Ohio Chapter/Air Force Chapters

Annual Abstract Competition

October, 2020

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(St. Elizabeth)

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Resident/Fellows Clinical Vignette

Abstracts 1-142

Unexplained CD4 lymphocytopenia in a non- HIV patient presenting as Diffuse Alveolar Hemorrhage

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Dr. Thomas Marnejon

Idiopathic CD4 lymphocytopenia (ICL) is defined as CD4+ T cell lymphocyte count of less than 300 cell per cubic millimeter or less than 20 % of total T cells without evidence of any condition known to lower the CD4 counts. While various infections, malignancies and medications are known to lower CD4 counts, HIV has been one of the most important cause. On the other hand, ICL represents a different subset of patients who are afflicted with an opportunistic infection without the presence of an identifiable immunosuppressive factor including HIV. This case report depicts a previously healthy man who presented with an opportunistic infection leading to the diagnosis of CD4 lymphocytopenia.

A 64- year old Hispanic male was seen at the ED due to a 2-week history of shortness of breath and pleuritic chest pain. Patient has recently returned from a vacation in Belize in Central America with his wife. He denied any associated symptoms of fever, chills, cough, sputum production, vomiting, diarrhea. Patient was a previous smoker, stopped 30 years ago. He denies any high-risk behavior nor hx of sexually transmitted disease and recurrent infection in the past. Initially, He also denied contact to other animals but later the wife mentioned about the patient's brief encounter with a bat during one of their cave explorations. CT scan of the chest showed extensive bilateral airspace diseases and mediastinal adenopathy. Labs revealed elevated ESR at 121, no leukocytosis and mild anemia with hgb at 8.3. Initial infectious work-up was unrevealing including negative HIV and antigens for legionella, streptococcus, histoplasma and cryptococcus. Immunoglobulin and the subsets were within normal levels but CD4 count was low at 275. Worsening respiratory status led to intubation. Bronchoscopy was done which showed diffuse alveolar hemorrhage. Final cultures from bronchial washing grew Cryptococcus Neoformans. He received Diflucan for 6 months. Repeat CD4 count normalize to 993 after 3 months of treatment.

Cryptococcus neoformans is a common infection to immunocompromised patient, most especially with AIDS/HIV patients. However, there has been number of diagnosed cases with low CD4 counts in the absence of HIV. From reports, Cryptococcal infections accounts to about 26.6% of the infections that afflict ICL patients. While the presence of Transient CD4 lymphocytopenia accounts to about 0.4- 4.1% of the healthy HIV- negative population, there has

been no proven cause that has led to this sequela. Most of cases are diagnosed after presenting with an opportunistic infection prompting an immunologic work-up. While there are noted normalization of CD4 counts in Transient CD4 lymphocytopenia, whether CD4 Lymphocytopenia recurs to some patients is still unknown.

Brugada syndrome, prolonged QT, and T-wave alternans? : a electrophysiologist's nightmare

Farouk Abu Alhana, Farouk Abu Alhana, Eric Kellett, Toshimasa Okabe
The Ohio State University Wexner Medical Center
David Wininger, MD

A 50-year-old man with a history of end-stage renal disease and hypertension presented with chest pain and palpitations while undergoing dialysis. He reported subjective fevers, abdominal pain, nausea, and vomiting for the past week. Shortly before presentation, he was tachycardic to the 110s and had a 30 second episode of polymorphic ventricular tachycardia with brief unresponsiveness that abated on its own. He was also noted to have a fever at 102 F. An amiodarone infusion was started and the patient was transferred to the emergency department.

On arrival, an ECG was obtained showing ST elevations in V1 and V2. He was taken urgently for cardiac catheterization, where he was found to have nonobstructive coronary artery disease.

While on the amiodarone infusion, a second ECG was obtained. Remarkably, his QTc had prolonged to approximately 750 ms. This prompted cessation of the infusion and initiation of a lidocaine drip. Importantly, this ECG also demonstrated macrovolt T wave alternans, a beat-to-beat variation in T wave amplitude. This ominous sign can often herald malignant ventricular arrhythmias (1).

Electrophysiology was consulted, who believed the patient to have a Brugada / prolonged QT overlap syndrome. In a type I pattern of Brugada syndrome (BrS), there are coved-type ST segment elevation of at least 2 mm followed by followed by a descending negative T wave in at least 1 right precordial lead (V1 to V3). A type II pattern ("saddle-back" type), however, is only suggestive of BrS. Patients affected by the disorder can have syncope, seizures, or nocturnal agonal breathing, the latter of which is believed to be secondary to polymorphic ventricular tachycardia or VF. Fever, as seen in this patient, is often a trigger in these affected (2).

Brugada / prolonged QT overlap is a rare phenotype of the cardiac sodium channel in which certain SCN5A mutations result in both gain- and loss-of-function at different phases of the action potential (3). Ultimately, given the diagnosis and high-risk features, this patient underwent placement of an ICD and referral for genetics counseling.

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Pleural effusion as the initial clinical manifestation of rheumatoid arthritis

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Introduction: Rheumatoid arthritis (RA) is a progressive destructive inflammatory disease characterized by symmetrical polyarthropathy of the small joints. It affects 1% of the population. Pulmonary complications occur in 60-80% of the cases and contribute to 10-20% of disease-related mortality. In this report, we review the case of a patient with cough and pleural effusion as the initial symptoms of underlying rheumatoid arthritis.

Case presentation: A 50-year-old Caucasian female with unremarkable past medical history presented with productive cough of 3-week duration, associated with mild shortness of breath, chills and low-grade fever. Review of symptoms were otherwise negative and vital signs were within normal limits. Physical examination was remarkable for decreased breath sounds over bilateral lower lung fields. Initial lab work including complete blood count and comprehensive metabolic panel were unremarkable. Chest X-ray showed bilateral pleural effusion. CT chest with contrast showed bilateral pleural effusions and centrilobular/paraseptal edema bilaterally. The patient was placed on high-flow oxygen and empiric IV ceftriaxone and azithromycin. She underwent left thoracenteses and chest tube placement. Pleural fluid analysis showed LDH 1517U/L, albumin 1.9g/L, total protein 4.2g/L, glucose <10mg/dL and pH of 7.0. Gram stain and culture were negative, and cell count showed a predominance of nucleated cells (1249/mL) with the following differential: 59% neutrophils, 14% lymphocytes, 25% macrophages and 2% monocytes. Pleural fluid showed no malignant cells, numerous leukocytic infiltrates and fibrous exudate. Further labs revealed ESR 117 mm/hour, CRP 22.9mg/dL, RF> 360IU/ml and anti-CCP >300U/ML. Antibiotics were discontinued, and the patient was diagnosed with rheumatoid arthritis. She was started on IV methylprednisolone which was transitioned to oral prednisone. Patient improved significantly, and chest tube was removed 3 days after placement. She was discharged on prednisone taper. Repeat chest X-ray two weeks following discharge showed resolution of her pleural effusions. Patient was treated with leflunomide as an outpatient.

Discussion: Rheumatoid pleural effusion occurs more commonly in men and is usually unilateral. It is rarely symptomatic and tends to occur concurrently with RA articular symptoms. Rheumatoid pleural effusion is a sterile exudative effusion characterized by low glucose, low pH, and elevated LDH. In our case, the presence of extremely low glucose in the pleural fluid in the absence of infection led to consideration of rheumatoid pleural effusion. Once diagnosis is made, patients respond very well to steroid therapy. Rheumatoid arthritis should be considered in the differential diagnosis for unexplained pleural effusion, even in the absence of articular manifestation.

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Hydrocephalus as a Presenting Feature in a Heroin User

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Introduction:
Inhalational heroin use, described as "chasing the dragon", leads to toxic leukoencephalopathy. Few cases of communicating and noncommunicating hydrocephalus after heroin use have been reported.

Case:
A 36-year-old female was brought to the ED after being found in a stranger's apartment. Past medical history was significant for bipolar disorder and polysubstance abuse. She received 8 mg of Narcan en route without improvement. GCS was 7 leading to emergent intubation. Labs showed mild transaminitis and elevated troponin. EKG showed T wave inversion in leads II, III, aVF, and V1-V4. UDS was positive for opiates and cocaine. Head CT revealed intracranial edema and hydrocephalus. She received decadron, and neurosurgery was consulted. Brain MRI revealed significant cerebellar edema, leading to tonsillar herniation and severe obstructive hydrocephalus. She underwent immediate ventriculostomy, with minimal change in neuro exam over the next two days. On the third day, she demonstrated decerebrate posturing on external stimuli. Over the next few days, she started opening her eyes spontaneously and following commands intermittently. By the end of the first week, she was fully following commands. On the tenth day of admission, she underwent VP shunt, followed by successful extubation. Imaging repeated after VP shunt showed marked improvement in ventricular volume with complete decompression of ventricular. On follow up three months after discharge, she exhibited residual headache, mild dizziness, increased thirst, and right Bell's palsy, but retained independent functional status.

Discussion:
Leukoencephalopathy refers to progressive white matter brain damage, particularly myelin. Since the advent of inhalational heroin use, many cases of toxic leukoencephalopathy have been described. Typical imaging illustrates symmetrically increased T2 and T2-FLAIR signal intensity of the cerebellar and posterior cerebral white matter. Posterior limb of the internal capsule is often affected, but the anterior limb and dentate nuclei are spared. Gray matter, extremely susceptible to hypoxic injury, is relatively spared. Our patient's brain MRI revealed diffusion restriction and severely decreased T1 and increased T2 signal within the bilateral cerebellar hemispheres with effacement of the cerebellar folia. Tonsillar herniation was present, leading to severe obstructive hydrocephalus. Additionally, bilateral acute cerebellar infarcts with evidence of laminar necrosis and 2 cm hemorrhage in the posterior right temporal lobe was noted. CTA excluded extra/intracranial vessel obstruction as a cause of the patient's brain imaging findings. Abrupt presentation leading to hydrocephalus after heroin use is rare.

Minimizing the Chance of Crisis - Diagnostic Reasoning in a Case of Multiple Malignancies

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Introduction: Here we present a unique case of a patient simultaneously diagnosed with two unrelated malignancies. Effective clinical decision making ensured that the chance of a potentially life-threatening misdiagnosis was minimized. The successful work-up illustrates the utility of a systematic approach to diagnostic reasoning when confronted with an unclear clinical picture.

Case Description: The patient was a 63-year-old female with a past medical history of CKD stage 4 and childhood Wilms tumor of the right side treated with surgery and radiation. She presented to the ED with acute abdominal pain located in the right upper quadrant. CT abdomen/pelvis showed a large right lobe liver mass.

The severity of her CKD contraindicated contrast imaging, so a PET scan was performed. The scan showed a large hypermetabolic liver mass and an incidental right breast mass. No lymphadenopathy or other sites of possible metastasis were seen. Colonoscopy and endoscopy were negative. Biopsy of the liver mass indicated an adenocarcinoma, likely cholangiocarcinoma.

Bilateral mammogram and ultrasound of the right breast both corresponded with PET findings. Right breast biopsy showed mammary carcinoma, confirming the presence of a second primary malignancy.

Discussion: The diagnostic work-up of this patient was greatly complicated by the incidental breast mass, as a missed diagnosis of another primary malignancy could lead to significant morbidity for the patient. Analysis of the final steps taken in making the diagnosis provides a demonstration of how a systematic method of diagnostic reasoning was employed to avoid such an outcome.

Beginning with the initial finding of an incidental breast mass in the setting of a confirmed liver malignancy, two potentially life-threatening differential diagnoses for the mass included an unrelated primary malignancy versus a related metastatic malignancy. The management decision to be made concerned the necessity of obtaining a biopsy of the mass. The potential cost of an unnecessary biopsy had to be weighed against the potential cost of missing a critical diagnosis.

Since an unrelated primary malignancy would require a drastically different treatment plan, any additional finding that would increase its likelihood would favor obtaining the biopsy. Close inspection of the patient's history revealed that she had received radiation treatment for a Wilms tumor as a child, on the same side of her body as the breast mass. This deceptively important finding made biopsy the clear decision.

While the use of systematic diagnostic reasoning must be coupled with the clinical judgment of an experienced clinician, proper implementation of this type of approach can serve to maximize the efficient use of healthcare resources and minimize unnecessary interventions that can cause increased morbidity. In this case specifically, it successfully reduced the chance of missing a diagnosis that could have potentially cost the patient her life.

CONGENITAL ANEURYSMAL CORONARY ARTERY TO PULMONARY ARTERY FISTULA PRESENTING AS NON-ST ELEVATION MYOCARDIAL INFARCTION

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INTRODUCTION: Anomalies of the coronary vessels such as a coronary artery fistula are rare, and typically asymptomatic. They are often detected incidentally on imaging such as coronary angiography. When symptomatic, patients could present with potentially life-threatening conditions that require immediate interventions. This is the case of a woman who was managed for NSTEMI, and coronary artery fistulae and coronary aneurysms were found incidentally on imaging. We describe her clinical course, imaging findings and management. We also highlight the reasoning behind specific management in this patient.

CASE: A 74-year old woman with a medical history of hypertension and hyperlipidemia presented to our emergency department complaining of typical chest pain which was associated with exertional shortness of breath. Upon presentation, vital signs were stable. On physical exam, she had a 3/6 systolic ejection murmur over the precordium.

Initial Troponin I was 0.026 ng/ml which was within range for our facility. After 2 hours, Troponin I increased out of normal range to 1.639 ng/ml, with creatinine of 1.0 mg/dL. EKG demonstrated sinus rhythm without ST segment or T wave changes. We performed early coronary angiography and found a non-obstructive 60% stenotic LAD with an aneurysmal and fistulous septal branch draining into the pulmonary artery.

We obtained a Coronary CT-angiogram for better characterization of the aneurysmal fistula of the septal branch of the LAD.

Echocardiogram demonstrated left ventricular systolic ejection fraction of 52.7%. We recommended percutaneous management with coiling of the aneurysm. She was clinically asymptomatic prior to discharge on Aspirin, Clopidogrel, Atorvastatin, and Metoprolol.

DISCUSSION: Coronary artery fistulae are very rare. They are mostly found incidentally on coronary angiography. Incidence ranges from 0.1% to 0.2% on coronary angiography, to 0.9% on a coronary CT-Angiography. Most cases arise from the right coronary artery, and they may drain into a cardiac chamber or into one of the great vessels. Morbidity has been described in children but rarely in adults, and when diagnosed, most adult patients are asymptomatic. Etiology can be congenital, post-infectious or postsurgical.

Symptoms could include chest pain, cardiac murmur, shortness of breath and heart failure due to steal syndrome. Management is typically reserved for symptomatic patients, as well as asymptomatic patients with high-flow shunting, and those at risk for complications such as endocarditis, aneurysm, thrombosis dissection, pulmonary hypertension and myocardial ischemia. Management could be surgical or percutaneous, based on a patient's age, comorbidities, and preferences. Our patient opted for outpatient percutaneous management with coiling, due to the patient's age and recurrent symptoms.

In conclusion, this case demonstrates a rare congenital anomaly of the coronary artery, its presentation, imaging findings and management.

Cirrhotic Liver: Putting the "Brakes" on Death

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Introduction: Methanol is a highly toxic alcohol, readily available in automotive solvents and solutions. Methanol poisoning occurs mostly from ingestion but also transpires from inhalation and skin absorption. Methanol is converted to a highly toxic metabolite that is responsible for metabolic acidosis, visual disturbances, neurologic dysfunctions and even cardiac arrest. Diagnosis is difficult as most victims of ingestion are unable to provide an accurate history. Furthermore, the clinical presentation can vary depending on the patient's comorbidities. Therefore, a high clinical suspicion and prompt lab testing are essential in order to avoid delay in therapy.

Case Presentation: A 51-year-old man with alcoholic cirrhosis presented to the emergency department 12 hours after inhalation of "O'Reilly" brake fluid cleaner. He had been abusing the cleaner as an inhalant for 3 months. He was asymptomatic on presentation but found to have elevated serum osmolality with high osmolal gap (OG). Serum anion gap (AG) was not elevated, and the patient did not have any signs of end-organ damage. Volatile panel reported methanol level of 50.6mg/dL (toxic >20 mg/dL). The patient was started on intravenous fomepizole and high-dose folic acid. Hemodialysis was not required given the lack of clinical indication or toxicity. Fomepizole was continued until methanol levels were <25 mg/dL. The patient had an uncomplicated hospital course and was soon discharged home in stable condition.

Discussion: Methanol is metabolized to formaldehyde and formic acid mainly in the liver. Accumulation of the formic acid is responsible for development of symptoms. Patients with underlying liver disease are unable to efficiently convert methanol to its toxic metabolite. Hence, these patients may not show any signs of clinical manifestations or end-organ damage. Methanol is responsible for the elevated OG while formic acid is responsible for the elevated AG. Consequently, patients with advanced liver disease may have a normal AG. However, the standard of care remains the same for all. A serum methanol > 20 mg/dL generally requires treatment. This includes competitive inhibition with fomepizole or ethanol along with removal of the toxic metabolites with hemodialysis if needed. Our patient, despite methanol levels well above the toxic threshold, did not have an elevated AG or clinical sequalae. We hypothesize that impaired hepatic metabolism due to advanced liver disease attenuated the conversion to formic acid responsible for the elevated AG and clinical sequelae.

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LEFT VENTRICULAR NON COMPACTION AND ASSOCIATED CARDIAC ABNORMALITIES

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Introduction: Left ventricular non compaction (LVNC) is considered a rare form of cardiomyopathy with the prevalence estimated between 0.014% to 1.3% in patients undergoing echocardiograms. While it was initially described as an isolated disease, there is emerging data that shows its association with other congenital abnormalities. We wish to emphasize on the associations of LVNC

Case description: 47-year-old male with past history of hypertension, diabetes and coronary artery disease presented with symptoms of congestive heart failure including shortness of breath and orthopnea and for 3 days. His family history was significant for sudden cardiac death in father at age 47 years. Physical exam showed raised JVD with positive hepatojugular reflux, 3+ pitting edema and bibasilar crackles. His labs were significant for BNP of 4911, troponin value of 0.6, BUN 31 and Creatinine of 1.74. Transthoracic echo showed depressed left ventricular systolic function and moderate to severe aortic regurgitation and a transesophageal echocardiogram (TEE) was ordered to further evaluate the valvular pathology. TEE showed left ventricular ejection fraction of 25-30% and prominent trabeculations in the left ventricle and a ratio of non compacted to compacted myocardium more than 2, and deep intertrabecular recesses. He was also noted to have moderately thickened and mildly calcified bicuspid valve with fusion of right and left commissures and moderate eccentric aortic regurgitation. Cardiac MRI confirmed the findings on echocardiogram and patient met diagnostic criteria for LVNC Patient was managed with guideline directed medical therapy.

Discussion: Left ventricular non compaction is increasingly found to be associated with other congenital cardiac abnormalities such as Ebstein anomaly, tetralogy of fallot, bicuspid aortic valve (BAV) and aortic coarctation. In addition it has been seen in association with skeletal myopathies like Becker's muscular dystrophy and Friedreich's ataxia. In a few retrospective studies, LVNC was seen in 2.8% to 11% of patients with BAV. Our case highlights the complex associations between non compaction and other congenital cardiac pathologies. Since BAV is a common congenital abnormality and has a prevalence of 1% of general population, it is also prudent to carefully look for LVNC in patients with BAV.

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Early Detection of Acute Cardiac Injury after Anthracycline-administration using Novel Cardiac Magnetic Resonance Imaging

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Case Presentation: While late-onset anthracycline induced dilated cardiomyopathy is well known, acute cardiotoxicity after anthracycline based-therapy is rare. The following is a case of acute inflammatory cardiomyopathy presenting as an accelerated idioventricular rhythm only hours after doxorubicin administration for diffuse large B-Cell lymphoma (DLBCL).

A 56 year old male with a history of sickle cell trait, HLD, CVA, recently diagnosed aggressive DLBCL and gastric cancer presented with de novo accelerated idioventricular rhythm on Cycle 1 Day 5 of EPOCH-R (Etoposide, Doxorubicin, Vincristine, Rituximab, Cyclophosphamide, Prednisone). He had several such episodes, lasting several minutes at a time over the following 24 hours. He denied chest pain, dyspnea, palpitations, and edema. He had no prior cardiac history. A week prior to initiation of chemotherapy, an echocardiogram showed normal ventricular function no regional wall motion abnormality and no evidence of valve disease. He denied family history of cardiac disease. He did not report any tobacco, alcohol or substance use.

A cardiac magnetic resonance imaging (CMR) was obtained for suspicion of acute toxicity secondary to his chemotherapy. CMR showed a dilated left ventricle with normal wall thickness and a left ventricular ejection fraction (LVEF) of 55%. However, there was diffusely elevated T2 signal in the myocardium, consistent with profound myocardial edema /inflammation as well as subepicardial fibrosis of the basal inferolateral left ventricle. Despite normal LVEF, these CMR-derived myocardial tissue changes findings were highly suggestive of acute drug-related inflammatory cardiomyopathy. He was initiated on beta-blocker and amiodarone, while anthracyclines were held for a few weeks to allow senescence of his myocardial injury. He was referred to cardio-oncology clinic and will initiate a less cardiotoxic regimen without doxorubicin, while maintaining close monitoring.

Discussion: Despite normal baseline cardiac function by echo and clinical assessment, vigilance should be given to the potential for acute change after administration of potentially cardiotoxic therapies. It can be seen in treatment naïve patients and can present with acute heart failure or any form of arrhythmia, including malignant ventricular arrhythmias. In this case, the use of CMR with T1/T2 and fibrosis imaging allowed for more clear guidance in the diagnosis and management of incident cardiotoxicity prior to a change in LVEF that would traditionally be seen on an echocardiogram with late onset cardiomyopathy in the setting of anthracycline use.

Conclusion: Acute clinical cardiotoxicity after anthracyclines is rare, but may be life-threatening. Early detection is associated with improved outcomes. Prompt selection of appropriate cardiac assessment tools such has CMR may prove critical to improving outcomes among those at risk for cardiotoxic and potentially life threatening events secondary to anthracyclines.

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Light chain amyloidosis presenting as gastroparesis and weight loss

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Introduction: Delayed gastric emptying, feeling of fullness, and weight loss can be the result of various causes such as gastroparesis secondary to autonomic dysfunction from diabetes mellitus, or mechanical causes such as ulcers or strictures. We present a rare case in which the initial presentation was that of gastroparesis and weight loss. Subsequent extensive investigations revealed amyloid light chain amyloidosis as the cause of our patient's symptoms. While amyloidosis is known to affect the gastrointestinal system, rarely do patients present with gastrointestinal symptoms as their first symptoms (<1%).

Case Description: A 49-year-old Caucasian male presented with early satiety, fatigue, and weight loss of approximately 30 pounds in 6 months. His medical history was only significant for hypertension, hyperlipidemia, and mild asthma. Initial imaging and endoscopy did not reveal any mechanical cause of obstruction or stricture. Both proximal and distal esophageal biopsies were negative. Our patient subsequently developed symptoms of severe orthostatic hypotension which prompted investigations for possible autonomic dysfunction. MRI of the brain revealed multiple focal hyperintensities in the white matter concerning for multiple sclerosis, vasculitic processes, toxic metabolic processes, infectious, paraneoplastic, or infiltrative disease. Extensive infectious and paraneoplastic workup was negative. Echocardiogram revealed posterior septal wall thickening. On CT imaging axillary adenopathy was noted and biopsy was performed in which no malignancy was found. Patient subsequently developed multiple post-biopsy hematomas requiring repeated surgical treatment. Subsequent CT of abdomen and pelvis demonstrated large perirectal hematoma without etiology. Serum protein electrophoresis showed elevated kappa and lambda light chain immunoglobulins suggestive of infiltrative disease. Abdominal fat pad biopsy stained positive with Congo red stain. Bone marrow biopsy was completed showing plasma cell abnormalities confirming amyloid light-chain amyloidosis. Gastric emptying study performed later confirmed severe gastroparesis. Our patient was started on Bortezomib with eventual goal of bone marrow transplant.

Discussion: Although extremely rare, gastrointestinal symptoms can be the initial presenting symptoms of Amyloid-light chain amyloidosis. If no other cause of these symptoms can be identified in early evaluation, internist and gastroenterologist should consider light-chain amyloidosis in light of other systemic symptoms such as autonomic dysfunction, unexplained proteinuria, bleeding abnormalities, or unexplained cardiomyopathy as earlier diagnosis of this disease can lead to better outcomes.

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Case Report: IFNβ causing Focal segmental glomerulosclerosis in Multiple Sclerosis patient

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Interferon β is the first-line treatment of relapsing-remitting multiple sclerosis. Side effects are both dose-dependent and idiosyncratic toxicity. There have been rare reports of Interferon β causing Acute Renal Failure and Nephrotic Syndrome, including Focal Segmental Glomerulosclerosis.

Our patient is a 53-year-old Caucasian female with a past medical history of Multiple Sclerosis diagnosed 12 years ago, medically treated with IFN-β. She presented to The Nephrology clinic as her routine labs showed elevated serum creatinine. She had no new complaints. Physical examination was significant for bilateral ankle swelling. Lab work was significant for elevated serum creatinine, elevated blood urea nitrogen, decreased albumin, and elevated total cholesterol.

Urinalysis showed heavy proteinuria (protein/creatinine ratio 4.8g/g), and albuminuria (albumin/creatinine ratio 3.2g/g).

Renal Ultrasound revealed increased cortical echogenicity in the right kidney. The patient subsequently underwent renal biopsy, which confirmed glomerulopathy with moderate glomerulosclerosis, segmental foot effacement, renal tubular injury, and microvillus changes. Due to the absence of classical risk factors for Focal Segmental Glomerulosclerosis, a presumptive diagnosis of medication-induced Focal Segmental Glomerulosclerosis was made. The offending drug was stopped and the patient was started on Steroids and Immunomodulators. She had complete recovery of her kidney function within 6 months.

This case represents a rare adverse effect of INF-β, the mechanism interferon β induces kidney damage is mediated by impairing the charge barrier of the glomerular basement membrane due to an interaction between its positive charge and the negatively charged glomerular basement membrane. Physicians should draw attention to symptoms and findings of nephropathy during the management of patients under treatment of IFN-β, as routine follow-up labs do not include renal function test and urinalysis.

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Not So Benign Early Repolarization Syndrome: A Case of Sudden Cardiac Death

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Sudden cardiac death (SCD) is so called due to its unexpected nature and the absence of other obvious causes of death. There are certain known syndromes which increase an individual's risk of SCD. Early repolarization (ER) is one of such syndromes. It is rare and usually benign, making it a particularly difficult clinical entity to recognize and manage.

A 21 year old gentleman presented following cardiac arrest. He was seen in ED earlier that day due to altered mental status and seizure-like activity. During his initial evaluation, the physical exam and workup were unremarkable except for J-point elevation on his EKG. He was discharged home with an outpatient referral to neurology. Later that day, he lost consciousness and was found unresponsive by his girlfriend. On EMS's arrival he was in PEA arrest which later converted to Ventricular Fibrillation during ACLS. The patient was subsequently intubated and transferred to the ED.

In the ED, he was profoundly acidotic with pH 6.48 and pC02 144. The ET tube was replaced with improvement in his ABG. Repeat EKG showed normal QT duration and again, J-point elevation. CT head and chest done at the time revealed diffuse anoxic brain injury and bilateral pulmonary edema with RLL infiltrate respectively. After ROSC was achieved, the patient was transferred to the ICU for hypothermia protocol and further management. Neurological testing after rewarming was unfortunately positive for brain death.

He had ED visits earlier in the year for chest and abdominal pain. This resulted in an extensive cardiac work up including TTE and a left heart catheterization with unremarkable results. He had no family history of SCD or premature MI. Post-mortem genetic testing and molecular panels for Brugada and Long QT were negative. Limited autopsy demonstrated borderline cardiomegaly with concentric LVH. A detailed analysis of the case points to ER as the cause of death in this young man.

ER pattern is diagnosed on EKG as a sharp positive deflection ($\geq$1 mm in $\geq$2 contiguous leads) at the beginning of the ST segment. If this pattern is seen in a patient with history of unexplained Ventricular Fibrillation, polymorphic Ventricular Tachycardia, or syncope, ER syndrome is diagnosed. Currently the guidelines regarding ER recommend ICD only for secondary prevention of SCD after surviving ventricular arrhythmia. However, there is sometimes no second chance. Often it is the history of episodes of syncope, "seizure-like" episodes, family history of SCD that are the tell-tale signs of possible fatality. By highlighting cases in which ER leads to SCD, it is hoped that this traditionally regarded as benign EKG abnormality will prompt detailed history taking and change in guidelines leaning towards primary prevention of SCD in these individuals.

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A very uncommon case of Myxedema coma: Rediscovery of an old presentation

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Myxedema coma is defined as severe hypothyroidism leading to decreased mental status, hypothermia, and other symptoms related to slowing of function in multiple organs. The term is largely a misnomer because quite a few patients are obtunded, rather than truly comatose. It is an uncommon but potentially lethal condition. Even with early detection and appropriate treatment, mortality ranges from 30 to 60 percent. Overt congestive heart failure is quite rare in the absence of preexisting cardiac disease. We describe a woman with myxedema coma, who developed respiratory failure, congestive heart failure as well as pericardial and pleural effusions, some of the uncommon manifestations.

Our patient is a 81 year old Afican-American female with a past-medical history of hypertension, transient ischemic attack, dementia, primary hypothyroidism and vitamin D deficiency presented with a one-day history of worsening shortness of breath(SOB). History was taken from the patient's husband. She developed altered mental status and became mute the week prior to presentation. She was unable to feed herself, consume medications or walk independently during that week. She was also noted to have facial swelling and lower limb edema.
On initial examination, the patient was vitally stable with the exception of hypothermia where her temperature was recorded to be 34.7 degree celsius. She was alert but disoriented and non-communicative. She was found to have facial puffiness, elevated jugular venous pressure, bilateral coarse crepitations on auscultation of the lung, muffled heart sounds and cool extremities with 3+ pitting edema in bilateral lower limbs. The patient soon developed cardiogenic shock with respiratory failure requiring intubation and pressor support.
Lab investigations revealed TSH of 103 uIU/mL, free T4 of 0.11 ng/dL, free T3 <0.5 pg/ml and random free cortisol of 68 ug/dL. Other labs showed no leukocytosis but bands of 8%, bicarbonate 18 mm/L, creatinine 1.94 mg/dL, BUN 38 mg/dL, ALT 136 U/L, AST 303 U/L, troponin 0.06 ng/ml. Chest X-ray revealed vascular congestion, cephalization and cardiomegaly. EKG revealed T-wave inversion in lateral leads and prolonged QT. Transthoracic echocardiogram revealed an ejection fraction of 25-30%, dilated cardiomyopathy, mild pericardial effusion and left pleural effusion.

Diagnosis of Myxedema coma with cardiopulmonary failure was subsequently made. Patient was managed in the ICU. She received IV levothyroxine, steroids and appropriate cardiopulmonary support. Patient gradually regained consciousness and was successfully extubated where her blood pressure was maintained off pressors and was eventually discharged. We present this case to emphasise the importance of early diagnosis and management of an almost forgotten and potentially fatal condition.

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Uncovering the true diagnosis: aortic stenosis masquerading as asthma exacerbation

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Syncope related to structural heart disease is associated with higher rates of sudden cardiac death and all-cause mortality when compared to syncope due to non-cardiac causes. In a 2013 study of patients with severe aortic stenosis (AS), roughly 3% of patients had symptoms of dizziness or syncope. It is therefore important to accurately identify the cause of syncope or arrest when there is an underlying structural cardiac abnormality.

A 40-year-old male presented to the emergency room after collapsing while pushing a vehicle out of an intersection. In the ER the patient was unresponsive, cyanotic, and had a questionable pulse. CPR was initiated and naloxone was given. Return of spontaneous circulation was achieved; however, he continued to have depressed mentation and was ultimately intubated. The patient had a medical history of hepatitis C, IV drug use (presently on methadone), and asthma. Of note, the patient had been hospitalized multiple times over the prior two months for asthma exacerbations. No mention of cardiac murmur was made on initial documentation, however, bilateral wheezes were noted on lung exam.

The patient was transferred to the ICU with the working diagnosis of acute hypoxic respiratory failure secondary to asthma exacerbation. CT head was negative for any acute pathology. CTA chest did not show evidence of PE but did note ectasia of the ascending thoracic aorta. The patient's respiratory status and level of consciousness improved, and he was extubated less than 24 hours after intubation. Given the evidence of thoracic aortic ectasia, a transthoracic echocardiogram was performed. The patient was found to have moderate aortic stenosis (AS) with a congenital bicuspid aortic valve, and moderate aortic arch dilatation with coarctation of the aorta. A transesophageal echocardiogram was performed, which showed severe AS with a valve area of 0.8 cm2. An MRA showed no evidence of aortic coarctation but confirmed aneurysmal dilatation of the proximal aortic arch. The patient underwent right and left cardiac catheterization, which confirmed AS and aortic aneurysm. The patient successfully underwent bioprosthetic aortic valve replacement along with an ascending transverse aortic arch replacement.

It is likely that the patient's syncope and brief cardiopulmonary arrest were in fact provoked by severe AS in combination with the physical exertion of pushing a stalled car. A careful review of the patient's medical record revealed an echocardiogram performed two years prior that identified a bicuspid aortic valve without evidence of significant AS. This case emphasizes the importance of accurately identifying the underlying etiology of syncope and arrest. It also highlights the significant sway of anchoring and framing effect which led to the initial diagnosis of asthma exacerbation. This case underscores the value of a well thought out differential, a thorough history, and an awareness of cognitive biases.

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Pembrolizumab-induced thyroiditis

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Introduction: Pembrolizumab is a humanized monoclonal antibody that acts on T cell programmed death receptor-1 (PD-1) resulting in activation of T-cell mediated response and destruction of malignant cells. This immunotherapy is implemented alone or in combination with other medicines to treat certain cancers, such as melanoma, lung, cervical, esophageal, and others. Administration of pembrolizumab is associated with immune-related adverse events (irAEs). Thyroid irAEs have a reported incidence of 3.2-10.1 % in patients receiving pembrolizumab. We present a case of pembrolizumab-induced thyroiditis in a patient with esophageal adenocarcinoma.

Case Presentation: A 54-year-old Caucasian female with past medical history of nicotine dependence, ulcerative colitis, hypothyroidism on levothyroxine, and stage IV poorly-differentiated distal esophageal adenocarcinoma presented to the emergency department with generalized weakness and confusion. On presentation she was lethargic and oriented to person and place but not time. Review of symptoms was positive for dizziness, lightheadedness, nausea, and anxiety. Physical exam was notable for cachexia and bilateral upper extremity resting tremor. No exophthalmos, thyromegaly, thyroid nodules or thyroid tenderness noted on thyroid exam. Vital signs were BP 116/79, pulse 133, RR 20, temperature 98.2° F, SpO2 99% on room air. Labs showed WBC 3.3, TSH 0.01 [0.49-4.67], free T4 3.01 [0.61-1.60], thyroglobulin 229.1 [0-35 ng/ml], free T3 3.01 [2.50 - 3.90 pg/mL], cortisol 21.2 (normal). EKG showed sinus tachycardia with nonspecific T-wave changes and QTC 432. CT chest showed esophageal stent with esophageal dilation, a 6-7mm pulmonary nodule in the right upper lobe, and multiple densely calcified nodules in the left lower lung. CT brain and CT abdomen/pelvis showed no acute pathology. She was admitted to the Internal Medicine service and Endocrinology was consulted. Thyroid ultrasound showed diffuse heterogeneous echotexture consistent with thyroiditis. Thyroid-stimulating hormone receptor (TSH-R), thyroid peroxidase (TPO), thyroglobulin, and thyroid-stimulating antibodies (TSI) were normal. T4 down trended as levothyroxine was held throughout the hospital stay.

Discussion/Conclusion: The patient's elevated T4 with elevated thyroglobulin makes iatrogenic hyperthyroidism unlikely; patient's family also reported appropriate levothyroxine compliance. Total T3:T4 ratio was >20 and free T3: free T4 ratio > 0.3, which is consistent with Graves' disease however, TSHR Ab was negative, and the thyroid ultrasound did not show hyperemia, instead supported destructive thyroiditis. The downtrend of T4 during hospital stay favors acute thyroiditis. As the patient was hypothyroid prior to admission and presented with hyperthyroidism in the setting of pembrolizumab use, Endocrinology attributed the hyperthyroidism to pembrolizumab-induced thyroiditis. The patient was treated symptomatically with metoprolol tartrate for tachycardia, and levothyroxine was held until outpatient Endocrinology follow-up. The patient was discharged in stable condition.
Pembrolizumab is known to have secondary side effects including thyroiditis. Active surveillance will help in the prevention, early detection, and treatment of future thyroid-related irAEs.

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Typhlitis as a Complication of Pasturella bacteremia in a Non-Neutropenic Patient

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Introduction: Typhlitis is a neutropenic enterocolitis whose pathology is poorly understood; it is most common in individuals with hematologic malignancies who are neutropenic. It occurs with various bacteria/fungal infections. Microbial infections lead to invasion and necrosis of intestinal layers. The most affected area of the bowel is the cecum, extending into the ascending colon and terminal ilium. On histology, inflammatory infiltrates are rarely identified. Typhlitis can be caused by bacteremia or fungemia, common pathogens include P.aeruginosa, E. coli, Candida, bacteroides spp, enterococci. Here, we report typhlitis secondary to Pasturella bacteremia in a non-neutropenic patient

A 66-year-old male with history of chronic lower extremity lymphedema, squamous cell carcinoma of the hypopharynx in 2018 with unknown treatment history was admitted to the hospital for lower extremity cellulitis and sepsis. On further investigation, the patient was found to have a dog that has licked an open wound on his right leg. Blood cultures were positive for Pasturella bacteremia. Two days after admission, the patient was leukopenic with WBC of 1.2 103/uL, etiology could not be determined. However, he developed leukocytosis with neutrophil predominance within two days. A CT abdomen/pelvis was done on admission that was negative for acute findings. 7 days after admission, patient was noted to have abdominal tenderness and constipation, repeat CT abdomen pelvis showed circumferential wall thickening in cecum, poorly distended inflammatory or infectious process with findings suggestive of Typhlitis. He was found to be a poor candidate for surgery due to other medical problems. He was started on antibiotics and underwent multiple changes in his antibiotic regimen. After antibiotic treatment and suppository, patient had a bowel movement, a repeat CT abdomen/pelvis 17 days later was negative for previous findings.

Conclusion: Typhlitis has a high mortality rate 50% or higher secondary to bowel necrosis, perforation or sepsis. Early recognition is key in reducing mortality rates. Typhlitis is usually suspected on neutropenic patients, however, this case highlights that it should be considered in patients with normal WBC count as well. Further research is needed on this topic to determine severity of typhlitis in immunocompetent vs. immunocompromised patients.

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Don’t Forget: Cerebral Amyloid Angiopathy as a Rare Cause of Subacute Decline in Cognition

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Introduction: Cerebral amyloid angiopathy (CAA) is caused by the deposition of amyloid beta peptides within small to medium-sized vessels involving the brain and leptomeninges. A common presentation of CAA is spontaneous lobar hemorrhage in elderly adults. Additionally, CAA is a known contributor to cognitive decline in patients with Alzheimer’s dementia (AD). In fact, previous autopsy studies have revealed the prevalence of CAA in Alzheimer’s disease to be between 70-90%. However, isolated CAA is rarely reported as a cause of progressive neurodegeneration and cognitive dysfunction in patients not previously diagnosed with dementia.

Case Presentation: An 82-year old male with a history of hypertension, coronary artery disease, atrial fibrillation, and type 2 diabetes mellitus presented for planned surgical resection of a left parietal brain mass. Prior to admission, he had a five-month history of ataxia, blurry vision, and intermittent confusion. Outpatient MRI brain imaging diagnosed a brain mass. He had no evidence of underlying dementia. In the post-operative course, he developed hyperactive delirium, requiring frequent re-orientation and scheduled low-dose antipsychotics. He also developed seizures during admission requiring antiepileptic therapy. Surgical pathology reported that the brain mass was cerebral amyloid angiopathy without evidence of malignancy or tau immunoreactivity. Neurology was consulted and recommended further evaluation at an outpatient dementia center. The patient’s mental status improved slightly throughout admission, but he did not recover baseline cognition before discharge.

Discussion: The prevalence of CAA is markedly age dependent. It can be identified pathologically in up to 8% of brains from individuals between the ages of 75 and 84. There is a strong association between CAA and Alzheimer’s dementia, but other proposed mechanisms of cognitive decline in patients with CAA are seldom reported. CAA-related inflammation (CAARI) is a distinct small subset of CAA. The clinical syndrome of CAARI is distinguished by subacute neurobehavioral symptoms, headaches, seizures, and stroke-like signs. Imaging of CAARI may show findings that are difficult to distinguish between cerebral neoplasm. Thus, CAA should be kept on the differential for subacute cognitive decline in previously functional patients with new lesions on brain imaging.

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Acute Pericarditis as an Initial Presentation of COVID-19

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Introduction: Acute pericarditis has various etiologies. Viruses are one of the most common causes of acute pericarditis, with coxsackievirus and echovirus being the most common viral agents. Coronavirus disease 2019 (COVID-19), caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has become a challenging global outbreak. Data regarding cardiovascular involvement due to SARS-CoV-2 infection are less described in the literature. We report a case affected by COVID-19 who presented with cardiac involvement in the absence of respiratory tract signs and symptoms.

Case presentation: A 25-year-old African American male with no significant cardiovascular history presented with fever, tachycardia, and bilateral non-purulent conjunctivitis. The patient denied any cough or shortness of breath. Initial lab work revealed ferritin 891 ng/mL, C-reactive protein 16.6 mg/dL, D-dimer 734 ng/mL, LDH 269 U/L, mild transaminitis and leukocytosis, normal troponin levels and negative respiratory pathogen panel and Monospot test. CT angiogram of chest ruled out pulmonary embolism. The chest X-ray was unremarkable. Electrocardiogram showed widespread ST elevation with PR depression consistent with acute pericarditis. The patient was eventually started on colchicine and ibuprofen. On day 2 of admission, the patient tested positive for SARS-CoV-2. During hospitalization, he developed chest discomfort. Repeat cardiac enzyme levels were normal. Transthoracic echocardiogram was unremarkable except for a trivial pericardial effusion. The patient was discharged on colchicine and ibuprofen after the fever has been resolved and a negative swab for COVID-19.

Conclusion: We describe a case of COVID-induced acute pericarditis. This case highlights acute pericarditis as a complication associated with COVID-19, which might be underdiagnosed during this pandemic. Our main conclusion from this case is to emphasize on the fact that COVID-19 may present with cardiac involvement even in the absence of respiratory tract signs and symptoms of infection. Therefore, EKG and troponin need to be considered in the evaluation of COVID-19 patients. Physicians should be aware of atypical presentations of COVID-19, such as acute pericarditis, which is crucial to detect and isolate patients with COVID-19 early.

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Autopsy confirmed pulmonary tumor emboli in the setting of metastatic breast cancer.

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Background: Pulmonary tumor emboli are likely under-diagnosed, with an estimated incidence of 3-26% of patients with cancer, and rarely diagnosed antemortem. We present a case where diagnosis was suspected and confirmed post-mortem.

Case Description: A 55 year old female with a history of triple negative, inflammatory stage IIIb breast cancer presented to the ED with dyspnea on exertion for two weeks. Cancer was diagnosed 14 months prior and she underwent neoadjuvant therapy, a right sided modified radical mastectomy, left total mastectomy and subsequent chest wall radiation. She had been traveling extensively and hiking up to 8 miles without issues the day prior to presentation. Additional complaints included bloating and abdominal fullness. CT scan of the chest was notable for a new streaky nodular opacity in the right upper lobe concerning for atypical pulmonary metastatic disease . CT of the abdomen and pelvis was notable for extensive hepatic metastatic disease and hepatomegaly. CT guided liver biopsy revealed metastatic carcinoma consistent with breast primary. Hypoxia worsened, requiring up to 6L via nasal cannula. CT pulmonary angiogram (CTPA) was performed and showed a non-occlusive sub -segmental thrombus of the left upper lobe. Anticoagulation was started;
patient then noted to have hemoptysis and anticoagulation was stopped and reversed. Hypoxia continued to worsen, requiring BiPAP. She was transferred to the ICU.
Echocardiography revealed a markedly dilated right ventricle and flattening of ventricular septum, new from 4 days prior. This raised concern for propagation of the pulmonary embolism. Repeat CTPA did not show any new thromboembolism with interval reduction in previously noted filling defect. Given the history of cancer, suspicion was for pulmonary tumor emboli. Condition deteriorated requiring intubation followed by cardiac arrest and death.

Autopsy was performed and showed a markedly enlarged liver weighing 3940 g extending 10 cm below the costal margin. Microscopic examination was notable for scattered tumor emboli in pulmonary vasculature in all sections of the lung examined. There was also right upper lobe lung metastasis noted. Microscopic examination of liver demonstrated geographic areas of replacement by metastatic carcinoma with focal necrosis.

Discussion: Although rare and likely under diagnosed, pulmonary tumor emboli should be considered in patients with dyspnea and hypoxemia, not responsive to common interventions. Although there are little management options, diagnosis provides option for palliative measures and prognosis is very poor with median survival around 3 days.

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Chordal Rupture with a Lesser Degree of Mitral Regurgitation secondary to Rheumatic Mitral Valve Disease

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Mitral regurgitation secondary to a calcified flail chordae tendineae is a rare pathology with a clinical presentation similar to other disease processes, potentially leading to initial misdiagnosis and a morbid outcome.

An 83-year-old female with a past medical history of heart failure with preserved ejection fraction, hypertension, hyperlipidemia and transient ischemic attack presented to the hospital with a fever of 38.1C, sudden onset dyspnea on exertion, and chest pain. Chest X-ray revealed bilateral infiltrates and she was treated for community acquired pneumonia. Initial EKG showed sinus tachycardia, ST depression in the inferior leads, and a right bundle branch block. Troponin was negative at 0.04. Due to the patient's current symptoms, an elevated BNP of 408, and a history of chronic congestive heart failure, a transthoracic echocardiogram was performed. This showed an echogenicity of the anterior mitral leaflet most likely consistent with a vegetation, suspicious for endocarditis. Due to negative blood cultures and no clinical stigmata of endocarditis, the patient underwent a transesophageal echocardiogram with 3D reconstruction revealing an elongated serpiginous structure measuring approximately 2.9 cm prolapsing into the left ventricle during diastole and into the left atrium during systole. The structure appeared to be affixed to the posterior medial commissure and was determined to be a ruptured flail calcified cord. Severe mitral valve stenosis secondary to a rheumatic valve with a mean gradient of 14 mmHg and moderate mitral valve regurgitation was noted. Given the findings, cardiothoracic surgery was consulted, and the patient underwent mitral valve replacement with a 27mm mosaic bioprosthetic valve two weeks later, following a negative cardiac catheterization.

Chordal rupture with subsequent flail mitral leaflet is a rare pathology overall, but a more common cause of pure mitral regurgitation. Severe mitral regurgitation due to a flail mitral leaflet has a high mortality rate of >6% if not treated by surgery. Even though there have been instances where a flail mitral leaflet has been mild and asymptomatic in patients, completely ruptured chordae tendineae normally cause patients to have severe regurgitation and need surgery urgently to alleviate symptoms. Though this patient did present with a ruptured chordae tendineae that caused a flail mitral leaflet, the regurgitation present was only mild to moderate. We believe this was likely due to the severely stenotic valve along with calcification causing diminished systolic sphincteric narrowing of the mitral annulus maintaining valvular competency that partially prevented an excess amount of regurgitation, showing a partial protective effect.

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Out of sight, but not out of mind. Vigilant follow up after obstructive pneumonia reveals pulmonary malignancy

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Introduction: We present a case of left upper lobe obstructive pneumonia after food aspiration. Follow up imaging for pneumonia resolution revealed unexpected left upper lobe small cell carcinoma.

Case Presentation: 69-year-old male veteran and lifelong smoker presented to the emergency department with fevers and productive cough following vegetable aspiration. Pulmonary exam demonstrated left-sided rales and labs showed a leukocytosis of 20,000 cells/uL. CT chest revealed abrupt tapering of the left upper lobe bronchus, and consolidation with air bronchograms suspicious for post obstructive atelectasis of the left upper lobe. Bronchoscopy demonstrated complete endobronchial obstruction of left upper lobe due to foreign body. Post procedure, the left upper lobe bronchus was patent with no obstructing lesion, and chest imaging showed significantly improved aeration. The patient clinically improved and was discharged with antibiotics. Six weeks later, follow up CT chest demonstrated reaeration of the left upper lobe with a new suspicious left upper lobe mass. Ultimately, fine needle aspiration biopsy revealed small cell carcinoma.

Discussion: Obstructive pneumonia is a common presentation after foreign body aspiration, but there is limited data on the clinical utility of routine follow-up chest imaging for patients with pneumonia. The incidence of lung cancer after pneumonia is low, with reported rates ranging from 1 to 4%. However, a recent Veteran Affairs study found previously undiagnosed pulmonary malignancy in 9.2% of inpatients above 64 years of age after being treated for pneumonia. This case emphasizes the need for close, vigilant follow up after post obstructive pneumonia to avoid overlooking pathology, especially in higher risk patients with smoking history. Further study may help clarify specific subgroups that would benefit from radiologic follow up after treatment for pneumonia.

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Burn-associated Delayed Dilated Cardiomyopathy Resulting in Pneumothorax in an Adolescent

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Acute heart complications of heart disease have been well defined in the literature, yet little has been reported about the delayed effects of burn injury on the heart. Dilated cardiomyopathy is a rare complication of burn injury with many causative factors and complicated pathophysiology.

In this case, a 16-year-old male sustained 95% total body surface area burns resulting in dilated cardiomyopathy. On initial presentation, a chest x-ray showed a non-enlarged cardiac silhouette that lacked apparent pathology and occupied less than 50% of the transverse diameter of the chest. Over the course of the hospital stay, complications occurred from multiple skin grafts and low blood pressure due to opiate medications used for pain management. The low blood pressure resulted in the inability to continuously utilize beta-blockade continuously as importance was given to pain medication over beta-blockade. 18 months after admission, the patient became dyspneic and a repeat chest x-ray showed dilated cardiomyopathy. The dilated cardiomyopathy became so severe that the heart restricted the expansion of the left lower lung lobe and resulted in secondary atelectasis.

Beta-adrenergic receptor antagonists have been utilized in burn injury to prevent tachycardia and hypermetabolism resulting in muscle catabolism. This case report suggests that proper beta-blockade may be beneficial in preventing delayed cardiomyopathy in burn patients.

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A rare case of urticarial skin rash associated with dexmedetomidine infusion

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Introduction: Dexmedetomidine is an α2 agonist which is commonly used for sedation in the ICU. Common side effects include hypotension, bradycardia, and nausea.1 We present a very rare case of urticarial rash which appeared hours after continuous infusion was started. Two other case reports were found in the literature, in the first a wheal and flare rash started within 4 hours of continuous infusion and resolved within 48 hours of discontinuation.1 In the second isolated case, immediate urticarial type cutaneous symptoms occurred within 90 seconds of localized nerve block administration of dexmedetomidine

Case Presentation: A 64-year-old man with a history of severe alcohol use disorder, COPD, CHF, CAD, hypertension, and opioid use disorder presented to the ED seeking treatment for his alcohol dependency. Initial labs were unremarkable, both with undetectable ethanol level and negative urine drug screen. In addition, patient was endorsing increased cough and dyspnea. He was admitted to the general medical floor with acute exacerbation of COPD for treatment with nebulizers and IV steroids. During his hospital stay, addiction medicine was consulted, and he was started on a phenobarbital taper. The patient's hospital stay was complicated with worsened tachypnea, wheezing, and increasing oxygen requirements requiring an ICU consult and transfer to the intensive care unit. Chest X-ray showed pulmonary vascular congestion, and he was given Lasix. Patient was noted to be disoriented and agitated, he was uncooperative with staff. He was started on non-invasive ventilator with a dexmedetomidine drip for agitation. Six hours later patient was noted to have bright red urticarial rash over his bilateral neck extending down his upper extremities. There was no angioedema of the face, airway was patent, but diffuse wheezing was heard on auscultation. Benadryl and IV epinephrine were given. Patients medication administration record was reviewed. The only new medication for the patient since arrival to ICU was dexmedetomidine, which was then appropriately discontinued. In the following hours the rash spread further down the arms, with the initial areas resolving. There was no peripheral eosinophilia noted on lab work. Less than 24 hours after discontinuation of dexmedetomidine, the rash had improved, and no residual rash was noted after six days from its initial appearance. Ultimately the patient made a full recovery and was discharged home.

Discussion: To our knowledge, this is the third documented case of a possible drug rash associated with dexmedetomidine. In this setting the rash was likely secondary to administration of dexmedetomidine, due to it being the only new agent started and the resolution with discontinuation. Though rare, providers should be aware of this possible side effect.

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Metastatic Small Cell Carcinoma Presenting as Acute Pancreatitis

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Introduction: Small cell lung carcinoma (SCLC) is an aggressive malignant lung cancer with poor prognosis. Acute pancreatitis is characterized by a combination of clinical symptoms such as abdominal pain, elevated pancreatic enzymes, and radiologic evidence of pancreatic inflammation. While SCLC commonly metastasizes to lymph nodes, lung, brain, bone, and adrenal glands, SCLC can also rarely metastasize to the pancreas, causing acute pancreatitis (1). Metastasis-induced acute pancreatitis (MIAP) is an extremely rare etiology for acute pancreatitis; retrospective studies of patients with lung carcinoma who developed MIAP note an incidence as low as 0.12% (2). Here, we present a case of MIAP secondary to incidentally-discovered SCLC in a 73-year-old Caucasian male patient.

Case presentation: A 73-year-old man with past medical history including hypertension, diabetes mellitus type II, GOLD stage D COPD, chronic respiratory failure on home two-liter per minute oxygen through nasal cannula, thirty-pack-year former smoker presented with a one-week history of sharp, non-radiating 8/10 (10 = worst) periumbilical/epigastric abdominal pain. Associated symptoms included nausea, anorexia, and fatigue. The patient denied gallbladder disease, alcohol use, hyperlipidemia, trauma, recent illness, history of pancreatitis, history of malignancy, occupational exposures, dietary changes, or known autoimmune diseases. Family history was positive for small cell lung cancer in his mother and cancer in his sister. Physical exam was notable for epigastric tenderness without hepatosplenomegaly, rebound tenderness, rigidity, or jaundice. Review of symptoms was otherwise negative. Vital signs were within normal limits, and labs showed white blood cells 13,300, hematocrit 41, lactic acid 1.2, glucose 169, BUN 25, creatinine 1.3, calcium 9.8, lipase 96, triglycerides 150, total bilirubin 0.4, AST 14, ALT 12, alkaline phosphatase 45. CT abdomen/pelvis with contrast revealed fat stranding around the pancreatic head, and gallbladder ultrasound showed no gallbladder stones or sludge but pancreatic duct dilation to 6 mm. CTA chest showed right paratracheal/hilar lymphadenopathy and a 4.8 x 2.7 cm subcarinal mass. Magnetic resonance cholangiopancreatography (MRCP) showed two 1-cm lesions in the pancreatic body, lymphadenopathy, and inflammatory pancreatic changes. Endoscopic ultrasound (EUS) re-demonstrated lymphadenopathy and showed that the two pancreatic lesions were communicating with the pancreatic duct. Mediastinal lymph node fine-needle aspiration (FNA) with biopsy confirmed metastatic SCLC. Oncologic evaluation included positron emission tomography-computed tomography which revealed an avid subcarinal mediastinal lymph node. Oncology started the patient on cisplatin and etoposide every three weeks for up to six cycles with concurrent radiation with the first two cycles, and he tolerated his first two cycles without complications.

Conclusion: Metastasis-induced acute pancreatitis secondary to small cell lung carcinoma is a rare but significant disease process with poor prognosis; however, accurate diagnosis and treatment may improve clinical outcomes and possibly positively impact survival in affected patients. MIAP should be considered in the differential diagnosis for acute pancreatitis.

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A Case of Prolonged Prosthetic Joint Infection due to Mycobacterium goodii

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Introduction: Mycobacterium goodii, an acid-fast, nontuberculous mycobacterium, is a rare but emerging cause of nosocomial infection. Its inherent antimicrobial resistance patterns and indolent presentation can lead to delays in diagnosis and prolonged periods of inappropriate treatment. To date, M. goodii has been implicated in a wide range of healthcare-associated infections from prosthetic joint septic arthritis and intraabdominal abscesses after hernia repair to pacemaker pocket infections and postcataract endophthalmitis after lens replacement. We present a case of prosthetic joint infection with M. goodii that was complicated by multiple surgical revisions and failed courses of antibiotic therapy prior to appropriate diagnosis and treatment.

Case Report: A 56-year-old male presented for stage one of a two-stage revision of a total knee arthroplasty (TKA) for suspected prosthetic joint infection in November 2018. The initial TKA was performed three years prior and had already been revised twice for suspected infections without a confirmed pathogen. On presentation, the patient had a chronic draining sinus tract and abscess over the joint. The prosthesis was removed, and an antimicrobial spacer was placed with complex wound closure involving a gastrocnemius flap and skin grafting. On day 14 of incubation, synovial fluid culture yielded growth of acid-fast bacilli. The patient was started on targeted treatment with amikacin 1 g QOD for 3 weeks in addition to tigecycline 50 mg BID, moxifloxacin 400 mg daily, and azithromycin 500 mg daily for 2 months. The mycobacterium was identified as M. goodii by MALDI-TOF and confirmed at CDC by 16s rRNA gene sequencing in February 2019. After two months of antibiotic therapy, the patient was transitioned to oral minocycline 100 mg BID and moxifloxacin 400 mg daily for an additional 4 months. The patient completed the two-stage revision of his right TKA in March 2019, and intraoperative bacterial, mycobacterial, and fungal cultures showed no growth. Postoperatively, the patient completed two months of minocycline and moxifloxacin. At 1 year follow up visit, the patient was without signs of TKA infection.

Discussion: M. goodii, which was first isolated in 1999, is an emerging cause of healthcare associated infections. M. goodii prosthetic joint infections are often associated with severe disease that requires debridement, prosthesis excision, and extended courses of broad-spectrum antibiotic therapy. The diagnosis is often missed initially because M. goodii can take over 2 weeks to grow in bacterial culture, which is beyond the typical 14 day incubation period. Our case demonstrates that samples from the infected material should be submitted for mycobacterial along with bacterial and fungal cultures, especially when initial cultures are negative, in order to optimize patient outcomes.

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Fusobacterium: A Rare Case of Septicemia in a Patient with Multiple Abscesses

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Fusobacterium, a gram-negative anaerobic bacterium that is part of the normal flora in the oropharynx, is frequently associated with pharyngitis and tonsillitis due to its native location. Fusobacterium septicemia is rare, with only 5.5 cases per 1,000,000 patients. We report a unique case of fusobacterium sepsis presenting with brain and lung abscesses.

A previously healthy 58-year-old male presented to the emergency department with left-sided weakness. He reported concurrent episodic left sided-arm jerking starting two weeks earlier. Examination revealed a frail patient with dry mucous membranes and poor dentition, with diffuse bilateral wheezing, worse in the upper lobes. Neurologic exam revealed 2/5 strength in the left upper and lower extremity with intact sensation. His laboratory studies revealed WBC 16.39 K/mcL with 84.8% neutrophils, hemoglobin 14.0 g/dL, and platelets 396 K/mcL. Computed tomography (CT) of the head demonstrated extensive vasogenic edema involving the right parietal lobe with a suspected underlying mass lesion, highly concerning for malignancy. Chest CT showed a cavitation in the right upper lobe measuring 7.5 cm with mediastinal lymphadenopathy, suspicious for bronchogenic carcinoma. The initial working diagnosis was lung cancer, possibly small cell, with metastasis to brain. Bronchoscopic evaluation with lavage was negative for malignancy. Brain biopsy revealed an abscess with ten milliliters of purulent drainage, positive for Fusobacterium. Neck and maxillofacial CT found a sizeable left maxilla lesion eroding into the nasal cavity. He underwent dental extraction and was ultimately treated with six weeks of ceftriaxone and metronidazole for his multifocal fusobacterium infection.

Fusobacterium accounts for only 6% of monomicrobial brain abscesses and 5% of lung cavitations, usually associated with the species F. nucleatum. The presenting symptoms of fusobacterium septicemia are often species dependent. Although the organism from our case was never speciated, our patient's clinical picture is most consistent with F. necrophorium infection. F. necrophorium infection typically presents in otherwise healthy individuals and is frequently accompanied by abscesses. The source of F. necrophorium septicemia is most commonly tonsillitis and pharyngitis. From this location, it can cause septic thrombophlebitis of the internal jugular vein, otherwise known as Lemierre's syndrome, or seed the blood stream directly. There are numerous case reports of abscesses from fusobacterium septicemia, but presentation with concomitant lung and brain abscesses is exceedingly rare and more frequently associated with Lemierre's syndrome. Our patient did not have tonsillitis, pharyngitis, or Lemierre's syndrome, but had profoundly poor dentition with periodontal abscesses and erosion into the nasal cavity, the likely source of his septicemia. Fusobacterium infection should be considered in patients with cavitary lung disease and brain lesions who present with significant dental pathology.

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Holey Esophagus! A case of pericarditis due to esophageal-pericardial fistula

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Background
Infectious pericarditis can be caused by multiple etiologies including viral, bacterial, and fungal. Fistulous connections with the pericardium can be predisposing risk factors for infectious pericarditis. In our case, we describe a case of esophageal-pericardial fistula as a cause of pericarditis and cardiac tamponade.

Case presentation
A 32 year old male who emigrated from India two years prior with a past medical history of HIV, well controlled on antiretroviral therapy, and latent tuberculosis (TB) presented to the hospital with shortness of breath and malaise. On presentation he was febrile to 102F, tachycardic to 140 bpm, and hypotensive to 80/53. He was ill appearing with exam findings notable for crackles in both lungs, distant heart sounds, and pulsus paradoxus. There was a high clinical suspicion for pericardial tamponade with subsequent echocardiographic confirmation. He was taken to the catheterization lab where 360mL of purulent, light green fluid was removed from the pericaricardial space resulting in improved hemodynamics. Further investigation for the underlying etiology included a chest CT and esophogram that noted a small fistulous communication between the upper thoracic esophagus to the pericardium. He was started on broad spectrum antibiotics. He was then taken to operating room where he underwent an esophageal stent placement with thoracotomy and drainage of a mediastinal abscess. Pericardial fluid analysis was notable for glucose <10 mg/dL, pH 6.6, 44,000 nucleated cells with 92% neutrophils. Pericardial fluid cultures grew Streptococcus anginosus, and he was treated with ceftriaxone and metronidazole for four weeks.

Discussion
We report a rare case of fistula formation that resulted in pericardial infection and tamponade. His fistula is believed to be from several bouts of vomiting leading up to admission, as a possible side effect of his antiretroviral therapy. Contiguous spread as a cause for pericarditis is rarely documented, but the case reports that do exist demonstrate that the patients at risk for this type of complication are those with esophageal malignancy, post cardiac procedure (such as ablation), or esophageal rupture. However, regardless of the etiology, when a fistula is suspected (noting bilious appearing pericardial fluid or history that suggests this as an etiology) it is important to identify and intervene quickly due to an associated high mortality rate.

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Epiglottic enigma: A challenging diagnosis of extra-nodal NK/T cell lymphoma

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Introduction: Epiglottitis, although generally characterized by slower progression of symptoms in adults as compared to children, has a broad differential diagnosis and, if untreated, may lead to life-threatening upper airway obstruction.

Case Description: A 26-year-old female without significant past medical history first presented to an outside emergency department (ED) with two weeks of throat pain and was diagnosed with acute pharyngitis. Indirect laryngoscopy one week later in the ED, again for throat pain, showed epiglottic and arytenoid edema. She was prescribed antibiotics and prednisone. On repeat indirect laryngoscopy six days later, the patient was noted to have a lateral epiglottic ulcer. She had at this point undergone several courses of antibiotics and steroids with only minimal interval symptomatic improvement.

At the time of evaluation for this case, about six weeks overall since symptom onset, she again presented to the ED with worsening throat pain. On assessment, she was afebrile, tachycardic, and hypertensive. The patient was noted to be in respiratory distress and was intubated by ENT who visualized edematous and narrowed supraglottic structures on laryngoscopy. The following diagnostic studies were collected over the course of several admissions for similar presentations. C-reactive protein and erythrocyte sedimentation rate were elevated at 33.2 mg/L and 34 mm/hr, respectively. Evaluation for infectious pathogens including Bordetella, Chlamydia, Gonorrhoeae, group A Streptococcus, adenovirus, herpes simplex virus, parvovirus, rhinovirus, influenza, HIV, RSV, histoplasmosis, tuberculosis, was unremarkable. EBV IgM was negative and IgG was positive. Broad rheumatologic evaluation was not diagnostic. Neck CT with contrast showed significant epiglottic and aryepiglottic fold edema with borderline enlarged cervical lymph nodes. Initial epiglottis and arytenoid biopsies showed reactive changes without evidence of malignancy. The patient's supraglottic edema improved, and she was extubated.

Eight months after initial presenting symptoms, the patient was ultimately diagnosed with extra-nodal NK/T cell lymphoma (ENKTL), nasal type on repeat biopsy of her laryngeal lesion following further extensive rheumatologic and infectious disease evaluation for symptom progression. She underwent tracheostomy and has completed multiple cycles of chemotherapy. Oropharyngeal and posterior pharyngeal wall radiotherapy was also performed. Thus far, she has had some improvement in lesion size, although the overall prognosis for this disease is poor.

Discussion: ENKTL, nasal type, is an aggressive and uncommon diagnosis in the United States but is more prevalent in regions of Asia and Latin America. Based on available literature, it rarely involves the epiglottis. As was the case for this patient, diagnosis is often delayed due to variable and nonspecific presenting symptoms. While the use of steroids in this patient was unavoidable given her airway compromise, it may have further delayed her histopathologic diagnosis. This case highlights the broad differential for recurrent epiglottitis as well as challenges in diagnosing ENKTL.

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Arterio-Enteric Fistula in a Kidney-Pancreas Transplant Patient: A Rare Case of Gastrointestinal Bleeding

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Gastrointestinal (GI) bleeding is one of the most common causes for hospital admission. Common etiologies for GI bleeds include peptic ulcer disease, esophageal varices, and diverticular bleeding. Arterio-enteric fistula is a rare but dramatic cause of GI bleeding. The best characterized arterio-enteric fistula is the aorto-enteric fistula, a known complication following aortic aneurysm repair. Arterio-enteric fistulas beyond the aorta are exceedingly rare, but should be considered in patients who have undergone kidney-pancreas (KP) transplant.

A 56-year-old female with a history of KP transplant and subsequent failure of the enteric-drained transplanted presented with increasing dyspnea, fatigue and a single episode of hematochezia. Outpatient laboratory evaluation had revealed anemia (Hgb of 5.1 g/dL). In the ED, patient was hemodynamically stable. Her abdominal exam was benign. Etiological evaluation revealed that iron studies were normal and markers of hemolysis were not detected. A CT scan of the abdomen and pelvis without contrast did not reveal any retroperitoneal or intraperitoneal hemorrhage. Despite initial response to blood transfusion, hemoglobin continued to trend down.

Patient ultimately underwent preparation for EGD/Colonoscopy. While undergoing bowel preparation, patient decompensated into hemorrhagic shock with multiple bloody bowel movements necessitating massive transfusion and ICU admission. She underwent emergent EGD which showed no source of bleeding, but red blood throughout the stomach and duodenum, suggesting an upper GI source. CT angiogram of the abdomen and pelvis identified an arterio-enteric fistula from her arterial anastomosis from the external iliac artery refluxing through her pancreas and into the duodenal-ileal anastomosis. Patient underwent emergent percutaneous placement of a right external iliac covered stent for temporary hemostasis. She subsequently underwent resection and anastomosis of the small bowel attached to her pancreas and pancreatic debridement. Patient recovered and was discharged from the hospital.

Arterio-enteric fistulas are a rare cause of GI bleeding, but should be considered in patients with KP transplant presenting with GI bleed. Fistula development is most commonly seen in those with failed enteric-drained pancreatic transplants, as was the case for this patient. The pathogenesis is multifactorial with leakage of digestive pancreatic enzymes and pseudoaneurysm rupture likely contributing to the development of this fistula. Accurate diagnosis includes a CT angiogram; percutaneous stent placement served as temporary hemostasis in this unstable patient. Resection of the donor duodenum graft with pancreatic debridement was definitive therapy to controlling the bleed.

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Statin-induced Necrotizing Myopathy

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While statins are one of the most prescribed medications, statin-induced necrotizing myopathy (NM) remains a rare disease. If unrecognized, it may lead to severe debilitation and therefore requires careful diagnosis and prompt initiation of treatment.

A 56-year-old woman presented with 2 months of progressive muscle weakness. Past medical history included HTN, HLD, DM, and depression for which she took: hydrochlorothiazide, atorvastatin, metformin, and citalopram. Notably, she had been on atorvastatin for two years prior to presentation. Physical examination revealed marked proximal upper and lower extremity muscle weakness. CPK was 16,180 U/L, AST 348 U/L, ALT 446 U/L, Aldolase 91.2 U/L; creatinine was normal. Her statin was discontinued, she was treated with IV fluids, and was discharged home. She returned to her PCP office with persistent symptoms and continued elevation of CPK ultimately requiring a second admission for further workup. An EMG was consistent with diffuse inflammatory myopathy, and subsequent muscle biopsy revealed necrotic muscle fibers with muscle regeneration and fatty infiltration. Anti-HMG-CoA antibody was positive, and the patient was diagnosed with statin-induced NM. She was initiated on high-dose glucocorticoids, without improvement; therefore IVIG therapy was added. At four months, she is starting to report improvement with continued IVIG infusions.

Autoimmune NM has been classified into three types by the European Neuromuscular Center: anti-SRP (signal recognition particle), anti-HMGCR (hydroxyl-3-methylglutaryl-CoA reductase), and seronegative. Anti-HMGCR autoantibodies were first described in 2010 by Sinet et al., and since that time, has been noted as part of the workup of statin induced NM. In the literature, previous statin exposure in these patients range from 63 to 90% in antibody positive patients. Additionally, the length of statin use prior to presentation may vary from weeks to greater than 20 years. It is hypothesized that statins up-regulate HMGCR expression by regenerating muscle fibers, which explains why despite statin discontinuation, symptoms persist. There is an association with malignancy which emphasizes the importance of up to date appropriate cancer screening and a full body CT scan, as performed in our patient. Seronegative NM is more consistent with malignancy, while anti-SRP is not associated with either statins or malignancy. Given its rarity, treatment for NM is guided by expert opinion. High-dose glucocorticoids, is recommended, with refractory cases requiring a second or a third agent, which may include immunosuppressive therapy (i.e. methotrexate, azathioprine, rituximab) or IVIG.

This case illustrates an uncommon side effect of a commonly used medication. Accurate diagnosis is imperative, as this guides treatment. Treatment of refractory anti-HMGCR NM takes time and improvement may be slow. Discontinuation of the statin, with initiation of therapy as soon as possible will give patients with severe disease, such as ours, a chance to regain normal muscle function.

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An Unusual Presentation of Cefepime Induced Encephalopathy

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Introduction: Cefepime is a commonly used fourth generation cephalosporin that has been associated with neurotoxicity, adverse effect is more prominent in the setting of renal impairment. The mechanism by which cefepime causes this adverse effect is not well understood but has been proposed to be related to its ability to cross the Blood Brain Barrier (BBB) and produce concentration dependent antagonism of the gamma aminobutyric acid. The most common neurotoxic signs and symptoms that have been reported include: depressed consciousness, confusion, aphasia, myoclonus, seizures, and coma. This case report is about a 52 year old man, with a past medical history of end stage renal disease on hemodialysis (ESRD-HD) who developed cefepime induced encephalopathy, with a unique physical finding of ankle clonus on examination.

Case presentation: A 52 years old African American man with a past medical history of Hypertension, Diabetes Mellitus Type 2 and ESRD-HD presented with a two weeks history of worsening right foot pain and a chronic ulcer with increased purulent discharge. He was diagnosed with gas gangrene and necrotizing fasciitis of the right foot and subsequently underwent emergency incision and drainage, excisional debridement, and fasciotomy. The patient was started on Vancomycin and Piperacillin/Tazobactam which later switched to IV Cefepime. Two days after cefepime was started, the patient reported clumsiness in the right upper extremity, inability to maintain a grip or hold on to items. The next day he was found to be disoriented and hypoactive. Pertinent findings on examination were; ankle clonus and hyperreflexia. Cranial nerves II-XII were grossly normal, cerebellar function, sensation and motor were also normal. Gait was not evaluated due to non weight bearing status of the right foot post surgery. Complete blood count, complete metabolic panel and computed tomography of the brain did not reveal any obvious etiology for these new onset neurological symptoms. Cefepime induced encephalopathy was suspected and the drug was stopped. Patient had his scheduled hemodialysis the following day and was found to have complete resolution of his altered mental status, hyperreflexia and clonus the following day.

Discussion: In critically ill patients with multiple comorbidities it might be challenging to identify cefepime induced encephalopathy. This case illustrates the importance of cautious use of cefepime in at risk patients and implementing a high degree of surveillance of neurologic symptoms. Close observation might lead to achieving an early diagnosis and avoidance of unnecessary testing or interventions. Cefepime discontinuation should result in improvement of the symptoms usually within 2-7 days.

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Phlegmasia Cerulea Dolens Presenting with Acute Compartment Syndrome

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Introduction: Phlegmasia cerulea dolens (PCD) is a fulminant, life-threatening condition caused by acute massive venous thrombosis. It is characterized by the triad of edema, pain, and cyanosis of the limb. This condition is poorly understood and due to its rarity there is no consensus regarding its management.

Case Presentation: A 67-year-old female with a history of DVT with IVC filter placed six months prior and recently diagnosed tonsillar squamous cell carcinoma presented with PEG-tube malfunction. She had recently completed her third week of carboplatin/paclitaxel with concurrent radiation therapy. She had PEG-tube placed two weeks prior to presentation due to poor oral intake. In the interim she developed leakage around the insertion site. CT imaging revealed buried bumper at her local hospital. She was transferred to our tertiary center for surgical evaluation. Upon arrival she was hemodynamically stable. She endorsed fatigue and chronic dysphagia. Physical exam was notable for leaking fluid around PEG insertion site; remainder of abdominal, extremity, and skin examinations were normal. Initial chemistries and CBC were unremarkable. Shortly after admission she developed severe lower extremity pain, hypotension, lactic acidosis, livedo reticularis to mid-thighs bilaterally, and weak lower extremity distal pulses. She underwent diagnostic laparoscopy which was unremarkable. Venous duplex imaging revealed extensive acute occlusive thrombus throughout the femoral, proximal deep femoral, popliteal, posterior tibial, and peroneal veins bilaterally. She was determined to have PCD resulting in acute compartment syndrome and cardiogenic shock. Intravenous unfractionated heparin was initiated. Her clinical status swiftly deteriorated as she developed anuric renal failure, shock liver, and bleeding requiring massive transfusion. She underwent emergent guillotine right above knee amputation, fasciotomy of the left leg, right iliac vein mechanical thrombectomy, and left femoral vein open thrombectomy. Despite these interventions she succumbed to her critical illness shortly after return from the operating room.

Discussion: PCD results from acute massive venous thrombosis which obstructs venous drainage of an extremity; most commonly the lower extremities are involved. PCD is most common during the fifth or sixth decades, is more common in females, and malignancy is the most common triggering factor. Other associated risk factors include inherited thrombophilias, surgery, and vena caval filter insertion. Cyanosis progressive from distal to proximal areas is the pathognomonic finding of PCD. IV UFH is the anticoagulant of choice while a decision to pursue more aggressive management such as thrombolysis or thrombectomy is considered. Acute compartment syndrome, characterized by ongoing tissue hypoperfusion due to raised intra-compartmental pressure, is a rare complication of DVT and has previously been described in PCD. While uncommon, it is important to quickly identify patients with PCD as it is associated with a high degree of morbidity. Delay in treatment may result in loss of limb or death.

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There's a Bug in the System - Finding an Uncommon Cause of Renal Abscess

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Ureaplasma species are fastidious bacteria that are known colonizers of the genitourinary tracts in sexually active females and males with unclear pathogenicity in immunocompetent hosts. Some reports have shown possible associations with urethritis and chorioamnionitis. Making the diagnosis of ureaplasma infection is challenging given the rarity of infection and inability of the bacteria to grow on regular microbiological culture media. We present a case of complicated urinary tract infection with renal and psoas abscesses in an immunocompromised patient failing multiple antibiotics courses and with negative routine infectious workup.

In our case a 70-year-old female with a history of chronic lymphocytic leukemia (on ibrutinib), immune thrombocytopenia (previously on rituximab), and hypogammaglobulinemia presented with approximately 3 months of urinary frequency/urgency and sterile pyuria. She had failed two courses of ciprofloxacin and a course of trimethoprim/sulfamethoxazole. An outpatient CT urogram showed cystitis, left sided pyelonephritis/pyeloureteritis with mild hydronephrosis, and a 3.5 by 3.7 cm renal abscess. She was admitted to the hospital and started on ceftriaxone. Urinalysis revealed blood, leukocyte esterase, white cells, and bacteria, but her urine cultures and blood cultures were no growth. She was discharged on amoxicillin/clavulanic acid and fluconazole. About a week later, she was readmitted to the hospital with fever, urinary frequency, and left-sided flank pain. A repeat CT scan showed progression of her previous CT findings, now with a 1.4 by 0.9 cm psoas abscess. Repeat urine and blood cultures were negative, and aspirated abscess fluid samples did not reveal bacterial or fungal growth. Pathology of the abscess showed suppurative inflammation. There was concern for an atypical organism, and therefore a urine mycoplasma hominis/ureaplasma culture was sent. She was started on levofloxacin. The culture returned positive for Ureaplasma urealyticum, and doxycycline was added later due to concerns for multidrug resistance, with improvement of her fever and symptoms. Upon outpatient follow-up about 8 weeks later, the patient reported continued resolution of her fevers and dysuria. Her follow-up imaging revealed resolution of the previously demonstrated abscesses and decreased kidney and urinary tract inflammation.

This case illustrates the need to consider Ureaplasma urealyticum as a rare causative organism for infection in a patient with hypogammaglobulinemia where routine infectious cultures are negative. Extra-urogenital infections with abscess formation have been reported in patients with humoral immunodeficiency or those who have received rituximab. Antibiotics that inhibit cell wall synthesis such as penicillins and cephalosporins have no activity against Ureaplasma species due to their lack of cell walls. Fluoroquinolones and tetracyclines are the antibiotics of choice. Physicians should consider checking for Ureaplasma urealyticum in the appropriate clinical setting.

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RHABDOMYOLYSIS- A RARE CAUSE OF BILATERAL BRACHIAL PLEXOPATHY

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INTRODUCTION- Brachial plexopathy is caused by inflammation, direct trauma, stretch injuries, pressure from tumors in the area, and radiation therapy to the brachial plexus. Bilateral brachial plexus injury is rare and usually a result of traction or compressive injury. We present a rare case of bilateral brachial plexus injury secondary to compression injury by rhabdomyolysis.

CASE-PRESENTATION- 36 years old male with past medical history significant for depression and seizure disorder presented with bilateral upper extremity paralysis. One month ago, patient was found unresponsive for 18 hours, on his right side on the ground after going through a seizure episode. Patient had a prolonged hospital stay for rhabdomyolysis. The patient was brought in because of suicide ideation due to progressive weakness of both upper extremities, right more than left, after the prior admission. Patient did not have any other symptom. He was afebrile and his vitals were stable. On examination, patient had flaccid paralysis with no strength of right arm, 3/5 strength of left arm with complete loss of sensation to touch, pain, and temperature. There was areflexia on the right with only +1 brachioradialis on the left. He, however, had 5/5 strength with normal sensation in the legs. CBC, kidney, liver, thyroid function, vit B12 and folate were normal. ESR, CRP, CK and myoglobin were normal. MRI brain, cervical and thoracic spine were normal. MRI Brachial plexus showed sub-acute myositis of bilateral serratus anterior, scapularis, right supraspinatus, and right intercostal muscles. Subsequently, EMG was done, which revealed sub-acute bilateral brachial plexus lesion affecting right side more than left side and involving all the trunks. Diagnosis of plexopathy secondary to compressive injury from rhabdomyolysis was made. He received high dose steroids for 3 days with minimal relief. Patient was discharged to rehab with follow up EMG in 3-4 months to determine any reinnervation of muscles.

DISCUSSION- Rhabdomyolysis induced compression brachial plexopathy is rare, but, possible complication and should be considered in differentials, while evaluating flaccid paralysis. Combination of compression and ischemia together with toxic damage to the muscles result in focal myopathy and secondary compression of peripheral nerves. Bilateral brachial plexus injuries are uncommon and associated with a poor functional outcome. The symptoms of the brachial plexus injury depend upon the site and extent of insult to the nerves and range from numbness and weakness in the involved arm to a complete motor paralysis of the limb. Immediate operative intervention is reserved for rare cases of ongoing compression of the plexus. In chronic paralysis, physical therapy and supportive treatment are the only options available. Brachial plexus injuries are not always reparable, however, and in such cases, neurotization or nerve transfers may offer a better functional outcome.

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Use of palliative radiotherapy in bleeding metastatic small bowel tumor

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Bleeding due to gastrointestinal tumors often leads to substantial blood loss requiring transfusion. This causes fatigue, malnutrition, dehydration, and interruption of chemotherapy, and bleeding control in these patients is crucial for improving quality of life. We report a case in which palliative external beam radiotherapy (EBRT) achieved hemostasis in severe bleeding due to jejunal metastasis of colon cancer.

A 74-year old male was admitted to ICU after hematemesis with ongoing grossly melenic diarrhea. Patient had hemoglobin of 5.9 g/dl. Patient had history of metastatic mucinous adenocarcinoma of the colon status-post right hemicolectomy with resection of gallbladder and duodenum, currently on maintenance therapy with 5-fluorouracil and bevacizumab. He required transfusion of 5 units pRBC's and underwent bedside esophagogastroduodenoscopy (EGD). On EGD, macerated tumor tissue with pulsatile vessel and active spurting bleed was seen. Tenuous hemostasis was achieved using epinephrine injection, laser coagulation and endoclip, with high rebleeding risk. Patient was not a surgical candidate and due to stage IV chronic kidney disease with prior renal cell carcinoma status-post left nephrectomy was a poor candidate for catheter embolization. Radiation oncology was consulted and determined patient to be appropriate for palliative EBRT. Treatment consisted of 5 fractions of 400cGy, which he tolerated well. Patient had stabilization of hemoglobin and was discharged home. After discharge, he was restarted on palliative chemotherapy with FOLFIRI without bevacizumab and at 8 weeks post-hemorrhage, had no recurrence of GI bleed.

Palliative EBRT has proven effective for hemostasis in tumor bleeds in multiple cancers including esophageal, gastric, rectosigmoid, anal, and uterine tumors when surgery is not an option. EBRT uses ionizing radiation to damage cells by causing DNA fragmentation and producing free oxygen radicals which irreversibly bind to these DNA fragments. In the case of bleeding vessels, EBRT produces hemostasis through vessel sclerosis. The majority of research on EBRT for upper GI bleeds has been focused on its use in primary or metastatic gastric cancer. The small bowel is a particularly difficult target for EBRT as frequent changes in bowel contents and volume lead to interfraction variations in shape and position of the target in addition to intrafraction variability that occurs in most other abdominal organs, largely related to respiratory motion. In our case, the patient had an endoclip placed during endoscopy, which assisted in localization of jejunal tumor on imaging for EBRT. After thorough literature review, we did not find any similar cases using palliative EBRT for small bowel tumor bleeding. We believe that EBRT could be considered for achieving hemostasis in the setting of small bowel tumor bleeding, particularly when other options such as surgery or embolization are not feasible or available, and when bleeding source can be localized on imaging studies.

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Prolonged Glucosuria with Euglycemic Diabetic Keto-Acidosis in Patient with a History of Empagliflozin Use.

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Background: Sodium-glucose cotransporter 2 (SGLT-2) Inhibitors are a novelty drug class for management of Diabetes Mellitus type II (DMII). SGLT-2 inhibitors have been increasingly prescribed given their favorable cardiovascular and renal outcomes. On the other hand, they also have been linked to the development of euglycemeic diabetic keto-acidosis (eDKA). Empagliflozin half-life is reported to be around 12.4 hours. Here we present a case of delayed euglycemic diabetic ketoacidosis that occurred 5 days after discontinuation with persisted glucosuria till day thirteen beyond the expected effect of SGLT-2 Inhibitors.

Case presentation: A 63year-old woman with a history of DMII on glimepiride, empagliflozin and pre-meal lispro insulin who presented with fatigue and altered mentation. Point of care glucose check done in the emergency department showed hypoglycemia with blood glucose of 40 mg/dl that was treated with D50% pushes followed by D10% infusion. Patient was intubated to protect her airway and was admitted to the ICU. On day 5 of admission, patient was persistently altered with blood workup showed evidence of high anion gap metabolic acidosis with elevated Beta-hydroxybutyrate, and normal serum lactate as well as normal blood glucose albeit with increased urine glucose. Patient was diagnosed to have eDKA and was started on insulin drip, IV fluids and electrolyte replacement protocol. On day 6, anion gap closed, insulin drip was discontinued, and patient was transitioned to subcutaneous insulin therapy. Despite that, the patient continued to have significant isolated glycosuria with normal blood glucose for a total of 13 days.

Discussion: Given empagliflozin's reported mean half-life of 12.4 hours, it is expected that the body will clear about 96.9% of the drug in about 2.5 days. For the drug to cause such prolonged glycosuria even after its discontinuation might imply that the clinical effects can persist much longer than the reported half-life and even longer than 5 half-lives. This should raise questions whether this effect is related only to the pharmacokinetics of the SGLT-2 inhibitors or could be related to the pharmacodynamic characteristics including genetic factors that may lead to a longer than expected effect. Reviewing the literature showed some case reports linking other various SGLT-2 inhibitors highlighting the possibility of this being a class rather than individual drug effect.

Conclusion: With the increasing use of SGLT-2 inhibitors in the current practice among diabetics and patients with heart failure, clinician should be aware about the potential side effects and prolonged effect of those medications. Further studies are warranted to better understand the pharmacokinetics of this medication class.

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Herpes is Here in My Head Again

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Mollaret's meningitis is a form of benign, recurrent aseptic meningitis characterized by as few as 3 to 10 episodes of fever and meningism lasting 2–5 days, followed by spontaneous recovery. The most common etiologic agent in Mollaret's meningitis is HSV-2, although some patients do not have the evidence of genital lesions at the time of presentation. Of great Importance it is to emphasize the benign and self limiting course of this type of meningitis to avoid unnecessary investigations and treatments, and to provide timely reassurance to patients.

A 52 year old African American woman presented with a two day history of severe headache. Headache was global, throbbing, rated 10 out of 10 and persistent with associated neck stiffness, nausea and vomiting, fever, photophobia, chills, myalgia, dizziness and lightheadedness.
The patient had been admitted twice in the past with a similar presentation and has had similar symptoms in the past without inpatient admission.
Past medical histories include postural orthostatic tachycardia syndrome, genital herpes, aseptic meningitis, hypertension and type 2 diabetes mellitus.
Examination was significant for painful distress, stiff neck and positive Kernig's sign.
Complete blood count revealed leukocytosis. Brain CT Scan was normal.
A lumbar puncture was done with CSF analysis revealing total CSF nucleated cell count of 556/mm3, with 52% neutrophils and 47% lymphocytes. CSF protein was elevated at 197mg/dl with a normal CSF glucose. HSV-2 DNA was detected on CSF PCR.
Patient was started on intravenous acyclovir and symptomatic treatment for vomiting and headache. Most of the symptoms resolved on the second day of admission with headache and lightheadedness resolving on the 4th day of admission. Having attained complete resolution of all symptoms, the patient was discharged on one week course of oral valacyclovir for outpatient follow up.

Studies suggest that recurrent meningitis occurs in approximately 20 percent of patients who present with primary HSV-2 infection with meningitis. Most cases of Mollaret's meningitis are caused by HSV-2, but some other causes including other viruses have been described.

No placebo-controlled clinical trial has been conducted to assess the efficacy of the currently available therapies for HSV infection in its acute treatment. It is still controversial whether acyclovir changes the natural course of the illness. A randomized controlled trial did not demonstrate any added benefit of suppressive therapy with valacyclovir in preventing HSV-2 related recurrent meningitis.
In conclusion, it is worthwhile to keep a high index of suspicion for Mollaret's meningitis in patients presenting with recurrent aseptic meningitis as an early diagnosis will help prevent unnecessary investigations and treatments, and provide timely reassurance to patients.

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Mycotic aortic aneurysm and bacterial pericarditis: two rare complications of community-acquired pneumonia

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Background: Both mycotic aortic aneurysm and bacterial pericarditis are conditions associated with significant morbidity and mortality. Risk factors for mycotic aneurysm include previous arterial injury, atherosclerosis, antecedent infection, and impaired immunity. Infection of the thoracic aorta is rare and most commonly occurs due to septic emboli, although bacteremic seeding can also occur. In the post-antibiotic era, bacterial pericarditis occurs mostly after thoracic surgery, trauma, and in immunocompromised patients. We present a case of methicillin sensitive staphylococcus aureus (MSSA) pericarditis and mycotic aortic aneurysm as a complication of community-acquired pneumonia.

Case Presentation: A 75-year-old male with recently diagnosed atrial fibrillation presented to the Cardiovascular Intensive Care Unit (CVICU) with chest pain and dyspnea. Approximately 3-4 weeks prior to presentation, the patient was treated for community-acquired pneumonia by his PCP. He completed a seven-day course of Doxycycline without significant improvement in his symptoms. At PCP follow-up ten days prior to admission, the patient was found to have atrial fibrillation with rapid ventricular rate and was admitted to an outside hospital. He was discharged on Diltiazem and Apixaban two days later.
On the morning of ICU admission, the patient presented to the outpatient echocardiogram lab as part of his workup for recent-onset atrial fibrillation. Echocardiogram demonstrated reduced ejection fraction (25%) and a large hemorrhagic pericardial effusion with concern for tamponade. No valvular vegetations were noted. The patient was subsequently admitted to our CVICU for further care. Review of systems was positive for productive cough, pleuritic chest pain, and mild neck pain. Physical exam was notable for stable vital signs, distant heart sounds, and diminished breath sounds in bilateral bases. Labs revealed anemia (hemoglobin 10.5 g/dL), leukocytosis (white blood cell count 16.9 x 103/L), thrombocytosis (platelets 532 x 103/L), and hyponatremia (sodium 126 mmol/L).
Repeat echocardiogram demonstrated increased size of the effusion and the patient's vitals worsened, therefore he was taken for pericardial drain placement. Pericardial fluid culture and blood cultures were positive for MSSA. Despite antibiotic treatment, the patient continued to have increasing leukocytosis and worsening respiratory distress. CT imaging demonstrated a 4x6 cm mycotic aneurysm of the brachiocephalic artery and left lower lobe consolidation. The patient was felt to be high-risk for surgical intervention, and he ultimately decided to pursue comfort care.

Discussion: This case illustrates an unusual complication of pneumonia and bacteremia. In the medical literature, there are few reported cases of mycotic aortic aneurysm associated with bacterial pericarditis. We suspect that both diseases occurred simultaneously as a result of hematogenous infection secondary to his antecedent pneumonia, despite what was considered appropriate outpatient treatment. Our case highlights the importance of recognizing rare complications of pneumonia, such as pericarditis and mycotic aneurysms, especially in the setting of high-risk pathogens like staphylococcus aureus.

Late-onset e-cigarette associated lung injury

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INTRODUCTION: E-cigarette or vaping product use associated lung injury (EVALI) is an outbreak syndrome of severe respiratory disease, with close to 2800 cases reported to CDC between July and February 2020. We present a case of EVALI with symptoms following more than a week after the most recent exposure.

CASE DESCRIPTION: In October 2019, a 19-year-old male presented to the ED after five days' worsening dyspnea, fever, cough, myalgias, and nausea. Past medical history of asthma, recent travel to Puerto Rico, tobacco/cannabis smoking, and e-cigarette use were elicited. He endorsed an 18-month cannabis history of 4-5 joints/week and a 2-year tobacco history of 1-2 packs/week. He also endorsed 1 year of intermittent e-cigarette use, most recently an NJOY device and non-refillable nicotine pods sourced from a gas station 10 days preceding symptoms. His most recent use of e-cigarette THC products was 3 weeks before symptoms began, using a Black Box device and a "Cookie"-branded pod sourced from Michigan.

At presentation, he was febrile, normotensive, tachycardic, tachypneic and saturating 96% on room air. Scattered ronchi were noted bilaterally. Initial labwork was unremarkable except for leukocytosis (21,800/μL) with neutrophilic predominance. CXR showed no focal opacifications, while CTPE revealed ground-glass opacities. Infectious work-up including Legionella, Mycoplasma, HIV, influenza, RSV, and a full RVP was negative. Additionally, blood and induced sputum cultures were negative, as were ANA, RF, Fungitell and QuantiFERON-TB assays.

The patient received broad-spectrum antibiotics, but his fever recurred and WBCs remained elevated, prompting additional atypical coverage with azithromycin/doxycycline. Procalcitonin was 0.2 ng/mL and CRP was 35 mg/dL, with bibasilar opacities on repeat CXR. Persistent fever and hypoxia were concerning for EVALI, and the patient was started on prednisone, but after respiratory worsening was transferred to the ICU on IV corticosteroids and high flow nasal cannula (HFNC). The patient then improved remarkably, both clinically and on imaging, and after weaning was discharged home on a week-long prednisone taper. At one-week follow-up, the patient was asymptomatic.

DISCUSSION: The CDC's EVALI screening criteria comprise infiltrates on imaging, exclusion of other etiologies, and e-cigarette use within 90 days of symptom onset.1 Here, the diagnosis of EVALI was made only after a thoroughly negative infectious work-up, failure to respond to broad-spectrum antibiotics, and dramatic improvement on corticosteroids.

Preliminary data on EVALI showed almost all patients having vaped in the week preceding symptom onset, and both the intermittence of this patient's e-cigarette use and latency since last exposure made the diagnosis appear less probable at admission. In November 2019, the CDC identified vitamin E acetate (an e-cigarette adulterant associated with THC products) as a toxicant of concern in EVALI, based on bronchoalveolar lavage fluid sample testing. Ultimately, the mechanism underlying this disease entity remains poorly understood.

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Cavitary Pneumonia Related to Vaping

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Lung injury from e-cigarette use and vaping (EVALI) has dramatically increased, with 2711 cases of vaping injury reported to the CDC as of January 2020. Most patients present with pulmonary symptoms including cough, chest pain, shortness of breath, and infiltrates and bilateral ground glass opacities on imaging. We present two cases of cavitary pneumonia related to vaping.

A 25yo female presented with 3 weeks of congestion, worsening cough with foul-smelling sputum, fever, and chills. Her past medical history included cyclic vomiting syndrome and anxiety, but she took no medications. She admitted to vaping marijuana and nicotine cartridges daily. She was hemodynamically stable without hypoxia. A CXR showed a 4cm cavitary lesion in the right lateral mid lung with pulmonary abscess, confirmed by CT, and antibiotics were started. Testing for influenza A/B, S. pneumoniae, HIV, mycoplasma, blood cultures, fungal studies, p-ANCA, c-ANCA and ANA were negative. Sputum cultures grew a polymicrobial mixture, including anaerobes. She was treated with 4 weeks of amoxicillin-clavulanic acid, and at 6 weeks, CXR showed nearly complete resolution of the cavitary density. She was advised against smoking and vaping.

A 21yo male presented with shaking chills, fever, sore throat, cough, and headache for one day. He denied prior medical history, took no medications, but used marijuana and e-cigarettes daily. Chest X-ray showed a right upper lobe infiltrate in the posterior segment with areas of cavitation, confirmed by chest CT; antibiotics were started. Testing for influenza A/B, rapid strep, mononucleosis, HIV, S. pneumoniae, Legionella, and tuberculosis were negative. Sputum cultures revealed a polymicrobial mixture including anaerobes. He was discharged on oral amoxicillin-clavulanic acid for 4 weeks, and was lost to follow-up.

Pulmonary vaping illnesses are primarily reported in young healthy adults; almost 90% of cases involve THC-containing products, though nicotine vaping is also associated with disease. Our patients vaped both marijuana and nicotine, and both had positive urine drug screens for marijuana. Infectious disease and pulmonary consultants believed that their cavitary pneumonia was related to vaping. The exact pathophysiology of vaping-related pulmonary disease is not defined. In animal models, oxidative stress from vaping increases platelet-activating factor receptor (PAFR) expression and allows for bacterial adherence to the airway lining, possibly increasing risk of bacterial infection. Studies note bacterial and fungal contamination of e-cigarette and vape liquids, though a definite link to infection is not established. Neither patient ever cleaned their vaping devices as recommended, but whether vaping lung disease and unclean devices are associated is not known. Cavitary pneumonia is uncommon in young, healthy adults. Vaping should be considered contributory to cavitary pneumonia in patients in the absence of other risks, including aspiration, HIV infection, tuberculosis, and tricuspid valve endocarditis.

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Diffuse Large B Cell Lymphoma Manifesting as Dyspnea and Cardiac Mass

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Introduction: Tumor formation in a cardiac chamber is uncommon. Cardiac envelopment as the initial presentation of malignant lymphoma is very rare. We report a case of diffuse large B-cell lymphoma presenting as progressive dyspnea and edema with cardiac mass on echocardiogram.

Case Description: A 57-year-old African American male in excellent health presented with a two-week history of progressive dyspnea and bilateral lower extremity edema. Patient described a cough with white productive sputum and moderate chest tightness exacerbated by exertion. On examination, patient was afebrile, blood pressure 143/100 mmHg, pulse rate 86/min, respiratory rate 16/min, and oxygen saturation 100% on room air. Decreased breath sounds were noted in the left middle and lower lung fields. Additionally, there was 2+ pitting edema of the bilateral lower extremities to the level of the mid-calf. Laboratory values were significant for PT 15.2 s, INR 1.5, Total bilirubin 2.0 mg/dL, Alkaline Phosphatase 131 U/L, AST 41 U/L, and AST 52 U/L. NT Pro-Brain Natriuretic Peptide was mildly elevated at 287 pg/mL. Echocardiogram revealed a large amorphous structure that appeared exterior to the heart with compression of the posterior left atrium free wall and a rounded echodensity was also noted within the right atrium. CT chest showed a lobulated soft tissue mass involving the right infrahilar and perihilar regions extending into the right atrium and right ventricle of the heart measuring 12.0 cm x 9.4 cm x 10.0 cm with involved narrowing of the right main pulmonary artery. CT guided core needle biopsy was performed with results showing diffuse large B cell lymphoma. Chemotherapy with regimen R-CHOP and Neulasta was initiated eight days after initial presentation. The patient has completed three of planned six cycles with the complication of acute anemia that resolved with blood transfusion after cycle one. An echocardiogram performed after the initial cycle revealed improvement in the intracardiac mass burden (3.7 cm x 4.6 cm to 2.9 cm x 3.1 cm).

Discussion: Diffuse large B cell lymphoma presenting with cardiac tumor is very rare. While the incidence of cardiac involvement in Non-Hodgkin lymphoma on autopsy is 18%, diagnosis typically occurs at a median of 20 months after initial presentation. This case illustrates a unique disease manifestation with cardiac symptoms and subsequent cardiac mass being the preliminary signs that led to the diagnosis of malignant lymphoma.

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Seize the day: Use of dialysis in the treatment of hyperammonemia

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INTRODUCTION: Clinical manifestations of hyperammonemia (HA) are typically neurologic in nature, including fluctuating levels of consciousness, seizures, cerebral edema and herniation (1). When HA is refractory to medical management, dialysis can be used as a treatment strategy (2). There are no established guidelines on when to initiate hemodialysis (HD) for the treatment of HA (3). We present the case of a patient with a history of hepatic encephalopathy and chronic liver failure who presented with HA manifesting as seizure activity after transjugular intrahepatic portosystemic shunt (TIPS) placement, which resolved after initiation of HD.

CASE PRESENTATION: A 63 year old male with a history of alcoholic cirrhosis, portal hypertension, and hepatic encephalopathy was brought to the emergency department for confusion. On presentation, he was found to have altered mental status requiring intubation, and shock requiring vasopressors. Initial lab work demonstrated a hemoglobin of 5.2mg/dl, normal electrolytes and renal function, and ammonia level of 62mg/dl. He underwent emergent esophagogastroduodenoscopy (EGD) to evaluate the acute anemia, which revealed bleeding varices not amenable to banding. The decision was made to proceed with TIPS placement to decrease the risk of bleeding. Post procedure, he remained in shock, with continued encephalopathy in the absence of sedative medications. Computerized Tomography of the brain did not reveal any acute process. He was placed on bedside video electroencephalogram monitoring, which detected seizure activity. His ammonia level was 157mg/dl at the time. Due to his EGD findings, he was not a candidate for nasogastric tube placement for administration of oral treatments for HA. His seizures were refractory to loading of multiple antiepileptic drugs including levetiracetam, lacosamide, and fosphenytoin. He was started on HD via continuous renal replacement therapy, and within three hours his ammonia level returned to 69mg/dl. After initiation of HD, his seizure activity ceased.

DISCUSSION: This case illustrates the role of HD as a treatment option for patients with symptomatic HA. Our patient presented with HA and neurologic manifestations of seizure activity following TIPS placement. Due to his variceal bleeding, he was not a candidate for the typical methods of treatment, which include the use of the gastrointestinal tract to modify production or excretion of ammonia (2). Prior research is limited, but the effective use of HD to lower ammonia levels has been studied in adults with acute or chronic liver disease with concomitant anuria, uremia, or metabolic acidosis (3). Our patient had no evidence of renal dysfunction or other indications for HD, yet it proved effective at reducing ammonia levels and resolving the neurologic manifestations of HA.

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Delayed Alemtuzumab Induced Neutropenia and Thrombocytopenia in Relapsing-Remitting Multiple Sclerosis

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Alemtuzumab is an anti-CD52 monoclonal antibody used to treat relapsing-remitting multiple sclerosis (RRMS). Common adverse effects of this drug include infusion reactions, increased risk of infections and secondary autoimmunity. Serious immune-mediated thrombocytopenia (ITP) secondary to alemtuzumab is well-reported, however significant neutropenia secondary to alemtuzumab is rare, with few case reports. We present an unusual case of delayed combined neutropenia and thrombocytopenia secondary to alemtuzumab.

A 45-year-old male with RRMS and acquired hypothyroidism (post radioiodine ablation for Grave's disease) presented with severe neutropenia (WBC 1,600 cells/μL, ANC 80 cells/μL) and thrombocytopenia (platelet 23,000 cells/μL). He was asymptomatic without fever, signs of infection or RRMS exacerbation. He had received two alemtuzumab infusions, 28 months and 15 months prior to presentation. Acyclovir 400mg twice daily was started prophylactically after his first alemtuzumab infusion for a CD4+ cell count below 200 cells/μL. The patient's ANC and platelets nadired at 70 cells/μL and 11,000 cells/μL, respectively, with hemoglobin of 15.8g/dL. Bone marrow biopsy revealed mild marrow hypocellularity without evidence of dysplasia. Other infectious/hematologic evaluations were unremarkable. Hematology consultants considered the etiology of bicytopenia to be an unusual delayed immune reaction to alemtuzumab. Dexamethasone was started and he was discharged on prednisone 60mg daily. At one month, the bicytopenia resolved and prednisone was discontinued. However, at 4 months, recurrent neutropenia (WBC 1800mL, ANC 385 cells/mL) was noted. Prednisone was restarted with resolution of neutropenia after one month. Subsequently, studies revealed recurrent ITP with platelets of 4 K/mL. He was admitted and treated with two IVIG infusions and dexamethasone 40mg daily with resolution within one week.

Alemtuzumab causes depletion of CD52-positive cells via antibody and complement-dependent cell-mediated cytolysis. CD52 is a surface glycoprotein primarily found on T and B-cell lymphocytes; neutrophils have approximately 22% CD52 of lymphocytes. Lymphocyte depletion is expected, but neutropenia from alemtuzumab is much less common. Studies suggest mild transient neutropenia can occur in approximately 16% of patients on alemtuzumab, but severe delayed neutropenia is rare and has only been reported in a few case reports within two months of last treatment dose. Severe delayed ITP is a well-documented adverse effect up to 48 months after last dose but has not been distinctly reported combined with neutropenia beyond a few months post treatment, as seen in our case. Our patient's unusual presentation of delayed and combined neutropenia and thrombocytopenia, with late recurrence, suggests neutropenia secondary to autoimmunity as a late adverse effect of alemtuzumab. Our patient was asymptomatic, but severe neutropenia and thrombocytopenia carry risk of serious infection and bleeding. Long term screening for delayed hematologic abnormalities are needed. Greater research is needed to understand the mechanism of alemtuzumab associated neutropenia.

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"Slow, Hoarse:" Gastroparesis and Vocal Cord Paralysis secondary to disseminated varicella-zoster virus

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Varicella-zoster virus (VZV) is a herpesvirus that most commonly affects children and reactivates in older adults (10-20% of cases). VZV remains dormant in cranial nerves, dorsal nerve roots, and autonomic ganglion prior to reactivation. The resulting complications include painful dermatomal rashes, facial nerve paralysis, viral meningitis, and more. This case discusses an atypical reactivation of VZV and the subsequent multi-organ sequelae.

A 66-year-old female presented with dysphagia, shortness of breath, and a productive cough. Treatment with antibiotics and steroids were ineffective. A chest computerized tomography (CT) scan showed groundglass opacities throughout the right lung. Given the history of dysphagia, a neck CT scan revealed right vocal cord paralysis without a laryngeal mass/lymphadenopathy. A modified barium swallow showed severe oropharyngeal swallowing with reflexive coughing following episodes of aspiration. Otolaryngology was consulted and laryngoscopy displayed mucosal ulcers on the right larynx, adenoids, and epiglottis. The nerve distribution of the affected tissues was concerning for a viral eruption. With concern of esophageal involvement, gastroenterology was consulted for an esophagogastroduodenoscopy (EGD). The study showed ulcerations throughout the mid-distal esophagus and a nasogastric feeding tube (NGT) was placed to aid with nutrition. Viral PCRs were positive for VZV. The patient was started on intravenous acyclovir in addition to empiric antibiotics for aspiration pneumonia. Despite appropriate therapy, the patient continued to develop fevers and worsening respiratory symptoms. A bronchoscopy revealed pooling of bile and gastric contents through the vocal cords and into the trachea, indicating persistent aspiration despite nutrition provided via NGT. Gastroenterology placed a percutaneous endoscopic gastrostomy (PEG) tube. The subsequent gastric emptying study yielded a 41% gastric clearance and a clearance half-time of 145 minutes – consistent with moderate-severe gastroparesis. The patient was started on metoclopramide to promote gastric motility and, after temporary escalation of nutritional support to total parenteral nutrition (TPN), was able tolerate enteral feedings. The patient's glottic incompetence was treated with intra-laryngeal calcium hydroxyapatite injection and the patient was discharged on oral acyclovir.

This case represents an atypical reactivation of VZV with its manifested nerve distribution (vagus and right laryngeal nerves) affecting a variety of organ systems. It reinforces the importance of maintaining a wide differential in patients experiencing symptoms of aspiration pneumonia. Of note, further analysis of the records showed that the patient was offered a zoster vaccination by her primary care physician but deferred due to a close contact with Non-Hodgkin's Lymphoma. If the patient had been educated about recombinant (non-live) zoster vaccination options, she may have avoided her VZV related illness and associated sequalae. Thus, this case offers two-fold educational benefit: 1) it educates medical providers regarding disseminated VZV and its rare manifestations, and 2) reinforces the importance of patient education and its role in preventative medicine.

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SGLT-2 Inhibitor Induced Euglycemic DKA after Total Knee Replacement Surgery

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Introduction: Euglycemic diabetic ketoacidosis (EDKA) induced by SGLT2i has been reported as DKA with normal or modestly elevated blood sugar. We introduce a case of a postoperative EDKA triggered by total knee replacement surgery (TKA) on postoperative day 5 in a patient using SGLT28 (Empagliflozin) for more than 1 year.

Case Report: A 68-year-old man with a medical history of non-insulin dependent diabetes mellitus on metformin and empagliflozin, hyperlipidemia, hypertension, and CVA on Plavix presented to the ER for nausea, vomiting and chest burning for 3 days after a total knee replacement surgery. Vital signs were only significant for sinus tachycardia. He denied any other symptoms. PE was unremarkable. Labs showed Leucocytosis of 12.9, high anion gap metabolic acidosis; CO2 12 mmol/L, anion gap of 23, and creatinine of 0.9 mg/dL. He was admitted to telemetry and later transferred to the ICU for worsening respiratory status requiring intubation. Repeat labs showed worsening severe high anion gap metabolic acidosis: pH 6.959, PCO2 <15, CO2 2, anion gap 34, creatine 1.2 and lactic acid 3.1. Urinalysis showed significant glucosuria >1000 and ketonuria >80 and beta-hydroxybutyrate >4.50. Treatment included sodium bicarbonate, insulin infusions and D5 containing fluid with continuous improvement of acidosis. Insulin infusion was continued for 8 days.

Discussion: Euglycemic DKA is defined as a clinical triad of high anion gap metabolic acidosis, and ketonemia or ketonuria with normal or modestly elevated blood glucose levels. Hence, it poses a challenge to physicians. Patients with normal BG levels in ketoacidosis may be overlooked, leading to a delay in appropriate management strategies. SGLT2 inhibitors were approved in 2013 by the (FDA) for the treatment of T2DM. These medications work by blocking the SGLT2 protein, which is involved in glucose reabsorption from the proximal renal tubule, causing an increase in renal glucose excretion and a decrease in blood glucose levels, lowering insulin production, increasing glucagon secretion and promoting a shift of glucose to fat metabolism. Ketogenesis is stimulated rendering the body susceptible to acidemia and continuous glycosuria mimicking starvation conditions, causing near-normal glucose levels despite DKA. Lately, SGLT2i have become an attractive class of medication for the management of T2DM given the growing evidence of decreased mortality from cardiovascular events. The warning also identified additional potential triggering factor among others, including reduced food and fluid intake and alcohol use. Recent reviews show that approximately 26-28% of these cases occur in post-operative patients.

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Not Your Typical Hypocalcemia

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Hypocalcemia in the setting of hypoparathyroidism is most commonly due to gland destruction-surgical or autoimmune. If no such process is present, a diagnostic dilemma may exist, and other etiologies of hypocalcemia with hypoparathyroidism should be considered. A 40-year-old female presented to the emergency department with nausea, vomiting, diarrhea, and abdominal pain. Her vital signs were stable on presentation other than slight tachycardia. Review of systems was positive for dizziness, weakness, and tingling in her hands. Physical exam was unremarkable other than focal tenderness to palpation in the peri-umbilical region. Lab work up was significant for a new hypocalcemia with a calcium level of 5.8 (reference range 8.4-10.4 mg/dl) and ionized calcium of 0.6 (reference range 1.10-1.40 mmol/L). During an admission two months prior for appendicitis, her calcium level was normal. CT abdomen/pelvis with contrast showed bowel wall thickening consistent with enteritis as well as enhancement of the peritoneal lining with free fluid showing evidence of peritonitis. There were multiple lesions in the liver that had increased in size and quantity since her appendicitis admission and also several sclerotic foci throughout the spine and pelvic bones. The patient was admitted to the medical service for treatment of suspected intra-abdominal infection and work up of her hypocalcemia. The patient was found to have a low normal parathyroid hormone level (21.3, reference range 12.0- 67.0 pg/mL) and normal vitamin D and D2 levels. Serum phosphorus level was elevated at 6.6 (2.5-4.8 mg/dL). She was started on oral calcitriol and calcium as well as a slow infusion of intravenous calcium per endocrinology recommendations. Despite receiving this therapy, her calcium level failed to improve. As her hospital course progressed, it was determined that her liver and bone lesions were due to metastatic lobular carcinoma of the breast. This case represents hypocalcemia with inappropriate hypoparathyroidism in the setting of occult malignancy. In patients that have refractory hypocalcemia and hypoparathyroidism without known glandular destruction, malignancy should be considered.

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Bartonella Subacute Bacterial Endocarditis Manifesting as Vasculitis

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Bartonella infection is a cause of subacute bacterial endocarditis that may initially manifest as glomerulonephritis with vasculitis findings.

A 71-year-old male with a history of trifecta aortic valve, coronary artery disease, diastolic heart failure, atrial fibrillation, and diabetes mellitus presented for bilateral lower extremity edema, rash, and acute kidney injury. He was admitted for further evaluation. The violaceous rash had migrated from his ankles to his calves over the last month. Biopsy demonstrated leukocytoclastic vasculitis. Autoimmune workup displayed a positive PR3, mildly low C3/C4 complements, elevated Cardiolipin IgG and IgM, elevated total IgG with a positive IgG Coombs. The C-ANCA, P-ANCA, and myeloperoxidase were negative. Blood cultures were repeatedly negative during his hospitalization. Outpatient initiated steroids were up titrated on admission with presumed glomerulonephritis in the setting of AKI and leukocytoclastic vasculitis. Worsening rash and renal function resulted in a kidney biopsy. Results demonstrated mild glomerular C3 dominant immune complex deposits and chronic renal injury without crescents, consistent with nephrosclerosis. He developed hypoxemia while supine during the biopsy, requiring short-term intubation. Echocardiogram revealed thickened prosthetic aortic valve leaflets, severe aortic valve stenosis, and a preserved ejection fraction. The left atrium and right ventricle were dilated with mild pulmonary hypertension. Bartonella henselae (>1:1024) and B. Quintana (1:512) IgG titers were positive. Aortic and pericardial biopsies exhibited strong positive Bartonella PCR. Steroids were discontinued, and doxycycline with IV gentamicin were prescribed. Hospital course was complicated by worsening AKI requiring hemodialysis and arteriovenous fistula placement. Continued worsening of his diastolic heart failure with rapidly progressive aortic stenosis lead to ascending aortic and mitral valve replacements with aortic root repair. He was discharged to a long-term care facility, where he recovered kidney function and eventually returned to independent living.

Subacute bacterial endocarditis (SBE) resulting from Bartonella can present with nonspecific symptoms with often delayed or even negative cultures. Autoimmune manifestations, such as glomerulonephritis and clinical vasculitis, can also occur from Bartonella. Appropriate pharmacotherapy can be delayed with these presentations and further complicated by the use of steroids when manifesting as an autoimmune process. Chronic infections, including SBE, can mimic ANCA vasculidities. Prior cases of Bartonella subacute bacterial endocarditis have been shown to demonstrate a variety of ANCA associated proteins. This case displays unusual characteristics of pauci-immune SBE, with negative ANCA other than PR3. The pauci-immune form and the PR3 positive only ANCA have few case reports and should be recognized as possible initial findings of SBE. Smaller studies propose that PR3 positive patients have a poorer prognosis. Knowledge of SBE manifesting as pauci-immune or largely ANCA negative infection can help clinicians consider Bartonella testing in patients with vasculitis manifestations and negative blood cultures.

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Immune-Related Adverse Events: More Than Just ITIS's.

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Introduction: Immune checkpoint inhibitors have shown remarkable results in treating a variety of metastatic solid tumors. While these advances are promising, the side effects are challenging. The adverse effects result in autoimmunity directed at any organ with some of the most common being dermatitis, pneumonitis, and thyroiditis. This contrasts with traditional cytotoxic chemotherapy which primary effects organs with rapidly dividing cells and occurs mostly within 1-2 weeks of therapy. We are increasingly seeing more irAEs as the use of immune checkpoint inhibitors has increased rapidly. Therefore, it is critical to recognize and manage side effects of these medications. Here we discuss two patients who presented with atypical reactions.

Case Presentation 1: Myocarditis and Myasthenia Gravis
An 81-year-old male with a history of recurrent renal cell carcinoma was started on nivolumab plus ipilimumab four weeks prior to presenting with severe fatigue, nausea, and blurry vision. He was found to have a troponin level of 75 ng/mL (reference <0.11) and a preserved ejection fraction but was in complete heart block. A left heart catheterization revealed no obstructive lesions. Cardiac electrophysiology was consulted and placed a temporary pacemaker. Ophthalmology was consulted for the blurry vision and their exam indicated likely ocular myasthenia. He was treated with high-dose steroids and may have initially improved but developed worsening respiratory failure and had a PEA arrest secondary to hypoxia. A myasthenia gravis panel showed a positive striated muscle antibody.

Case Presentation 2: Diabetic Ketoacidosis
A 60-year-old female with a history of non-small cell lung cancer who was started on pembrolizumab six months prior presented with urinary frequency and nausea. She had a glucose of 508 mg/dL, anion gap of 22, beta-hydroxybutyrate of 9.78 mmol/L (reference 0.02-0.27) and a pH of 7.126. Her only previous abnormal glucose level was a week prior which was 130 mg/dL. She did not have thyroid or pituitary dysfunction. She had an HbA1C of 6.9%, low C-peptide level, negative antibody testing and normal lipase. She was treated for diabetic ketoacidosis related to new autoimmune diabetes and discharged on insulin.

Discussion: Given the increasingly prevalent use of immune checkpoint inhibitors, internists need to be familiar with the adverse events presentations and management. Risk factors for irAE remain poorly defined. Recognition and initiation of therapy as well as changing therapeutic course in a timely manner is essential. Multiple adverse events can occur simultaneously and each need to be appropriately addressed. High-dose steroids are often but not always the answer. Patients with irAEs should be managed by a multidisciplinary team with pertinent expertise given the complexity of disease and multi-organ system involvement.

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Acute Portal Vein Thrombosis Post Laparoscopic Sleeve Gastrectomy

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Introduction: Portal vein thrombosis (PVT) can be a potentially devastating complication of laparoscopic sleeve gastrectomy (LSG). Portomesenteric thrombosis can lead to mesenteric ischemia or infarction which can be life-threatening.

Case Presentation: A 34-year-old African American female with a recent history of laparoscopic sleeve gastrectomy presented 10 days following surgery with increasing abdominal pain. The pain was present since the procedure but significantly worsened 3 days prior to presentation. She also complained of anorexia, nausea and vomiting. Her past medical history was significant for morbid obesity and obstructive sleep apnea. There were no hypercoagulable conditions in her family medical history. She did not use oral contraceptives or smoke cigarettes. Physical examination revealed tenderness in the right upper quadrant without peritoneal signs. Her recent surgical incisions were healing well, without erythema or drainage. Ecchymosis was seen in the left lower abdomen where she had been injecting lovenox as appropriately prescribed. There were no significant laboratory abnormalities. CT abdomen with intravenous contrast revealed an asymmetric irregular low-attenuation change along the course of the left portal vein branch of the liver indicative of portal vein thrombosis. She was started on a therapeutic dose of lovenox then transitioned to warfarin and discharged to the INR clinic.

Discussion: High clinical suspicion for PVT is required after LSG, given the non-specific presentation. Most patients tend to present with vague abdominal pain, nausea, and vomiting which can also be seen early in the normal postoperative course of LSG. Surgical manipulation of the portomesenteric vessels can predispose patients to PVT and is a known complication of splenectomy. Gastric bypass procedures have a slightly higher risk due to some manipulation of gastric and mesenteric veins, however, sleeve gastrectomy involves only the short gastric veins and therefore has minimal risk. Increased intraabdominal pressure with carbon dioxide pneumoperitoneum, the pro-inflammatory state associated with baseline obesity, and a steep reverse Trendelenburg position are believed to contribute to the prothrombotic state in the portomesenteric circulation during LSG. Risk factors include age, gender, smoking, hormone use, BMI, previous VTE, thrombophilia, immobility, pulmonary hypertension, and obesity hypoventilation syndrome. Though not based on randomized controlled trials, current data suggest that LMWH is most effective in preventing thrombosis with no increase in bleeding risk for bariatric patients. Higher dose LMWH in those with higher BMI is usually recommended. After diagnosis, therapeutic anticoagulation is necessary, along with appropriate treatment of possible predisposing conditions.

Conclusion: PVT is an uncommon complication following LSG with potentially life-threatening consequences. Presenting symptoms are often vague, and a high index of suspicion is required to confirm the diagnosis and begin prompt treatment.

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Recurrent Pleural Effusions and Elevated Parathyroid Hormone-Related Peptide: An Unusual Case of Sarcoidosis

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Sarcoidosis is a disease that has been established for several years, and it is a clinical, radiologic, and histopathologic diagnosis. The variable presentations of sarcoidosis, especially its ability to mimic malignancy, make it a diagnostic challenge. A 63-year-old male with a past medical history of stage 3 chronic kidney disease, type 2 diabetes mellitus, hypertension, and coronary artery disease presented with low back pain, unintentional weight loss, and recurrent pleural effusions associated with shortness of breath. On physical exam, the patient had decreased breath sounds in his left lung base. Labs revealed elevated serum calcium and parathyroid hormone-related peptide, but normal parathyroid hormone and vitamin D levels. His erythrocyte sedimentation rate and C-reactive protein level were elevated. As parathyroid hormone-related peptide is commonly associated with malignancy and the patient had localizing symptoms along with weight loss and hypercalcemia, he underwent MRI of his lumbar spine which showed salt-and-pepper appearance of the bone marrow consistent with multiple myeloma. His kappa to lambda light chain ratio was elevated, his serum protein electrophoresis showed polyclonal gammopathy, and urine protein electrophoresis was negative for monoclonal bands. CT of chest, abdomen, and pelvis was done to further evaluate for malignancy, and it showed scattered calcified granulomas in the spleen, large left-sided pleural effusion and no evidence of neoplastic process. Angiotensin converting enzyme level was normal. Due to recurrent, large pleural effusions, the patient underwent a therapeutic and diagnostic thoracentesis that was remarkable for lymphocytic exudate and significant amount of blood, without malignant cells on cytology. Pleural fluid analysis was consistent with sarcoidosis. The patient had a PleurX catheter placed. Bone marrow biopsy of the lumbar region revealed non-caseating granulomas suggestive of sarcoidosis. Patient was treated with intravenous fluids and started on prednisone 60 mg daily after which his serum calcium levels normalized. Drainage from his PleurX catheter continued to decrease during the hospital stay, and the catheter was removed two weeks after discharge. Patient continued to remain asymptomatic with no recurrence of pleural effusion.
This case highlights pleural effusions, parathyroid hormone-related peptide and MRI findings mimicking multiple myeloma as features associated with sarcoidosis. These are rarely reported findings of sarcoidosis, and recognizing them in association with the disease can allow for a more cost-effective and focused approach to management, especially because sarcoidosis can mimic malignancy.

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Knocked down and out by Cefepime

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Cefepime is a 4th generation cephalosporin that has an extended spectrum of activity against many gram-positive and gram-negative bacteria. Because of its rarity, cefepime induced neurotoxicity is a diagnosis that needs a very high index of suspicion to be made especially in patients with renal dysfunction.

A 51 year old gentleman with past medical history of ESRD on hemodialysis who presented to the ED with complaints of pain and drainage from his right foot and leg which was progressively getting worse for 3 days. Physical exam showed a full thickness wound to the plantar right 5th metatarsal with underlying fluctuance, crepitus and bogginess noted to the right forefoot, lateral foot along the 5th metatarsal and lateral ankle with malodorous discharge. Imaging demonstrated osteomyelitis of the small toe. Gas gangrene with osteomyelitis of the right foot was diagnosed and the patient underwent immediate excisional debridement with 5th ray amputation and fasciotomy. Blood cultures were negative. Wound cultures grew Citrobacter Amalonaticus, Enterococcus Faecalis and Klebsiella Oxytoca. The initial antibiotic regimen was Zosyn, Clindamycin and Vancomycin which were changed to Cefepime and Vancomycin on the second day. On day 4, the patient was noted to be lethargic, clumsy and disoriented with reduced strength and hyperreflexia in his upper and lower extremities and ankle clonus. Head CT did not reveal any acute intracranial abnormality. Cefepime was discontinued on day 5 with improvement noted on day 7 after 2 dialysis sessions on day 6 and 7 respectively. By day 8, the patient was back to his baseline.

The risk of cefepime induced neurotoxicity is high in patients on high dose cefepime, patients with renal dysfunction due to prolonged elimination and patients with systemic inflammatory states due to disruption of the blood brain barrier. The FDA has recognized the neurotoxic potential of cefepime and has included the risk of seizure in patients with underlying kidney disease. An extensive case series literature review that included 37 studies comprising 135 cases, of whom 80% had renal dysfunction reported that all patients exhibited altered mental status, with reduced consciousness (47%), myoclonus (42%), and confusion (42%). The mechanism responsible for these adverse events is thought to be due to a concentration dependent competitive GABA-A receptor antagonism leading to excitatory neurotransmission. Three-hour hemodialysis sessions can effectively remove 70% of a given dose of cefepime but may take up to 2-3 dialysis sessions for complete resolution of symptoms.

In conclusion, cefepime neurotoxicity should be suspected in patients who become encephalopathic while receiving cefepime, especially patients with risk factors like renal dysfunction. Additional findings of hyperreflexia and clonus which were not described in most case reports should also be noted highlighting the upper motor neuron component of cefepime neurotoxicity.

Statin-Associated Autoimmune Myopathy

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Introduction. Statins have revolutionized treatment of hypercholesterolemia and have improved cardiovascular outcomes for millions of Americans. Adverse effects of statins are usually mild, but data suggest that 1 in 10,000 people will develop a serious myopathy. In very rare cases, 2 in 100,000 patients will develop an autoimmune myopathy. This is characterized by muscle weakness, muscle-cell necrosis on biopsy, and the presence of autoantibodies against 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase.

Case Presentation. A 59 year old woman with a prior myocardial infarction on a high intensity statin presented to the emergency department (ED) with 4 months of progressive fatigue and myalgias. Three weeks prior, her primary care doctor checked a creatinine kinase (CK) level and it was 6,620 U/L (normal range 30 – 170 U/L). Her statin was subsequently stopped. Patient presented to the emergency department secondary to continued myalgias. A repeat CK drawn in the ED was 11,372 U/L and she was admitted for rhabdomyolysis. She was treated with aggressive fluid resuscitation and loop diuretics but her CK levels remained elevated > 11,000 U/L with normal renal function. As a result, a muscle biopsy was performed and consistent with necrotizing autoimmune myopathy. HMG-CoA reductase IgG antibody was elevated at 148 units (normal range 0 – 19 Units). Her muscle biopsy report revealed changes consistent with central core myopathy. She was referred to rheumatology for immunosuppressive therapy and set to begin on high dose prednisone, intravenous immunoglobulin (IVIG), and other immune modulating therapies. Patient tolerated well with improvement in functional status, however patient did expire in a separate admission due to complications of systolic heart failure.

Discussion. Statin-associated autoimmune myopathy is an exceptionally rare side effect of statin use, occurring in 2 of 100,000 patients treated with statins. The pathogenesis remains unknown, however a model suggests that statin-induced overexpression of HMG-CoA reductase in genetically susceptible patients may cause autoimmunity against HMG-CoA reductase. Few patients have spontaneous remission and most have to undergo immunosuppressive therapy which include high dose prednisone, methotrextate, mycophenolate mofetil, IVIG, and the monoclonal antibody Rituximab. Triple immunosuppressive therapy has been used to treat nearly half of all patients with statin-induced autoimmune myopathy described in literature.

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Disease or drug? Multi-organ failure in a patient with myasthenia gravis

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Azathioprine (AZA), a prodrug of the purine nucleoside analogue 6-mercaptopurine (6-MP), has been a useful immunosuppressant since its debut in the 1960s. Commonly reported adverse reactions to AZA include nausea and rash, however severe reactions including myelosuppression and hepatitis have also been reported. In this case, we present a 72-year-old male who suffered severe hypotension and multi-organ failure secondary to AZA toxicity.

The patient had a complex medical history including CAD, diabetes, and myasthenia gravis (on AZA) and initially presented to the hospital for 1 week of increasing fatigue, anorexia, "burning" epigastric pain, light-headedness and nausea. He denied fevers, cough, or other complaints upon presentation. On exam, he was afebrile and hypotensive to the 80s/40s, which responded well to fluid resuscitation. The remainder of his exam was unremarkable, without stigmata of liver disease including scleral icterus, jaundice, asterixis, or lower extremity edema.

Initial laboratory evaluation was significant for coagulopathy (INR 3.6), elevated aspartate aminotransferase (AST) and alanine aminotransferase (ALT) 90 and 127 U/L, total bilirubin 12 mg/dL, and direct bilirubin 7.4 mg/dL. A CBC revealed pancytopenia with a WBC count of 1.27 K/uL, platelets of 26 K/uL, and Hgb of 11.3 g/dL with macrocytosis. Electrolyte panel revealed acute kidney injury with elevated creatinine of 1.92 and BUN of 61. Right upper quadrant ultrasound, abdominal and chest CT scans were unrevealing. A hepatitis panel was ordered and positive for both anti-smooth muscle antibody (1:40) and hepatitis B surface antibody. At this time, it was revealed that while the patient had been stable on AZA for over a year, 4 months ago, his dose was increased from 200mg BID to 300mg TID. A liver biopsy showed evidence of acute hepatitis with cholestasis and no fibrosis, consistent with a drug-induced liver injury (DILI).

Initially, due to concern for infection, AZA was held, and the patient was started on empiric antibiotics. While off AZA, his laboratory evaluation showed modest improvements in total/direct bilirubin (8.6 mg/dL and 5.0 mg/dL), WBC count (10.88 K/uL) and platelets (213 K/uL), however his Hgb declined to 7.4 g/dL, presumably from hypotension-induced acute tubular necrosis, requiring hemodialysis. To confirm the diagnosis of AZA-induced liver toxicity, AZA metabolites were measured including the known hepatotoxin, 6-Methylmercaptopurine nucleotide (6-MMPN), which was elevated to 40,837 pmol/8x108 red blood cell, roughly 7x the upper limit of normal. Genetic testing for TPMT deficiency, which predisposes an individual to AZA toxicity, was negative.

This case demonstrates an incidence of multi-organ failure secondary to the accumulation of the toxic AZA metabolite 6-MMPN. Clinicians should be vigilant when prescribing azathioprine to reduce the risk of potentially life-threatening organ dysfunction. Awareness of this risk may also lead to early cessation of therapy in affected patients so that recovery can begin.

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Not All Fevers Are Due to Covid-19

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Staphylococcus lugdunensis, like other coagulase negative staphylococci, was previously viewed as a likely contaminant on blood cultures. However, several case reports and research studies over the past 20 years have demonstrated that it can cause severe infections ranging from soft tissue infections to prosthetic joint infections and endocarditis. Many of these studies also comment on the acute to sub-acute presentation for patients diagnosed with endocarditis secondary to S lugdunensis. Our case highlights a delay in diagnosis and treatment of S lugdunensis right-sided native valve endocarditis directly due to self-isolation in the era of Covid-19.

A 65-year-old female presented in December 2019 after several episodes of syncope. She was subsequently diagnosed with an infra-his block and treated with an implanted biventricular pacemaker. Five months later, she developed fever, chills, nausea, vomiting, and diarrhea for 37 days. Per the patient, she presented for in-person care twice during this period and was "turned away" and told to self-isolate for possibility of Covid-19. Two weeks later she presented to the emergency department for persistent fever, chills, and rigors. On initial presentation she was afebrile, normotensive, and not tachycardic. Physical exam was unremarkable. Labs demonstrated a neutrophilic predominant leukocytosis (11.7 K/uL) and elevated inflammatory markers, erythrocyte sedimentation rate (97 mm/Hr) and c-reactive protein (6.8 mg/dL, normal <0.8 mg/dL). A CT scan of the lungs showed a possible left lower lobe sub-segmental pulmonary embolus and numerous small nodules throughout the peripheral bilateral lung fields. Blood cultures grew coagulase negative staphylococci, later speciated as Staphylococcus lugdunensis and the patient was started on intravenous vancomycin. Culture sensitivities revealed a pan-sensitive strain and the patients antibiotics were deescalated to intravenous cefazolin. A transthoracic echocardiogram did not reveal any valvular disease or vegetations. Due to concern for a subacute pacemaker endocarditis, a transesophageal echocardiogram was performed and showed a 1.7 cm by 0.63 cm vegetation attached to the anterior leaflet of the tricuspid valve. The pacemaker and leads were extracted and two days later the fevers resolved. The patient was discharged on 6 weeks of intravenous ceftriaxone and is doing well.
This case demonstrates a delayed diagnosis and treatment of S lugdunensis native tricuspid valve pacemaker associated endocarditis due to self-isolation recommended during the era of Covid-19. It is important to remember S lugdunesis as a serious pathogen, especially in a patient with a recent device implant. It is dually important to educate patients and providers about Covid-19 so that there are not delays in diagnosis and treatment of other serious medical illnesses.

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An Atypical Presentation of Nivolumab Induced Hypophysitis in a Patient with Malignant Melanoma

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Hypophysitis is a rare condition caused by pituitary gland inflammation leading to hypopituitarism. Disease can be primary or secondary. Immune checkpoint inhibitors, (used for treatment of melanoma, renal cell carcinoma, lung, bladder, and head and neck cancers) are associated with secondary hypophysitis, particularly ipilimumab. This case emphasizes the importance of considering secondary hypophysitis with adrenal crisis secondary to nivolumab, an anti-PD1 immune checkpoint inhibitor.
Case A 72-year-old male with a history of chronic leukocytosis, primary hypothyroidism and malignant melanoma presented to an outside hospital with confusion and fever. He was taking nivolumab for metastatic melanoma and was due to complete his year-long treatment in 3 weeks. Evaluation for metabolic and infectious causes was negative and he was treated empirically with acyclovir, piperacillin/tazobactam, and vancomycin. He was subsequently discharged to inpatient rehabilitation, but confusion and agitation persisted, and he was readmitted to acute care. Extensive evaluation was negative, including routine chemistries, HIV testing, and blood and urine cultures. Chest, abdominal and pelvic CT was nonrevealing, and brain MRI was interpreted as chronic microvascular changes. An LP revealed WBC 4 (20% neutrophils, 60% lymphocytes, 20% eosinophils) protein 107, and glucose 59, but neurology consultants thought findings were secondary to a traumatic tap. CSF cultures were negative. A right chest port was removed, but fever persisted, and he was transferred to our facility for further evaluation. On admission, he was encephalopathic with temperature 100.7, BP 122/74, RR 18 and oxygen saturation of 93%. His WBC was elevated at 18.04 K/mcL with hemoglobin 11.6 g/dL. Chemistries showed elevated sodium of 151 mmol/L, potassium 3.7 mmol/L, creatinine 2.10 mg/dL, and glucose 119 mg/dL. Chest X-Ray was non-acute. A TSH was 58.2, free T4 0.9, ESR 44, CRP 77.2, ACTH <1.5, 8:30 am cortisol 2.3, TPO 8.4, thyroglobulin antibody <0.9, FSH 10.3, LH 14.7, and total testosterone of 90. Based on laboratory studies, secondary hypophysitis from nivolumab therapy was diagnosed. Steroid therapy was initiated, and his levothyroxine dose was adjusted. His encephalopathy resolved and steroids were tapered to a maintenance dose of prednisone 7.5mg daily. At 6 months, he was doing well.
Discussion: Hypophysitis secondary to nivolumab is rare; incidence is only 0.4-0.5% with nivolumab, compared to 3.2-13.6%, with ipilimumab. Headache and pituitary enlargement are less common in nivolumab-associated hypophysitis, and onset is later in the treatment course, thus, diagnosis can be delayed. In our case, treatment was delayed due to atypical presentation of persistent fever and encephalopathy, with no pituitary changes seen on MRI. Importantly, abnormal brain imaging and biopsy are not required for diagnosis; pituitary imaging is normal in approximately 25% of patients. Diagnosis of hypophysitis from nivolumab is clinical, based on studies indicating pituitary dysfunction and temporal relationship of symptoms with drug therapy.

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Drug Induced Acute Liver Injury-Fulvestrant can be Responsible!

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Background: Drug induced liver injury is one of the most common causes of acute liver failure in the united states. With an estimated annual incidence between 10 and 15 per 10,000 to 100,000 persons exposed to prescription medications. Adults are at higher risk of developing DILI than children and women may be more susceptible due to smaller size. Patients usually present with cytotoxic hepatocellular injury, cholestatic injury, or a combination of both. Histologic findings include hepatitis, cholestasis, steatosis. Here we present a case of DILI induced by Fulvestrant.

Case: An 81-year-old female presented with complaints of nausea, generalized weakness. She has a history of metastatic breast cancer. She received radiation therapy followed by adjuvant chemotherapy with anastrozole, four years ago. Recent bone scan revealed abnormal uptake compatible with metastatic breast cancer in bilateral ribs (L>R) and spine (mainly T10). She was started on Fulvestrant. She developed intolerance to PEG tube feedings and became increasingly weaker. She was admitted to the hospital for further evaluation. On admission, she was found to have elevated ammonia level, elevated liver enzyme levels. CT abdomen/pelvis remarkable for new ascites, retroperitoneal lymphadenopathy; no liver/splenic metastasis noted. MRCP showed no obstruction or masses. She was found to have a predominantly cholestatic liver injury with elevated alkaline phosphatase level. At this point, we considered Fulvestrant as the cause of liver injury as it was recently started. Given her poor prognosis, aggressive measures such as liver biopsy were not recommended by gastroenterology. Fulvestrant was discontinued and ammonia level trended down and her mentation improved.

Discussion: Fulvestrant is a steroidal antiestrogen that works as a direct antagonist of estrogen receptors. It is commonly used for the treatment of hormone-receptor positive metastatic breast cancer. DILI most commonly caused by hepatocellular injury (cytotoxic), cholestatic injury or a mixture of both. Serum bilirubin and tests of synthetic function can be abnormal in both conditions. Drugs associated with DILI may cause injury in a dose-dependent, predictable way or in an unpredictable (idiosyncratic) fashion. Idiosyncratic reactions may be immune-mediated or metabolic. Fulvestrant therapy can be associated with elevation of liver enzymes in 15% of the cases. Fulvestrant is mainly metabolized in the liver through cytochrome P450 enzyme. Hepatotoxicity can be caused by a toxic or immunogenic metabolic product of the drug.
Trending points in this case are:

- DILI should always be considered in a patient presenting with acute encephalopathy and elevated liver enzymes
- Fulvestrant can cause DILI
- When Fulvestrant is suspected as a cause of DILI, immediate discontinuation may result in clinical improvement

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Rapidly progressive glomerulonephritis with associated diffuse alveolar hemorrhage

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Pulmonary renal syndrome with respiratory failure secondary to diffuse alveolar hemorrhage can be a deadly complication of connective tissue disease. Knowing how to identify key aspects of a patient's history and clinical presentation, and then taking appropriate action may prove life-saving.

A 58 year old woman with a history of SLE and CKD stage 3 secondary to obstructive uropathy requiring intermittent hemodialysis presented to the ED with abnormal laboratory results.

She had seen her PCP one week ago with symptoms consistent with an URI and was started on doxycycline. She endorsed recent fatigue, chills, malaise, abdominal cramping, watery diarrhea and vomiting. On the day of presentation, laboratory evaluation had revealed creatinine of 23.49 on a baseline of 2.7 and BUN of 131. On physical exam in the ED, vital signs were stable and she appeared lethargic with dry mucous membranes. Exam was otherwise unremarkable. Laboratory workup revealed wbc 22.36, BUN 136, and Cr. 23.64. Urinalysis revealed large leukocyte esterase with elevated protein and pH 7.5. Urine microscopy revealed bacteria with elevated RBC and WBC. Chest CT revealed multiple bilateral pulmonary nodules and generalized ground glass airspace disease with patchy atelectasis at the posterior lung bases. We started her on IV vancomycin and cefepime, and admitted her to general medicine with concern for sepsis. Over the next several days, she had slow, but steady improvement in her condition.

However, on HD 12 she had an episode of hypercapnia requiring BiPAP and close monitoring in the ICU. On HD 14, she returned to the floor, but subsequently developed recurrent fever with continued oxygen requirement. We added Flagyl. Repeat CT chest revealed extensive airspace disease. We then proceeded with bronchoscopy, which revealed diffuse alveolar hemorrhage. We transferred her back to the ICU, starting her on IV solumedrol 500mg BID and nebulized tranexamic acid q8h.

She improved over the next few days on high dose steroids and was discharged on HD 26 in stable condition on PO prednisone 60mg with taper and PO cyclophosphamide 75mg. On follow up, she demonstrated improvement in functional status with decreased dyspnea. We plan to start her on rituximab for maintenance therapy after her steroid taper.

Pulmonary renal syndrome can develop from lupus, as is most likely in our case. Optimal treatment is based on the glomerular injury pattern found on biopsy, ranging from Class 1 to 5. For RPGN, evidence suggests high dose steroids and either cyclophosphamide or mycophenolate mofetil (MMF) for 2 to 6 months, then maintenance therapy with lower dose steroid and MMF or azathioprine. Rituximab may also be as effective as CYC for ANCA associated vasculitis, but data is limited. Plasmapheresis may also be considered in refractory cases.

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Renal Failure from Beta Blocker Bradycardia - A Bigger Picture of BRASH

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Introduction: BRASH (Bradycardia, Renal failure, AV node block, Shock, Hyperkalemia) is a condition which is difficult to treat unless identified correctly. In this condition, a patient with renal failure develops hyperkalemia in the setting of taking medication which blocks the AV node. The initiation of BRASH occurs when hyperkalemia occurs synergistically with the toxicity of AV nodal blocking medications. The resultant effect on the AV node is significant bradycardia which reduces perfusion to the kidney worsening the hyperkalemia and a feedback loop is initiated with potentially fatal effects. We present a case of BRASH in a critically ill patient with severe bradycardia and refractory hyperkalemia that was treated with early dialysis.
Case: A 76 year old Caucasian female with past medical history of chronic kidney disease, coronary artery disease with multiple stents, diabetes mellitus type 2, hypertension on Toprol XL, and hypothyroidism presented from nursing home for altered mentation. EMS arrived and found she was bradycardic with a heart rate of 30-40 BPM. Atropine was administered followed by transcutaneous pacing. In the ER, the patient was hypotensive with MAP < 65 and heart rate was between 40-50 bpm with an electrocardiogram revealing junctional rhythm. Pertinent labs included hyperkalemia with potassium of 6.4, acute kidney injury with creatinine of 3.96 (baseline 2.12) and significant acidosis with pH at 7.16 and TSH 2.01. Despite initial resuscitation measures, mental status continued to deteriorate resulting in admission to ICU and mechanical intubation. She was given calcium gluconate, insulin and dextrose, but her hyperkalemia persisted. Given refractory symptomatic hyperkalemia and kidney injury, dialysis was initiated urgently. Upon dialysis treatment, hyperkalemia and bradycardia resolved. Patient was then extubated the next day without further recurrence.
Discussion: BRASH was only relatively recently described as a unique diagnosis by Josh Farkas in 2016. To our knowledge, there have been nine reported cases of BRASH in literature. Geriatric age group is mostly affected since hypovolemia leads to kidney injury which is particularly common in elderly patients. Compensatory mechanisms to increase perfusion are inhibited by AV nodal blockers thus precipitating the feedback loop. The severity of illness also varies, but refractory shock can develop given the hypovolemia with inability to compensate. The existing ACLS protocol focuses solely on the potassium aspect and thus it does poorly in management of BRASH as it does not recognize the multifactorial cause contributing to decreased cardiac output with no compensation. It is suggested to focus on perfusion when treating these cases and thus pressor support should be considered promptly along with overall treatment of AV nodal block.
In conclusion recognition of BRASH and prompt initiation of dialysis for hyperkalemia refractory to medical treatment as in our case can be invaluable for treatment.

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Drug Rash with Eosinophilia and Systemic Symptoms associated with levetiracetam, a case report and review of the literature

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Multiple medications have been implicated in the precipitation of Drug Rash with Eosinophilia and Systemic Symptoms (DRESS syndrome). The most common class of medications to do so are anticonvulsants. The limited awareness regarding this side effect of levetiracetam can be life threatening in complicated cases.

A 40 y.o. male with past medical history of alcoholic liver cirrhosis, history of seizure disorder requiring ICU treatment, history of adrenal insufficiency due to corticosteroid withdrawal, and alcohol use disorder presented with a five-day history of diffuse rash. The patient stated that his symptoms first started on his left foot. He noticed an erythematous, itchy, warm, diffuse macular rash which spread from the foot to the soles and up his left leg. Patient initially presented to his Primary Care Physician (PCP) who attributed his rash to thrombocytopenia secondary to his liver cirrhosis. His PCP referred him to Hematology. The rash spread to his abdomen, chest, back, bilateral arms and his lips. He decided to seek acute care in the emergency department.

On physical exam, the rash was erythematous, non-pruritic, palpable, and blanching. Notable labs included normal white count with 8.6% eosinophils, IgE 945, absolute eosinophil count 1.0 (ref 0-0.4) and elevated D-dimer 1191. He underwent bilateral venous duplex ultrasonography that was negative for deep vein thrombosis (DVT). CT angiogram of chest showed bilateral pulmonary embolism. There was no history of recent travel, environmental exposures or changes to dietary habits. Of note the patient had recently been discharged after treatment for alcoholic liver cirrhosis complicated by seizures. At that time he was started on levetiracetam which was four weeks prior to this presentation.

The patient was treated with oral prednisone 40 mg twice daily. His steroid dose was increased to 250 mg daily for three days. Levetiracetam was held on admission. The patient was treated for his pulmonary embolism with a heparin infusion bridged to enoxaparin. His rash improved during his hospital stay. Levetiracetam was discontinued and he was started on lacosamide. He was discharged on a taper of prednisone 60 mg daily.

There are a very limited number of cases recorded in literature regarding the use of levetiracetam with DRESS syndrome. Routine monitoring is crucial to the proper management of patients initiated on levetiracetam as DRESS syndrome is frequently associated with anti-epileptic drugs. Typically, patients present with fever, hematologic irregularities, diffuse and severe skin changes, and organ involvement coalescing over a period of a few weeks. The patient in question developed the rash four weeks after initiation of an antiepileptic with elevated absolute eosinophil count. Though there is minimal literature associating this syndrome with levetiracetam, it is an important side effect to be discussed with patients upon prescription of this drug.

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Multiple Rare Manifestations of a Systemic Lupus Erythematosus Flare Related to Double Stranded DNA Antibodies

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INTRODUCTION: Systemic lupus erythematosus (SLE) is an inflammatory disease which can cause both direct autoantibody and indirect complement-mediated damage to nearly every organ system. Anti-dsDNA parallels renal disease, including lupus nephritis, and less frequently results in indirect neuropsychiatric symptoms from striatal encephalitis. Large pericardial effusions and atypical hemolytic uremic syndrome (aHUS) are rare manifestations of SLE and are associated with renal involvement.

CASE REPORT: A 34-year-old female with discoid SLE presented with angioedema, dyspnea, and pleuritic chest pain. Initial labs revealed thrombocytopenia (30x10^9/L) and acute kidney injury (AKI) with creatinine 1.82 mg/dL. She self-discontinued her immunosuppressants 6 months prior.

Anti-dsDNA was elevated at >1000 IU/mL with low C3/4. LDH was 2703 U/L, haptoglobin <10 mg/dL, and peripheral smear showed schistocytes. Urinalysis had 3+ hemoglobin and >300 mg/dL protein with microscopy revealing muddy brown casts, dysmorphic RBCs and RBC casts. TTP was ruled out with normal ADAMTS13. Renal biopsy was deferred due to thrombocytopenia. She received pulse dosed steroids, hydroxychloroquine and cyclophosphamide for lupus nephritis. Eculizumab was started empirically for management of aHUS. She became anuric requiring dialysis and eculizumab was discontinued due to lack of renal recovery. Her course was complicated by resistant hypertension likely associated with aHUS.

Electrocardiogram showed no findings of pericarditis but an echocardiogram revealed a large pericardial effusion with partial RV collapse consistent with early tamponade. A pericardiocentesis yielded 625ml of serosanguinous fluid which was ANA positive. Re-accumulation required repeat pericardiocentesis 3 days later.

A MR brain was performed for acute aphasia and altered mental status showing restricted diffusion in the bilateral caudate heads/bodies concerning for antibody-mediated striatal encephalitis. CSF analysis ruled out infectious meningitis and serum NMDA Ab was negative. Plasmapheresis and cyclophosphamide were started with improvement in neurologic status.

DISCUSSION: Large pericardial effusions are uncommon in SLE with an incidence of cardiac tamponade of less than 2.5%. Pericardial fluid may express antibodies and pathogenesis is believed to be immune complex-mediated. Large effusions are associated with aggressive disease such as nephrotic syndrome. Treatment involves steroids and immunosuppressants including cyclophosphamide.

Lupus nephritis is a recognized trigger for aHUS with a classical presentation of severe hemolytic anemia, thrombocytopenia, and AKI. aHUS is related to uncontrolled activation of the complement system and is associated with high mortality and progression to ESRD. Treatment with plasmapheresis and eculizumab significantly improve renal recovery and mortality. Severe hypertension may be associated with aHUS.

Antibody-mediated striatal encephalitis is due to anti-dsDNA antibodies entering the CNS and cross-reacting with NMDA receptor antigens resulting in excitotoxicity. This is rare, affecting 7% of patients with neuropsychiatric symptoms. Symptoms are nonspecific. CSF shows a lymphocytic pleocytosis. MRI shows bilateral FLAIR hyperintensity within the striatum without restricted diffusion or post-contrast enhancement. Patients generally respond to steroids and plasmapheresis.

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A case of Raoultella ornithinolytica urinary tract infection spontaneously resolving

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Introduction: Roultella ornithinolytica formally known as Klebsiella ornithinolytica belongs to the Enterobateriaceae family and is a gram-negative, oxidase-negative, histamine-producing non-motile encapsulated aerobic bacillus. It was re-named after the French bacteriologist Didier Raoult. It had been reclassified from K ornithinolytica with the use of new genetic technology. It inhabits aquatic environments and has been underreported in the past, however it is an emerging pathogen and now occasionally seen in hospital acquired infections, mainly systemic and urinary tract infections.

Case Presentation: A 76-year-old female with history significant for clostridium difficile and rheumatoid arthritis (taking infliximab and methotrexate) presented to the outpatient clinic complaining of dysuria. Vital signs and physical exam were normal. Urinalysis of a clean catch specimen revealed amber-colored, hazy urine with specific gravity 1.016, pH 5.0, positive nitrite, small (1+) leukocyte esterase. Microscopic urinalysis revealed 103 white blood cells per high power field without red blood cells. Urine culture revealed >100,000 colony forming units per milliliter of R ornithinolytica. Antibiotic susceptibility testing demonstrated resistance to ampicillin, indeterminate resistance to nitrofurantoin, and sensitivity to amoxicillin-clavulanate, cefazolin, ciprofloxacin, and trimethoprim-sulfamethoxazole. The patient was prescribed nitrofurantoin because of the patient's reported allergies to many antibiotics. However, due to concern for possible recurrent clostridium difficile colitis, the patient elected not to take any of the prescribed antibiotic. The patient continued to take phenazopyridine and maintained adequate hydration. Repeat urinalysis performed weekly for two consecutive weeks after initial presentation yielded resolution of pyuria. Urine cultures also obtained weekly for two consecutive weeks after initial presentation revealed no significant growth.

Discussion: In a retrospective study by Seng et al. at four hospital centers, R ornithinolytica was found to contribute to UTIs in 32%, respiratory infections in 24%, gastrointestinal infections in 14%, wound and skin infections in 13%, and bacteremia in 5% of the cases. In the same study, the mortality rate was 9% while there were no deaths attributed to a UTI. Seng et al. also looked at 69 case reports that showed a mortality rate of 20% in which 34-44% of the 69 cases involved bacteremia. This appears to agree with the well-known fact that most bacterial infections have a higher mortality rate in bacteremia than in UTI. Our case report illustrates the first time a patient resolved spontaneously with only symptomatic treatment from a UTI caused by R ornithinolytica. In addition, our patient was on both methotrexate and infliximab for rheumatoid arthritis. This suggests that mildly symptomatic UTIs caused by R ornithinolytica may be treated without antibiotics.

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Thyrotoxic Periodic Paralysis with Features of Anderson-Tawil Syndrome

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Introduction: Thyrotoxic periodic paralysis (TPP) is a rare manifestation of hyperthyroidism. Thyrotoxic periodic paralysis is more common in the Asian population; fewer cases are reported in Caucasian and black population. TPP is characterized by recurrent episodes of reversible muscle weakness and hypokalemia. Studies support that hyperthyroidism, hyperinsulinemia, and androgen stimulate Na+/K+ ATPase activity; also, increased thyroid levels inhibit K efflux channels, leading to cell trapping of K+ and subsequent alteration in skeletal muscle repolarization. Andersen-Tawil Syndrome (ATS) is a primary periodic paralysis that can present as an autosomal dominant or a sporadic disorder. Some cases of ATS are caused by a mutation in KCNJ2 gene coding for inward rectifier potassium channel, which stabilizes the resting membrane potential in skeletal and cardiac myocytes. Unlike TPP, ATS tends to affect multiple sites; including cardiac and skeletal cells, leading to its clinical presentation, a triad of hypokalemia, prolonged QTc, and facial and skeletal dysmorphism (low-set ears, mandibular hypoplasia, orbital hypertelorism).

Case Report(s): A 30-year old Caucasian male with a past medical history of periodic paralysis with no home medications presented to the emergency department with lower extremity and upper extremity weakness after drinking about ten cans of soda and energy drinks. Family history insignificant for a similar disease process and was only positive for maternal hypertension. Social history was unremarkable. On presentation, he was tachycardic, 105 beats/minute. On examination, he exhibited orbital hypertelorism, mandibular hypoplasia, 3/5 strength in the upper extremities bilaterally, 2/5 strength in the lower extremities bilaterally, and +1 on reflexes in the bilateral upper and lower extremities. Laboratory was significant for hypokalemia 2.2 meq/L, Magnesium 1.6 meq/L, P, a suppressed TSH level, elevated free T3 and T4, elevated Thyroid-stimulating immunoglobulins, elevated thyroid peroxidase antibody levels, spot urine calcium 12.0 mg/dl, spot urine phosphorus <4 mg/dl, and urine K to creatinine ratio 2.18. Initial EKG was remarkable for a manually calculated QTc interval using the Bazett technique of 537 msec. He was given a total of 140 meq of KCL during 48 hours of hospital stay and discharged on Methimazole and KCL tablets.

Discussion: Andersen Tawil syndrome is diagnosed based on a triad of Periodic paralysis (hypo or hyperkalemia), dysmorphic features, and cardiac abnormalities such as prolonged QTc interval, prominent U wave, and ventricular arrhythmias. Our case is unique highlighting the possibility of co-existence or triggering-relationships between TPP and Andersen Tawil, syndrome, with overlapping clinical features.

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Lance-Adams Syndrome: A Challenging Diagnosis and Management

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It is uncommon for a patient who survives cardiac arrest to experience significant neurologic impairment such as acute and chronic post-hypoxic myoclonus. This chronic post-hypoxic myoclonus is also known as Lance-Adams syndrome (LAS). LAS was first reported by Lance and Adams in 1963 after they observed muscle cramps characteristic of patients who survived cardiac arrest. Less than 150 cases of Lance-Adams syndrome have been recognized worldwide. We present a rare case of Lance Adams syndrome below.

A 41-year-old male with history of drug abuse and suicide attempts was admitted on 5/15/2020 after being found down and unresponsive. Cardiopulmonary resuscitation was done by EMS and he was given 12 mg of Narcan with minimal response. Urine drug screen was positive for opiates and cocaine. The patient was intubated and sedated. The patient was noted to follow commands off sedation and had diffuse intention myoclonic jerking. Patient was loaded with Keppra followed by maintenance dosing while he was hooked up to long-term monitoring for epilepsy. The patient was extubated after 4 days and was alert and oriented to person, time, and place. He did not have any neurological deficits but continued to have myoclonic jerking. Electroencephalography was reflective of a moderate to severe encephalopathy with no epileptiform discharges. MRI brain was unremarkable. CT thoracic and lumbar spine showed multilevel spondylosis but no fracture. Action myoclonus continued and the patient was discharged on Depakote due to concern of LAS.

Post-hypoxic myoclonus can occur both in acute and chronic types. It may occur after the patient remains in a hypoxic coma for several hours to days. As the patient regains consciousness, he may immediately develop action myoclonus, or it may develop in a short period of time. Patients can have action and reflex myoclonus, which subside with rest. Other symptoms, such as ataxia and mental status changes can frequently be seen. Cerebrospinal fluid analysis often shows decreased serotonin metabolites. The physiology of chronic post-hypoxic myoclonus is generally both cortical action and cortical reflex myoclonus. Failure to diagnose LAS may lead to inappropriate therapy and delayed treatment. If a patient develops intention myoclonus after receiving cardiopulmonary resuscitation and regaining consciousness but shows minimal response to anticonvulsants, a possibility of Lance-Adam Syndrome should be considered. This can lead to minimizing disabilities and improved prognosis. When a patient is suspected of having LAS, aggressive drug treatment to reduce the myoclonus and rehabilitation to prevent disabilities are necessary.

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Acute Myeloid Leukemia Presenting with Hyperleukocytosis

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Case Presentation: A 71-year-old male presented with an elevated white blood cell count in the setting of one month of recurrent infections. In the last month, he was treated for recurrent lung infections and diverticulitis with multiple courses of antibiotics. He was also treated for a pulmonary embolism and DVT. He presented again to an outside hospital with fever, chills and sweats and was found to have WBCs greater than 200x10^9/L. He was transferred for further workup. On additional history, he endorsed malaise, recent weight loss and five days of blurry vision. On exam, temperature was mildly elevated and left lower extremity edema and tenderness were noted. Labs revealed elevated WBCs of 168x10^9/L with 85% blasts. Hemoglobin was low at 10.4 g/dl and platelets were low at 47x10^9/L. LDH was elevated to 552/L. The initial concern was for chronic lymphocytic leukemia.

The workup included a peripheral smear significant for small mature looking lymphocytes. However, further review revealed results may also be consistent with small blasts. Peripheral flow cytometry showed an abnormal myeloid blast population with 90.8% blasts. Bone marrow biopsy was consistent with FLT3+ AML. During hospitalization, the patient developed chest pain, shortness of breath, a new oxygen requirement and worsening blurry vision, concerning for leukostasis. Treatment was promptly initiated with leukapheresis and hydroxyurea, resulting in a decrease in WBCs from a peak of 346x10^9/L to 198x10^9/L. He was then started on induction chemotherapy and WBCs had recovered to 1.83x10^9/L by discharge.

Discussion: This patient represents a unique case of AML presenting with hyperleukocytosis. Hyperleukocytosis is defined as WBCs greater than 50-100x10^9/L and is seen in 5-20% of patients with newly diagnosed AML. While leukocytosis may result from infection and inflammation, levels generally do not reach hyperleukocytosis. It is a risk factor for both high early death rates and high relapse rates. Hyperleukocytosis alone may have a nonspecific presentation. However, critical hyperleukocytosis can lead to leukostasis, a medical emergency with a 20-40% mortality rate.

Leukostasis is, most commonly, seen as respiratory and neurologic distress in patients with AML or CML in blast crisis. In our patient, specifically, this presented as a new oxygen requirement and worsening vision changes. Rigid blasts create white cell plugs in the microvasculature, resulting in decreased tissue perfusion. Cytokine production causes endothelial damage, further exacerbating tissue hypoxia. Leukostasis is frequently an empiric diagnosis that requires a high degree of suspicion for prompt treatment. While asymptomatic hyperleukocytosis may be treated with hydroxyurea alone, leukostasis necessitates cytoreduction. Chemotherapy is often combined with therapeutic leukapheresis to lower WBC count, as seen in our patient. Additional complications of leukostasis that must be monitored include disseminated intravascular coagulation in 40% of patients and spontaneous tumor lysis syndrome in 10% of patients.

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A late-delayed periodic hyperammonemic encephalopathy and normal anion gap metabolic acidosis secondary to ureterosigmoidostomy

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Introduction: Hyperammonemia is most commonly associated with liver disease, metabolic disorders such as urea cycle enzyme deficiencies, and medication-induced toxicities such as valproate. Hyperammonemia can manifest with neuropsychiatric symptoms, including confusion, lethargy, sleep disorders, hallucinations, ataxia, seizure, and even coma. Ureterosigmoidostomy, where urinary diversion to the colon is performed, has been rarely with delayed onset hyperammonemia. Periodic hyperammonemic encephalopathy is an extremely rare complication of ureterosigmoidostomy, which may occur even decades after ureterosigmoidostomy. The condition mimics hepatic encephalopathy but without underlying liver disease and metabolic disorders. We report a rare case of late-delayed periodic hyperammonemic encephalopathy related to ureterosigmoidostomy.

Case presentation: A 71-year-old male patient with a history of epispadias and bladder exstrophy treated with ureterosigmoidostomy shortly after birth, who presented with episodes of confusion and hallucinations for three years. During these three years, he was hospitalized multiple times for episodes of confusion when he was found to have hyperammonemia and was started on lactulose. However, He continued to have hyperammonemia and bouts of confusion and hallucinations. He was not on valproate. On further investigations, there was no underlying liver disease or metabolic disorders. He was also on sodium bicarbonate, which was started by his nephrologist for hyperchloremic metabolic acidosis that he had for years. The patient was started on rifaximin in addition to lactulose. His ammonia levels have since then decreased significantly, and his mental status was back to baseline.

Conclusion: We describe a rare case of periodic hyperammonemic encephalopathy as a late-delayed complication of ureterosigmoidostomy. However, the condition rarely occurs following ureterosigmoidostomy. It can be a potentially life-threatening condition. Early recognition is pivotal. Physicians should be aware of this condition, especially in the setting of absence of obvious underlying liver disease or metabolic disorders causing hyperammonemia in patients with ureterosigmoidostomy.

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Acute Pulmonary Histoplasmosis in an Immunocompetent Host after a Deadly Tornado Outbreak

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Background: Acute pulmonary histoplasmosis results after infection by Histoplasma Capsulatum, a fungus endemic to the Ohio and Mississippi River Valleys. Infection follows soil-disturbing events, most commonly landscaping and construction projects, but has only rarely been described following devastating natural disasters such as tornadoes. As a result, it is imperative to maintain sporulating fungal infections within the differential for acute respiratory illness succeeding natural disasters.

Case: A 26-year-old female with a medical history significant for obesity with body mass index 58, hypertension, hyperlipidemia, and tobacco dependence presented with a three-week history of progressively worsening shortness of breath, fever, and cough productive of thick yellow sputum. She was febrile, hypoxemic, and tachycardic. Her chest radiograph demonstrated bilateral pulmonary infiltrates. Initial laboratory studies were notable for leukocytosis (13.8 K/uL) and an elevated procalcitonin (0.24 ng/mL). Viral respiratory PCR panel, blood cultures, mycoplasma IgG/IgM assay, and urine Streptococcus and Legionella antigen assays were all negative. During the diagnostic evaluation she was treated with empiric antibiotic therapy for pneumonia without nosocomial risk factors. Her respiratory failure was treated supportively with heated high-flow nasal cannula oxygen therapy. Further diagnostic workup demonstrated negative urine Histoplasma antigen; however, serum antigen testing was positive, confirming acute pulmonary histoplasmosis secondary to H. capsulatum infection. HIV testing was negative and there was no evidence of disseminated disease. Antibiotics were discontinued and she began to recover by the time of diagnosis and did not require systemic antifungal therapy. Further questioning revealed that her workplace and surrounding buildings were struck by the multiple tornadoes that had ravaged the Montgomery County area, exposing her to debris from the immediate wreckage and providing the necessary inoculum to spur infection.

Discussion: Histoplasma capsulatum is a soil-dwelling fungus found world-wide and is endemic to the Ohio and Mississippi River Valleys. Most cases of acute pulmonary histoplasmosis have been reported in association with soil disturbing events such as large construction projects or major landscaping where individuals are exposed to soil contaminated with bird or bat droppings containing the fungal spores. Cases of pulmonary histoplasmosis associated with devastating natural disasters are rarely reported. On Memorial Day, May 27th, 2019, eighteen tornadoes touched down in Ohio setting a record for the most twisters in a single, local event in recorded history. The disruption to the environment harboring H. capsulatum was sufficient to disseminate fungal spores and provide an inoculum adequate to elicit acute pulmonary infection in an otherwise young and immunocompetent patient. This case highlights the importance of including sporulating fungal infections in the differential for respiratory illness after significant soil-disturbing events such as tornadoes and other natural disasters. Recognition of this can lead to earlier diagnosis and treatment of fungal infections that may otherwise be under recognized.

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Secondary Syphilis Mimicking Autoimmune Disease

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Syphilis has been a sexually transmitted disease of concern for decades, and its prevalence has been increasing over the last twenty years. Most patients present with secondary, latent, or tertiary syphilis. Secondary syphilis is known as a "mimicker" of many other disease processes such as viral hepatitis, autoimmune conditions, dermatologic conditions, or other infections. Here we present a less common case of secondary syphilis presenting as sore throat and oral ulcers in a patient with chronic hepatitis C and positive autoimmune markers.

A 41-year-old female with a history of untreated chronic hepatitis C presented to clinic with the complaint of a sore throat and a non-pruritic, non-painful rash on her forehead for one month after multiple trips to the emergency department. Rapid streptococcal PCR was negative twice. She was noted to have crusted, erythematous lesions on her forehead, and back as well as oral ulcers inside her bottom lip and on her tongue. Symptoms had failed multiple treatments including nystatin swish and swallow, oral steroid rinses, hydroxychloroquine, and oral prednisone as per ENT, rheumatology, and dermatology recommendations. Autoimmune work-up revealed positive ANA and RNP antibodies. Biopsies of lesional and perilesional oral mucosal ulcers revealed findings of chronic inflammation and spirochete organisms on immunohistochemistry. She subsequently had a positive syphilis total antibody and positive RPR. Due to a penicillin allergy, she was instead treated with two weeks of doxycycline with improvement of her rash and sore throat.

Secondary syphilis is often considered the "great imitator" of many different diseases as it can affect almost every organ system. Classically it presents as painless macules across the trunk, extremities, palms and soles. Other symptoms include fatigue, myalgias, headaches, kidney injury, or hepatitis. Dermatologic presentations can often be overlooked especially in patients with little to no risk factors. These lesions may present as plaques, ulcers, or gummata, and can overlap with rashes found in autoimmune conditions such as lupus erythematous (SLE), drug-rash, impetigo, pemphigus vulgaris, and lichen planus.

It can also be safely assumed that coinfection of syphilis and viral hepatitis B or C is not uncommon given their similar risk factors. Hepatitis C has complications and extrahepatic manifestations that have the potential to overlap with signs of secondary syphilis. Hepatitis C has also been shown to be associated with autoantibodies including ANA or RF. Thus, patients co-infected with hepatitis C and syphilis can present in a multitude of ways and complicate the clinical scenario or be mistaken for autoimmune conditions. Thus, syphilis should always remain in the differential diagnosis in any patient who is being evaluated for dermatologic or autoimmune conditions. Its incidence is rising, and it can mimic so many other diseases, which may lead to delayed diagnosis.

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Euglycemic DKA in the a patient with Cyclical Vomiting Syndrome

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As diabetic guidelines evolve prescription of SGLT-2 inhibitors will likely increase. Euglycemic DKA is a known complication of SGLT-2 inhibitors and predicting which patients are at risk should be considered.

In this case, a 54 year old Female presented to ED with acute on chronic nausea and vomiting. She did have previous episodes in the past, typically 4 days in duration while being asymptomatic between episodes. Prior outpatient workup had been unrevealing. Pertinent past medical history also included a history of type 2 diabetes mellitus previously on insulin which had been discontinued about 6 months prior to presentation. Current diabetic regimen included metformin and empagliflozin.

Venous blood gas revealed a pH of 7.068 with pCO2 of 28 and HCO3 7.9 as well as a lactate of 2.1, concerning for mixed anion gap / respiratory acidosis. Additional testing performed in ED included urinalysis with ketones >80 mg/dL and urine glucose of 500 mg/dL. Serum glucose level was 142. Beta-hydroxybutyrate was also significant at 9.57 mmol/L. Workup also revealed an osmolar gap of 14 with volatile alcohol panel significant for acetone of 30.

She denied any prior ingestion. Salicylate and acetaminophen levels were undetectable. Differential also included starvation ketoacidosis however acidosis in starvation ketoacidosis is typically not as profound with a pH typically above 7.30 and bicarbonate above 18.

She was started on continuous insulin infusion with half normal saline / dextrose maintenance fluids. Subsequent chemistry ~24 hours following presentation revealed bicarbonate level of 20 with closed anion gap. The patient was transitioned off of insulin drip with long-acting subcutaneous insulin.

Given ketonemia with ketonuria and serum glucose <200 euglycemic DKA had been the suspected diagnosis. Euglycemic DKA has been associated with SGLT-2 inhibitor use, often in the setting of illness or fasting.

While the mechanism for euglycemic DKA in SGLT-2 inhibitor use has not been clearly elucidated, it is suspected to be similar to hyperglycemic DKA with the combination of insulin deficiency / resistance and glucagon excess pushing toward ketone production. Ketogenesis is further promoted and hyperglycemia is not observed due to the renal glucose clearance promoted by the SGLT-2 inhibitor. Euglycemic DKA is typically triggered by stress or illness due to increased insulin demand or fasting due to less available carbohydrates.

In this case the patient was at increased risk for euglycemic DKA due to her fasting state in the setting of cyclical vomiting syndrome. Furthermore, given that her diabetic control previously required insulin, there is further suspicion that she was insulinopenic at baseline.

The diagnosis of euglycemic DKA requires a strong clinical suspicion, as does the identification of those at increased risk.

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Atypical Manifestation of Atypical Mycobacteria - Recurrent Orogenital Ulcerations

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Introduction: Atypical or Non-Tuberculous Mycobacterium (NTM) are a group of environmental mycobacteria consisting of numerous species that are known to cause wide variety of syndromes. Pulmonary disease and disseminated manifest in immunocompromised patents. Lymphadenitis and skin and soft tissue infections (SSTIs) can be found in immunocompetent individuals exposed to trauma, surgery, marine/lake water exposure and have increased in incidence over the last decade with around 2 per 100000 person years. These patients have non-specific clinical presentations making diagnosis difficult. Here, we present a rare oro-genital presentation of SSTIs caused by NTM.
Case: A 38-year-old male, with history of brief imprisonment had experienced recurrent maculo-papulo-nodular skin lesions intermittently for 1-year with episodes of oro-genital ulcers that resolved by itself. He presented currently with 2-month history of intermittent non-specific symptoms of arthralgia, low-grade fever, night sweats, weight loss, tender swelling in the anterior neck (possible lymphadenitis not elicited on presentation). He was found to have painful testicular swelling (Unremarkable ultrasound except for skin thickening) with non-draining ulcers, and oral ulcers. Infectious disease work-up was negative for HIV, sexually transmitted infections, HSV, other cutaneous viruses, Hepatitis B&C and TB QuantiFERON test. ANA positive with no other significant antibody titers. Biopsy of scrotal ulcer revealed rare multiple acid-fast bacilli with beaded appearance associated with deep ulcerations, non-granulomatous, leukocytoclastic vasculitis, folliculitis, and cellulitis. This threw a surprise diagnosis of Atypical Mycobacteria given the pathology and negative TB QuantiFERON testing. Unfortunately, sample was insufficient for Paraffin block PCR to detect the species. Patient had all scrotal ulcers removed for biopsy and no residual ulcer left, he was treatment with a short course of Doxycycline and has been doing well since.

Discussion: SSTIs caused by NTM have varied presentations including papules, nodules, plaques, ulcers, panniculitis, cellulitis especially in trunk and extremities. NTM infection presenting in an immunocompetent individual as recurrent oro-genital ulcers along with non-specific B symptoms is a unique manifestation. Most cases reveal granulomatous histopathology, which are pathognomonic of atypical mycobacterial infections, but our patient's biopsy was non-granulomatous in nature. This was reported in few patients with early Mycobacterium ulcerans (M. ulcerans) infection which could well be the culprit in our case. M. ulcerans is known to cause large Buruli ulcers which is not seen in our patient. Also, leukocytoclastic vasculitis associated with NTM SSTIs has not known to be reported. NTM infections are known to be unresponsive to common antibiotics and often require longer duration of treatment. However, removal of ulcers and short course antibiotics have shown success in our patient. The non-specific multi-systemic symptoms mimics other infections and rheumatological diseases, however suspicion/differential of NTM is important so initiation of early testing, diagnosis and treatment can be offered thereby preventing complications.

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A CASE OF CHLAMYDIA INFECTION IN ASSOCIATION WITH FOURNIER'S GANGRENE

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Fournier's Gangrene is a urologic emergency characterized by necrotizing fasciitis of the perineal, perianal or gluteal areas. The underlying infection is polymicrobial, the organisms usually being commensal bacteria of the surrounding gastrointestinal tract or perineum. This case describes a patient with Fournier's Gangrene and identification of Chlamydia Trachomatis in the urine, which is not a typical causative organism. Chlamydia Trachomatis should be considered in the spectrum of disease-causing organisms in high-risk patients.

We are reporting a case of eighty-five-year-old male, nursing home resident, with a significant past medical history of peripheral arterial disease status post bilateral above-knee amputations presented with dysuria, penile discharge, and scrotal pain for one day. He is sexually active with one female partner and the last sexual encounter was one month prior to the admission. He participated in sexual encounters without any protection. The patient was septic on evaluation, with a temperature of 39.1, pulse rate of 101 beats per minute, respiration of 22 breaths per minute, and blood pressure of 126/69. On physical examination, there was diffuse swelling of penis and scrotum with overlying erythema, exquisitely tender to palpation, no relief in pain with scrotal elevation. There was no discharge at the urethral meatus. No crepitus was appreciated. Laboratory workup was significant for leukocytosis of 20.7 K/uL with 11% band neutrophils. Lactic acid was elevated at 2.1. Urine and blood cultures were taken prior to the initiation of antibiotic therapy. Urine culture grew Providencia Sutartii and blood culture was positive for Staphylococcus Pettenkoferi. Nucleic acid amplification testing (NAAT) performed on the urine sample was positive for Chlamydia Trachomatis. He was initially managed for scrotal cellulitis, urinary tract infection, and sexually transmitted infection with Ceftriaxone and Doxycycline. On day two of admission, the patient was noted to have scrotal gangrene. He received urgent scrotal debridement and commenced on broad-spectrum antibiotics including Vancomycin, Clindamycin, and Doxycycline. Scrotal tissue microscopy confirmed the presence of edema, necrosis and inflammation. The debrided scrotal tissue was cultured which grew Enterococcus faecalis and Enterococcus Raffinosus. He had an uncomplicated postoperative recovery in the intensive care unit and was transferred to the long term acute care facility for continued wound management.

The culture of the debrided tissue is the most reliable method of determining the bacterial organisms which should be covered with antimicrobial therapy when treating Fournier's Gangrene. Chlamydia Trachomatis however, is not easily cultured. It can be detected via NAAT which is simple, quick, and accessible. Chlamydia Trachomatis and other sexually transmitted bacterial infections should be considered as possible causative organisms, especially in the setting of high risk sexual behavior. This would allow for the early utilization of adequate antimicrobial coverage.

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A case of myxedema coma with acute heart failure

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A 42-year-old female with a history of Grave's disease status-post radioiodine ablation and subsequent hypothyroidism presented with progressive shortness of breath for a week. She was prescribed levothyroxine for her hypothyroidism but had self-discontinued all medications six months prior at the suggestion of her pastor.

Her physical exam was remarkable for ¬somnolence, elevated jugular venous pressure, diffuse nonpitting edema including her face and hands, and cool extremities. Cardiac exam revealed tachycardic rate with normal S1 and S2 and no murmurs. Vitals were remarkable for severe hypotension at 90/60. The patient quickly required intubation and mechanical ventilation to protect her airway, in addition to norepinephrine and dobutamine support to improve her arterial pressure.
The patient was subsequently admitted to ICU. EKG showed sinus tachycardia. Notable labs included troponin within normal limits, elevated BNP, TSH five times greater than the upper limit of normal, and an undetectably low T4. Estimated Fick cardiac index was low at 1.4L/min/m2. Echocardiography showed severe global hypokinesis of the left ventricle with ejection fraction less than 20%. Atria were normal and there was a small pericardial effusion present. Left heart catheterization was performed after the patient was stabilized and showed mild diffuse disease in all three coronary arteries; no intervention was required.

She was diagnosed with acute cardiogenic shock due to myxedema coma and required extensive supportive care in the ICU including dobutamine, steroids, and intravenous thyroid hormone replacement. After three weeks in the hospital, thyroid labs normalized and she was stabilized on inotropic support. However, she was unable to be weaned entirely and discharged on ambulatory inotropic support.

Considering the broad reaching effects of thyroxine in organ regulation, it is no surprise that hypothyroidism can produce cardiovascular side effects. Typically, these are bradycardia, pericardial effusion, peripheral edema, and increased vascular resistance with hypertension. Hypothyroidism can also impair cardiac contractility via decreased expression of Ca-ATPase in the sarcoplasmic reticulum. However fulminant clinical heart failure without pre-existing cardiac disease is very rare. Our patient had no prior heart disease, yet she presented with acute cardiogenic shock and profoundly low cardiac output.

In cases of acute heart failure from myxedema coma, systolic dysfunction is typically reversible with thyroid replacement therapy in as few as four weeks. While we generally expect clinical improvement within 6 months of normalized TSH and T4, our patient continues to manifest chronic systolic heart failure requiring inotropic support. However, medical optimization is ongoing with the continued hope of systolic recovery. Should cardiac function not improve, then the myxedema coma may have induced significant remodeling or a more permanent cardiomyopathy, which would be a very rare pathophysiologic process.

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Not Your Typical Streptococcal Meningitis

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Group B streptococcal meningitis is a bacterial infection caused by Streptococcus agalactiae (GBS), a gram-positive coccus that is found usually in the gastrointestinal and genital tract. GBS meningitis is more prevalent in neonates, but is an infrequent cause of meningitis in adults, both immunocompromised and immunocompetent. This report presents an uncommon case of adult meningitis with concomitant bacteremia.

A 76-year-old Caucasian male with a history significant for alcoholic cirrhosis and chronic kidney disease presented from home with new seizures. In the emergency department, his seizure was refractory towards anti-epileptic medications. He required intubation and was admitted to the ICU. Initial CT head showed no acute process. Blood cultures were sent, and he was empirically covered with cefepime 2 mg every 8 hours, vancomycin 15 mg/kg every 12 hours, and acyclovir 10 mg/kg once. Corticosteroid was also administered. The ICU physician performed a lumbar puncture, and CSF analysis was significant for neutrophilic predominance of 98%, a protein level of 3056.5 mg/dL, and a glucose less than 20 mg/dL. MRI of the brain was interpreted as "laminar ischemia, which may have been a sequelae of his status". A meningitis panel detected GBS, which also grew on previously collected blood and urine cultures. At this point, the empiric antibiotics were deescalated to ampicillin 2g every 4 hours. Over the remaining ICU stay, sputum culture grew Methicillin-resistant Staph aureus and he was treated with linezolid 600 mg every 12 hours. His CXR also displayed worsening atelectasis, which followed with respiratory decline and unresponsiveness despite sedative discontinuation. The hospital's palliative care team was consulted for goals of care discussion with the family. On hospital day 13, the patient was made DNR and he expired 2 days later in the hospice general inpatient care.

This case illustrates an uncommon manifestation of invasive GBS in a non-pregnant adult with a severe medical condition. GBS meningitis is a unique cause of bacterial meningitis because GBS does not readily penetrate the blood-brain barrier; it accounts for about 4% of all meningitis in the elderly population. Physicians need to recognize the abrupt nature of this illness, especially in the elderly population with multiple comorbidities, as it is associated with concomitant bacteremia and most importantly, a high case-fatality rate. Among adults > 65 years of age, the case-fatality rate was as high as 56%.

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MDA-5 Dermatomyositis in the Internal Medicine Clinic

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Introduction: MDA-5 positive dermatomyositis is a rare amyopathic dermatomyositis characterized by melanoma differentiation-associated gene 5 (MDA-5) antibodies and is associated with aggressive interstitial lung disease (ILD). We present a case of 53-year-old African American man who was initially evaluated by his internist for facial erythema. He was diagnosed with MDA-5 positive dermatomyositis associated with rapidly progressive ILD and succumbed to this disease.

Case Presentation: Our patient initially presented to his internist with a 5-month history of facial erythema, unimproved with antibiotics and steroids prescribed by an urgent care provider. He was referred to a dermatologist who diagnosed cutaneous lupus erythematous after a skin biopsy showed interface dermatitis. The patient subsequently presented to his internist with polyarthralgias and was then referred to Rheumatology.

The patient was admitted to the hospital from this Rheumatology appointment due to concern for respiratory failure related to amyopathic dermatomyositis associated with ILD. Along with facial erythema and polyarthralgias, his constellation of symptoms now included dyspnea and night sweats. His exam findings were notable for characteristic skin lesions, such as digital ulcers, Gottron's papules, "mechanics hands," and palmar papules, along with normal muscle strength. Rheumatologic serologies including ANA, ENA battery, anti-Scl 70, Jo-1 Ab, and ANCA were negative. His CK level was within normal limits. High resolution CT of chest revealed airway inflammatory changes and lymphadenopathy. Per patient preference, he left the hospital prior to the completion of his evaluation.

After his discharge, the patient's myositis panel returned with elevated levels of MDA-5 and aldolase confirming the diagnosis of MDA-5 positive dermatomyositis. The patient was readmitted to the hospital due to worsening dyspnea which improved with high-dose steroids. He was started on mycophenolate mofetil since he declined treatment with Rituximab.

Thereafter, the patient's respiratory status declined, and he was soon admitted to the MICU with respiratory failure. Amid intravenous steroids, broad spectrum antibiotics, and intravenous immunoglobulin, his respiratory status failed to improve, and the patient expired.

Discussion: MDA-5 dermatomyositis is an inflammatory myopathy that has a strong association with ILD. Even with treatment, the 6-month mortality rate is 60 percent due to respiratory involvement. This is a rare case of MDA-5 positive dermatomyositis associated with rapidly progressive ILD which emphasizes the importance of early recognition of rheumatological diseases by primary care physicians. As demonstrated in our case, classic cutaneous manifestations of dermatomyositis and retained muscle strength are characteristic of this disease. This patient's internist appropriately identified the initial manifestations of this disease and referred the patient to appropriate subspecialists. Unfortunately, despite this prompt recognition, referral, and treatment, our patient passed, highlighting the graveness of this disease.

Kreb's Cycle Not Just for Undergrads: A Case of Thiamine Deficiency Causing Type B Lactic Acidosis

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We present a case of a 61-year-old man who presented with difficulty in ambulation, weakness, numbness, and frequent falls progressive over a three month period. He was found on initial presentation to have a profoundly elevated lactate level. The patient endorsed a history of heavy daily alcohol use and poor oral intake for the past three years. His physical exam was pertinent for heart rate of 113 bpm, blood pressure of 138/81 mm Hg, tremulousness, diminished sensation to light touch in the bilateral lower extremities with associated truncal ataxia, and difficulty ambulating. His sensorium was clear. Laboratory investigation was significant for macrocytic anemia with Hgb 12g/dL (13.9-16.3 g/dL), Hct 33.9% (41.0-53.0%), MCV of 115fL (80-100 fL), and normal WBC count of 5.1 K/uL (4.5-11.5 K/uL). Other significant laboratory values included B12 of 87pg/mL (>300 pg/ml), folate of 1.7 ng/mL (5.9-24.7 ng/mL), lactate of 13.5 mmol/L with elevated anion gap of 28, and positive ethanol level on urine toxin screen. A lactate was repeated after intravenous crystalloid administration and remained elevated at 12 mmol/L. Thiamine deficiency was suspected given his alcohol use disorder and poor nutritional intake and was aggressively repleted parenterally. A repeat lactate value fell to 4 one hour after thiamine administration. Vitamin B12 and folate were also supplemented after return of these values. In the days following admission, the patient had substantial improvement in his neuropathy with vitamin B12, folate, and thiamine repletion. Lactate levels completely normalized within 24 hours of admission. An MRI of the spinal cord was done to assess posterior columns and was found to be normal. He was subsequently discharged to a skilled nursing facility with gradually improving neurologic abnormalities.

Lactate is a normal byproduct of carbohydrate metabolism with levels exceeding 2 mmol/L considered elevated. Most lactic acidosis reflects inadequate tissue perfusion, known as Type A lactic acidosis. Type B lactic acidosis, in which tissue perfusion appears intact, is less common. Type B lactic acidosis may be caused by, e.g., malignancy, diabetes, and beta-adrenergic agonists, via mechanisms not completely understood. Thiamine deficiency represents a rare but well-recognized etiology of Type B lactic acidosis with a pathway that is well-elucidated. Thiamine is an essential cofactor for the enzyme pyruvate dehydrogenase. Its deficiency may block conversion of pyruvate to acetyl-CoA prior to entry into the Kreb’s cycle leading to potentially profound accumulation of lactate. Parenteral administration of thiamine has previously been documented to dramatically correct lactic acidosis, as occurred in our patient. Thus, it is important for clinicians to consider the use of this simple and benign therapy in the setting of severe lactic acidosis that is otherwise unexplained.

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Archnidism Induced Saddle Pulmonary Emboli, An Unusual Complication of Furrow Orb Spider Bite.

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Introduction: The spider bites often resolve quickly without leaving complicated outcomes. Species such as the black widow spider (Latrodectus) and rarely brown recluse spider (Loxosceles) are capable of causing necrotic and systemic manifestations in humans. The Furrow orb spider (Larinioides cornutes) bites are typically superficial bites that do not require medical attention. Spider bites can be fatal due to cytotoxic and hemotoxic effects of envenomation. The underlying pathomechanism involves the primary toxin Sphingomyelinase D which dysregulates the coagulation cascade. We report a case of saddle pulmonary embolism secondary to Furrow Orb spider bite.

Case: A 26-year-old male with no known comorbidities presented to the emergency department with sudden onset shortness of breath and pleuritic chest pain. 1 week ago, he was bitten by a spider over the groin area, following which he developed swelling and pain in his right calf and he also felt a cordlike structure evolving on his right lower extremity. He experienced chest pain, intermittent fevers, and chills. History and blood work was unremarkable for inherited coagulation disorders. The patient was found to be tachycardia, with elevated JVD, hypoxia, and cyanotic lips. Lab work showed elevated troponin and D- dimer. CT PE revealed saddle pulmonary emboli in the distal main pulmonary artery extending into bilateral multiple segmental and subsegmental branches of all pulmonary lobes with evidence of right heart strain. Bilateral lower extremity venous ultrasound showed venous thrombosis extending from gastrocnemius into the external iliac vein. The patient underwent emergent mechanical thrombectomy, IVC filter placement, and discharged on oral anticoagulation.

Discussion: Earlier studies have shown that spider bite can cause dermo-necrosis, arterial thrombosis, and gangrene due to cytotoxic effects of the venom and there are no reported venous complications. Our patient had a unique presentation of acute onset and rapid progression of extensive venous thrombosis. In addition to this, another distinctive element, in this case, is the species of the spider, Furrow Orb, which is not known to cause medically significant bites. Hence, management should comprise wound care, close monitoring for thrombotic complications, and educating patients about the consequences of a spider bite to seek early medical attention. A review of the literature has shown that physicians should keep a low threshold for antivenom administration. In conclusion, Knowledge of this potentially rare complication of a spider bite can help clinicians in early detection and timely intervention so that rare fatal complications like PE can be prevented.

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An Unusual Case of Syncope

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Introduction: Syncope is a highly prevalent condition accounting for about 250,0000 hospital admissions each year. This is a case of Syncope which turned out to be Waldenstrom's Macroglobulinemia (WM), a rare and indolent B-cell lymphoma characterized by monoclonal IgM overproduction. Hyperviscosity Syndrome is a common sequelae of WM that can manifest with neurologic symptoms such as syncope and visual disturbances.

Case Description:

A 63 year-old female with Hypertension presented to the ED following a syncopal episode. She admitted to recurrent episodes for two months with blurred vision. No preceding prodrome of palpitations, light-headedness or warmth was noted and recovery was spontaneous without sequelae. She also reported fatigue, night sweats, weight loss, generalized weakness and dyspnea without chest pain. A previous cardiac history was denied. On exam, she was tachycardic and appeared cachectic with mucosal pallor and splenomegaly. Her visual acuity was impaired, but she had no motor or sensory deficits. Initial work-up revealed a normal serum glucose. Emergent brain CT and EKG were unremarkable. Lab data revealed pancytopenia (Hb 3.2g/dL, platelets 28K/uL, WBC 3.9k/uL) and peripheral blood smear showed plasmacytoid lymphocytes and normochromic red cells with rouleaux formation. She was admitted to the ICU for stabilization and transfused with three units of packed red blood cells, improvement in her dyspnea and fatigue ensued. An elevated serum protein-albumin gap of 9.6 (total protein 11.7gm/dL, albumin 2.1gm/dL) was also noted and subsequent SPEP and Immunofixation revealed an M-spike of 4.9 g/dL with significantly elevated IgM (11.9 g/dL). Serum viscosity was elevated at 6.2 Centipoises. A tentative diagnosis of Hyperviscosity Syndrome secondary to IgM Paraproteinemia was made and the patient was transferred to a tertiary center for emergent plasmapheresis. Fundoscopy done on site showed dilated, tortuous veins suggestive of retinal vein occlusion. Following 3 days of plasmapheresis, serum IgM level decreased to 3.5mg/dL with resolution of her visual disturbance. Bone marrow biopsy confirmed B cell lymphoma with 20% plasma cells. MYD88 L265P gene mutation was detected, confirming the diagnosis of WM and treatment with Bendamustine was commenced.

Discussion: WM is a rare disorder with an incidence of approximately three per million per year. Typical presentation is insidious and nonspecific with symptoms such as weakness, anorexia, night sweats and neuropathy. Syncope as a presentation of WM could therefore be overlooked and attributed to the more common orthostatic, neurologic and cardiogenic etiologies. Clues that led to the diagnosis of WM in this case were associated pancytopenia, high protein-albumin gap, M-spike on SPEP and an elevated serum viscosity. This case therefore highlights the importance of a detailed clinical evaluation, even when faced with often nonspecific symptoms.

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A Case of A Rapidly Enlarging Neck Mass: A Clinical Conundrum

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Introduction: Primary thyroid lymphoma (PTL) is a rare disease, accounting for only 1-2.5% of all lymphomas and 2-5% of thyroid malignancies. We present a case of a patient known to have Hashimoto's thyroiditis (HT), presenting with a rapidly enlarging thyroid mass, causing compressive symptoms. On further workup, the patient was found to have a biopsy-proven diffuse large B-cell lymphoma (DLBCL) of the thyroid, treated with chemotherapy.
Case Report: A 52-year-old male was admitted for new-onset hoarseness, associated with a visible and growing anterior neck mass. Physical examination revealed thyromegaly with no thyroid nodules and no tenderness on palpation of the thyroid gland. Computed Tomography (CT) scan of the neck revealed a heterogeneous enlargement of the thyroid gland compressing the larynx. He had elevated thyroid peroxidase antibodies and was initially diagnosed with Hashimoto's thyroiditis and treated with levothyroxine and steroid taper. Before a biopsy could be done, he returned to the hospital with worsening dyspnea and hoarseness. Repeat CT scan showed a progressively enlarged thyroid gland, now encasing the trachea. A fine-needle aspiration (FNA) biopsy was done which was concerning for lymphoma. However, flow cytometry and bone marrow biopsy were nonrevealing. Repeat biopsy done through thyroid isthmusectomy showed malignant lymphomatous infiltrate composed of large cells in sheets - consistent with diffuse large B-cell lymphoma. He was treated with Rituximab, Cyclophosphamide, Doxorubicin, Vincristine, and Prednisone (RCHOP).
Discussion : The typical thyroid lymphoma patient is an elderly female with painless thyroid enlargement causing compressive symptoms and airway obstruction. There is an estimated 60-fold increase in the risk of developing thyroid lymphoma in the setting of HT. Thyroid lymphomas can mimic anaplastic thyroid carcinomas; differentiation is key as anaplastic carcinoma entails poorer prognosis. Although fine needle biopsy with cytology has an established role in the diagnosis of thyroid nodules and goiters, its role in diagnosing thyroid lymphoma is limited due to its small yield. Therefore, a core needle biopsy and even thyroidectomy are occasionally required. The significantly increased incidence of primary thyroid lymphomas in patients with HT strongly suggests a pathogenetic link between this autoimmune disorder and malignant thyroid lymphoma. Chronic inflammation elicits an immune response leading to repetitive damage of surrounding stromal cells, contributing to genetic alterations, inappropriate cell proliferation, and subsequent neoplastic transformation. In our case, a probable sequence of events may have included severe chronic HT leading to MALT lymphoma and subsequent transformation to DLBCL, although DLBCL could certainly arise de novo in the absence of a low-grade precursor.
Conclusion: Thyroid lymphoma should be considered in a rapidly enlarging neck mass in the setting of Hashimoto's thyroiditis. This condition rapidly responds to chemotherapy and surgery is almost always utilized for diagnostic biopsy only.

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COVID-19 It's not just thrombotic: A Case of Unexpected Hemorrhagic Stroke in SARS-CoV-2

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Introduction: Neurologic manifestations in patients with SARS-CoV-2 include headache, confusion, delirium, anosmia, hypogeusia, cerebrovascular accidents (CVA), seizures and encephalitis. CVAs have been found to be a strong negative prognostic marker in patients admitted for SARS-CoV-2. The association of ischemic and thrombotic stroke with SARS-CoV-2 has been well documented. Hemorrhagic stroke associated with SARS-CoV-2 is not well established. We report a case of a patient with SARS-CoV-2 who was clinically improving but unexpectedly developed a neurologic catastrophe.
Case Description: A 62 year old African-American female with chronic obstructive pulmonary disease, hypertension, diabetes and history of transient ischemic attacks (TIA) presented to the emergency department for worsening dyspnea. She initially had dry cough, intermittent fevers and shortness of breath for two weeks which did not resolve despite outpatient azithromycin. She was admitted for SARS-CoV-2 and was started on empiric antibiotics and hydroxychloroquine. She was eventually intubated for severe hypoxia and acute respiratory distress syndrome (ARDS). Workup was suggestive of cytokine storm and she received Tocilizumab (IL-6 inhibitor). She was not on systemic anticoagulation and her coagulation parameters were normal. She developed labile hemodynamics suggesting autonomic instability and transient hypotension due to high PEEP and necessary sedation. Early in her ICU course, nursing staff noted asymmetric pupils. She had no focal deficits and followed one step commands but neurologic assessment was limited due to sedation. A computed tomography (CT) scan of the brain did not show acute abnormalities. Her ARDS eventually resolved but her mentation did not improve. A repeat CT brain was done which showed a large 7.0 x 8.4 x 4.9cm acute intraparenchymal hemorrhage in the left frontal lobe with extension into the ventricles. There was vasogenic edema and midline shift concerning for uncal and tonsillar herniation. Due to her grim prognosis, goals of care were readdressed and she unfortunately died.
Discussion: The suggested mechanism of stroke associated with SARS-CoV-2 is hypercoagulability and although there are no consensus guidelines, patients are often started on higher doses of anticoagulation to reduce thromboembolic risks. This case demonstrated that first, although there is a higher likelihood of ischemic and thrombotic strokes in SARS-CoV-2, hemorrhagic strokes can also occur. Clinicians must therefore be cautious of escalating empiric anticoagulation in certain patient populations. Second, frequent neurologic exams in SARS-CoV-2 patients are inherently difficult due to PPE constraints and patients often being sedated and paralyzed. Despite these challenges, a high index of suspicion for CVAs is warranted as recent data shows that its presence is a strong negative prognostic marker with mortality of up to 50%. It is unclear how hemorrhagic CVAs develop in SARS-CoV-2. In this case, autonomic dysfunction with cytokine storm catecholamines resulting in hemodynamic lability was likely the predisposing risk factor.

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An older man with eosinophilic asthma, abdominal pain and diarrhea.

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Introduction: Strongyloidiasis is characterized by infection of the human host with Strongyloides stercoralis, an intestinal nematode, and has both acute and chronic manifestations. Due to the rarity of this disease in North America, as well as its insidious nature, chronic strongyloidiasis is often unrecognized. In the setting of immunosuppression, it may disseminate and lead to hyperinfection syndrome with life-threatening consequences.

Case description: A 70-year-old man was hospitalized with abdominal pain and a one-week history of non-bloody diarrhea. He had a history of severe persistent eosinophilic asthma with elevated serum IgE, for which he was receiving chronic oral prednisone. There had been several recent admissions for unexplained dyspnea, and he was discharged on prednisone each time. Additionally, years prior to presentation, a colon biopsy had revealed tissue eosinophilia. On physical examination he was afebrile, hemodynamically stable, and exhibited diffuse tenderness on palpation of the abdomen. CT scan revealed findings consistent with colitis. Blood cultures and stool studies were obtained. Testing for Clostridium difficile was negative.

On hospital day 3, the patient developed fever, increased oxygen requirements, and encephalopathy. Empiric antibiotics were administered. Blood cultures grew Streptococcus mitis and Enterococcus faecium, and a stool specimen was positive for Strongyloides stercoralis larvae. Microscopic sputum inspection showed eggs consistent with Strongyloides stercoralis. Cerebrospinal fluid exam revealed neutrophilic pleocytosis; no organisms were visualized on Gram stain and culture was negative. Additionally, sputum and urine cultures grew Klebsiella pneumoniae.

Ivermectin was administered for disseminated Strongyloidiasis. Treatment for polymicrobial bacteremia, presumed bacterial meningitis from parasitic life cycle migration, and Klebsiella infection was initiated with cefepime and daptomycin. The patient's neurological status improved within 3 days. The antibiotic regimen was later adjusted to ceftriaxone and linezolid for improved CSF penetration while continuing ivermectin. Follow up blood cultures were negative. The patient was discharged on ivermectin and linezolid in stable condition. Subsequent stool specimens confirmed clearance of the parasite at five and twenty-four days after the initial positive specimen.

Discussion: This case demonstrates how chronic strongyloidiasis may elude diagnosis and masquerade as another more common eosinophilic disorder. The apparent persistence of this patient's infection for a number of years dating back to an earlier colonoscopy and elevated serum IgE demonstrates the insidious nature of chronic strongyloidiasis. This case is an example of how the introduction of immunosuppression drives chronic strongyloidiasis into dissemination and hyperinfection syndrome. The involvement of multiple body systems due to this syndrome is of particular interest as it is reflective of the organism’s life cycle and migration. Excluding strongyloidiasis is crucial for the physician-patient team when establishing the diagnosis of any disease characterized by systemic or tissue eosinophilia.

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Malignant Peritoneal Mesothelioma: An Ominous Cause of Diffuse Abdominal Pain

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Malignant peritoneal mesothelioma (MPM) is a rare, serosal-based malignancy with an approximate incidence of 300 cases in the United States annually. Disease can be difficult to diagnose because symptoms and imaging findings of MPM are often non-specific. The average interval between symptom onset and diagnosis is 4-6 months. We present a case of MPM in a young male and highlight important epidemiologic and clinical pearls with regards to this disease.

Case: A previously healthy 40-year-old male without a history of asbestos exposure presented to the emergency department with acute onset of diffuse abdominal pain and distension. He was seen at an outlying hospital with similar symptoms roughly 1.5 years prior. At that time, computed tomography (CT) of the abdomen and pelvis demonstrated omental nodularity and ascites, and omental biopsy was interpreted as most consistent with inflammatory changes without evidence of malignant cells. The findings were thought to be secondary to chronic infection. He was subsequently discharged and lost to follow up before presenting to our hospital 14 months later. Now, abdominal CT findings were concerning for peritoneal carcinomatosis with mild ascites. Peritoneal biopsy was consistent with malignant epithelioid mesothelioma. The patient required extensive cytoreductive surgery and intraoperative hyperthermic intraperitoneal chemotherapy (HIPEC) with carboplatin. Diffuse abdominal disease was present, and pathology demonstrated malignant epithelioid mesothelioma involving the liver capsule, falciform ligament, mesoappendix, posterior abdominal wall, hepatic segment 8, and the left and right hemidiaphragm. The patient recovered well from surgery and adjuvant chemotherapy was deferred on initial oncologic follow up.

Discussion: While MPM shares similar epidemiologic characteristics with pleural mesothelioma, there are a few important differentiating features. Only 33-50% of patients with MPM have a history of asbestos exposure as compared to 80% in pleural mesothelioma. The average age at diagnosis for MPM is 51-59 years of age, while the average age at diagnosis for pleural mesothelioma is 75-79 years in women and 80-84 for men. Women and men are affected equally in MPM. Presenting symptoms of MPM include abdominal distension (30-80%) and diffuse, non-specific abdominal pain (27-58%.) Typical CT findings include peritoneal-based masses, irregular nodular peritoneal thickening, omental masses, and ascites. The peritoneal carcinomatosis index (PCI) staging system has been proposed for MPM due to its propensity for local spread without distal or nodal metastasis. Early recognition of disease is critical, as 5-year survivals for disease are 87% for stage I, 53% for stage II, and 29% for stage III. There are no randomized control trials for optimal treatment strategy; first line therapy generally involves cytoreductive surgery (CRS) and peritoneal chemotherapy, usually heated intraperitoneal chemotherapy (HIPEC) with cisplatin. For non-surgical candidates, pemetrexed with cisplatin/carboplatin systemic chemotherapy has been used.

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Is it Really Rare? A Case Series of Fournier's Gangrene in Type 2 Diabetes Patients Treated with SGLT-2 Inhibitors

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INTRODUCTION: Sodium-glucose cotransporter 2 (SGLT2) inhibitors prevent glucose reabsorption in the proximal renal tubule. SGLT2 is preferred for patients with increased ASCVD risks, heart failure (HF) or chronic kidney disease (CKD). Fournier's gangrene (FG) is a severe polymicrobial infection resulting in necrosis of perineal and genital fascias. Although rare, the FDA issued warnings that SGLT2 inhibitors are associated with FG.

CASES: From 2017 to 2019, there were 3 observed cases of FG in diabetic patients previously treated with SGLT2 inhibitors in an outpatient endocrine clinic.
Case 1: A 54yo class 3 obese Caucasian male with type 2 diabetes, nephropathy, retinopathy, neuropathy, CKDG2A3, peripheral vascular disease (PVD), and tobacco dependence started Canagliflozin 100mg daily as an adjunctive therapy to insulin and metformin. He subsequently developed genital infection and was diagnosed with FG. He underwent surgical debridement and diverting colostomy. Hospital course was complicated with septic shock and DKA. Case 2: A 50-year-old class 3 obese Caucasian female with type 2 diabetes, CKDG2A3, diabetic neuropathy, PVD, frequent UTI, recurrent left foot infection and tobacco dependence started Canagliflozin 100 mg daily since along with insulin. She was diagnosed with FG and underwent multiple surgical debridements. Case 3: A 44-year-old class 3 obese Caucasian female with type 2 diabetes, CKDG2A3, diabetic neuropathy and tobacco dependence started Empagliflozin 10 mg in addition to Liraglutide and metformin. She was diagnosed with FG and underwent multiple surgical debridements. Risk factors, such as diabetes, obesity, immunosuppressed states, smoking, alcohol abuse and end-stage renal or liver failure, may increase risk of FG. All three cases exhibited smoking, diabetes and obesity as risk factors. All had diabetes induced neuropathy. Two had PVD. Although all had GFR>60, their microalbumin/creatinine ratios were > 100. Lasix use was found in all cases.

CONCLUSION: FG may be rare in early stage diabetic patients without comorbidities. However, diabetes with neuropathy and nephropathy, and other predisposing risk factors such as smoking, obesity, kidney failure, chronic alcoholism, liver failure, immunosuppression and cancer, may pose increased risk for FG. Our observations also identified other possible predisposing risk factors: microalbuminuria, concurrent Lasix treatment, PVD, and frequent UTIs, along with SGLT2 treatment, as FG risk factors. While SGLT2 inhibitors may reduce cardiac risk, patients with increased risk for FG should consider avoiding this class of medication. Timely cessation of SGLT2 inhibitors in individuals with multiple risk factors may help prevent progression to severe genital infections.

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Unilateral Renal Infarction Secondary to Paradoxical Embolism

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Introduction: Patent foramen ovale (PFO) is the most common congenital cardiac abnormality, affecting 25% of the general adult population. PFO is generally an incidental finding and asymptomatic in most patients. However, PFO can allow thrombi, vasoactive substances, air or fat in venous circulation to cross into systemic arterial circulation; i.e. paradoxical embolism, causing cryptogenic stroke, MI, mesenteric or limb ischemia and solid organ infarction.

Case Report(s): A 49yo man presented with right-sided flank pain and hematuria associated with fever and chills. He denied personal or family history of thrombosis, recent prolonged immobility or travel, congenital cardiac abnormalities, chest pain, or lower urinary tract symptoms. PE was significant for a faint systolic murmur over the left and right second intercostal spaces, right CVA tenderness and digital clubbing. Laboratory studies demonstrated elevated serum creatinine 1.3 mg/dL, increased from a baseline of 0.9 mg/dL; serum lactic acid of 2.4 mmol/L; modest leukocytosis with white blood cell count of 13,800/L with neutrophilic predominance; and C-reactive protein 16.9 mg/dL. CT of the abdomen with contrast demonstrated two wedge-shaped regions of low attenuation in the right kidney, concerning for infarction. Renal ultrasound revealed decreased echogenicity within the mid to lower pole of the right kidney with correlating diminished Doppler flow. Transesophageal echocardiography (TEE) subsequently revealed PFO with right to left shunting on bubble study. Duplex ultrasound of the bilateral lower extremities was negative for DVT. Thrombophilia panel was negative. Renal infarction secondary to paradoxical embolism was suspected. Cardiology consultation recommended direct oral anticoagulation with apixaban with deferment of PFO closure in the absence of additional risk factors.

Discussion: Systemic non-cerebral paradoxical embolism is exceptionally rare, accounting for less than 5% of all paradoxical emboli. Therapeutic modalities remain unclear for non-cerebral paradoxical emboli. Secondary prophylaxis can include pharmacotherapy alone or with either surgical or transcatheter PFO closure. In the context of cryptogenic stroke, three recent, randomized, multi-center trials (CLOSE, REDUCE, DEFENSE-PFO) have corroborated the superiority of PFO closure over medical management alone in the reduction of recurrent embolic events. While there has been extensive research into the relationship between PFO and cryptogenic stroke, there is minimal data regarding PFO and systemic non-cerebral emboli. Renal infarction is a unique manifestation of paradoxical embolism. Literature pertaining to such events is relegated to a limited number of case reports, with PFO closure considered on a case-by-case basis.

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Diabetic Ketoacidosis in a Previously Well-controlled Diabetic,

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Introduction: Diabetes is one of the most common chronic medical conditions in the world today. Here we present a case of Flatbush diabetes or ketosis-prone diabetes, an increasingly recognized and developing clinical entity.

Case: A 49 y.o. African-American male with past medical history of hypertension, gastroesophageal reflux, and previously well-controlled, insulin independent diabetes presented to the emergency room with epigastric pain. Symptoms began 1 week ago with severe heartburn and progressed to nausea, vomiting, and epigastric pain on the day of presentation. Patient was diagnosed with diabetic ketoacidosis (DKA) on initial lab work-up in the Emergency Room.

Of note, the patient had been diagnosed with diabetes approximately 1 year prior to presentation with an Hgba1c 7.5% on screening. Patient's most recent Hgba1c just 5 months prior was well-controlled at 6.8% on low dose Metformin monotherapy. He had a strong family history of diabetes type II, mostly insulin independent, and no family history of autoimmune disorders. On further history, patient endorsed increased polydipsia and polyuria over the past few months, but did not endorse significant changes in his diet or lifestyle. His BMI was stable at 35 compared to 5 months prior.

Hba1c testing on admission was 14.0%. During his hospitalization, the patient was diagnosed with esophageal candidiasis as a possible inciting factor for his DKA. HIV, other infectious, pancreatitis, and cardiac work-ups were negative. Islet cell antibodies and glutamic acid decarboxylase (GAD) antibodies were negative as well. He was discharged home on a basal-bolus insulin regimen in addition to Metformin.

Discussion: This patient was a previously well-controlled, non-insulin dependent diabetic who developed rapidly worsened control of his diabetes and DKA concerning for Flatbush diabetes, or ketosis-prone diabetes.

Flatbush diabetes is part of a developing and heterogeneous category of diabetes where patient present with DKA. There are several subsets of ketosis-prone diabetes that are categorized based on the presence/ absence of autoantibodies and the presence/ absence of beta islet cell function. Flatbush diabetes commonly refers to autoantibody-negative ketosis-prone diabetes with preserved islet cell function. These patients tend to behave clinically more like a type II diabetic, as opposed to a type I. Flatbush is commonly seen in middle-aged, African-Americans and African-Caribbean males who are often overweight. After intensive insulin therapy, these patients may be able to regain insulin independence.

Conclusion: Flatbush diabetes is a clinic phenomenon where previously well-controlled diabetes, especially African-American or African- Caribbean males, suddenly develop worsening control and DKA. It is important to recognize this clinical scenario, as these patients may be able to regain insulin independence later in their course

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PFO closure in Biatrial Clot

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59 year old male with significant past medical history presented with nonproductive cough and shortness of breath that had been persisting for 4 days. Patient noticed that there was also some swelling and pain in his leg that was worse with ambulation that started that day. Patient underwent CT Chest with contrast showing marked bilateral Pulmonary emboli with greatest involvement in the left main pulmonary artery. RV/LV ratio is 1.38. Patient underwent placement of EKOS catheters in right and left pulmonary arteries. After therapy bedside transthoracic Echo reported as Normal Left ventricle size, wall thickness and function with an estimated EF >55%. Small atrial thrombus noted with connection to intra-atrial septum, likely a continuation of large right atrial thrombus which is attached to IAS.

Cardiology and CT surgery were stat consulted. Decision was made to take patient urgently to the OR for Right and left atrial clot removal PFO closure, Left atrial ligation and bilateral pulmonary embolectomy. Right atrial clot found to be 6 cm and left atrial clot 2.5 cm.. Patient continued to improve and was started on Coumadin for anticoagulation and was on amiodarone for atrial fibrillation that spontaneously converted to sinus rhythm.

PFO is considered to occur in about 25% of the general population. Most cases atrial clots are secondary to atrial fibrillation. Prior guidelines had recommended that candidates for PFO were reserved for those who were unable to tolerate anticoagulation. Randomized Evaluation of Recurrent Stroke Comparing PFO Closure to Established Current Standard of Care Treatment trial (RESPECT) compared PFO closure to anticoagulation alone showed a decrease in occurrence of recurrent ischemic stroke when compared to medical management alone. Cases have been reported of catheter directed therapy failing but not involving bi-atrial clot with PFO.

There are strong recommendations for the use of PFO closure devices in patients with PFO for preventing ischemic strokes and anticoagulation in patients under 60 years of age has a benefit in preventing ischemic strokes with minor adverse events.

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A Rare Case of CNS Vasculitis with Two Primary Malignancies

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Case report: A 59 year old female presented with bilateral blurry vision, floaters and light flashes for a two-week period. Symptoms preceded by diffuse headaches associated with temporal tenderness two weeks prior to the vision symptoms. Review of systems was notable for fatigue, fever, chills, night sweats, dry mouth, and weight loss of 64 pounds in the past year. Physical exam revealed palpable left submandibular lymph node, impaired visual acuity to counting fingers in the left eye at 3 feet and 20/200 in the right eye and fundus exam revealed yellow-white placoid lesions with areas of chorioretinal atrophy. Brain MRI showed a subacute lacunar infarct in the dorsal left putamen/capsular junction. Ophthalmologic findings triggered a concern for cerebral vasculitis, immunologic workup was done which was unremarkable. CSF demonstrated lymphocytic pleocytosis and elevated protein levels. CSF studies for varicella zoster virus PCR, IgG and IgM were negative. Patient was started on methylprednisolone intravenously for 3 days with transition to prednisone to manage her suspected primary central nervous system vasculitis.

A whole body PET CT imaging showed increased uptake in the right breast with associated hypermetabolic right axillary lymphadenopathy and left submandibular lymph node, but no evidence of large-vessel vasculitis. A breast biopsy revealed invasive ductal carcinoma while a needle core biopsy of the patient's submandibular lymph node was consistent with follicular lymphoma. A catheter angiography of the brain demonstrated multifocal regions of vessel wall narrowing leading to occlusion most pronounced in distal bilateral middle cerebral arteries and distal left posterior cerebral artery territories concerning for vasculitis.

Patient was started on cyclophosphamide and prednisone was tapered during CYC treatment, however one week later she had an episode of right leg weakness and numbness and repeat MRI showed new subacute/acute infarcts in both cerebral hemispheres and the left genu of the corpus callosum. Rituximab was added to CYC and prednisone was increased. Clinically, the patient did well with no recurrence of neurologic deficits.

Summary: Physicians should consider the existence of an occult malignancy when CNS vasculitis is suspected in a patient with recurrent cryptogenic strokes. Cancer-associated CNS vasculitis might represent an immune-mediated paraneoplastic neurologic syndrome. CYC may be an appropriate first-line treatment in a patient with CNS vasculitis, however in the setting of underlying malignancy, our patient did not respond well to cyclophosphamide. Adding rituximab to CYC was beneficial in our patient possibly due to the underlying follicular lymphoma. This may point to the possibility of a paraneoplastic etiology of the CNS vasculitis. Further studies are needed to help guide treatment for PCNSV when associated with malignancy.

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To treat or not to treat, that is the question: A case of simultaneous hemorrhagic conversion of stroke, STEMI and LV thrombus

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INTRODUCTION: Cardio-cerebral infarction (CCI) is an uncommonly encountered challenge. Here, we present a case where we were faced the predicament of treating an anterolateral STEMI and LV thrombus, when the patient had a concomitant hemorrhagic conversion of her stroke.

CASE REPORT: A 71 yo female with history of hypertension was admitted to the ICU from another hospital for left sided weakness, facial droop, slurring speech after she was found to have right middle cerebral artery (MCA) ischemic stroke. The next morning her neurological deficits worsened. A repeat CT head showed hemorrhagic conversion with mild local mass effect and she CTA head confirmed right MCA involvement and CTA neck showed 50 % left carotid artery stenosis. MRI confirmed right MCA infarct with hemorrhagic conversion, edema and 5mm midline shift. Echocardiogram revealed EF of 50-55%, hypokinesis of apical myocardium with 1.0 x 1.2 cm apical thrombus. Neurosurgery was immediately consulted, and in an effort to minimize her edema, 3% hypertonic saline and mannitol were started. Due to her hemorrhagic conversion and shift, we initially chose not to anticoagulate her. On Day4, the patient complained of nausea, telemetry showed ST changes, and EKG showed anterolateral STEMI. Cardiology agreed to proceed for emergent cardiac catheterization only after Neurosurgery agreed to manage potential worsening of hemorrhage and cleared the patient for dual-antiplatelet therapy (DAPT). She underwent suction thrombectomy, PCI with drug eluding stents to LAD, and was started on DAPT. Daily head CTs were done and patient was kept on hypertonic saline and mannitol until day5. Repeat echocardiogram showed worsening LV systolic function with a drop in EF to 25-30% and two apical thrombosis: 0.6 cm x 1.6 cm and 1 cm x 0.5 cm. On day6, after CT head showed no further bleed or worsening of edema, cardiology, neurology and neurosurgery mutually agreed to start the patient on Coumadin, using heparin IV as a bridge. Patient was continued on clopidogrel and aspirin was discontinued. She was later transferred to the floor where her medications including for heart failure were optimized and she was discharged to rehab on a 30 day monitor, with a plan for close follow up.

Discussion: The term Cardio-cerebral Infarction (CCI) was first introduced in 2010. Multiple mechanisms have been proposed, in our case it was secondary to embolization from an LV thrombus. Our case was unique in the fact that our patient developed hemorrhagic conversion of her infarct, further complicating matters. Each scenario was life threatening and hence we chose to treat her STEMI and thrombus despite her intracranial bleed as a delay in treatment of her STEMI was potentially fatal, and a delay in anticoagulation would further increase her risk for thromboembolic phenomenon.

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A Case of Nivolumab-Induced Pneumonitis

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Introduction:
Nivolumab is an anti-programmed cell death-1 receptor (PD-1) monoclonal antibody. It is fast becoming the standard of care for various types of malignant tumors, including metastatic melanoma (MM), non-small cell (NSCLC), small cell lung cancer (SCLC), and renal cell carcinoma (RCC), to name a few. Various immune-related adverse effects of nivolumab could be potentially severe and fatal. Pneumonitis is a relatively uncommon, but potentially life-threatening adverse effect which has been reported.

Case Report:
We present a 67-year-old male with a past medical history of MM BRAF negative. He was seen by oncology and initially started on 4 cycles of nivolumab/ipilimumab combination, later switched to nivolumab monotherapy for 3 cycles with a total treatment duration of 11 months. After the last cycle, he developed progressively worsening cough and exertional shortness of breath and was referred to pulmonology. Chest CT revealed bilateral ground glass opacities, a new finding compared to imaging done 2 months previous. Bronchoscopy was normal with BAL and EBUS negative for malignant cells. As a result, he was started on systemic corticosteroids with clinical improvement and scheduled for 2 month follow up.

Discussion:
Nivolumab is a human IgG4 monoclonal antibody against the PD-1 receptor that works as an immune checkpoint inhibitor. Commonly used against various types of malignant tumors, including metastatic melanoma, it was approved in 2014. Immune-related adverse effects have been reported, but not well characterized, including pneumonitis, hepatitis, nephritis, colitis and neuropathy. Of the afore-mentioned, pneumonitis is relatively uncommon but potentially life-threatening. Interestingly, nivolumab-induced pneumonitis presents with various patterns, including cryptogenic organizing pneumonia, non-specific interstitial pneumonia and idiopathic pulmonary fibrosis. Early recognition and prompt institution of high dose corticosteroids result in pneumonitis resolution. Due to increasing popularity, clinicians should have a high index of suspicion in order to facilitate treatment and to prevent morbidity and potential mortality from nivolumab-induced pneumonitis.

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Propofol Embolism via previously unknown Atrial Septal Defect resulting in Opisthotonos and Chemical Meningitis

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Introduction. Opisthotonos is a disorder classically associated with hyper-extension of the extremities and spine in a conscious patient, and is a known complication of many anesthetic drugs, usually when used in combination. The main culprits are propofol, fentanyl, or alfentanil and there are only proposed mechanisms, with current speculation pointing to gamma-aminobutyric acid (GABA) and glycinergic pathways. However, in patients with structural defects allowing for shunting, paradoxical embolus of medications have the capacity to cause direct damage to vital organs with potentiated effects.

Case Presentation. A 64-year old woman with a past medical history of diabetes mellitus type 2, hypertension, hyperlipidemia presented to the hospital for elective repeat esophagogastroduodenoscopy (EGD) with dilation for management of a mid-esophageal stricture. EGD sedation was achieved with peripheral intravenous propofol, during the dilation there was concern for perforation requiring stent placement. After the procedure, patient did not awaken from anesthesia and subsequently developed diffuse peripheral extremity rigidity with muscle fasciculations. Patient was immediately transferred to the intensive care unit and evaluated by the on-call neurologist. Patient was treated empirically with antiepileptics, antibiotics, and antiviral therapy. Urgent electroencephalogram (EEG) noted increased contractions but no epileptiform activity. Computed tomography (CT) of the head was done which showed concern for “air embolism vs lipoma”. Repeat EEG revealed no epileptiform activity. Lumbar puncture was performed and was not indicative of any infectious process. During that time frame, patient’s muscle tone changed from rigid to flaccid. Brain magnetic resonance imaging was performed showing mild cerebral edema and did not comment on the presence of lipomas or emboli, findings were confirmed with CT head. Transcranial doppler with bubble study was performed but did not show evidence of shunting. Trans-thoracic echocardiogram was performed but also did not show evidence of shunting. Patient had intermittent periods of lucency but was otherwise somnolent throughout hospital stay. Patient eventually received a tracheostomy and percutaneous endoscopic gastronomy tube and subsequently discharged to extended care facility for continued supportive care. Upon removal of the esophageal stent, transesophageal echocardiogram with bubble study revealed evidence of atrial septal defect. Eventually patient was admitted to hospice with no discernible improvement and expired soon after.

Discussion. A case of propofol embolus via a peripheral IV line through a previously unknown atrial septal defect that resulted in opisthotonos and direct chemical meningitis has never been previously documented. With limited data for direct propofol chemical meningitis, supportive care became the mainstay of treatment, although there may be evidence that supports the use of high dose steroids. Opisthotonos is a known complication of propofol, although very rare and usually self-limiting, there has been debate on the pathophysiology, and thus treatment options are mainly speculative at this point.

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Pitfalls in diagnosing an unusual presentation of microscopic polyangiitis.

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Introduction: Microscopic Polyangiitis (MP) is a rare condition often having a non-specific presentation that increases the risk of misdiagnosis due to low levels of suspicion. Common presentations involve respiratory symptoms, weight loss and low-grade fever. This report describes an unusual presentation of abdominal pain and non-bloody diarrhea, subsequently diagnosed as MP.

Case Report: A 72-year-old Caucasian female presented to the emergency department with epigastric pain and diarrhea. Her past medical history was significant for breast cancer on anastrozole, atrial fibrillation and hypertension. Physical examination was remarkable only for epigastric tenderness. Urinalysis was positive for significant proteinuria, large amounts of leukocyte esterase, moderate hemoglobin, elevated red and white blood cells, but negative for nitrites and bacteria. New renal impairment was noted on biochemistry. CT-abdomen was negative for acute pathology. She was discharged on cephalexin for presumed hemorrhagic cystitis.

She subsequently presented three weeks later with complaints of continued epigastric, and now, periumbilical pain, with one episode of vomiting. Upon examination, she had mild diffuse abdominal tenderness, which had worsened from the previous epigastric tenderness alone. Urinalysis and renal impairment were largely unchanged. CT-abdomen now showed right middle lobe consolidation, but no acute abdominal pathology. This presentation, she was suspected to have pneumonia and was admitted on ceftriaxone and azithromycin.

Day four of admission was complicated by atrial fibrillation with rapid ventricular response so she was started on IV heparin and metoprolol. On Day eight, she developed hypoxic respiratory failure. CT-chest showed worsened bilateral diffuse infiltrates. In the MICU, bronchoscopy revealed bloody bronchoalveolar lavage consistent with diffuse alveolar hemorrhage (DAH). Additionally, a gradual decline in renal function was noted. Serologies for a vasculitic cause were significant for positive p-ANCA, and a subsequent renal biopsy confirmed the diagnosis of MP.

The patient was started on steroids and rituximab, with six sessions of plasmapheresis. Despite improvement in renal function, she remained ventilator-dependent and was discharged to a LTAC. The family later changed her code status to comfort care, and she expired the following day.

Discussion: This report describes an atypical presentation of MP with epigastric pain and non-bloody diarrhea only. This patient also had gradual progression in renal impairment, contrary to the step-wise rapid progression typically seen with MP. As observed in this case, lung involvement in MP is a major contributor to morbidity and mortality. Hematuria with proteinuria and renal failure on initial presentation, should've been investigated further. The presence of red cell casts, suggesting a glomerular source, may have prompted earlier suspicion and diagnosis prior to the development of DAH, possibly improving this patient's outcome. This unique presentation underscores the importance of increased awareness of atypical presentations of MP. Early diagnosis and treatment are recommended to avoid subsequent morbidity and mortality.

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Rash from fire- a case series

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Introduction: Erythema ab igne is an uncommon hyperpigmented dermatological disorder caused by repeated exposure to heat sources. Its reticulated configuration closely resembles that of livedo reticularis and as such it can be easily mistaken for the latter. Due to this, extensive workup usually ensues for a disorder that is benign and easily treated. This case review seeks to be a clear resource on the presentation and diagnosis of erythema ab igne, helping the reader differentiate it from livedo reticularis with the goal of sparing our patients from the unnecessary investigations that are undertaken.
Case Reports: We present three patients who presented with non pruritic, painless, hyperpigmented or erythematous, reticulated rash of weeks duration. They all reported a significant history of repeated heat exposure to the affected area. Patient 1:49 year old Caucasian woman, with a reddish hyperpigmented rash to pretibial region of legs for three weeks – prior space heater use. Patient 2:34 year old Caucasian woman whose hyperpigmented rash was located to the lower back for six months– prior heating pad use; Patient 3: 28 year old African American woman, hyperpigmented rash to plantar aspect of feet for 5 months – foot warmer use. This was the first occurrence of this type of rash in all three patients, without a prior significant rheumatological history. Reviews of symptoms in all three patients were negative for systemic symptoms or joint involvement. Examination of each patient was significant for a hyper pigmented reticulated rash in the affected areas. The diagnoses were confirmed by an experienced dermatologist, in which the clinical diagnosis of Erythema Ab Igne was made without further clinical investigations. Counseling and avoidance of continuous direct heat exposure without pharmacological management was advised with complete resolution of rash after a few months of follow up.
Discussion: Erythema Ab Igne is an uncommon disorder, its exact incidence is unknown. Historically it was often seen due to use of open fires for cooking and heating. Its resurgence is linked to the increase use of heating pads and laptop use. Repeated and chronic exposure to infrared radiation causes venule dilation with resultant red blood cell extravasations and hemosiderin deposition. Its main mimic is livedo reticularis, which is not localized and frequently associated with systemic symptoms. Once the offending source is removed, resolution is expected. In rare cases due to chronic exposure, thermal keratosis and squamous cell carcinoma have occurred.
Conclusion: Erythema ab igne is a highly localized hyperpigmented reticulated rash, associated with prolonged heat exposure. Due to its reticulated nature it is often mistaken for livedo reticularis, the latter of which sometimes requires extensive evaluation. Erythema ab igne is diagnosed clinically and treated with removal of the offending agent.

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TB or not to be? A 41 year old prison guard with migraine headaches

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Introduction: Cryptococcal meningitis is a rare but severe complication of sarcoidosis. This is associated with the known predilection for lymphocytopenia in sarcoidosis patients. Both neurosarcoidosis and central nervous system (CNS) fungal infections can present with cranial neuropathies, which makes diagnosis challenging.

Case Description: A 41 year-old man presented to the emergency department after sudden onset of severe headache and double vision. Additionally, his right eye deviated outward and on arrival was unable to adduct past midline. Thirty minutes later, symptoms resolved. Ten months prior, the patient began experiencing daily retro-orbital headaches. The patient's primary care provider suspected sinusitis, then migraines, however various treatment regimens proved ineffective.
The patient's medical history included pulmonary sarcoidosis diagnosed via paratracheal lymph node biopsy a decade earlier. No further respiratory symptoms over the next ten years resulted in a loss to follow up. He worked for the County Correctional Department and approximately one year prior had contact with a tuberculosis (TB) positive inmate. His purified protein derivative (PPD) skin test one week later was negative.
On morning rounds, the patient divulged fifteen pounds of recent weight loss. He also reported fatigue and anorexia. Vital signs were unremarkable. Physical exam revealed no horizontal diplopia, nuchal rigidity, or neurological deficits, and was otherwise benign.
Complete blood count was significant for a lymphocytopenia (absolute count 0.50K/microliter (mcL), normal range 1.00 to 4.80K/mcL) and chest x-ray showed bi-apical infiltrates with no prior comparison. His HIV screen was negative. A non-contrast CT chest revealed fibrotic changes and hilar adenopathy consistent with pulmonary sarcoidosis. No active cavitary processes were seen. A lumbar puncture with cerebrospinal fluid (CSF) studies showed a mixed neutrophilic/mononuclear pleocytosis (total nucleated cell count 39 cells/mcL) with hypoglycorrhachia (20 mg/deciliter (dL)) and elevated protein (154mg/dL). Chronic tuberculosis meningitis became the working diagnosis and treatment was started with rifampin, ethambutol, isoniazid, and pyrazinamide with adjunctive corticosteroids.
Later that afternoon, the patient's CSF cryptococcus antigen came back positive. His TB regimen was stopped and intravenous liposomal amphotericin and oral flucytosine began. In the ensuing days, Cryptococcus Neoformans grew in his CSF culture. A lymphocyte panel was obtained and the patient's absolute CD4 count was 210 cells per cubic millimeter. The patient's condition remained stable and he was discharged. By two-week follow up he noted the complete resolution of his headaches.

Discussion: This interesting case demonstrates the importance of a broad differential in sarcoidosis patients with cranial neuropathies. Any suspicion for CNS infection warrants a CSF exam with full diagnostic studies. General CSF studies are very similar for infective chronic meningitis versus neurosarcoidosis. The only decisive discriminant is a positive CSF cryptococcus antigen and/or CSF culture.

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Spontaneous Atraumatic Splenic Rupture in Systemic Infiltrative AL Amyloidosis

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Introduction: Atraumatic spontaneous splenic rupture (SSR) is rare, with estimated incidence of 0.1-0.5%. Risks for SSR include malignancy, antiaggregant-anticoagulation drug use, infection, and amyloidosis. Systemic amyloidosis involves the spleen in 5-10% of cases. If SSR occurs, it is associated with a high 30-day mortality of 26%, thus prompt recognition and treatment are required, with either splenectomy or angioembolization.

Case Description: A 50-year-old female presented with sudden diffuse abdominal pain, positional chest pain, and hypotension with no precipitating event. She had systemic AL Amyloidosis causing cardiomyopathy, congestive heart failure and end-stage renal disease requiring hemodialysis. Physical examination revealed BP 85/48, grade 2/6 systolic murmur, no peritoneal signs, masses, nor hepatosplenomegaly. Her platelet count was 144 K/mcl, hemoglobin was 8.8 g/dL and hematocrit 26.6 %. Non-contrast computed tomography of the chest, abdomen and pelvis showed a sub-capsular hematoma involving the spleen as well as extension of hemorrhage within the abdomen; a same-day repeat CTA showed interval increase in high attenuation free fluid, consistent with intraperitoneal bleeding. Given significant co-morbidities, the patient was considered a poor surgical candidate, and distal splenic artery embolization (SAE) was completed. She was immediately stabilized following the procedure, and ultimately discharged with close follow up.

Discussion: SSR secondary to AL amyloid has several proposed mechanisms, including direct amyloid damage, amyloid angiopathy, and factor X deficiency from absorption to amyloid fibrils predisposing a bleed. Our patient had ESRD, with possible uremic coagulopathy, creating a complex of disorders leading to spontaneous rupture of a normal sized spleen. The less-invasive approach of embolization prevented confirmatory tissue sampling to determine exact etiology. Mortality of SSR can range up to 26% and requires prompt management. Splenic artery embolization has gained increasing acceptance over splenectomy in hemodynamically stable patients, with overall mortality of 12.7% in splenectomy and 5.4% in SAE. Early infectious complication rate is 23.1% with splenectomy and 11.7% in SAE. Hemodynamic instability is an independent risk factor for SSR mortality, but infectious complications or procedure used (splenectomy vs SAE) do not appear to be independent risk factors for mortality. SAE preserves some splenic function; however, pathologic spleens with reduced baseline function are unlikely to benefit from embolization. While the decision of embolization was based on surgical candidacy in our patient, it is a non-definitive treatment of a pathologic spleen, and recurrent hemorrhage may occur. This case is an example of SAE as successful treatment for hemodynamically unstable SSR.

Conclusion: This case illustrates highlights a potential complication of AL amyloidosis. SSR has a high mortality rate, and is an important inclusion in the differential of an acute abdomen. Embolization may be successful in hemodynamically unstable patients that are poor surgical candidates.
Key words: AL Amyloidosis; Amyloid Angiopathy; Factor X deficiency; Hemoperitoneum; Angioembolization

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A case of Takotsubo cardiomyopathy (stress cardiomyopathy) triggered by diabetic ketoacidosis and hypothermia.

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Background: Takotsubo cardiomyopathy (TC), also recognized as stress-induced cardiomyopathy, is a transient condition of left ventricular (LV) dysfunction, which presents similarly to acute coronary syndrome (ACS), but with normal coronaries. Physical or emotional stressors usually precipitate TC. It is typically a benign condition with the complete resolution once the triggering cause resolves. There have been a few cases of TC induced by diabetic ketoacidosis (DKA) that have been reported in the literature.

Case presentation: A 50-year-old Caucasian female patient presented with lethargy, in addition to hypothermia and hypotension. Further investigation showed hyperglycemia with metabolic acidosis and ketonemia. Eventually, she was diagnosed with diabetic ketoacidosis (DKA). On day 2 of the admission, the patient's condition further deteriorated despite appropriate treatment of DKA. An EKG showed ST-segment elevation in inferior leads, and troponin levels were elevated. Cardiac catheterization showed non-obstructive coronary arteries but severely reduced cardiac index. Echocardiography showed an ejection fraction (EF) of 25% with global hypokinetic LV. Eventually, the patient was diagnosed with TC or stress-induced cardiomyopathy.

Conclusion: TC should always be suspected in any patient presenting with acute heart failure during DKA treatment. TC is a transient syndrome. However, it can result in dreadful complications, including cardiogenic shock, arrhythmias, or thromboembolic events. Early recognition and timely treatment are pivotal in such cases.

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Positive Pressure Ventilation and Right-Sided Heart Failure- A Hemodynamic Challenge

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Maintaining appropriate hemodynamics in right-sided heart failure can become challenging when the need for positive-pressure ventilation arises. The following case highlights an extreme instance of the cardiovascular collapse that can ensue.

A 26-year old male with a history of intravenous drug use developed septic shock from methicillin-sensitive Staphylococcus aureus (MSSA) bacteremia. A surface echocardiogram revealed infective endocarditis with a large tricuspid valve vegetation leading to torrential tricuspid regurgitation and right ventricular dysfunction, while a CT of the chest showed diffuse septic pulmonary emboli leading to significant pulmonary hypertension. He had profuse anasarca secondary to right-sided heart failure, and required intermittent hemodialysis for volume control. He was deemed not a surgical candidate given ongoing drug use and a poor support system. Despite appropriate antibiotic therapy, he continued to be febrile and endorsed new back pain. To rule out an epidural abscess, MRI of the spine was proposed. The patient suffered from severe anxiety and agitation; thus, anesthesia was required for him to tolerate the imaging. He underwent an uneventful induction with propofol, rocuronium and midazolam. Shortly after intubation, oxygen desaturations into the 60's were noted despite increasing FiO2 to 100% and raising the PEEP to 10 mmHg. He subsequently became hypotensive with MAP's in the 60's requiring intermittent boluses of pressors. He was transferred to the medical ICU, where an ABG showed a pH of 6.918, a pCO2 of 122.7 and a pO2 of 51.9 on 100% FiO2. His respiratory rate was increased, his PEEP was minimized to 5 mmHg and he was started on inhaled epoprostenol. His oxygen saturation initially was refractory, and his MAP's continued to decline, requiring initiation of epinephrine, norepinephrine and vasopressin drips. He ultimately suffered PEA arrest. Return of spontaneous circulation was obtained after one round of compressions, after which his oxygen saturations slowly normalize with an improvement in his mental status. Over the next 12 hours, pressors were weaned off entirely and he was able to tolerate minimal vent settings. CRRT was initiated for assistance with volume removal. Less than 48 hours after being intubated, he passed a spontaneous breathing trial and was extubated successfully to heated high flow while still receiving epoprostenol, which was ultimately weaned off as well. His oxygen requirements continued to decline and he was transferred out of the ICU. He was discharged from the hospital on room air, just 12 days after his cardiac arrest.

Positive pressure ventilation affects both preload and afterload, which can be detrimental in right-sided heart failure, particularly when pulmonary hypertension is present. Cardiac anesthesia should be involved in such cases to reduce the risk of hemodynamic compromise.

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Presentation of Chronic Anemia: A Near Miss of Medical Emergency

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Anemia is more prevalent in women of childbearing age. In few cases the cause may be readily apparent, however, in many, it is multifactorial or overlooked. Anemia in the setting of concomitant cytopenias with atypical clinical presentation requires high degree of clinical suspicion and thorough evaluation. An accurate diagnosis of the underlying condition is essential for timely intervention to improve patient prognosis. We report a case of acquired thrombotic thrombocytopenic purpura (TTP) who presented with chronic anemia, thrombocytopenia and mild, transient neurological manifestations.

A 37-year-old G6P5 African American female presented to the emergency department (ED) with slurred speech, right upper extremity and right facial numbness for an hour. She had a significant medical history of menorrhagia, hypertension and marijuana use disorder. Her symptoms including slurred speech and numbness resolved shortly after arriving to ED. She also reported heavy vaginal bleeding at presentation. Initial vital signs showed elevated blood pressure. Physical exam was notable for active vaginal bleeding with clots. Labs at presentation were significant for normocytic anemia with hemoglobin of 8.0 g/dL, thrombocytopenia (59 k/Ul) and mild hypokalemia (3.3 mmol/L). Brain CT was negative for intracranial pathology. Abdomen/pelvis ultrasound revealed markedly heterogeneous bulky uterus with extensive fibroid. She was further evaluated for severe thrombocytopenia. Lab results showed low haptoglobin (<8 mg/dL), elevated lactate dehydrogenase (512 U/L). Prothrombin time, partial thromboplastin time, fibrinogen activity and creatinine were normal. Peripheral blood smear (PBS) showed numerous schistocytes and nucleated red blood cells, suggestive of microangiopathic hemolytic anemia (MAHA). Presumptive diagnosis of thrombotic thrombocytopenic purpura was made. The patient received IV steroids, two units of fresh frozen plasma (FFP) and was transferred to tertiary care center for plasma exchange (PEX) therapy. ADAMTS13 activity drawn prior to FFP transfusion was measured as <2% and the diagnosis of acquired TTP was established. At the tertiary care center, she received PEX therapy for 3 days along with IV steroids. Platelet count (59-->410) and ADAMTS13 activity (2%-->40%) were significantly improved following PEX therapy.

This case exemplifies the unusual presentation of TTP with mild clinical features and underscores the importance of careful evaluation of PBS in any patient presenting with cytopenias. TTP is a clinical diagnosis suspected in patients presenting with MAHA and thrombocytopenia with or without clinically evident etiology. The diagnosis is established by autoantibody-mediated deficiency of ADAMTS13 activity, typically <10%. It is a medical emergency, almost always fatal with a mortality rate of 90% without prompt intervention. PEX is the mainstay of treatment. Administration of glucocorticoids and rituximab is suggestible for presumptive or confirmed cases of TTP. Caplacizumab treatment is recommended for severely ill patients with neurological symptoms or elevated troponin levels.

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Malignant pleural effusion: a rare presentation of urachal adenocarcinoma

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Introduction: Urachal adenocarcinoma is a very rare form of vesical malignancy, it accounts for 0.1-0.7% of all bladder cancer. It usually presents at an advanced stage and carries poor prognosis. We present a case of a previously healthy 35-year-old female who was diagnosed with urachal adenocarcinoma in the setting of pleural effusion. Malignant pleural effusion secondary to urachal adenocarcinoma is uncommon and if present, typically occurs at the late stages of the disease. This case is unique due to the rarity of this malignancy and unusual early pleural involvement that led to diagnosis of urachal adenocarcinoma.

Case Presentation: A 35-year-female presented with pleuritic chest pain and progressive dyspnea of one-week duration. Her physical examination revealed respiratory distress and decreased breath sounds more on the right side. A chest CT scan with contrast was obtained to rule out pulmonary embolism, it showed large right-sided pleural effusion with complete lung collapse, mediastinal shift to the left and incidental small left lung pulmonary nodules. A diagnostic and therapeutic thoracentesis was performed. The pleural fluid cytology revealed cells were positive for urachal adenocarcinoma tumor markers, including: CK8, MOC-31, CK20, PAX-8 and negative lung markers. Subsequent abdominal CT revealed an infiltrative lesion in the pelvis involving the dome of the urinary bladder and surrounding mesenteric soft tissue. Transvaginal ultrasound revealed non-gynecological source. Patient underwent transurethral resection of bladder tumor, with following tissue histology confirming urachal adenocarcinoma. Patient was discharged with follow up appointment with medical oncology, however, she presented after 3 days with recurrent dyspnea. Repeat imaging revealed a complete opacification of right hemithorax with the recurrence of effusion. The patient had repeat thoracentesis for therapeutic purposes. She was evaluated by thoracic surgery and received talc pleurodesis and video-assisted thoracic surgery for the pleural effusion.

Discussion: Urachal adenocarcinoma is an extremely rare form of bladder cancer. It rises from remnant of urachus associated with intestinal metaplasia and adenomatous changes. Patients usually present with painless hematuria, irritative voiding symptoms, mucusuria or abdominal mass. Systemic metastasis occurs in more than half of patients with disease progression. The most common sites of metastases are liver, lung, bone, peritoneal and lymph nodes. Malignant pleural effusions rarely arise from genitourinary cancers, and proposed mechanisms are lymphatic, blood or transcoelomic metastasis with cells passing from peritoneal cavity to pleural space through diaphragmatic pores. This case describes an unusual metastasis to the pleura and lung in a patient with underlying urachal adenocarcinoma as a presenting symptom. Few cases have reported urachal adenocarcinoma with lung metastasis. This is the first case of malignant pleural effusion secondary to urachal adenocarcinoma as presenting symptoms.

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Amiodarone-Induced Thyroiditis Resulting in Pericardial Effusion

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Amiodarone is well known to be toxic to the lung, thyroid, and liver. However, amiodarone-induced thyroiditis resulting in pericardial effusion is rarely described in the literature. The broad impact of amiodarone on human physiology makes it important to thoroughly examine the entire clinical presentation when evaluating patients on amiodarone therapy.

A 76-year-old male with history of hypertension, atrial fibrillation, and prostate cancer on surveillance was admitted three days after presenting to his cardiologist. He had experienced one month of new onset bilateral lower extremity edema, 20 pound weight gain, and paroxysmal nocturnal dyspnea. He denied fever and was started on furosemide 20mg daily. The following day he developed a cough, dizziness, and urinary incontinence. He was on a beta blocker and amiodarone for seven months for atrial fibrillation. Physical exam revealed pulmonary and peripheral edema, elevated jugular venous pressure, but no muffled heart sounds. Blood pressure was 141/91 and pulse was 70 in normal sinus rhythm. Cardiac workup found a BNP of 12 and echocardiogram revealed a small to moderate pericardial effusion with right atrial collapse without tamponade. Thyroid studies showed a heterogeneous and mildly vascular gland, TSH of 96, untraceable free-T4 levels, and positive thyroid peroxidase antibody titers. Pulmonary studies showed no evidence of pneumonia or amiodarone toxicity. Liver function tests showed an AST of 86 and an ALT of 82. CBC showed a WBC of 3.3, RBC of 4.25, and platelets of 119. The amiodarone, furosemide, and blood pressure regimen were stopped and he was started on levothyroxine 50mcg with subsequent resolution of symptoms. The patient was scheduled for outpatient follow-up with cardiology, endocrinology, and internal medicine to verify resolution of these toxicities.

The observed hypothyroidism was attributed to the hypothyroid phase of an acute amiodarone-induced thyroiditis because he lacked the constitutional symptoms of hypothyroidism. The subacute pericardial effusion was then attributed to his hypothyroidism. The diuretics likely decreased his preload to the point of right atrial collapse which caused his worsened dizziness and paroxysmal nocturnal dyspnea. The expected reflex tachycardia could have been masked by his beta blocker and hypothyroidism. The transaminitis was also attributed to amiodarone as he had normal LFTs at the medication initiation seven months prior. The mild pancytopenia was secondary to his profound hypothyroidism.

This intricate case presents an important lesson for clinical practice. Namely, the importance of looking at the whole clinical presentation. This patient had several concurrent toxicities which could have been missed without a holistic approach. Additionally, we present a rare presentation of an amiodarone-induced thyroiditis resulting in pericardial effusion. This caused edema and paroxysmal nocturnal dyspnea that worsened with diuresis. Had we not considered amiodarone as the culprit, we would not have been able to properly correct his symptomatology.

Screening for Food Insecurity and Addressing with Home-Delivered Meals and Nutrition Assistance Programs in Geriatric Patients

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Introduction: Food insecurity exists whenever the availability of nutritionally adequate and safe foods is limited or uncertain. Prevalence of food insecurity is under-recognized and has been cited to be as high as twenty-three percent and is associated with female gender, social isolation, home-bound status, and financial vulnerability. Primary treatment relies on home-delivered meals and community-based nutrition services. Studies have demonstrated improvements in nutritional status, loneliness, and delayed entrance into nursing homes in the geriatric population.
Case Presentation: A 66-year- old female patient with medicare presented with fatigue, presyncopal symptoms and 7 lbs of unintentional weight loss. Social history was obtained and revealed her husband had passed three months prior yielding a fifty percent reduction in household income, yet unchanged monthly expenses. Discretionary income, which was limited to $50 per month, was spread between household items, cigarettes and food. Patient lived alone, relied exclusively on public transportation, and did not participate in social or religious activities. She denied symptoms of depression and screened negative PHQ-9. Clinical picture was consistent with a diagnosis of food insecurity. Meals on Wheels referral and food-stamps enrollment were initiated, which improved her presyncopal symptoms and weight trajectory on follow-up.
Discussion: This is a case of geriatric food insecurity induced by a recent passing of a primary income-producer with resolution following home-based meal delivery and the addition of government-sponsored food stamps. Initial screening and diagnosis of food insecurity is often under-recognized and under-performed by clinicians. A meta-analysis assessing the effectiveness of home-delivered meal programs demonstrated 6 of 8 studies showed noticeably improved diet quality and nutritional risk among participants. Other reported outcomes included increased socialization opportunities as well as the ability for volunteer drivers to serve as safety checks to report needs of home-bound older adults. At a health policy level, the financial incentives are significant. The average cost of a one-month nursing home stay is equivalent to providing home-delivered meals five days a week for seven years. One study found that each additional $25 states spent on home-delivered meals per year, per person aged 65 or older was found to be associated with a reduction in the low-care nursing-home population by one percent. Despite these benefits, home-delivered and congregate meals reach less than 5% of eligible older Americans. As in our case, physicians should remain vigilant and appropriately screen geriatric, socially isolated, home-bound, and low-socioeconomic status patients as this high-value intervention has tremendous potential to improve medical, financial and social outcomes in this vulnerable population.

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Severe Tertiary Hyperparathyroidism Leading to Myelofibrosis and Pancytopenia in a Young Female

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Introduction: Secondary hyperparathyroidism can progress to tertiary hyperparathyroidism through unregulated parathyroid function after a prolonged period of parathyroid stimulation. If left untreated, significantly elevated parathyroid hormone can rarely lead to myelofibrosis, with associated pancytopenia, through releasing cytokines and stimulating marrow fibroblasts. We present a case of a young female with fatigue, pathological fractures, and pancytopenia who was found to have myelofibrosis and severe tertiary hyperparathyroidism.

Case presentation: 27 year old female patient presented to the hospital with progressively worsening fatigue and weakness. Her past medical history is significant for end-stage renal disease (ERSD) on hemodialysis, chronic pancytopenia due to biopsy proven myelofibrosis, and recent history of right femur fracture two months before presentation status post retrograde intramedullary nail of the right femur. To note, patient was bedridden in a nursing home for six months prior to presentation due to weakness and inability to walk.

On presentation, patient's vital signs were within normal limits. Physical exam was significant for right hip swelling and tenderness. Initial laboratory findings revealed pancytopenia. Calcium was 9 mg/dL and phosphorus was 5.4 mg/dL. Liver function tests were significant for an alkaline phosphatase of 1947 U/L. Gamma-glutamyltransferase was within normal limits. Vitamin D was less than 7 ng/ml. Right hip X-ray showed diffuse osteopenia. During her hospital stay, parathyroid hormone was ordered, and it was elevated at 4221 pg/ml. Nuclear medicine parathyroid imaging showed probable parathyroid adenoma inferiorly on the left.

Tertiary hyperparathyroidism was diagnosed, and patient underwent near complete parathyroidectomy as it was indicated due to the recent pathological fractures and significantly elevated markers of bone turnover. Patient's bones were very fragile, and she had a distal fracture of the left femur as she was getting transferred to the X-ray machine during her stay. After her parathyroidectomy, she developed hungry bone syndrome, and she required IV calcium for more than twenty days.

Discussion: It is important to look for secondary hyperparathyroidism in ESRD patients and to treat it appropriately with medications such as calcitriol and phosphate binders to prevent progression to tertiary hyperparathyroidism. This patient had untreated hyperparathyroidism which progressed to severe tertiary hyperparathyroidism with significantly elevated PTH of 4221 ng/ml. This led to very fragile bone which manifested with multiple fractures with minimal movement in addition to myelofibrosis which was diagnosed on bone marrow biopsy as part of her pancytopenia workup. Myelofibrosis can rarely occur when elevated parathyroid hormone leads to the release of cytokines such as tumor necrosis factor-α and interleukin-6 which stimulates bone marrow fibroblasts. Pancytopenia in a patient with ESRD and severe hyperparathyroidism should point towards possible marrow fibrosis which can be diagnosed through bone marrow biopsy. Parathyroidectomy can reverse both myelofibrosis and pancytopenia.

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Lofgren's Syndrome-: Not every redness is cellulitis.

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Introduction: Lofgren's syndrome (LS) is a form of Sarcoidosis characterized by erythema nodosum, bilateral hilar lymphadenopathy, migratory polyarthralgia and fever. In the USA, Sarcoidosis initially presents as LS in 10% of patients. The disease has a strong association with the HLA-DRB1 allele in Scandinavian populations. Typical sarcoidosis tends to have an insidious onset whereas, LS tends to present acutely. Young middle-aged females are affected more commonly, with the median age of onset being 37. LS has a good prognosis, with more than 90% of patients experiencing complete resolution after 2 years.

Case report: A 29-year-old Caucasian female with no medical history who initially presented to the emergency department with bilateral foot pain and subjective fevers from 2 weeks. She had been treated for foot tendinitis and plantar fasciitis by a podiatrist but symptoms remain unresolved. She was diagnosed with right foot cellulitis and sent home on oral clindamycin. She returned to the emergency department 24 hours later due to uncontrolled pain. Physical examination revealed normal vital signs, tender, erythematous nodules of 1 cm in size on anterior middle third of both legs. Both ankles were tender and erythematous. CT thorax with contrast showed bilateral hilar lymphadenopathy. CBC and CMP were normal, ESR and CRP were 50 and 4.95, respectively. Workup for rheumatological conditions and venereal diseases were negative. Ankle joint aspiration showed no crystals and grew no organisms. Angiotensin converting enzyme (ACE) level was 41 U/L. The patient was initially treated with opioids and NSAIDs but did not get much pain relief. She however, showed a remarkable response to glucocorticoids. At 2 year follow up, she had been off steroids for 6 months and was asymptomatic.

Discussion: There are 3 situations where a presumptive diagnosis of sarcoidosis can be made without the need for a biopsy; Heerfordt syndrome, LS and asymptomatic bilateral hilar lymphadenopathy.

Apart from the aforementioned characteristics of LS, ACE levels and type of affected joints can increase the likelihood ratio of a patient having this disorder. With ACE levels < 25 U/L, 25-71 U/L and >71 U/L, the likelihood ratios of sarcoidosis/LS are 0.12, 1.31 and 7.15 respectively. LS also has a predilection for ankle joints with symmetric involvement.

Our patient had erythema nodosum, symmetrical ankle pain, subjective fevers and radiological findings suggestive of stage 1 pulmonary sarcoidosis made LS the highly likely diagnosis. Her elevated ACE levels and improvement with glucocorticoids further increased the likelihood of LS as correct diagnosis.

Conclusion: The purpose of doing this case report is to highlight the importance of clinical exam, less invasive diagnostic tests including chest X ray, serum ACE levels in making diagnosis of LS so that invasive procedures for tissue biopsy like bronchoscopy, mediastinoscopy etc. can be avoided.

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A Stimulating Case of Ischemic Colitis

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Background: The excessive daytime sleepiness associated with central hypersomnias is treated with stimulant medications, including amphetamine-like stimulants. Amphetamine toxicity usually presents with cardiac, neurologic or psychiatric symptoms, but these findings are less likely at therapeutic doses. Abdominal pain is reported in up to 14% of patients taking traditional stimulants, but incidence of ischemic colitis has not been reported. Acute ischemic colitis due to stimulants is most likely due to hypertension and splanchnic vasoconstriction.
Case history: A 63 year-old female, with history of hypertension, hyperlipidemia, remote tobacco use, and idiopathic hypersomnia with long sleep time (treated with long-acting amphetamine/dexamphetamine (Adderall™) 60mg daily) presented with near syncopal episode followed by 3 episodes of watery diarrhea and hematochezia. CT abdomen/pelvis with angiogram showed acute colitis involving the transverse and descending colon, without evidence of arterial thrombotic or stenotic process. Colonoscopy with histology confirmed severe ischemic colitis. Telemetry and echocardiogram were unrevealing for a cardioembolic etiology; infectious and autoimmune work-up was negative. During her hospitalization, Adderall was discontinued and the patient was started on armodafinil at discharge.
Discussion: Amphetamine-induced systemic effects are mediated by the release of vasoactive amines (dopamine and norepinephrine). Intestinal ischemia results from splanchnic vasoconstriction and/or necrotizing angiitis. Newer generations of stimulant medications are associated with fewer cardiovascular complications compared to traditional stimulant medications. Causality assessment based on the Naranjo probability scale indicates the acute ischemic colitis is probably due to the Adderall.[5] Amphetamine-induced ischemic colitis has been reported most frequently with cocaine, but also with phentermine, pseudoephedrine, methamphetamines.[1-4] Amphetamine-like stimulant prescription is indicated for chronic illnesses, including idiopathic hypersomnolence, narcolepsy, and attention deficit hyperactivity disorder, but the risk/benefit ratio should be re-evaluated regularly as the patient ages.
Conclusions: We have presented a case of ischemic colitis probably due to Adderall use for hypersomnia. While cocaine-induced ischemic colitis has been well described, prescription amphetamine-like psychostimulants can present with similar complications. Heightened awareness for adverse events is warranted in patients on amphetamine-like stimulant medications for chronic indications like idiopathic hypersomnolence, particularly as they age and develop further risk factors for vascular disease.

Key words: amphetamines, ischemic colitis

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Utility of Glucocorticoids in Acute Liver Failure

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Introduction: Oncologic emergencies can be categorized as structural, metabolic, hematologic, or treatment related. Structural oncological emergencies include but are not limited to acute liver failure, superior vena cava syndrome, and airway obstruction. In this case report, we look at a rare case of hyperacute malignant liver failure in the setting of invasive anal carcinoma with neuroendocrine features, and the utility of empiric glucocorticoids in the absence of a histological specimen.
Case Presentation: We present a 45 year-old woman with a history of HTN with recently diagnosed invasive anal carcinoma with neuroendocrine features who presented to the ED with a two day history of progressive dyspnea, abdominal pain, and one episode of "moderate" hematemesis. Physical exam was notable for a tender RUQ and palpation of a firm mass in the RUQ. Laboratory studies revealed elevated transaminases, and she subsequently underwent an abdominal CT demonstrating multiple lesions throughout her liver and lung bases prompting admission. Vascular Interventional Radiology was consulted for a possible liver biopsy for concerns of metastatic pathology. The following day, the patient had an episode of witnessed hematemesis in conjunction with hypotension. Her clinical status progressively worsened; subsequently became altered mentally necessitating intubation and was transferred to the ICU. While in the ICU, the patient's clinical state progressively declined with an INR of 7.1 and transaminases well above 2000 U/L. She went into PEA and underwent multiple rounds of ACLS before having successful ROSC. Her condition continued to deteriorate, and a discussion was had with the family in which her code status was changed to DNR-CCA shortly before she passed away. Post mortem liver biopsy revealed that 90% of her liver was replaced with tumor.
Discussion: Acute liver failure remains a challenging multi-organ clinical syndrome to manage, with transplant being the definitive treatment. There have been limited data on the utility of empiric glucocorticoids in the setting of ALF. Karkhanis et al. conducted a retrospective study examining the overall and spontaneous survival outcomes of patients given glucocorticoids who had autoimmune, indeterminate, or drug induced ALF. They found that glucocorticoids were not associated with improved survival (61% vs 66%, $p=0.41$), and found that glucocorticoid use was associated with diminished survival in certain subgroups of patients (Karkhanis et al). A study conducted by Auzinger et al. found supra-physiological hydrocortisone lowers norepinephrine dosing in vasopressor dependent hypotensive ALF, but did not improve survival outcomes. There is no data on treating fulminant neuroendocrine tumors with steroids, and given the extent of tumor burden as well as the rapidity of deterioration, it is unlikely that glucocorticoids would have provided any benefit to our patient. More research is needed to identify treatments that can stabilize critically-ill patients presenting with advanced malignancies and organ failure.

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The Nervous Jerks: Pregabalin-Induced Neurotoxicity

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Introduction: Pregabalin is a gabapentinoid, originally designed as an antiepileptic drug used in the management of neuropathic pain and fibromyalgia. Albeit rare in medical literature, neurotoxicity associated with the use of pregabalin, presenting as myoclonic jerks, aphasia and dysarthria, has been mentioned in the setting of renal dysfunction. Due to its potentially debilitating effects, clinicians should have a high index of suspicion in order to facilitate prompt treatment.

Case Report: We present a 72-year-old man with a history of systemic hypertension and insulin dependent diabetes mellitus type 2 complicated by diabetic neuropathy. He presented with a one-week history of tremors, progressively worsening over time, associated with difficulty forming and pronouncing words. Prior to that, he mentioned a reduction in his oral intake following a brief viral illness. He had no focal neurologic deficits, and no recent changes to his medication regimen, which included Pregabalin 300 mg twice daily and losartan 50 mg daily. Physical examination was significant for generalized myoclonic jerks with dysarthria. Labs revealed a creatinine of 6.7 mg/dL, BUN 142 mg/dL and a toxic pregabalin level of 36.5 mg/L. Imaging of the brain was unremarkable. Pregabalin was promptly discontinued, and he underwent 2 sessions of hemodialysis with complete resolution of symptoms and full recovery of renal function.

Discussion: Pregabalin is a gabapentinoid developed as a successor to gabapentin, used initially for the treatment of seizures, but now used for managing neuropathic pain and fibromyalgia. Its adverse effect and safety profile have been widely reported since its approval for use in 2004. However, the neurotoxic effects seen in patients with acute or chronic renal dysfunction have been scarcely reported. Pregabalin is completely excreted into the urine without hepatic metabolism, with its clearance directly proportional to that of creatinine. Patients typically present with generalized myoclonic jerks, drowsiness, aphasia and dysarthria. Once neurologic symptoms occur, the drug must be immediately withdrawn. Due to its pharmacokinetics properties, relatively small molecular weight, no protein binging and low volume of distribution (Vd; 0.5 L/kg), hemodialysis is effective in removing pregabalin. Prudence must thus be exercised in prescribing pregabalin in patients with or at risk of renal dysfunction.

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Unusual Presentation of Adult-Onset IgA Vasculitis as Upper Gastrointestinal Bleeding

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Introduction: IgA vasculitis, also known as Henoch-Schoenlein purpura (HSP), is a multi-system small vessel autoimmune disease. In adult HSP, systemic manifestations include non-destructive arthritis, renal derangement, abdominal pain, and a classic lower extremity palpable purpura. Rarely, life-threatening gastrointestinal side effects may occur, such as intussusception, bowel necrosis, bowel perforation, or destruction of the gastric mucosa. This may lead to significant hematemesis. Here, we present a case of an adult male with recurrent hematemesis as the initial presenting symptom of IgA vasculitis.
Case Presentation: An 18-year-old Caucasian male without any significant past medical history presented with a one-day history of hematemesis; Symptoms included meal-independent centralized, constant, non-radiating abdominal pain, diarrhea, fever (T-max 101 F), and chills. Two weeks prior to presentation, the patient had symptoms consistent with a viral upper respiratory infection (URI). One week after the URI, the patient noticed bilateral knee and ankle arthralgias which limited weight-bearing and ambulation. This was followed by a painless, purpuric, non-blanchable rash on the left ankle that extended proximally and bilaterally. The patient presented to the emergency department after experiencing recurrent hematemesis. In the emergency department, patient was afebrile and hemodynamically stable; physical exam was notable for anterior cervical lymphadenopathy, oropharyngeal edema, uvular petechiae, and the palpable purpuric bilateral lower extremity rash. Notable labs on admission included WBC 10.8, hemoglobin 15.8, hematocrit 46.5%, platelets 243, creatinine 0.97, CRP 4.8. Urine dipstick was positive for trace hemoglobin and proteinuria. The following labs were within normal limits: ANA, cANCA, pANCA, C3, C4, HIV screen, and serum electrophoresis. Upper endoscopy showed non-bleeding erosive gastropathy, duodenal erosions, and erosive mucosal changes. Skin biopsy of the left leg showed leukocytoclastic vasculitis and deposition of IgA and fibrin within the walls of the superficial dermal vessels. This confirmed the diagnosis of IgA vasculitis. The patient received IV methylprednisolone 500mg daily for three days. He then required two outpatient methylprednisolone infusions and was transitioned to prednisone taper in tandem with mycophenolate mofetil 1000mg BID due to relapse after initial treatment with steroids only. This regimen was maintained for three months, then it was tapered off gradually, and the patient remained in remission.
Discussion/Conclusion: IgA vasculitis should be included as a rare but significant differential diagnosis in adult patients who present with hematemesis. While HSP is often self-limited in pediatric populations, adults with HSP may suffer devastating, irreversible consequences if their symptoms are not properly identified or promptly treated. Adults with HSP should be screened for renal involvement and evaluated for serious GI manifestations. Here, we present an unusual presentation of IgA vasculitis with upper gastrointestinal bleeding.

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CRRT/UF for therapeutic cytokine removal in a patient with COVID-19-induced (HLH)-like syndrome

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Introduction: COVID-19-related Hemophagocytic Lymphohistiocytosis (HLH)-like syndrome is an increasingly recognized constellation of symptoms involving cytokine storm in the presence of COVID-19 infection. Certain inflammatory biomarkers, including procalcitonin, lactate dehydrogenase (LDH), C-reactive protein (CRP), and ferritin, are proxy indices that can be used to identify the syndrome. Currently, there are no FDA-approved therapies to combat the cytokine storm or the ensuing immune over-stimulation that follows. Extracorporeal removal of cytokines with RRT (renal replacement therapy) has been theorized as a mode to decrease overall cytokine burden and early RRT may be a temporizing measure in the ICU setting.

CRRT /UF are common interventions in critically ill patients requiring renal replacement therapy. Due to the novelty of SARS-CoV-2 and its management, there is a paucity of data in treating AKI in patients with acute COVID-19 infection. Our case describes obstacles secondary cytokine storm in (HLH)-like COVID-19 syndrome, and the potential benefit that early identification and treatment can provide.

Case Presentation: The patient is a 65 year old man with a PMHx of psoriasis who was admitted to the ICU after a week of progressive shortness of breath, fatigue, cough, fever, and rash. Upon admission nasopharyngeal swab for COVID-19 was obtained and eventually resulted positive . His viral pneumonitis evolved into fulminant acute respiratory distress syndrome (ARDS) and multiorgan failure, requiring intubation, mechanical ventilation, and continuation renal replacement therapy (CRRT). Due to his worsening illness and high acuity, the patient also received convalescent plasma exchange for three cycles.

Results: The patient met diagnostic criteria for HLH and cytokine storm with elevated biomarkers (LDH, ferritin), transaminitis (elevated AST), hematologic abnormalities (pancytopenia with low fibrinogen) and fever in the absence of HIV or HCV. Subsequently, the patient was initiated on IV methylprednisolone therapy for suspicion of HLH-like syndrome.

After initiation of CRRT, the patients fever curve dramatically improved. Additionally, his kidney function improved and progressed from anuria to non-oliguric urine production and CRRT was discontinued; he was kept on UF for fluid overload. As evidenced graphically, ferritin, fibinogen and other biomarkers improved with RRT.

Discussion: In the critical care setting, early identification of the syndrome is crucial, as CRRT / UF may be more likely to reduce cytokine burden and improve mortality. COVID-19 + patients presenting with an AKI may be less likely to clear cytokines, and earlier initiation of CRRT may prove to be beneficial. During the COVID-19 pandemic era, with high infection and high mortality rates, patients with certain risk factors, including rheumatological disease as seen in this patient, may present as higher risk for development of HLH/cytokine-mediated multiorgan failure. While cytokine-blocking medications are in development, RT may be a considerable benefit to lower cytokine burden in patients with critical illness due to COVID-19.

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A Rare Glomerulonephropathy in a Common Presentation: Nephrotic Range Proteinuria

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Introduction: Fibrillary glomerulonephropathy (FGN), a rare cause of primary glomerular diseases, is characterized on electron microscopy (EM) by haphazardly arranged, straight fibril deposits, measuring 10-30 nm in thickness, in the mesangium and glomerular basement membranes. We present two cases presenting with nephrotic range proteinuria, of which biopsies revealed FGN.

Case Report(s): Case 1: A 70-year-old female with a history of hypertension (HTN), hypothyroidism, degenerative joint disease, perforated gastric ulcer, and nephrolithiasis, was admitted with altered mental status and volume responsive acute kidney injury. Creatinine was 2.0 mg/dL; baseline was 0.9 mg/dL. Physical exam was unremarkable without edema. Because of large proteinuria, urine microalbumin/creatinine ratio (UACR) of 7837.5 mg/g and protein/creatinine ratio (UPCR) of 10.3 g/g, kidney biopsy was obtained. Light microscopy (LM) showed mesangial expansion with hypercellularity; immunohistochemical stain was positive for DNAJB9, and fibrillary deposits were seen on EM. Workup for autoimmune disease was unremarkable. The most recent serum creatinine was 1.4 mg/dL, 8 months after biopsy.
Case 2: A 67-year-old male with a history of uncontrolled HTN, iron deficiency anemia, osteoarthritis, and gout presented for outpatient evaluation, referred by his PCP after a finding of proteinuria. Creatinine was 1.4 mg/dL. Spot UPCR was 7.7 g/g and UACR was 4227 mg/g. Physical exam was unreamarkable; he had no edema. Albumin was 3.8 g/dL. Kidney biopsy showed EM findings consistent with FGN with mesangial expansion and positive immunohistochemical stain for DNAJB9. Autoimmune and hepatitis workup were negative except for a low titer (1:40) of anti-double-stranded DNA. He continues on home dialysis.

Discussion: FGN is a rare cause of primary glomerular diseases (0.5 – 1% of native kidney biopsies). FGN overall has a poor prognosis; 86% of patients either reach end-stage renal disease or persistent renal dysfunction with no statistical benefit from immunosuppressive therapy. Both patients exhibited a preserved renal function at presentation, with large nephrotic range proteinuria. Kidney biopsy in both revealed FGN, with the presence of fibrillar deposits, a positive immunohistochemical stain for DNAJB9. One of the patients is presently on renal replacement therapy. Since described in 1977, more and more FGN cases have been described in the literature. Given its silent presentation and limited access to electron microscopy, clinicians may miss the diagnosis. With recent advancements in technology, especially the immunohistochemical staining for DNAJB9 which is very specific for FGN, these cases can be diagnosed by obtaining a kidney biopsy.

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An Extreme Case of Renal Tubular Acidosis Type 1 from Malnutrition and Chronic Interstitial Nephritis

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Introduction: Renal tubular acidosis can cause rapid and progressive electrolyte abnormalities. Unrecognized, complications can wreak havoc. In this case, we highlight complications from Type 1 RTA in conjunction with poor oral intake and cocaine abuse.

Case Report(s): A 49-year-old Caucasian female with history of polysubstance abuse (cocaine and opiates) and anxiety was admitted for obtundation and evaluation of acute encephalopathy. Blood cultures, lumbar puncture and head CT were all negative. She exhibited severe hypokalemia (<1.5), hypophosphatemia (0.4), HAGMA, NAGMA, respiratory alkalosis, and hypocalcemia. She was admitted to the medical ICU. Electrolytes were replaced at an appropriate rate; however, the patient developed respiratory distress and was unable to protect her airway. After intubation and during her ICU stay, testing revealed electrolyte abnormalities consistent with RTA Type 1. Extensive workup ruled out extra-renal causes. Electrolyte abnormalities stabilized, and she was extubated. Mentation returned to baseline, and she was discharged home with supplementation of potassium citrate, 30 mEq BID.
Discussion: Type 1 Renal Tubular Acidosis (Distal RTA) occurs when damage occurs to the alpha-intercalated cells of the distal tubule, preventing further production of bicarbonate and hydrogen ions. Urine is acidified, preventing excretion of acids. The inability to excrete H+ ions results in malfunctioning H/K ATPase. Symptoms include fatigue, muscle weakness, respiratory distress, neurological tingling, muscle spasms, heart rhythm abnormalities such as prolonged QTc, nephrolithiasis, dehydration, and osteoporosis. In our case, electrolyte abnormalities resulted in acute hypoxic respiratory failure. Hypophosphatemia was due to hypovitamin-D-osis which has been linked with other forms of renal tubular acidosis as well as osteopenia. Since discharge, the patient has not been evaluated for osteopenia with DEXA, but with evidence of rib fractures, it should be considered. There are various causes of distal RTA including: autoimmune, hypergammaglobulinemic state, drugs (lithium, amphotericin B, NSAIDS, lead, antivirals), toluene, and tubulointerstitial disease. In the case of our patient, all extra-renal causes were ruled out through laboratory testing. We summarize that the patient developed RTA Type 1 due to either acute or chronic interstitial nephritis. Testing ruled out other causes of distal RTA; yet, the patient never had a formal renal biopsy to definitively note interstitial nephritis. Interstitial Nephritis has a variety of causes, including cocaine abuse, as in our patient. Further evaluation of the patient to monitor for changes, as well as follow her hepatic anatomical changes, is needed.

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Successful Management of Acquired Ventricular Septal Defect Secondary to Late Presentation of ST-Elevation Myocardial Infarction

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Ventricular Septal Defect (VSD) is a rare complication of ST elevation myocardial infarction in current era of early reperfusion. Immediate recognition and urgent management can be lifesaving in these patients.

A 68-year-old man with no significant past medical history presented to the emergency department with progressive chest pain and shortness of breath one week after symptom onset. He was tachycardic with hypotension and physical examination findings consistent with cardiogenic shock. Cardiac examination was significant for a holosystolic murmur at left sternal border and at the apex. EKG showed sinus tachycardia with ST elevation in anterior leads along with Q waves. Troponin I was elevated at 0.349 ng/mL (upper normal 0.120). Brain natriuretic peptide was elevated at 1350 pg/mL. Chest X-ray showed pulmonary edema and cardiomegaly. A stat echocardiogram showed akinetic mid anteroseptal and apical walls with 1.7cm ventricular septal defect in the mid-distal anteroseptal wall. Emergent left heart catheterization confirmed mid LAD occlusion and a significantly left ventricular end-diastolic pressure of 30 mmHg (normal <12mmHg). An intra-aortic balloon pump (IABP) was placed and patient required pressors for hemodynamic support. He underwent urgent cardiac surgery with anterior VSD closure with Hemashield patch and coronary artery bypass graft on hospital day 2. He did well post-operatively and was discharged home on atorvastatin, aspirin, and furosemide. Subsequent echocardiogram showed EF of 46% with mild to moderate mitral regurgitation. Patient has completed cardiac rehabilitation and remains symptom-free.
This case highlights prompt recognition and treatment of VSD secondary to STEMI is critical for patient survival. The complication carries a high in-hospital mortality; 45% for surgical vs. 90% for medically managed patients. Key learning point of this case: (1) Acute left ventricular heart failure and cardiogenic shock may be presentation of a mechanical complication of STEMI (2) The presence of holosystolic murmur in an MI patient should prompt emergent evaluation of VSD, acute mitral regurgitation or free wall rupture. (3) Early recognition and diagnosis, prompt mechanical support and urgent surgery are key to successful survival in these patients.

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Oncogenic osteomalacia – brittle bones and a hidden tumor

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Introduction: Severe hypophosphatemia, defined as a level under one mg/dL, can pose significant morbidity and mortality secondary to adenosine triphosphate depletion. Most cases of hypophosphatemia occur secondary to renal losses as diagnosed by a urinary fractional secretion of phosphate greater than five percent or twenty-four-hour urine phosphate excretion greater than 100mg. Below we present an unusual case of severe hypophosphatemia, which upon exclusion of alternative causes, is most likely related to tumor-induced osteomalacia (TIO). TIO, also know as oncogenic osteomalacia, is a rare paraneoplastic syndrome in which mesenchymal tumors secrete fibroblast growth factor 23 (FGF23). This acts on the renal tubule to impair phosphate reabsorption and 1α-hydroxylation of 25-hydroxyvitamin D, resulting in chronic hypophosphatemia which causes bone pain, nontraumatic fractures and muscle weakness.
Case Presentation: A 36-year-old male presented to the ED with four weeks of progressively worsening lower extremity weakness and myoclonic jerking, now requiring a walker to ambulate. He denied saddle anesthesia, decreased sensation, trauma, recent upper respiratory infection or diarrheal illness. The physical exam was significant for bilateral five-beat ankle clonus, hyperreflexia in the achilles and patella, and quadriceps weakness. Laboratory studies were significant for a phosphate of <0.7 mg/dL, 1,25-dihydroxyvitamin D 60 ng/mL, and 24-hour urine phosphorous excretion of 2278 mg. Despite aggressive intravenous (IV) phosphate repletion, the patient continued to be hypophosphatemic with high urinary phosphate. Standard bone scan and octreotide scan, ordered to rule out a neuroendocrine tumor, were both normal. Given the severity of the hypophosphatemia, nontraumatic fractures, and osteoporosis with muscle weakness, the patient was treated for hypophosphatemic rickets. Therapy with oral potassium 1000 mg four times daily, calcitriol 0.5 mg twice daily and elemental calcium 400 mg twice daily yielded symptomatic improvement and potassium levels rose to 2.7 mg/dL. Laboratory analysis showed a FGF23 of 5460 RU/mL concerning for oncogenic osteomalacia. A subsequent fluorodeoxyglucose (FDG)-positron emission tomography (PET) and whole body magnetic resonance imaging (MRI) searching for definitive tumor location was inconclusive. The patient was continued on elemental replacement therapy with the intentional of observing further tumor growth for better evaluation of the source.
Conclusion: The major limitation for definitive treatment of TIO stems from the difficulty in tumor localization. Unfortunately, advanced imaging modalities such as FDG-PET, octreotide scintigraphy, and whole body MRI are often unsuccessful in localizing the tumor as seen in this case. Selective venous sampling with FGF 23 measurement can also be used. If the tumor is not localized, phosphate supplementation and active vitamin D can be used to successfully manage the disease. This case highlights an uncommon presentation of hypophosphatemia secondary to an undiscovered tumor, which was treated with conservative management.

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Atypical presentation of Nodular lymphocyte-predominant Hodgkin Lymphoma

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Nodular lymphocyte-predominant Hodgkin Lymphoma (NLPHL) is an extremely rare subtype of Hodgkin Lymphoma (HL), characterized by slow growth, and usually presents as early stage disease with peripheral lymphadenopathy. Early stage NLPHL has excellent survival rates, whereas patients with advanced disease have high relapse rates. Treating NLPHL can be challenging as there is limited literature regarding its management.

A 51-year-old male with a medical history of depression and anxiety presented at the time of admission with acute encephalopathy and expressive aphasia. Initial diagnostic work-up showed enhancement in the right basal ganglia and thalamus, edema, and leftward midline shift on MRI brain, as well as an elevated protein level and lymphocyte count in the cerebrospinal fluid (CSF). Neurology was consulted and he was treated immediately with a five-day course of intravenous methylprednisolone and acyclovir. CSF cytology had no immunophenotypic evidence of malignancy. Full work-up revealed large soft tissue lesions in the right lower quadrant mesentery on CT abdomen and pelvis. PET scan confirmed the hypermetabolic abdominal mass and showed additional hypermetabolic mesenteric lymph nodes, compatible with malignant involvement. Biopsy of the mass was positive for NLPHL.

Repeat MRI brain after completing intravenous treatment revealed significant resolution of the lesions, associated edema and shift. He was discharged with close hematology and neuro-oncology outpatient follow up.

Since discharge, he has been undergoing serial imaging and his symptoms have largely improved without any cognitive deficits. Due to the localization of his disease, radiation therapy was not a viable option; this left observation or systemic therapy with chemoimmunotherapy or rituximab monotherapy. Given the patient's low disease burden, he opted for observation. A 6 month follow up MRI brain showed majority of the enhancing lesions had resolved. CT abdomen and pelvis showed interval decrease in size of the mesenteric soft tissue lesions with no new lymphadenopathy. PET imaging showed improvement of abdominal adenopathy, and no evidence suggesting transformation without interval treatment. He continues with close follow up and surveillance imaging.

This case demonstrates the importance of completing a thorough diagnostic work-up and starting immediate treatment for the patient's symptoms. It is still unclear what caused the neurologic symptoms; due to the time course, imaging findings, and no evidence supporting transformation of NLPHL to diffuse large B-cell lymphoma, central nervous system involvement by lymphoma was considered unlikely. An inflammatory or infectious process seemed more probable, although paraneoplastic syndrome was not excluded given the findings of NLPHL. If there is an association between NLPHL and neurologic manifestations, an in-depth neurologic evaluation in patients with a high suspicion of disease could aid in its recognition. Early detection and a comprehensive analysis are vital to initiate timely therapy and prevent disease transformation.

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Disease Modifying or Disease Causing? A case of methotrexate induced colitis

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Methotrexate is a widely used drug, reputable for its anti-neoplastic and anti-inflammatory properties. Via its mechanism of action, methotrexate can paradoxically cause gastrointestinal side effects such as nausea, vomiting and stomatitis. The development of colitis however, is a rare phenomenon and we report this case in order to highlight this toxic effect.

The patient is a sixty three year old female with a past medical history of psoriatic arthritis and stage 3 chronic kidney disease. She was on low dose methotrexate therapy of 15mg weekly for the past three years. She presented with a one week history of painful mouth ulcers, abdominal pain, non-bloody vomiting and diarrhea. Vital signs on presentation were temperature of 36.8 C, blood pressure 139/82 mmHg, pulse of 101 beats/minute, respirations of 20 breaths/minute and saturating 99% on room air. Her physical exam was remarkable for multiple oral aphthous ulcers. There was generalized tenderness in the abdomen without guarding or rigidity. Bowel sounds were present and normal. The remainder of the physical examination was non-contributory.

Her lab work was significant for WBC of 3.4 K/uL, Hb of 12.9 g/dL and platelet count of 126 K/uL. The patient was initially managed conservatively for acute gastroenteritis. She did not improve clinically. The presence of aphthous ulcers, persistent diarrhea and elevated ESR of 59 led to the possible diagnosis of Crohn's disease for which the patient underwent endoscopy. Upper endoscopy revealed gastritis, duodenitis and a superficial duodenal ulcer. Colonoscopy was significant for patchy areas of ulceration in the transverse and descending colon. Microscopy of the biopsies taken revealed acute inflammatory pseudomembranes and marked stromal inflammation. There was minimal cryptitis and mild superficial crypt abscess formation. No granulomata were seen. The histopathological pattern was not typical of that of inflammatory bowel disease. Clostridium difficile testing was negative. The possibility of CMV colitis was entertained however, viral inclusion bodies were not identified. The result of CMV DNA PCR testing was negative. On day three of admission, the complete blood count was alarming for a pancytopenia and worsening thrombocytopenia of 35 K/uL. Methotrexate induced colitis was concluded to be the cause and was supported by the presence of concurrent pancytopenia and mucositis. The patient was treated with oral folinic acid which resulted in significant clinical improvement and recovery of cell lines.

A few case reports have described the manifestation of colitis with methotrexate use, with severe complications such as toxic megacolon. Folic acid supplementation can reduce the incidence of gastrointestinal side effects however, patients are still susceptible to methotrexate toxicity. Physicians should be cognizant of the uncommon potential for methotrexate induced colitis despite folic acid use.

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A case of rhabdomyolysis associated with re-initiation of levetiracetam

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Introduction: Rhabdomyolysis is a very rare but documented adverse drug reaction of the anti-epileptic levetiracetam (1). We present a case of rhabdomyolysis associated with re-initiation of the drug. The case is particularly unusual in that the patient had previously been taking levetiracetam before discontinuing over a year prior.

Case Description: A 22 year old man with a history of epilepsy, developmental disability, and schizoaffective disorder presented from a jail after being found unresponsive. On arrival, he had a witnessed seizure which was treated with lorazepam 1 mg plus levetiracetam 1000 mg. Admission labs were notable for elevated creatinine kinase (CK) at 377 U/L, lactic acid of 3.4 mmol/L, and a serum sodium of 127 mmol/L. He was given a 2L fluid bolus and started on continuous fluids for treatment of rhabdomyolysis. Levetiracetam was continued at a dose of 1500 mg twice daily. He was previously on levetiracetam and lamotrigine but he had not filled his anti-epileptics for over 1 year. CK increased to 9708 U/L, and delayed CK peak due to the initial seizure was thought to be the explanation, since he had no additional seizures in the interim. However, despite appropriate treatment of rhabdomyolysis with boluses, continuous fluid replacement, and normal renal function, CK rose rapidly. Last dose of levetiracetam was given at 72 hours before it was identified as a possible cause of the rhabdomyolysis; CK rose to a peak of 27,501 U/L at 79 hours. Patient was not on any other possible causative medications. Concerns were discussed with Neurology who replaced levetiracetam with lacosamide. After discontinuation, and with continued fluid replacement, CK gradually fell to 9497 U/L on hospital day 6, and the patient was discharged in good condition.

Conclusion: We suspect that levetiracetam was the cause of the rhabdomyolysis. This is supported by the dramatic rise in CK after re-initiation, peak >72 hours after initial seizure, lack of improvement despite aggressive fluid replacement, absence of other identified culprit medications or causes, and improvement with discontinuation. In one similar case, a 42 year old man treated with levetiracetam for hyponatremic seizure had CK rise to 30,000 U/L on hospital day 3, and levels promptly improved after discontinuation (1). Overall, our case highlights the need to consider newly initiated or re-initiated medications in cases of rhabdomyolysis, especially when other causes no longer explain the clinical course.

Reference: 1. Rastogi V, Singh D, Kaur B, Arora P, Gadikota JP. Rhabdomyolysis: A Rare Adverse Effect of Levetiracetam. Cureus. 2018;10(5):e2705.

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IgA Nephropathy as a Rare Cause of Rapidly Progressive Glomerulonephritis

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Introduction: IgA nephropathy (IgAN) is the most common cause of glomerulonephritis worldwide; however, it is seldom reported as a cause of rapidly progressive glomerulonephritis (RPGN). Furthermore, it is not commonly reported as a cause of glomerulonephritis in the African American population. Nearly one-third of patients with IgAN present only with asymptomatic hematuria with or without proteinuria. However, IgAN can rarely present as a more aggressive RPGN, and patients may display different degrees of renal dysfunction, hypertension, edema, and proteinuria. Prompt renal biopsy is often needed in these patients, as management often differs depending on the underlying pathology.

Case Description: A 26-year old African American man with no prior medical history presented with acute-onset shortness of breath, cough, and musculoskeletal chest pain. Lab work showed a new acute kidney injury with a creatinine of 4.02 mg/dL and notable proteinuria with a urine microalbumin-creatinine ratio of 7.5 mg/g. Further lab work showed a negative ANA, negative ANCA, normal C3 and C4 levels, negative HIV screen, negative hepatitis panel, normal plasma aldosterone-renin activity ratio, and normal plasma metanephrine level. Although urinary free kappa light changes and free lambda light chains were elevated, the free kappa-lambda light chain ratio was within normal limits. Physical exam was only significant for chest wall tenderness on the left side, LLQ pain, and L costovertebral angle tenderness. Renal biopsy was performed, and surgical pathology revealed IgA-mediated immune-complex glomerulonephritis with marked arteriosclerosis and glomerulosclerosis. He received high-dose intravenous steroids for three days and was started on diuretic therapy. The patient improved significantly and was discharged on a prolonged steroid taper. Upon follow-up with nephrology, the patient was started on immunomodulatory therapy with mycophenolate mofetil therapy for his biopsy-proven rapidly progressive IgA crescentic glomerulonephritis.

Discussion: Rapidly progressive crescentic IgA nephropathy is rare, and there are few reported cases showing evidence of high risk of progression to end-stage renal disease (ESRD) with variable response to immunosuppression. In fact, current data suggests that the renal survival in cases of rapidly progressive crescentic IgAN is 50% at one year and 20% at five years. There is only recently published data supporting the use of high-dose steroids and immunomodulator therapy in patients with crescentic IgAN. Therefore, although a rare cause of RPGN, IgAN should be considered, and kidney biopsy should be pursued early in order to prevent delays appropriate renal-saving therapy.

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Partial Anomalous Pulmonary Venous Return: An Incidental or Consequential Finding?

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Partial anomalous pulmonary venous return (PAPVR) is a congenital cardiovascular anomaly characterized by failure of embryologic connection of one or more, but not all, of the pulmonary veins to the left atrium. We present a case of PAPVR in a patient with exertional chest pressure and dyspnea that highlights the symptomatology, management, and diagnostic workup of PAPVR.

A 53-year-old male with a history of recurrent deep vein thrombosis (DVT), pulmonary embolism (PE) status-post inferior vena cava (IVC) filter placement, and failure of multiple anticoagulants presented with months of exertional dyspnea and chest pressure. He presented to an outside hospital several weeks before admission for the same symptoms and was diagnosed with a small, sub-segmental PE and switched from enoxaparin to apixaban. A computed tomography (CT) scan with pulmonary artery protocol performed in the emergency department did not reveal acute PE, but did reveal partial anomalous pulmonary venous return with several large pulmonary veins draining from the right upper lobe into the superior vena cava (SVC) with right ventricular prominence. He underwent a left and right heart catheterization. Left heart catheterization (LHC) did not reveal any significant coronary artery disease. Right heart catheterization demonstrated pulmonary artery pressure (PAP) of 23/2 (mean 13 mmHg), pulmonary capillary wedge pressure of 6 mmHg, and right ventricular pressure of 26/10 (mean 9 mmHg). Oxygen saturations were 80% in the RV, 76% in the pulmonary artery, 81% in the IVC, 95% in the aorta, and 96% in the SVC with anomalous return of right upper lobe vein to the SVC. Transesophageal echocardiogram demonstrated moderate dilatation of the right ventricle without atrial septal defect (ASD) or patent foramen ovale (PFO). He was discharged with a plan to obtain an outpatient cardiac MRI.

PAPVR is a rare condition that occurs in only 0.4-0.7% of the general population. Patients are most commonly asymptomatic; however, they can present with dyspnea, decreased exertional tolerance, fatigue, and peripheral edema. Those who do have symptoms from right-sided cardiac volume overload can present any time from early childhood to late adulthood. Left-to-right shunting in patients with PAPVR leads to chronically increased pre-load that can cause progressive dilation of the tricuspid annulus and right ventricle. Right ventricular (RV) enlargement, development of symptoms, or presence of significant shunt (pulmonary-to-systemic flow ratio >1.5) is an indication for surgical intervention. In asymptomatic patients with low shunt fraction and no evidence of RV dysfunction, close monitoring is recommended. We concluded that in our case, the shunt might not be significant, and the lack of pulmonary hypertension warrants close monitoring. The cardiac MRI will aid in accurately assessing the anatomy of the shunt, quantify the shunt fraction, and provide information on the RV function.

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Three False Negative RT-PCR Tests: A Case of COVID-19

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Introduction: The testing of severe acute respiratory syndrome coronavirus 2 (SARS-CoV2), the virus known to cause COVID-19, has been inconsistent and at times unreliable as health care facilities, laboratories, and clinicians struggle to tackle the pandemic. We present a critically ill patient who tested negative for COVID with three tests before testing positive on the fourth test.

Case Description: A 74 year old male presented to the ED for shortness of breath in an area with a high number of early COVID-19 cases. He was hypoxic on arrival with tachypnea requiring supplemental oxygen. Despite high flow oxygen and furosemide, he remained hypoxic. He was intubated due to worsening hypoxia, respiratory distress, and to limit spread of suspected SARS-CoV2. The first COVID PCR test was sent using a nasopharyngeal swab and transferred to our hospital, located in a different state. The first test was negative. His hospital course was complicated by acute respiratory distress syndrome and septic shock with little improvement on broad spectrum antibiotics and negative cultures. Inflammatory markers on admission included LDH 875 U/L, D-Dimer 1030 ng/mL and Ferritin 2800 ng/mL. These trended upwards before decreasing. Serial chest imaging showed worsening diffuse airspace opacities. A second nasopharyngeal swab was collected and PCR testing at a different laboratory in state was negative. The third test performed at the same laboratory was also negative. Due to high clinical suspicion for COVID-19, the decision was made to obtain a sample by brochoalveolar lavage (BAL). The PCR from this fourth sample was positive for SARS-COV-2 virus. He received Tocilizumab once during admission but ultimately passed away.

Discussion: Our patient tested negative for SARS-COV-2 virus with three PCR tests obtained from nasopharyngeal samples sent to two seperate labs in two states before finally testing positive on the fourth test using a BAL sample. As evidence has emerged throughout the pandemic, the sensitivity of these PCR tests has been a growing concern. There has not been a systematic review. However, one small study examining known COVID cases showed BAL samples were correctly positive in 95% of patients while pharyngeal samples were only positive in 32% of known cases [1]. Two other studies showed 11-29% of patients were repeatedly negative but became positive after four to five tests [2,3]. As such, the accuracy of the test likely depends on the assay used, duration of illness when tested, and type of specimen collected. Viral loads are likely higher in the lower respiratory tract. This could explain the significant sensitivity difference between nasopharyngeal and BAL samples. Nasopharyngeal samples remain an appropriate first line test given the low risks and costs, but clinicians should use clinical suspicion to guide further workup when initial testing is negative.

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Steroid-unresponsive Immune-mediated Hepatitis Induced by Durvalumab: A Case Report

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Background: Lung cancer is the leading cause of cancer death in the United States. Durvalumab is a monoclonal antibody against programmed cell death ligand (PD-L1) and CD80 used for the treatment of stage III non-small cell lung cancer. Immune-mediated hepatitis is a common side effect of durvalumab, which is reported in 12% of patients. However, most durvalumab-induced hepatitis is mild and progression to severe (grade 4) immune-mediated hepatitis is rare and seen in only 0.4% of patients. Of these patients, only 1.7% required corticosteroids, and mycophenolate was required in 0.1%. We report a case of grade 4 immune-mediated hepatitis induced by durvalumab, which was unresponsive to high-dose corticosteroids and needed treatment with mycophenolate.

Case presentation: A 78-year-old female with a history of lung adenocarcinoma presented with abnormal liver function tests on routine screening after two cycles of durvalumab. The patient reported jaundice, pale stools, dark-colored urine, and pruritus. On admission, her vitals were normal. Initial labs revealed a significant elevation in total bilirubin, alkaline phosphatase, alanine aminotransferase, and aspartate aminotransferase. She was started on high-dose steroids for grade 4 immune-mediated hepatitis. Initially, she showed temporary improvement on steroids but declined on day 2 of admission with an increase in total bilirubin and alkaline phosphatase. Mycophenolate was added on day 4, and Magnetic resonance cholangiopancreatography was done and ruled out obstruction. The administration of mycophenolate provided a gradual improvement of hepatitis. However, on day 6, a sharp decline in her pulmonary function prompted a transfer to ICU for acute respiratory failure, which was likely secondary to immunotherapy. On day 9, the patient elected to withdraw her treatment and be admitted to hospice.

Conclusion: We describe a rare case of steroid-unresponsive severe immune-mediated hepatitis induced by durvalumab. As the use of durvalumab is rising following FDA approval, physician cognizance of immune-mediated hepatitis induced by durvalumab is important. This requires careful monitoring of liver function tests in cancer patients on immune checkpoint inhibitors such as durvalumab, and demonstration of acute liver injury should be evaluated and managed promptly.

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A Case with Successful Targeted Therapy of BRAF Positive Esophageal Cancer

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Introduction: With better understanding of molecular pathways involving different types of cancers, targeted therapies (TT) have rapidly emerged as viable treatment options. Unfortunately, TT for metastatic esophageal adenocarcinoma (EAD) has not shown promising results. Currently, the only biomarker driven therapy for advanced EAD is trastuzumab which is used in HER-2 positive disease. We present a unique case in which combination therapy with BRAF and MEK inhibitors achieved surprising results in metastatic EAD.

Case: A 68 year old male with PMH of GERD presented with dysphagia and unintentional sixty pound weight loss. Multiple esophagogastroduodenoscopies (EGD) were performed with initial findings of atypical squamous cells but final EGD results demonstrated eosinophilic esophagitis. Due to the severity of presentation and high suspicion of malignancy, patient underwent esophagectomy. Pathology showed moderately to poorly differentiated adenocarcinoma with invasion of muscularis propria and focal advancement into the adventitia. Pleural effusion was noted and fluid cytology also showed adenocarcinoma. Initially, there was no evidence of metastatic disease on CT imaging, but PET scan showed metastasis of vertebral bones and subcarinal lymph nodes.
Patient completed seven cycles of FOLFOX with minimal improvement. Second line therapy with irinotecan was discontinued due to side effects. Patient developed worsening bilateral pleural effusion and bony metastasis. Next generation sequencing (NGS) resulted in BRAF and PIK3CA mutations with negative microsatellite instability. Subsequently, combination therapy with BRAF and MEK inhibitors (dabrafenib and trametinib) was initiated. Pleural effusion improved within 3 months. Patient has been on combination therapy for more than sixteen months and he has stable disease based on last imaging.

Discussion: EAD is a fatal malignancy with a six fold increase seen in recent decades. The primary modality of treatment for EAD is cytotoxic chemotherapy. A multicenter, Phase III clinical trial lead to the most effective regimen (epirubicin, oxaliplatin and capecitabine) with an overall median survival of only 11.2 months. Even with other first line chemotherapy options, survival remains low.
Several studies were conducted to characterize the molecular basis of EAD. Researchers examined success of current trials targeting different molecular alterations (FGFR2 and MET mutations) but with unsatisfactory results.
BRAF TT is considered to be an active treatment for BRAF-positive metastatic melanoma, lung and colon adenocarcinomas. However, clinical resistance is typically seen within six to seven months of treatment. Current data suggests that MAPK reactivation, which is downstream from the BRAF pathway, is a possible cause of resistance. Therefore, combined therapy with BRAF and MEK (MAPK kinase) inhibition has shown better results.
After thorough literature review, we could not find similar cases of metastatic EAD where combination therapy with BRAF and MEK inhibitors has shown such positive results. This emphasizes the importance of NGS, offering different treatment modalities for various types of cancers.

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A Rare Case of Pulmonary Inflammatory Myofibroblastic Tumor (PIMT): A Diagnostic Challenge

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Inflammatory myofibroblastic tumors (IMT) are mesenchymal neoplasms that can arise in the soft tissues of nearly any organ. IMTs have a predilection for the abdomen and pelvis and occur primarily in pediatric and adolescent populations. They are rarely found in adults and constitute less than 1% of all adult lung tumors. Due to the rarity of these tumors, the biological behavior, natural course, and response to therapy are not well understood. We present a case of an adult female who presented with a pulmonary IMT to illustrate the challenge of clinical and radiological diagnosis of IMTs and subsequent treatment approach, due to non-specific patient presentations and variable pathogenesis.

Case: A 28-year-old female with hypogammaglobulinemia presented to the ED with a one-day history of hemoptysis. Review of systems was positive for fever, chills, night sweats and progressive shortness of breath for 3 months. A CT pulmonary-artery scan demonstrated a large right hilar mass (55x47 mm) narrowing the bronchus intermedius and encasing the right lower lobe pulmonary artery. Infectious workup was negative. CT-guided needle biopsy of the mass showed hypercellular spindle cell lesion with occasional cells showing nuclear atypia, concerning for neoplastic versus benign process. Repeat biopsy and analysis found positive staining for muscle specific actin (MSA), desmin, and strongly-positive anaplastic lymphoma kinase-1 (ALK-1), along with morphology supporting a diagnosis of IMT. Positron-emission tomography showed markedly increased F-fluorodeoxyglucose uptake, consistent with biopsy-proven IMT. Several smaller hypermetabolic lung nodules concerning for metastasis were also noted. ALK-1 inhibitor-therapy with alectinib was initiated. At 3 months, CT chest with intravenous contrast found significant decrease in size of the mass, now 29x20 mm, and of pulmonary nodules. Surgical resection is planned.

Discussion: IMTs include an array of pulmonary lesions. Synonyms most commonly include plasma cell granulomas, and inflammatory-psuedotumors. It remains unclear whether IMTs constitute a primary inflammatory response, or whether they are an inflammatory response to a low-grade malignancy. Diagnosis is obtained via biopsy or surgical resection; however, biopsy can often yield a false-negative result, requiring resampling and delay in treatment. There is significant controversy regarding pathogenesis and natural course of progression. Much of the controversy stems from the varying degree of inflammatory cells present on pathology. Though histological findings vary, the most common finding are myofibroblastic/fibroblastic spindle cells with mononuclear inflammatory cells. Due to their diverse origin, IMTs tend to stain positive for vimentin, desmin, smooth muscle actin, MSA, and most distinctively ALK-1. Treatment most often includes surgical resection; recently ALK-1 inhibitors have been used for cytoreduction followed by surgical intervention showing favorable response.

Teaching Point: PIMTs must be considered in the differential of pulmonary masses, as treatment and surgical intervention can be curative.

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A Deceptive Case of Conjugated Hyperbilirubinemia

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Conjugated bilirubinemia is generated by diseases that cause the hepatobiliary system to reflux conjugated bilirubin from the liver back into the blood. In addition to causing liver damage from hypotension, infections causing septic shock may release endotoxins that can affect bile acid transportation as well as injure hepatocytes and disturb bile flow. (2) Cholestasis and conjugated hyperbilirubinemia from infection are most often described in the setting of septic shock. (3) Here we describe a case of conjugated hyperbilirubinemia despite the resolution of sepsis suggesting these endotoxic effects can play a role in cholestasis and/or liver dysfunction even in settings without sepsis.

Case Presentation: A 66-year-old male with atrial fibrillation on warfarin, end-stage renal disease requiring hemodialysis, and chronically elevated alkaline phosphatase presented with right lower extremity (RLE) pain (distal to the knee and proximal to the ankle) rated as 9/10 and described as "knife-like" with the onset of 2 days along with fever (102.6 F) and chills. Physical exam revealed +1 pitting edema of the bilateral lower extremities and erythema of the right pretibial area. Initial labs were notable for WBC 29, total bilirubin 2.9, and subtherapeutic INR. RLE ultrasound revealed acute occlusive deep vein thrombosis (DVT) of the bilateral posterior tibial veins. He was diagnosed with cellulitis & DVT. Initial therapy consisted of broad-spectrum antibiotics, heparin infusion, and consults to gastroenterology and infectious disease. Blood cultures initially grew Serratia marcescens, which the antibiotic regimen (including piperacillin/tazobactam) should have covered. Despite significant improvement in his leukocytosis and negative repeat blood cultures, the conjugated hyperbilirubinemia continued to worsen. Abdominal ultrasound, Magnetic resonance cholangiopancreatography, and hepatitis panel were unremarkable. However, hepatobiliary scintigraphy was concerning for acute cholecystitis. General surgery was consulted but deduced that suspicion for acute cholecystitis was low considering the lack of abdominal complaints. After multiple days in the hospital, a large fluid-filled blister developed on the dorsum of the patient's foot and unroofed. Podiatry was consulted on day 17 of admission, evaluated the lesion, and debrided the new wound. The day after debridement, the patient's hyperbilirubinemia started to decrease and trended downward for the rest of the hospitalization.

As previously stated, the differential for conjugated hyperbilirubinemia is broad and includes sepsis. In this case, the signs of sepsis were resolving (improvement in leukocytosis, negative repeat blood cultures, improvement in the area of initial concern of cellulitis, etc.) but it was not until a dorsal foot blister appeared and was then debrided that there was an improvement in the hyperbilirubinemia. This suggests that inflammation and cytokine release may be involved in hyperbilirubinemia beyond sepsis bacteremia.

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Encephalopathy at the Right Hand of Lactate

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D-lactic acidosis is a well-known cause of anion gap metabolic acidosis in pediatrics with short bowel syndrome due to necrotizing enterocolitis or Crohn's disease. Recognition of this condition in adults with short bowel syndrome and unexplained anion gap metabolic acidosis is crucial to avoid morbidity.

A 30-year-old male presented with abdominal pain associated with weight loss of 14 lbs over one week. Eight years previously, he had suffered a gunshot wound to the abdomen requiring partial small bowel resection complicated by short bowel syndrome, malnutrition, and intermittent obstruction. On presentation, he was confused with slurred speech and appeared cachectic with abdominal distension. Vital signs were stable. Labs were remarkable for severely low vitamin levels and pre-existing pancytopenia, and serum bicarbonate was 28 (range 22-30 mmol/L). An abdominal X-ray was negative for obstruction. A nasogastric tube was placed and his mental status improved with nutritional support. Rifaximin was started for possible small intestinal bacterial overgrowth. On day 12, he had worsening abdominal cramping and again became confused with slurred speech. Serum bicarbonate was now 10, serum anion gap elevated to 20, and chloride was 118, with an elevated urine anion gap. Blood gas showed a pH of 7.28 with PaCO2 of 40.5. Serum L-lactate was not elevated. Sodium bicarbonate (1300 MG TID) and oral vancomycin (125 MG QID) were started with concerns for D-lactic acidosis. His D-lactate level returned at 3.13mmol/L (upper limit 0.25mmol/L). Two days later the patient's anion gap had closed and his chemistries normalized.

This case illustrates D-lactic acidosis as a cause of anion gap metabolic acidosis in short bowel syndrome from trauma, a syndrome classically associated with children and young adults having undergone small bowel resection for necrotizing enterocolitis or Crohn's disease. Lactate is synthesized from pyruvate into L- and D-lactate by L- and D- lactate dehydrogenase (LDH), respectively. Humans normally form L-lactate, but in patients with short bowel syndrome and no short bowel metabolism, carbohydrates are metabolized to D-lactate in the large intestine by bacterial flora, which produces D-LDH. Due to low levels of the metabolizing enzyme D2-HDH, D-lactate accumulates, resulting in an anion gap metabolic acidosis with an elevated urine anion gap. A plasma D-lactate level >3mmol/L is used to diagnose D-lactic acidosis. Symptoms include encephalopathy, slurred speech, and often Kussmaul breathing, although some patients present solely with gastrointestinal discomfort. The predominance of neurological symptoms is likely a result of D-lactate crossing the blood-brain barrier and lack of production of D2-HDH in the brain. Treatment options include low-carbohydrate diet, bicarbonate replacement, and use of poorly absorbable antibiotics. Due to increased prevalence of children surviving to adulthood with short bowel syndrome, as well as adults acquiring it after surgery, increased recognition of D-lactic acidosis is essential.

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To Blame or Not to Blame: A Diagnostic Dilemma on Immune Check-point Inhibitor Therapy

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As checkpoint inhibitors become prevalent in oncologic management, immunotherapy-related adverse effects (IRAEs) are increasingly seen by general and field-specific practitioners. As IRAEs have potentially lethal consequences, treating physicians should be familiar with IRAEs and their diagnosis/management.

A 55-year-old male with metastatic melanoma who received first cycle of nivolumab/ipilimumab five days prior presented to the ED with dyspnea and productive cough progressing over the past week. At presentation, he was febrile (38.3C), tachycardic, tachypneic, and saturating 86% on room air. CT demonstrated diffuse mosaic attenuation throughout lungs and stable innumerable pulmonary metastases. Patient was started on broad spectrum antibiotics as empiric treatment for pyogenic pneumonia in an immune-compromised host. On day four of hospitalization, respiratory status worsened. ABG revealed PaO2 47. CT demonstrated extensive ground-glass opacities at lung bases with subpleural sparing, suggestive of subacute organizing pneumonia. Differential was broadened to include immunotherapy-related pneumonitis and pneumocystis pneumonia (PJP) as patient had a two-month history of steroid use without PJP prophylaxis. IV trimethoprim/sulfamethoxazole (TMP-SMX) and high dose steroids were initiated to empirically treat for PJP and pneumonitis. Serum (1→3)-β-D-Glucan was elevated >500 pg/mL (ref: <80pg/mL). Bronchoalveolar lavage was performed from which PJP PCR was detected, but below level of quantification. As a definite diagnosis for PJP could not be established, patient was to be treated with a 21-day course of TMP-SMX and prolonged steroid taper to treat for both PJP and pneumonitis. Respiratory status improved with treatment. Immunotherapy was discontinued and patient was switched to BRAF-targeted therapy.
Hospital course was also complicated by melena. Patient had melena for one month prior, with known metastasis to the transverse colon. During admission, bowel movements became more frequent and of liquid consistency. Stool studies were negative. There was concern for immune-therapy related colitis; however, diarrhea did not improve despite high dose steroids. Further review of prior CT revealed a suspected colo-jejunal fistula that was initially difficult to ascertain due to extensive metastases. As patient was receiving higher amounts of tube feed through PEG tube (initially placed due to dysphagia two months prior) than patient was taking at home, the feed was traversing the fistula leading to severe diarrhea. Patient had a repeat MBS and was switched to a high-fiber dysphagia-advanced diet. Patient's diarrhea notably improved after dietary modification.

This case illustrates importance of exclusion of other etiologies when suspecting IRAEs. Immune-therapy related pneumonitis has median onset time of 2.7 months, but literature review revealed it can happen within days and is more common with dual-inhibitor use, which is what prompted collateral treatment for pneumonitis. Similarly colitis occurs 5-10 weeks after 1-2 doses of immunotherapy, but rarely occurs sooner. IRAEs are difficult diagnosis to make and should remain on initial differential diagnoses to avoid delayed treatment.

Invasive Candida glabarta in a patient with a new diagnosis of diabetes presenting as a retroperitoneal mass mimicking cancer

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Introduction: Primary retroperitoneal masses are often asymptomatic and are most commonly malignant in nature. We present a rare case of a retroperitoneal mass caused by invasive Candida glabarta infection in a patient with newly-diagnosed diabetes.

Case presentation: A 54 year-old woman presented with a twenty pound weight loss, progressive lower back pain, progressive right lower extremity weakness, and polyuria. She originally presented to an urgent care center and was diagnosed with new type 2 diabetes mellitus with a blood glucose level of 500. She saw her primary care physician the next day, where she was also diagnosed with a urinary tract infection, and blood work showed a hemoglobin A1C of 17%. Outpatient management of her diabetes was originally planned. However, her symptoms worsened in the interim, and she developed confusion and slurred speech. She was admitted to the hospital for diabetic ketoacidosis and suspected sepsis related to her UTI. Urine and blood cultures were obtained, ceftriaxone was started, and she underwent a CT scan to evaluate her back pain. This revealed a 19cm left retroperitoneal mass that was highly suspicious for a mucinous tumor. CT chest was also obtained and was unremarkable. The leading differential diagnosis for the mass was malignancy, with dedifferentiated liposarcoma suspected. She underwent a CT-guided biopsy that surprisingly yielded purulent fluid with minimal solid material. Fluid culture grew Candida glabarta, and her initial blood cultures were also positive for C. glabarta. Urine cultures were negative. The patient was treated with caspofungin and was transitioned to oral fluconazole by the time of discharge. Plans were made for her to have a retroperitoneal drain inserted prior to discharge, but repeat imaging revealed almost complete resolution of the mass after starting antifungal therapy.

Discussion: Primary retroperitoneal masses are most commonly malignant tumors and are often identified at a more advanced stage due to the lack of symptoms associated with masses in that region. Other differential diagnoses for retroperitoneal lesions include fibrosis, infection or abscess, extramedullary hematopoiesis, or Erdheim-Chester disease. Fungal retroperitoneal infections, as in this patient, are incredibly rare when there are no preceding invasive measures or other obvious underlying structural abnormalities to act as a nidus for infection. In fact, there is only one case of a C. glabarta retroperitoneal mass reported in the literature, and in that case, the patient had overlying skin changes concerning for cellulitis. In this case, the patient's uncontrolled diabetes was likely the risk factor that led to an immunocompromised state, making an invasive fungal infection possible.

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Significant Hemopericardium with Subsequent Cardiac Tamponade Secondary to Rivaroxaban

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Introduction: The use and utility of novel oral anticoagulants (NOAC) has been increasing in clinical practice due to their relatively lower incidence of side effects such as intracranial hemorrhage, particularly in the elderly, when compared to Vitamin K antagonists. Despite an additional risk of gastrointestinal bleeding, there is an overall lower risk of thromboembolic events. In addition, a predictable clinical profile along with a lack of need for periodic monitoring makes them an ideal option for patients with baseline debility or dementia. Rivaroxaban is a factor Xa and prothrombinase inhibitor indicated for stroke and venous thromboembolism prophylaxis in non-valvular atrial fibrillation as well as treatment of venous thromboembolism based on the ROCKET AF, EINSTEIN DVT, and EINSTEIN PE studies. Hemopericardium is not listed as a known side effect, but previous cases of hemopericardium secondary to rivaroxaban have been described in the literature. Here we present a case of hemopericardium presumably secondary to Rivaroxaban use.

Case presentation: An 84 year-old male with a past medical history of paroxysmal atrial fibrillation on rivaroxaban, chronic anemia and leukopenia, coronary artery disease presented with general malaise, lightheadedness and dizziness. The patient was previously worked up for chronic anemia via colonoscopy which was unremarkable. Laboratory findings were significant for hepatic and renal dysfunction. Chest radiography showed worsening cardiomegaly. Due to hemodynamic compromise and impending respiratory failure, he was transferred to the intensive care unit where he was found to have a significant pericardial effusion with tamponade physiology. Following urgent reversal of his INR with prothrombin concentrate, the patient successfully underwent pericardiocentesis which yielded 1.5 liters of grossly hemorrhagic fluid. He was noted to have new onset LV dysfunction and severe mitral regurgitation following his procedure. Workup for malignancy was negative.

Conclusions: Despite an overall favorable pharmacokinetic and side effect profile compared to previous agents, Rivaroxaban can have potentially significant hemorrhagic complications necessitating urgent reversal and intervention. A wider differential with higher clinical suspicion for less likely sequelae such as hemopericardium should be considered in patients who are taking novel anticoagulants and present with nonspecific symptoms, due to the possibility of rapid decompensation. Other novel anticoagulation medications that have been associated with hemopericardium include Apixaban and Dabigatran. Typically, grossly hemorrhagic pericardial fluid is suggestive of underlying malignancy and, given our patient's history of chronic anemia, a thorough workup was conducted which ruled out this possibility. The worsening cardiomegaly on chest radiography and history of chronic anemia also suggested a subacute to chronic process. Typically, anti-Factor Xa chromogenic assays are used to measure the drug concentration of Rivaroxaban but these levels reportedly do not have any direct correlation with anticoagulant activity further emphasizing the need for careful assessment of concurrent medications and supplements for potential interactions.

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Cannon Ball appearance that disappeared? a novel case of COVID-19 presentation with cannon ball appearance on imaging

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Introduction: COVID-19 diagnosis requires high suspicion with clinical presentation & imaging, where imaging plays key role in early decision-making before serology returns. The characteristic findings on CT include ground glass opacities, consolidation, crazy-paving pattern, and air bronchogram sign. We present a case of COVID-19 which misleadingly presented with cannon-ball appearance on imaging, causing a management dilemma.

Case presentation: 35-year-old with past medical history of type 1 Diabetes mellitus, Factor V Leiden deficiency, history of DVT (on warfarin) and recently treated osteomyelitis 3 months prior. He presented with altered mental status and respiratory distress. Denied fever, chills, cough, sore throat, recent travel or sick contacts. Non-alcoholic and non-smoker. He was treated 2 months prior for sepsis secondary to osteomyelitis of the foot. He did not have any respiratory problems and his chest xray was clear.

On presentation patient was obese, afebrile, tachycardic, hypotensive 80/50mmHg, tachypneic, saturating 89% on room air. Chest exam revealed rhonchi and decreased breath sounds bilaterally. Labs showed leukocytosis 15,000, creatinine of 1.45, lactate 4.0. Troponin & BNP were negative while EKG showed sinus tachycardia. Oxygen saturation did not improve with oxygen. ABG showed worsening acidosis, PaO2 70, PaCO2 50. He developed grunting respirations and was intubated for airway protection and respiratory distress.

Chest xray showed cannon-ball appearance in all lung fields bilaterally which was new compared to xray from prior admission. CT chest revealed prominent diffuse bilateral and rounded soft tissue densities, displayed throughout all lobes, but most prominent in the right middle lobe and upper lobes. Patient was transferred to ICU. Procalcitonin was elevated, respiratory pathogen panel, streptococcus and legionella urine antigen were negative. However, patient tested positive for COVID-19.

Pulmonary and Infectious disease services were consulted. Echo done for suspected septic emboli but was normal. TB, fungal cultures, autoimmune panel was also negative. ARDS was suspected secondary to COVID-19. He received Hydroxychloroquine for 5 days however little improvement was seen. Supportive therapy was continued with high PEEP 12 and FiO2 100%. Subsequent chest xrays showed gradual improvement with clearing of cannon ball opacities. Patient was extubated and follow-up chest xray showed near complete resolution. The patient continues to be in the hospital, recovering slowly but doing well.

Discussion: Cannon-ball appearance in radiology describes the presentation of multiple round, nodular opacities with well-defined borders, usually bilaterally. The most common cause of cannon-ball appearance is metastatic malignancy; however, other infectious diseases such as pulmonary tuberculosis, histoplasmosis, cryptococcosis, and nocardiosis can also present with granulomas that match this finding. Our case is the first to be reported with this finding and adds to the literature to identify this as a potential presentation to be cognizant of for COVID-19.

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Sarcoidosis Imitating a Genetic Cardiomyopathy

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Non-ischemic dilated cardiomyopathy can be a diagnostic challenge. This case highlights the importance of the patient history in the evaluation of new onset heart failure.

A 48-year-old male with a history of atrial fibrillation status post ablation presented to the Emergency Department with lightheadedness and palpitations for two hours. He had not had palpitations since his ablation, although he did endorse several presyncopal episodes. At the time of presentation, he denied anginal symptoms, drug use, or viral illnesses. Upon further discussion, he reported a significant family history of cardiac disease. Specifically, his maternal grandmother died suddenly in her 30-40s of a "massive heart attack", his mother had a pacemaker and died in her 40s, and his brother died of a myocardial infarction at the age of 56. In the emergency department he was found to be in hemodynamically stable ventricular tachycardia. He spontaneously converted to sinus rhythm and was admitted for further workup.

His physical exam and laboratory work were unremarkable. His EKG showed normal sinus rhythm. On Echocardiogram, he was found to have a dilated left ventricle with an ejection fraction of 35%. Coronary angiography was obtained to assess for ischemic disease in the setting of a new cardiomyopathy and was unremarkable. His cardiac MRI showed late gadolinium enhancement in the septum and base. He then underwent a cardiac PET scan which revealed FDG uptake in a patchy distribution concerning for sarcoidosis.

Following the MRI and PET findings, he was started on immunosuppression with medical therapy for heart failure, and an ICD was placed for secondary prevention. Over the next year, his systolic function worsened, and he had additional episodes of VT. Given his lack of extra-cardiac involvement, as well as his overall worsening cardiac function, the presumed diagnosis of cardiac sarcoidosis was reconsidered. An endomyocardial biopsy was done and was negative for sarcoidosis. This, in combination with his significant family history of premature death, was suggestive of a genetic process. A genetic workup ultimately found a disease causing mutation in the LMNA gene, the second most common cause of familial cardiomyopathy. LMNA cardiomyopathies are inherited in an autosomal dominant fashion and are characterized by life threatening arrhythmias and progressive heart failure.

This case demonstrates the importance of the patient presentation and background history. The diagnosis of a genetic cardiomyopathy was important for the patient and can have profound implications for his family. The imaging findings showing marked inflammation are not usually seen in genetic cardiomyopathies. Because of this, he has been continued on immunosuppression in addition to heart failure therapy, and his cardiac function is stable at the current time.

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CNS Tuberculosis in the 21st Century at a Large Community Teaching Hospital: A 4-Day Journey to Rapid Diagnosis and Effective Th

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Introduction: Central nervous system (CNS) tuberculosis is a devastating form of extrapulmonary tuberculosis with mortality rates between 20-69%. Manifestations may include those of meningitis, tuberculomas, brain abscess, or spinal cord infection. The clinical presentation is often subtle with few distinguishing features and an insidious course of progression. Cerebrospinal fluid (CSF) acid-fast bacilli (AFB) smear is not sensitive, and culture results are positive in only 38-88% of patients. Therefore, a high index of suspicion should guide diagnostic testing in order to avoid delay in diagnosis and therapy.

Case Presentation: A 29-year old woman, an Indian immigrant to USA 4 years ago, presented to hospital with a 5-month history of worsening global headache only recently associated with nausea, vomiting, weakness and 20-lb unintentional weight loss. Computed tomography (CT) of head showed six ring-enhancing lesions throughout both cerebral hemispheres and the cerebellum. Chest CT showed a new left upper lobe mass-like lesion and some nodularity right upper lobe. Lab data included negative HIV serology and positive interferon-gamma release assay (IGRA) for M. tuberculosis. AFB stain and Mycobacterial nucleic acid amplification testing (NAAT) of expectorated sputum were negative. Bronchoscopy was performed and BioFire®Film Array® pneumonia panel and AFB smears of 3 BAL samples were negative. 2 of 3 Mycobacterial NAATs performed on BAL samples were positive. Lumbar puncture revealed clear fluid with 23 nucleated WBCs (81% lymphocytes, 5% monocytes, 14% PMN), 200 RBCs, glucose of 35 mg/dL (serum = 125 mg/dL) and CSF mycobacterial NAAT was positive. Treatment was begun on hospital day 4 with isoniazid, rifampin, pyrazinamide and ethambutol. Repeat head imaging two weeks later showed dramatic decrease in size of all 6 tuberculomas. Patient improved significantly and was discharged three weeks after admission.

Discussion: A large population-based analysis in the United States from 2010-2017, showed that > 50% of patients with CNS tuberculosis die or develop neurological sequelae despite appropriate therapy. Symptoms are subtle and non-specific and AFB staining of CSF is not sensitive. As in this case, mycobacterial NAAT should be performed when the clinical suspicion is high. Establishing an early diagnosis of CNS TB and initiating early treatment have long-term positive implications for ultimate outcome.

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Late Presentation of a Rare Congenital Cardiopulmonary Defect: Scimitar Syndrome

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Introduction: Scimitar syndrome a rare congenital disease, is characterized by abnormal drainage of the right pulmonary vein into the inferior vena cava or right atrium, instead of the left atrium. Commonly associated with hypoplasia of the right lung and right pulmonary artery, as well as dextroposition of the heart, the syndrome is derived from radiographic manifestation of the anomalous vein resembling a Turkish sword.

Case presentation: A 45-year-old woman with history of obstructive sleep apnea, hypertension, morbid obesity and recurrent mild respiratory infections presented with cough and dyspnea on exertion for four months. Over that period, she was repeatedly treated by her PCP with several courses of antibiotics and steroids. With a 40-pound weight gain and progression of dyspnea at rest, she was referred to pulmonology. Chest CT reported hypoplastic right lung with a prominent right pulmonary vein draining into the junction of the inferior vena cava and right atrium, compatible with Scimitar Syndrome. Full PFT showed moderate restrictive pattern with air trapping and decreased diffusion capacity. Echocardiogram showed evidence of mild pulmonary hypertension. She underwent surgical anastomosis of the Scimitar vein via the present atrial septal defect into the left atrium, with patch enlargement of the ostium of the scimitar vein at its entrance and tricuspid valve repair commissuroplasty. Postoperatively, dyspnea improved significantly. Supplemental oxygen was weaned to 2 liters of oxygen via nasal cannula.

Discussion: Scimitar syndrome accounts for 0.5-1 percent of all congenital heart disease cases. Prevalence of disease is very low and estimated to be 1-3 out of every 100,00 live births. Two types of syndrome have been described: infantile and adult forms. The infantile form is associated with other thoracic and vascular abnormalities. Adult variants have a smaller shunt and are more commonly isolated. Atrial septal defect is the most common associated cardiac anomaly in both adult and infantile forms. Echocardiogram with color doppler studies and CT are the mainstay diagnostic tools. Electrocardiogram may be identical to ASD. Most patients are asymptomatic at birth. Clinical symptoms are similar to ASD. Exercise intolerance is the most common complaint. Depending on the shunt volume, patients may be asymptomatic or exhibit dyspnea and recurrent respiratory infections. Treatment is mostly surgical, diverting the anomalous connection between pulmonary and systemic veins or draining the flow to the left atrium. No mortality difference has been reported between different surgical techniques.

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Kaposi's Sarcoma Inflammatory Cytokine Syndrome (KICS) Presenting with Diffuse Alveolar Hemorrhage

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Kaposi's Sarcoma Inflammatory Cytokine Syndrome is a condition in patients with HIV and Human Herpesvirus-8 infection (HHV-8) that presents as a sepsis mimic with respiratory failure. It does not respond to antibiotic therapy. Cytokines, specifically interleukin (IL)-6 and IL-10 are elevated. This is a rare condition in the era of anti-retroviral therapy with poorly understood pathophysiology. KICS can be treated with chemotherapeutic agents and supportive care, but misdiagnosis and delayed treatment can lead to significant mortality.

Our patient was a 41-year-old male with a history of HIV/AIDS and Kaposi's Sarcoma (KS) who presented with four days of night sweats, nausea and fatigue. The patient endorsed intermittent compliance with anti-retroviral therapy. On exam, the patient had worsening of his existing lymphadenopathy. His CD4 count was 7/cmm. He had new thrombocytopenia to 12,000 and was admitted for a bone marrow biopsy for further investigation. This showed a hypercelluar marrow without KS, evidence of Castelman's Disease, or fungal/mycobacterial disease. Chest x-ray on the day of admission showed mild bibasilar opacities.

One day after admission, the patient developed hypoxia and chest discomfort. EKG and troponin were unremarkable. Chest x-ray showed dramatic worsening of the bilateral opacities and he was treated with broad-spectrum antibiotics. The patient's platelet count had dropped to 5,000 and he received multiple platelet transfusions. He developed a new anemia. On the third day of admission, his respiratory status worsened, requiring endotracheal intubation, and he developed hemoptysis and petechiae. Hemolysis and diffuse intravascular coagulation (DIC) labs were unremarkable. Bacterial, fungal, and acid-fast cultures returned negative. HHV-8 PCR level was greater than 700,000 copies/ml blood.

Bronchoscopy was pursued given lack of respiratory status improvement and demonstrated Diffuse Alveolar Hemorrhage (DAH). He became febrile and hypotensive requiring vasopressor support. Ganciclovir, IVIG, and steroids were administered without initial improvement. He was then treated with Doxorubicin (20mg/m2) and Rituximab (375mg/m2), and was able to wean off vasopressors within 48 hours. His thrombocytopenia improved. Shortly thereafter, he was extubated and ultimately discharged without a resting oxygen requirement.

The patient's cytokine panel showed dramatic elevations in (IL)-6 to 1200 pg/ml (normal <2) and IL-10 to 1800 pg/ml (normal <2.8). He also had elevations of several other cytokine levels including IL-2r, IL-8, and IL-13. He improved clinically with administration of anti-neoplastics, making KICS the most likely diagnosis. The combination of cytokine storm and profound thrombocytopenia in this patient likely led to DAH. It is important to recognize that a patient with KS presenting with signs of systemic inflammation, cytopenias, elevated HHV-8 level, and respiratory distress may have KICS rather than sepsis. Prompt recognition of this rare diagnosis can lead to survival, such as in this case, but lack of treatment has led to mortality in other cases.

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Undetected Splenic Artery Pseudoaneurysm on CT Angiogram: A Rare Cause of Gastrointestinal Bleeding

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Introduction: Splenic artery pseudoaneurysms (SAPs) are rare compared to true aneurysms and are much more likely to rupture. The mortality rate could reach 90% if ruptured cases left untreated. SAPs usually arise in patients with a history of pancreatitis, gastric ulcers, or trauma. SAP can present with hematochezia, hematemesis, or intraperitoneal bleeding. SAPs can sometimes be missed on Esophagogastroduodenoscopy (EGD) or colonoscopy, even during active bleeding. Thus, CT angiography has become the key to diagnose SAPs as it is less invasive and can detect most nonbleeding lesions. However, as our case illustrates, CT angiography may still fail to detect SAPs, and a definitive diagnosis requires mesenteric angiography. We report a rare case of undetected SAP on CT angiography, which presented with gastrointestinal bleeding.

Case Presentation: A 59-year-old female with a history of pancreatic pseudocyst status post drainage five years ago presented with bloody bowel movements and signs of hemodynamic instability. Initial investigations revealed hemoglobin of 9.6, and, subsequently she required multiple blood transfusions. EGD, colonoscopy, and Nuclear Medicine bleeding scan all showed no source. Interestingly, even CT angiography was negative. On the tenth day, she had episodes of hematemesis and hematochezia with an acute drop of hemoglobin to 7.1 g/dL, and became hypotensive. She was taken to ICU, and emergent EGD was performed, which showed active bleeding into the gastric lumen. A mesenteric angiography was subsequently done and revealed a proximal splenic pseudoaneurysm. The intraoperative embolization coiling of the pseudoaneurysm was performed successfully. The post-procedural course was uneventful.

Conclusion: SAPs are challenging to diagnose with endoscopy; thus, CT angiography has become the primary mode of diagnosis. However, as in our case, CT angiography can sometimes fail to detect SAPs. The suspicion for SAPs should be high in patients with a history of pancreatitis who present with upper gastrointestinal bleeding. Therefore, in patients with a history of pancreatitis and abdominal interventions, a mesenteric angiogram should be considered as an earlier option if the CT angiography fails to detect a source of bleeding.

Keywords: Splenic artery pseudoaneurysm, CT angiogram, Gastrointestinal bleeding

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An Unusual Case of Recurrent Angioedema

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Angioedema is characterized by episodic submucosal and subcutaneous tissue swelling and is classified as histamine or bradykinin-mediated. Bradykinin-mediated angioedema can be hereditary or acquired angioedema (AAE); the latter is often drug-induced, commonly from ACE Inhibitors and gliptins. Rarely, AAE occurs secondary to C1 inhibitor deficiency from other causes, including lymphoproliferative disease, infection and autoimmune disorders. Evaluation for rare causes of AAE is necessary to ensure correct therapy and best outcomes.

Case: A 74-year-old female with a history of hyperlipidemia, HTN and osteoarthritis presented with angioedema which began during an outpatient dental procedure. This was her fourth episode of angioedema within the last several years, all of which occurred after age 50. All common culprit medications had previously been discontinued; she endorsed no family history of angioedema. She denied histaminergic symptoms of urticaria and/or bronchospasm; thus, a non-medication related, bradykinin-mediated cause of angioedema was suspected. Testing revealed a low C1 esterase inhibitor quantification and low C1 esterase inhibitor antigen, which supported AAE secondary to acquired C1 esterase deficiency. She was referred for outside evaluation. Additional workup included serum protein electrophoresis which showed atypical restricted bands. A bone marrow biopsy was consistent with marginal zone lymphoma. She was started on an as-needed bradykinin receptor antagonist, icatibant, for acute episodes. Treatment of her underlying lymphoproliferative disorder with rituximab was planned.

Discussion: AAE due to acquired C1 inhibitor deficiency is rare; precise incidence is unknown, but it is estimated to be between 1:100,000 and 1:500,000. AAE is not associated with urticaria, but is characterized by episodic edema of the face, lips, tongue, extremities, and genitals, abdominal pain from gastrointestinal mucosal swelling, and possible life-threatening edema of the upper airways. Patients often present in or after the fourth decade of life. Disease is classified into AAE types I and II: type I is often associated with underlying disease, particularly lymphoproliferative disorders. With B-cell proliferative disorders, anti-idiotypic antibodies attach to surface immunoglobulin of the B-cells resulting in C1 inhibitor deficiency. The bradykinin pathway is mediated by C1 esterase inhibitor; deficiency leads to overproduction of bradykinin and subsequent angioedema. Evaluation for AAE includes measurement of C4 levels which are usually low during and between episodes, and measurement of qualitative and functional C1 inhibitor levels. C1q levels assist in distinguishing hereditary and AAE. Treatment necessitates evaluation and treatment of underlying associated disease states. Case reports describe successful use of rituximab for AAE in patients with B-cell lymphoproliferative disorders. Acute attacks can be treated with C1-inhibitor concentrates, fresh frozen plasma, or bradykinin receptor antagonists, as used for our patient.

Teaching Point:
Evaluation for cause of AAE is necessary to ensure proper treatment of possible underlying disease process, and prevention of further life-threatening episodes of angioedema.

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Rapidly Progressive Pulmonary Fibrosis Heralded by Early Hair Graying

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Short telomere syndrome (STS) is a rare hereditary disorder with diverse clinical presentations, making diagnosis difficult. This accelerated aging disorder affects organs with high cell turnover like the lungs, bone marrow, liver, and skin. Idiopathic pulmonary fibrosis (IPF) is the most common manifestation of STS.

A 40 year old man with emphysema, COPD, and pulmonary fibrosis presented to an outside hospital for shortness of breath. He recently required home oxygen (2L) after an episode of pneumonia 3 months prior. He had a 20 pack-year smoking history, but quit several years ago and had started vaping daily. Family history was significant for pulmonary fibrosis in his parent. His exacerbation was initially thought to be a vaping associated lung injury. He was intubated, treated for acute respiratory distress syndrome, improved, and extubated. However, due to concern for aspiration, he required reintubation. Bronchoalveolar lavage grew Staphylococcus aureus. Despite appropriate medications including epoprostenol, high dose steroids, ceftriaxone, and sedatives and analgesics for ventilator dyssynchrony, he developed worsening hypoxia. Repeat CT chest showed worsening basilar pulmonary fibrosis. Extensive connective tissue disease, autoimmune, and infectious workups were unrevealing. He was transferred to our MICU for further evaluation and management. He remained intubated for over 20 days without much improvement despite aggressive medical management. Further history revealed early graying of his hair and beard. Additional workup was significant for lymphocyte and granulocyte telomere length less than 1st percentile for age. At this point, he was not a lung transplant candidate. The remainder of his hospital course was complicated by a pulmonary embolism, worsening hemodynamic status, and continued respiratory decline. After family discussion, a decision was made to pursue comfort care and he was palliatively extubated.

Though rare, this case demonstrates why a younger patient with pulmonary fibrosis, positive family history, and/or premature hair graying should prompt evaluation for STS. IPF is a devastating progressive lung disease that often has no identifiable cause. IPF usually affects male smokers over age 60 years. It is rarely seen in patients under 50 except in those with familial pulmonary fibrosis or telomeropathies. Younger adults usually have other forms of interstitial lung diseases related to connective tissue diseases. While STS has many clinical presentations, IPF is the most common manifestation. Extra-pulmonary abnormalities including early hair graying, cytopenias, and liver cirrhosis also clue to STS. Telomere-mediated pulmonary fibrosis is accelerated in smokers and often presents with overlapping emphysema like in our patient. Telomere shortening predicts poor prognosis and worse post-transplant survival. Still, early referral for lung transplant remains the mainstay treatment. Use of antifibrotic medications has also been described in the literature. Pulmonary STS has an autosomal dominant inheritance, so this patient's family should receive genetic counseling.

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Acute Pancreatitis Induced by Itraconazole

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The annual incidence of acute pancreatitis in the United States ranges from 4.9-35 per 100,000 persons, with gallstone disease and alcohol being the leading etiologies. However, in 25-30% of patients, the etiology is inconclusive following initial testing. In recurrent disease, additional testing with more invasive procedures including MRCP, ERCP, and sphincterotomy fails to find a precipitating cause in 15-20% of cases. Drug-induced pancreatitis is rare, constituting 5% of all cases, but proton pump inhibitors, sulfonamides, and didanosine are known causal agents. However, there is a scarcity of literature discussing the association of acute pancreatitis and azole-ring antifungal administration.

A 73 year old male with a past medical history of recurrent Clostridium difficile (C.diff), Cytomegalovirus (CMV) viremia, and disseminated Histoplasmosis presented with altered mental status. He previously completed a course of amphotericin and was started on continuous itraconazole with a planned course of 12 months. Prior to and after initiation of itraconazole, the patient had experienced several bouts of altered mental status and sepsis, including this admission for sepsis secondary to left lower extremity cellulitis resulting in altered mentation. He was started on cefazolin and continued on previously prescribed metronidazole, valacyclovir and itraconazole for recurrent C. difficile, CMV viremia and disseminated histoplasmosis, respectively. The patient’s mental status continued to improve until day eight of admission when he started having waxing and waning altered mental status. He complained of severe epigastric abdominal pain. Patient had no history of alcohol abuse, smoking abuse or drug addiction. Physical examination demonstrated epigastric tenderness, abdominal guarding and tense abdomen. Laboratory evaluation was significant for mildly elevated serum lipase (105 U/L) and triglycerides (172 mg/dL). Liver function tests were normal. Infectious etiologies were entertained, considering the patient’s medical history, but CMV PCR was < 200 units. CT abdomen and pelvis was consistent with acute pancreatitis. Right upper quadrant ultrasound did not show gallstones and esophagogastroduodenoscopy (EGD) was non-concerning. With common etiologies (gallstones, alcohol, and elevated triglycerides) effectively ruled-out, attention was turned to iatrogenic causes and the patient's medications were reviewed. Itraconazole was stopped and the patient’s symptoms slowly improved.

The mechanism of drug-induced pancreatitis is unknown but proposed mechanisms include immunologic reactions, direct toxic effect, accumulation of a toxic metabolite, ischemia, intravascular thrombosis and increased viscosity of pancreatic fluids. Known side effects of azole-antifungals, including hypertriglyceridemia and hypercalcemia, may also play a role. This case of itraconazole-induced pancreatitis offers perspective on a rare case of iatrogenic disease and emphasizes the importance of maintaining a wide differential in patients with pancreatitis

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An Atypical Presentation of Chronic Disseminated Intravascular Coagulation

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Disseminated Intravascular Coagulation (DIC) carries a mortality of 20-50% due to the positive feedback loop of hypercoagulation, factor consumption, hemorrhage and multiorgan failure. DIC can present as either an acute or chronic form, triggered by a variety of insults including sepsis, malignancy and trauma. Discerning the acute and chronic presentations of DIC can be diagnostically challenging early in the disease process. We present the case of an 80 year-old African American male admitted for community acquired pneumonia and jaundice. Past medical history was notable for a bladder mass discovered three weeks prior that was pending biopsy. Admission labs were significant for total bilirubin 7.4, Hgb 12.1, platelets 57; all of which had been unremarkable three weeks prior. Further workup revealed D-dimer >20, fibrinogen <60 and INR 2.6. Factor VIII was normal suggesting DIC and not acute hepatic failure. Imaging, including right upper quadrant ultrasound, a CT abdomen/pelvis, and MRCP were all negative except for the known bladder mass. HIV, viral hepatitis panel, ADAMTS 13, and UDS were also negative. The patient received antibiotics and blood products resulting in improvement in his clinical status. He was subsequently discharged home per the patient's wishes and consultant recommendations. Six hours later, he returned to the ED due to multiple falls. He had a core body temperature of 90.1°F and bilateral pedal edema. Venous doppler ultrasound revealed bilateral lower extremity DVTs. Given his persistent DIC, an IVC filter was placed. The patient then developed hemorrhagic shock from an upper GI bleed. EGD revealed an ulcerative gastric mass that was positive for H.pylori and CMV, but negative for metaplasia. Goals of care were discussed, the patient was referred to hospice and passed away shortly thereafter. Our case is unique in that it presents the complete disease course of DIC but the inciting trigger remains a mystery. Acute DIC secondary to pneumonia was originally considered. However, our patient lacked the hemolytic anemia typical for acute DIC and the pneumonia was relatively mild, lacking any criteria to qualify as sepsis. Moreover, DIC persisted despite adequate CAP treatment. Chronic DIC secondary to suspected bladder malignancy was possible, however, the severity of the patient’s presentation was atypical for the compensated state classically seen in chronic DIC. Ultimately, the case was hypothesized to be an atypical presentation of chronic DIC, illustrating the challenges of differentiating DIC and its causes even when early diagnosis is achieved.

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What's in a Rapidly Expanding Mass?

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A 56 year old female with a history of chronic systolic heart failure status post LVAD implantation and recent tricuspid valve repair complicated by candidal fungemia, presented to our hospital with the sudden appearance of multiple soft tissue masses. She denied abdominal pain, diarrhea or additional GI symptomatology. Over the course of two days, the patient developed progressive pain and swelling involving the left breast, left medial thigh, and left buttock. She denied trauma and review of systems was otherwise unremarkable. Physical examination revealed multifocal, nonfluctuant, mildly erythematous masses with moderate tenderness to palpation. Of note, the left breast lesion was markedly indurated. Admission labs demonstrated a mild leukocytosis with an INR of 2.9. Computed tomography of the chest, abdomen, and pelvis demonstrated soft tissue masses at the areas of concern without additional pathology. The operating diagnosis was multifocal hematomas given chronic warfarin use with concern for secondary abscesses given physical exam findings and leukocytosis. On day two, thick bloody fluid was aspirated from the left buttock and left breast lesions. Samples of the fluid were sent for culture and a drain was placed in the breast. At 48 hours, the fluid cultured from the left breast grew nonlactose fermenting Gram negative bacilli and the patient was started empirically on meropenem. The organism was identified as Shigella sonnei and antibiotic therapy was transitioned to levofloxacin. Blood and left gluteal cultures and a second breast culture showed no growth. The patient improved although was hospitalized for two weeks.

This case is interesting because extraintestinal Shigella sonnei infection is uncommon and focal soft tissue infection is rare. Extraintestinal Shigella species infection reported in the medical literature include meningoencephalitis, bacteremia, osteomyelitis, septic arthritis, splenic abscesses, vaginitis as well as endomyometritis. The most frequent extraintestinal manifestation is seizure, which is reported in 5 to 30% of children hospitalized with shigellosis. Additional common extraintestinal manifestations in Shigella species infection include alterations in consciousness, severe hyponatremia, leukemoid reaction, and hemolytic uremic syndrome (HUS). Both leukemoid reaction and HUS are thought to be related to Shiga toxin production. Focal soft tissue infection appears to be very rare outside of the genitourinary tract. In this case, either occult hematogenous dissemination or focal secondary infection of the breast hematoma remain possible. Contamination would be highly unlikely. This presentation further raises concern for asymptomatic intestinal shigellosis, as asymptomatic infection can occur, particularly in previously infected individuals.

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A particularly challenging diagnosis of HLH

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Introduction: Hemophagocytic lymphohistiocytosis (HLH), a rare life-threatening syndrome of excessive immune activation and tissue destruction, affects diverse populations with a varied presentation, often leading to delay in diagnosis. HLH triggers include infections, hematologic malignancies, immunocompromised states, and autoimmune disorders.

Diagnosis is confirmed by meeting 5 of 8 of the following criteria:
- Fever (≥38.5C)
- Splenomegaly
- Cytopenia (Hb<9, plt <100, ANC<1000)
- Hypertryglyceridemia >265 and/or hypofibrinogenemia <150
- Hemophagocytic lymphocytes in biopsy
- Low or absent NK cell activity
- Ferritin >500
- Elevated soluble CD 25 (or IL-2)

Case: 56-year-old woman presented with worsening confusion and shortness of breath for one week. Past medical history was significant for Burkitt lymphoma (chemotherapy treatment 2004), hyperlipidemia, and anxiety. She presented with hypotension, hypoxia, and intermittent fevers. CBC showed hemoglobin of 8g/dL with normal MCV and platelet count. Liver function tests showed alkaline phosphatase 268IU/L, ALT 62IU/L, and AST 92IU/L (previously normal). D-dimer was 970ng/ml (normal, <230). Abdomen/pelvis computed tomography demonstrated splenomegaly. Autoimmune work up and investigations for an infectious source (including blood/respiratory cultures, a respiratory viral panel, and studies for HIV, EBV, CMV, HSV, parvovirus, tuberculosis, and histoplasmosis) were negative. Other laboratory studies included fasting triglycerides 451 mg/dl (previously normal) and Ferritin >3000 ng/ml.

Hospital course was complicated by a new leukopenia, worsening thrombocytopenia/anemia requiring multiple blood transfusions. Hematology/oncology was consulted for worsening pancytopenia and concern for possible HLH. Peripheral smear showed toxic granulations in PMN and a leftward shift, while cerebrospinal fluid analysis was unremarkable. IL-2 receptor level was elevated at 83,379 (normal <1,891). Bone marrow biopsy showed hemophagocytosis and findings consistent with Myelodysplastic syndrome-Multilineage dysplasia (MDS-MLD).

Patient improved clinically and cytopenias slowly improved without specific therapy, leading to a conclusion that HLH was less likely. She was discharged, but returned 1 week later with similar symptoms; hematology/oncology was reconsulted for concerns of HLH. The consult service concurred that HLH was likely, and dexamethasone was initiated. Patient's mental status returned to baseline, and laboratory abnormalities improved.

She was discharged with close hematology/oncology follow up, with plans to start outpatient treatment for MDS/HLH. Unfortunately, patient returned a few days later with altered sensorium and worsening cytopenias. She rapidly developed cardiopulmonary arrest and expired.

Discussion: HLH is a rare syndrome with poor prognosis. Due to its varied presentation, nonspecific lab findings, and overlap in symptoms with other disease processes, diagnosis is often delayed. Thus, a high level of suspicion for HLH should be raised in patients presenting with fever, cytopenias, elevated liver markers, and elevated inflammatory markers.

Malignancy associated HLH is believed to have the poorest prognosis. MDS-HLH is rare and few case reports have been published. This case presentation was particularly challenging given the patient's waxing/waning course, which led to cognitive bias of premature closure.

Although the prognosis for HLH is poor, treatment options available. Treatment aims at suppressing immune response and treating underlying conditions.

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Rare zooinotic infection in patient with polysubstance abuse

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Case Report: A 44 year-old male with a history of IV drug use presented to our hospital with severe upper abdominal pain, arthralgias, myalgias, and night sweats of 3 days duration. His physical exam was significant for hepatosplenomegaly that was also demonstrated by subsequent CT of the abdomen and nuclear imaging of the liver and spleen. On imaging, his hepatosplenomegaly was consistent with either an infectious or inflammatory process. His spleen was measured at 21 cm in length, almost double of what we would expect in the average adult. His initial labs suggested a mixed warm and cold autoimmune hemolytic anemia with a hemoglobin of 6.2 g/dL, haptoglobin < 8 mg/dL, and lactate dehydrogenase of 421 U/L. His direct antiglobulin test (DAT) was positive for anti-complement 1 and IgG. Further workup was negative for Hepatitis B, HIV, and CMV. However, his hepatitis C antibody screen was positive with a viral load of 1,180,000 IU/mL. The patient began treatment with methylprednisolone, but daily labs showed continued hemolysis and constitutional symptoms remained unabated. Rituximab was considered for his acute hepatitis C infection and splenectomy for his hemolysis. It was not until hospitalization day 7, however, that his underlying condition finally became clear. His brucella antibody screen suggested an acute brucellosis with an elevated IgM but normal IgG level. This was later confirmed with a brucella antibody titer of 1:320 (normal < 1:80).
The patient began treatment with doxycycline, gentamicin, and prednisone. Rifampin was later added when his TEE revealed a small mobile vegetation of his mitral valve. MRI and LP showed no CNS infection. With his new treatment regimen, the patient's hemolysis and symptoms improved dramatically. Soon after, the patient left against medical advice and was lost to follow up.

Discussion: This patient is unusual due to no prior history of contact with cows or consumption of unpasteurized milk within the last 25 years which are the most common risk factors for brucellosis. Contact with or usage of cocaine contaminated with levamisole is the suspected source, a common cutting agent for cocaine which is a cow de-wormer. Levamisole acts as an immunomodulator by increasing macrophage chemotaxis and T-cell lymphocyte function. It was used for rheumatoid arthritis prior to being pulled from US markets in 2000. Levamisole is used to cut heroin and cocaine because of its euphoria potentiating effects. The U.S. Drug Enforcement Agency in 2012 reported that more than 80% of cocaine seized by law enforcement officials contained levamisole. Due to the unusual nature of his infection in accordance with significant polysubstance abuse further research needs to be done on whether cutting agents can lead to an increased risk of unusual infections like brucella.

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Recognizing Lemierre's Syndrome

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Introduction: Lemierre's syndrome is a rare life threatning bacterial infection. Quick recognition and treatment are important to ensure recovery. In this case we discuss Lemierre's Syndrome as well as the difficulty in treating. It is extremely unusual to have thrombosis of bilateral jugular veins.

Case presentation: 43 year old male presenting with throat pain and muffled voice that started 3 days prior. Pain is on left side associated with fevers measured at home 100.6. Denies any sick contacts no recent travel. Has significant history of Type B dissection treated for 1 year prior. In the ED patient was found to be tachycardic 111, respiratory rate 20 saturating on room air. Labs significant for leukocytosis of 13,000 with neutrophilia. Sed rate 36 and CRP 225.4. CT reveiled pharyngitis and bilateral internal jugular clots. Patient was treated for Lemire's syndrome and ENT was consulted due to concern for abscess drainage. Vascular surgery was also consulted due to thrombosis in bilateral jugular veins. Ultimately patient was given Decadron, clindamycin and ampicillin-sulbactam. 2 of 2 blood cultures showed no growth. Patient was continued on clindamycin on discharge. No decision to further anticoagulate patient as he was symptomatically improving. Patient to go for follow up CT angiogram of head and neck as outpatient to monitor for thrombosis.

Discussion: Lemierre's syndrome is a rare life threatening bacterial infection generally caused by Fusobacterium necroforum. Some cases of lemierre's syndrome have been reported with Streptococcus species as well as EBV. The infection can lead to thrombosis of the internal jugular vien, generally unilaterally. Diagnosis is made with imaging as well as blood cultures. However blood cultures can return negative due to Fusobacterium necroforum being anaerobic gram negative rod. Surgical treatment for Thrombosis is usually reserved in cases of persistent sepsis or progressive thrombosis into cavernous sinus or causing septic pulmonary emboli. It is important to remember Lemierre's syndrome and that prompt treatment is required. Antibiotics recommendations for treatment include antibiotics with beta lactamase resistance and can possibly need further surgical intervention. There is no recommendations on anticoagulation but is considered if thrombosis progresses. It is unusual to have bilateral internal jugular veins having thrombosis.

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The case of the missing testis: diagnostic challenges in an obese patient with testicular cancer

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Background: One to 4.5% of males are born with cryptorchidism and are at increased risk of testicular cancer compared to the general male population. The gold standard for diagnosis of cryptorchidism is a testicular exam under anesthesia followed by exploratory laparoscopy. We present a case of testicular cancer in a patient with an undetectable testis on scrotal ultrasound.

Case presentation: A 35-year old male with a past medical history of morbid obesity (BMI 83), type 2 diabetes, hypertension, and hepatic steatosis came to the hospital with a two-month history of progressively worsening abdominal pain, emesis, dizziness, and dyspnea.
He presented in acute renal and liver failure (Cr 9.1 mg/dL, AST 575 units/L, ALT 177 units/L, total bilirubin 6.7 mg/dL, INR 1.8) and met the Cairo-Bishop criteria for tumor lysis syndrome (uric acid 17.5 mg/dL, phosphorus 6.4 mg/dL). A CT of his abdomen showed retroperitoneal lymphadenopathy and pulmonary nodules. Tumor markers alpha-fetoprotein and CA 19-9 were elevated at 4398 ng/mL (ULN 9) and 77.8 units/mL (ULN 37), respectively. A testicular exam was limited due to the patient's body habitus. The left testicle was not visualized on scrotal ultrasound, concerning for cryptorchidism. A bronchoscopy with lymph node biopsy was performed, and pathology was consistent with yolk sac tumor, leading to a diagnosis of metastatic testicular cancer. Shortly after starting chemotherapy, he developed hypoxic respiratory failure and died.

Discussion: This patient's diagnosis of cryptorchidism was limited by difficult physical exam due to his weight and body habitus. He had no knowledge of abnormal testicular exams during childhood, but cryptorchidism could have been missed due to obesity. A study of in-office scrotal examinations for boys with cryptorchidism showed only a 20% positive predictive value (PPV) for the detection of non-palpable testis in obese patients, compared to 72% in the non-obese. Importantly, there was no difference in PPV when the exam was performed under anesthesia1.
Ultrasound has a sensitivity of 45% for detecting an intra-abdominal testis in children and of 65% for detecting unilateral cryptorchidism in adults2,3. Given the 35% false-negative rate, we cannot definitively confirm cryptorchidism or conclude that the patient's cancer was related to cryptorchidism. His severe presentation highlights the need for thorough testicular examination during childhood followed by diagnostic laparoscopy if abnormal. This facilitates appropriate management to aid in the early detection of possible testicular malignancy in adulthood.

1Breyer, B et al. Obesity does not decrease the accuracy of testicular examination in anesthetized boys with cryptorchidism. J Urol 2009; 181(2): 830-834
2Tasian, G et al. Diagnostic imaging in cryptorchidism: utility, indications, and effectiveness. Journal of Pediatric Surgery 2011; 46: 2406-2413
3Pekkafali, M et al. Comparison of ultrasonographic and laparoscopic findings in adult nonpalpable testes cases. European Urology 2003; 44(1): 124-127

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Black Tar Botulism

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Botulism is a life-threatening neuroparalytic syndrome with 110 cases annually in the US. Wound botulism accounts for 5-10% of cases. Illness is characterized by neuromuscular impairment without the typical gastrointestinal prodrome of foodborne botulism. Rarity of presentation can impede recognition and delay critical treatment of Clostridium botulinum infection.

Case: A 23-year-old female with history of IV drug use presented to an outside hospital with a right-sided sternocleidomastoid abscess and dysphagia. She left against medical advice (AMA), however returned two days later. The abscess was incised and drained, IV vancomycin was started, but again, she left AMA. Within hours, she was re-admitted with altered mental status, and within 24 hours, became unresponsive with acute hypercapnic respiratory failure, requiring intubation. Urine drug screen was positive for benzodiazepines, cocaine, and opioids, but she did not improve with naloxone. Chest x-ray demonstrated mild left-sided consolidation and bronchoscopy cultures grew Haemophilus influenza. She completed 7 days of ceftriaxone, but low oxygenation persisted despite trials of multiple ventilation modes. She failed extubation attempt on day 6, quickly becoming unresponsive, and was re-intubated. Brain imaging was negative for acute process and EEG demonstrated moderate global encephalopathy without seizures. Visitor restrictions were placed given concern that patient may have obtained an illicit substance and overdosed. On day 9, she again failed extubation attempt, and progressive weakness and ptosis were noted. She was transferred to our hospital with presumed myasthenia gravis. On arrival, she was intubated but alert and communicative, with significant ptosis, and weakness in neck and upper extremities. Sensation and reflexes were intact. Pyridostigmine and intravenous immunoglobulin were started, but a myasthenia gravis panel returned negative. An EMG showed pathology at the neuromuscular junction and fast repetitive stimulation EMG was indicative of botulism. The department of public health was contacted and she received botulinum anti-toxin on day 16. The next day, she was successfully extubated. The patient admitted to regular injection of “Black-Tar” heroin into her neck and upper extremities.

Discussion: Wound botulism is rare and diagnosis can often be delayed. Presenting symptoms can include dyspnea, blurry vision, dysphagia, weakness, constipation, and urinary retention, or symptoms similar to acute intoxication. This patient demonstrated a number of key findings including ptosis, failure to wean from ventilator, abscess at injection site, and significant IV drug use, specifically “Black Tar” heroin. The decision to treat with antitoxin is made by presumptive diagnosis, which is supported by electromyography. Diagnosis is confirmed by isolation of the toxin in serum, which is positive less than 70% of the time, or identification of the toxin in wound cultures.

Teaching Point: Recognition of wound botulism in “at-risk” patients is key for initiation of anti-toxin in order to prevent significant morbidity and mortality.

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SLE: A Story That Starts with Cardiac Tamponade

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Introduction: While Systemic Lupus Erythematosus (SLE) can present with a variety of cardiac findings that involve the pericardium, myocardium, and coronary arteries, it is rare for SLE to initially present as cardiac tamponade, specifically with bloody pericardial fluid. Similarly, although SLE is rarely the cause of hemorrhagic cardiac tamponade, it should be within the differential diagnosis to ensure appropriate therapy.

Case: A 21-year-old female with a history of vitamin B12 deficiency called emergency medical services (EMS) for nausea, vomiting, non-bloody diarrhea, diffuse abdominal pain, and mild chest discomfort. She was recently diagnosed with community-acquired pneumonia, but never filled her antibiotics. EMS personnel reported a HR of 140 and a SBP in the 90s. On arrival to the outside ED, her pulse was 150, BP 136/106, temperature 97.5, respiratory rate 20, and SpO2 98% on room air. Physical exam revealed mild RUQ tenderness but was otherwise unremarkable. An EKG was read as sinus tachycardia with a rate of 145 without ischemic changes. Hemoglobin was 6.7 g/dL with an MCV of 76.4; she admitted to non-compliance with her B12 supplement, but denied recent bleeding/bruising. A chest x-ray showed an enlarged cardial/pericardial silhouette suggesting pericardial effusion as well as pleural effusions and perihilar edema. Bedside ultrasound confirmed a large pericardial effusion with tamponade physiology. Re-evaluation of the EKG revealed electrical alternans. She was transferred to our hospital, where she became acutely dyspneic, requiring intubation. 1 L of serosanguinous fluid was removed during emergent pericardiocentesis. Viral pericarditis was suspected, and colchicine monotherapy was initiated. Bacterial and fungal cultures returned negative. Eventually SLE was considered and an ANA titer was found to be 1:320. Anti-dsDNA and anti-Smith returned positive and hydroxychloroquine was added for SLE.

Discussion: SLE most commonly presents with skin manifestations, arthralgias, and generalized fatigue; however 15% of patients present with cardiac findings at onset of disease. While pericardial effusions can occur relatively commonly, cardiac tamponade only occurs in 1-6% of patients during the course of their lupus. Moreover, tamponade occurs as the presenting feature in about half of these patients. Although autoimmune disease is high on the differential for pericarditis or a small pericardial effusion, it is lower on the list in patients presenting with large effusions. Furthermore, SLE is a rare cause of hemorrhagic pericardial effusion. Therefore, in patients without risk factors for the most common causes of hemorrhagic effusions, SLE must be considered. Per EULAR guidelines, treatment with hydroxychloroquine is recommended for all patients with SLE. Glucocorticoids, usually methylprednisolone, are reserved for cases of severe pericarditis and tamponade. Our patient was not treated with steroids, as she improved clinically before the diagnosis of lupus was made, but misdiagnosis and lack of timely treatment can worsen the prognosis of systemic SLE.

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A Case of Chronic Diarrhea and Dyspnea

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Scleroderma is a multisystem disease caused by vascular dysfunction and fibrosis of the skin and internal organs. It often affects multiple organs and thus leads to a variety of clinical manifestations and a heterogeneous presentation. There are multiple types of scleroderma, and some patients with the limited cutaneous subtype of scleroderma exhibit manifestations of the CREST syndrome (Calcinosis, Raynaud's phenomenon, Esophageal dysmotility, Sclerodactyly, Telangiectasia). Prognosis is variable, however most deaths are related to pulmonary or cardiac involvement.

Here we present the case of a 73 year old female who came to the hospital with dyspnea on exertion. She was found to have bilateral pleural effusions secondary to severe hypoalbuminemia. She had evidence of malnutrition with a weight loss of 60 pounds within a year. Past medical history was significant for severe chronic diarrhea of unclear etiology despite extensive workup including colonoscopy with biopsies. After multiple hospitalizations for failure to thrive, she was found to have esophageal dysmotility on esophagogastroduodenoscopy. A more directed history revealed that the patient also exhibited symptoms of Raynaud's phenomenon during cold weather. Closer physical examination revealed telangiectasias and mild skin thickening on the distal extremities. A diagnosis of scleroderma was considered and rheumatologic labs were initiated. She was found to be ANA positive with a high titer of 1:1280. She was also positive for RNP and Centromere B; however, SSA, SSB and anti-Smith antibodies were negative. Additionally, an echocardiogram revealed an elevated Right Ventricular Systolic Pressure of 49 and possible pulmonary hypertension. The patient was eventually stabilized from a nutritional perspective and discharged, and was officially diagnosed with CREST syndrome as an outpatient. She re-presented to the hospital one month later with acute hypoxic respiratory failure. A chest CT was performed and showed recurrent bilateral pleural effusions as well as partial left lung collapse. A repeat echocardiogram showed an elevated pulmonary artery systolic pressure of 57 mmHg further supporting a diagnosis of pulmonary hypertension. The patient was initially treated with diuresis but quickly showed signs of dehydration. She also was unable to tolerate non-invasive ventilation due to issues with mental status. In a discussion on whether intubation would fit the patient's goals of care, the patient and her family decided to forgo further treatment and focus on comfort; the patient passed away shortly after.

This case illustrates the compounding nature of a multisystem disease. The patient displayed mild symptoms of the typical CREST syndrome, but had very profound gastrointestinal manifestations. It took years of these severe symptoms before discovering an explanation, which ultimately caused severe malnutrition, hypoalbuminemia and deconditioning. This compounded with the underlying lung and cardiac involvement likely exacerbated symptoms and may have contributed to the patient's death.

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Heparin-Induced Thrombocytopenia in a Patient on Multiple Anti-Epileptic Drugs

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Heparin-induced thrombocytopenia (HIT) is an uncommon but clinically important and potentially life-threatening condition that should be considered in all patients with acute thrombocytopenia and heparin exposure. Driven by an immune response to complexes formed by platelet factor 4 (PF4) and heparin, a subset of patients with HIT progress to more serious complications such as thrombosis. Here we illustrate a case of a patient with refractory seizures provoked by a primary brain cancer status post craniotomy on multiple antiepileptic drugs (AEDs) who developed thrombocytopenia during his admission and was discovered to have HIT with thrombosis.

A 55-year-old male was admitted to the hospital for status epilepticus following a recent left parietal subtotal resection for glioblastoma multiforme 11 days prior. He was started on a prophylactic dose of unfractionated heparin upon admission and was noted to have a decline in platelets greater than 50% from baseline on hospital day five. The initial leading diagnosis was drug-induced thrombocytopenia (DITP) secondary to antiepileptic use, a well-known cause of DITP. The patient required significant escalation of AEDs for his refractory seizures and was up to 5 different agents at one point. However, a 4T score of 5 placed him at an intermediate risk for HIT, hence heparin was discontinued and a heparin-induced platelet antibody screen was obtained which returned weakly positive with an optical density (OD) of 0.684. A diagnosis of HIT requires serologic evidence, and the most commonly used screen for PF4-heparin antibodies is an ELISA immunoassay which reports the OD of a sample that positively correlates with the probability of HIT. With an equivocal value, as this patient had, a platelet activation assay such as the serotonin release assay is performed. The patient was initiated on fondaparinux, and subsequent functional serotonin release assay confirmed the diagnosis of HIT. An ultrasound of the lower extremities obtained earlier in the hospital course did not reveal thrombosis, but further imaging showed acute upper extremity deep vein thrombosis and bilateral pulmonary embolisms. The patient was continued on fondaparinux at discharge. This medication was chosen due to interactions that other anticoagulants such as novel oral anticoagulants (NOACs) and warfarin have with AEDs. Many AEDs are strong CYP3A4 inducers which may reduce the efficacy of NOACs and cause challenges in maintaining a therapeutic INR while on warfarin.

Thrombocytopenia with concomitant heparin administration is not unusual in the inpatient setting. While HIT constitutes a minority of the causes with other etiologies such as DITP being more prevalent, this case demonstrates the importance of maintaining a high clinical suspicion for HIT, even with evidence that suggests other explanations for thrombocytopenia, and initiating prompt work-up in those with an intermediate to high pre-test probability.

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Outcomes and Predictors of 30-day readmission in patients with Diabetic Ketoacidosis in the United States:A Nationwide Analysis

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Introduction: Diabetes ketoacidosis (DKA) is one of the most critical and life-threatening emergencies in patients with Diabetes Mellitus (DM), resulting in considerable morbidity and mortality along with health care resource utilization. There is limited data regarding clinical and financial cost of readmission in patients with DKA. Therefore, we conducted this study to investigate the outcomes and predictors of 30-day readmission in patients with DKA.

Methods: We queried the 2017 Nationwide Readmission Database (NRD) using ICD-10-CM diagnosis codes to identify all adult patients admitted with a primary diagnosis of DKA from January to November 2017. Outcomes assessed were 30-day readmission rates, mortality, length of stay (LOS) and hospitalization costs. A multivariate cox regression was done to identify independent predictors of readmission. Statistical analysis was performed using STATA software.

Results: A total of 105,151 adult patients were admitted with a primary diagnosis of DKA in 2017, with in-hospital mortality rate of 0.26%. Of the patients discharged alive, 19.6% (20,572) patients were readmitted within 30-days. The most common primary diagnosis at readmission was Type 1 DM with ketoacidosis without coma. When compared to index admission, readmitted patients had higher in-hospital mortality (0.26% vs 0.57%, $p<0.01$), increased mean LOS (3.05 days vs 4.07 days, $p<0.01$) and higher mean hospitalization charges (\$30,089 vs \$38,724, $p<0.01$). Readmission added 83,794 inpatient days and \$796 million in hospitalization cost to the healthcare burden. Higher Charlson comorbidity score (HR 1.19, $p<0.01$), discharge other than home discharge (HR1.78, $p<0.01$), End Stage Renal Disease (ESRD) (HR 1.52, $p<0.01$) and female gender (HR 1.16, $p<0.01$) were associated with higher likelihood of readmission, whereas increasing age (HR 0.98, $p<0.01$), Type 2 DM (HR 0.78, $p<0.01$) and obesity (HR 0.74, $p<0.01$) were associated with decreased likelihood of readmission.

Discussion: In the US, almost 20% patients with DKA are readmitted within 30 days of discharge. Readmission is associated with significantly increased in-hospital mortality, LOS, and hospitalization costs. 30-day readmissions resulted in an increased burden to healthcare resource utilization, adding considerable in-patient stay days and overall hospitalization costs. Female patients, ESRD, discharge other than home discharge and higher comorbidity burden was significantly associated with higher readmission. Patients with Type 1 DM were more likely to be readmitted compared to patients with Type 2 DM. Further studies are needed to assess the high-risk populations to understand the reasons predisposing to early readmission. This will allow physicians to guide clinical practice to reduce readmission and improve quality and cost of care.

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Awareness of HIV Pre-Exposure Prophylaxis (PrEP) Among Michigan State University Undergraduate Students

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Background: HIV infection remains a public health concern with college aged people being among those with the highest incidence of newly diagnosed HIV annually in the United States. Underutilization of HIV Pre-Exposure Prophylaxis (PrEP) remains an overlooked public health issue despite evidence of PrEP efficacy.
Purpose: To determine if undergraduates at higher risk of HIV acquisition lack awareness of HIV PrEP and assess barriers to PrEP use. The study hypothesizes a major barrier to HIV PrEP utilization is lack of awareness of PrEP amongst undergraduate students.
Methods: This is a cross-sectional survey of Michigan State University (MSU) undergraduate students aged 18 or older. The MSU IRB approved the study as "exempt research". An anonymous 15-item multiple-choice online survey with questions that assessed demographics, HIV acquisition risk factors, PrEP awareness and use was utilized. Key variables included gender, sexuality, race, use of PrEP. The study also assessed non-usage factors such as knowledge, perception of need, and cost. Initial sample size was 108 random MSU undergraduate students. Subsequently, a convenience sample was collected through the University Registrar, who emailed a survey link to approximately 40,000 undergraduate students each week for a 3-month period. During this period, oversampling of undergraduate students in select venues (Counseling & Psychiatry Services and the MSU LBGT Student Resource Center electronic newsletter) was done with the intent to reach more high risk students. Using a potential population of 40,000 students and a 3% sampling error, it was estimated that 1039 students would provide the ability to estimate 95% confidence intervals for the data. EpiInfo (CDC, version 7.2.2.6) was used to perform frequency estimates of key variables and Chi- square analyses to compare PrEP usage and PrEP awareness in study population.
Results: 3030 unique survey responses were collected. Eighteen were either incomplete or not usable, resulting in 3012 usable surveys. Only 28.5% of MSU undergraduate students were aware of PrEP. No difference was found in PrEP awareness among racial groups. Of individuals not aware of PrEP, 28% reported behavior at risk for acquiring HIV. Among individuals who reported high risk behaviors, 67% are not aware of PrEP. 92% of homosexual, lesbian, gay and queer individuals having sex with men were aware of PrEP, vs only 24.7% of heterosexual, straight individuals having sex with men.
Conclusions: The study reveals a significant lack of PrEP awareness among the undergraduate student population at MSU. Awareness of PrEP in homosexual, lesbian, gay and queer individuals was highest in this study. This suggests current targeted interventions towards the LGBT+ community may be effective in increasing awareness. A high percentage of self-reported high risk individuals had no awareness of HIV PrEP. High risk and average risk populations may benefit from HIV PrEP education.

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Hospital readmission rates for Chronic Obstructive Pulmonary Disease (COPD) and Congestive Heart Failure (CHF) exacerbations in

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Introduction: Untreated obstructive sleep apnea (OSA) has been linked to increased readmission rates and worsened prognosis of chronic obstructive pulmonary disease (COPD) and congestive heart failure (CHF). We hypothesize that undiagnosed obstructive sleep apnea is an underrecognized risk factor for hospital readmissions due to decompensated COPD and CHF. And that treating OSA can decrease the hospital readmission rates for these conditions.

Methods In this upcoming retrospective chart review we will enroll patients with an admitting diagnosis of COPD or CHF exacerbation. We will use the Berlin sleep and STOP-BANG questionnaires to risk stratify patients risk for obstructive sleep apnea. We will recommend those who are high risk for OSA and should undergo a sleep study. Of those enrolled we will review their medical records from the prior 12 months to compare admission rates for decompensated COPD or CHF between those who high a high or low likelihood of OSA based on our risk stratification.

Results: We plan to enroll 50 patients with COPD and 50 patients with CHF. We expect to measure primary outcomes of inpatient hospital admission rates over the previous 12 months and estimate patient's riskof OSA using questionnaires. We expect to have data of the retrospective study by the ACP meeting. We will analyze these comparisons using Pearson chi-square and calculate odds ratio for the data.

Conclusion: We expect to show that patients at high risk of OSA have a higher odds ratio of previous hospital admissions for decompensated heart failure or COPD.

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Diagnostic utility of bronchoscopy in newly diagnosed acute leukemia patients with respiratory failure

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Background: One important complication of acute leukemia is acute respiratory failure, which has been reported to occur in over 40% of patients receiving induction therapy. Bronchoscopy is a valuable tool for the evaluation of airway disease. The diagnostic yield of bronchoscopy in this setting has not been fully explored.
Methods: We performed a retrospective chart review of 75 newly diagnosed acute leukemia patients who had bronchoscopies during their initial hospitalization in 2011-2015. Data recorded prior to bronchoscopy included age, diagnosis, induction treatment regimen, chest imaging findings or need for assisted ventilation, duration of neutropenia and antibiotic therapy and presence of positive microbiological studies. Bronchoscopy culture data were sorted by organism found, as well as by other pathologic findings such as diffuse alveolar hemorrhage. The primary outcome was antibiotic change supported by culture data.
Results: The study population included 67 AML, 2 APL, 5 B-ALL and 1 T-ALL patients with a median age of 57 (range 18-86). Induction regimen backbones included 7+3 (51), 7+4+ATRA (2), decitabine (12), hyperCVAD (2), AYA (3), other (1) and none (4); 18 patients were treated on clinical trial. Average days of neutropenia was 10.92 and average days of antibiotic therapy was 10.57. Thirty-eight patients had positive chest imaging, 11 received NIPPV, and 17 were mechanically ventilated. We identified 24 patients with positive bronchoalveolar lavage studies. Of these, 37.5% (9) had positive culture data before bronchoscopy, with only 3 having cultures positive for the same organism as the BAL. Of 51 patients without bronchoscopy findings, 33.3% (17) had negative culture data beforehand. Identified infections were most commonly bacterial (15/24), followed by viral (5/24) and fungal (4/24). The most common organisms were rhinovirus, vancomycin sensitive Enterococcus (each 4/24) followed by VRE, Candida albicans and MRSA (each 2/24). Additionally, 5 patients had alveolar hemorrhage. Of the 24 patients with +BAL cultures, bronchoscopy findings supported changing antibiotics in 18. In contrast, antibiotics were changed in 16 patients without +BAL cultures.
Conclusion: We investigated the utility of bronchoscopy on AL patients during their initial admission. BAL cultures were positive in 32% of newly diagnosed acute leukemia patients undergoing bronchoscopy. However, BAL cultures guided antibiotic therapy in 75% of patients with positive cultures. Prior culture data did not predict bronchoscopy findings. Further studies will be needed to establish predictors of bronchoscopy findings in this patient population.

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Use of CHADS2VASC and other factors to assess for risk of thromboembolic events in patients with Takostubo Cardiomyopathy

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Purpose: Similar to patients with anterior myocardial infarctions, patients with Takotsubo Cardiomyopathy (TCM) are known to be at risk of thromboembolic events. Providers are uncertain if patients with TCM should be routinely placed on anti-coagulation. The primary purpose of this study was to evaluate risk factors for thromboembolic events (e.g. left ventricular thrombus, stroke, and arterial embolus) in patients diagnosed with Takotsubo Cardiomyopathy, specifically CHADS2VASC, Ejection Fraction, Anticoagulation Status as well as other disease state and demographic variables.

Methods: A retrospective chart review was performed on 134 patient diagnosed with TCM from 2010 to 2019 who survived at least one year after their TCM event.

Results: Of 134 patients diagnosed with TCM, 8 had a thromboembolic event (6.0%). 6.6% of 61 patients with a low CHADS2VASC score (0-2) had a thromboembolic event, compared to 7.4% of 58 patients with a medium score (3-5) and 0% of 11 patients with a high score (6-8), P-value 0.673. 9.5% of 23 males had a thromboembolic event, compared to 5.4% of 111 females, P-value 0.544. 10% of 60 patients with an ejection fraction less than 35% had a thromboembolic event compared to just 2.7% of 74 patients, P-value 0.038. Finally, of the 8 patients with TCM who had a thromboembolic event, only 1 was on anticoagulation at the time of their event.

Conclusion: TCM has been a disease state that has now been identified for close to 30 years. Still to this day, clinicians are very heterogenous with regards to which patients to place on anticoagulation. At this juncture, very few additional risk factors have been identified as to which patients are at most risk of a thromboembolic event after being diagnosed with TCM. Additional research is warranted.

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The Impact of Community Outreach and Advocacy in an Internal Medicine Residency Program

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Introduction: The American Medical Association and American Council for Graduate Medical Education (ACGME) have stated that professionalism standards for physicians extend beyond clinical practice, including participating in community advocacy to promote societal health and well-being. Graduate medical education (GME) is an important time for community outreach4. It is a time when professional knowledge and attitudes are solidified, and may provide a platform for physicians to continue to participate in outreach activities during their careers. Currently there are no standards for incorporating community advocacy into training programs.

Methods: Sixty internal medicine residents were divided into 5 groups according to their pre-assigned outpatient clinic groups. Three of the five groups participated in one half-day of community outreach at either 1) Homeless shelter, 2) An interactive exhibit exploring the relationship of structural racism in the Cleveland housing market to subsequent healthcare disparities, and 3) Local community organization that promotes healthy living through farmer's markets and outdoor exercising space for local residents. The other 2 groups were unable to complete their assigned session due to the COVID-19 pandemic. We collected anonymous pre-surveys on the same day as the outreach and post-surveys 5 weeks later to understand residents' overall views about outreach and interest in future participation. Unpaired t-tests and chi-square tests of independence were used to analyze differences between pre- and post-surveys.

Results: Thirty-six residents completed the pre-study survey and 41 residents completed the post-study survey. Although no statistically significant differences were found in pre/post responses, 91% of residents would participate in community advocacy work again in the future, and 85% of residents would like community outreach to be incorporated into the outpatient curriculum. Additionally, residents felt that the outreach experience increased their understanding of the communities where their patients live and the daily health barriers they encounter.

Discussion: Our study's results are consistent with prior studies which have found that trainee's experience of community advocacy was generally positive, and improved trainee's knowledge and attitudes regarding the communities where their patients live. Our study also found that most internal medicine residents would be motivated to continue participating in community outreach activities in the future if incorporated into an outpatient didactic curriculum. Prior research has identified several key areas needed for successfully implementing advocacy curricula, including: 1) Requirements from ACGME, 2) Institutional support (e.g. protected time from clinical duties, funding), and 3) Faculty volunteers. Barriers include competing curricular demands, time constraints, lack of local opportunities, and faculty/resident turnover.

Our study further demonstrates that integrating community outreach into GME-level training is well-received. More work is needed to create guidelines and expectations surrounding professional advocacy, as well as tools for ensuring sustainability given their importance for trainees and local communities.

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Identification of a Risk Profile for New Onset Diabetes After Acute Pancreatitis

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Background: Impaired glucose tolerance in common after an episode of acute pancreatitis (AP) and nearly one in four patients develop new-onset diabetes, yet there is a paucity of studies evaluating the predictors of new-onset diabetes mellitus following AP (AP-related DM). We sought to utilize a population-based database to evaluate predictors of AP-related DM.

Methods: The Nationwide Readmissions Database (2010-2014) was utilized to identify all adult subjects (age ≥18 years) with a primary discharge diagnosis of AP. Multiple exclusion criteria were applied to identify defined cohorts of subjects with and without DM after an index episode of AP. A case-control study with multivariable analysis was conducted to identify population-level risk factors for developing AP-related DM within the calendar year.
Results: During the study period, 2,510 subjects with AP-related DM and 40,308 controls with AP who did not develop DM were identified. After adjusting for multiple covariates, multivariable analysis revealed that increasing age (50-64 years of age, adjusted Odds Ratio (aOR) 1.35, 95% CI 1.14-1.60), male sex (aOR 1.2, 95% CI 1.03-1.40), lowest income quartile (aOR 1.48, 95% CI 1.18-1.84), Elixhauser comorbidity index ≥3 (aOR 1.47, 95% CI 1.23-1.75), metabolic syndrome associated AP (aOR 2.12, 95% CI 1.21-3.70), severe AP (aOR 1.60, 95% CI 1.34-1.90), and recurrent AP (aOR 1.46, 95% CI 1.24-1.72) were independently associated with increased risk of AP-related DM within the same calendar year following index hospitalization.

Conclusion: In this national survey, we identified key predictors of new onset DM after hospitalization with AP. The risk profile may be useful to identify patients who may benefit from closer follow-up and intensive education, and a high risk group to study for preventative strategies.

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The Prevalence of Venous Thromboembolism and its Impact on the Severity of COVID-19: A Systematic Review and Meta-analysis

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Introduction: Coronavirus Disease 2019 (COVID-19) is associated with a hypercoagulable state, which can result in venous thromboembolism (VTE). The purpose of our meta-analysis was to investigate the prevalence of VTE and its impact on the severity of COVID-19.
Methods: We performed a comprehensive search in the literature for studies that evaluated VTE in COVID-19. We searched the databases of PubMed/MEDLINE, Embase, World Health Organization COVID-19 Database, LitCOVID, and Web of Science Core Collection databases from January 1, 2020 until May 11, 2020. The search was not limited by language, study design, or country of origin. Two researchers (SG and YK) independently selected the studies; discrepancies were resolved by a third researcher (CN). We considered randomized controlled trials, cohort studies, case-control studies, and case series. We excluded animal studies, case reports, reviews, editorials, and letters to editors. We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The random-effects model was used to calculate the prevalences, odds ratios (OR), and confidence intervals (CI). Our main outcomes were the prevalence of pulmonary embolism (PE), the prevalence of deep venous thrombosis (DVT), and the need for intensive care unit (ICU) admission based on the presence of PE or DVT. Publication bias was assessed visually by generating a funnel plot of the studies that reported the prevalence of PE and DVT. We also performed Egger's regression to quantitively assess publication bias, where $p < 0.05$ was considered statistically significant for publication bias.
Results: We included 13 studies with a total of 1321 patients. The prevalence of PE was 17.4% (95% CI: 11.1% - 23.7%) and the prevalence of DVT was 14.4% (95% CI: 8.7% - 20.1%). The percentage of patients who required ICU admission was significantly higher in patients who had PE compared with patients who did not have PE (69.2% vs 22.7%, OR: 5.8, 95% CI: 3.2 – 10.6, $p < 0.001$, $I2 = 3.8\%$). The percentage of patients who required ICU admission was significantly higher in patients who had DVT compared with patients who did not have DVT (81.3% vs 18.4%, OR: 10.3, 95% CI: 1.3 – 83.0, $p = 0.029$, $I2 = 70.5\%$). There was visible symmetry in the funnel plot of the studies that reported the prevalence of PE and DVT, suggesting no publication bias. Similarly, Egger's test was not statistically significant implying no publication bias for the prevalence of PE ($p = 0.51$) or DVT ($p = 0.79$).
Conclusions: In conclusion, COVID-19 patients have a high prevalence of VTE. VTE can worsen the clinical course of COVID-19 and result in higher rates of ICU admissions. We recommend testing for VTE when clinically appropriate, especially when COVID-19 patients fail to improve clinically.

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Successful treatment of Rheumatoid Arthritis Associated Interstitial Lung Disease with Rituximab; A case series.

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Rheumatoid arthritis (RA) is a chronic autoimmune disease affecting nearly 1% of the population. RA associated interstitial lung disease (RA-ILD) is one of the extra-articular manifestations of RA associated with a significant increase in morbidity and mortality. Existing literature is scarce on best approach to treatment of RA-ILD. This report describes three RA-ILD patients who experienced significant improvement of RA-ILD after treatment with Rituximab, an anti-CD20 monoclonal antibody. All patients initially received two 1000 mg doses of Rituximab two weeks apart. Repeated every six months.

Patient #1, 61 years old African American male with seropositive (RA) and ILD, RA manifested by inflammatory arthritis and synovitis of hands small joints bilaterally associated with synovial thickening of interphalangeal joints, (ILD) has been worsening despite multiple courses of Prednisone, Hydroxychloroquine and continuous O2, Pulmonary function test (PFT) was strikingly diminished FEV1 42%, FVC 38% and DLCO 23%, was being evaluated for lung transplant. Rituximab initiated, after first dose PFT improved, FEV1 47.18%, FVC 43.88, more improvement in PFT was achieved, O2 sat is 94% on ambient air (off the O2 for the first time in two years), significant improvement reported in joint pain, PFT is FEV1 69%, FVC 65%. Our patient is able to walk 2.3 mile, maintain normal O2 sat, lung transplant plans were held off.

Patient #2, 65 years old female patient with seropositive RA and ILD, ILD related arthritis, Rituximab led to significant improvement in respiratory function, over the course of treatment he received two cycles of rituximab, in the meantime she was on small dose prednisone, leflunomide 20 mg and hydroxychloroquine. FVC improved significantly from 50% to 64% with significant improvement in DLCO. After resuming the full dose of rituximab her symptoms improved significantly.

Patient #3, a 74 years old male patient with seropositive RA and ILD since 2004 on Infliximab, methotrexate and prednisone, rituximab started early 2015 for lack of symptoms control and worsening lung function, within few months of initiating the rituximab infusion, he reported significant improvement in hands pain, breathing and exercise tolerance. The PFT significantly improved FVC of 99% and DLCO on 60%, to FVC 107% and DLCO 62%.

Conclusion: In conclusion, we report a favorable outcome in patients with RA-ILD after treatment with Ritixumab. Our findings suggest a potential role for rituximab in treatment of RA-ILD.

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Relationship of Body Mass Index to Mortality after Colorectal Cancer Diagnosis

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Introduction: Colorectal cancer (CRC) in young to middle-aged adults has been rising in developed countries. This increase has paralleled the rising prevalence of obesity, with worldwide obesity having nearly tripled since 1975 according to the World Health Organization. To study how obesity relates to mortality after CRC diagnosis, we examined the association between body mass index (BMI) and mortality in 676 patients diagnosed with CRC at the Dayton Veterans Affairs Medical Center (Dayton VAMC) for a period of 18 years.

Methods: This study was a retrospective chart review. After IRB approval, patients with a diagnosis of colorectal cancer from January 01, 2000 to December 31, 2018 were identified from the electronic database at the Dayton VAMC. The demographic variables of age, gender, and race were collected as were the clinical variables of BMI, family history of colorectal cancer, cancer type, histology, cancer stage at diagnosis, treatment, and mortality.

Results: For the 676 patients the mean age at diagnosis was 68.2±10.3 years with 97.8% male, 85.4% Caucasian and 14.2% African American. There were 221 (32.7%) patients under/normal weight, 208 (30.8%) overweight, and 247 (36.5%) obese, and mortality rates were 77.8%, 66.3%, and 59.9%, respectively ($p<0.001$). In addition, mean survival increased as BMI category increased: 57 months, 68 months, and 77 months ($p<0.001$). Consequently, survival and survival time increased with progression of BMI. Further, the Kaplan-Meier survival curve graphically displays survival experience reported in months.

To determine whether BMI was an independent predictor of mortality, we entered BMI into a multiple logistic regression equation with those predictors related to mortality on a univariate basis at $p<0.05$ (age, gender, site of cancer, ASA, statins, tobacco use, CAD, HLD) and family history of colorectal cancer. Lower BMI was an independent risk factor for mortality after controlling for these nine covariates: odd ratio = 1.04 (95%CI = 1.01 to 1.07, $p=0.012$). Again, as BMI increased, the risk of mortality decreased.

Discussion: BMI is an important consideration in patients, including those with colorectal cancer. Obesity is often among the first risk factors about which physicians counsel their patients. Nevertheless, we found that lower BMI is an independent risk factor for mortality among colorectal cancer patients. We postulate that weight loss has a deleterious prognostic effect in CRC patients, especially in advanced disease. In our study, we recorded patient BMI at time of diagnosis. For some patients, the diagnosis of CRC was at an advanced stage, and lower BMI may have correlated with malnutrition secondary to their disease. Previous studies have shown that lifestyle modification such as exercise and weight loss are protective against development of CRC. However, after diagnosis of CRC, losing “healthy weight” is not encouraged due to the deleterious effects of undernourishment.

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Myocardial Infarction in a 28-year old male with Neurofibromatosis Type 1

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Type 1 Neurofibromatosis (NF1) is an autosomal dominant genetic disorder that affects multiple systems throughout the body. Although there are multiple documented vasculopathies that can be seen in NF1, there are very few documented cases of coronary artery aneurysms with thrombosis of the ectatic vessel resulting in myocardial infarction. The low incidence, lack of clinician awareness, and severity of a life-threatening event in a young individual can make diagnosis and management challenging.

A 28-year old male with past medical history of NF1 presented with dull, left sided, substernal chest pain. On physical exam, he had a regular rate and rhythm without any murmurs, rubs, or gallops. His initial EKG showed 4-5mm ST segment elevations leads V1-V5 and an initial troponin level was 35.50. The patient's troponin continued to trend upwards to 49.27 and peaked at 62.01. He was initially given nitroglycerin, aspirin, and started on heparin. Cardiac catheterization was performed and was significant for severe thrombotic occlusion of the mid left anterior descending artery (LAD) with TIMI flow 0. Percutaneous coronary angioplasty with thrombectomy was attempted and was unsuccessful as the operator was unable to clear the thrombus, with TIMI flow 0 after the intervention attempt. The LAD was noted to be severely ectatic. The unsuccessful PCI was due to aneurysmal dilatation and complete thrombotic occlusion of the vessel. The mid-inferior, apical-anterior and apical-inferior segments of the LV were akinetic on angiography. After cardiac catherization, the patient was started on tirofiban and dual antiplatelet therapy. An echocardiogram was performed which showed ejection fraction (LVEF) of 30-35% and LV hypokinesis with no clot formation. He had multiple episodes of non-sustained ventricular tachycardia (NSVT) the day after his catheterization. Due to low LVEF, the patient was placed on a life vest and later received an implantable cardioverter defibrillator (ICD) device. After reviewing his previous echocardiogram and ventriculogram, there was suspicion of aneurysmal behavior of the left ventricle. A cardiac MRI (CMR) was performed to assess this suspicion and showed thrombus formation in the left ventricular apex.
This case report is presented to familiarize physicians with the rare vasculopathies that can occur in patients with NF1. Occlusive or aneurysmal disease can occur almost anywhere in the body in patients with NF1 due to proliferation of fusiform endothelial cells in the blood vessels. These coronary vasculopathies are especially difficult to manage and can lead to long term structural and functional abnormalities in cardiac function.

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BRASH Syndrome: A Novel Clinical Constellation of Bradycardia, Renal Failure, AV-Nodal Blockade, Shock and Hyperkalemia

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Introduction

The constellation of Bradycardia, Renal insufficiency, Atrioventricular (AV) nodal blockade, Shock and Hyperkalemia is a novel clinical syndrome which was recently described in 2016. We report a case of 75-year-old male who was admitted with severe bradycardia and hyperkalemia associated with chronic beta-blocker use and acute kidney injury.

Case Description

A 75-year-old male with underlying coronary artery disease, ischemic cardiomyopathy, peripheral arterial disease and chronic kidney disease presented to emergency department with three days history of worsening fatigue, confusion and slurring of speech. His blood pressure was borderline on presentation with heart rate (HR) in low 40's. His home medications included use of carvedilol 6.25 mg twice daily. CT head was unremarkable for any acute intracranial abnormality. Initial laboratory work-up was pertinent for acute kidney injury (AKI) with Cr of 6.47 mg/dl and potassium of 5.8 meq/L. 12-lead ECG showed junctional rhythm at rate of 30 and left bundle branch block. Patient was intubated for airway protection and admitted to ICU. Patients' HR further dropped with associated hypotension requiring initiation of two vasopressors, norepinephrine and dopamine. Cardiology and Nephrology consultation was obtained. Beta-blockers were held. Patient underwent urgent hemodialysis with correction of renal parameters and subsequent improvement in his HR. Vasopressors were then successfully weaned off. Patients was extubated on day 2. His Cr started returning to the baseline and dialysis was eventually stopped. Patient was transferred out of ICU on day 4 and is currently recovering well with stable hemodynamics.

Discussion

Concomitant use of AV nodal blocking agents and hyperkalemia from acute on chronic kidney injury is the likely etiology of BRASH syndrome. Renal failure not only causes hyperkalemia but also impaired clearance of AV nodal blockers. This leads to synergistic blockade of AV node causing severe bradycardia and hypotension. A vicious cycle then starts with further worsening of AKI from renal hypoperfusion. The treatment in this scenario is directed towards correction of hyperkalemia and discontinuing all negative chronotropic agents. Temporary pacemaker might be necessary in cases of refractory bradycardia and hypotension.

Conclusion

Through this case report, we highlight the importance of cautious use of AV nodal blocking agents in patients with multiple co-morbidities particularly chronic renal insufficiency.

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Gender Related Variation in the Aorto-iliofemoral Arteries in Patients Undergoing Transcatheter Aortic Valve Replacement

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Background: Determining aortic valve and vascular size is needed in order to determine feasibility of performing a transcatheter aortic valve replacement (TAVR). Variations in size of the aortic valve and iliofemoral arteries can impact TAVR size and deliverability, respectively. To determine if gender variations exists in the aorto-iliofemoral arteries this study was undertaken.

Methods: One-hundred fifty-one patients undergoing TAVR were studied. Aorta and iliofemoral artery areas were obtained from pre-procedural computed tomography imaging and compared between genders. Specifically, the following areas were determined: aortic annulus (mid systole), sinotubular junction, ascending aorta (4 cm above annulus), right and left common iliac arteries, right and left external iliac arteries, and right and left femoral arteries. Non-adjusted area and adjusted area for body surface area (BSA) were determined.

Results: Non-adjusted areas were significantly greater in men for aortic annulus, sinotubular junction, ascending aorta, left and right iliac arteries, and left and right femoral arteries (5.29±0.83 vs. 4.27±0.78 cm2, p<0.0001; 6.86±1.57 vs. 5.70 ±1.43 cm2, p<0.0001; 8.80±1.69 vs. 7.98±1.64 cm2, p<0.005; 0.52±0.19 vs. 0.42 ±0.15 cm2, p<0.005; 0.50±.016 vs. 0.38±0.13 cm2, p<0.0001, 0.48±0.18 vs. 0.40±0.14 cm2, p<0.005; 0.47±0.16 vs. 0.40±0.12 cm2, p=<0.05, respectively). After adjusting for BSA, areas were significantly greater in men for aortic annulus, sinotubular junction, and left iliac and femoral arteries (2.63±0.42 vs. 2.36±0.36 cm2, p<0.0001; 3.40±0.73 vs. 3.16±0.71 cm2, p <0.0001; 0.26±0.09 vs. 0.23±0.08 cm2, p= 0.04; 0.25±0.08 vs. 0.21±0.07, p= 0.01 cm2, respectively).

Conclusions: Gender variations in aorta-iliofemoral artery areas persisted even after adjusting for BSA. This information may be useful when planning TAVR procedures.

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Fenofibrate and CKD: A Case Series

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Introduction: Fibrates are indicated in treatment of dyslipidemia in patients with diabetes and provide renoprotection by preventing atherosclerosis. Few RCT and retrospective studies hypothesized a dose dependent reversible increase in serum creatinine with use of fibrates. The objective of our case series study was to evaluate the reversibility of changes in serum creatinine levels in chronic kidney disease (CKD) patients by discontinuing fibrates.

Methods: Patients whose creatinine changed abruptly, with no other change in medications, no new nephrotoxic drugs added, and no explainable reason for worsening renal function were included in this series with the sole intervention consisting of discontinuing fibrate therapy. Eight patients on fibrate therapy were included. Charts were retrospectively reviewed. Clinical data, including comorbidities, medications and laboratory data before and after discontinuing fibrate therapy were collected. Statistical analysis was done using a one-tailed paired student's t test in SPSS.

Results: Fenofibrate with variable doses were used in 8 outpatients to address dyslipidemia in patients with a history of CKD. Serum creatinine and calculated GFR levels improved in six out of eight patients, whereas in two patients improvement after discontinuation of fibrates was not observed. At baseline initiation of fenofibrate in the small patient series (n=8), BUN was 37.3 + 18.1, decreasing after discontinuation to 33.8 + 10.3 (one tailed paired $t = 0.955$, $p = 0.186$). GFR was 32.0 + 11.1, rising to 35.0 + 11.4 (one tailed paired $t = -1.664$, $p = 0.070$) after cessation. Creatinine declined significantly from 2.1 + 0.6 at baseline administration to 1.9 + 0.5 (one tailed paired t test = 2.491, $p = 0.021$) after fenofibrate was discontinued.

Discussion: Studies report fibrates cause dose dependent decline in renal function. The mechanism of renal dysfunction is due to impairment of vasodilatory prostaglandins, with resultant decrease in renal plasma flow and glomerular pressure.
According to our results, discontinuation of fenofibrate therapy resulted in statistically significant variations in serum creatinine and GFR. We observed that an increase in creatinine could occur at any time from initiation of the fibrates. Though we included only fenofibrate, literature associates implicates other fibrates with the same adverse effects; gemfibrozil is a much safer option. In summary, if serum creatinine rises > 30%, fibrate therapy should be discontinued. As our case series is very small, there is a need for a larger powered study to address standard guidelines for the dose, efficacy and management of fibrate side effects.

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Injectafer-induced hypophosphatemia

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Introduction: Hypophosphatemia is a commonly observed side effect to medications such as antibiotics, insulin, antacids, diuretics, and bisphosphonates. We present an interesting case of hypophosphatemia in a patient who received parenteral iron administration.

Case: A 50 y/o male with a past medical history of coronary artery disease, heart failure reduced ejection fraction, iron deficiency anemia, and gastric ulcer disease presented to his primary care physician for two months of worsening fatigue and generalized weakness significantly affecting his mobility. Prior to this, he was very active. Previously the patient was referred to hematology for further workup of anemia and he was started on Injectafer. After a few months, his fatigue improved but now the patient started to have new pain in lower extremities mostly in the left hip. Physical examination revealed decreased strength and limitation in hip joint motility bilaterally. MRI showed osteomalacia and compression fracture in the femoral head on the left hip. Further workup for osteomalacia was negative for other etiologies. Pertinent labs were serum phosphate 1.4 mg/dl, calcium 9.3 mg/dl, ALP 198, Vit D 25 hydroxy 25 ng/dl, 24-hour urinary phosphate 614, PTH 67.3 pg/dl. Injectafer was discontinued. The patient was started on phosphate supplements and encouraged to eat a phosphate-rich diet. After a few weeks, serum phosphate levels normalized, and lower extremity strengths improved.

Discussion: Iron deficiency anemia (IDA) is a commonly seen condition in daily clinical practice. Usually, oral iron supplements are the first-line treatment but if there are insufficient absorption and frequent side effects, the other option is the administration of IV iron formulations. One of the undesired and frequently under-recognized side effects of IV iron supplementation is severe hypophosphatemia (Megapanou et al., 2020). Patients can develop numerous side effects such as osteomalacia, fragility fractures, and hypoxemia (Megapanou et al., 2020). As per the literature review, the underlying mechanism involves increased FGF-23 levels, which inhibits 1-alpha-hydroxylation of vitamin D, leading to decreased intestinal and renal phosphate absorption. FGF-23 is also associated with the downregulation of sodium-phosphate co-transporter NaPi-2a in the proximal tubule (Wolf et al., 2018). Of all the parenteral Iron formulation, ferric carboxymaltose poses the greatest risk of hypophosphatemia (Schaefer et al., 2016). As witnessed in this case report, this can lead to significant impairment and disability. Frequent monitoring of blood phosphate levels and addressing other factors that can contribute to Hypophosphatemia is important to prevent disability associated with IV iron.
In conclusion, increased awareness of the side effects of IV Iron formulation is crucial in order to recognize, prevent, and actively manage the potential side effects of parenteral iron supplement. This can lead to significant improvement in compliance and a better quality of life.

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Improving Cardiovascular Risk Assessment in Rheumatoid Arthritis and Systemic Lupus Erythematosus Patients in a Resident Clinic

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Introduction: Cardiovascular disease is one of the leading causes of death in rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) patients. Its risk in RA and SLE is comparable to that of diabetes. Observational studies show that RA patients frequently have unidentified and untreated risk factors due to gaps in screening. There are currently no guidelines in the United States on cardiovascular risk reduction for patients with autoimmune diseases. EULAR guidelines have suggested multiplying 10-year ACC/AHA atherosclerotic cardiovascular disease (ASCVD) risk scores by 1.5. A 2015 study by Ozen et al. found that screening for 10-year ASCVD risk scores of >5% led to increased detection of subclinical atherosclerosis. Our aim is to increase the rate of annual lipid screening in RA and SLE patients at our resident clinic. We also aim to increase the use of statin therapy in patients with 10-year ASCVD risk scores ≥5%.

Methods: Baseline data was established by reviewing electronic medical records (EMRs) of patients with SLE and RA in the internal medicine resident clinic. Two plan-do-study-act (PDSA) cycles were performed over a four-month period. First, we developed a custom EMR note template for the resident rheumatology clinic that prompted residents to review lipid screening, calculate 10-year ASCVD risk score, determine if patients were on appropriate statin therapy, and notify the patient's primary care provider as needed. We also provided resident education on ASCVD risk assessment and utilization of note template. Second, we adjusted the note template to prompt residents to notify clinic staff to schedule an office visit for cardiovascular risk assessment if indicated and provided additional resident education.

Results: We reviewed 79 patients in the pre-intervention period (73.4% RA, 20.3% SLE, 6.3% SLE and RA). Ninety-two percent of patients were ≥ 40 years old, 82% were female, and 41% had comorbid conditions of diabetes mellitus, coronary artery disease, or cerebrovascular accident. After PDSA cycle 2, 88 patients with similar baseline characteristics were reviewed. Rates of documented lipid screening (92.4% vs 93.2%, $p = 0.846$) and annual lipid screening (62.0% vs 60.2%, $p = 0.812$) did not show improvement. The proportion of patients with 10-year ASCVD risk scores ≥ 5% on statin therapy in the current population (33.3% vs 57.9%, $p = 0.038$) showed improvement; a subgroup analysis of the original 79 patients (33.3% vs 58.3%, $p = 0.038$) also showed improvement.

Conclusion: In our clinic, there was a statistically significant increase in the proportion of patients on statin therapy with 10-year ASCVD risk scores ≥5% in the current and baseline populations. The rate of yearly lipid screening remained unchanged despite the use of a custom EMR template. Future PDSA cycles will focus on improving the rate of annual lipid screening.

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Reducing the all-cause-30-day hospital readmission rate of patients presenting with acute congestive heart failure. A QI project

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Introduction: An estimated 6.5 million of adult Americans have heart failure (CHF) [1]. CHF is the most common cause of hospitalization in people older than 65 years of age in the US [2] accounting for 15% of total in-patient costs [2] and it has the highest 30-day rehospitalization rate amongst other medical and surgical conditions, accounting up to 26.9% [3]. We conducted a quality improvement project at our hospital aiming to reduce 30-day all cause readmissions of patients with CHF by improving the transition of care through providing patient education material and setting up follow up appointments within two weeks of patient discharge.

Methods: Retrospective data collection before the implementation of the project included a list of patients with a primary diagnosis of CHF who were admitted during November 2017. Readmission rates were calculated as the percentage of CHF patients who were readmitted to the hospital within 30 days of discharge for any unplanned cause. Similarly, prospective data included patients who were enrolled in the project during November 2018. Patients who were younger than 18 years old, were on dialysis or those with discharge disposition other than their homes were excluded from both the retrospective and prospective components of the study.
Written patient education materials regarding CHF exacerbation symptoms, life style modifications and importance of medication compliance and follow up were provided to the nurses to distribute to and educate the patients before their discharge. In addition, all nursing staff and health unit coordinators (HUC) were asked to ensure there was a follow-up appointment scheduled with the patient's primary care physician, cardiologist or the heart failure clinic before they are discharged.

Results: All patients admitted with CHF in November 2018 were provided with patient education materials. Of the enrolled patients, 58% had a follow-up appointment scheduled within 2 weeks of discharge written on the discharge instructions compared to only 30% in 2017 (figure 1). Fifty-six percent of enrolled patients kept follow up compared to 37% in 2017 (figure 2). The 30-day readmission rate of CHF patients was cut in half after the implementation of the project, with 14% readmission rate for enrolled patients compared to 28% in 2017 (figure 3).

Conclusions: Providing patient education and ensuring post- discharge follow up with the help of the nursing staff and HUC can lead to substantial decrease in the readmission rate of CHF patients.

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An EMR-based approach to improve physician compliance to the standard of care in pre-chemotherapy pregnancy screening

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Cancer in pregnancy is a growing field of interest with the increasing average age of pregnant women. Pregnancy excludes several conventional treatments and all chemotherapy is relatively contraindicated in the first trimester. This poses an ethical and therapeutic dilemma to patients and physicians alike. As such, NCCN guidelines suggest that all premenopausal women be counselled regarding the potential impact on fertility of chemotherapy, radiation therapy or endocrine therapy and recommend pregnancy testing in all women of child-bearing age prior to its initiation. Nevertheless, physician compliance to the standard of care in pre-chemotherapy pregnancy screening was only 20.5-35% in previous studies. As such, this quality improvement project set out to establish an EMR-based intervention to improve physician compliance in a medium-sized academic hospital setting. Our pre-intervention review showed that our internal compliance to pregnancy screening was only 18%. By implementing a best-practice advisory through the EMR within the chemotherapy order-set for eligible patients, we were able to increase physician compliance to 75%. This project highlighted a major shortcoming in patient care that was greatly improved via EMR-based interventions. Further quality improvement interventions via EMR can be an easy and effective method for ensuring patient safety and improving outcomes.

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Improving hospital follow-up care for patient with acute heart failure, COPD exacerbation and pneumonia

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The Affordable Care Act established the Hospital Readmissions Reduction Program with the goal of reducing cost of excessive readmissions. Diagnoses under scrutiny included chronic obstructive pulmonary disease (COPD), heart failure and pneumonia. 30-day readmission rates for these diagnoses are 22.6%, 20%, and approximately 17% respectively(1-3) with annual cost totals in the billions. Data in heart failure shows that 7-day follow-up is associated with lower 30-day readmissions(4). Our health system tracks 7-day follow-up rates for acute heart failure (AHF), acute exacerbation of COPD (AECOPD) and pneumonia. Our residency clinic's 7-day follow-up rate was reported as 46.2%. The aim was to match the system goal of >55.9% 7-day follow-up.

A review of our process found multiple deficiencies regarding how high-priority follow-up appointments were facilitated. We conducted a chart review on our clinic's patients discharged to home with AHF, AECOPD and pneumonia over a 3-month period. This demonstrated a 7-day follow up rate of 66% with 71% having had follow-up scheduled for within 7 days. Over the next 9 months, we undertook three PDSA cycles. First, we educated our program on the need for 7-day follow-up for these diagnoses, then standardized post-discharge calls were coupled with a transitions of care (TOC) nurse rounding with the inpatient service 3 days per week, and finally the TOC nurse began rounding 5 days per week. Charts were collected and reviewed directly by the resident authors for accuracy. Chi-square analysis was used.

With education alone, we found no improvement in scheduling of appointments within 7 days, follow-up attendance within 7 days or readmission rates at 30 days. With the addition of a rounding TOC nurse 3 days per week and a standardized post-discharge phone call we found improvement in scheduling of follow-up within 7 days (86.7% vs 67.9%, p=0.027) but no improvement in attendance and higher readmission rate (33.3% vs 28.6%, p=0.022). Improved scheduling was sustained with the TOC nurse rounding 5 days per week, though again no change in the rate of follow-up attendance or readmissions was seen. Analysis of patients interacted with by the TOC nurse compared to those not interacted with demonstrated improvement in follow-up being scheduled within 7 days (88.1% vs 63.4%, p=0.004) but no improvement in attendance and higher readmission rates (35.7% vs 29.6%, p=0.010).

Our rates of follow-up for AHF, AECOPD and pneumonia were actually above our health system goal, likely due to the way the statistics are gathered. Our data showed that adding a TOC nurse to a busy resident inpatient service increased the percentage of appointments scheduled within 7 days. Work remains to improve actual follow-up rates, but the establishment of a TOC nurse dramatically improved our process of linking inpatient to outpatient care for all diagnoses and patients.

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Hypertension Control in a High Risk Population: Multidisciplinary QI Project

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Introduction: Hypertension (HTN) affects 1 in 3 Americans and is often poorly controlled, with 52% of hypertensive Medicare patients having uncontrolled hypertension (>140/90). Controlling HTN remains one of the most effective ways to reduce risk for cardiac disease and stroke. We report the outcomes of a two-year QI project that involved close multidisciplinary follow-up of uncontrolled HTN patients in a federally qualified, resident run clinic with the goal of increasing the percentage of patients with controlled HTN by 50% over 24 months.

Method: All patients presenting to the Five Rivers Health Center (FRHC; Dayton, Ohio) with diagnosed HTN were followed serially. Resident physicians were instructed to provide HTN education, prescribe antihypertensives and refer all patients with uncontrolled HTN (>140/90) for a two-week multidisciplinary visit for BP monitoring and further medication titration. Patients were provided with BP monitor logs with remote access monitoring. Patients with persistently elevated blood pressures were referred for repeat two week visits until control was achieved. The percentage of patients scheduled for a two week follow-up visit and that achieved blood pressure control were calculated every month.

Results: An average of 342 HTN patients were seen per month from January 2018 to January 2020; 53% were African-American and 51% males. Prior to the intervention, 10% of HTN patients had close follow-up appointments and 42% had well controlled HTN. After the intervention, 72% of patients had close follow-up appointments (620% increase, p-value <0.001) and 57% had achieved control (36% increase, p-value <0.005). Of the patients that achieved blood pressure control, 75% of patients with a two week follow-up visit were controlled in 6 months as opposed to 30% of patients without two week follow-up. In the uncontrolled group, heart failure (4.6%, 95% CI -3.80-16.94%, P=0.31), diabetes (7.9%, 95% CI -7.04-23.96%, P=0.320) and smoking (12.1%, 95% CI -3.92%-28.16, P=0.146) did not have a significant causal relationship.

Conclusion: Implementation of a two-year multidisciplinary QI project resulted in a statistically significant increase in the follow-up of HTN patients (10% pre vs 72% post) and in the percentage of HTN control (42% pre vs 57% post). In patients that gained BP control, participation in close multidisciplinary follow-up yielded faster control at six months. This QI project allowed for the highest level of BP control ever seen at FRHC, but ultimately fell short of the goal of increasing BP control by 50% in 24 months. Our next steps include involving pharmacy and social work to address the social determinants of healthcare. This QI project illustrates the power of close multidisciplinary follow-up in increasing hypertension control in a high risk population served by a federally qualified, resident run clinic.

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Limitations of MALDI-TOF in a Clinical Setting

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Background: Matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) is an integral part of rapid identification of causative organisms for bloodstream infections (BSIs). Compared to traditional microbiology methods, MALDI-TOF has been shown to decrease the time to organism identification on the order of 1.5 days. More rapid organism identification allows for decreased time to appropriate antimicrobial therapy. Multiple studies demonstrate that with the use of a sensityper, MALDI-TOF is able to identify 66%-100% of bacterial isolates on the genus level and 31%-94% on the species level. Variables suspected to impair MALDI-TOF identification include incomplete reference database, encapsulated bacteria, polymicrobic samples, and low inoculum infection.

Methods: This review consisted of 361 bacteria positive blood cultures collected from February to June of 2018 that underwent MALDI-TOF analysis. MALDI-TOF failed to identify an organism in 101 of these cultures. These 101 cultures were further evaluated to compare conventional microbiology results and to identify common variables that could explain the inability of MALDI-TOF to identify the bacteria.

Results: Of the 101 blood cultures that MALDI-TOF could not identify, 75 grew bacteria likely to be contaminants. There were 6 blood cultures that grew uncommon bacteria. Of the 101 cultures, 4 were polymicrobial. After stratifying the data by known limiting variables, 19 cultures did not fall into any of the aforementioned categories. Of these 19 cultures, 5 were culture positive for methicillin-resistant Staphylococcus aureus (MRSA) and 14 grew a variety of bacteria and had no readily apparent variable that could explain why the MALDI-TOF would not have been able to identify the bacteria in those blood samples.

Discussion: MALDI-TOF is a useful tool for rapid organism identification to assist in guiding antimicrobial therapy. In this analysis, we determined that MALDI-TOF failed to identify an organism in 101/361 (28%) of available cultures. While 82 of these were associated with variables that could explain the inability to identify the organisms, 19 remained with no explanation as to why the MALDI-TOF failed. Variables that could not be quantified in our study include poor blood sample or low inoculum infection at the time of sample collection. Having shown its ability to improve patient outcomes and reduce clinical costs, MALDI-TOF has great potential for clinical use. In order to expand the benefits of this technology, it is important to identify existing limitations in order to develop strategies to correct them.

Medical Student Clinical Vignettes

Abstracts 400-435

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Hydralazine-induced Liver Injury: A case study and review of the mechanisms of this rare side effect.

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Introduction: Hydralazine is a vasodilator that is used for hypertension management. Despite its well-known side effects, little is known about its hepatotoxic effects. We present a case of a middle-aged female who presented with a hydralazine-induced liver injury pattern and her constellation of symptoms, which progressively resolved following discontinuation of the medication.

Case Description: A 54 year-old Caucasian female with a past medical history of hypertension, stage three chronic kidney disease, asthma, Crohn's disease, and stroke presented to the office for hypertension management. Due to insurance issues, intolerance of certain antihypertensives, and suboptimal control of blood pressure, medication regimen was adjusted. She was subsequently switched from carvedilol to hydralazine 50mg three times daily with titration to 100mg after two weeks on the medication. Regimen also included triamterene 100mg daily and losartan 100mg daily.
Three days after increasing hydralazine, she presented to the emergency room for flu-like symptoms including fevers, chills, non-bloody diarrhea, myalgias and a dry cough. Labs and infectious disease work up was mostly unrevealing apart from transaminitis with elevated AST of 170 U/L and ALT of 155 U/L, but normal bilirubin and alkaline phosphatase. An EKG revealed sinus tachycardia which eventually resolved with IV fluid hydration. She was sent home with a diagnosis of a non-specific viral syndrome.
Five days later she had complaints of generalized malaise, fatigue, myalgias, right upper quadrant abdominal pain, darkened urine and exertional dyspnea. Fevers persisted despite taking acetaminophen every eight hours with temperatures reaching 102 Fahrenheit. Labs were again unrevealing apart from elevated transaminases, now with AST 249 U/L, ALT 519 U/L, and alkaline phosphatase 282 U/L. An acetaminophen level was undetectable. An acute hepatitis panel was negative for infection as was a second COVID19 test. An abdominal ultrasound revealed hepatic steatosis, but no gallstones, dilated bile ducts or wall thickening was seen. Further studies revealed a negative ANA, AMA, anti-smooth muscle antibody, LKM antibody and anti-Histone antibody levels. Immunoglobulin, ceruloplasmin and ferritin levels were normal. Testing for viral infections was negative for EBV, CMV, and HSV. An MRCP demonstrated a normal pancreas and biliary tract with only small side-branch intraductal papillary mucosal neoplasms (IPMNs) and hepatic steatosis.
Following stabilization and follow up with Internal Medicine, her hydralazine was discontinued. Six weeks following discontinuation, her liver enzymes normalized completely. After consultation with gastroenterology, it was determined that her symptoms resulted from a hydralazine-induced liver injury, a rare condition. She returned to her baseline state of health with no liver biopsy performed.

Discussion: Few case reports and studies have examined the mechanism by which hydralazine induces a liver injury pattern. With this case, we highlight a clinical scenario that led to this finding, the pathogenesis involved, and the eventual management of this condition.

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Full Throttle: Immune Checkpoint Inhibitors and Risk Stratification of Immune Related Adverse Events, A Case Series

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Introduction: Immune modulating therapies targeting CTLA-4, PD-1 and PD-L1 have recently emerged as effective treatments for a variety of cancer types, however; Immune Related Adverse Events (irAEs) may limit the use of these effective therapeutic modalities.

Case 1: A 72-year-old woman with lung adenocarcinoma presented with two weeks of worsening exertional dyspnea and fatigue after finishing her 9th cycle of Pembrolizumab. On arrival, her pulse oximetry was 74% and decreased to 69% after ambulating a short distance. Computerized Tomography (CT) abdomen and pelvis demonstrated development of extensive airspace consolidations throughout the left lung , right upper, and right lower lobes suggestive of multifocal pneumonitis, likely related to irAE pneumonitis, CTCAE grade 2/4. Patient was started on solumedrol 120 mg and empiric antibiotics. Infectious workup returned negative. Over the course of her 3-day hospitalization, patient expressed symptomatic improvement with steroids. Patient was weaned off of oxygen and discharged home with a pulse oximeter, oral antibiotic therapy and a 6-week steroid taper.

Case 2: A 36-year-old woman with metastatic melanoma to bone, liver, and lymph nodes treated with immunotherapy (pembrolizumab and ipilimumab) presented to the infusion clinic with a chief complaint of worsening left lower abdominal pain and more than four watery bowel movements a day. Infectious work up returned negative. CT abdomen and pelvis demonstrated pancolitis consistent with irAE colitis, CTCAE grade 2/4. Patient was started on 80 mg solumedrol. Patient improved symptomatically and was discharged home with plans to taper steroid dose on an outpatient basis.

Discussion: These cases presented to a single comprehensive cancer center within a two-week period and illustrate the prevalence of irAEs. The most common irAEs are colitis and pneumonitis, but other involved sites include skin, liver, muscle, pituitary gland and heart. IrAEs are graded 1-4 from asymptomatic to life threatening with treatment recommendations escalating with increasing grade.

Preliminary studies have shown that tumors with higher mutational burdens, such as non-small cell lung cancer and melanoma, are associated with a higher likelihood of irAEs. These tumors may have a higher antigenic load and may spark cross reactivity with native wild-type tissue. Pre-existing rheumatologic disease, solid organ transplantation, allogenic stem cell transplant therapy, HIV status and viral hepatis may be risk factors for development of irAEs. Outcomes did not differ between patients with advanced melanoma who discontinued therapy due to irAEs and those who continued therapy. This suggests that cancer patients will still benefit from a trial of the immune-modulating therapy even if therapy is discontinued due to irAEs. Identifying genetic, epigenetic, microbiomic, tumor, and host immunological markers may guide criteria to risk-stratify patients' chances of developing high grade irAEs and severe long term sequalae.

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Multiple Etiologies of Pulmonary Hypertension

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A woman in her 40s with a complex medical history presents with worsening dyspnea on exertion. She has systemic lupus erythematosus complicated by lupus nephritis, nephrotic syndrome, secondary Sjogren's syndrome, and fibromyalgia. In addition, she has recurrent pulmonary emboli, multiple episodes of pneumonia, sickle cell trait, and asthma. Notably, she has had a stable, large pulmonary embolus in her right lower pulmonary artery for several years. The patient's previous right heart catheterization 2 years prior demonstrated elevated pulmonary artery pressures consistent with pulmonary hypertension. However, a pulmonary angiogram was not performed at this time because the patient was lost to follow-up.

During the present admission, the patient underwent another right heart catheterization, which demonstrated worsening pulmonary hypertension as well as signs of right heart enlargement and decreased ejection fraction. In addition, a pulmonary angiogram was simultaneously performed. It revealed the major etiology of her pulmonary hypertension was small vessel disease secondary to lupus and not chronic thromboembolism. She was started on an infusion of epoprostenol, a synthetic analogue of prostacyclin.

Determining the appropriate etiology of pulmonary hypertension is crucial since there are vastly different treatments for each etiology. Pinpointing a specific etiology can be difficult when a patient has multiple comorbidities that can each contribute to the pathogenesis of pulmonary hypertension, such as in this case. Both the patient's lupus and chronic pulmonary embolism could be likely etiologies. Lupus can cause small vessel disease in which endothelial dysfunction results in hyperplasia of both the intima and media. Chronic pulmonary emboli result in vascular remodeling secondary to increased shear forces in occluded vessels. A pulmonary angiogram can distinguish between the two etiologies, as the pulmonary vascular resistance will be markedly elevated relative to the degree of obstruction in pulmonary hypertension secondary to small vessel disease.

Pulmonary vasodilators such as prostacyclin analogous have the most evidence in treating pulmonary hypertension caused by small vessel disease. Epoprostenol results in pulmonary vasodilation through both direct and indirect mechanisms, in addition to having anti-inflammatory and anti-thrombotic properties. Unfortunately, those with an underlying connective tissue disease have a worse prognosis than those with idiopathic pulmonary hypertension. If the patient's chronic pulmonary embolus was determined to have been the major etiology, a thromboendarterectomy would have been indicated instead.

Pulmonary hypertension has a diverse range of etiologies. A thorough and complete evaluation to determine the primary etiology is necessary to provide proper treatment.

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A False-Positive HIV Test Revealed on Fecal Microbiota Transplantation Donor Screening

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Fecal microbiota transplantation (FMT) has gained popularity as a therapeutic option for recurrent CDI refractory to antibiotic therapy, with a recent meta-analysis citing success rates as high as 92%1. Patients with refractory CDI receiving FMT have a decreased CDI-related mortality (20.0% vs 82.4%) and all-cause mortality (20.0% vs 94.1%) when compared to patients treated with routine antimicrobial therapy2. Due to similarities in microbiota, selecting a family member as a FMT donor can be advantageous. However, the screening process may present hereunto unconsidered ethical ramifications. We describe a case of a false-positive HIV test revealed on routine FMT donor screening..

A 54-year-old male was identified as an appropriate potential donor for fecal transplantation. His father, hospitalized for a fourth recurrence of C. difficile colitis after multiple failed antibiotic courses, was referred for FMT. Upon routine screening to evaluate his suitability as a donor, the son's HIV p24 antigen returned positive. The son was in a monogamous relationship with his wife and had no risk factors for HIV infection. Confirmatory testing using a Multispot HIV-1 and HIV-2 antibody differentiation assay returned negative, and subsequent HIV viral load was negative. The son was then counseled that he had a false-positive HIV test.

As FMT is a relatively new treatment modality, new ethical considerations are raised regarding how potential donors with positive screening tests are best handled. In this case, the donor received a false positive HIV test during the FMT screening process. This imposes significant burden including potential isolation from spouse or family and significant stigma. In addition, the physician must inform the father of his son's incompatibility as donor while protecting the son's autonomy.

Procuring stool from a stool bank from pre-screened donors can cost between $1500 and $20003. Selecting a family member as donor is an encouraging alternative. Screening any potential donor for transmittable infectious diseases is an important step before donor selection. Several organizations have provided screening recommendations for donors, but no evidence-based guidelines have been developed; although, HIV testing has been recommended by all organizations4. However, those ordering testing in this circumstance may not be well-versed on counseling after a positive screening test. Current HIV testing has an extremely high specificity; however, in populations of low prevalence the positive predictive value is extremely low5.

Many obstacles prevent FMT from becoming mainstay treatment for CDI. Challenges include donor recruitment and consistent regulation of donor stool screening4. Utilization of stool banks would have avoided this ethical dilemma. They provide a reliable donor and spare time spent undergoing pre-screening to improve the quality of recurrent CDI treatment for patients while avoiding the risk of false positive tests and the associated psychological harm to their family members.

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Loperamide Toxicity in Opioid Addicts: A Potential Treatment Option

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Introduction

Over past decade, the incidence of loperamide toxicity has increased alarmingly among opioid users. Loperamide is a widely available over-the-counter anti-diarrheal that works primarily on intestinal opioid receptors. However, when taken in very high doses, it crosses the blood-brain barrier and produces euphoric effects similar to opioids making it popular as "poor man's methadone." We present a case of loperamide toxicity in a young patient with possible clinical improvement after therapeutic plasma exchange (TPE).

Case Presentation

A 25-year-old female with history of Brugada syndrome, paroxysmal ventricular tachycardia and IV opioid abuse presented with fever and altered mental status. She was taking 200-300 mg/day of loperamide for the past several weeks to cope with opioid withdrawal symptoms. Physical exam revealed confusion, dilated pupils and clonus. Labs revealed a WBC count of 20,000/uL, lactic acid 6.5 mmol/L, magnesium 1.3 mEq/L, positive influenza B, negative serum tox screen but positive benzodiazepine and buprenorphine on urine screen. EKG showed a type 1 Brugada pattern with a markedly elevated QTc interval of 693 msec. She developed progressive bradycardia with prolongation of QTc (761 msec) and suffered a cardiac arrest 24 hours after admission with return of spontaneous circulation after CPR. Serum loperamide and its inactive metabolite (desmethylloperamide) levels were high at 19 ng/mL (therapeutic level ~3 ng/mL) and 210 ng/mL respectively. TPE was initiated 44 hours after admission, resulting in improved QTc and decreased loperamide and desmethylloperamide serum levels. A second TPE session was administered at 68 hours leading to improvement, extubation and discharge home on suboxone.

Discussion

The role of TPE in drug toxicities is still unclear and, to the best of our knowledge, it has not previously been used for the treatment of loperamide toxicity. For TPE to be effective in rapid removal of a drug, it must be highly protein bound (>80%) and have a low volume of distribution (Vd < 0.2L/Kg of body weight). Loperamide is 95% protein bound and has very low bioavailability (0.3%) after oral ingestion. In our patient, serum loperamide level decreased from 19 ng/mL to 9 ng/mL after 1st session of TPE (1.5 plasma volume) over a period of 24 hours. However, it was unclear whether this could be attributed to plasmapheresis or was simply related to loperamide's normal half-life elimination. Nonetheless, the patient showed evidence of significant clinical improvement and her QTc interval improved with resolution of bradycardia and arrhythmias. In conclusion, loperamide toxicity is becoming increasingly common among opioid addicts and may result in life-threatening cardiac arrhythmias. Due to its favorable pharmacokinetics, loperamide can potentially be removed more rapidly with the help of TPE. Further studies are warranted to explore the use of this modality in the treatment of loperamide and other drug toxicities.

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A Surprise Co-infection: Neurocysticercosis meets Fusobacterium Pyogenic Liver Abscess

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Fusobacterium, an anaerobic gram-negative bacterium, can be an infectious pathogen in Lemierre's syndrome, bacteremia, or pyogenic liver abscess, particularly in patients with poor dentition. Neurocysticercosis, a CNS infection caused by Taenia solium larvae, is endemic to Mexico and Central America; most cases in the United States are seen in Latin American immigrants or near the southern border with Mexico. Bacteremia and parasitic co-infection is exceedingly infrequent.

An 85-year-old male presented with progressive dyspnea, generalized weakness, altered mental status, and intermittent fevers for one week. He had dental extractions ten days prior to admission. Four years earlier, the patient traveled to Southern Texas for several weeks. Examination revealed an afebrile, tachypneic male oriented only to person with diminished breath sounds bilaterally and right upper quadrant abdominal tenderness. Laboratory testing revealed leukocytosis of 36 x10^9/L, bandemia of 16.0%, thrombocytopenia, acute kidney injury, and elevated LFTs. CT of the abdomen and pelvis revealed a large heterogeneous liver lesion in the medial left lobe measuring 13 centimeters transversely. He subsequently became febrile, and he was intubated and placed on antibiotics. The hepatic abscess was drained; aspirate cultures and blood cultures revealed Fusobacterium nucleatum. Antibiotics were de-escalated to ceftriaxone and metronidazole. He remained obtunded upon weaning from sedation. An MRI of the brain revealed innumerable ring-enhancing lesions throughout the cerebral hemispheres, brainstem, and cerebellum. Our differential diagnosis included neurocysticercosis and disseminated Fusobacterium abscesses. An MRI from three years earlier lacked these lesions. Toxoplasmosis and HIV serologies were negative. A transesophageal echocardiogram revealed no vegetation. Neurosurgery declined biopsy due to the innumerable lesions and poor prognosis. The patient continued to deteriorate, and family declined empiric antiparasitic coverage with albendazole and praziquantel. The patient was terminally extubated, transferred to hospice, and expired. Serology for Taenia solium IgG antibodies then returned positive, suggesting neurocysticercosis as the likely cause of the brain lesions.

We report a patient with Fusobacterium nucleatum pyogenic liver abscess and bacteremia, with multiple ring-enhancing lesions subsequently identified on MRI of the brain. The Fusobacterium nucleatum bacteremia was likely related to his recent dental procedure, and Fusobacterium has been described as an etiology of ring-enhancing brain lesions. However, he had traveled to an area endemic for neurocysticercosis four years earlier. When considering the median incubation period for cysticercosis is 3.5 years prior to symptom onset, and pairing it with the numerous characteristic ring-enhancing lesions and confirmatory serology for Taenia solium, neurocysticercosis emerged as the more likely diagnosis of his brain lesions. This case highlights the importance of obtaining a careful travel history in composing a differential diagnosis. As the patient's prognosis was poor and outcome from treatment was uncertain, the family opted for hospice.

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Hemoptysis in a 75 Year Old Female

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Case Presentation: A 75 year old woman with history of invasive left breast ductal carcinoma presented with hemoptysis. She denied chest pain, fevers, or shortness of breath. Two previous episodes of hemoptysis, several years prior, were presumed secondary to a sinus infection. Patient was afebrile and had a normal lung exam. CBC was normal. CT angiogram was negative for PE, but revealed right upper lobe ground-glass opacities in a "tree-in-bud" pattern and bronchiectasis, as well as middle lobe atelectasis and volume loss. She was diagnosed with atypical pneumonia and discharged on levofloxacin. The patient's cough persisted, but she had no further hemoptysis.
Pulmonary testing was ordered to work up patient for ABPA, immunodeficiency, and cystic fibrosis. Aspergillus (fumigatus, flavus and niger) antibodies were negative. All immunoglobulins were within reference ranges. Endobronchial US showed mediastinal and hilar lymphadenopathy, and edematous mucosa of the R lower lobe and lingula, with no endobronchial lesions or secretions. Bronchoalveolar lavage had no malignant cells, but grew Bordetella bronchiseptica. Sweat chloride test was 63 mol/L (normal <30, indeterminate 30-59), and CF carrier study was ordered. Results were negative for 32 analyzed mutations, and a repeat sweat chloride test was ordered. Second positive sweat chloride test, 66 mol/L, confirmed diagnosis of CF with bronchiectasis. Subsequent genetic testing showed a single CFTR mutation of "unclear clinical implication."

Discussion: The average age of diagnosis of CF is 4 years old, with 75% of patients diagnosed by age 2. However, due to the over 1,700 known CFTR gene mutations, there is a wide spectrum of disease presentation. Patients who present with atypical CF tend to have milder symptoms, leading to later diagnosis. For those presenting in adulthood, respiratory symptoms tend to be mild or intermittent, including chronic sinusitis or recurrent pneumonia. Additionally, those with atypical CF tend to have a lower prevalence and more subtle presentations of CF-related diagnoses—such as pancreatic insufficiency. When there is clinical suspicion of CF, evidence of CFTR dysfunction is required for diagnosis: two CF mutations, two positive sweat chloride tests or CF-consistent nasal potential difference testing. For this case, the two positive sweat chloride tests confirmed CF despite the finding of only one CFTR mutation. Atypical CF patients may have normal results on sweat chloride tests and nasal potential difference testing. Furthermore, genetic analysis may be normal, as testing is limited by the number of mutations screened. The combination of atypical symptoms and normal test results may increase the difficulty of diagnosing atypical CF. Due to the spectrum of disease presentations and possibility for atypical symptoms, CF should be considered in adults with sino-pulmonary symptoms and unexplained bronchiectasis.

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Autoimmune myelofibrosis presenting as pancytopenia in a patient with systemic lupus erythematosus

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A 48 year old woman with a history of SLE, Antiphospholipid syndrome, and Immune Thrombocytopenic Purpura presented to the emergency room with fatigue and syncope. She had been recently hospitalized for syncopal episodes with anemia and thrombocytopenia. During this past hospitalization, blood counts normalized following transfusion and she was discharged without steroids due to a co-occurring GI bleed. On presentation of the current episode, she was hypotensive with anemia (hemoglobin 6.6 g/dL), thrombocytopenia (platelet 23 K/uL), and a WBC count of 8.23 K/uL. Early labs were inconsistent with hemolysis, with an elevated haptoglobin of 261 mg/dL, LDH of 175 U/L, low reticulocyte index of 0.4, and normal total and direct bilirubin values. Peripheral blood smear demonstrated teardrop cells and neutrophils with Pelger-Huet anomaly. The patient's hospital course was complicated by Pseudomonas putida bacteremia. She also received multiple red blood cell transfusions due to symptomatic anemia, and she was persistently thrombocytopenic.

Due to concern for a primary myelodysplastic syndrome, a bone marrow biopsy was performed. Results showed myelofibrosis with increased megakaryocyte precursors, decreased erythroid genesis, and no blast cells. Follow up testing for JAK2, CALR, and MPL exon 10 mutations were negative, and abdominal ultrasound did not demonstrate splenomegaly. The patient's symptoms were attributed to lupus-related autoimmune myelofibrosis, a rare cause of pancytopenia that is reported to respond to treatment of the underlying autoimmune disease.
Following antibiotic treatment for bacteremia, the patient was started on a course of high-dose steroids, leading to subsequent improvement in cell counts. Over the course of the hospitalization, platelet counts improved from 12 K/uL to 71 K/uL and hemoglobin improved from 6.1 g/dL to 10 g/dL. WBC counts increased to 25 K/uL, though it is unclear if this was secondary to steroid treatment or sepsis from an unknown source. Once medically stable with improving cell counts, the patient was discharged with a two-week course of steroids and a four-week course of low dose methotrexate.

Although autoimmune myelofibrosis is a rare phenomenon, it is important to recognize it as distinct from primary myelodysplastic syndrome. The pathophysiology is not fully understood, but one proposed mechanism for fibrosis involves immune complex-mediated activation of megakaryocytes with subsequent release of platelet derived growth factor and fibroblast activation. While further investigation may inform future treatment of myelofibrosis, current treatment approaches tend to focus on addressing the underlying autoimmune disease. Further, although this patient has a well-documented history of SLE, previous case reports have identified pancytopenia due to autoimmune myelofibrosis as the primary presenting symptom of an autoimmune disease. For this reason, it is important for providers to not only recognize myelofibrosis as a potential complication of known autoimmune disease, but also an atypical cause of pancytopenia that would require further hematologic workup.

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Bartonellosis Presenting as Branch Retinal Artery Occlusion

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Introduction: Bartonella henselae is the causative agent of cat scratch disease (CSD). This disease tends to occur in immunocompetent hosts and is typically characterized by regional lymphadenopathy, with approximately 12,000 cases diagnosed annually. Cats serve as the natural reservoir for B. henselae. Following inoculation of the organism into humans, B. henselae typically causes a local infection that manifests as local lymphadenopathy. In some individuals, the organism can disseminate and infect the liver, spleen, eye or central nervous system. Patients with localized disease generally have self-limited illness, while those with disseminated disease can have life-threatening complications. We present a patient with CSD that presented with acute, painless vision loss.

Case description: A 30-year-old female developed acute painless scotoma in her left visual field, followed by blurry vision in the right eye on the next day. Her vision loss was painless, and no associated redness or drainage was noted. An ophthalmology evaluation revealed a left branch retinal artery occlusion and right-sided retinal hemorrhage; there was no evidence of macular pathology in either eye. She presented to the emergency department, whereupon a brain MRI with and without contrast and an MRV head and MRI orbits with and without contrast were negative for an acute cerebrovascular accident. Hypercoagulable workup revealed a positive heterozygous prothrombin g20210a mutation. She was initiated on dual antiplatelet therapy with aspirin and clopidogrel with plans to perform cerebral angiography as an outpatient as her visual symptoms subsided on their own.

Further history revealed she had been caring for 17 stray cats and had been bitten and scratched multiple times in the previous several months; none of these cats had been evaluated by a veterinarian. She noted no tender nodules near the bite and scratch sites. She developed worsening generalized weakness, fatigue, lightheadedness and poor appetite. B. henselae IgG titers were elevated at 1:1024 with normal B. henselae IgM titers at 1:16. She was treated with a four-week course doxycycline and rifampin. Her initial course was complicated by persistent headache and dizziness, but her symptoms resolved with completion of treatment.

Discussion: Ocular complications of bartonellosis are rare but can be devastating if left undiagnosed. Ocular involvement typically presents as neuroretinitis; examination generally reveals macular exudate and retinal hemorrhage. Branch retinal artery occlusion secondary to bartonellosis is even more rare, occurring in ~1-2% of patients with bartonellosis, and usually presents with acute, painless, monocular vision loss. If left untreated, ocular bartonellosis with neuroretinitis may be complicated by retinal vascular occlusion causing permanent visual loss. Obtaining a careful history, including animal contacts, can prevent severe complications and reduce diagnostic error.

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Failure to identify a retropharyngeal foreign body leading to anoxic brain injury

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Introduction: Chicken and fish bones are the foreign bodies most commonly ingested by accident in adults [1]. Therefore, it is important to make sure to utilize the most efficient imaging modalities to identify these two particular foreign bodies in the head and neck with regularity in order to prevent adverse outcomes.
Case Presentation: A 66-year-old male presented to the Emergency Department at an outside hospital reporting a scratching sensation when he swallowed and concern that he may have ingested a bone. Per patient history, he recently consumed both chicken and fish. He denied dyspnea or dysphagia at initial presentation. X-rays of the neck were performed and were read as normal with no foreign bodies identified; no further imaging was conducted. Three days later, the patient returned to the same Emergency Department diaphoretic and in acute distress. He endorsed significant neck pain, fullness of the throat, and was unable to clear his own secretions. The patient was now dyspneic and could not lie flat for CT imaging. New X-rays were performed which demonstrated air in the retropharyngeal space and leukocytosis was present on the CBC. Emergent airway management was deemed necessary and the patient was taken to the OR for sedation and emergent airway placement. He was, per report, hypoxic for approximately 20 minutes as the emergency tracheostomy was performed. The patient was unresponsive following the procedure. CT imaging of the neck was performed, and the patient was subsequently transferred to the closest tertiary care center. The CT images showed extensive soft tissue swelling and a prominent curvilinear density at the level of the hyoid bone worrisome of a foreign body. Swelling prevented ENT physicians from visualizing and removing the potential foreign body. ENT also noted the development of a retropharyngeal abscess. A multidisciplinary approach by the Surgical ICU, ENT, and Neurology teams took place over several weeks, but the patient remained unresponsive and continued to require mechanical ventilation. Swelling was reduced with medical management and the retropharyngeal abscess was eventually identified and drained. However, no foreign body could be recovered. Neurology diagnosed the patient with anoxic brain injury and encephalopathy. He was eventually transferred to a long-term acute care facility.
Conclusion: Previous studies have shown a much higher and more consistent detection rate of chicken and fish bones in the head and neck by both CT imaging and direct endoscopic visualization compared to plain radiography [2-4]. This case highlights the importance of using these further imaging modalities in cases involving potential ingestion of either of these foreign bodies.

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NOTHING TO SNEEZE AT: PASTEURELLA PNEUMONIA IN A HEALTHY CAT OWNER

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Introduction: Pasteurella multocida is a zoonotic bacterium that is most commonly associated with cutaneous infections following domestic animal bites or scratches. We present a case of Pasteurella pneumonia in the healthy owner of a sneezy cat with no known cutaneous portal of entry.
Case Presentation: A 61-year-old healthy non-smoking, active female presented with a persistent cough from a URI 6 weeks prior, new left pleuritic chest pain, and low-grade fever. Although the lung exam was benign, CXR revealed consolidation in the lingula. She completed a 10-day course of moxifloxacin but re-presented approximately one week later for continued chest tightness, worsened fatigue, and occasional dry cough. Physical exam and EKG were unremarkable, with repeat CXR showing improved consolidation in the lingula. After six weeks without intervention, her cough worsened to 2-3 coughing spells daily, productive of minimal yellow sputum. At that time, CXR showed non-specific RML and lingular airspace disease, and she was referred to pulmonology for further work-up. CT showed a centrilobular distribution of micronodules and subsegmental atelectasis in the RML and lingular segment, suggestive of TB or an atypical mycobacterium; however, cultures from BAL grew Pasteurella multocida, confirming the diagnosis. The patient recovered following a 7-day course of levofloxacin.
Discussion: P. multocida infection can present in a variety of ways, including cellulitis, osteomyelitis, pneumonia, and meningitis, with recent animal bites or scratches nearly always leading the clinical work-up. Pneumonia is the most common respiratory infection caused by P. multocida and is characterized by non-specific symptoms such as fever, malaise, dyspnea, and pleurisy. The majority of cases have been documented in the elderly (>65), immunocompromised, or those with chronic lung disease; however, few if any are preceded by direct animal exposure. Review of the literature reveals several case studies that refer to Pasteurella pneumonia as the “cat cuddler’s cough” or “pig trotters lung” and suggest likely indirect airborne transmission from the domestic animal in question. The pathophysiology of infection is described first by inhalation of aerosolized particles of organism, colonization of the oro- or nasopharynx, and subsequent aspiration of the secretions into the lungs. With cats having P. multocida carriage rates of 70-90%, it is feasible that a sneezy cat could create an environment teeming with potential for human airway colonization. Regardless, Pasteurella pneumonia is extremely rare in middle-aged immunocompetent patients with benign exposure histories, such as our patient. Thus, even in healthy patients, it is important for clinicians to consider P. multocida as an airborne pathogen that can cause ambulatory pneumonia.

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A NOVEL TREATMENT FOR BURNING FEET. UNDERSTANDING PATHOPHYSIOLOGY IS KEY

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Introduction: Erythromelalgia is an extremely rare disease which affects blood flow to the distal extremities causing debilitating episodes of burning pain. Thus far, therapeutic options have been limited and varied in efficacy. We present a case of erythromelalgia in a 51-year-old female who was treated successfully with an infusion of epoprostenol and subsequently maintained on sildenafil.

Case Presentation: A 51-year-old female with a history of autoimmune hepatitis, SLE, and Raynaud's presented with daily episodes of redness, swelling and pain of her bilateral lower extremities which was progressively worsening in severity and frequency. She was diagnosed and treated for erythromelalgia but symptoms worsened despite trials of topicals, aspirin, Lyrica, nortriptyline, and propranolol. She also complained of decreased exercise tolerance, worsening skin lesions on her toes and difficulty sleeping due to exacerbations at night.
Having failed prior therapies, she was admitted for an IV epoprostenol infusion. She was initiated on 2ng/kg/min of epoprostenol and titrated up to 9ng/kg/min over six days. She tolerated the treatment well and was discharged on phosphodiesterase inhibitors (PDE5-I) therapy with sildenafil for maintenance. At 1 week follow up, she reported improvement in sleep quality and reduced frequency and severity of daily flares. At 1 month follow up, she reported continued improvement in symptoms, most notably a reduction in the skin lesions, but continued daily lower intensity flares.

Discussion: Erythromelalgia is a rare disease characterized by intense, burning pain with erythema, most commonly of the distal extremities. Symptoms are debilitating as these episodes occur multiple times daily and are brought on by mundane stimuli such as warm water, blankets, or shoes.
The pathophysiology is still unclear and likely multifactorial, but vascular changes are an important component. During the episodes, blood flow to the extremities is increased which has been shown via Doppler imaging. Paradoxically transcutaneous oximetry remains unchanged or decreased. This implies that large portions of blood are being shunted via microvascular anastomoses leading to skin hypoxia, which causes further arteriolar dilation.
Substances that augment cutaneous blood flow, such as prostacyclins and PDE5-I, can improve oxygenation of the skin. There has been success treating erythromelalgia with infusions of the prostacyclin analog, iloprost, and the oral PGE1 analog, misoprostol. However, these are not first line treatments given the side effects of prostacyclins. There have been no previous reports of sustained symptom relief with chronic PDE5-I maintenance therapy, or maintenance therapy with sildenafil. Our patient tolerated the prostacyclin infusion and sildenafil well. She reported alleviation of symptoms including reduced frequency of flares, improved sleep quality, and decreased skin lesions. Patients with erythromelalgia that have failed multiple first line treatments should consider treatment with a prostacyclin or prostacyclin analogue followed by continued oral vasomodulating therapy with sildenafil.

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Optic Neuritis: A Manifestation of Many Possible, and Occasionally Multiple, Etiologies

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Introduction: Optic neuritis is defined as inflammation of the optic nerve as characterized by blurry vision, sudden vision loss, color blindness, visual field defects, and painful eye movements. It is diagnosed clinically and can be seen on ophthalmoscopy showing papilledema. The most common etiology of optic neuritis in adults is multiple sclerosis (MS), but it can also be seen in neuromyelitis optica, pseudotumor cerebri, sarcoidosis, lupus, infections (ex: Lyme, syphilis), and many others.

Case Presentation: A 42-year-old morbidly obese African American woman with a history of migraines, hypertension, and type 2 diabetes mellitus presented to the ED for four-day history of blurred vision and worsening vision loss of her right eye. She first noted that her vision was less sharp than usual, and progressed to complete loss of her right lower quadrant vision. She also reports discomfort with lateral gaze and associated photophobia and frontal headache. She denies any fever, chills, nausea, vomiting, abdominal pain, or dizziness. Of note, patient states that she was pregnant at age 17 but had an abortion, and believes that she recently had a miscarriage.
On admission, patient's exam was remarkable for inferior altitudinal hemianopia and Marcus-Gunn pupil on the right. Ophtho exam showed bilateral optic disc edema and inferonasal > inferotemporal vision loss in the right. MRI head showed enhancement of the right retrobulbar optic nerve and nonspecific multiple white matter signal abnormalities. CT chest revealed no pulmonary nodules or adenopathy. Lumbar puncture (LP) showed elevated opening pressure of 32 cm/water, presence of gamma oligoclonal bands, elevated total protein, normal glucose, and culture without PMNs or organisms. Labs showed ESR 83, normal complement levels, and negative ANA, anti-cardiolipin Ab, Lyme Ab, VDRL, and aquaporin-4 receptor Ab.
Neurology determined etiology to be most likely pseudotumor cerebri given her elevated opening pressure on LP, on a background of MS given the presence of white matter changes on MRI and oligoclonal bands on LP. Patient was given pulse IV solumedrol followed by oral steroid taper, as well as acetazolamide with improvement but not complete resolution. Repeat LP and ventriculoperitoneal shunt were considered but deemed not necessary at this time. Patient was discharged home with steroid taper and acetazolamide to follow up with neurology outpatient.

Discussion: This case illustrates the difficulty and complexity of establishing the etiology of optic neuritis in the context of competing differential diagnoses with widely varied and non-specific overlapping presentations. Although MS is the most common cause, a comprehensive workup involving imaging, LP, autoimmune panel, and infectious diseases labs is necessary to rule out other causes. Most importantly, patient should be closely followed up to address response to selected treatment and changes in presentation over time that may elucidate the correct diagnosis.

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Hemorrhagic stroke and persistent fevers, an unusual presentation of pheochromocytoma

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Introduction: Pheochromocytomas are rare catecholamine-secreting neoplasms and present in just 0.2% of hypertensive patients. The classic triad of episodic headaches, sweating, and tachycardia is often described in medical education. However, most patients with pheochromocytoma do not have these classic symptoms. The following case illustrates the importance of recognizing atypical presentations of this rare but potentially lethal condition.
Case Description: A 70-year-old-man with a history of hypertension presented with acute-onset right-sided hemiplegia and aphasia. Computed tomography of the brain without contrast demonstrated a large left parietal cerebral hemorrhage with intraventricular extension, as well as subdural hematoma and subarachnoid hemorrhage. Initial blood pressure was 176/133 and was controlled with nicardipine; later the patient was transitioned to carvedilol, amlodipine, and losartan. He developed a temperature of 39.7 degrees on the fourth hospital day, initially thought to represent central fevers secondary to the intracerebral hemorrhage. His hospitalization was complicated by aspiration pneumonia, for which he received a seven-day course of antimicrobials, and yet his fever persisted. As part of the workup for fever of unknown origin, computed tomography of the abdomen and pelvis revealed a multiloculated cystic lesion which appeared to contain enhancing septation in the right upper quadrant inseparable from portions of the right adrenal gland and right kidney. An MRI of the abdomen showed a right sided retroperitoneal mass that appeared to extend from the right adrenal gland. Evaluation of this adrenal incidentaloma demonstrated markedly elevated plasma free fractioned metanephrines at 468 pg/mL (12-67, up to 72 in a hypertensive adult) and free normetanephrine at 633pg/mL (18-101 normotensive, up to 145 in a hypertensive adult), which confirmed the diagnosis of pheochromocytoma. He was transitioned to prazosin but was not a candidate for surgical resection. The patient was discharged to rehabilitation on hospital day 12 but remained persistently febrile on discharge.
Discussion: This case illustrates the potential for pheochromocytoma to present as a hemorrhagic stroke in the setting of severe hypertension. Pheochromocytoma has also been described as a cause of fever of unknown origin, with fevers often persisting for several months after diagnosis. Stroke is a leading cause of death in the United States and is most commonly attributed to systemic conditions such as hypertension and diabetes, with pheochromocytoma comparatively less common as a potential etiology. Recognition of pheochromocytoma presenting as a stroke is critical to the prevention of iatrogenic harm. In this patient, beta-blockers were initially offered as part of the regimen to control hypertension prior to the recognition of the pheochromocytoma; unopposed alpha-adrenergic tone resulting from administration of beta-blockers for hypertension following hemorrhagic stroke can worsen outcomes.

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Aspiration Pneumonitis and Acute Renal Failure in a Patient with Severe Malnutrition

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Introduction: Aspiration pneumonitis is a chemical injury of the lungs caused by macroaspiration of noxious liquids. Reports in the literature and animal models describe progression of aspiration pneumonitis to ARDS and nonpulmonary organ failure. Patients with severe protein-calorie malnutrition are at higher risk of aspiration and complications of aspiration-related lung injury due to higher incidence of dysphagia and tenuous physiological status. Recognition of and management of risk factors for aspiration as well as distinguishing between aspiration pneumonitis and aspiration pneumonia (i.e. acute and subacute aspiration events) will help to reduce adverse outcomes in this patient population.

Case: A 35-year-old woman was admitted for severe, acute-on-chronic protein malnutrition secondary to restrictive eating patterns (BMI 11.4). Her care plan included IV electrolyte repletion and PO nutrition after patient request to discontinue trial of nasogastric tube feeding. On the evening of hospital day 10, agitation and urinary retention were observed and managed with straight catheterization and 0.25mg Ativan IV. The following morning, the patient was found hypotensive with increased respiratory effort and mixed acidosis. During intubation, gastric suction and pulmonary suction produced contents of the same appearance, and CXR showed near complete whiteout of bilateral lung fields, consistent with aspiration pneumonitis.
In the MICU, pressors, broad-spectrum antibiotics (Vancomycin + Zosyn due to suspicion for aspiration pneumonia) and stress dose steroids were started. The patient's respiratory status improved, and by hospital day 13 she was extubated. On hospital day 15, the patient decompensated again with increased oxygen demands, mixed acidosis, profound hypotension, oliguria, and worsening elevations of BUN and creatinine. The patient was transitioned to comfort care and expired on hospital day 16.

Discussion: Patients who suffer from severe protein-calorie malnutrition are known to experience dysphagia due to pharyngeal muscle weakness. Two case studies describe benefits of swallow evaluation and recommends the use of feeding tubes or thickening agents for such patients.
Gastric aspiration, which is the most common cause of aspiration pneumonitis, frequently occurs in patients who are critically ill, have altered mental status, or are sedated. 1/3 of patients with aspiration pneumonitis progress to ARDS and are more vulnerable to nonpulmonary organ failure. Supportive treatments are the mainstay of aspiration pneumonitis treatment, in contrast to antimicrobial therapy for aspiration pneumonia. However, the two conditions are difficult to distinguish in the acute setting without a witnessed aspiration event, which is the gold standard for diagnosis of aspiration pneumonitis. Evidence of inciting events such as seizure, AMS, or absence of antecedent history of pulmonary symptoms help to rule out pneumonia. Previous case reports recommend holding antimicrobial therapy at the time of a witnessed or suspected aspiration event to reduce the risk of treatment-related complications. In patients with aspiration pneumonitis, potential medication-related toxicities can be avoided through a conservative approach when inciting factors for an acute aspiration event are present.

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Persistent Pain in a Patient with a Prosthetic Elbow Joint

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Introduction: Tuberculosis is one of the top 10 causes of death and is the leading cause of death from a single infectious agent worldwide. When left untreated, tuberculosis kills up to 80% of those affected (1). We report a case of osseous tuberculosis in a patient that was discovered after removal of an infected left total elbow prosthesis.

Case presentation: A 52-year-old Filipino female with a history of latent TB (treated with INH in 1986) and subsequent re-exposure to infected mother in 2008 and left elbow replacement (2012) complicated by MRSA infection and removal of hardware with spacer insertion in 2014 had recurrent presentations between 2012-2015 with elbow pain. MRSA was discovered on intraoperative cultures during initial elbow revision (2/2014) and was treated with 6 weeks of IV vancomycin with subsequent normalization of ESR and CRP. The patient continued to report pain, and a CT scan showed osseous irregularity at which time the patient underwent left elbow arthrotomy and removal of deep hardware with placement of new antibiotic cement. After discharge, operative cultures grew acid fast bacilli and DNA probe resulted positive for Mycobacterium Tuberculosis. The patient was prescribed INH, Rifampin, Pyrazinamide, and Ethambutol.

Discussion: The incidence of prosthetic joint infection after an elbow replacement is 2.53% (2). Mycobacterium tuberculosis prosthetic joint infection is increasingly rare, accounting for <1% of these infections (3). Therefore, joints are more frequently tested for the typical bacterial pathogens, staphylococcus aureus and staphylococcus epidermidis, which can lead to delays in further testing, especially when bacterial organisms are present such as in our patient. There is an increased need for consideration of atypical infections, such as TB, especially in high risk populations or prior history of atypical infections. Patients in high-risk groups for TB include immigrants, immunocompromised individuals, or those in close contacts with a person who has active TB as in our patient. Extrapulmonary tuberculosis infection is usually sputum negative and there are no current guidelines to re-assess immigrants who may return to endemic countries. These factors make diagnosing latent and extrapulmonary tuberculosis challenging. Consequently, it is necessary to rely on a thorough history and exam in patients with a history/known risk factors of TB. Current guidelines recommend screening travelers for TB 8-10 weeks post-travel if they have possible prolonged exposure to TB, such as working in healthcare or homeless facilities, or those who plan a prolonged stay in an endemic country (4). In patients such as ours, with a history of immigration from a TB endemic region, it is crucial to discuss risk of re-exposure with travel to such endemic locations and implement plans for future TB screening.

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Ventricular Tachycardia Caused by Obstructive Sleep Apnea

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Introduction:
Symptoms of ventricular tachycardia vary depending on its rate and duration. Some patients do not experience symptoms while others may have palpitations, chest pain, and shortness of breath. If the rate is rapid enough to result in hemodynamic compromise and the duration approaches twenty to thirty seconds, patients may experience pre-syncope or syncope. The differential diagnosis of ventricular tachycardia is broad and can include electrolyte imbalances, myocardial ischemia, hypoxia, adverse drug effects, anemia, and infection.

Case Description:
A 71-year-old man with hypertension, benign prostate hyperplasia, heart failure with reduced ejection fraction (25-30%), and an automatic implantable cardioverter defibrillator presented to the emergency department with a two-hour history of generalized weakness and an episode of syncope. He was in his usual state of health until he awoke that morning and felt weak. He had an unwitnessed loss of consciousness when rising from bed and fell. Pre- and post-syncope he denied experiencing palpations, chest pain, dyspnea, orthopnea, dizziness, fever, incontinence, and sick contacts. Emergency department evaluation revealed hypokalemia, hypomagnesemia, and multiple episodes of ventricular tachycardia with his defibrillator firing. The patient was given intravenous metoprolol and amiodarone and admitted to telemetry for further workup. By hospital day two, electrolyte abnormalities had been corrected and acute decompensated heart failure, myocardial ischemia, and infectious etiologies had been ruled out. The ventricular tachycardia was controlled during the day but the patient continued to have episodes during the night. Although the patient denied morning headaches, snoring, and daytime sleepiness, these nocturnal episodes led to sleep apnea consideration as a likely etiology. Polysomnography revealed obstructive sleep apnea (OSA) and after continuous positive airway pressure machine optimization, all episodes of ventricular tachycardia resolved.

Discussion:
Although sleep apnea is an uncommon cause of ventricular tachycardia, it must be considered in the differential. A 2018 study with more than 900,000 patients found that ventricular tachycardia and fibrillation were more prevalent among patients with OSA compared to those without a diagnosis of OSA. The exact mechanism of increased ventricular tachycardia in sleep apnea is unclear and is most likely multifactorial. Etiologies may include sympathetic vagal tone imbalance, hypoxia and hypoxemia, and large intrathoracic pressure shifts. Screening for sleep apnea should be considered as a standard part of the workup of patients with ventricular arrhythmias due to its prevalence, risk of adverse events, and potential to be abolished with appropriate treatment such as continuous positive airway pressure therapy.

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C. acnes Presenting as Brain Abscesses

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Cutibacterium spp. (formerly Proprionibacterium) colonizes skin and mucosal surfaces and is known for its relationship to acne vulgaris, but can occasionally cause invasive infections. Specifically, Cutibacterium acnes is a leading pathogen for prosthetic joint infections of the shoulder. Although a low virulence pathogen, Cutibacterium acnes can also cause endovascular or central nervous system (CNS) infection associated with implantable devices. Due to its slow growth and potential to contaminate cultures it can often be overlooked as a pathogen. Herein, we present a case of multiple brain abscesses due to Cutibacterium acnes.

A 65-year-old man with a history of bipolar disorder, no known IV drug use, and well-controlled HIV on Genvoya (elvitegravir, cobicistat, emtricitabine and tenofovir alafenamide) with a viral load of <40 copies/ml and CD4 count of 1284 cells/μL presented complaining of four weeks of frequent falls, headache, nausea, and fatigue. The patient underwent brain computed tomography angiography (CTA) and was referred to the Emergency Department due to multiple ring-enhancing lesions in the inferior frontal, left posterior parietal and temporal lobes. The patient denied fever, unsteady gait, seizures, neck pain, numbness, weakness, or nausea/vomiting. Review of systems and physical examination revealed no abnormalities. Laboratory evaluation revealed a white blood cell count of 6.82 10*3/μL and toxoplasma IgG <3.0 IU/mL. Blood cultures were obtained.

The differential diagnoses included malignancy and infection; the patient was admitted for left-sided craniotomy. Lesions in the left anterior and posterior temporal lobe were resected and appeared necrotic centrally. Preliminary pathology revealed possible abscesses. While inpatient the patient was given four days of empiric vancomycin, ceftriaxone, and metronidazole for suspected cerebral abscess. Blood cultures were negative. Hospital course was uneventful. While awaiting final pathology and culture results, the patient was discharged on amoxicillin-clavulanate and sulfamethoxazole-trimethoprim to cover likely pathogens.

On follow-up ten days after sample collection, the patient admitted poor antibiotic adherence. Pathology was still pending but cultures revealed C. acnes from both lesions, which was initially believed to be possible contamination. The patient was prescribed ceftriaxone and oral metronidazole. During the second follow-up five weeks after sample collection, the cultures continued to reveal only C. acnes. Pathology did not suggest any fungal, neoplastic, or demyelinating etiology. It was determined that C. acnes was the causative organism for the brain abscesses. The patient was transitioned from ceftriaxone to amoxicillin-clavulanate as there was difficulty maintaining the PICC line due to psychosocial factors. Follow-up MRI two months after craniotomy showed abscess resolution.

In this case initially positive cultures were believed to be due to contamination. This highlights the importance of maintaining C. acnes in the differential of brain abscesses despite absence of traditional risk factors such as CSF shunt placement or other neurosurgical intervention.

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Abnormal Presentation of Post-Transplant Lymphoproliferative Disorder in an Orthotopic Liver Transplant Patient

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Case Description: A 47-year old male with past medical history of liver transplant in 2015 due to alpha-1 antitrypsin deficiency and alcoholic cirrhosis taking tacrolimus and mycophenolate mofetil presented with an 8-day history of shortness of breath, productive cough, fever, and left neck swelling. The patient was in mild respiratory distress with course crackles in his left upper lobe. Chest x-ray showed bilateral patchy consolidations concerning for multifocal pneumonia. Chest CT revealed multifocal consolidative and glass opacities with multiple cystic changes in upper lung zone, splenomegaly, and an enlarged left thyroid gland. He was treated empirically for multifocal pneumonia with cefepime, trimethoprim-sulfamethoxazole, and isavuconazole. Despite this, his respiratory status continued to worsen. A left thyroid lobe ultrasound was performed on the enlarging neck mass and indicated an enlarged lobular hypoechoic thyroid concerning for malignancy. A thyroid biopsy revealed monomorphic post-transplant lymphoproliferative disorder (PTLD) classified as diffuse large B cell lymphoma. His disease was staged with PET/CT revealing hypermetabolic lesions of the thyroid gland, lungs, and gastric wall. He was transferred to the Bone Marrow Transplant/Oncology service for further management.

Discussion: PTLD is the most common malignancy associated with solid organ transplantation (SOT), accounting for 20% of all malignancies that occur with SOT. However, the overall incidence of disease is low and varies by transplanted organ, about 1% following liver transplant. Most new diagnoses occur within the first year post-transplant (>80%). Risk factors for developing PTLD include higher level of T-cell immunosuppression, EBV-negative recipient and EBV-positive donor, time post-transplant less than 12 months, and age less than 25 years. There is a possible link between tacrolimus specific use post-transplant and PTLD development; however, most studies conducted on this topic evaluated patients who had received kidney or heart transplants. Half of cases of PTLD in heart, lung, and liver transplants involve the transplanted organ resulting in dysfunction of that organ. PTLD typically presents with the classic B symptoms, including night sweats, low-grade fever, and weight loss. Compared to general lymphomas, PTLD cases are more likely to have extranodal involvement, high grade and aggressive clinical courses, and poorer outcomes.
This patient had an unusual presentation of PTLD. The atypical presenting symptoms mimicking infection, lack of liver dysfunction, and time since transplant greater than 12 months delayed his time to diagnosis and treatment. This case illustrates the importance of keeping the differential diagnosis broad and maintaining a high index of suspicion for malignancy in transplant recipients.

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Anticoagulation Management of COVID-19-Associated Coagulopathy

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Introduction: COVID-19-associated coagulopathy is characterized by elevations in fibrinogen and D-dimer levels that correlate with inflammatory changes distinct from disseminated intravascular coagulation. The risk for thromboembolism is markedly increased, especially in the critically ill, with case series reporting prevalences of 20 to 43 percent in ICU patients, often despite prophylactic anticoagulation.

Case Presentation: A 72 year-old man with coronary artery disease s/p percutaneous coronary intervention, diabetes mellitus, and hypertension was transferred for management of subdural hematoma, atrial fibrillation with RVR, and COVID-19 infection. One week prior, he was hospitalized for syncope and URI symptoms. COVID-19 test was positive and CT chest showed ground-glass opacities. No supplemental O2 was required. Vasovagal syncope was the purported cause of his fall and he was discharged home. The patient presented again for a pre-syncopal fall. Physical exam revealed tachycardia with irregular rhythm. Labs showed normal platelet count, WBC 15.1, PT 17.4, fibrinogen 693, and D-dimer 927. He tested positive for COVID-19. CT showed a small, stable subdural hematoma in the right temporal lobe. No surgical intervention was required, but anticoagulation prophylaxis was held. CT showed lung opacities, enlarged pulmonary artery, and mild cardiomegaly. ECG showed atrial fibrillation with RVR and diltiazem was given, but recurrent episodes required metoprolol and digoxin loads. Hypoxemia required increasing O2 flow rates. Sputum grew methicillin-sensitive S. aureus and IV cefazolin was started. D-dimer increased to 28,686. CT angiography revealed an acute pulmonary embolism involving the right pulmonary artery, extending into lower lobe segmental and subsegmental branches with no evidence of right heart strain or pulmonary infarction. Duplex ultrasound showed non-occlusive DVT of the left posterior tibial vein. Heparin was initiated after CT showed stable hematoma. Patient was transitioned to enoxaparin. By day 14, he was saturating well on room air, hemodynamically stable, and discharged home on warfarin and aspirin.

Discussion: COVID-19-associated thromboembolism has prompted treatment with anticoagulation for patients admitted to the hospital even though the etiology of the coagulopathy is incompletely understood. An autopsy study reporting on lung tissue from patients who died from COVID-19–associated respiratory failure found endothelial injury, microangiopathy, alveolar capillary microthrombi, and vascular angiogenesis. Our patient presented the extra challenge of bleeding prior to admission and coagulopathy. Management of patients at higher bleeding risk was addressed by Bowles et al who shared their evaluation of blood specimens obtained from 216 COVID-positive patients. Twenty percent of these patients had a prolonged aPTT and a large percentage of them were lupus anticoagulant positive. They recommended anticoagulation and even thrombolytic therapy for these patients despite lab findings. Treatment of our patient's thromboembolism with anticoagulation was initiated because his subdural hematomas had stabilized. The coagulation abnormalities related to COVID-19 need further clarification to help clarify best patient management.

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Intractable Hemoptysis in an African Immigrant

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Case Presentation: A healthy 31-year-old female from the Democratic Republic of Congo presented with chronic dyspnea since age 17. Her symptoms had worsened over the past 2 weeks, as she had developed a dry cough and orthopnea. CTPA demonstrated no acute thrombus but revealed a lack of arterial opacification in the right middle and lower lobes with dilation of the pulmonary arteries and right ventricle. Right heart catheterization established a pulmonary artery pressure of 118/35 mm Hg (normal < 25/10 mm Hg) and echocardiogram revealed moderate tricuspid regurgitation. Right ventricular pressures were elevated at 115/17 mm Hg and cardiac output was decreased at 2.6 L/min. Hypercoagulability workup was negative. A diagnosis of chronic thromboembolic pulmonary hypertension (CTEPH) was made and the patient started anticoagulation and vasodilatory therapy. She was referred for surgical intervention but declined. 5 months later she presented with hemoptysis (240 mL/24 hours). Exam demonstrated a 3/6 systolic murmur near the LLSB with a palpable right ventricular heave. Repeat imaging showed persistent pulmonary artery thrombus, as well as ground glass opacities consistent with extravasation into the lung parenchyma. Due to intractable hemoptysis the patient was recommended again for surgery. After undergoing a bilateral pulmonary endarterectomy with tricuspid valve repair, her hemoptysis resolved, there was dramatic improvement in her pulmonary artery pressures (49/16 mm Hg), and her symptoms improved.

Discussion: CTEPH is characterized by the limited resolution of thromboemboli in the pulmonary artery, which results in decreased flow through the pulmonary circuit, subsequent formation of pulmonary hypertension, and right-sided heart failure. Because of increased resistance in the pulmonary arteries, blood may preferentially be routed to the lung parenchyma through the bronchial arteries, branches of the thoracic aorta. In normal circulation, these arteries are responsible for only 1% of the cardiac output and are connected to the pulmonary arteries through microscopic anastomoses at the alveoli and respiratory bronchioles. However, in times of chronic pulmonary ischemia, as in CTEPH, the bronchial arteries undergo hypertrophic dilation and may observe an 18-30% increase in cardiac output in an attempt to preserve gas exchange. Because of this increased flow through the bronchial circuit, patients are at an increased risk of hemoptysis. This hemoptysis is thought to result from vasodilation of thin-walled anastomotic connections between the pulmonary and systemic circulations which rupture under systemic pressures. Potential therapeutic interventions for hemoptysis secondary to bronchial artery hypertrophy include bronchial artery embolization or pulmonary endarterectomy. As was demonstrated in this case, removal of pulmonary artery thromboemboli can help reduce pressures in the pulmonary circulation and improve symptoms of pulmonary hypertension, as well as reduce the flow burden present in bronchial arteries and minimize the risk of potentially fatal hemoptysis.

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ACUTE ESOPHAGEAL NECROSIS PRESENTING AS UPPER GI BLEED IN THE SETTING OF SEVERE DECOMPENSATED HEART FAILURE.

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Introduction: Acute esophageal necrosis (AEN), otherwise known as black esophagus, is an extremely rare condition with an incidence of <0.01%. As of 2006, there were only 88 cases formally diagnosed. The etiology of this condition is unclear and thought to be multifactorial. Here, we present an elderly gentleman with clinicopathologically confirmed AEN in the setting of decompensated heart failure.

Case Description: A 66-year-old Caucasian male with a history of systolic heart failure, atrial fibrillation s/p ablation, and CAD presented after a fall. He had not been compliant with medications and had been attempting to control his peripheral edema with bandages and compression devices. On admission, patient was in atrial fibrillation with RVR, hypotensive, and hypoxemic. He had significant anasarca consistent with decompensated heart failure. Ultrasound showed a nodular liver, which, along with an elevated INR, high total bilirubin, and thrombocytopenia, was suggestive of cardiac cirrhosis. Transthoracic echocardiography revealed an ejection fraction of 20%. He was determined to be in cardiogenic shock and stabilized on milrinone. His hospital course was complicated by MRSE bacteremia from severe LE cellulitis. On hospital day 4, he developed coffee ground emesis. EGD findings included friable ulcerated mucosa in the middle and distal thirds of the esophagus. Pathology confirmed esophageal necrosis. He became acutely encephalopathic and expired soon after from multi-organ system failure.

Discussion: Acute necrotizing esophagitis is pathological diagnosis. Clinically, it is characterized by black or dark pigmented ulcerated mucosa in the lower third of the esophagus, as in our patient. Patients usually present with hematemesis, coffee grounds emesis, and/or melena. It is usually seen in the context of severe, life-threatening systemic disease. Also known as Gurvit's syndrome, AEN was formally described in 1990, but necrotizing esophagitis has been mentioned in medical literature prior to this. Information on the topic remains sparse. From 1970 to 2006, only 88 cases of necrotizing esophagitis have been reported. It is likely more prevalent than this, but commonly misdiagnosed. In our patient, we presume that acute decompensated heart failure led to cardiogenic shock, followed by esophageal hypoperfusion, resulting in AEN.

The pathophysiology of esophageal necrosis is not well understood and is likely multifactorial. AEN may be triggered by severe reflux in the context of gastritis/PUD. This affects the distal and middle esophagus more. The upper esophagus, supplied by the inferior thyroid artery, is virtually never involved. Hemodynamic instability causes hypoperfusion in esophagus, but additional insult is required to cause frank necrosis. These include malignancy, diabetes, atrial fibrillation, CKD, and liver disease; many of which were seen in our patient. Mortality is as high as 50% in AEN when associated with co-morbid conditions. Treatment includes an IV proton pump inhibitor, supportive care, and stabilization of underlying systemic disease.

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Corneal Ulcer Due to Atypical Nontuberculous Mycobacterium

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Introduction: Over the past decade, the number of nontuberculous mycobacterial (NTM) infections have increased, specifically with Mycobacterium Abscessus/chelonae groups for ocular infections. Their course is indolent and difficult to treat due to their inherent resistance to many therapies.

Case Description: 70-year-old female patient with sarcoidosis on azathioprine and a history of multiple ocular procedures presented with worsening vision and right eye pain over 4 months. On exam, the patient had severe visual impairment of both eyes with corneal ulceration of the right eye. Culture obtained was positive for Mycobacterium Abscessus with resistance to multiple agents. She was started on an ophthalmic formulation of tigecycline, amikacin, bacitracin, and chlorhexidine for 6-12 months. Treatment was complicated by recurrent ulceration of right eye patch site requiring removal of right glaucoma drainage implant. Repeat swab cultures demonstrated persistence of acid-fast bacilli on stain and cultures were sent for synergy testing. The patient was transitioned to rituximab and prednisone for immunosuppression, acutely treated with vancomycin and amikacin injection, and continued indefinitely on the same eye drop regimen.

Discussion:
Mycobacterium Abscessus complex is a group of rapidly growing, multidrug-resistant, nontuberculous mycobacteria that are responsible for a wide spectrum of skin and soft tissue diseases, central nervous system infections, bacteremia, and ocular and other infections. This atypical mycobacterium has the unique expression of the gene erm(41) that contributes to its inherent resistance to macrolides and overall multidrug-resistance. Moreover there are 3 different subspecies of M. Abscessus, each with different patterns of erm(41) that further changes treatment outcomes. With respect to ocular infections, M. abscessus may present as keratitis, endophthalmitis, scleritis, or infection of other ocular origin. Our patient had a presentation concerning for endophthalmitis, an intraocular infection that can be seen after ocular procedures. Typically, this infection can occur within days and up to 35 weeks after intervention. On examination, anterior chamber inflammation with hypopyon reaction is typical and at times corneal infiltrates or abscess formation may be noted. The indolent course of this infection is important to note, as a delayed postoperative inflammation should bring NTM infection into the differential. This patient had several risk factors for ocular NTM infection including multiple corneal transplants, ocular surgery for glaucoma, and immunosuppression. In terms of treatment, NTM species have different susceptibilities to therapy making identification with AFB stain and culture critical. Intraocular antibiotic administration is most effective against NTM. Studies have shown that M.abscessus is generally susceptible to amikacin and clarithromycin followed by agents such as fluoroquinolones. Ophthalmic formulations of these antibiotics are crucial, as many systemic antibiotics have poor ocular penetration. Lastly, surgical intervention may be required for refractory cases.

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Primary Chickenpox in an Immunocompromised Patient

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Introduction: Adult primary chickenpox is a severe infection in immunocompromised patients. We present a case of chickenpox in a young patient on mTOR inhibitors.

Case Description: A 27-year-old male with a past medical history of tuberous sclerosis and seizures, on everolimus, presented with vomiting, fever, and altered mental status. He was treated empirically for meningitis. Within 24 hours, he developed an evolving vesiculopapular rash, beginning on his trunk and progressing to his limbs and face. Separate lesions were in different stages of development simultaneously. Vesicle fluid was PCR positive for VZV. CXR showed multifocal infiltrates and right-sided nodularity. CT head showed radiographic proptosis, left periorbital swelling, and paranasal sinus opacification. No lumbar puncture was obtained due to vesicles on his back. He was intubated 4 days after admission due to respiratory distress. During intubation, vesicular and ulcerated lesions were noted in his oropharynx and upper airway. A chest CT showed multifocal pneumonia. VZV PCR was positive on broncho-alveolar lavage. His immunization history was unknown, but VZV IgM and IgG were positive, supporting a case of primary chickenpox, rather than reactivation zoster. He was treated with 21 days of acyclovir, and within 18 days of the initial lesion, all lesions were scabbed over. Respiratory and mental status improved, and he was discharged after 36 days in the hospital.

Discussion: Chickenpox is a primary infection by the varicella zoster virus, typically occurring in children. Incidences of chickenpox have decreased drastically since the introduction of the Varivax vaccine. As a live attenuated vaccine, Varivax is contraindicated in immunocompromised patients, who depend on herd immunity. While childhood chickenpox is a self-limited disease, adult chickenpox can be deadly. Chickenpox is characterized by diffuse skin lesions in multiple stages of evolution. Adult patients may develop pneumonia and encephalitis. This presentation may appear similar to disseminated zoster, a reactivation of the varicella virus, or as part of other viral illnesses, like coxsackievirus and echovirus. The best way to determine the causative organism is through PCR of blister fluid. In a patient whose immunization status is unknown, VZV IgM and IgG titers can determine if the patient had previous exposure to varicella. In cases of primary VZV infection, IgM and IgG antibodies appear 2-5 days after the rash. IgM antibodies are undetectable by year-1, after the initial infection. IgG antibodies persist long after the infection is gone. Patients who have primary chicken pox are at risk for developing shingles, and vaccination should be considered. Unvaccinated contacts of an adult with chickenpox should be vaccinated. Acyclovir is the recommended treatment for adult chickenpox, although its utility in pneumonia and encephalitis is not well studied. Length of treatment depends on symptom severity.

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Presentation and Treatment Considerations of Disseminated Histoplasmosis in Renal Transplant Patients.

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Introduction: Disseminated histoplasmosis is a potentially fatal disease seen primarily in immunocompromised individuals. Historically, disseminated histoplasmosis has been seen in AIDS patients. However, with the broader availability of ART therapy and the proliferation of solid organ transplants, transplant patients increasingly makeup cases of disseminated histoplasmosis. We report a case with septic shock in a renal transplant patient.

Case Description: A 38-year-old female Ohio native with SLE complicated by ESRD, status post two living donor renal transplants on mycophenolic acid and tacrolimus, Libman-Sacks endocarditis, and dilated cardiomyopathy presented with abdominal pain, transaminitis, and a thickened gallbladder wall on ultrasound. She had an uncomplicated cholecystectomy but subsequently became septic. She was started on vancomycin and meropenem and transferred to the MICU for vasopressor therapy. MICU workup included: WBC 26.8 x 109/L, CXR with bibasilar opacities, and CT Chest with multiple right upper lobe groundglass nodular opacities and innumerable nodules. (1,3)-Beta-D-Glucan was positive. She was started on Isavuconazole. Liver enzyme elevations persisted with peak ALP 709 IU/L, AST 383 units/L, ALT 151 units/L. She developed melena, prompting an EGD showing multiple duodenal and linear esophageal ulcers. Liver biopsy demonstrated Histoplasma within the sinusoidal Kupffer cells. Histoplasma species were also noted on duodenal, transverse, and ascending colon biopsies. Isavuconazole was transitioned to Amphotericin 3mg/kg IV. She improved, and Amphotericin was transitioned to voriconazole 200 mg BID indefinitely

Discussion: Infection with Histoplasma capsulatum is common in endemic regions, with up to 80% of individuals demonstrating previous infection. Among solid organ transplant patients, symptomatic disseminated histoplasmosis incidence is still low, with reported 1 case per 1000 transplant-person-years. Though any organ can be affected, pulmonary and gastrointestinal, specifically colon and small bowel, involvement is most common. Almost all patients have abnormal chest radiograph or CT results like the groundglass opacities and nodules in this case.

Treatment of disseminated histoplasmosis consists of amphotericin B with transition to an oral azole. Amphotericin is poorly dialyzable with a maximum recommended dose of 35 mg/kg and should be used judiciously in ESRD patients. This case required 45 mg/kg, but still no adverse effects were seen. Our patient received the liposomal formulation of amphotericin, possibly increasing tolerability. Patients receiving amphotericin prior to azole therapy show faster serum and urine antigen clearance than azole therapy alone.

Itraconazole is an effective and well tolerated step-down therapy, with fluconazole, ketoconazole, and voriconazole considered second line agents. However, retrospective studies show voriconazole (our therapy) has comparable efficacy to itraconazole. Mycophenolate should be held and steroid doses reduced during infection in renal transplant patients. There are no reported cases of transplant rejection during or following histoplasmosis treatment. Patients require lifelong azole prophylaxis once immunosuppressants are restarted.

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An Unusual Case of Postoperative Anaerobic Bacterial Meningitis

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Introduction: Bacterial meningitis is a significant cause of morbidity and mortality despite effective antibiotic regimens. Clinicians should be aware of uncommon organisms and when to suspect them.
Case Description: A 56-year-old female with a history of non-functioning pituitary adenoma status post uncomplicated transsphenoidal resection presented post-operatively with headache, nausea, vomiting and altered mental status. She was afebrile. Exam was notable for confusion, meningismus and sonorous respirations. Initial workup revealed leukocytosis and elevated inflammatory markers. CT showed new interventricular hemorrhage, cerebral edema and pneumocephalus. She was intubated and treated empirically with vancomycin, cefepime, metronidazole and dexamethasone. CSF analysis showed many neutrophils, low glucose and high protein. Preliminary culture results suggested an anerobic bacterium. On hospital day 2, she was neurologically stable and extubated. Follow-up MRI suggested the presence of sellar abscess and showed extensive mucosal thickening of the paranasal sinuses with air-fluid levels concerning for new sinus infection or inflammation. Final organism identification showed Veillonella spp. and therapy was deescalated to intravenous ceftriaxone and metronidazole. After gradual improvement she was discharged on hospital day 7 on 4 weeks of IV antibiotics. Follow-up 4 weeks from discharge showed no sequelae of CNS infection and normalization of lab values.
Discussion: Major mechanisms for the development of bacterial meningitis include nasopharyngeal spread to the bloodstream or bacteremia with subsequent invasion into the CNS, and neurosurgical or traumatic inoculation. Bacterial meningitis due to anaerobic organisms is uncommon, accounting for 2.4% of cases. Of those cases, half had recent neurosurgery. This patient had several interesting findings in her workup that may have given clues toward her final diagnosis.
Referred to as the “anerobic counterpart” of Neisseria, Veillonella is an anaerobic gram-negative diplococcus considered normal flora of the mouth, gastrointestinal tract and vagina. The organism has been identified in cases of periodontitis, aspiration pneumonia, endocarditis and is the most predominant anaerobe in chronic maxillary sinusitis. However, few sources have identified Veillonella meningitis. Interestingly, a 2000 report discussed a case of Veillonella meningitis in which CT reported similar signs of sinusal inflammation, leading the authors to conclude the sinuses as the most likely source of infection. In addition, pneumocephalus is often due to trauma and surgical procedures. However, there are case reports in which pneumocephalus has been associated with meningitis, notably Clostridium perfringens and mixed aerobic-anaerobic infections. Veillonella has been implicated in destructive gas-producing abscesses, however in the current case it is difficult to identify the cause due to recent surgery. Moreover, anaerobic bacteria are important causative organisms of intracranial and epidural abscesses.
Ultimately, clinicians should be aware of unusual causes of meningitis and have suspicion for these organisms when features such as sinus infection or abscess is observed.

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Atypical COVID-19: Striking Lab Abnormalities with a Mild Disease Course of Acute Abdominal Pain

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Introduction: The understanding of COVID-19 presentations is limited due to the recent emergence and ongoing evolution of the pandemic. A common protocol to prevent spread is to screen for typical symptoms including fever, cough and shortness of breath. However, less common presentations as well as asymptomatic carriers have become potential roadblocks to tracking and containing spread.

Case Presentation: A 54-year-old African American male with hypertension, chronic kidney disease, colonic diverticulosis, and a family history of colon cancer presented with 3 days of acute epigastric pain. On the day of admission, the pain was stabbing in character and progressed to 9/10 in severity. There was no radiation and no association with specific behaviors or positioning. His pain decreased oral intake, and he described orthostatic hypotension along with a headache. The patient notably denied any recent cough, dyspnea, fever, nausea, emesis, melena, hematochezia, constipation, diarrhea, falls/trauma, travel, and exposure to sick contacts. Paramedics brought him to the emergency department (ED) following his sister's insistence, due to appearing acutely ill. Upon arrival, the patient appeared unkempt, with poor personal hygiene, leading to suspicion that he was currently homeless.

In the ED, he was afebrile, pulse =110 bpm, blood pressure =127/94 mmHg, breathing comfortably with (18 bpm), no supplemental oxygen requirements. CBC showed lymphopenia with white blood cells at 3.2x10 3 cells/microliters and an absolute lymphocyte count of 0.37x10 3 cells/microliters. BUN/creatinine reported as 105/2.85 mg/dl and a GFR-AA EKG were all non-concerning.

Abdominal CT incidentally visualized the left lower lung lobe, showing a diffuse ground glass infiltrate. COVID-19 PCR nasal swab was positive. His D-dimer level was 79,000 mcg/L, with a negative DVT workup. The patient was isolated and treated supportively, leading to uncomplicated discharge.

Discussion: While COVID-19 is associated with predominant pulmonary symptoms, this case illustrates concerning GI presentations which seem to present in a milder form than patients with primary respiratory symptoms. Literature suggests that over 80% of patients have mild disease (1). COVID-19 testing initially focused on febrile patients with respiratory symptoms—not digestive symptoms—leaving a large cohort of undiagnosed patients with low severity illness and digestive symptoms, such as diarrhea, who unknowingly spread the virus. It is critical to define clinical characteristics of patients with mild disease in the outpatient setting as this group has the potential to be a major driver of the pandemic.

Han, C, Duan, C, Zhang, S, et al. Digestive Symptoms in COVID-19 Patients With Mild Disease Severity: Clinical Presentation, Stool Viral RNA Testing, and Outcomes. Am J Gastroenterol. 2020 Apr 15 : 10.14309/ajg.0000000000000664. Published online 2020 Apr 15.

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Thiamine-responsive megaloblastic anemia, an extremely rare inherited disorder mimicking MDS, megaloblastic anemia, and TTP

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Fewer than 80 cases of thiamine-responsive megaloblastic anemia (TRMA), or Rogers syndrome, have been reported in the world's literature. The diagnosis is confirmed by detection of a mutation of the SLC19A2 gene. Clinical features include the triad of megaloblastic anemia with normal serum B12 and folic acid levels, diabetes mellitus, and progressive sensorineural hearing loss. Additional medical issues including optic atrophy, high-output heart failure, congenital heart defects, arrhythmias, mood disorders, developmental delay, and seizure disorders have also been described in association with TRMA. Megaloblastic anemia is reversed with high dose thiamine replacement. Other manifestations of this genetic disease, however, do not improve or respond to thiamine therapy. Symptoms of the disorder present between early childhood and adolescence.

The patient is a 27 y/o AA male who presented to a community hospital with hemoptysis and pancytopenia. His medical history included hearing loss, insulin-dependent diabetes, seizures, hypertension, congestive heart failure, and developmental delay. He discontinued thiamine a month prior to admission. He was accompanied by his mother who reported a history of TRMA and Usher syndrome. No confirmatory records were available at the time of admission. Family history was significant for sudden death of an adolescent brother with similar medical issues.

Physical exam was unremarkable, with the exception of bilateral subconjunctival hemorrhages and mildly elevated blood pressure (142/76). Laboratory findings showed pancytopenia with a white blood cell count of 2.1 and normal differential, hemoglobin 8.1 with normal indices, and platelet count of 17,000. Rare schistocytes were seen on review of peripheral smear. The comprehensive metabolic profile was unremarkable.

Plasma exchange was instituted on the day of admission due to suspicion of thrombotic thrombocytopenic purpura (TTP). This was discontinued after a single exchange. ADAMSTS-13 level was normal (77%). Vitamin B12 and folate levels were normal (590 pg/ml and >1000 ng/ml). Bone marrow biopsy revealed granulocytic and erythroid hyperplasia with megaloblastic features and ringed sideroblasts. Marrow karyotype was normal (46, XY[20]).

Records were obtained one day after admission, confirming that patient had homozygous biallelic mutations in gene SLC19A2 and indicating a diagnosis of thiamine-responsive megaloblastic anemia (TRMA) or Roger's syndrome. The patient was started on high dose IV thiamine followed by oral replacement. At discharge, platelets had improved to 52,000, white blood cell count was 12,000, and hemoglobin was 8.5. The patient was subsequently lost to follow up.

The extreme rarity of this condition and the absence of documentation on admission presented clinicians with a diagnostic dilemma: to trust the family's verbal report, or pursue other diagnostic possibilities, in particular, TTP. Hospital and ER physicians may have little familiarity with these rare entities. Patients and families would benefit from having genetic tests readily available.

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To Give Hemin or Not to Give Hemin? The Spot Test Answers The Question.

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Introduction: Acute intermittent porphyria (AIP) is a metabolic disorder of the heme synthesis pathway at the step of porphobilinogen deaminase (PBGD), resulting from one of over 400 identified mutations of the hydroxymethylbilane synthase (HMBS) gene. The disease has low penetrance, modulated by environmental and genetic factors outside of the HMBS mutation, resulting in latent or manifest presentations that inform separate treatment plans. Symptoms are variable and non-specific, featuring neurovisceral attacks characterized by abdominal pain and neuro-psychiatric dysfunction.

Case Presentation: A 27-year-old man with a family history of AIP and a positive mutation in the HMBS gene presented to the ED with an episode of severe abdominal pain and nausea, associated with a sensation of near-syncope. He reported episodes of abdominal cramping since age 18 that increased following a hernia surgery 3 years ago. Abdominal pain persisted days at a time, with pain on average 15-20 days each month. He endorsed "fainting spells" and lack of control over racing thoughts associated with abdominal pain. He endorsed headaches, blurry vision, paraesthesias in bilateral lower extremities, back pain, a recent skin rash and darkening of urine to orange.

On inpatient admission, he was stable but anxious and started on a carbohydrate-loading diet, with urine spot tests taken immediately. He received hemin for 4 days. Echocardiogram, ECG, EEG and CT abdomen/pelvis were unremarkable. Upon discharge, he was started on fluoxetine for anxiety and referred to outpatient psychiatry. Tilt table testing and event monitoring were ordered outpatient, to rule out alternative causes of syncope. Following discharge, spot urine porphobilinogen, urine total porphyrins and blood tests for porphyrins, ALA and porphobilinogen all returned within normal limits.

Discussion: This case illustrates the variable penetrance and presentation of AIP as manifest versus latent disease. A diagnosis of manifest AIP requires: symptoms, elevated urine porphobilinogen and an HMBS mutation and necessitates treatment. Latent AIP is characterized by an HMBS mutation in an individual without consistent clinical or biochemical symptoms and requires regular monitoring. Despite acute neurovisceral symptoms, this patient does not have manifest AIP, given negative biochemical findings. He exhibits latent AIP as a carrier, with potential to develop disease. In both cases, regular outpatient hematology and psychiatry follow-up is critical.

Typically, neurovisceral symptoms lead to genetic sequencing and a subsequent AIP diagnosis. Perhaps increasingly, a known genetic mutation may precede neurovisceral symptoms, as shown here. Genetic screening of patients with a known AIP mutation or family history should be followed by education to recognize symptoms and obtain spot porphobilinogen testing as early as possible at the start of symptoms, to conclusively determine an AIP attack.

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Generalized Tonic-Clonic seizure induced Takotsubo Cardiomyopathy

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Introduction: First described in Japan in 1990, Takotsubo Cardiomyopathy (TC) is a syndrome characterized by transient regional systolic dysfunction of the left ventricle in the absence of angiographic evidence of obstructive CAD or acute plaque rupture. While the pathogenesis of the disorder is not well understood, it occurs in approximately 1 to 2 percent of patients presenting with suspected Troponin positive ACS or STEMI with a higher prevalence in post-menopausal women(1-3).

Case: 46 y/o female with a history of epilepsy (on Keppra) and HTN (on Amlodipine and Carvedilol) presented to the ED after a breakthrough GTC seizure while asleep. Upon arrival, she was unresponsive with initial vitals notable for HR of 136 bpm, BP of 170/108 mm Hg, RR 40-50s, and Oxygen saturation in the 60s on RA, which improved on non-rebreather mask. Initial VBG was concerning for lactic acidosis without respiratory compensation; EKG showed sinus tachycardia with nonspecific ST and T wave changes diffusely; BSUS revealed decreased ejection fraction with bilateral B lines; and a CXR showed diffuse parenchymal airspace opacities bilaterally. Patient was given Fentanyl in preparation for intubation for concern for an ICH. Her breathing improved after fentanyl administration and she became responsive. She was placed on BiPAP and head CT ruled out any acute intracranial pathology. Her VBG improved and she became A&O x 3, but she was unable to be weaned off BiPAP. Her initial Troponin came back at 3.29 ng/mL with a repeat BSUS showing significantly reduced EF with no pericardial effusion or right heart strain –prior Echocardiogram in 2015 showed no abnormalities. Despite initial concern for demand ischemia, this episode was thought to be less likely related to an MI in the setting of coronary artery calcium score of '0' 14 months prior to presentation. Patient was started on ASA, Brilinta, and heparin and admitted. Her Troponin stabilized overnight (3.29→5.57→4.25). Echocardiogram the next day showed LVEF of 35% with multiple wall motion abnormalities. Patient was managed symptomatically and a repeat echocardiogram on hospital day 4 showed EF of 45-50%. Her hospital course was complicated by aspiration PNA and an AKI with probable ATN. On hospital day 8, patient had no active complaints and was discharged with plan for outpatient coronary CT once renal function improved.

Discussion: Takotsubo Cardiomyopathy is increasingly recognized in the neurocritical care population especially in postmenopausal females. Subarachnoid hemorrhage and seizures are recognized as the most common neurologic triggers for TC. One study found that seizure related Takotsubo patients were younger (61.5 vs. 68.5 years, $p<0.0001$), more frequently males (17 vs. 9%, $p=0.004$), had less frequent chest pain (6 vs 76%, $p<0.005$), higher risk of cardiogenic shock (25 versus 8%, $p=0.003$), and higher risk of recurrence (14 versus 3%, $p=0.004$)(4). This case illustrates the importance of cardiac evaluation in patients with seizure, as the initiation of proper medical treatment for Takotsubo Cardiomyopathy can be lifesaving.

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Vitamin B12 masquerading as TTP

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Introduction: Microangiopathic hemolytic anemia (MAHA) is characterized by Coombs-negative hemolysis producing schistocytes on peripheral blood smear. There are many conditions that cause MAHA with thrombocytopenia, the most classic being primary thrombotic microangiopathies (TMA) such as thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS). However, severe vitamin B12 deficiency may produce findings suggestive of TTP, known as "pseudo-TTP".
Case Presentation: A 56-year-old African American woman with history of HTN, radiculopathy, and asthma presented with progressively worsening lower extremity swelling of 2-week duration. She denied trauma, immobilization, or travel. She also denied diarrhea, nausea/vomiting, altered mental status, or urinary changes.
Vital signs were within normal limits. Laboratory analysis revealed hemoglobin 5.9 g/dL, hematocrit 16.6%, platelet count 96 x 1012/L, white blood cell count 7.3 x 109/L, mean corpuscular volume 97 fL, lactate dehydrogenase 4301 IU/L, haptoglobin <30mg/dL, total bilirubin of 3.2 mg/dL (direct 0.5 mg/dL), prothrombin time 14.5 seconds, and INR 1.3. Peripheral smear showed schistocytes and anisopoikilocytosis.
Due to concern for TTP, HUS, or other TMAs, patient was started on glucocorticoids and plasmapheresis without complication.
Follow-up diagnostic testing revealed inappropriately normal reticulocyte percentage of 1.3% (reticulocyte production index 0.21, appropriate marrow response >2) and negative DAT. ADAMTS13 activity showed 52%, ruling out TTP. Rheumatologic testing revealed low titer +ANA 1:40, but was otherwise unremarkable. Notably, patient had markedly decreased vitamin B12 with high homocysteine and methylmalonic acid levels, raising suspicion that the patient's near-macrocytic anemia and MAHA was mediated by severe vitamin B12 deficiency.
At this point, plasmapheresis was stopped, and the patient began B12 supplementation. Because bone marrow pathology could not be excluded, patient underwent biopsy, which revealed hypercellularity with megaloblastic changes and near absent iron stores despite bland serum labs. Genetic studies were unremarkable, excluding myeloid clonal processes.
Since bone marrow biopsy is the gold standard for assessing iron deficiency, patient was placed on IV iron and scheduled for an outpatient EGD for workup of pernicious anemia, although anti-intrinsic factor antibodies testing was negative.
Discussion: While B12-deficiency driven pseudo-TTP is a rare cause of MAHA, workup of a patient with MAHA should include B12, homocysteine, and methylmalonic acid levels. Consistent with other reported pseudo-TTP cases, this patient's case had higher levels of LDH (>2500 IU/L) and more profound reticulocytopenia compared to typical TTP or TMA cases. Additionally, symptoms of heart failure secondary to severe anemia are common at presentation.
Prompt identification of pseudo-TTP and appropriate B12 repletion can reduce the need for higher acuity care, saving patients from aggressive treatments such as plasmapheresis.
This case also highlights the diagnostic challenge of a common condition, anemia, as the patient was both B12 and iron deficient, despite her serum iron studies being misleadingly normal.

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Therapeutic Use of Remdesivir in the Management of COVID-19

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Introduction: The novel coronavirus SARS-CoV-2 (severe acute respiratory syndrome coronavirus-2) was identified as the pathogen behind cases of pneumonia-like illness comprising that of COVID-19 (coronavirus disease 2019) in Wuhan, China in December 2019. COVID-19 quickly grew into a pandemic, necessitating advances and investigations in screening and treatment options. This case was one of the first cases of COVID-19 at this institution at the advent of the pandemic, and was one of the first to use remdesivir.

Case Presentation: A 58-year old gentleman with no reported PMH presents with 12 days of fever, fatigue, anosmia, dysgeusia, anorexia, nausea, and diarrhea. He reported recent travel to New York with potential exposure to COVID-19. In the ED, he was febrile with 89% SpO2 on room air. Influenza and RSV tests were negative and CT chest showed peripheral ground glass opacities and patchy consolidation. CBC showed lymphopenia with WBC of 4.8, 87.7% PMN and 8.8% lymphocytes. D-dimer level was 659, ferritin was 1259, LDH was 269, and procalcitonin was 0.09. Patient was admitted and placed on 3L nasal cannula due to oxygen saturation of 89-91% on 2L, despite lack of symptomatic dyspnea. Patient tested positive for COVID-19 PCR and began receiving a 10-day trial of remdesivir infusion. The patient improved steadily with remdesivir infusion and was weaned back to room air with resolving symptoms. He was discharged after completing 5 days of infusion due to his stable condition.

Discussion: The use of the nucleotide analogue remdesivir in the treatment of COVID-19 is currently debated. The US government has permitted compassionate use of remdesivir in hospitalized patients with severe COVID-19. Remdesivir is being studied in moderate and severe cases of COVID-19 in the US and Asia in the National Institute of Health's Adaptive COVID-19 Treatment Trial (ACTT). Preliminary results from ACTT, conducted among 1063 patients and released in April 2020, demonstrated that hospitalized patients with advanced COVID-19 and lung involvement who received remdesivir recovered faster than those with placebo [1]. In May, these findings were published in the New England Journal of Medicine demonstrating that adults with COVID-19 and lower respiratory tract infections had a median recovery time of 11 days compared with 15 days to those on placebo and mortality rates by 14 days were 7.1% in patients on remdesivir and 11.9% on placebo [2]. However, a multicenter Chinese trial published in Lancet of 237 patients demonstrated no statistically significant difference in the time to clinical improvement for patients with remdesivir versus placebo [3]. Our patient improved on remdesivir, although it is unclear if this medication was key to his recovery. In this ever-changing landscape of understanding regarding COVID-19, the demand and necessity for data regarding potential treatment options like remdesivir is critical.

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Rare Guillain-Barre Syndrome Variant following Proteus mirabilis and Clostridioides difficile Infections Masquerading as a CVA

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Introduction: Guillain-Barre syndrome classically presents as quickly ascending motor paralysis, beginning symmetrically in the legs after acute respiratory or gastrointestinal infection, most commonly with Campylobacter jejuni. GBS rarely follows a urinary tract or Clostridioides difficile infection. Moreover, facial diplegia characterizes a rare GBS facial variant, making it a further diagnostic challenge.

Case Presentation: A 68-year-old woman with hyperlipidemia, hypertension, and history of left lower extremity ischemia (status-post percutaneous angioplasty for left superficial femoral artery occlusion, on dual antiplatelet therapy) presented to the ED with acute-onset right leg weakness, right facial droop, and dysarthria for one day. NIH stroke scale score was 7. She had recently received nitrofurantoin and ciprofloxacin for Proteus mirabilis UTI and was undergoing oral vancomycin treatment for Clostridioides difficile-associated diarrhea. CT angiography showed no acute intracranial abnormalities but revealed high-grade left vertebral artery stenosis and mild-moderate right cavernous ICA narrowing. She was admitted to Medicine for ischemic CVA evaluation.
On physical exam, she exhibited 3/5 right lower extremity strength, notable right facial weakness with droop, and relative forehead sparing; however, she retained 5/5 strength in her deltoids, biceps, and triceps. This unusual right arm sparing suggested two simultaneous, separate ischemic lesions within the brain may be responsible. Cardiac source for ischemic CVA was unlikely, as TTE and telemetry revealed no interatrial shunt or paroxysmal atrial fibrillation. Therefore, either two separate intracranial thrombotic events - given her extensive multifocal atherosclerotic disease - or emboli from a left vertebral artery occlusive plaque could mechanistically explain two separate lesions and sparing of her right arm. However, MRI revealed no evidence of acute infarct or other intracranial abnormality.
Two days after admission, she experienced progressive facial weakness extending to the right forehead and spreading bilaterally, suggesting a peripheral - rather than central - pathology. CSF was negative for Lyme, AFB, fungus, HSV, VZV, and VDRL. Prednisone was initiated for suspected idiopathic bilateral Bell's palsy. Persistent facial and leg weakness, as well as new-onset bilateral paresthesia in her feet and hands, prompted electromyography. EMG demonstrated peripheral polyneuropathy, acute axonal loss, and demyelinating features concordant with an uncommon variant of Guillain-Barre syndrome known as facial diplegia with distal limb paresthesia. She was started on IVIG for five days and discharged to SNF with significant improvement in facial and lower extremity weakness and resolution of dysarthria.

Discussion: A rare facial variant of GBS in its early stages can mimic CVA, particularly in a patient with high atherosclerotic burden and risk factors. Knowledge of neurologic and vascular localization, with careful physical examination, distinguishes the two. Awareness of this entity is crucial to avoid misdiagnosis of CVA and to initiate proper treatment, especially for patients who would fall within the 4.5-hour window of thrombolytic therapy.

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Elevated Liver Enzymes in a Patient with COVID-19

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Introduction: Elevation of liver enzymes can be due to a variety of etiologies, including viral hepatitis, alcohol abuse, medications, and non-alcoholic fatty liver disease (NAFLD). Studies have suggested that SARS-CoV-2 primarily enters epithelial cells of alveoli via the human ACE2 receptor, which is also expressed by bile duct epithelial cells and, to a lesser degree, hepatocytes. There have been many reports of abnormal liver tests with COVID-19 infection, though the associated mechanism is not clear.

Case: A 53-year-old female presented to the ED with six weeks of dry cough, chills, SOB with minimal exertion, nausea, reduced appetite and taste sensation, weakness, fatigue, intermittent sore throat, intermittent chest tightness, headaches, body aches, and diarrhea, for which she had taken Tylenol 650 mg every other day with partial relief. She also endorsed NSAID use for chronic back pain. She tested persistently positive for COVID-19 for five weeks before presentation. She denied recent worsening symptoms, but reported to the ED as directed by employee health.

In the ED, the patient had elevated AST (388 U/L), ALT (237 U/L), alkaline phosphatase (960 U/L), and total bilirubin (2.7 mg/dL), and decreased albumin (3.1 g/dL) and total protein (6.0 g/dL). Her blood ethanol level was 24 mg/dL, although she reported drinking three glasses of wine per week for the past two years and no illicit drug use. Her acetaminophen level and serologies for hepatitis A, B, or C infection were negative. CT of the abdomen and pelvis showed decreased attenuation throughout the liver, consistent with hepatic steatosis. There was no evidence of gallbladder pathology. The patient's obesity (BMI 33.8), hypertriglyceridemia, and PSH of Roux-en-Y gastric bypass surgery pointed to underlying NAFLD.

The patient's liver enzymes began to downtrend after administration of IV fluids, encouraging PO intake, and avoidance of hepatotoxic medications. She was discharged after five days with AST 122 U/L, ALT 139 U/L, alkaline phosphatase 652 U/L, and total bilirubin 2.0 mg/dL.

Discussion: The etiology of this patient's presentation included elements of pre-existing NAFLD, hepatotoxic medications, alcohol, and COVID-19 infection. A study published in the Journal of Hepatology suggests that liver damage in COVID-19 may be caused directly by infection of liver cells. Of patients with liver test abnormalities, 20.75% had hepatocyte type abnormalities (AST and/or ALT greater than 3x upper limit of normal (ULN)), 29.25% had cholangiocyte type (alkaline phosphatase or GGT twice ULN), and 43.4% had mixed type. The greater presence of ACE2 receptors on bile duct epithelial cells compared to hepatocytes may explain the greater rate of cholangiocyte type injury. This may explain this patient's dramatic increase in alkaline phosphatase and notable increase in total bilirubin, though her concurrent increase in AST and ALT does suggest a mixed type of liver injury.

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A Tough Catch: Miller Fisher Syndrome Masquerading as Vitamin B12 Deficiency

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Guillain Barré syndrome (GBS) encompasses a spectrum of acute immune-mediated polyneuropathies. While typically presenting with ascending paralysis following infection, GBS variants present with heterogeneous symptoms. Diagnosis involves clinical suspicion in addition to lumbar puncture demonstrating albuminocytologic dissociation. Glycolipid antibodies are present in up to 60% of GBS patients. Anti-GQ1b antibodies are present in 80-90% of patients with Miller Fisher syndrome (MFS), a variant of GBS characterized by ataxia and ophthalmoplegia. This case reports a patient who presented with clinical features of vitamin B12 deficiency, later determined to have MFS.

A 55-year-old male with history of diabetes on metformin presented to the hospital with acute onset unsteady gait, extremity numbness, and diminished taste. He was diagnosed with sinusitis one week prior. Exam was notable for a broad based ataxic gait, heal-shin dysmetria, decreased distal vibratory sensation, and absent lower extremity reflexes. He was found to have a vitamin B12 level of 126 pg/mL (>300 pg/mL). Hemoglobin and hematocrit were normal. His dorsal column spinal cord deficits were initially attributed to vitamin B12 deficiency and supplementation was started; however, the acuity of his symptoms was inconsistent with B12 deficiency. An MRI of the brain and cervical and thoracic cord was normal. An LP was planned but delayed because the first attempt was unsuccessful. On hospital day two, the patient developed diplopia with a lateral gaze palsy. Lumbar puncture demonstrated eight nucleated cells, two RBCs, and a total protein of 46 mg/dL. Anti-GQ1b titer came back positive at 1:25600. The patient was treated with high dose IVIG with prompt symptom improvement. The patient was discharged with physical therapy for residual ataxia.

As seen in this patient, Miller Fisher syndrome can initially present similarly to vitamin B12 deficiency with paresthesia and ataxia. There are reports of MFS causing abnormalities in dorsal spinal column, similar to subacute combined degeneration of vitamin B12 deficiency. It has been demonstrated that brainstem involvement occurs with the GQ1b antibody syndrome as well as posterior spinal column involvement, resulting in ataxia and loss of vibratory sensation with preserved pain, temperature and light touch. The distinguishing ophthalmoplegia found in MFS presented late in this case, demonstrating the importance of monitoring patients with abrupt onset of ataxia. This case also exemplifies the importance of careful history taking, as his recent history of sinusitis and acute onset of symptoms increased our suspicion for MFS. Loss of taste is a common symptom of B12 deficiency; however, it is important to note that any cranial nerve can be involved in MFS and dysguesia is a rare presenting symptom. The diagnosis of MFS may not always be clear given the varied symptomology, however recognition and treatment of this disease is important to maximize patient outcomes.

Medical Student Clinical Research

Abstracts 500 – 507

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The Impact of Surgical Chronology on Outcomes of Patients Receiving Lumbar Spine and Lower Extremity Joint Surgeries

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Introduction
The aging population and obesity epidemic suggest that increasingly more patients with lumbar spine pathology will likely also develop lower extremity joint (LEJ) disease (hip and/or knee). These patients may have co-existing lower back and extremity pain, the major source of which (spine vs. LEJ) is difficult to determine. There remains a lack of outcomes data of patients who receive surgeries for both an LEJ and the lumbar spine within a short period based on chronology of surgery type. This is the first report that evaluates the differences in surgical outcomes of patients receiving lumbar surgery following LEJ surgery compared to receiving lumbar surgery before LEJ surgery.

Methods
Retrospective chart review was performed of patients undergoing lumbar surgery between 2008-2015 and receiving hip/knee surgery within three years prior/ following lumbar surgery at a single institution. Pre- and post-operative outcome measures were assessed using the EuroQol five dimensions questionnaire (EQ5D) and Pain Disability Questionnaire (PDQ).

Results
670 patients were included. Patients receiving lumbar surgery first were 4.75 times more likely to reach the minimal important difference in PDQ. Patients receiving LEJ surgery first had a higher mean Charlson Comorbidity Index. Male patients were 37% less likely to be readmitted within 90 days of either procedure.
Conclusion
Postoperatively, patients who received lumbar surgery before LEJ surgery had greater improvement than patients who received LEJ surgery before lumbar surgery. These findings suggest that in patients with a co-existing need for lumbar and LEJ surgery, greater consideration should be given to providing lumbar surgery before LEJ surgery. The findings of this study are applicable to multiple disciplines including primary care, orthopedics, and neurosurgery, and warrants further large-scale prospective investigation to determine the cause and generalizability of these surgical outcomes results.

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Written Prescription for OTC Non-Opioid Pain Medications Does Not Increase the Likelihood of Use After Ambulatory Hand Surgery

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Purpose: This study looked to determine how providing written prescriptions for non-opioids affects postoperative pain medication usage and pain control in patients undergoing outpatient hand and upper extremity surgery.

Materials and Methods: Patients undergoing hand and upper-extremity surgery (n = 244) were recruited at an outpatient hand and upper extremity clinic after implementation of a post-operative pain control program encouraging non-opioids before opioids. Patients were grouped based on procedure type: bone (n=66) or soft tissue (n=178). Patients reported postoperative medication consumption and pain control scores. Two-tailed t-tests assuming unequal variance were performed to look for differences in postoperative pain control and medication consumption between those who were and were not given written prescriptions for non-opioids.

Results: For both soft tissue and bone procedure patients, a written prescription did not significantly affect patients' postoperative pain control or medication consumption. Regardless of receiving a written prescription, patients who underwent soft tissue procedures consumed significantly more daily non-opioids than opioids ($p < 0.001$).

Conclusions: Receiving written prescriptions for non-opioids may not have a significant effect on postoperative pain control or medication consumption. Patients undergoing soft tissue hand and upper extremity procedures may be more likely to consume more daily non-opioids than opioids postoperatively compared to bone procedure patients regardless of whether they receive a written prescription for non-opioids. This underscores the importance of the implementation of an effective pain plan regimen emphasizing non-opioids as first line therapy for pain control for patients to adhere to after surgery. This may be especially important for hand and upper extremity patients undergoing bone procedures where the postoperative period may entail a longer duration of symptoms requiring pain relief.

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ROLE OF BILIRUBIN IN PROTECTION OF KIDNEY FUNCTION

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Introduction: Traditionally, bilirubin was considered merely a waste product of heme metabolism. More recent evidence, however, suggests bilirubin has antioxidant and anti-inflammatory effects, and may provide cardio-nephroprotection. Its effects in renal transplant recipients have not been well studied. We sought to determine the impact of bilirubin on outcomes after renal transplantation.

Methods: We retrospectively reviewed all transplants completed at a single transplant center from December 2014 to December 2018. Patients who had undergone prior transplantation were excluded. Pre- and post-transplant total, indirect, and direct bilirubin levels, eGFR, and rejection status were abstracted. eGFR was measured by MDRD method. Rejection was confirmed by transplant biopsy. Time to rejection and post-rejection eGFR were correlated with pre-rejection bilirubin levels. Data were analyzed using mixed model ANOVA in SAS statistical software.

Results: Most patients (n=279) were male (67.4%) and recipients of deceased donors (229/279; 82.1%). Lower pre-transplant direct bilirubin levels were correlated with higher post-transplant eGFR ($p = 0.006$). Lower post-transplant direct and total bilirubin levels were associated with higher post-transplant eGFR (both $p < 0.01$). Post-transplant direct and total bilirubin were negatively correlated with graft survival (both $p < 0.05$). 35 of 279 patients (12.5%) experienced a biopsy-proven rejection episode. Higher total bilirubin predicted a shorter time to rejection for those patients that experienced a rejection event ($p = 0.008$).
Conclusions: Our data suggest that higher bilirubin levels may predict poorer renal function, deceased graft survival, and faster time-to-rejection after renal transplantation. Additional research is needed to confirm these findings.

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Correlation Between Nitroglycerin-Induced Headaches and Coronary Artery Disease

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Background:
Nitroglycerin (NTG) is used to treat and prevent angina and is often given to patients experiencing chest pain in the emergency department (ED). NTG is thought to exert its vasodilatory effects in an endothelium-dependent manner. One of the most common side effects of NTG is headache, which is thought to be due to cerebral artery dilation that occurs within 10 minutes of NTG administration.

Objective:
The objective of this IRB-approved study is to investigate whether the presence or absence of headache within 10 minutes of NTG administration is indicative of coronary artery disease (CAD) status. We will also assess for contributing risk factors such as gender, race, diabetes, hyperlipidemia, hypertension, smoking history, or family history of CAD. We hypothesize that those with occlusive CAD (>/= 50% stenosis of any coronary artery) experience fewer headaches following NTG administration than those without occlusive CAD.

Methods:
Patients aged 18-100 presenting to the ED at a university medical center with a chief complaint of chest pain were enrolled in the study if they were given NTG either en route to the hospital or in the ED. After informed consent was obtained, patients were asked whether or not they experienced a headache after NTG administration, and if so, its timing of onset, severity, location, and quality. Patient demographics, risk factors, and cardiac test results were obtained from the electronic medical record. Patients found to have a positive history of migraines were excluded from the study. A total of 29 patients were analyzed with Chi-squared tests through IBM SPSS software.

Results:
Overall association between lack of headache and occlusive CAD was borderline significant ($P = 0.057$). Lack of headache was found to be a significant predictor of occlusive CAD for females ($P = 0.024$) but not for males ($P = 0.297$). The absence of headache following NTG was also a significant predictor of occlusive CAD in Caucasian ($P = 0.014$) and diabetic patients ($P = 0.03$) as well as those with a smoking history ($P = 0.045$).

Conclusions:
These results suggest that lack of a headache following NTG administration may be predictive of the level of CAD in certain population

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Projecting primary care shortages secondary to the COVID-19 pandemic

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Introduction: The COVID-19 pandemic disproportionately affects individuals over age 60. As a result, physicians over age 60 are at increased risk of discontinuing their practice, whether due to severe disease course, early retirement, or decreases in productivity because of transitions to virtual clinics. A shortage of primary care physicians (PCPs) already persists across the nation. Here, we use COVID-19 migration models and workforce data to project which U.S. states will face the largest decreases in access to primary care by 1 October 2020.

Methods: Publicly available workforce data (AAMC) and SARS-CoV-2 infection data (Johns Hopkins University) were combined with a COVID-19 migration model (University of Washington) using the R programming language to identify areas of current PCP shortage and predict areas of PCP shortage by 1 October 2020. All data was standardized to values per 100,000 population to avoid bias favoring populous states. Since the data involves populations, not samples, exploratory analysis without statistical inference suffices. A fully reproducible pipeline is available at https://github.com/rrrlw/covid-primary-care.

Results: As of 4 June 2020, there were nearly 2 million cases (cumulative) of SARS-CoV-2 infection in the United States with a projected increase to over 20 million by 1 October 2020. 34% of practicing U.S. PCPs are over age 60. The U.S. states with the highest number of PCPs over age 60 (per 100,000 population) are Vermont (54), Maine (46), Hawaii (41), New Hampshire (40), and New York (40). As of 4 June 2020, New Jersey has the highest ratio of SARS-CoV-2 cases per PCP under age 60 (28), followed by New York (26), Connecticut (18), Rhode Island (17), and Louisiana (16). Using current COVID-19 migration projections, the states with the most SARS-CoV-2 cases per PCP under age 60 by 1 October 2020 will be North Carolina (262), New Jersey (261), Nebraska (258), New York (253), and Utah (220).

Conclusion: Many states will soon have over 200 cases of SARS-CoV-2 infections per PCP under age 60. Although PCPs over age 60 – over one-third of all PCPs – will contribute to patient care, decreased productivity as a consequence of the COVID-19 pandemic will certainly increase barriers to accessing primary care, with large variations between states. These barriers are currently highest in the East Coast region of the United States and are projected to spread to the Pacific Northwest and Southern regions within a few months. Primary care funding programs (e.g. National Health Service Corps scholarships) should account for this geographical variation to effectively address the impending burden of PCP shortfalls. Otherwise, reduced primary care access will exacerbate an already concerning shortage.

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The Effect of PDE-5 Inhibitors in Renal Allografts

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Renal transplantation (RT) recipients are often prescribed calcineurin inhibitors (CNIs), immunosuppressive drugs that mitigate allograft rejection. CNIs are effective in increasing renal allograft survival, but long-term usage produces serious side effects for the allograft. Phosphodiesterase-5 inhibitors (PDE5i), vasodilatory agents used to treat erectile dysfunction (ED), prevent degradation of cyclic guanosine monophosphate (cGMP), a secondary messenger necessary for NO-induced vasodilation. The nephroprotective effects of PDE5i in renal allografts have previously been established in animal models. The purpose of this study was to determine whether oral administration of the rapid-acting PDE5i, avanafil, reduces renal allograft resistive indices (RI). Such improvements in RI would benefit RT patients with ED by potentially prolonging allograft survival.

Male RT patients with ED over age 18 were included in this two-phase study. In both Phase 1 and Phase 2, participants were screened with the sexual health inventory for men (SHIM), baseline vital signs were recorded, a comprehensive metabolic panel was drawn, and a transplant Doppler ultrasound performed. In Phase 1 (n=10), patients were administered 100mg of avanafil and re-examined after 30-minutes. Phase 1 had a mean age of 59.1 years, SHIM score of 12.2, and 5.2 years duration since RT; 80% of patients were using CNIs. In Phase 2 (n=4), patients were administered 200mg of avanafil and re-examined after 60-minutes. Phase 2 had a mean age of 55 years, SHIM score of 8.7, and 7.75 years duration since RT; 75% of patients were using CNIs. Upon re-examination, each participant's vital signs were re-measured, and a repeat transplant Doppler ultrasound was performed.

Results indicate that in Phase 1, RI were statistically significantly increased from baseline at the kidney's upper, middle and lower poles 30-minutes after 100mg avanafil administration. While in Phase 2, RI was statistically significantly decreased at the kidney's upper pole 60-minutes following 200mg avanafil administration—decreases were not statistically significant at the middle and lower poles, but suggest a trend towards a decrease in RI.

Future directions include increasing Phase 2 study enrollment to 10 participants, assessing the impact of daily administration of a long acting PDE5i on RI (Phase 3), and longitudinally tracking renal allograft outcomes via creatinine levels in a randomized double-blind controlled trial in patients using a PDE5i vs. placebo (Phase 4).

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The effect of number of imputations on accuracy predicting thirty-day all-cause mortality after transvenous lead extraction

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Introduction: Data sets used to develop new clinical risk prediction tools are often incomplete. To avoid possible selection bias by including only complete cases, missing values can be imputed by multiple imputation by chained equations (MICE). However, MICE can be computationally intensive in terms of time and storage, and there is significant heterogeneity in how many imputations are performed in these analyses, even within a single institution. Here we assess how the performance of a prediction tool generated from real patient data varies with the number of imputations.

Methods: A prediction tool for thirty-day all-cause mortality in patients undergoing transvenous lead extraction (TLE) was developed from a dataset of consecutive patients with transvenous pacemakers who underwent the procedure at the Cleveland Clinic between 1996 and 2011. Missing values in this data set were imputed using MICE over five, ten, twenty, and fifty repetitions. For each series of imputations, multivariable logistic regression models were built independently on each copy of the imputed dataset and subsequently pooled. Discrimination and calibration were assessed for each pooled model using bootstrapping for internal validation.

Results: A total of 5521 (4137 pacemaker and 1384 defibrillator) leads were extracted during 2999 TLE procedures (patient age 67.2 [55.2, 76.2] years, 30.2% female). Sixty-seven patients (2.2%) had died by thirty days after TLE. Variables included in the complete model with missing values included BMI, New York Heart Association functional class, left ventricular ejection fraction, and hemoglobin. The range of missing values in these variables was 15.2%-33.2%. After imputation, the bootstrap-corrected concordance indices for the pooled models were 0.871 for five imputations, 0.866 for ten imputations, 0.863 for twenty imputations, and 0.875 for fifty imputations. The bootstrap-corrected Brier scores for the pooled models were 0.0201 for five imputations, 0.0202 for ten imputations, 0.0201 for twenty imputations, and 0.0202 for fifty imputations. All pooled models were well-calibrated.

Conclusion: Thirty-day all-cause mortality after TLE could be predicted with good discriminative power from this data set. Pooled models did not vary greatly in discrimination or calibration with the number of imputations. When the proportion of missing information is moderate, reliable inference may be made after relatively few imputations.

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ASSOCIATIONS BETWEEN PHYSICAL ACTIVITY AND RISK FACTORS FOR TYPE II DIABETES IN PREDIABETIC ADULTS

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Diabetes is a national healthcare crisis related to both macrovascular and microvascular complications. We hypothesized that higher levels of physical activity are associated with lower total and visceral fat mass, lower systolic blood pressure, and increased insulin sensitivity. Participant inclusion criteria: 21-50 years old, BMI ≥ 30 kg/m2, hemoglobin A1C 5.7-6.4, fasting glucose 100-125 mg/dL, and HOMA IR ≥ 2.5. Exclusion criteria: history of diabetes, hypertension, HIV, renal disease, hearing loss, alcoholic intake over four drinks daily, use of organic nitrates or PDE5 inhibitors, and decreased cardiac function. Total physical activity was measured using accelerometers, body composition using DXA, and insulin resistance via fsIVGTT. Clinical and biochemical cardiometabolic risk factors, blood pressure and heart rate obtained using a calibrated sphygmomanometer. Anthropometric measures, fasting glucose, insulin, lipid profile, C-reactive protein, and BMP analyzed using standard procedures. Within our study, we found correlations between levels of physical activity in a heterogenous group of prediabetic adults. Patients with more physical activity had a higher degree of insulin sensitivity, lower blood pressure, total visceral adipose tissue, and overall lower total mass. Total physical activity levels showed small, but significant correlations with systolic blood pressure, visceral fat, lean mass and insulin sensitivity. After normalizing for race, age, and gender using multiple regression, these associations were no longer significant considering our small sample size. More research into prediabetes will decrease the population of diabetics overall. In the future we could increase sample size and conduct cross sectional and longitudinal studies in various populations with prediabetes.

Resident/Fellows High Value Cost Conscious Care

Abstracts 600-602

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The Effect of the COVID-19 Pandemic on Geriatric Depression

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Background: The COVID-19 pandemic in Spring 2020 brought a period of encouraged social distancing and a stay at home order for Central Ohio residents. Research has shown much is still unknown about COVID-19, but the elderly are known to be a particularly vulnerable population1, leading to a higher rate of social distancing in older adults. The geriatric population is known to experience minor depression at a prevalence between 4-13% and major depression between 1-4%,2 with the prevalence among geriatric women up to twice that of geriatric men.3 Studies have shown that low social support, marital/relationship status, and social isolation influence disease mortality and psychological processes in the geriatric population.4,5

Objective: This study was performed to assess the level of depression in geriatric patients during the COVID-19 Pandemic compared to prior depression screenings.

Materials and Methods: This prospective cohort study was performed at the Internal Medicine clinic in a Primary and Specialty Care office. The nursing staff began by completing ìGeriatric Check-Inî phone calls, documented as a nurse visit, to the Internal Medicine patients above the age of 60 years old. During the phone call, patients were asked about general depression symptoms, access to food, exercise, and overall well-being. A chart review was then completed on the check-in visits to determine if certain parameters such as having a pet, food access, physical activity, and living with others affects the presence of depression in this vulnerable population.

Results: There were 30 patients contacted by phone between 4/21/2020 and 5/13/2020. The mean (SD) age was 67.8 (6.9) with a range of 60-85. Nine (30%) patients were female; 21 (70%) were male. Five (55.6%) of the nine females had a PHQ-2 score of zero compared to 19 (90.5%) of the 21 males (p=0.0496). Statistical analysis determined that the PHQ-2 scores taken during the COVID-19 Pandemic were not significantly different from prior (p=0.8750).

Conclusions: Males were significantly more likely to have a PHQ-2 score of zero compared to females. The level of depression in geriatric patients during the COVID-19 Pandemic did not significantly change compared to prior PHQ-2 depression screenings.

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Healthcare Economic Burden in Adult Patients with Pancreatitis

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Significance: While episodes of acute pancreatitis can often be managed with supportive care, severe episodes, the development of complications, or progression to chronic pancreatitis can lead to advanced inpatient and procedural care. In this study we look to investigate the healthcare economic burden of pancreatitis and factors that drive increased costs.

Design: The Medical Expenditure Panel Survey (MEPS) was used to retrospectively analyze patient data from 2007-2015. MEPS provides a data set that compiles data on the cost and usage of health care, as well as health insurance coverage for a nationally representative population. The study population included 221,273 respondents (adults aged >18 years), 599 of whom reported having pancreatitis. We used a cross-validated 2-part generalized linear regression model to estimate the incremental pancreatitis related expenditures independent of respondent demographics and comorbidities. Furthermore, we examined specific healthcare service sectors (inpatient, outpatient, emergency room, home health, and medications) in addition to total expenditures.

Results: Mean expenditures for cohorts with and without pancreatitis were $18031.91±2260.98 and $5362.53±60.39, respectively. Cost of care for patients with pancreatitis was 2.05 times higher (95% CI, 1.69, 2.48; $p<0.001$) than patients without pancreatitis after controlling for comorbidities and socioeconomic factors. This suggests an additional annual healthcare burden of $9235.86 for patients with pancreatitis compared to individuals without pancreatitis. The health care service sectors that saw a difference in expense between patients with and without pancreatitis were inpatient services (OR 3.14, 95% CI 2.52, 3.90; $p<0.001$), emergency rooms (ER) (OR 2.04, 95% CI 1.61, 2.59; $p<0.001$), and medication expenditures (OR 1.28, 95% CI 1.03, 1.60; $p<0.029$). When looking further at the trends of inpatient and ER costs, demographics that led to significantly increased costs in these settings included African American race, any income bracket outside of high income (4x federal poverty limit), public insurance, and increasing Charlson Comorbidity Index. Of these demographics, race and income did not play a significant role in determining total costs.

Conclusions: These results suggest that patients with pancreatitis have a significantly increased economic burden on the healthcare system compared to those patients without pancreatic disease. Inpatient hospital management and ER visit expenditures were where the bulk of the economic burden was increased, with medication expenditures also contributing significantly. Initial management of pancreatitis after an acute episode involves lifestyle modifications such as the cessation of alcohol or tobacco usage and dietary modifications. Strategies that improve patient education and adherence to these lifestyle modifications, particularly African American and those of poorer income populations, may help reduce recurrent episodes, progression to chronic disease, and the development of complications in these patients. Further research into why these populations experience increased cost would be beneficial in reducing the disproportional healthcare burden of pancreatitis.

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Trends in Health Care Expenditure among U.S. Adults with Gout

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Significance: Although the management of gout primarily takes place in the ambulatory setting, flares and comorbidities lead patients to require care in emergency rooms and, even, inpatient settings. There is limited population-based national data on the trends in expenditures related to gout across healthcare services. We aim to estimate the incremental healthcare burden of gout, controlling for comorbidities and sociodemographic factors, and offer insights into the cost drivers behind this burden.

Design: This is a retrospective analysis of the Medical Expenditure Panel Survey (MEPS) patient data from 2007-2015. MEPS is a nationally representative dataset for the noninstitutionalized US population. This study analyzed 221,273 respondents (adults aged ≥18 years) and 3096 patients with gout (1.4% of respondents) between 2007 and 2015. A cross-validated 2-part generalized linear regression model estimated the incremental gout expenditures independent of demographics and comorbidities. Healthcare service sectors (inpatient, outpatient, emergency room, home health, and medications) and total expenditures were assessed.

Results: The cohorts with and without gout had mean expenditures of $12000.44±1365.87 and $5294.28±116.47, respectively. Patients with gout were more likely to be male (68% vs 46%), elderly (45 vs 16%), non-Hispanic (90% vs 73%), residing in the South (42% vs 38%), publicly insured (Medicare/Medicaid) (33% vs 22%), and have a higher Charlson Comorbidity Index ($p<0.001$ for all). Controlling for comorbidities and socioeconomic factors, cost of care for patients with gout was 1.36 times higher (95% CI, 1.22-1.52; $p<0.001$) than patients without gout. After accounting for demographics and comorbidities, patients with gout incur an additional annual healthcare burden of $3176.59 (95% CI, 2164.01-4105.41, $p<0.001$) when compared to individuals without gout, translating into an additional $13.3 billion in population-level US healthcare expenditures. Gout patients had higher total healthcare spending than those without gout across the 9-year span analyzed, as well as higher expenditure associated with inpatient care ($p<0.001$), outpatient care ($p=0.029$), and prescription medications ($p<0.001$), controlling for demographics and comorbidities. Between 2007 and 2015, there was 38% increase in emergency care expenditures for patients with gout ($p=0.042$).

Conclusions: Gout patients have substantially increased incremental economic healthcare burden after accounting for demographics and comorbidities. Higher healthcare expenditure were across inpatient and outpatient settings and with services such as prescription medications. A recent increase in emergency care expenditure among those with gout is consistent with a similar increase among those without gout. Strategies aimed at lowering utilization and expenses for this patient population in settings such as the emergency room and the inpatient setting may help alleviate the higher economic burden associated with the condition. Increasing frequency of outpatient follow-up, monitoring medication adherence, and patient education about avoidance of triggers are suggestions that can aid in prevention of flares and disease complications among this patient population.

Medical Students Quality Improvement

Abstracts 700-702

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Provider Performance and Patient Satisfaction of Telehealth During the COVID-19 Pandemic

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Background: The unprecedented SARS-CoV-2 pandemic thrusted the relatively novel approach of telemedicine to the center stage of healthcare infrastructures within the United States and around the globe, leading to a dramatically increased utilization of telehealth services. Since the first reported case of SARS-CoV-2 in the U.S. on January 20, 2020, telemedicine has played an instrumental role in reducing viral transmission by preserving personal protective equipment and supporting crippled healthcare revenue streams. The convenience of telehealth services has also reduced caregiver burden and travel costs for patients. Prior to the SARS-CoV-2 pandemic, studies have consistently demonstrated a strong association between telehealth utilization and increased patient satisfaction. The impact of telemedicine on patient satisfaction during the current pandemic has yet to be fully understood.

Objective: This study aimed to identify patient perspectives and satisfaction with the virtual appointments during the SARS-CoV-2 pandemic to help guide future implementation of telehealth services. We also attempted to discern statistically significant differences between the utilization of video conferencing versus telephone-only telehealth visits.
Materials and Methods: This prospective patient survey study was performed at a Primary and Specialty Care Clinic in Columbus, Ohio on patients seen in April and May of 2020. Patients completed a survey either in person at their in-office visit or were contacted by phone following their telehealth visit. The survey analyzed multiple satisfaction metrics including friendliness of registration staff, convenience of appointment times, and communication with physicians.

Results: Between April 17th – May 1st, 2020, the no-show rate for telehealth visits (7.5%) was statistically significantly lower than in-office visits (36.1%) ($p<0.0001$) and lower than the baseline non-SARS-CoV-2 no-show rate of 21.9%. Patients who had telehealth visits had similar overall satisfaction metrics to those who had in-office visits with the exceptions of the registration staff not being rated as friendly during telehealth visits ($p=0.0479$) and patients feeling like they did not spend as much time with their doctor during telehealth visits ($p=0.0018$). There were no statistically significant differences in overall satisfaction between patients who had a video telehealth visit versus those who had a phone-only telehealth visit. The most frequently suggested improvement from patients was related to improving technology (20% of patients).

Conclusions: Telehealth appointments resulted in comparable and positive overall patient satisfaction metrics with a significant reduction in missed appointments compared to those who had in-person office visits, despite registration staff not being rated as friendly and patients feeling like they did not spend as much time with their doctor during telehealth visits compared to in-office visits. There were no differences in satisfaction metrics between patients who had a video versus a phone-only telehealth visit. Collectively, this data strongly supports the widespread utilization of telemedicine during and following the SARS-CoV-2 pandemic.

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Analyzing Trends in Patients with Ureteral Stents Who are Lost to Follow-up

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The ureters are muscular vessels that run from the kidneys to urinary bladder and act as a conduit for flow of urine into the lower urinary tract. Ureteral stents are devices that are inserted into the ureter to maintain flow of urine or debris into the bladder and/or to reestablish the passage. Common indications for ureteral stents are to relieve or prevent intraluminal obstruction caused by calculi (stones) and genitourinary malignancies or obstructions, typically caused by compression of the ureter by a related disease process1,4. Generally, ureteral stents are made of silicone based material3. The stent itself may also be coated to be dissolvable or to help administer medications locally7. Ureteral stents are also associated with potential risks and adverse events. Common complications of ureteral stents include urinary tract infections (UTI), stent migration and stent encrustation4,5. The risk of stent encrustation increases the longer the stent remains in the ureter. Stent retention is a rare but serious complication associated with failure to remove the stent in the indicated timeframe2. For similar reasons, self-removal can also lead to complications3. As such, this study explored potential risk factors for retained and self-removed stents. A retrospective chart review using Clinical PortalTM and AthenaTM was performed to examine follow-up compliance, specifically age upon stent placement, age upon stent removal, race, sex, chronic versus first-time stents, urological status, patient zip code, 12 month UTI history, and drug use. August 2016 to August 2017 222 patients were found to have underwent stenting procedure(s), of which 172 qualified for this study. Patients were considered lost to follow-up if the stent was either self-removed or there was no record of the patient for follow-up stent removal. Patients that expired with stents in were excluded from the dataset. Multivariable analysis was performed to determine predictors of loss to follow-up. Females were more likely than males to miss their follow-up for stent removal (12% vs. 20.5%, $p<0.05$). In the 41-70yrs old range, there is an increase in non-compliance (9% increasing to 15%, $p<0.05$). Patients either younger or older than this range were approximately 25% likely to miss their follow-up, suggesting that middle-age individuals are more compliant. The role of race in non-compliance is difficult to determine, other than that African-Americans are just as likely as Whites to be non-compliant. In the future, we will expand dataset to include patients past mid-2017 so that more conclusive results can be delivered on the other aforementioned parameters and increase the study power.

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The I in Team: Integrated Rounds are Changing the Game

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Intro: In the hospital setting, there exists a positive correlation between the level of professional engagement felt by staff members and the level of reported satisfaction from the patients with whom they interact. The rise of productivity, retention, innovation, and intimate interaction that comes with increasing employee engagement can have a profound impact on not only the patient themselves, but an entire healthcare system when considering patient-centered quality metrics and their effect on both ratings and reimbursements. Interested in improving some of these outcomes, our hospital implemented an integrated, structured, and multidisciplinary rounding system. We hypothesized that the implementation of our own interdisciplinary rounding at the University of Toledo Medical Center, entitled "Rocket Rounds" would lead to increased employee engagement

Methods: To measure employee engagement, we used the criteria set forth by the ADP Research Institute of People and Performance (ADPRI). Using their own global study of employee engagement, ADPRI isolated five parameters as measured by eight specific survey questions which affected the likelihood an employee was engaged: trust in leadership, being on a team, sense of purpose, team support, and prospects of future success. Using these specific questions, we created a survey and distributed it to hospital staff members to measure the differences in employee engagement between floors who had and had not implemented Rocket Rounds.

Results: 82 hospital providers took the survey, with 35 providers identifying as having participated in Rocket Rounds and 47 providers specifying that they did not participate in Rocket Rounds. Only providers who answered "strongly agree" to questions qualified as exhibiting the parameters being measured. Our results showed that those participating in Rocket Rounds were 16% more likely to trust their leader, 23% more likely to have a strong sense of purpose, 6% more likely to identify as a member of a team, 2% more likely to feel supported, and 10% less likely to have a positive sense of the future when compared to those who did not participate in Rocket Rounds.

Discussion: Rocket Rounds improved nearly every ADPRI parameter affecting employee engagement. According to ADPRI, the two strongest indicators of employee engagement were having trust in leadership and being a member of a team, which increased the likelihood that an employee was engagement by factors of five and two, respectively. These were two areas that Rocket Rounds improved staff response. Moving forward, we would like to look at different measures of outcomes to analyze if improving employee engagement through Rocket Rounds is truly affecting levels of patient satisfaction and quality measures like HCAHPS in our hospital. In all, we believe we have identified interdisciplinary rounding as a way to wield employee engagement as a tool for the betterment of hospitals and patients alike.

Resident/Fellows

Oral Presenters

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Lance-Adams Syndrome: A Challenging Diagnosis and Management

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It is uncommon for a patient who survives cardiac arrest to experience significant neurologic impairment such as acute and chronic post-hypoxic myoclonus. This chronic post-hypoxic myoclonus is also known as Lance-Adams syndrome (LAS). LAS was first reported by Lance and Adams in 1963 after they observed muscle cramps characteristic of patients who survived cardiac arrest. Less than 150 cases of Lance-Adams syndrome have been recognized worldwide. We present a rare case of Lance Adams syndrome below.

A 41-year-old male with history of drug abuse and suicide attempts was admitted on 5/15/2020 after being found down and unresponsive. Cardiopulmonary resuscitation was done by EMS and he was given 12 mg of Narcan with minimal response. Urine drug screen was positive for opiates and cocaine. The patient was intubated and sedated. The patient was noted to follow commands off sedation and had diffuse intention myoclonic jerking. Patient was loaded with Keppra followed by maintenance dosing while he was hooked up to long-term monitoring for epilepsy. The patient was extubated after 4 days and was alert and oriented to person, time, and place. He did not have any neurological deficits but continued to have myoclonic jerking. Electroencephalography was reflective of a moderate to severe encephalopathy with no epileptiform discharges. MRI brain was unremarkable. CT thoracic and lumbar spine showed multilevel spondylosis but no fracture. Action myoclonus continued and the patient was discharged on Depakote due to concern of LAS.

Post-hypoxic myoclonus can occur both in acute and chronic types. It may occur after the patient remains in a hypoxic coma for several hours to days. As the patient regains consciousness, he may immediately develop action myoclonus, or it may develop in a short period of time. Patients can have action and reflex myoclonus, which subside with rest. Other symptoms, such as ataxia and mental status changes can frequently be seen. Cerebrospinal fluid analysis often shows decreased serotonin metabolites. The physiology of chronic post-hypoxic myoclonus is generally both cortical action and cortical reflex myoclonus. Failure to diagnose LAS may lead to inappropriate therapy and delayed treatment. If a patient develops intention myoclonus after receiving cardiopulmonary resuscitation and regaining consciousness but shows minimal response to anticonvulsants, a possibility of Lance-Adam Syndrome should be considered. This can lead to minimizing disabilities and improved prognosis. When a patient is suspected of having LAS, aggressive drug treatment to reduce the myoclonus and rehabilitation to prevent disabilities are necessary.

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Significant Hemopericardium with Subsequent Cardiac Tamponade Secondary to Rivaroxaban

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Introduction: The use and utility of novel oral anticoagulants (NOAC) has been increasing in clinical practice due to their relatively lower incidence of side effects such as intracranial hemorrhage, particularly in the elderly, when compared to Vitamin K antagonists. Despite an additional risk of gastrointestinal bleeding, there is an overall lower risk of thromboembolic events. In addition, a predictable clinical profile along with a lack of need for periodic monitoring makes them an ideal option for patients with baseline debility or dementia. Rivaroxaban is a factor Xa and prothrombinase inhibitor indicated for stroke and venous thromboembolism prophylaxis in non-valvular atrial fibrillation as well as treatment of venous thromboembolism based on the ROCKET AF, EINSTEIN DVT, and EINSTEIN PE studies. Hemopericardium is not listed as a known side effect, but previous cases of hemopericardium secondary to rivaroxaban have been described in the literature. Here we present a case of hemopericardium presumably secondary to Rivaroxaban use.

Case presentation: An 84 year-old male with a past medical history of paroxysmal atrial fibrillation on rivaroxaban, chronic anemia and leukopenia, coronary artery disease presented with general malaise, lightheadedness and dizziness. The patient was previously worked up for chronic anemia via colonoscopy which was unremarkable. Laboratory findings were significant for hepatic and renal dysfunction. Chest radiography showed worsening cardiomegaly. Due to hemodynamic compromise and impending respiratory failure, he was transferred to the intensive care unit where he was found to have a significant pericardial effusion with tamponade physiology. Following urgent reversal of his INR with prothrombin concentrate, the patient successfully underwent pericardiocentesis which yielded 1.5 liters of grossly hemorrhagic fluid. He was noted to have new onset LV dysfunction and severe mitral regurgitation following his procedure. Workup for malignancy was negative.

Conclusions: Despite an overall favorable pharmacokinetic and side effect profile compared to previous agents, Rivaroxaban can have potentially significant hemorrhagic complications necessitating urgent reversal and intervention. A wider differential with higher clinical suspicion for less likely sequelae such as hemopericardium should be considered in patients who are taking novel anticoagulants and present with nonspecific symptoms, due to the possibility of rapid decompensation. Other novel anticoagulation medications that have been associated with hemopericardium include Apixaban and Dabigatran. Typically, grossly hemorrhagic pericardial fluid is suggestive of underlying malignancy and, given our patient's history of chronic anemia, a thorough workup was conducted which ruled out this possibility. The worsening cardiomegaly on chest radiography and history of chronic anemia also suggested a subacute to chronic process. Typically, anti-Factor Xa chromogenic assays are used to measure the drug concentration of Rivaroxaban but these levels reportedly do not have any direct correlation with anticoagulant activity further emphasizing the need for careful assessment of concurrent medications and supplements for potential interactions.

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The Prevalence of Venous Thromboembolism and its Impact on the Severity of COVID-19: A Systematic Review and Meta-analysis

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Introduction: Coronavirus Disease 2019 (COVID-19) is associated with a hypercoagulable state, which can result in venous thromboembolism (VTE). The purpose of our meta-analysis was to investigate the prevalence of VTE and its impact on the severity of COVID-19.
Methods: We performed a comprehensive search in the literature for studies that evaluated VTE in COVID-19. We searched the databases of PubMed/MEDLINE, Embase, World Health Organization COVID-19 Database, LitCOVID, and Web of Science Core Collection databases from January 1, 2020 until May 11, 2020. The search was not limited by language, study design, or country of origin. Two researchers (SG and YK) independently selected the studies; discrepancies were resolved by a third researcher (CN). We considered randomized controlled trials, cohort studies, case-control studies, and case series. We excluded animal studies, case reports, reviews, editorials, and letters to editors. We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The random-effects model was used to calculate the prevalences, odds ratios (OR), and confidence intervals (CI). Our main outcomes were the prevalence of pulmonary embolism (PE), the prevalence of deep venous thrombosis (DVT), and the need for intensive care unit (ICU) admission based on the presence of PE or DVT. Publication bias was assessed visually by generating a funnel plot of the studies that reported the prevalence of PE and DVT. We also performed Egger's regression to quantitively assess publication bias, where $p < 0.05$ was considered statistically significant for publication bias.
Results: We included 13 studies with a total of 1321 patients. The prevalence of PE was 17.4% (95% CI: 11.1% - 23.7%) and the prevalence of DVT was 14.4% (95% CI: 8.7% - 20.1%). The percentage of patients who required ICU admission was significantly higher in patients who had PE compared with patients who did not have PE (69.2% vs 22.7%, OR: 5.8, 95% CI: 3.2 – 10.6, $p < 0.001$, $I2 = 3.8\%$). The percentage of patients who required ICU admission was significantly higher in patients who had DVT compared with patients who did not have DVT (81.3% vs 18.4%, OR: 10.3, 95% CI: 1.3 – 83.0, $p = 0.029$, $I2 = 70.5\%$). There was visible symmetry in the funnel plot of the studies that reported the prevalence of PE and DVT, suggesting no publication bias. Similarly, Egger's test was not statistically significant implying no publication bias for the prevalence of PE ($p = 0.51$) or DVT ($p = 0.79$).
Conclusions: In conclusion, COVID-19 patients have a high prevalence of VTE. VTE can worsen the clinical course of COVID-19 and result in higher rates of ICU admissions. We recommend testing for VTE when clinically appropriate, especially when COVID-19 patients fail to improve clinically.

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Hypertension Control in a High Risk Population: Multidisciplinary QI Project

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Introduction: Hypertension (HTN) affects 1 in 3 Americans and is often poorly controlled, with 52% of hypertensive Medicare patients having uncontrolled hypertension (>140/90). Controlling HTN remains one of the most effective ways to reduce risk for cardiac disease and stroke. We report the outcomes of a two-year QI project that involved close multidisciplinary follow-up of uncontrolled HTN patients in a federally qualified, resident run clinic with the goal of increasing the percentage of patients with controlled HTN by 50% over 24 months.

Method: All patients presenting to the Five Rivers Health Center (FRHC; Dayton, Ohio) with diagnosed HTN were followed serially. Resident physicians were instructed to provide HTN education, prescribe antihypertensives and refer all patients with uncontrolled HTN (>140/90) for a two-week multidisciplinary visit for BP monitoring and further medication titration. Patients were provided with BP monitor logs with remote access monitoring. Patients with persistently elevated blood pressures were referred for repeat two week visits until control was achieved. The percentage of patients scheduled for a two week follow-up visit and that achieved blood pressure control were calculated every month.

Results: An average of 342 HTN patients were seen per month from January 2018 to January 2020; 53% were African-American and 51% males. Prior to the intervention, 10% of HTN patients had close follow-up appointments and 42% had well controlled HTN. After the intervention, 72% of patients had close follow-up appointments (620% increase, p-value <0.001) and 57% had achieved control (36% increase, p-value <0.005). Of the patients that achieved blood pressure control, 75% of patients with a two week follow-up visit were controlled in 6 months as opposed to 30% of patients without two week follow-up. In the uncontrolled group, heart failure (4.6%, 95% CI -3.80-16.94%, P=0.31), diabetes (7.9%, 95% CI -7.04-23.96%, P=0.320) and smoking (12.1%, 95% CI -3.92%-28.16, P=0.146) did not have a significant causal relationship.

Conclusion: Implementation of a two-year multidisciplinary QI project resulted in a statistically significant increase in the follow-up of HTN patients (10% pre vs 72% post) and in the percentage of HTN control (42% pre vs 57% post). In patients that gained BP control, participation in close multidisciplinary follow-up yielded faster control at six months. This QI project allowed for the highest level of BP control ever seen at FRHC, but ultimately fell short of the goal of increasing BP control by 50% in 24 months. Our next steps include involving pharmacy and social work to address the social determinants of healthcare. This QI project illustrates the power of close multidisciplinary follow-up in increasing hypertension control in a high risk population served by a federally qualified, resident run clinic.