



New Jersey
Chapter

New Jersey Chapter
American College of Physicians
Residents Abstract Competition
2017 Submissions

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Marian Sidor	Bishoy Hanna, Amit Misra, Nanda Nair, Dominik Zampino	Atlantcare Regional Medical Center (Dominik Zampino)	Systolic carotid flow as a possible marker in prediction for volume responsiveness	<p>Introduction/Hypothesis: Calculating Systolic Carotid Flow (SCF, ml/min) in Common Carotid Artery (CCA) based on the systolic Volume Time Integral (VTI, cm) can be used as an alternative parameter to cardiac output (CO, L/min) in volume status assessment. Changes of SCF from baseline upon preload challenge with various maneuvers can be a useful tool in predicting volume responsiveness in patients with shock before they get overloaded with fluids. It is important to understand that patients in shock may not respond to fluids; therefore administration of early vasopressors is essential to maintain hemodynamic support. Maintaining hemodynamic stability is a crucial task for an intensivist, however this requires further research for the most objective, readily available and preferably, noninvasive methods of volume status assessment in the ICU. The primary focus of this study was to establish the correlation between CO and SCF as well as to examine a pattern of carotid flow changes upon different preload challenges by utilizing diagnostic ultrasound.</p> <p>Methods: In this study, we examined 20 healthy and hemodynamically stable subjects by using diagnostic ultrasound. Increased venous return was achieved by passive leg raise. Preload reduction was achieved by compressing one thigh and then both thighs simultaneously to decrease venous flow but sparing arterial blood flow. CO and SCF were measured at rest and then with each of the maneuvers described above.</p> <p>Results: This study demonstrated a direct correlation between CO and SCF of 0.67 with a p value < 0.001. The percentage of left ventricle output tract blood flow measured in the right CCA was approximately 10.9%. Assuming similar flow in both carotids, the total blood flow adds up to 21.8% in CCAs. The ratio of systolic volume flow to CO was very similar (21-22%), regardless of the preload challenging maneuver applied. Two leg raise test induced a 14.9% increase in CO (p<0.001), and a 15.6% increase in SCF (p<0.001). The one thigh compression test induced an 11% reduction in SCF (p<0.007). This test elicited a 6% reduction in CO (p<0.08). Comparatively, two leg compression test elicited an average of 19% reduction in CO (p<0.001) and 29.7% reduction in SCF (p< 0.001).</p> <p>Conclusions: This pilot study provides evidence to support the potential for the use of SCF as an alternate method to estimate CO, which may aid in predicting the volume responsiveness by preload challenge with different maneuvers. Our study also demonstrates the feasibility of venous leg compression testing for the induction of measurable reduction in venous return, which can be used for preload challenge and volume status assessment</p>

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Clinical Vignette	John Oghene	John Oghene, MD [1], Anokhi Patel, MD [1], Nanda Nair, DO[2]	Atlanticare Regional Medical Center (Dominik Zampino)	Bioprosthetic Aortic Valve Thrombosis Complicated by Embolic CVA	Degenerative valvular heart disease is becoming more prevalent in the aging population resulting in an increase in bio-prosthetic and mechanical valve replacement, with the former being predominant. A bio-prosthetic valve is recommended in any patient in whom anticoagulation is contraindicated or cannot be managed appropriately, however it is a reasonable option in individuals >70. Typically there is a higher risk of thromboembolic events within the first 90 days after bio-prosthetic aortic valve replacement (AVR) We report a case of an 86 year old male who received a bio-prosthetic aortic valve 6 years ago and received antiplatelet therapy with aspirin during the recommended postoperative period. He presented to the Emergency Department with a 5 day history of visual field deficit consisting of diminished peripheral vision of the right eye. He had no previous history of stroke, transient ischemic attack or atrial fibrillation. On physical exam, heart and lung exam was unremarkable with NIH score of 2. ECG showed sinus rhythm. MRI of the brain revealed an acute non-hemorrhagic infarct of the left occipital lobe. A TEE revealed an echogenic 1.4 cm mass on the ventricular side of the bio-prosthetic aortic valve consistent with a thrombus. The patient was started on anticoagulation therapy with intravenous heparin and oral warfarin to prevent further embolic events. He was subsequently discharged from the hospital on oral warfarin therapy. There is sparse literature on bio-prosthetic aortic valve replacement resulting in thrombosis and subsequent embolic cerebrovascular accidents. The optimal thrombo-prophylaxis of bio-prosthetic valve thrombosis still remains a matter of debate.

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Clinical Vignette	John Oghene	Siddharth Verma MD. Second Author: Edward Hamaty, DO	Atlanticare Regional Medical Center (Dominik Zampino)	Hypermagnesemia - A Case Report	<p>Introduction: Magnesium is an essential mineral nutrient that acts an important signaling ion for various biochemical reactions. In adults, 300 mg of magnesium is ingested daily out of which 25-75% is absorbed through the intestines. The kidneys filter approximately 2.1 grams of Magnesium daily. Hypermagnesemia occurs due to the excessive oral administration of magnesium containing salts, laxatives and antacids particularly in the elderly as renal function declines with age. We present a case of iatrogenic hypermagnesemia with unusually high magnesium levels.</p> <p>Case presentation: A 55-year-old woman was brought to the ER with altered mentation. This patient had a history of CVA with residual right hemiparesis, expressive aphasia, chronic dysphagia requiring a PEG tube for long-term nutrition and CKD Stage IIB/III with a baseline creatinine of 1.4. On presentation she was lethargic, hypotensive and bradycardic. She was unresponsive to verbal stimuli, minimally responsive to pain, her pupils were fixed and dilated, DTRs were absent and she had decreased muscle tone. GCS was 6. She was intubated and placed on mechanical ventilation. EKG showed sinus bradycardia of 58 b/min, first-degree atrioventricular block, QRS of 0.9 secs, QT of .48 secs and a PR of 0.23 secs. Laboratories revealed a potassium of 5.4, magnesium of 15.8 mg/dL, BUN of 65 mg/dL and creatinine of 1.68 mg/dL. CT head showed no acute intracranial changes. She was given IV normal saline, calcium gluconate 2 gm, IV naloxone 2 mg, kayexalate 30 gm and transferred to the ICU. In the ICU a repeat magnesium of 13 mg/dL confirmed the diagnosis of hypermagnesemia. Within 36 Hours after presentation, the patient became gradually more responsive and was successfully extubated. At that time, her serum magnesium level was 8.1 mg/dL. After further investigation, it was discovered that the patient was given 2 tablespoons of "Magnesium Oil" by mistake through the PEG tube, when her caretaker confused it with a bottle of aloe vera. The preparation contained 560 mg of elemental magnesium in each teaspoon as per the label. The patient's condition and serum magnesium levels improved after treatment with IV calcium, normal saline, and supportive therapy. At the time of her discharge, her cardiac, respiratory, and neurological function were back to her baseline.</p> <p>Discussion: The patient presented with classical symptoms of respiratory depression, lethargy, hyporeflexia and EKG changes consistent with hypermagnesemia. She initially had to be intubated for respiratory arrest. Her underlying poor renal function further complicated her electrolyte disturbances. The patient was managed with IV fluids and supportive management and her magnesium level gradually dropped from 15.8 to 3.65 in 3 days.</p>

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Clinical Vignette	Ahmed Elshazly	John Oghene M.D.; Scott Urban, D.O	Atlanticare Regional Medical Center (Dominik Zampino)	Dental extraction complicated by unilateral Facial Nerve Palsy	Bells palsy is a subacute weakness of the facial nerve that occurs in 22 to 33 per 100,000 people in the population annually. The etiology may be idiopathic, infectious, or even traumatic. Facial nerve paralysis due to dental extraction is extremely rare. This article illustrates the case of a 59-year-old male with past a medical history of type 2 diabetes mellitus who presented to the emergency department with complaints of unilateral facial droop that occurred 72-hours after extraction of his left lower molar. The physical examination revealed a complete left side facial droop, a normal intraoral exam (lack of abscess or swelling), a normal ear exam (considering Ramsay Hunt syndrome), and a normal neurological exam except for an isolated left sided peripheral CN VII deficit. Initial laboratory studies revealed a blood sugar of 309; otherwise his basic metabolic profile and complete blood count were unremarkable. CT of the brain and CT facial bones with IV contrast were both unremarkable. The patient did not receive any acute intervention in the emergency department and was referred to otorhinolaryngologists for further workup. There is some evidence showing benefit from treating bells palsy with corticosteroids given its potent inflammatory effect. In this patient, corticosteroids were never initiated before discharged from the emergency department due to his presentation 72 hours after the onset of symptoms and his history of diabetes. A thorough history and physical examination is essential in ruling out other causes of facial nerve paralysis before making the definitive diagnosis. The diagnosis of Bells palsy is often made clinically and no testing is required. We suggest that trauma with inflammation to the area secondary to tooth extraction might provide a causal explanation for his presenting symptoms, although there's no way to prove this.

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Clinical Vignette	Izzah Vasim	John Oghene MD2, Katherine Abella MD, Sara Dean APN, Sivashankar Sivaraman MD	Atlanticare Regional Medical Center (Dominik Zampino)	Excessive Amphetamines in a weight-reducing regimen leading to cardiac arrest – Case Report	<p>Introduction: Over the years dietary supplements have been used as a popular method to aid weight loss. These weight loss supplements sometimes have ingredients such as amphetamines that can lead to cardiac dysrhythmias. Amphetamines are a class of sympathomimetic amines that are known to cause cardio-toxic effects including myocardial infarction, arrhythmias, cardiomyopathy and acute heart failure. There have been very few reported cases of fatal ventricular arrhythmias with the use of dietary supplements containing amphetamine related compounds.</p> <p>Case: 47-year-old female with no significant medical history presented after being found unresponsive, cyanotic and in agonal respirations. CPR was immediately initiated and EMS was activated. On EMS arrival, the patient was found to be in a shockable rhythm on the AED and she converted to sinus rhythm and achieved spontaneous circulation after one shock. Upon arrival to the emergency department, hypothermia protocol was immediately initiated and she was transferred to the intensive care unit. The patient had no prior medication use, smoking, alcohol or recreational drug use. On further questioning, the patient's husband revealed that she had recently started a rigorous workout regimen as well as ingesting several dietary supplements called "310 MetaBoost –Enhanced Wight Loss Formula" and "Hydroxycut". Her initial laboratory studies revealed an AST of 397U/L, ALT of 371U/L, Albumin of 3.3g/dl, Troponin level of 1.196ng/ml and 0.781ng/ml. Her drug screen was positive for amphetamines. Her initial EKG was not suspicious for any acute injury or ischemia. A CT angiogram revealed no evidence of pulmonary embolism except for mucous plugs in the right main stem. A 2-D echo did not reveal any significant finding. Her cardiac catheterization revealed no coronary artery disease. Given her presentation of sudden cardiac arrest due to Ventricular fibrillation, an AICD placement was deemed necessary for secondary prevention of sudden cardiac death. The patient tolerated the procedure without any complications and was later discharged home.</p> <p>Discussion: Hydroxycut has been known to contain amphetamine related compounds and another sympathomimetic amine EPIGALLOCATECHIN that potentially causes ventricular arrhythmias via inhibiting cardiac ion channels. Other serious cardiac side effects include hypertension from its alpha and beta-adrenergic stimulation, coronary vasospasm and increased thrombogenicity due to catecholamine induced platelet aggregation. Ultimately, over the counter dietary supplements must be scrutinized carefully for their adverse cardiovascular effects. Our case suggests a possible causal relationship between Hydroxycut /Meta Boost use and Ventricular Fibrillation.</p>

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Clinical Vignette	Siddharth Verma	Dr. Bishoy Hanna, MD; Dr. Aditya Bansal, MD, FCCP	Atlanticare Regional Medical Center (Dominik Zampino)	A Rare Case of Extranodal Lymphoma	<p>Introduction: Extranodal marginal zone B-cell lymphoma constitute 0.4% of lymphomas and 3.6% of Non-Hodgkin Lymphomas. The usual imaging patterns include pneumonia like alveolar consolidation with air-bronchograms, tumor like well circumscribed nodular opacity or infiltrative pattern with diffuse poorly defined ground glass opacities. Pleural effusions are unusual. We describe a rare presentation of extranodal lymphoma in the lungs with pleural effusion.</p> <p>Case Description: The patient is an 87-year-old woman with past medical history of diabetes mellitus, hyperlipidemia and hypertension who presented with flank pain, hematuria and passage of a 9 mm kidney stone. She also reported unintentional weight loss of 35 lbs and poor appetite. Her laboratory evaluation was unremarkable. CT scan of the chest showed right middle lobe, right lower lobe and left lower lobe consolidations with left pleural effusion. She was diagnosed with pneumonia and treated with antibiotics. Thoracentesis revealed exudative effusion with fluid protein of 3.8 g/dL, LDH of 175 U/L, pH of 8 and glucose 172 mg/dl (serum protein 7.4 g/dL, LDH of 167 U/L). Pleural fluid was lymphocyte predominant raising concern for lymphoproliferative disorder. She clinically improved and was discharged. Follow up CT scan in 2 months showed moderate left pleural effusion, slightly increased from the prior study and bilateral airspace consolidation without improvement. PET scan showed persisting consolidation of both lower lobes, left greater than right with diffuse hypermetabolic activity consistent with infection/inflammation. No other areas of uptake were noted. Bronchoscopy with transbronchial biopsy from the left lower lobe showed fragments of peribronchial tissue with dense lymphocytic infiltrate and no apparent lymph node architecture. Majority of these lymphocytes were positive for CD20 and BCL-2; negative for CD5 and BCL-1. Histologic and immune-histochemical staining features showed low-grade B-cell lymphoma, favoring marginal zone lymphoma, Stage 1E involving the lung only. All cultures from the pleural fluid and BAL were negative. The patient is negative for HIV and hepatitis C. Treatment with Rituximab is planned.</p> <p>Discussion: Pulmonary MALT lymphomas are low grade lymphomas. They may be associated with underlying autoimmune disease, immunodeficiency or chronic infections. Less than 50% of patients present with cough, dyspnea and chest pain. Radiological imaging is nonspecific however pleural effusions are unusual. Diagnosis is confirmed by biopsies with histologic and immune-histochemical staining suggesting features of lymphoma. Current data favors systemic treatment over surgical resection. Prognosis is excellent with an indolent and localized prolonged course. Five-year survival of greater than 80% and evolution to high grade lymphoma in less than 5% patients. Close follow up is crucial to monitor for recurrence due to a high rate of recurrence.</p>

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Clinical Vignette	Vishal Patel	Anokhi Patel, MD; Bishoy Hanna, MD	Atlanticare Regional Medical Center (Dominik Zampino)	Aortoenteric Fistula as a Rare Complication after Endovascular Abdominal Aortic Aneurysm Repair	<p>Secondary aortoenteric fistulas (AEF) are rare and life threatening complications after abdominal aortic aneurysm (AAA) reconstruction seen in 0.36 to 1.6% of cases. The incidence of AEF is much lower in endovascular repair (EVAR) compared to open repair. It is an abnormal communication between the aorta and the gastrointestinal tract via erosion of prosthetic graft or stent from AAA repair into adjacent viscera, most commonly the third and fourth portions of the duodenum. While the precise mechanism of formation of the fistula is unknown, several risk factors play a role including: AAA with surgical repair, malignancy, and aortic infection. AEFs are associated with significant mortality is high and nearly 40% even after surgical repair.</p> <p>We present a case of an 86 year old man with a history of colon cancer resected 5 years ago who underwent endovascular repair of an infrarenal aortic aneurysm three years later. The patient was without complaints for 18 months after the initial AAA repair until he presented to the emergency department with complaints of two bloody and melanotic stools over 24 hours. On presentation, the patient was hemodynamically stable with a 5g/dL drop in hemoglobin from baseline. The following day an upper esophagogastroduodenoscopy revealed non-bleeding diverticula in the second and third portions of the duodenum. The patient's hemoglobin continued to drop and patient became progressively unstable. A computed tomography of the abdomen revealed leakage of intravenous contrast surrounding the aortobiiliac stent graft and intra-aortic air suggestive of a duodenoaortic fistula. A large retroperitoneal hematoma communicating with the third portion of the duodenum was also noted. Emergent surgical evaluation deemed intervention futile. Patient continued to deteriorate despite aggressive resuscitation with 7 units of packed red blood cells and 6 liters of intravenous fluids as the patient continued to have hematemesis and large bloody bowel movements. Patient passed away within 9 hours of onset of hemorrhagic bleeding. AEF is an unusual complication seen after open surgical graft repair and rarely presents after endovascular repair. There are currently 14 reported cases of this lethal complication. The largest study consisting of 11 patients showed a 36% mortality rate. AEF are difficult to diagnose and treat. Esophagogastroduodenoscopy may confirm the diagnosis but do not exclude AEF if a fistula is not visualized. The sensitivity of CT scan is limited and varies between 33% and 80%. Treatment options include explantation of defective graft after extra-anatomical bypass or in situ grafting using prosthetic material. The true incidence of AEF after EVAR is unknown but believed to be lower than open repair. Given these points, it is crucial to consider AEF as a potential diagnosis in patients with gastrointestinal bleeding after aortic abdominal aneurysm repair.</p>

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Clinical Vignette	Daniel Rhoades	Aileen Hocbo, Lynn Manganiello, Karen Alexander, Hinna Mannan, Amina Mehrab	Atlanticare Regional Medical Center (Dominik Zampino)	A Fashion Nightmare: DRESS Syndrome With Leukocytosis and Acalculous Cholecystitis After Bactrim Ingestion	<p>DRESS syndrome is a life-threatening idiosyncratic event cause by many medications with an incidence of 1 in 1,000 to 1 in 10,000 exposures and carries a 10% mortality. Many cases of DRESS syndrome involve hematologic derangements, but few reported cases document leukocytosis as a presenting symptom.</p> <p>CASE: The following is a case of DRESS syndrome in a 23 year old African American female presenting with nausea and vomiting with a fever to 104 degrees after one month of Bactrim treatment. A maculopapular rash was noted on her trunk during admission that spread over her entire body, but spared her oral mucosa. This was accompanied by nausea, vomiting, ana sarca, acalculous cholecystitis from liver edema, an increase in her AST/ALT to 625/756 and a leukocyte count increasing from 14,300 to 42,500. She was successfully treated with systemic steroids, topical steroids and a sauna suit.</p> <p>Discussion: DRESS syndrome induced by Bactrim is rarer than the other commonly implicated drugs. When examining which blood count abnormalities that are usually found, many times it is lymphocytosis, eosinophilia or thrombocytopenia. However, very high leukocytosis and liver edema causing acalculous cholecystitis are not usual features of DRESS syndrome. Even with these findings, this patient still improved on high dose topical and systemic steroids, which is still one of the main treatments for this life threatening disease.</p>

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Clinical Vignette	Aparna Daley	Daniel Goldsmith, M.D., Michael S. Beede, M.D.	Capital Health Regional Medical Center (Saba Hasan)	Smoking, Scleroderma and Minor Trauma – A Design for Digital Amputation	<p>Background: Diffuse scleroderma is a multi-system disease involving vascular and immune dysfunction. Poor wound healing and microvascular manifestations of digital ischemia may occur. Minor trauma compounded by factors like smoking may result in non-healing digital ulcers ultimately leading to infection, necrosis and ischemic digital loss.</p> <p>Case: A 61 year-old female smoker with a history of scleroderma and rheumatoid arthritis overlap syndrome reported sustaining a minor needle stick injury to her right index finger while sewing. Her medical history was significant for severe pulmonary hypertension, moderate to severe aortic stenosis, moderate mitral regurgitation, a total left knee arthroplasty ten months ago (initially complicated by poor wound healing), cerebrovascular accident without focal deficit, medical non-compliance and history of 15 years of smoking. Her Anti-Centromere and Anti-CCP antibodies had been notably elevated on previous admissions. Despite a course of Trimethoprim/sulfamethoxazole from her primary care physician, she suffered a painful, non-healing wound of the injured finger. After 4 weeks, she complained of persistent pain and fever and presented to the ED. Physical examination was remarkable for necrosis and loss of tissue from the dorsal and volar aspects of the right index finger distal interphalangeal joint to the fingertip, with overlying eschar, but no exposed bone or drainage. X-rays showed soft tissue loss of the distal phalanx. We initially treated the patient with intravenous Vancomycin and Piperacillin/tazobactam and wound care. Blood cultures were positive for Methicillin-sensitive Staphylococcus aureus. We consulted plastic surgery, and the surgeon found the fingertip unsalvageable. We changed the antibiotics to Ampicillin/sulbactam and after outpatient follow-up, the patient underwent amputation of distal right index finger at level of middle phalanx.</p> <p>Discussion: This case illustrates the serious consequences that even minor trauma can cause in scleroderma patients who tend to have poor wound healing. Impaired wound healing is compounded by smoking which directly affects the vascular endothelium. Educating patients with scleroderma to avoid minor trauma and encouraging smoking cessation are critical to avoid devastating consequences of digital amputation in this population. Every primary care visit is an opportunity for the physician to address smoking cessation and encourage awareness of avoiding minor trauma.</p>

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Clinical Vignette	Nabil Mesiha	Abisoye Fakayode MD, Manish Gugnani MD, Ramninder Nagra MD, Bipinpreet Nagra MD	Capital Health Regional Medical Center (Saba Hasan)	Dramatic Nocturnal Complete Heart Block – What is the management?	<p>Background: Obstructive sleep apnea (OSA) is a common chronic disorder that requires lifelong care. It has substantial cardiovascular morbidity and mortality including tachyarrhythmias and bradyarrhythmias. The most common treatment is continuous positive airway pressure (CPAP). We report a case of transient marked nocturnal advanced heart block in a patient with OSA.</p> <p>Case presentation: 61-year-old man with known OSA, morbid obesity with BMI of 39.22, Hypertension, Type 2 Diabetes and non-compliance with CPAP was referred to cardiology for abnormal holter monitor. Twenty-four hour holter monitor showed a surprising finding of a minimum heart rate of 15 beats per minute overnight. He had a total of 1,113 nocturnal pauses with longest pause being 5.6 seconds at 00:29 hours. Holter also showed multiple episodes of nocturnal complete heart block, 2-1 AV block, wide complex escape beats and PVCs while sleeping. No arrhythmias noted during daytime. Patient adamantly refuses to wear CPAP. Patient is asymptomatic. Cardiac work up showed normal resting electrocardiogram, normal electrolyte, and TSH. Patient had an unremarkable surface echocardiogram, and negative exercise stress test with appropriate chronotropic response to exercise. Here, we need to ask a question!! Is cardiac pacing indicated for this asymptomatic patient with significant nocturnal bradyarrhythmia with nocturnal complete heart block and pauses? Is the patient at an elevated risk of nocturnal sudden cardiac arrest?</p> <p>Discussion: It has been suggested that OSA is a systemic illness rather than simply a local airway abnormality. Aim of treatment is to reduce the number of episodes of apnea , oxygen desaturation, and number of arousal during sleep and subsequently improvement of blood pressure, baroreceptor sensitivity and reduction in sympathetic nervous system activation and cardiac arrhythmia especially bradyarrhythmias. The mainstay for therapy has been CPAP, unfortunately despite its effectiveness, refusal of using, and non compliance still considered a big issues. Alternative treatment should be proposed. Cardiac Pacing in some small studies was found to be effective in patients with OSA who have significant symptomatic bradyarrhythmias and frequent variations in heart rate and found to reduce the number of episode of apnea. However, in other studies, researchers were unable to show any beneficial effect of pacing in reducing the number of episodes of apnea or increasing arterial saturation during sleep. Cardiac pacing is generally restricted in guidelines to symptomatic patients. There is no absence of significant body of literature to guide management of asymptomatic patients with severe nocturnal bradyarrhythmias resulting from OSA. More research is needed on this important subject.</p>

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Clinical Vignette	Nabil Mesiha	Stephen Tieku, MD; Naresh Nagella, MD; Pirouz Parang, MD; Daniel Goldsmith, MD	Capital Health Regional Medical Center (Saba Hasan)	Kill Two Birds with One Stone	<p>Background: Acute lower GI bleeding is a medical and potentially surgical emergency, requiring urgent attention of the treating team. Management includes resuscitation with intravenous crystalloids and blood products. After stabilization, it is mandatory to find the bleeding source. We report one case of recurrent GI bleeding found to be from angiodysplasia, secondary to a cardiac cause.</p> <p>Case Presentation: A 71 year old woman with history of paroxysmal atrial flutter, hypertrophic cardiomyopathy, and transient ischemic attack, was admitted three times in one year with massive GI bleeding and severe symptomatic anemia, requiring multiple blood transfusions. During the first admission the patient was on rivaroxaban for atrial flutter. A GI work up, including upper GI endoscopy, colonoscopy, and capsule endoscopy showed duodenal angiodysplasia. Rivaroxaban was discontinued, and after a period of time with no bleeding, she was placed on warfarin. A few months later, she had another lower GI bleed. At that time, upper and lower endoscopy found no significant abnormality. Warfarin was discontinued, but she had a third massive lower GI bleed, despite cessation of anticoagulants. Echocardiogram showed moderate concentric left ventricular hypertrophy with normal EF, and hematological evaluation showed high partial thromboplastin time. Routine testing for von Willebrand disease(vWD) was normal. The patient was sent to a tertiary center for a left ventricular myomectomy, maze procedure, and left atrial appendage excision. She resumed anticoagulants after surgery and is currently in good health in outpatient care. For the last 8 months, she has not had any further episodes of bleeding.</p> <p>Discussion: Heyde syndrome is a triad of acquired vWD, aortic stenosis, and bleeding from intestinal angiodysplasia. The mechanism of bleeding is from degradation of vW factor multimers by shear stress through the diseased aortic valve leading to significant blood loss through areas of angiodysplasia. We postulate that hypertrophic cardiomyopathy, or any cause of shear stress of blood flow similar to aortic stenosis, is another condition which may be associated with acquired vWD. Definitive management of GI bleeding in the setting of Heyde syndrome should be directed to the cause of turbulence of the blood flow inside or outside the heart. Routine screening tests for acquired vWD are usually normal, therefore to confirm acquired vWD a gel electrophoresis is required. Recurrent GI bleeding in patients with a clinical reason for turbulent blood flow should undergo more detailed laboratory testing for acquired vW factor deficiency than the routine screen.</p>

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Clinical Vignette	Sayedmajidreza Alavidehkordi	Daniel Oldsmith, Lee Glickman, Ekaterina Vypriskaya	Capital Health Regional Medical Center (Saba Hasan)	Reverse takotsubo associated with suicide attempt by drug overdose	<p>Background: Takotsubo Cardiomyopathy (CM) also called stress CM, as classically defined, involves wall motion abnormalities confined to the LV apex believed to be triggered by the sudden endogenous release of harmful levels of catecholamines. The majority of Takotsubo patients recover cardiac function in three to six months, and long term prognosis is generally good. Early recognition and evaluation is important for exclusion of other acute causes of cardiomyopathy and for treatment considerations. Increasingly, other patterns of segmental abnormalities have been recognized, including exclusive basal wall motion abnormalities, termed "Reverse Takotsubo" and we report such a case here.</p> <p>Case: A 29 year old male with 3 year history of heroin and cocaine addiction, presented reporting abdominal pain, acutely increased depression and having injected as many as 18 bags of heroin in a suicide attempt 3 days prior to presentation. He changed his mind and called the suicide hotline, who arranged transport to the ED. There was no chest pain and physical examination was unremarkable including normal vital signs, normal cardiac and neurological exams. EKG was normal. A screening troponin was negative. Urine drug screen was positive for cocaine and opiates.</p> <p>Later that day, he began complaining of chest and neck pain and became gradually more obtunded. Repeat EKG showed sinus tachycardia, with ST depressions infero-laterally. Troponin rose to 6 ng/mL. Echocardiogram showed the basal to mid portion of the anterior, septal, lateral, and inferior walls were akinetic to dyskinetic while function of the distal LV and apex was preserved. Visually estimated LVEF was 32%. He was initially treated as a non-ST-segment-elevation myocardial infarction (NSTEMI) with aspirin, carvedilol and heparin. The patient required intubation for tachypnea and hypoxia although chest X-ray was clear. Troponin peaked at 12.3 ng/dL. Cardiac catheterization showed normal coronary arteries, but demonstrated basal LV "ballooning" (dilatation and dyskinesia) and normal apical contraction. Heparin was discontinued. The patient was able to be extubated after 48 hours. Repeat echocardiogram on day 4 of admission showed a persistent pattern of segmental abnormality but slight improvement in overall function to an EF 35-40%.</p> <p>Discussion: Previous cases of classical pattern Takotsubo CM have been reported as associated with psychiatric stress such as a severe depressive episode. Other cases have been reported associated with cocaine overdose. We are unaware of any previously reported case of Reverse Takotsubo CM associated with one or both of these potential triggers. Despite mimicking a NSTEMI, the long-term prognosis of stress cardiomyopathy is good and with adequate treatment of the underlying psychiatric and substance abuse diagnoses, a positive outcome is expected.</p>

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Clinical Vignette	Ola Adams	Albana Mihali. Schumacher, Hermann	Capital Health Regional Medical Center (Saba Hasan)	Narcolepsy with cataplexy. The drop attacks.	<p>Background: Narcolepsy with cataplexy is a disabling sleep disorder, characterized by severe, irresistible daytime sleepiness and sudden loss of muscle tone (cataplexy). It can be associated with sleep-onset or sleep-offset paralysis and hallucinations, frequent movement and awakening during sleep, and weight gain. Sleep monitoring during night and day shows rapid sleep onset and abnormal, shortened rapid-eye-movement sleep latencies. It usually begins between the ages of 10 and 20 years with the sudden onset of persistent daytime sleepiness, although it can also develop gradually.</p> <p>Case: A 43-year-old Hispanic male was admitted for evaluation of multiple episodes of falls for 3 weeks. He gave a history of generalized fatigue and weakness, brief episodes of syncope when laughing or with emotion of anger, associated with inability to talk for a couple of seconds. The episodes last for seconds without loss of consciousness. He denied urinary incontinence, jerky movements, or tongue biting. He normally sleeps 7 hours per night, and wakes up fresh afterward. He reported increased daytime sleepiness lately that necessitated taking naps during work in landscaping. MRI's of the brain and cervical spine were negative. Blood work for HIV, Acetylcholine receptor antibodies, B12 level and Lyme antibodies were all negative. Multiple Sleep Latency Test revealed severe hypersomnia across the entire day with a mean sleep latency of 2.1 minutes. In addition to four sleep onset REM episodes were noted and therefore the study was consistent with narcolepsy. Patient was started on methylphenidate with titration and was followed up regularly as an outpatient. He showed significant improvement in daytime sleepiness with no more cataplexy attacks.</p> <p>Discussion: Narcolepsy, one of the most common causes of chronic sleepiness, affects about 1 in 2000 people. Despite the frequency of narcolepsy, the average time from the onset of symptoms to diagnosis is 5 to 15 years, and narcolepsy may remain undiagnosed in as many as half of all affected people, since many clinicians are unfamiliar with this disorder. Clinicians now recognize two types of narcolepsy. Type 1 is caused by extensive loss of hypothalamic neurons that produce the neuropeptides orexin-A and -B (also referred to as hypocretin-1 and -2); type 2 includes most of the same symptoms, but its cause is unknown. In contrast to people with disorders such as obstructive sleep apnea who have poor-quality sleep, those with narcolepsy usually feel refreshed after a full night's sleep or a brief nap, but their sleepiness returns 1 to 2 hours later, especially when they are sedentary.</p> <p>Teaching point: Narcolepsy is an under-recognized disease, and general practitioners are uniquely poised to identify symptoms early and differentiate them from sleep apnea.</p>

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Clinical Vignette	Natalia Plotskaya	Daniel Goldsmith MD, Asher Hussein MD	Capital Health Regional Medical Center (Saba Hasan)	Integrated endovascular approach to treatment of acute renal vein thrombosis manifested as primary membranous glomerulopathy.	<p>Background: Renal vein thrombosis (RVT) is a very common complication of nephrotic syndrome, which can be symptomatic or chronic and asymptomatic. It is associated with imbalance between procoagulant and anticoagulant factors, and endothelial dysfunction. Multiple modality treatment might be required in symptomatic RVT with extension to inferior vena cava.</p> <p>Case: A 51 year old male with a long history of hypertension and 4 months lower extremity edema presented with severe sharp left flank pain with radiation to low back for 4 days. He had similar pain in the right flank 2 months ago. Physical examination revealed elevated blood pressure of 190/110 mmHg , left lower abdominal tenderness and moderate lower extremities edema. No family history of kidney disease or hematologic disorders was identified. Laboratory showed increased creatinine of 1.35 mg/dL for 2 months, and decreased albumin of 2.6 g/dL. CT scan of abdomen with intravenous contrast detected left renal vein thrombosis extending into the suprarenal inferior vena cava, with possible extension of thrombus into the left gonadal vein, and an enlarged and heterogeneous left kidney. Immediately patient was started on heparin. Despite tissue plasminogen activator (tPA) infusion at 3 mg/hr for 6.5 hours, follow up left renal venogram still showed large clot. AngioJet thrombolysis and mechanical removal of residual clot finally led to resolution confirmed by venogram. Next day, creatinine decreased to 1.19 mg/dL. On day 5 after intervention, nuclear renal scan demonstrated symmetric flow to both kidneys, and no evidence of hydronephrosis. Hypercoagulability work up was unremarkable. Urinalysis showed severe proteinuria of 11.6 g/24 hours. For this reason, patient underwent successful left kidney biopsy which shown membranous glomerulopathy (MGN) stage1-2. Indirect immunofluorescence staining was positive for phospholipase A2 receptor (PLA2R) which supports primary MGN.</p> <p>Discussion: Acute RVT should be highly suspected in symptomatic patients with acute kidney injury associated with lower extremity edema, and sudden flank pain. Timely diagnosis with CT angiography is necessary to assess extension of thrombosis and initiate combined intervention to preserve renal function and prevent further thromboembolic events.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Bushra Saleem	Sree Yelamanchili, Ola Adams, Sarah Sordo.	Capital Health Regional Medical Center (Saba Hasan)	Rare initial presentation of Lupus in young female_ Cardiac Temponade.	<p>Background: Systemic lupus erythematosus (SLE) is a chronic inflammatory disease of unknown cause, however, usually attributed to immunological abnormalities. More seen in women as compared to men, this disease is found to be associated with multiple organ involvement. Patients may have constitutional symptoms along with skin, serological, musculoskeletal, hematologic, renal, or central nervous system manifestations. The clinical course is variable among patient who may have chronic or acute relapses and periods of remissions.</p> <p>Case: A 30 year old African American female with history of asthma and GERD, was sent to the ER from the office due to very high blood pressure. She reported generalized fatigue, joint pains and stiffness, since one year, worsening over last few months. Her other symptoms include mild exertional dyspnea, occasional dry mouth, bilateral elbow rash and 40 lbs unintentional weight loss in 6 months. She was found to be in hypertensive urgency and was admitted on tele-monitor floor. Work up was started with high suspicion of autoimmune etiology. Significant initial labs include leucopenia, microcytic anemia, proteinuria, occult blood in the urine, high c – reactive protein. Echocardiogram showing no wall motion abnormality but small pericardial effusion. Her BP was initially difficult to control. On 3rd day of admission her blood pressure stabilized to normal range, however, she developed persistent tachycardia with a new complaint of dizziness. She also had a spike of fever that later subsided. There was high suspicion of pulmonary embolism as well as infection. A CT chest suggested cardiac temponade, confirmed with urgent echocardiogram. Patient was transferred to ICU and pericardial window was performed, 244 ml of pericardial fluid was drained suggestive of chronic active inflammation. She was started on steroids and colchicine. The lab results returned showing high ANA titer 1:640 and dsDNA 4844. She was diagnosed with SLE, further stabilized and discharged home with outpatient follow up.</p> <p>Discussion: Patients with SLE can have involvement of various cardiac structures including pericardium (in 25% of cases) however myocardium, valves, conduction system, and coronary arteries can also be affected. Cardiac tamponade as a first presentation of SLE is rare found in less than 2% of the cases. There should be high suspicion of cardiac temponade, if such patients get hemodynamically unstable.</p>

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Clinical Vignette	Bushra Saleem	Sree Yelamanchili, Iram Mahmood, Noorain Mazhar.	Capital Health Regional Medical Center (Saba Hasan)	Acute Interstitial Nephritis_ An overlooked cause of Acute Kidney Injury.	<p>Background: Acute interstitial nephritis is seen in 1 to 3 percent of all renal biopsies. However, because analysis is restricted to biopsies performed in the setting of AKI, the percentage rises to 13 to 27 percent. Almost 70% cases of AIN are found to be drug induced and others associated with infections, systemic diseases, malignancies and idiopathic. There is need to evaluate all the patients who present with acute kidney injury for the underlying cause and consider AIN in cases of unexplained renal insufficiency.</p> <p>CASE: 59 year old asymptomatic male was sent from the nursing home when the routine labs were found to be abnormal. His past medical history is HTN, HLP, PAD, BPH, COPD, right AKA due to ischemic limb and CVA with left hemiparesis. His labs showed BUN 104 mg/dl, Creatinine 14.4 mg/dl (Baseline 11/0.82, 5 months ago). Hemoglobin 5.6 g/dl, HCT 17.2% MCV 83.2fl, RDW 14.6 %, platelet count 13600/ul, eosinophils 10.0%. Urinalysis shows protein 30 mg/dl, moderate eosinophils, RBCs 25-50, moderate bacteria, negative nitrite and leukocytes. Fractional excretion of sodium calculated as 3%, BUN: Creatinine ratio was 7. Work up was initiated, medication list review showed, the only new medication that he was started on was pantoprazole, 5 months ago. Pre-renal and post renal causes were excluded, and renal biopsy was performed. At this stage there was high suspicion of AIN however the patient developed perinephric abscess and bacteremia and treatment was delayed. Patient had to be started on hemodialysis because of lethargy secondary to refractory uremia complicated with metabolic acidosis and infection. The lab data resulted showing normal level of ANA, ANCA, SPEP, UPEP, C3, C4, ACE (initial mild high complement followed by normal level). Biopsy resulted as acute interstitial nephritis. After treatment with antibiotics and resolution of bacteremia, he was started on steroids with a plan to continue for 12 weeks and was discharged to nursing home on dialysis.</p> <p>Conclusion: Proton pump inhibitors are one of the most frequently prescribed drug classes worldwide and are considered to have favorable safety profile. However, they have been found to be associated with development of not only acute interstitial nephritis but also chronic kidney disease. Prevention and early identification with prompt treatment of these cases may be helpful to avoid the development of severe chronic kidney disease or end-stage renal disease.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Nagham LeAnne Jafar	Bipinpreet Nagra,Sevil Aliyeva , Maryam Siddiqui , Sofia Chaudhry	Capital Health Regional Medical Center (Saba Hasan)	When is imaging the right answer for syncope?	<p>Background: Syncope is the cause of more than 1% of all emergency department visits in the United States. Out of these, 30% - 40% of such patients are subsequently admitted for further investigation at an annual cost of \$2.4 billion according to the Medicare database. Usual assessment of syncope varies case by case, but imaging only rarely reveals the correct diagnosis.</p> <p>Case: An 83 year old female with history of hypertension and lymphedema, presented with an episode of lightheadedness and ataxia. She denied any prodromal symptoms prior to the episode, and no chest pain or dyspnea. Her EKG, echocardiogram and CT head were negative. She was diagnosed with TIA and shortly upon becoming asymptomatic she was discharged home. Two months after this episode, she presented again for evaluation of frequent episodes of witnessed syncope. During that admission, cardiac stress test was negative for ischemia and she was discharged home. Nonetheless, she continued to have syncopal episodes over the course of 8 months, and as a result, an extensive cardiac and neurological workup was undertaken. An MRI of the neck showed a carotid body tumor at the right carotid bifurcation. Surgical removal of the tumor led to complete resolution of the symptoms. Histology confirmed the diagnosis of carotid body tumor.</p> <p>Discussion: Syncope is a frequently encountered complex medical condition and the work up is based on multiple factors including history, age and co-morbidities. The 2009-revised European society of cardiology guidelines on the management of syncope stated that there are no studies evaluated the use of brain imaging for syncope. CT or MRI in uncomplicated syncope should be avoided. Imaging may be needed based on a neurological evaluation. AHA/ACCF Scientific Statement on the Evaluation of Syncope published in circulation 2006, stated that when a neurological basis of syncope is suspected, imaging of the brain with either CT or MRI is indicated. If cerebrovascular disease is suspected, imaging of the extra-cranial and intracranial carotid arteries is appropriate. They also stressed on the importance of history taking and physical exam in deciding on how to proceed with the work up.</p> <p>Teaching point: Carotid body tumor rare neoplasms represent less than 0.5% of all head and neck tumors. They are usually benign. Symptoms are generally due to local involvement of the nerves and vessels. They rarely presents as syncope or TIA, In the absence of an explanation, Carotid body tumor needs to be ruled out as a cause of syncope. High index of suspicion, taking thorough history and physical exam are keys, that helps detect neurological causes of syncope and order the right diagnostic test. Prompt diagnosis can lead to curable treatments and thus prevent readmission and help relieve patient anxiety due to frequent syncope.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Majlinda Xhikola		Capital Health Regional Medical Center (Saba Hasan)	Successful surgical treatment of a large, well differentiated Neuroendocrine Tumor	<p>Introduction: Midgut neuroendocrine tumors are rare tumors that arise in the jejunum, ileum, and proximal colon. Patients tend to present late, after a long history of vague nonspecific symptoms, and disseminated metastases are often discovered at presentation. Patients may experience symptoms secondary to the release of active substances by the primary tumor, such as serotonin and its metabolites, which have local and systemic effects.</p> <p>Case report: The patient, a 55 year old male with history of hypertension and chronic alcohol abuse was incidentally found to have a large liver mass, while being worked up for the cause of his septic shock admission. CT scan of abdomen showed a large hepatic mass of 11.4 cm and a 3.4 cm soft tissue mass in the right lower quadrant. Serum chromogranin A level was 5800 ng/mL and 24 hours urine collection for 5-HIAA showed a level of 308 mg/24 hr. CT guided biopsy of the liver showed a well differentiated neuroendocrine tumor.</p> <p>After detailed history from the patient, there are no specific symptoms reported other than intermittent abdominal discomfort. Patient underwent surgical resection of the liver mass, ileal mass and pelvic lymph node excision. Pathology revealed well differentiated metastatic neuroendocrine carcinoma of the liver with primary ileal lesion and 1/10 lymph nodes involved, stage T3N1M1. No chemotherapy was introduced. Follow up chromogranin A levels at 3 and 6 months post surgery were 19 ng/mL and 24 ng/mL. PET scan done 6,12 and 18 months after surgery revealed no evidence of new lesions. Patient continues to be asymptomatic.</p> <p>Discussion: Patients with metastatic midgut neuroendocrine tumors often die of local complications of the primary tumor, such as small bowel obstruction or ischemia. These complications can potentially be avoided by resection of the primary tumor, with recent literature showing that resection has a survival benefit. We described a case of a successful surgical treatment of a well differentiated, nonfunctioning neuroendocrine tumor with primary ileal lesion and a large hepatic metastasis. This case once again emphasizes how indolent neuroendocrine tumors can be even in the setting of an impressive size.</p>

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Clinical Vignette	Noorain Mazhar	Nabil Mesiha,MD; Stephen Tiekou, MD; Saba Hasan, MD; Daniel Goldsmith,MD	Capital Health Regional Medical Center (Saba Hasan)	Not every orthopnea means heart failure	<p>Background Orthopnea is defined as dyspnea that occurs while lying down. The mechanism is redistribution of body fluids, which is tolerated in patients with normal cardiac function, but in those with heart failure, the heart is unable to accommodate the minimal extra fluid in the lung. Orthopnea is often associated with heart failure, but can be observed with other, less obvious diagnoses.</p> <p>Case: A 61 year old woman presented with gradual onset of dyspnea, orthopnea, paroxysmal nocturnal dyspnea for 2 months. On physical exam she was slightly tachycardic, oxygen saturation was 96% on room air while sitting but she desaturated to 89% on supine position. Lung exam showed fine crackles bilaterally but there was no leg swelling. Initial diagnosis was heart failure, and she required non-invasive mechanical ventilation especially at night. Lab testing revealed mildly elevated LFTs, normal troponins. Chest X-ray revealed bibasilar subsegmental atelectasis versus infiltrate. Furosemide resulted in no significant improvement in symptoms. Echocardiogram was normal. BNP was not elevated. Review of the chest X-ray showed small lung volumes and on further questioning she reported mild dysphagia. Neurologic exam revealed mildly decreased proximal muscle strength in the upper and lower extremities. PFTs in the sitting and supine position showed that in the sitting position, the FVC was 1.12 liters (46% predicted) while in supine position FVC was 0.56 liters (24% predicted). Sitting FEV-1 was 0.97 liters (52% predicted) while supine FEV-1 was 0.42 (23% predicted) showing moderate restrictive ventilatory impairment worse in the supine position. ABG showed hypercapnia and hypoxemia. Additional lab testing showed elevated CK, ANA titer 1:160, elevated aldolase 25.2 U, negative acetylcholine receptor antibody test, and negative anti-MUSK antibodies. EMG showed acute myopathy. Muscle biopsy was consistent with mild, active myopathy. Prednisone resulted in significant improvement in her symptoms. Patient was discharged home with no ventilatory support.</p> <p>Discussion: Myopathy is a systemic disease that can affect all muscles of the body including chest wall and diaphragm, resulting in dyspnea and orthopnea especially during supine position. Arriving at the correct diagnosis starts with high clinical suspicion, supported with high level of CK, aldolase, and transaminases. EMG and muscle biopsy confirm the diagnosis. Positional PFT's can help establish the diagnosis of myopathy when there is significant worsening of volumes in the supine position, and should be considered in certain patients with dyspnea and orthopnea, when heart failure is less likely the correct diagnosis.</p>

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Clinical Vignette	Maryam Siddiqui	Bushra Saleem MD, Nagham L. Jafar MD, Daniel F. Goldsmith MD.	Capital Health Regional Medical Center (Saba Hasan)	PAIN: AN OVERLOOKED PRESENTATION OF MULTIPLE SCLEROSIS.	<p>Introduction: Multiple sclerosis (MS) is a chronic immune-mediated disease of the central nervous system that causes long-term physical disability. Throughout the course of the disease, pain is common and it is associated with many symptoms and problems. Studies have reported that 23% of patients had pain at the onset of being diagnosed with MS and the prevalence of pain in MS ranged from 29% to 86%.</p> <p>Case Presentation: A 30 year African American female presented to the ER 33 times over the course of 10 years for evaluation of different symptoms including multiple visits for lower back pain, neck pain, and extremity pain. The patient underwent multiple X-rays, CT of lumbar and cervical spine which were all inconclusive. She also underwent Electromyography for evaluation of possible carpal tunnel syndrome after presenting with intermittent wrist pain. The patient was discharged multiple times on various pain medications to treat her “chronic pain syndrome”. She was also referred to a psychiatrist for evaluation and was started on antipsychotic medications which the patient refused to use. In 2016 patient came to the ER complaining of headache and ambulatory dysfunction from leg weakness leading to falls which required admission. Motor examination revealed a mild drift with decreased hand grip on the right arm and right lower extremity drift. Sensory exam revealed a sensory loss to touch, pinprick and temperature at various cervical and thoracic distributions more on the right arm and leg. An MRI of brain was done which showed multiple regions of white matter hyper intensity, both above and below the tentorium, with a distribution and appearance compatible with a clinical diagnosis of demyelinating disease. The patient subsequently underwent lumbar puncture which showed the presence of oligo clonal bands . The patient was started on high dose IV steroids and currently on Disease modifying therapy for MS with significant clinical improvement requiring no further hospital admissions or ER visits since diagnosis.</p> <p>Discussion: MS is a devastating disease with a wide spectrum of presentations making it difficult to diagnose. It is the most common non-traumatic cause of disability in young adults, affecting 350,000 individuals in the USA and millions worldwide. Pain without sensory deficits is the predominant symptom in 10-15% of people diagnosed with MS and is classified into 4 major groups: continuous or intermittent central neuropathic pain, musculoskeletal pain, and mixed neuropathic and non-neuropathic pain, A high index of suspicion is required to evaluate young people visiting the ER/PCP multiple times for nonspecific pain symptoms and should prompt the clinicians to consider MS in the differential diagnosis.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Sofia Chaudhry	Naghm LeAnne Jafar	Capital Health Regional Medical Center (Saba Hasan)	Clinical Picture of Posterior Reversible Encephalopathy Syndrome (PRES)	<p>Background: Posterior Reversible Encephalopathy Syndrome (PRES) can present with severe headache, visual changes, and encephalopathy in patients with elevated blood pressures. When the systemic blood pressure is higher than the cerebral auto-regulation range in a chronic hypertensive patient, then there is a breakthrough of auto-regulation, leading to an increased cerebral blood flow and capillary leakage into the interstitium, and thus edema. The most frequent focal deficit is cortical vision loss due to the involvement of the occipital lobes but any focal deficit can be seen. Nonconvulsive seizures can also occur in this hyper-perfusion state. On the MRI, it is seen as symmetrical vasogenic edema most often in the occipital area of the brain but can be seen in other areas as well. The key treatment is to lower the blood pressure with IV antihypertensives and provide supportive care.</p> <p>Case Presentation: A 79 year old Caucasian female with a history of atrial fibrillation, hypertension, type 2 diabetes was last seen in her usual state of health two days prior to presentation. She lived alone and was found by her son to be unresponsive on the floor of her home and thus was intubated in the field. Vital signs were blood pressure 201/156 mmHg, and heart rate 202. Patient was given 10 mg IV lopressor and then subsequently diltiazem drip plus IV heparin. MRI showed: "Multiple patchy regions of FLAIR hyperintensity in bilateral cerebral hemispheres, left basal ganglia, right greater than left cerebellum, and brainstem. Small foci of susceptibility in the right cerebellum may reflect petechial hemorrhage. The findings are concerning for acute ischemic changes from ischemic/anoxic injury. Differential considerations include posterior PRES, which may be less likely given clinical history, infectious etiologies such as viral encephalitis, and inflammatory demyelinating diseases"</p> <p>During the hospital stay, on the first 2 days, the patient was only able to withdraw unilateral upper and lower extremities to pain. By the third day in ICU, she was beginning to follow commands, and able to move all extremities. She was extubated and did well thereafter, having returned completely to baseline neurological state. She was discharged on adequate antihypertensives and instructed on the importance of adhering to therapy.</p> <p>Discussion: The preliminary MRI report read that PRES was unlikely given the clinical scenario and that it was more likely that the patient underwent anoxic brain injury. As a result, the patient was deemed to have a poor prognosis. This case demonstrates that PRES can mimic anoxic brain injury and detection of the characteristic MRI findings, along with appropriate blood pressure management and supportive care is crucial to identify and treat this reversible condition and lead to good outcome.</p>

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Quality Improvement/Patient Safety	Olga Tarasova	Aparna Daley, Delaram Moazami	Capital Health Regional Medical Center (Saba Hasan)	KEEP CALM AND GET VACCINATED - IMPROVING ADULT IMMUNIZATION	<p>Introduction: Acute respiratory infections, including pneumonia and influenza, are the 8th leading cause of death in the United States, accounting for 56,000 deaths annually. Many of these diseases can be avoided by timely use vaccines. Vaccines are one of the most cost-effective clinical preventive services. All adults need immunizations to help prevent acquiring and spreading serious diseases that could result in poor health, missed work, medical bills and the inability to care for family. However, the rates of adult immunization remain poor. One of the reasons for this, as research suggests, is because physicians often neglect to discuss immunization with patients.</p> <p>Method: Prior to initiating this project, medical residents and staff were educated on adult immunization through grand rounds. Residents were identified as Vaccination Champions on each day of the week and nurses acted as a driving force of the project logistics and promoted immunization and documentation. An educational document was provided to patients at the time of check-in. At our clinic, we identified patients requiring immunization with Tdap and the Pneumococcal vaccine for patient above 65 years old. Standing orders for vaccination were implemented. The importance of immunization was emphasized during daily team huddles. Patients were asked at every visit about immunization by both the nurse and the resident physician. Vaccination rates before and after implementation of these strategies were extracted from Capital Health medical records and compared.</p> <p>Results: In 5 month period Tdap rates increased from 5.4% to 38% and immunization rates for Pneumococcal vaccine on eligible patients increased from 18.8% to 37%.</p> <p>Conclusion: Improving Tdap and pneumococcal vaccination rates in adults is essential to prevent associated complications. Effective strategies include educating both staff and patients, addressing immunization with patients at every visit and encouraging educational dialogue.</p>

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Clinical Vignette	Aparna Daley	Albana Mihali, M.D., Delaram Moazami, M.D., Daniel Goldsmith, M.D.	Capital Health Regional Medical Center (Saba Hasan)	Don't Miss Malaria! - Microscopy Diagnosis lies in the Eye of the Beholder	<p>Introduction: Although not endemic in the United States, approximately 1500 – 2000 cases of Malaria are reported in the country annually. Microscopic examination of thin and thick blood smears is considered the gold standard for diagnosis of malaria. However, this modality may be limited by the quality of reagents, experience of the laboratory staff or a low level of parasitemia. The Centers for Disease Control and Prevention recommend that an initially negative smear should be repeated every 12-24 hours for a total of three sets in order to rule out malaria in patients suspected of having the disease. It is important for healthcare providers who may not be exposed to many patients with malaria to recognize that a single negative is insufficient to rule out the diagnosis.</p> <p>Case: A 58 year old male who has lived in the USA for several years presented to the ED complaining of one week of flu-like symptoms, fever, chills, headache, vomiting and diarrhea. His maximum temperature was measured as 100.6°F at home. He reported having returned two weeks ago from a six-week long trip to Ghana. He denied any sick contacts or previous history of Malaria. He had been seen at the same ED three days prior and was discharged on anti-inflammatory agents after a peripheral smear resulted negative for Malaria. The patient was afebrile and the physical examination was unremarkable. A peripheral smear was repeated and resulted as positive for Malaria (<i>Plasmodia falciparum</i>) with 3% parasitemia. The patient also had mild thrombocytopenia with a platelet count of 140 x10 000/microliter and a mildly elevated total bilirubin of 1.5 mg/dL. The patient was admitted to the hospital and completed a three-day course of Atovaquone-proguanil and improved clinically.</p> <p>Discussion: Malaria, especially when caused by <i>Plasmodium falciparum</i> species, can be rapidly fatal. Therefore, prompt diagnosis and appropriate treatment are of the utmost importance. The clinical features of Malaria are often non-specific. A suggestive travel history should alert a physician to the possibility of the diagnosis. Once suspected, the physician should not be reassured by a single negative microscopic result but should repeat the smear as suggested by the CDC. Other options include concurrent testing with an FDA-approved rapid antigen detection test which can rapidly detect the presence of Malaria but cannot confirm the species or the parasitemia. PCR can also be used as an adjunct; however the time constraints are a disadvantage.</p>

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Clinical Vignette	Maryam Siddiqui	Muhammad Y. Khan MD, Abisoye Fakayode MD, Daniel F. Goldsmith MD	Capital Health Regional Medical Center (Saba Hasan)	ISCHEMIC COLITIS IN A YOUNG PATIENT: NOT ALWAYS ATHEROSCLEROTIC DISEASE.	<p>Introduction: Ischemic colitis as the commonest form of gastrointestinal (GI) ischemia accounts for 50 to 60% of all cases. Frequently seen in the elderly, younger patients may also be affected. Predisposing factors for colonic ischemia in young adults include vasculitis, abdominal surgery, use of cocaine, oral contraceptive pills and nonsteroidal anti-inflammatory drugs, hypercoagulable states, colonic obstruction and marathon running. We present a rare case of sigmoid ischemic colitis in a young black female resulting from external compression by uterine fibroids.</p> <p>Case Presentation: A 37 year old African female with no significant past medical history presented with recurrent episodes of lower abdominal pain and bloody diarrhea for 1 year. She reported up to 20 diarrheal episodes with bright red blood. Abdominal pain was localized to left lower quadrant with radiation to the back. Patient denied fever, chills, arthralgia, mouth ulcers, recent travel or weight loss. Patient also recently noticed having menorrhagia. Patient denied smoking, alcohol or illicit drug use. Work up was negative for viral hepatitis and HIV. Stool studies were negative for infectious etiology and Clostridium difficile. Abdominal CT Scan showed colitis and multiple uterine masses, the largest one being 11.9 cm with myomatous changes confirmed on MRI. Colonoscopy showed severe sigmoid colitis with obvious external compression from the pelvic mass correlating with the fibroids. Colonic biopsies were negative for inflammatory bowel disease, microscopic colitis or malignancy. Patient refused hysterectomy but agreed to leuprolide therapy and possible myomectomy. The abdominal symptoms and diarrhea resolved while being on leuprolide therapy.</p> <p>Discussion: The colon has a relatively low blood flow compared with the rest of the GI tract predisposing it to early ischemic changes. Reversible vascular occlusion of the colon was first described by Boley et al in 1963. Increased intraluminal pressures leading to compression of the blood vessels and resultant decreased mucosal vessel flow has been identified in literature as a risk factor for the development of ischemic colitis like in patients with constipation. Clinical improvement is mostly seen within 24 to 48 hours with complete clinical recovery taking 1 to 2 weeks to allow for colonic mucosa to regenerate and heal. This case highlights atypical presentation of intermittent ischemic colitis in a young female due to massive uterine fibroids. It will remind physicians to look for reversible and atypical causes of ischemic colitis, especially in young patients, which can easily be missed leading to frequent visits and morbidity.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Abisoye Fakayode	Sevil Aliyeva MD, Mazhar Noorain MD, Raji Anandakrishnan MD, Daniel Goldsmith MD	Capital Health Regional Medical Center (Saba Hasan)	HYPERFERRITINEMIA IN ADULT ONSET STILLS DISEASE MASQUERADING AS SEPSIS	<p>BACKGROUND: Adult onset Still's disease (AOSD) is a known cause of Hyperferritinemia greater than 10,000mcg/L. It is a rare systemic inflammatory disease of unknown etiology. AOSD is typically considered as a diagnosis of exclusion and a definitive diagnosis should be made based on the Yamaguchi criteria only after excluding infectious, malignant, and other connective tissue diseases.</p> <p>CASE PRESENTATION: A 40 year old Female presented with a three and a half week history of high-grade fever, sore throat, dry cough and a generalized papular rash. She also had complaints of generalized myalgias, arthalgias and an eight pound weight loss over this period. There was a positive family history of rheumatoid arthritis in her mother. On admission she was febrile with temperature of 103, tachycardic with heart rate of 122 and blood pressure of 120/60. She had a papular rash on her right upper back, arms and legs. She also had palpable bilateral axillary and inguinal lymphadenopathy. Workup done revealed elevated ESR of 120 and CRP of 26.5, normal white blood cell count with bandemia of 47% , high neutrophil count of 84.5% and Normocytic Normochromic anemia with hemoglobin of 9.5. Her iron panel did show a Ferritin level of 13,902, total iron of 30, TIBC of 245 and transferrin saturation of 12 . She was admitted and started on empiric antibiotics for suspected sepsis as she fulfilled three SIRS criteria. However, blood cultures returned negative with no infiltrates on chest XRAY, and negative results on the Rapid strep throat test, HIV, hepatitis panel, urine analysis and 2D echo, hence antibiotics were stopped. An extensive rheumatology workup was done with negative Anti-nuclear antibody (ANA), rheumatoid factor (RF), and cyclic citrullinated peptide (Anti-CCP) antibody. Lymph Node Biopsy showed a Reactive lymphoid hyperplasia and dermatopathic lymphadenopathy, negative for malignancy. Hence a diagnosis of adult onset Still's disease was made based on the Yamaguchi criteria. She was started on 60mg of prednisone with rapid improvement in symptoms and fever and discharged home on steroids.</p> <p>DISCUSSION: Hyperferritinemia is a common feature of AOSD and is associated with a sepsis-like clinical picture. It is easy to initially misdiagnose AOSD as Sepsis as highlighted in this case. Diagnosis of AOSD requires at least 5 features, with at least 2 of these being major diagnostic criteria. The patient fulfilled three Major criteria including Fever of at least 39°C for at least a week, Arthralgia or arthritis for at least 2 weeks, Non pruritic salmon colored rash on trunk/extremities and four Minor criteria including Sore throat, Lymphadenopathy, Abnormal liver function tests and Negative tests for RF and ANA.</p>

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Clinical Vignette	Olga Tarasova	Bipinpreet Nagra, Daniel Goldsmith	Capital Health Regional Medical Center (Saba Hasan)	Deadly arrhythmia in postpartum period	<p>Background: In inherited long QT syndrome, female sex is associated with a longer QT interval duration and a higher risk for potentially lethal polymorphic ventricular tachycardia and sudden cardiac death.</p> <p>Case: A 36 year-old African-American female with baseline mild non-ischemic cardiomyopathy (diagnosed by cardiac MRI 1.5 years ago with left ventricle ejection fraction (LVEF) of 45% and normal cardiac catheterization) and mildly prolonged QT interval presented to her cardiologist complaining of progressively worsening dyspnea, three pillow orthopnea, intermittent chest tightness and leg edema. Four weeks ago, she delivered healthy twins via C-section. She was not breastfeeding. She had two prior pregnancies and the last pregnancy was uneventful. Family history was positive for sudden cardiac arrest in her 1st degree cousin at the age of 26 years. The patient was sent to the emergency room. Initial EKG showed sinus rhythm with prolonged QT interval of 649msec. While in the emergency room, the patient became pulseless and developed multiple episodes of sustained Torsade de Pointe (TdP) requiring emergent defibrillation. The EKG showed T wave alternans with QTc of 567msec. A bolus of 300 mg of IV Amiodarone was administered by emergency room physician followed by IV Amiodarone drip. After resuscitation the patient was intubated and admitted to the ICU for acute heart failure. Patient on consultation with cardiology was started on IV Lidocaine with no farther recurrence of TdP. The EKG taken after administration of Amiodarone showed QTc interval of 682msec and the next day QTc was 698 msec. Electrolytes were within normal range. ECHO-cardiogram revealed mildly dilated left ventricle with mild concentric left ventricle hypertrophy and severe global hypokinesis of left ventricle with LVEF of 15-20%. Patient was aggressively diuresed and the following day she was successfully extubated. Spironolactone was added and a low dose beta-blocker was resumed. Later the same day, the patient was transferred to a tertiary care center under the management of a heart failure care team, where after stabilization, an AICD was placed.</p> <p>Conclusion: In patients with prolonged QT syndrome, while the arrhythmogenic risk is relatively low during pregnancy it is particularly pronounced during the first 9 months of the postpartum period. Amiodarone may aggravate ventricular arrhythmias in such patients with prolonged QT syndrome. This case illustrates the markedly increased arrhythmogenicity in patient with prolonged QT syndrome in postpartum settings especially if there is an onset of postpartum cardiomyopathy. In-addition it highlights that Lidocaine should be the preferred antiarrhythmic drug in such patients.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Jawad Malik	Bushra Saleem, Iram Mahmood, Emily Chen, Lindita Shehu	Capital Health Regional Medical Center (Saba Hasan)	Hypertensive Crisis From Hematologic Phenomena	<p>Background: Hypertensive emergency is a common condition encountered in the hospital. Identifying the underlying etiology is very important because it can alter clinical outcome and management.</p> <p>Case: A 42-year-old man with Hypertension presented with exertional dyspnea. He admitted to taking his blood pressure medication inconsistently. While in the ED, blood pressure was 215/163mmhg with bilateral lower extremity edema. Chest x-ray showed cardiomegaly with bibasilar interstitial prominence. Creatinine was found to be 10.14 with unknown baseline, but patient denied any history of kidney dysfunction. Also, patient had hemoglobin of 8.5g/dl with unknown baseline. Patient was admitted to ICU and initially managed with Intravenous furosemide and nitroglycerine for hypertensive emergency with flash pulmonary edema, which resolved his shortness of breath. However, blood pressure remained elevated so nicardipine drip was started. Creatinine continued to rise to 13.41 and patient was developing metabolic acidosis. Also, Hemoglobin dropped to 7.3g/dl. Nephrology was consulted for potential initiation of dialysis. However, due to acute onset of presentation secondary causes were being considered, so plans for renal biopsy were made. For workup of anemia, LDH elevated (1134), haptoglobin decreased (<15) consistent with hemolysis. Moreover, peripheral smear showed schistocytes, platelet count was 87000, and results of kidney biopsy showed thrombotic microangiopathy. Due to clinical picture of uncontrolled blood pressure, hemolysis, schistocytes, thrombocytopenia and thrombotic microangiopathy, diagnosis of Atypical Hemolytic Uremic syndrome was made. Next, plans were made to hold dialysis and initiate treatment with eculizumab. Over the course of next few months blood pressure was well controlled on oral medications, creatinine came down to 3.5, hemoglobin improved to 12g/dl, and platelet count normalized.</p> <p>Discussion: Atypical Hemolytic Syndrome is a rare genetic disorder that primarily effects kidney function. It occurs by unregulated complement activation leading to thrombi formation in small blood vessels. AHUS has three features: hemolytic anemia, thrombocytopenia, and renal failure. Uncontrolled blood pressure can also result when thrombi block small vessels in the kidney, causing elevation of renin. Also, renal failure occurs secondary to thrombi causing ischemia to the kidneys. Eculizumab is a recombinant humanized monoclonal antibody that inhibits the complement cascade, which makes this drug effective treatment for AHUS. This case highlights importance of clinicians identifying underlying etiology in kidney disease, even if a common cause of CKD appears likely. By identifying the underlying etiology, this case was successfully managed with drug therapy as opposed to commitment to dialysis.</p>

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Clinical Vignette	Sree Teja Yelamanchili	Bushra Saleem, MD; Sarah Sordo, MD; Rajashree Anandakrishnan MD	Capital Health Regional Medical Center (Saba Hasan)	PITFALLS OF IMMUNE THERAPY- A CASE REPORT OF MYCOBACTERIUM KANSASII ASSOCIATED WITH AZATHIOPRINE USE	<p>Background: Non-tuberculous mycobacteria (NTM) are found ubiquitously in nature including soil and water. The incidence of NTM pulmonary disease is increasing throughout the world with some estimates of an increase of about 8.2% per year. Currently NTM pulmonary disease is more common in the US than tuberculosis. In HIV-negative patients, the major predisposing factor is pre-existing lung disease. However, it is also associated with malignancy, immunosuppressive medications and alcohol use.</p> <p>Case Report: A 64-year-old male with history of hypertension and pauci-immune glomerulonephritis (GN) characterized by focal segmental necrotizing GN and p-ANCA positivity presented to the emergency room with fatigue, cough and weight loss for 2 months. Prior to this presentation, he was treated with prednisone and cyclophosphamide as induction therapy for his GN, and had been on azathioprine maintenance therapy for nearly 7 months. Physical examination revealed cervical lymphadenopathy. A CAT scan of chest showed bilateral pulmonary nodules and bilateral mediastinal lymphadenopathy. HIV screen was negative. A cervical lymph node biopsy was performed with final cultures growing Mycobacterium kansasii. Neither tuberculin skin test (TST) nor an interferon gamma release assay (IGRA) were done during the induction or maintenance phase of immunosuppressive drugs. Therapy with azathioprine was continued due to obvious normalization of kidney function and patient was started on a three-drug regimen with clarithromycin, rifampin and ethambutol.</p> <p>Discussion: The association of reactivation of latent tuberculosis and NTM infections with TNF- alpha inhibitors use is well documented. Here we present a case of Mycobacterium kansasii infection associated with long term azathioprine use for pauci-immune GN. We have guidelines for testing for active and latent tuberculosis infection before starting anti-TNF agents. However, M. tuberculosis and NTM infections have also been described in patients receiving non-anti-TNF immunosuppressive therapy. Nonetheless, testing for latent tuberculosis is currently not the standard of care for non-TNF-alpha-inhibitor immunosuppressive therapy. Further case reports and prospective trials will be useful and necessary to assess whether to recommend routine testing for latent or active tuberculosis in this setting.</p>

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Clinical Vignette	Ellen Wang	A. Hakim Shoustari, O. Tarasova, S. Tiekou, D. Goldsmith	Capital Health Regional Medical Center (Saba Hasan)	Inevitable Extrapontine Myelinolysis	<p>Introduction: Hypernatremia is most commonly due to free water loss, therefore resulting in elevated plasma osmolality. It becomes pathologic when the body fails to adequately compensate the volume and fluid deficits through thirst mechanism or prompt rehydration. In a critical setting of symptomatic hypernatremia, careful fluid replacement and closely monitored sodium level are warranted to slowly lower the serum sodium to avoid pontine demyelination. In practice, correction of hypernatremia is often clinically challenging despite careful fluid replacement. We report a case of extrapontine myelinolysis occurred in a setting of very gradual correction of hypernatremia.</p> <p>Case: A 44 years old female was found in the woods after being reported missing for 6 days by family. She was tachycardic, hypertensive, tachypneic, febrile, and appeared dry, warm, with moderate distress. She was lethargic but oriented and followed commands. Physical exam is significant for multiple bruises, excoriations, and moderate epigastric tenderness. The laboratory workup revealed sodium of 191 milliequivalents/liter, osmolality of 431 milliosmoles/kilogram, creatinine of 5.06 milligram/deciliter, and calcium of 10.4 milligram/deciliter. Leukocytes were 14.4 with 30% bandemia, and creatine kinase was 7558 units/liter. Trauma workup was negative. After receiving resuscitation with IV fluids and antibiotics in emergency room, she was admitted to intensive care unit for severe hypernatremia, acute renal injury and rhabdomyolysis. Intravenous normal saline was initiated with the goal to correct the hypernatremia at 1 milliequivalents/liter/hour or 10 milliequivalents/liter/24 hours. Sodium level was followed every 4-6 hours, and infusion rate was adjusted per recalculation by the formula. On day 3 with sodium of 177 milliequivalents/liter, she developed respiratory distress and appeared more lethargic with left proximal drifting, hypoactive reflex, downward plantar responses, and bilateral leg weakness. She was intubated. Her MRI revealed symmetric restricted diffusion in the internal capsules and brachium pontis but spared globus pallidus, which was not typical for myelinolysis. The intravenous fluid treatment continued with frequent sodium level check. Sodium level was normalized on day 7, however she remained unresponsive. The repeat MRI revealed increasing diffusion restriction in brachium pontis, internal capsules, cerebellum, crura of fornices. Diagnosis of extrapontine myelinolysis was made. The patient developed extrapontine myelinolysis despite adequate sodium correction. Through time and supportive management, she slowly improved clinically and was extubated on day 14. She was alert, awake, and oriented on day 22 and discharged to rehabilitation.</p> <p>Discussion: The recommended rate of correction for hypernatremia is 10 milliequivalents/day, however data have shown most cases have slower rate at < 0.25 milliequivalents/hour. Furthermore, most reported cases of pontine demyelination are among pediatric population. We speculate the adequate fluid replacement therapy in adult population, especially in cases of severe hypernatremia, can be further reduced to even 5 milliequivalents/day, give no significant focal neurologic deficits or seizures occurred.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Srinivas Ayyala	Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	A Rare Case of Sweet Syndrome with Pemphigus Vulgaris	<p>Introduction: Reactive neutrophilic dermatosis is known as Sweet’s syndrome, which is usually associated with an underlying malignancy but rarely represents an autoimmune association.</p> <p>Case: A 50-year-old Caucasian female with history of fibromyalgia, depression and pemphigus on venlafaxine and dapsone presented with a diffuse painful and mildly pruritic rash on her abdomen, chest, upper back, and extremities for three days. The rash was associated with recent onset fatigue and multiple joint pains without swelling. She denied oral mucosal involvement, fever, photosensitivity, recent travel, new medication or food, diarrhea, vaccination or ill contacts. Her vital signs were within normal limits. Physical examination revealed diffuse red annular papules, plaques and nodules that were mildly tender with scattered flaccid bullae. A punch biopsy showed dermal perivascular and interstitial lymphocytic and neutrophilic infiltrate around the vessels in the papillary and reticular dermis, consistent with a diagnosis of Sweet’s syndrome. She had mild leukocytosis (11,900/mcL), elevated ESR (55 mm/hr) and elevated CRP (6.0 mg/L). She had normal rest of the complete blood count, complete metabolic panel, antinuclear antibody, thyroid stimulating hormone, rheumatoid factor, urinalysis, anti-CCP antibodies and ferritin. She was treated with prednisone 60 mg/day for two weeks on a tapering regimen. The cutaneous lesions reached near resolution after two weeks. Work up for an underlying malignancy was negative.</p> <p>Discussion: Pemphigus vulgaris is an autoimmune blistering disease of the skin and mucous membranes. Sweet’s syndrome typically presents with acute onset of fever, leukocytosis and tender erythematous skin lesions infiltrated by neutrophils. Internal organ involvement is common. Clinically, patients with sweet’s syndrome typically present in one of three ways including idiopathic, malignancy-associated or drug-induced forms. The idiopathic form typically presents in women between the ages of 30 to 50 years, is often preceded by an upper respiratory tract infection and may be associated with inflammatory bowel disease and pregnancy. The pathogenesis is multi-factorial, mostly cytokine mediated. Diagnosis requires fulfillment of both major criteria (1, 2) and two of four minor criteria (4-6): (1) Abrupt onset of painful erythematous plaques or nodules (2) Dense neutrophilic infiltrate without evidence of leukocytoclastic vasculitis (3) Pyrexia >38°C (4) Association with an underlying hematologic or visceral malignancy, inflammatory disease, or pregnancy, OR preceded by an upper respiratory or gastrointestinal infection or vaccination (5) Excellent response to treatment with systemic corticosteroids or potassium iodide (6) Abnormal laboratory values at presentation (three of four): erythrocyte sedimentation rate >20 mm/hr; positive C-reactive protein; >8,000 leukocytes; >70% neutrophils. Systemic corticosteroids improve skin lesions however, recurrence is common. Although rarely reported, underlying autoantibodies with pemphigus vulgaris might explain Sweet’s syndrome in our case.</p> <p>Conclusion: Our case of Sweet’s syndrome with pemphigus vulgaris suggests that Sweet’s syndrome may be associated with autoimmune diseases.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Agnes Adarkwa	Satyajeet Roy MD	Cooper University Hospital (Brian Gable)	A rare case of diffuse large B-cell lymphoma of the nasal sinus in a nonagenarian	<p>Introduction: Malignant lymphoma of the nasal sinuses are rare. Early diagnosis is challenging due to nonspecific clinical presentation.</p> <p>Case: A 95-year-old Caucasian woman with history of well controlled hypertension and hypothyroidism on amlodipine, candesartan and levothyroxine presented because her “eyes were bothering.” She had received 3 courses of antibiotic therapy in the previous 4 months for sinusitis. Subsequently she noticed a painless purple mass in the right nostril. She denied fever, diplopia, nasal discharge, headache or epistaxis. CT scan of the nose and sinuses showed a 30 mm right inferior nasal soft tissue mass with paranasal sinus mucosal thickening without fluid level. Excision biopsy of the mass showed diffuse large B-cell lymphoma consisting of infiltrates of large cells with variably prominent nucleoli and scant cytoplasm. Frequent apoptotic bodies were noted. Immunostains confirmed B cells with the following immunophenotype: CD20+PAX5+BCL6+(subset)CD10+MUM1+ BCL2-. Ki67 revealed a proliferative index of approximately 90%. T cells were admixed in the background. In-situ hybridization for EBV was negative. Pancytokeratin highlighted squamous epithelium. Imaging of the whole body revealed no other tumors or lymphadenopathy. Considering the involvement of the nasal passage a CSF examination was performed which showed no abnormality. She was diagnosed with nasal diffuse large B cell lymphoma (DLBCL). The patient was treated with rituxmab and craniofacial radiation with resolution of previously noted hypermetabolism in the nasal mucosa. She remained asymptomatic after 1 year.</p> <p>Discussion: DLBCL of the primary nasal and paranasal sinuses is rare and represents only 0.17% of all lymphomas. It is most commonly found in Caucasians and is diagnosed in 7 out of 100,000 people in the United States. The average age of diagnosis is 64 years. It is a neoplasm of the B lymphocytes. Mutations within the B cells facilitate their proliferation in an unchecked manner. DLBCL encompasses different histological lymphoma entities with different clinical profiles and outcome. It has high propensity for CNS dissemination. Other differential diagnoses should be considered, such as infectious mononucleosis, melanoma and other lymphomas. When considering treatment, patients are categorized as having either limited or advanced stage disease. Limited stage disease implies that involvement of lymph nodes within one irradiation field. Treatment includes chemotherapy, rituximab and possibly radiation. Advanced stage disease involves more than one irradiation field. Treatment is with systemic chemotherapy and rituximab. Hematopoietic cell transplantation is considered in patients who have relapsed. Some patients are successfully cured and about 45.9% of DLBCL patients remain alive 10 years after therapy.</p> <p>Conclusion: DLBCL of the primary nasal and paranasal sinuses may present with clinical features suggestive of sinusitis. A high index of suspicion and early imaging studies can provide a favorable outcome.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Agnes Adarkwa	Satyajeet Roy MD	Cooper University Hospital (Brian Gable)	Hyperlipidemia and electrolytes – Laboratory values that need deeper analysis	<p>Introduction: Dyslipidemia in diabetic patients is common. It can present with severe electrolyte abnormalities due to indirect laboratory measurement of the electrolytes.</p> <p>Case: A 58-year-old Caucasian woman with history of hypertension and type-2 diabetes mellitus presented with malaise and tiredness for 1 week. Patient denied fever, vomiting, diarrhea, headache, dizziness, constipation, dysuria, or consumption of alcohol. She was managed with metformin, insulin glargine, losartan and simvastatin. Her fasting blood glucose ranged between 150 and 320 due to poor compliance to a carbohydrate restricted diet. Her vital signs were within normal range. Her body mass index was 42.6 kg/m². Physical examination was unremarkable. Her laboratory tests showed fasting glucose (FBG) 256 mg/dL, sodium (SNa) 124 mEq/L, blood urea nitrogen (BUN) 20 mg/dL, total cholesterol 488 mg/dL, triglyceride (TG) 1,680 mg/dL, HDL 42 mg/dL, and measured plasma osmolality (Posm) 286 mOsm/kgH₂O. The rest of her complete metabolic panel, anion gap, complete blood count, urine osmolality, serum ketone, thyroid stimulating hormone, serum alcohol level were normal. Her Posm-[2xSNa+BUN/2.8] was greater than Glucose/18, and Posm-[2xSNa+Glucose/18] was greater than BUN/2.8. She denied use of mannitol, sorbitol or glycine. She was diagnosed as pseudohyponatremia secondary to severe hypertriglyceridemia due to uncontrolled type-2 diabetes mellitus. She was managed with tight glycemic control (carbohydrate restricted diet and insulin) without change in sodium or water intake. At 3-day follow up she remained asymptomatic. Her lab results showed FBG 108 mg/dL, TG 520 mg/dL SNa 131 mEq/L. At 7-day follow up her lab results showed FBG 98 mg/dL, TG 158 mg/dL SNa 139 mEq/L.</p> <p>Discussion: Patients with hypertriglyceridemia or hyperproteinemia can have falsely low SNa levels. These patients usually do not exhibit any symptoms or signs of hyponatremia. This falsely low reading of SNa occurs due to indirect ion selective electrodes usage by the laboratories in the United States. This process includes significant dilution and specific calculation that assumes 93% water and 7% fats and proteins in patients' plasma. Patients with hyperlipidemia and hyperproteinemia have less than 80% of water in plasma, which upon further dilution reduces the calculated value of SNa. SNa is reported as low while the overall sodium concentration remains normal. This problem can be resolved by use of direct ion selective electrodes which do not require dilution and do not depend on plasma water content.</p> <p>Conclusion: It is important to identify pseudohyponatremia in patient with hypertriglyceridemia or hyperproteinemia who require a prompt diagnosis and correction of the underlying etiology without water restriction or sodium based therapy.</p>

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Clinical Vignette	Matthew Varner	Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	Double whammy - Staghorn calculus and horseshoe kidney	<p>Introduction: Horseshoe kidney is a common congenital abnormality that most of the time is asymptomatic. However some patients may present with urinary tract infection or obstruction. Urolithiasis, which is also common, can affect these patients, however management of urolithiasis is challenging and effective management is required in order to prevent serious complications. The case below illustrates the devastating combination of horseshoe kidney and renal calculi.</p> <p>Case: A 71 year-old male with history of horseshoe kidney and urolithiasis presented with acute onset persistent sharp pain on left the side of his back, radiating to the flank associated with fevers and chills. On physical exam, he was afebrile and normotensive. His left flank was tender to palpation. He had leukocytosis (WBC 17,100/ul) and elevated serum creatinine (1.9 mg/dl). Urinalysis showed pH 6.5, WBC >180, RBC >180, 3+ bacteria, 3+ leukocyte esterase, negative nitrates. A plain abdominal X-ray of the kidney revealed a left sided staghorn calculus. A non-contrast abdominal CT scan showed a staghorn calculi within the left side of the horseshoe kidney encompassing the mid to lower pole region with severe hydronephrosis and significant cortical thinning of the left kidney. Dynamic Radionuclide Renal Imaging Scan with Furosemide showed delayed left kidney perfusion with relative renal function of 8% on the left and 92% on the right. Urine cultures were positive from Escherichia coli sensitive to Cephalosporin. The patient underwent a left sided nephrectomy.</p> <p>Discussion: Horseshoe kidney is the most common fusion anomaly with a reported incidence of 0.4 to 1.6 in 10,000 live births. It occurs when one pole of each kidney fuses. In more than 90 percent of cases, fusion occurs at the lower poles; as a result, two separate excretory renal units and ureters are maintained. Majority of the patients with horseshoe kidneys are asymptomatic and are diagnosed incidentally. However occasionally patients present with infection or obstruction. Urolithiasis is also a common entity. In patients with congenitally abnormal kidneys as such with horseshoe kidney, renal calculi are reported to occur in 20 percent of cases. Stone formation causes obstruction that leads to urinary stasis and increased risk of infection. The specific renal anatomy associated with this condition such as ureteropelvic junction obstruction is a factor that may influence treatment and makes it challenging. This patient had a history of horseshoe kidney as well as urolithiasis with previous urinary tract infection that progressed towards the development of staghorn calculi. As a result patient lost significant renal function and required referral for nephrectomy.</p> <p>Conclusion: Patients with horseshoe kidney, recurrent UTI and urolithiasis may progress to significant loss of unilateral renal function. An early diagnosis and management may save the functioning kidney from deterioration.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ethan Goldstein	Samantha Lee, MD; Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	Biopsy Proven Soft Tissue Metastasis as First Sign of Metastatic Lung Adenocarcinoma	<p>Introduction: Gradual onset painless thoracic subcutaneous mass mostly represents a benign etiology but it can rarely be a manifestation a serious underlying disorder.</p> <p>Case: A 60 year-old woman with asthma and rheumatoid arthritis presented with a gradual onset and slowly progressive lump on her back. She denied fever, local trauma, pain, drainage of pus or similar lumps anywhere else on her body. She had a history of more than 30-pack-year cigarette smoking. She also complained of intermittent cough with mild wheezing. Her vital signs were within normal range. She had a 10 mm size soft to firm mass on the dorsal aspect of her thorax about 2 cm right to the mid thorax. She had mild bilateral scattered rhonchi. Rest of her physical examination was normal. Her cough prompted a routine chest X-ray which showed large hilar mass. A chest CT confirmed a large hilar mass and soft tissue densities on her back. Bronchoscopy was unsuccessful for biopsy, but biopsy of back mass showed poorly differentiated adenocarcinoma. Originally her masses were not bothersome but grew over time and became painful. She was diagnosed with stage 4 poorly differentiated adenocarcinoma of lung with subcutaneous metastasis. With chemotherapeutic management she did not do well and was eventually transitioned to hospice within a few months of diagnosis.</p> <p>Discussion: Metastasis of lung cancer to soft tissue as first finding has been reported in case studies, however most literature discussing soft tissue spread comes as a late finding with poor prognosis. The frequency of metastatic soft tissue tumors has been reported as 0.2-2.7%. The common location of the metastases are reported as abdominal wall (25%), back (25%), thigh (19%), chest wall (13%) and arm (6%). The most common source of primary malignancies vary in different case series studies. In one study (Torigoe, et al, 2011) lung cancer (38%), lymphoma (31%), stomach cancer (13%), esophageal, pancreatic and uterine cancer (6%) represent the likely primaries. To date, there is still no established treatment, as this typically is found with aggressive disease. According to one review (Damron, et al, 2000) combination treatment with radiation and chemotherapy is indicated for metastatic soft tissue tumors in 40% of cases, radiation alone in 24%, radiation and resection in 20%, radiation and resection and chemotherapy in 13%, and no treatment in 3%.</p> <p>Conclusion: Patients who present with suspicious subcutaneous masses warrant full evaluation including thorough history to screen for cancer risk factors, physical examination for possible metastatic disease, and diagnostic tests as indicated as subcutaneous masses can be the only presentation of a serious systemic disorder, such as lung cancer in our patient.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Megan Gooch	Margaret Manus, MD; Kunal Dalal, MD; Paul Kannarkatt, MD; Satyajeet Roy, MD	Cooper University Hospital (Brian Gable)	Low-Dose CT (LDCT) Scan Findings Among Patients who Underwent Lung Cancer Screening in Community-Based Practice	<p>BACKGROUND: The USPTF received the first official guidelines for lung cancer screening in December 2013 and the results of which in the general population may now be observed. Patient between ages 55-80 years with a 30 pack year smoking history who are current smokers or who quit within the past 15 years are eligible for lung cancer screening with Low-Dose Computed Tomography (LDCT).</p> <p>AIMS: Review the results of LDCT ordered for the purpose of lung cancer screening based on the 2013 USPTF Lung Cancer Screening Guidelines in the Southern New Jersey population.</p> <p>METHODS: We retrospectively reviewed the electronic medical records patients between 55 and 80 years of age who underwent LDCT for purpose of lung cancer screening between January 2014 and January 2015. Results of the LDCT were placed into one of three groups: normal lung findings (Group 1), abnormal without malignancy (Group 2), abnormal with malignancy (Group 3). Factors such the length of time since smoking cessation (if applicable), previously diagnosed lung disease, and comorbid conditions such as diabetes mellitus, hypertension, hyperlipidemia, and coronary artery disease were also analyzed. Patients found to have pulmonary nodules were further stratified into groups based on Fleischner Criteria. The groups were statistically compared using the Chi-Square Test, Independent T-Test, and Simple T-Test.</p> <p>RESULTS: A total of 124 patients were included in the study. The age range of the patients was 55-78 years old with an average age of 62.3 years. Group 1 included 32 patients (25.8%), Group 2 included 92 patients (74.2%), and Group 3 included zero patients. The majority of patients, 68.6%, who underwent LDCT were Caucasian; 78.9% of which had abnormal LDCT ($p < 0.001$). Two-thirds of patients included in the study were current tobacco users at time of LDCT. The majority of patients who underwent screening had hyperlipidemia (53.2%), hypertension (47.9%), diabetes mellitus (22.9%), and CAD (21.8%). Only 5.6% had COPD. Patients with abnormal LDCT had significantly increased prevalence of hyperlipidemia (46.9%) and CAD (26.1%) compared to patients with normal findings ($p < 0.0001$). The majority of patients with abnormal findings (Group 2) demonstrated newly identified pulmonary nodules (59.9%). Most pulmonary nodules were found to be less than 4mm (56.9%), followed by 4-6mm (27.3%), 6-8mm (12.7%), and greater than 8mm (9.1%). Of other abnormal findings, most were found to have emphysema (43.5%) or pleural thickenings/opacifications (10.9%). Of pulmonary nodules discovered, 5 (9.1%) were greater than 8 mm.</p> <p>CONCLUSION: Although no malignant lesions were identified on LDCT, pulmonary nodules requiring close follow-up or biopsy were noted. Additionally, pulmonary abnormalities other than nodules were identified in the majority of patients.</p>

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Clinical Vignette	Manisha Jakkidi	Manisha Jakkidi, MD ; Satyajeeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	An atypical presentation of Marginal Zone Lymphoma associated with Hepatitis C Infection	<p>Introduction: Mucosa Associated Lymphoid Tissue (MALT) lymphomas of the ocular adnexa can be associated with certain infections. We present a rare case of EMZL of ocular adnexa with nodal and bone marrow involvement likely due to HCV infection.</p> <p>Case: A 68-year-old male presented with right eye swelling, painful eye movements and diplopia for 3 weeks. He had no vision loss, redness, floaters, eye trauma, fever, headache or weakness. He smoked cigarettes and used cocaine. He denied personal or family history of malignancy, vasculitis, autoimmune or thyroid disease. His vital signs were normal. He had marked proptosis of the right eye and bilateral eyelid swelling with no congestion or visible discharge. He also had bilateral non tender axillary and inguinal lymphadenopathy. Complete blood count revealed mild anemia(hemoglobin 12g/dL). He had an elevated hepatitis C viral load. CT scan of the head and orbits showed a right superior rectus muscle mass, proptosis of the right globe, asymmetrically enlarged right temporalis muscle and subcutaneous nodules overlying the left temporalis muscle. CT scan of the chest, abdomen and pelvis revealed mediastinal, axillary and scattered mild lymphadenopathy in the abdomen. Bone marrow biopsy revealed hyper cellularity and morphologic evidence of malignant lymphoma. A tandem-flow-cytometry demonstrated small lambda light chain restricted B-cell population with immunophenotypes CD5-CD10-CD19+CD20+CD22+. Excision biopsy of the right axillary lymph node established the diagnosis of Marginal Zone Lymphoma (MZL). His proptosis and blurred vision improved with a five day course of Prednisone 80 mg daily. He was discharged home with a plan to initiate chemotherapy and anti-Hepatitis C therapy.</p> <p>Discussion: MZLs are a group of indolent Non-Hodgkin B-cell lymphomas. They include: Extranodal MZL (EMZL or MALToma), splenic MZL (SMZL) and nodal MZL (NMZL). EMZL accounts for 70% of all MZLs and can arise from any extra nodal site due to chronic antigenic stimulation either as a result of infection (example, Helicobacter pylori and stomach) or autoimmune disease (example, Sjogrens syndrome and salivary glands). Long-standing antigenic stimulation explains how lymphoid infiltrates may appear in extranodal sites that are normally devoid of lymphoid tissue (example, stomach, salivary glands, orbits). Infections associated with MLZs include Helicobacter pylori, Hepatitis C, Campylobacter Jejuni, Borrelia Burgdorferi. Orbital MALT Lymphomas are commonly associated with Chlamydia Psittaci infections. There is growing evidence that Hepatitis C virus is associated with B-cell non-Hodgkin lymphomas, such as diffuse large B cell lymphoma, small lymphocytic lymphoma and MZLs. Among the MZLs, SMZL and NMZL are more commonly associated with chronic HCV infection, rather than EMZL.</p> <p>Conclusion: MALT lymphoma is among the most common malignant lymphomas of the ocular adnexa .It is important to be aware of the infections associated with EZML for management of these malignancies.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Shivani Gandhi	Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	Acute exertional compartment syndrome after chemotherapy	<p>Introduction: Acute exertional compartment syndrome (ACS) after chemotherapy is extremely rare.</p> <p>Case Presentation: A 35 year-old female athlete presented with diffuse muscle pain, malaise, weakness and inability to walk for two days. Her past medical history was significant for BRCA-positive, triple-negative (ER-, PR-, and HER2-) breast cancer. She underwent one treatment of neoadjuvant dose-dense chemotherapy with doxorubicin, 60 mg/m² and cyclophosphamide, 600 mg/m² nine days prior to presentation. This was followed by pegfilgrastim 3 mg. The patient was a seasoned athlete who had participated in an extreme exercise-training program prior to her breast cancer diagnosis. She attended an exercise training class several days after getting chemotherapy, and prior to the onset of her symptoms. She was tachycardic (128 beats/min), in moderate distress, and had bilateral lower extremity edema with tenderness to palpation, mostly in both of her thighs. She had an elevated serum creatinine (1.61 mg/dl), metabolic acidosis (anion gap of 27), and creatine kinase of 9,174 U/L. She was adequately treated with intravenous fluids and empiric antibiotics. Repeat bloodwork showed worsening metabolic acidosis (pH 7.01), hyperkalemia (7 mmol/L) refractory to medical therapy with calcium gluconate and bicarbonate, increasing serum creatinine (2.51 mg/dl), and rising creatine kinase (34,120 U/L). Stryker Intra-Compartmental Pressure Monitor recorded pressures in the right and left anterior thighs as 24 mmHg and 35 mmHg, respectively (normal < 10 mmHg). She underwent emergent bedside fasciotomies, but continued to have rhabdomyolysis and renal failure requiring continuous venovenous hemodialysis. Despite our best efforts, the patient suffered a cardiac arrest from severe hyperkalemia, and she could not be resuscitated.</p> <p>Discussion: ACS of the bilateral anterior thighs after exercise is exceedingly rare, and only several case reports exist in the current literature. Infections, medications, and congenital storage disorders can predispose myocytes to injury, which may increase the risk for the development of ACS. Anti-neoplastic agents can rarely cause myotoxicity. Doxorubicin is known to cause cardiotoxicity, and has been shown to have some toxicity to skeletal muscle in animal models. When synthesizing this information in relation to our case, it is possible that our patient, who was previously a physically fit and seasoned athlete, might have been predisposed to the development of exertional ACS after chemotherapy. In our day and age, intense exercise training programs continue to gain widespread recognition amongst various populations, some of which may include our patients. ACS must be on the differential, as a delay in diagnosis can be fatal. Furthermore, clinicians administering certain medications should be especially aware of potential myotoxicity, and they should counsel their patients accordingly.</p> <p>Conclusion: Exertional compartment syndrome can be rapidly fatal. Physicians should counsel patients on the risks of extreme exercise programs, especially with concomitant use of myotoxic medications.</p>

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Clinical Research	Shivani Gandhi	Karim Nathan, MD; Iris Hagans, MD; Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	Prevalence, comorbidity, and investigation of anemia in the primary care office	<p>Background: Anemia has a myriad of causes and its prevalence is growing. Anemia is associated with increased all-cause hospitalization and mortality in community-dwelling individuals above age 65 years. Aim: To determine the prevalence and severity of anemia in adult patients in our primary care office and to determine relationships between anemia and medical comorbidities.</p> <p>Materials and Methods: Electronic medical records of 500 adult patients in our suburban internal medicine office were reviewed who had had at least one hemoglobin value and did not undergo moderate to high-risk surgery in the preceding 30 days.</p> <p>Results: 21.6% (108/500) of patients had anemia. Of these patients, 60.1% had mild anemia (hemoglobin 11-12.9 g/dL) and 39.8% had moderate anemia (hemoglobin 8-10.9 g/dL). For every year of increase in age, there was 1.8% increased odds of having anemia. African-Americans had 5.2 greater odds of having anemia than Caucasians. Hispanics had 3.2 greater odds of having anemia compared to Caucasians. Patients with anemia had a greater average number of comorbidities compared to patients without anemia (1.74 and 0.96, respectively; p 0.049).</p> <p>The mean age of patients with anemia was 62.6 years. Comorbid medical conditions associated with anemia were diabetes mellitus (19.4%), hypertension (59.3%), hypothyroidism (21.3%), chronic kidney disease (11.1%), malignancy (18.5%), rheumatologic disease (7.4%), liver disease (6.5%), congestive heart failure (6.5%), chronic obstructive pulmonary disease (5.6%), and coronary artery disease (18.5%). 41% patients with mild anemia and 62% patients with moderate anemia underwent additional diagnostic studies. 14.8% patients had resolution of anemia without therapy in one year, 15.7% were on iron replacement therapy, and 6.5% were on cobalamin therapy. No specific etiology of anemia was found in 24% of patients.</p> <p>Discussion: Our results suggest that anemia is prevalent in the outpatient setting, and that African-American and Hispanic race have greater odds of having anemia within a population. Given the growing prevalence of anemia in the adult population, several studies have sought to determine the impact of anemia on health. Recent evidence suggests that anemia is not only a symptom or result of underlying disease, but it can also be an independent risk factor for morbidity and mortality. In 2006, Culleton et. al showed that anemia was associated with increased all-cause hospitalization and mortality in community-dwelling elderly above age 65. Furthermore, even mild anemia was associated with decreased physical performance, increased prevalence of dementia, and poorer quality of life in the elderly. Studies show that improving hemoglobin level improves symptom burden. Further studies are required to demonstrate a long-term benefit in correcting underlying anemia.</p> <p>Conclusion: It is important to be aware of the demographic factors and their relationship to anemia in primary care. Furthermore, it is crucial to identify reversible causes of anemia to prevent potential complications.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	James Liu	Vinay Palli, MD; Satyajeet Roy, MD	Cooper University Hospital (Brian Gable)	May-Thurner Syndrome in a 44 year old man with flank pain	<p>Introduction: May-Thurner Syndrome (MTS) is characterized by the compression of the left common iliac vein by the right common iliac artery.</p> <p>Case: A 44-year-old man with a past medical history of nephrolithiasis presented with acute left flank and thigh pain associated with left leg swelling. His left leg swelling progressed gradually to diffuse left thigh swelling and pain in next 3-4 hours. He had no chest pain or shortness of breath. His vital signs were within normal limits. Physical examination revealed diffuse tenderness and edema in his left lower extremity and left lower abdomen. An ultrasonography of his left lower extremity showed extensive left iliofemoral occlusive thrombus extending to the left popliteal vein. A contrast venogram showed compression at the left common iliac vein with radiographic findings consistent with MTS. He underwent urgent thrombectomy and iliac and femoral vein stent placements. He was discharged on warfarin. Patient gradually improved and remained asymptomatic at 3 months follow-up.</p> <p>Discussion: MTS or Iliac vein compression syndrome results from the compression of the left common iliac vein by the right common iliac artery. Estimated prevalence of this anatomic variant is 22-24%, although frequency of iliac vein thrombosis is much lower (1-3 individuals per 1,000 persons). The actual risk of developing symptomatic disease is high in presence of coagulopathy. Middle aged women who are exposed to a high estrogen state (during pregnancy or are on oral contraceptives) are commonly described in the literature. Patients with MTS are thought to progress through distinct clinical stages. Stage 1, asymptomatic compression; stage 2, venous spur development (fibrous lesions within the vein from chronic compression); and stage 3 development of deep vein thrombosis (DVT). Typical history includes recurrent left lower extremity DVT, edema, varicose veins, venous claudication, and venous ulcerations. Diagnostic modalities include venous ultrasonography, CT with contrast, MRV, intravenous ultrasound, and contrast venography. Though there are no accepted diagnostic criteria for MTS, contrast venography is considered the gold standard to confirm the diagnosis. High pressure gradient across the stenosis and presence of collaterals are common findings in MTS. Interventional thrombolysis, thrombectomy and stenting are the standard of care for patients presenting with acute DVT in the setting of MTS with overall patency of the iliac vein reported to be 86%. The classic demographic for MTS is a middle aged female with high estrogenic state. Our patient represents a rare presentation in a man with initial atypical presentation of flank pain.</p> <p>Conclusion: MTS is less commonly associated male gender, it is essential to be aware of this anatomic variant in all patients who presents with diffuse left lower extremity pain and swelling.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	NICHOLAS CALDER	Pratik Shah, MD; Satyajeet Roy, MD, FACP	Cooper University Hospital (Brian Gable)	Association of Sweet syndrome and granulomatosis with polyangiitis	<p>Introduction: Sweet syndrome presents a clinical challenge in establishing its association with a specific underlying malignancy or infection, and rarely an autoimmune etiology.</p> <p>Case: A 50-year-old female with SLE, COPD and hypertension presented with rapidly worsening diffuse skin lesions, arthralgias, myalgias, abdominal pain and weight loss for 2 weeks. Prior to her presentation she had hematuria, epistaxis and cough with occasional hemoptysis. She denied fever, chills, nausea, diarrhea, flank pain, recent travel or sick contacts. Her past managements included hydroxychloroquine, mycophenolate mofetil and corticosteroids. Her temperature was 99 degrees F, heart rate 102/minute, blood pressure 174/104 mmHg, and room-air oxygen saturation 92%. Her skin lesions were heterogeneous and involved various erythematous, edematous papules and papulovesicles in arms, legs, face, and hands bilaterally along with periungual telangiectasias. The rest of her physical exam was unremarkable. Diagnostic studies showed serum creatinine 4.19 mg/dL, positive ANCA, MPO Ab and proteinase 3 Ab; small kidneys with generalized atrophy and increased echogenicity on renal ultrasonography, and small ground-glass opacities in upper lung fields on high-resolution CT. She had negative or normal ANA, Sm/RNP, anti-DNA, anti-GBM Antibody, HIV, hepatitis B and C, C3 and C4 complement levels. Renal biopsy showed crescentic change consistent with pauci-immune etiology. Skin biopsy of lesions from her left forearm and from her left thigh showed neutrophilic dermatosis consistent with Sweet's syndrome.</p> <p>Discussion: Sweet's syndrome is a cutaneous disorder characterized by erythematous, usually painful plaques that are accompanied by diffuse systemic symptoms. The diagnostic criteria includes 2 major criteria: (1) the abrupt onset of tender or painful erythematous or violaceous plaques or nodules, and (2) a predominantly neutrophilic infiltrate in the dermis, without leukocytoclastic vasculitis. Both major criteria should be met for consideration of Sweet syndrome. At least 2 of the 4 following minor criteria should also be met: (1) fever or infection preceding the eruption; (2) concurrent fever, arthralgia, conjunctivitis, or underlying malignancy; (3) leukocytosis; and (4) response to systemic steroid therapy but not to antibiotic therapy. Classical Sweet's syndrome occurs more commonly in women and is often associated with an upper respiratory tract or gastrointestinal infection, inflammatory bowel disease or pregnancy. Malignancy associated Sweet's syndrome typically arises in the setting of haematological malignancies. A study of 50 patients with Sweet's syndrome and autoimmune disorders found only one patient with granulomatosis with polyangiitis (GPA). Our patient was diagnosed with Sweet's syndrome and GPA. The patient progressed to kidney failure but the skin lesion resolved after a pulse dose course of methylprednisolone-sodium-succinate followed by cyclophosphamide.</p> <p>Conclusion: In the setting of Sweet's syndrome the work-up should not only include associated malignancy and infection, but other autoimmune etiology as well, such as the rare association GPA. Early detection offers a favorable prognosis.</p>

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Clinical Vignette	Kennedy Ukadike, MD, MS	Brittany Carter, MD; Yelena Galumyan, MD	Englewood Hospital And Medical Center (Jon Shammash)	A rare fistulating cardiac anomaly with atrial fibrillation and heart failure	<p>Case Description: A 63-year-old female with no PMH or FH of heart disease presented to the ER complaining of dyspnea and pressure-like central chest pain. She reported a 3-week history of cough, sore throat and rhinorrhea. On evaluation, her vital signs were T 97.8°F, BP 143/85 mmHg, HR 150/min, RR 20/min and SpO2 97%. Physical exam was significant for tachycardia with irregularly irregular pulse and trace bilateral lower extremity pitting edema. Initial diagnostic investigation was notable for elevated BNP 317 pg/mL, normal Troponin, CKMB, TFT, BMP, CBC and CXR. ECG revealed AFib with RVR at 152/min. She was admitted and started on diltiazem and heparin infusion given a CHA2DS2-VASc score of 2. TTE and TEE both revealed dilated chambers with an estimated LVEF of 25% and no evidence of intracardiac thrombus. Electrical cardioversion was successful at achieving normal sinus rhythm, and she was given a short course of amiodarone infusion with subsequent addition of metoprolol, digoxin and furosemide. Cardiac catheterization revealed a left diagonal artery to LV fistula and otherwise normal coronary arteries in addition to the echocardiographic findings. Cardiac MRI revealed cardiomegaly with global hypokinesia and an LVEF of 21.5% without evidence of ischemic disease, valvulopathy or cardiomyopathy. Given an increased risk of sudden cardiac death, she underwent placement of dual chamber ICD/pacemaker. She remained mostly in normal sinus rhythm and was eventually discharged home on apixaban, digoxin, metoprolol and furosemide with ACEI deferred due to hypotension.</p> <p>Discussion/Conclusion: This case illustrates a previously undiagnosed coronary artery fistula identified in the setting of AFib and heart failure. Coronary artery fistula is an abnormal connection between one of the coronary arteries and a cardiac chamber or great vessel. This rare congenital cardiac anomaly has an estimated incidence of 0.2-0.4% found incidentally on diagnostic cardiac catheterizations. Most reported fistulae originate from the RCA and insert into the RA, RV or PA with no significant effects on cardiac function. Few originate from the LAD or LCX but insertion into the LV as seen in this case is extremely rare. When hemodynamically significant fistulae are present, their effects depend upon their sizes and locations, resulting in clinical symptoms in ~19% of patients <20 years old and ~63% of those >20 years old. Some reported clinical sequelae include rhythm abnormalities, cardiomyopathy or heart failure, chronic myocardial ischemia or angina, and fistula thrombosis or aneurysm. The incidence of these reported clinical sequelae is unknown and warrants further investigation. In the case presented here, it is plausible that the coronary artery fistula was part of a spectrum of congenital cardiac anomalies that predisposed the patient to AFib and heart failure which had been compensated for a long time until unmasked by a recent viral illness.</p>

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Clinical Research	Alice Chedid	Prabin Sharma, Rodrigo Aguilar, Armenia Mordan, Joy-Ann Tabanor, Alex Montero, Jon Shammash, Ping Li, Mark Abi Nader	Englewood Hospital And Medical Center (Jon Shammash)	Epidemiology and predictors of Granulomatosis with Polyangiitis in Inflammatory Bowel Disease	<p>Objectives: Concurrent Granulomatosis with Polyangiitis (GPA) and IBD is exceedingly rare, and the association between the two is poorly understood. The aim of this study is to identify factors associated with concomitant Wegener and IBD.</p> <p>Methods: Patients hospitalized from 2004 to 2012 were identified using the Nationwide Inpatient Sample and ICD 9 codes for IBD (555.0, 555.1, 555.2, 555.9, 556.0, 556.1, 556.3, 556.4, 556.5, 556.6, 556.8, 556.9) and Wegener (446.4). IBD patients with Wegener were compared with those without Wegener. The Pearson's X2 test and paired t-test were used for categorical and continuous variables respectively. Statistical analysis was performed using SAS version 9.3 (SAS Institute, Cary, North Carolina).</p> <p>Results: Between 2004 and 2012, we identified 268,170 patients with Crohn's disease (CD) and 152,804 patients with ulcerative colitis (UC) admitted to the hospital. Among the CD and UC groups, we further identified 66 and 58 patients with Wegener respectively. Compared to UC patients without Wegener, the UC-Wegener group was significantly associated with male gender (58.73% vs 41.27%, p 0.04), renal osteodystrophy (1.59% vs 0.12%, p 0.0006), acute diastolic CHF (1.59% vs 0.15%, p 0.0027), secondary hyperparathyroidism (1.59% vs 0.05%, p 0.0027), and autoimmune hepatitis (1.59% vs 0.19%, p 0.01). Similarly, the CD-Wegener group compared to CD without Wegener was significantly associated with anemia of chronic disease (9.09% vs 3.37%, p 0.01), COPD (10.61% vs 3.18%, p 0.001), HIV (1.52% vs 0.18%, p 0.01), hypothyroidism (16.67% vs 7.8%, p 0.007), transaminitis (4.55% vs 0.35%, p< 0.001), and sarcoidosis (1.52% vs 0.23%, p 0.03). Smoking (4.55% vs 18.39%, p 0.004) had a significantly lower association. In both UC and CD groups, mortality difference remained unchanged compared to those affected with GPA.</p> <p>Conclusion: GPA should be considered in the differential of IBD patients presenting with Acute Kidney Injury and having some of the associations described above.</p>

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Clinical Vignette	Raghav Chaudhary	Joseph Shatzkes, MD ; Ritu Kathuria, MD;Hisham Hakeem,MD	Englewood Hospital And Medical Center (Jon Shammash)	Idiopathic Epicardial Ventricular Tachycardia (VT)	<p>Case: 80 year old male with no significant cardiac history presented with worsening dyspnea (NYHA I to NYHA III) over a period of 3 months with episodic dizziness. He underwent Holter monitoring, which showed 7% episodes of monomorphic nonsustained VT coinciding with symptoms. Echocardiogram showed preserved left and right ventricular systolic function. Stress testing to rule out ischemia revealed a possible of reversible posterior basal ischemia but cardiac catheterization was negative for obstructive coronary artery disease. His symptoms did not improve with long acting beta blockers. On a follow up visit, he was again noted to have long runs of slow NSVT and admitted for initiation of medical cardioversion with amiodarone which was discontinued due to prolonged QTc interval. On close inspection of EKG by electrophysiologists, it was determined that the source of his VT was epicardial as against the more commonly seen endocardial focus. Cardiac MRI was negative for structural abnormalities or scars. Patient opted for a trial of medical therapy with sotalol instead of radiofrequency ablation.</p> <p>Discussion: Majority of VTs of chronic ischemic disease are subendocardial in origin, though up to 12-17% of VTs can originate from an epicardial focus. Prevalence can be as high as 25-35% in dilated nonischemic cardiomyopathy and 41-53% in arrhythmogenic right ventricular cardiomyopathy. Most remain idiopathic and manifest as a pattern mimicking RBBB with left axis deviation. It can be identified on an EKG, even though no single finding is considered gold standard. When initial activation starts at an epicardial site, there is an intramyocardial delay in the activation of the His Purkinje system which is reflected as initial slurring of QRS complex and is referred to as the “pseudo delta” wave. A pseudo delta wave of >34 msec and an intrinsicoid deflection time >85 msec in lead V2 has >90% specificity in predicting epicardial origin of VT. Other parameters include a maximum deflection index (MDI) >0.55msec, Q waves in lead 1, shortest RS complex duration >121 msec. Theoretically, there may be a difference in effectiveness of antiarrhythmic pharmacological therapy as there are differences in action potential characteristics between endo and epicardium, but no difference in efficacy has been found in studies. Epicardial radiofrequency ablation is the treatment of choice and is deemed immediately successful in 76% of patients, with about a quarter having recurrent VT by end of a year.</p> <p>Conclusion: Epicardial VTs are more common than thought, tend to be idiopathic and are mostly not a risk factor for sudden cardiac death. Consider looking for it on EKG in a patient with no underlying ischemic disease or with a failed endocardial radiofrequency ablation.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Quality Improvement/Patient Safety	Hanaa Benchekroun Belabbes	Namrata Sekhon, Layal Esper, Johnthan shammash, Karlen Williams	Englewood Hospital And Medical Center (Jon Shammash)	Impact of follow up on diabetes control	<p>Background: Diabetes is a leading cause of disability and death in the United States, affecting an estimated 17 million people. Patients with HbA1c values >9% are at highest risk of morbidity and mortality. (For every 1% reduction in A1c, the risk of developing eye, kidney and nerve disease is reduced by 40% while the risk of heart attack is reduced by 14%.)</p> <p>Purpose: Our aim statement was to increase the percentage of diabetics with a HbA1C < to 9% from 86% to 90% over a 6 month period (1/17/16 to 7/17/16) at a resident-staffed Internal Medicine clinic in Northern New Jersey.</p> <p>Methods: From April 2015 to September 2015, we collected data on a cohort of patients (Group 1) with HbA1c >8%, and analyzed factors presenting impediments to diabetes control. Analysis showed that patient follow up was the most modifiable variable. We devised an EMR-based intervention which included identifying a diabetic coordinator and sending messages to her through an order-based action message. During the post intervention period, from January to July 2016 residents and other clinic providers sent an action telephone encounter to the diabetes coordinator for each patient with a Hba1c >9%. The diabetic coordinator called patients 1 week before their appointments, utilizing a script to encourage the patients to attend their appointment, bring necessary information, and reinforce the treatment plan.</p> <p>Results: In Group 1, 18% of patients had follow up appointments scheduled, 53% had appropriate medication changes for poorly controlled DM2, 26% of these patients were establishing new care, and 26% were provided diabetic educator referrals. Follow up of Group 1 after intervention showed the following: 83% of these patients had scheduled appointments, 75% had medication changes performed, 72% were provided diabetic educator referrals. 69% of these patients had a significant improvement in HbA1C (with reduction or 1% or more), and another 39% experienced reduction in HbA1c below 9%. Analysis of diabetics with HbA1c >9% from January to July of 2016 (Group 2) revealed that 81% had appointments scheduled, 63% had medication changes performed, 48% were provided diabetic educator referrals, and 47% were new patients. The percentage of diabetics with HbA1c <9% was 86% in Group 1, and increased to 88% in Group 2.</p> <p>Discussion: Application of the EMR-based action resulted in improved patient follow up appointments, diabetic referrals, and medication changes. Limitations of the study include a small sample size, different practice patterns for the non-resident providers who did not utilize the action order, and a significant number of new uncontrolled diabetics in each group.</p> <p>Conclusion: Interventions which increase diabetics' follow up can improve glycemic control.</p>

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Clinical Research	Joy-Ann Tabanor	Namrata Sekhon, MD, Sahai Donaldson MBBS, Jonathan Shammash, MD	Englewood Hospital And Medical Center (Jon Shammash)	OSTEOPOROSIS SCREENING AT A RESIDENT-STAFFED INTERNAL MEDICINE CLINIC	<p>Introduction: Only 52% of eligible women get bone mineral density screening based on CMS data for DXA scan reimbursements. Rates as low as 31% have been previously reported. These low screening rates are concerning given the relatively high incidence of osteoporotic fractures, the associated morbidity, mortality and health-care costs. The aims of our study are to determine the osteoporosis screening rate at an internal medicine clinic at a Federally-qualified Health Center in northern New Jersey and to explore the potential barriers to screening.</p> <p>Methods: Based on current screening guidelines we formed two risk group categories: women 65 years and older and patients with risk factors for secondary osteoporosis. We conducted surveys of residents, recent graduates and clinic preceptors. The percentage of respondents that reported screening 5 or more out of ten patients in each risk group category was determined. This threshold was chosen because it is closely related to the reported 52% national screening rate. The mean screening rates for respondents was also determined. Chart reviews were performed for patient visits from October to December, 2015.</p> <p>Results: 13% of respondents were PGY1, 44% were PGY2, 30% were PGY3, 4% were recent graduates and 9% were attendings with 10 years or more of experience. 95% thought that it is important to screen for osteoporosis. 42% of respondents reported screening 5 or more out of 10 women 65 years and older for osteoporosis. The mean reported screening rate was 44%. The most commonly reported barriers to osteoporosis screening in this group were: not remembering to screen (65%), other screening tests taking priority (55%), no reminders in the EMR (50%) and not having the time (30%). 32% of respondents reported screening 5 or more out of 10 of patients with risk factors for secondary osteoporosis. The mean reported screening rate was 37%. The barriers reported in this group were not remembering to screen (75%), other screening tests taking priority (35%), no reminders in the EMR, not knowing to screen that population of patients (30%) and not having the time (25%). From the chart reviews, the overall screening rate was 31% (35% for the resident-staffed clinic and 21% for other providers). Only 46% of ordered DXA scans were done.</p> <p>Conclusion: Low osteoporosis screening rates nationally and at our resident-staffed internal medicine clinic are concerning. Possible interventions include systems-based measures, provider and patient education.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Chaitali Patel	Chinwe Ogedegbe, Joseph Degregorio	Humc Mountainside (Douglas Zaeh MD)	Recurrent spontaneous coronary dissection in a post-partum young patient	<p>Spontaneous coronary artery dissection (SCAD) is relatively rare and unexplored type of coronary artery disease. It has been increasingly acknowledged to be an important cause of acute coronary syndrome in women, especially in the peri-partum period. Revascularization in patients with SCAD is technically challenging in part due to fragility of the vessel wall and associated with higher failure rates or complications. In percutaneous coronary intervention (PCI) for SCAD, instrumentation (wiring, angioplasty, or stenting) can propagate dissection and occlude side branches.</p> <p>A 35-year-old female presented to the emergency department with complaints of sudden onset left sided chest pain, radiating to the left shoulder, accompanied by shortness of breath, diaphoresis, nausea and vomiting. She was six days post-partum (assisted vaginal vacuum delivery) and breast feeding with no other obstetric complications. She was diagnosed with acute ST-segment elevation myocardial infarction, and immediately taken to a cath lab. Coronary angiography showed coronary artery dissection and narrowing in the proximal and mid segment of the left anterior descending (LAD) artery and the first diagonal branch (D1). A stent was deployed across the ostium of D1 in the mid and proximal LAD with TIMI 3 flow. Troponin I, creatine kinase, and creatine kinase-MB were elevated up to 27.84 ng/mL, 1185 IU/L, and 109.2 ng/mL, respectively, with the constant chest pain subsequently for the next three days. She was taken to the cath lab on the 2nd day and repeat coronary angiography showed patent stents without any new dissection. Echocardiography consistently showed an ejection fraction (EF) of 35%. Post procedure the patient was started on aspirin, clopidogrel, beta blocker, and statin. The patient was discharged home on the 6th day with a wearable defibrillator due to the low EF. She presented with chest pain 40 days after her first PCI and was brought to a cath lab. Coronary angiography showed extension of the dissection of the mid LAD and the mid D1 with TIMI 3 flow. Thereafter, due to expansion of the intramural hematoma occurring distally to the stent in the mid LAD, another stent was deployed. Troponin I was slightly elevated. Echocardiography findings were consistent with anterior septal hypokinesia with EF of 45%. On the 4th day, repeat coronary angiography showed no coronary dissection. She was discharged and patient came back 3 days later for exertional chest pain. She was taken for an elective diagnostic cath and wearable defibrillator was discontinued on this visit. Patient underwent a total of 5 catheterizations in a span of 3 months.</p> <p>SCAD is relatively rare, however more common in women, especially peri-partum. Although conservative management may be optimal for stable patients, those with symptoms of ongoing ischemia or hemodynamic compromise should be considered for emergent catheterization and revascularization.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Chaitali Patel	Chinwe Ogedegbe	Humc Mountainside (Douglas Zaeh MD)	Death of a patient awaiting ECHO	<p>Aneurysms and dissections of aorta are associated with high degree of morbidity and mortality despite continued improvements in diagnostic and surgical techniques. Sinus of Valsalva aneurysms (SVAs) are rare anomalies, most often caused by the congenital absence of muscular and elastic tissue in the aortic wall behind the sinus of Valsalva. The diagnosis is usually found accidentally, and surgical repair provides a long lasting resolution. Open-heart surgery with or without aortic valve replacement can be an optimal intervention with transcatheter device closure of a ruptured SVA.</p> <p>A 45 year old female was brought to the hospital with complaints of feeling dizzy, burning in the chest and flushing, starting the night before presentation. Patient had taken antacid and ibuprofen without any relief last night, and experienced a “blackout” on the morning of presentation. Her past medical history was significant for hypertension, obesity and vertigo. Patient had stopped taking her antihypertensives for at least 6 months as she “did not like” the medications. She denied any past surgical history, allergies, history of smoking, alcohol abuse or family history of any cardiac disease or early death. The patient had a normal physical examination, but widely ranging blood pressure (120/50-180/110) in the observation unit. An Electrocardiogram during admission, showed normal sinus rhythm with left ventricular hypertrophy (LVH) and lateral T-wave inversion, troponin 0.01 at 0 hours and 0.11 after 6 hours. Chest x-ray showed enlarged heart border and central prominence of bronchovascular markings. An echocardiogram was ordered, and while waiting on the stretcher for the echo, she complained of headache and stated "I am getting that feeling again." Immediately the patient seized for approximately 30 seconds with urinary incontinence and was unresponsive thereafter. A code blue was called and patient received 6 doses of epinephrine, 2 ampules of bicarbonate and 2mg of Atropine. Patient went into asystole after 20 minutes of resuscitation. The code was unsuccessful and cardiopulmonary resuscitation was terminated. Autopsy revealed a ruptured aortic aneurysm in the root of the ascending aorta (Sinus of Valsalva), measuring 1.7 cms in maximum extent with dissection extending into the pericardium with secondary hemopericardium and surrounding soft tissue on left side close to pulmonary hilum. There was no evidence of organization or lamination of clot showing that it was fresh blood and was an ante-mortem event, reflecting the acuteness of the event. SV aneurysms may rarely dissect through the interventricular septum and rupture into left ventricular cavity, resulting in a presentation clinically indistinguishable from aortic regurgitation (AR). This case serves to highlight the importance of early identification and treatment of a ruptured SVA and to keep it on our list of differentials is essential as, not making timely diagnosis may lead to fatal outcome.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract																																			
Clinical Research	Chaitali Patel	Chinwe Ogedegbe	Humc Mountainside (Douglas Zaeh MD)	Does low risk chest pain, utilizing the HEART Score for earlier discharge in the ED, have a decreased 30 day readmission rate?	<p>INTRODUCTION: Over the years the number of patients presenting with chest pain to the Emergency departments (EDs) throughout the US has increased, creating a rising health care concern that needs to be addressed. The objective of this retrospective case-control study is to focus on the low risk chest pain population who may have benefited from an early discharge, and still have decreased hospital readmission.</p> <p>We utilized the HEART Score, which incorporates 5 elements to risk stratify: History, Electrocardiogram (ECG), Age, Risk factors, and Troponin. Patients were sorted into groups of, High (Score of 7-10), Intermediate (Score of 4-6) and Low risk (Score of 0-3), The primary end point of this study is a composite of acute myocardial infarction, percutaneous coronary intervention, coronary artery bypass graft surgery and death, together called the Major Adverse Cardiac Event (MACE) and the secondary end point being 30-day readmission rate.</p> <p>This is a retrospective, case-control study, taking place in a tertiary care community teaching hospital, enrolling patients who came in between 01/01/2013 to 01/31/2013 to the adult Emergency Department.</p> <p>METHODS & MATERIALS</p> <table> <tbody> <tr> <td rowspan="3">HISTORY</td> <td>Highly Suspicious</td> <td>2</td> </tr> <tr> <td>Moderately Suspicious</td> <td>1</td> </tr> <tr> <td>Slightly or non-suspicious</td> <td>0</td> </tr> <tr> <td rowspan="3">ECG</td> <td>Significant ST depression</td> <td>2</td> </tr> <tr> <td>Non-specific repolarization</td> <td>1</td> </tr> <tr> <td>Normal</td> <td>0</td> </tr> <tr> <td rowspan="3">AGE</td> <td>>65 years</td> <td>2</td> </tr> <tr> <td>>45 to <65 years</td> <td>1</td> </tr> <tr> <td><45</td> <td>0</td> </tr> <tr> <td rowspan="3">Risk Factors</td> <td>>3 risk factor</td> <td>2</td> </tr> <tr> <td>1 or 2 risk factor</td> <td>1</td> </tr> <tr> <td>No risk factor</td> <td>0</td> </tr> <tr> <td rowspan="3">Troponin</td> <td>>3 times Normal limit</td> <td>2</td> </tr> <tr> <td>>1- <3 normal limit</td> <td>1</td> </tr> <tr> <td>Normal limit</td> <td>0</td> </tr> </tbody> </table> <p>RESULTS</p> <p>During the study period, a total of 160 patients with chest pain presented to the ED. The main reason for admission was chest pain which included cardiac as well as non-cardiac causes. Amongst the excluded cases were twenty four (15%) cases who were admitted for non-cardiac reasons, twenty four (15%) cases had STEMI, known CAD, CHF, previous MI/stent placement and six (3.8%) were patients who were</p>	HISTORY	Highly Suspicious	2	Moderately Suspicious	1	Slightly or non-suspicious	0	ECG	Significant ST depression	2	Non-specific repolarization	1	Normal	0	AGE	>65 years	2	>45 to <65 years	1	<45	0	Risk Factors	>3 risk factor	2	1 or 2 risk factor	1	No risk factor	0	Troponin	>3 times Normal limit	2	>1- <3 normal limit	1	Normal limit	0
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				<p>pregnant, deceased, transferred to other hospital, left AMA or had missing ECG/Troponin on their chart. The moderate and high risk patient were admitted for clinical observation, and focus was for the low risk group. The low risk group had sixty three patients, out of which, two (3.2%) had non-cardiac related readmission and one (1.6%) had a cardiac related readmission. The cardiac related admission was not described as MACE and the patient underwent objective cardiac testing.</p> <p>CONCLUSION: The HEART Score provides a useful tool in making accurate disposition decisions in the ED in low risk patients with chest pain who could be safely discharged with low likelihood of ACS and 30 day readmission.</p>
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Category	Name	Additional Authors	Program	Abstract Title	Abstract
Quality Improvement/Patient Safety	Ankita Patel	Valerie Allusson, Sunya Ashraf, Ashley Jenkins	Humc Mountainside (Douglas Zaeh MD)	Can Highly Reliable Rounding Reduce Inappropriate Telemetry?	<p>Telemetry services are over utilized and can be harmful to patients that do not meet American College of Cardiology (ACC) guidelines. Using ACC telemetry admission guidelines, Benjamin et al. (2013) observed that 35% of telemetry days were classified as inappropriate, and not clinically indicated. Moreover, Ivonye et al. (2010) concluded that with the inappropriate use of telemetry services, “there is also the potential for harm to patients if artificial telemetry findings in low-risk patients lead to the performance of inappropriate invasive procedures.” The inappropriate use of telemetry is associated with excessive medical expenditures, unnecessary resource utilization and potential harm to patients.</p> <p>Highly Reliable Rounding (HRR) involves sharing real time information with all care providers. Unique to this approach, is the active involvement of the patient who is viewed in this model as a partner in the center. Activities performed by the HRR model include performing quality and safety checks, engaging the patient and the team in discharge planning and assuring a safe transition of care at discharge.</p> <p>The study conducted a retrospective chart review to determine whether the HRR paradigm impacted telemetry discontinuation rates prior to day of discharge. The control group consisted of patients on telemetry between July 2014 and August 2014, time prior to the implementation of HRR at the hospital. Patients after the implementation of HRR were grouped into non HRR on telemetry or HRR on telemetry. The months of July 2015 and August 2015 were reviewed. A chi square analysis was done on the control group and the HRR on telemetry group to see if there was a statically significant difference between the two groups.</p> <p>In the control group 46 out of 517 patients (8.9%) were discontinued from telemetry prior to the day of discharge. In the non HRR on telemetry group 34 out of 319 patients (10.7%) were discontinued. In the HRR on telemetry group 42 out of 115 patients (36.5%) were discontinued. The differences between the groups was statistically significant (p<.05) using chi square analysis.</p> <p>Patients in the HRR on telemetry group, who had telemetry discontinued were reviewed for 30 day readmission. There were 7 patients readmitted, of which 4 were not placed on telemetry. The 3 patients placed on telemetry were admitted for non-cardiac complaints.</p> <p>HRR can increase the number of patients discontinued from telemetry monitoring prior to the day of discharge, who no longer meet ACC guidelines for monitoring. The study found a statistically significant difference in telemetry discontinual rate between patients who had HRR versus patients who did not. Patients who were discontinued from telemetry on HRR did not have any cardiac adverse effects. Highly Reliable Rounding significantly improves quality patient care by decreasing unnecessary costs due to inappropriate telemetry monitoring.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Luis Dominguez	Marian D Valentin, MD.	Jersey City Medical Center (Amer Syed)	Macrophage Activation Syndrome: A nearly fatal case presentation, and review of diagnosis and treatment guidelines.	<p>A 33 year-old Hispanic male without significant history presented with an unremitting fever for 5 days, associated with myalgias, fatigue, night sweats, and a rash. Upon admission, he was tachycardic, hypotensive and febrile at 103F. Physical exam revealed soft palate red papules, a diffuse non-blanching, maculo-papular rash, sparing the face, palms and soles. Labs revealed pancytopenia, and elevated ferritin, alongside transaminitis, and low fibrinogen. Imaging showed hepatosplenomegaly. He was started on empiric antibiotics. Blood cultures were drawn and returned negative. Despite antibiotics, he continued to spike fevers. On the 4th day, he suffered hypoxemic respiratory failure, was found to be in ARDS and was intubated and transferred to the ICU. He continued to deteriorate. Serum titers for viral and atypical bacterial antibodies, along with a bone marrow biopsy and lumbar puncture were inconclusive.</p> <p>Given the clinical picture of his persistent fever, pancytopenia, hepatosplenomegaly with transaminitis, hypofibrinogenemia and hyperferritinemia, Macrophage Activation Syndrome was considered based on recent classification criteria. He was started on high-dose methylprednisolone. He rapidly recovered, was extubated within 24 hours and discharged later that week.</p> <p>Macrophage Activation Syndrome (MAS) is a rare but life-threatening systemic inflammatory disorder, most commonly arising as a complication from juvenile idiopathic arthritis, but also associated with other rheumatological conditions, malignancies, immunological deficiencies and infections – especially viral. MAS is characterized by an uncontrolled and dysfunctional immune response, involving the expansion of T-Cells, Macrophages and the hypersecretion of pro-inflammatory cytokines. This results in fever, cytopenia, hepato-splenic involvement and hyperferritinemia.</p> <p>Previous diagnostic criteria, focusing on hemophagocytic macrophages found on biopsy lacked sufficient sensitivity. More recent criteria, by Filipovich in 2009, and now an international expert panel just released new classification in 2016. The 2016 criteria offers greater sensitivity and specificity, especially laboratory guidelines.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Sujata Panthi	Acharya S, Mehran P, Matta J	Jersey City Medical Center (Amer Syed)	A rare case of marantic endocarditis causing recurrent stroke: Case report	<p>Background: Nonbacterial thrombotic endocarditis (NBTE) is a rare condition, often referred as marantic endocarditis when associated with cancer. While many patients with NBTE are often asymptomatic, constitutional symptoms like fever or focal symptoms due to systemic embolism are found in up to half of the patients. Here we present a case of recurrent ischemic stroke caused by NBTE associated with cancer.</p> <p>Case presentation: A 71 year old woman with history of non-small cell lung adenocarcinoma stage IIIB on chemotherapy and radiation presented to the emergency department with altered level of conscious. Physical examination revealed lethargic and disoriented patient with apical systolic murmur. Trans thoracic echo-cardiogram showed thickened mitral valves with mild to moderate mitral regurgitation. Transesophageal echocardiogram revealed a moderate sized vegetation on the anterior mitral leaflet. During the admission, the patient remained afebrile and blood cultures were negative. The patient had no predisposing heart condition and no IV drug abuse history. Tele-monitoring did not show atrial fibrillation. Magnetic resonance imaging (MRI) brain showed acute large right temporal and parietal infarcts and small left frontal and cerebellar infarcts. Patient could not be anticoagulated due to thrombocytopenia. Patient was readmitted within 2 weeks of discharge due to additional stroke in left frontal areas. The diagnosis of non-bacterial endocarditis and cerebral infarcts due to embolism from the endocarditis was the most likely diagnosis in this clinical setting.</p> <p>Conclusions: Cardioembolic stroke in a cancer patient could be explained by NBTE when other common source of embolic stroke like atrial fibrillation, carotid stenosis, artery dissection and mural thrombus are ruled out. NBTE and embolic phenomenon could be related to hypercoagulable state associated with malignancy. Anticoagulation is routinely performed when appropriate. Thrombocytopenia might be common in the patients undergoing chemotherapy and pose a major contraindication to anti-coagulation hence rendering the management of NBTE and stroke very challenging.</p>

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Clinical Vignette	Muhammad Khakwani	Rehman, Uzair, MD Rohit, Kumar, MD Onwochei, Francis, MD Acharya, Saurav, MD	Jersey City Medical Center (Amer Syed)	Type B Lactic Acidosis with Albuterol use	<p>Introduction: Lactic acidosis is often used as a marker for tissue hypoperfusion and hypoxia in the setting of sepsis and shock. It can also be seen in the setting of β-2 agonist therapy. Mechanism of lactic acidosis with β-2 agonist use is poorly understood however, with increasing use of the treatment physicians must keep in mind other etiologies of lactic acidosis, to avoid unnecessary interventions.</p> <p>Case: 61 y/o M with past medical history of COPD, HTN, DM, HIV, Hepatitis C, presented for COPD exacerbation. He was found to have an increased anion gap and lactic acid of 4.4. Patient received 5 doses of albuterol every hour for total of 5 doses. Patient improved symptomatically however his Lactic acid increased to 7.3 at 7 hours and 4.6 at 19 hours. After extensive work up all causes of lactic acidosis including sepsis, drug induced, liver dysfunction, diabetes, salicylates and carbon monoxide poisoning were ruled out. Albuterol was discontinued and substituted with ipratropium. Ten hours after discontinuing albuterol treatment lactic acid levels trended down to 1.5, confirming albuterol as the cause of lactic acidosis.</p> <p>Discussion: Albuterol use stimulates β-2 receptor, activating glycogenolysis and lipolysis pathways leading to increased production of pyruvate. Excess pyruvate is shunted through the anaerobic cycle producing lactic acid. Furthermore, lipolysis produces fatty acids which inhibits pyruvate dehydrogenase leading to a buildup of pyruvate. Pyruvate is then converted to lactic acid. Another mechanism that may contribute lactic acidosis could be excessive respiratory muscle activity and muscle fatigue that can add to accumulation of lactate in the setting of acute COPD exacerbation and severe asthma.</p> <p>Conclusion: In our case, excessive respiratory muscle activity or hypoxia may have contributed to the elevated lactic acid on admission. Elevated lactic acid seen at 7 hours and then at 19 hours were likely due to β-2 agonist therapy as lactic acid improved after discontinuation of the treatment. In patients with elevated lactic acid and in the absence of tissue hypoperfusion, other etiologies should be considered to avoid unnecessary and even harmful medical treatments.</p>

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Clinical Vignette	Muhammad Khakwani	Rehman, Uzair,MD Rohit, Kumar,MD Onwochei, Francis,MD Gongireddy,Srinivas,MD	Jersey City Medical Center (Amer Syed)	Curious case of anemia associated with Squamous cell carcinoma of forearm	<p>Introduction: Non Melanoma skin cancers such as Basal cell carcinoma (80%) and squamous cell carcinoma (20%) are the most common cause of cancers in U.S. and most common malignancy amongst the white population. Non melanoma skin cancers appear most commonly on sun exposed areas. If left untreated these tumors can be aggressive, metastasize and invade underlying tissue requiring extensive surgeries. We present a case of atypical location of Squamous cell carcinoma associated with severe anemia.</p> <p>Case: 66 year old male with no significant past medical history, presented with progressive shortness of breath for the past three months. Shortness of breath was more pronounced with exertion, associated with dizziness and generalized weakness. Patient also mentioned a growth on his Left forearm which started about 5 years ago. Initially, patient visited a dermatologist who prescribed a topical ointment but growth progressively increased in size over time. Patient notes bleeding at the site of the growth when he takes a shower and occasional burning sensation. Patient was hemodynamically stable. On physical examination, a large mass 9.5cm x 8cm in size, foul smelling, and fungating in appearance was seen on the left forearm. Laboratory findings were significant for a hemoglobin level of 4.1. Patient was transfused 6 units PRBC. Patient underwent colonoscopy which was unremarkable for any source of bleeding. Shortness of breath improved markedly after transfusion. On MRI, the mass had spread to underlying muscles. Biopsy of the mass was sampled and reported as moderately differentiated invasive squamous cell carcinoma with a 2.2cm x 1.5cm lymph node in left axilla positive for metastasis. After, a multidisciplinary team discussion decision was made to amputate the left forearm.</p> <p>Discussion: Squamous cell carcinoma has multiple risk factors, most common being UVB light exposure. UVB can inactivate or delete p53 genes leading to metaplasia. Most common sites of sunlight exposure are head and neck. Tumor starts from a precursor lesion known as actinic keratosis. These are scaly, pinkish or brown colored lesions and have cumulative lifetime progression risk of 6-10%. In squamous cell carcinoma, mass greater than 2cm if untreated can lead to invasive disease and thus requiring amputation.</p> <p>Conclusion: There are over 600,000 new cases yearly from non-melanoma type cancer that cause significant disfigurement and morbidity . Squamous cell carcinoma has an increased risk of metastasis, that increases mortality if not diagnosed early. Most invasive Squamous cell carcinomas occur on the head and neck and 2nd most common site is the trunk. Lesions greater than 2cm recur and metastasize at a rate of 15% to 30% with a 5-year cure rate of 70%.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Jennifer Mouriz	Ang Zhang, Neil Ibrahim, Naved Munir, Anthony Mangia and Mahesh Bhatt	Jersey City Medical Center (Amer Syed)	Atypical presentation of Zika Virus in a young Dominican female	<p>A 27 year old Dominican female without a significant past medical history presented with complaints of a pruritic non-tender, erythematous maculopapular rash of different stages on her torso for five days. The patient had emigrated from the Dominican Republic six days before the rash appeared. The patient noted that the rash started on her abdomen, spread to her back, chest and neck two days after the initial eruption began. On admission, the rash spared her arms, face, legs, buttocks and perineal area. The patient had an associated periodic severe headache, but denied photophobia, neck stiffness, aura, nausea or vomiting. The patient had subjective fevers at home and was found to have a fever of 102.8 during the hospital stay. The patient was in moderate distress secondary to the pruritic nature of the rash and the headache that was described as throbbing and severe. During her hospital course, the rash spread and included her thighs, neck, back, arms and palms of her hands. Pt was given Benadryl and solumedrol for a possible allergic reaction secondary to an oven cleaner that was used the day before the rash appeared. NSAIDS and ASA were used sparingly as they have been associated with hemorrhage during other flavivirus infections. Anti-viral medication was given due to the patient's unknown childhood immunization status while titers were being obtained. Rheumatology as well as infectious disease physicians were consulted. The patient tested negative for ASO, C3, C4, RF, ANA and a skin biopsy showed superficial dermal periadnexal and perivascular chronic inflammation composed of lymphoplasmacytic infiltration with occasional eosinophils. Blood samples were sent to the Department of Health and the Centers for Disease Control and were found to be preliminarily positive for Zika virus. The patient did not develop conjunctivitis, muscle or joint pain and was discharged from the hospital after resolution of the fever and headache.</p> <p>The patient's risk factors for developing Zika virus include a long period of exposure in an endemic area, the Dominican Republic. Patient's sexual history included one heterosexual partner two years prior to admission with protection used during every sexual encounter. The patient was outdoors while in the Dominican Republic, but did not remember specific mosquito, tick or animal bites. The patient did not have any family or friends with similar rashes.</p> <p>The highlights of the clinical presentation of Zika are typical for not only a maculopapular rash, fever and headache, but also conjunctivitis, muscle and joint pain. The findings in this case presented above are atypical for a Zika virus presentation. It is necessary to identify the signs and symptoms that may be absent in an active Zika infection to assure a proper diagnosis is not missed.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Yasmine Elamir	Dr. Laura Riley, Dr. Robert Mcloughlin, Dr. Neil Ibrahim, Dr. Paul Woroch	Jersey City Medical Center (Amer Syed)	Pheochromocytoma Mimicking Sepsis	<p>Patient is a seventy year old Egyptian female with past medical history of coronary artery disease, systolic heart failure, chronic kidney disease stage 3, hiatal hernia, and a benign pheochromocytoma previously worked up at a neighboring hospital, who was admitted for dyspnea and subjective fever intermittently for the past two weeks. Vitals on admission were normal except for a pulse oximetry of 93% and heart rate of 110. Chest x-ray revealed bilateral pulmonary infiltrates. She was treated for community acquired pneumonia with Rocephin and azithromycin. Four days after admission patient had a rapid response for respiratory distress and code sepsis called for fever of 102 degrees, leukocytosis of 23 (from 11.3 on admission), respiratory rate of 30, heart rate of 131, with blood pressure of 183/56 and pulse oximetry of 84%. Patient was intubated and transferred to ICU, where antibiotic coverage was broadened with vancomycin and Zosyn. Pan cultures obtained on admission returned negative and her symptoms resolved two days later. Patient's antibiotics were deescalated and patient was then discharged home. It was thought that her sepsis was likely secondary to pneumonia resulting in acute hypercapnic hypoxic respiratory failure.</p> <p>Patient returned four days later and was readmitted directly to the ICU for worsening dyspnea with a temperature of 102.0, leukocytosis of 27.4, heart rate of 149, respiratory rate of 30, with a blood pressure of 172/90 and pulse oximetry of 83%. Chest x-ray revealed new bilateral infiltrates from previous admission. Patient was intubated and treated with Zosyn, vancomycin, and gentamycin. Cultures again were obtained and again returned negative. At this point it was thought patient's hiatal hernia was leading to cough and aspiration during sleep causing recurrent pneumonia, but this did not explain the negative cultures. It was then thought after researching similar cases that the patient's pheochromocytoma could be leading to a "pseudo" septic picture. Records were obtained from a neighboring hospital that were significant for a PET scan revealing a 2.6x1.9cm left adrenal nodule with increased FDG avidity, elevated serum total metanephrines (676 mcg), urine total metanephrines (2021 mcg), urine normetanephrines (173 mcg). The pathology from biopsy study results were most consistent with pheochromocytoma with neoplastic cells, reactive for chromogranin, synaptophysin and vimentin. It was then determined that patient's septic symptoms were likely due to pheochromocytoma as patient's blood pressure would coincide with febrile episodes and dyspnea. Patient was placed on prazosin and blood pressure normalized during admission without return of fever. Pheochromocytoma should be considered as part of the differential diagnosis in the setting of recurrent dyspnea and sepsis when more common causes have been ruled out. It is also important to do this early as to prevent unnecessary antibiotic use and to prevent antibiotic resistance.</p>

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Clinical Vignette	Waheed Abdul	Dhruv Vasant MD, Mathurika Jeyasingam MD, Abdul A Ameen MD, Mohammad T. Javed MD	Jersey City Medical Center (Amer Syed)	ST alternans and coving simulating Brugada Syndrome induced by Flecainide toxicity: A case report.	<p>Flecainide is a class IC anti-arrhythmic used in treatment of a variety of cardiac arrhythmia including paroxysmal supraventricular tachycardia (atrial fibrillation, AVNRT, WPW) and ventricular tachycardia in patients with normal structured heart. One of the major trials called CAST (Cardiac Arrhythmia Suppression Trial) showed higher mortality when Flecainide was used to suppress ventricular arrhythmia in the setting of acute myocardial infarction or left ventricular dysfunction. Also, Flecainide has a negative inotropic that can worsen or cause heart failure especially in patients with pre-existing NYHA class III or IV.</p> <p>A 55-year-old Caucasian male with history of Atrial fibrillation, s/p cardioversion admitted to CCU for lethargy after ingesting 15 tablets of Flecainide acetate (approx. 2.5 grams). The 1st EKG showed sinus rhythm, HR of 68bpm with widened QRS (140ms), RBBB, and prolong QTc. The next EKGs showed 1st degree AV block, prolongation of QRS and QTc with ST alternans in V1 and V2 leads. Cordis® catheter was inserted in his right interval jugular vein for emergency transvenous pacing. He was treated with continuous IV fluids, Sodium bicarbonate IV drip. Potassium IV was initiated as well to prevent hyperkalemia secondary to metabolic alkalosis. Continuous serial EKGs showed resolving QRS and QTc prolongation and shortly after, he was discharged to home.</p> <p>Flecainide blocks the sodium channels slowing phase 0 leading to prolong depolarization and slows the AV node conduction. These changes will result in the prolongation of PR interval, QRS duration, first or second-degree heart block.</p> <p>Flecainide toxicity causes conduction disturbances with widening of QRS complex, which can lead to ventricular fibrillation, torsade de pointes and asystole due to its narrow therapeutic index. Overdose can be lethal with a mortality rate of up to 22%. There have been few studies on canine heart that show ST alternans correlated with flecainide use. One of the most prominent studies done by Tachibana, et al., showed intracoronary flecainide induces ST alternans and reentrant arrhythmia in canine heart. Advanced studies are required to establish this mechanism of action in humans.</p> <p>Supratherapeutic flecainide levels can unmask brugada type pattern in the absence of underlying channelopathy in our patient. As shown in this patient's EKG, we see QRS broadening, 1st degree AV block and right precordial lead ST alternans. Few recent case reports have been published showing Brugada phenocopy in flecainide toxicity in patients with Atrial fibrillation just like our patient. Brugada Phenocopy is defined as an acquired brugada like ECG pattern findings of ST alternans in the absence of true Brugada syndrome.</p> <p>In conclusion, Flecainide can unmask brugada type pattern showing ST alternans in the absence of underlying channelopathy in patients. Therefore, it is important to be vigilant and to do routine EKG monitoring for patients on Flecainide medication.</p>

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Clinical Vignette	Waheed Abdul	Dhruv Vasant MD, Mathurika Jeyasingam MD, Abdul A Ameen MD, Mohammad T. Javed MD	Jersey City Medical Center (Amer Syed)	T wave alternans preceding Torsades de Pointe induced by a single loading dose of Amiodarone	<p>Amiodarone is a class III antiarrhythmic drug works in prolonging the repolarization phase (phase 3 of action potential) by decreasing calcium and increasing potassium permeability. This results in increased effective refractory period of both heart chambers. Amiodarone has excellent therapeutic value and low rate of arrhythmia compared to other anti-arrhythmic drugs. One of the downside of this prolonged repolarization phase may cause early after depolarization which can induce short coupled PVCs leading to ventricular tachyarrhythmias especially Torsades de pointes. Although the rate of efficacy far outweighs the side-effects of amiodarone, we cannot ignore them in acute clinical settings as they can be life threatening.</p> <p>85-year-old woman admitted for Atrial flutter with rapid ventricular response. She was administered Metoprolol followed by diltiazem resulting in unsuccessful treatment. As the heart rate remained uncontrolled, 150mg Amiodarone was administered followed by an infusion at 1mg/min for 6 hours and 0.5mg/min for 18 hours. At the end of the infusion, the rhythm converted to sinus, however, the QT wave was prolonged. In a rapid sequence, the QTc was further prolonged followed by macro T wave alternans and then paroxysmal polymorphic ventricular fibrillation. Amiodarone was discontinued and Magnesium was administered. The rhythm reverted to sinus without any subsequent arrhythmias. Amiodarone has been established in successful conversion of arrhythmia to sinus rhythm and in long-term therapy of cardiac arrhythmias. Presence of severe co-morbidities (infection, heart failure, recent surgery, dilated cardiomyopathy, myocardial infarction and underlying cardiac genetic conditions) in patients is considered to be pro-arrhythmic and there is a decreased in the rate of conversion or a higher recurrence of arrhythmias compared to normal populations.</p> <p>Macroscopic T wave alternans (TWA) is described as variation in the beat-to-beat amplitude or shape of T wave. It shows temporal heterogeneity in the repolarization of ventricles involved in underlying reentrant arrhythmias. TWA is used in the risk stratification of predicting sudden cardiac death and life-threatening arrhythmias.</p> <p>In addition, TWA is a part of long QT syndrome (LQTS) score that is often used for diagnosis of LQTS. Macroscopic TWA is a rare finding in congenital LQTS; Zariba et al. showed occurrence of 1.2% in a large series. The mechanism of action causing prolong QT. The exact mechanism of this phenomenon is unknown but it has been hypothesized that an early after-depolarization–induced, triggered response initiates torsade de pointes but that the arrhythmia is maintained by a reentrant mechanism. Torsade de Pointes is observed in <1% of patients treated with Amiodarone but the occurrence can increase significantly in patients with various underlying pathological conditions, most commonly Torsades de Pointes. It is important to perform further testing for prolong QT syndrome in this patient which is our next step.</p>

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Clinical Research	Dhruv Vasant	Waheed Abdul MD, Yaoyun S Shen MD, Mathurika Jeyasingam MD, Akbar Mohad BSc, Abdul A Ameen MD, Mohammad T. Javed MD	Jersey City Medical Center (Amer Syed)	Utility of BNP as a Diagnostic Marker for Cardiac Tamponade	<p>Purpose: The utility of serum N-terminal pro-B-type natriuretic peptide (BNP) is well recognized in determining presence, severity and prognosis of patients with congestive heart failure, which is attributed to myocardial stretch in fluid overload. Given the restriction present in cardiac tamponade, it is possible that the myocardial stretch becomes reduced. The purpose of this study is to investigate the possible utility of BNP levels in determining presence or absence of cardiac tamponade aside from the patient's clinical presentation.</p> <p>Methods: This retrospective study looked at 44 patients over the last 5 years (2004-2014), which included 9 patients who were treated, by pericardiocentesis, pericardial window or thoracotomy after a diagnosis of cardiac tamponade and or pericardial effusion. We compared before and after pericardial effusion drainage values of BNP in these patients.</p> <p>Results: The mean NT-proBNP level during cardiac tamponade/pericardial effusion was 2825.3 + 3251.5 ng/L (range 146 to 9450), and the median level was 796 ng/L (interquartile range 730 to 4050). The mean NT-proBNP level after drainage was 10938.6 + 13209.2 ng/L (range 887 to 37500), and the median level was 5800 ng/L (interquartile range 2010 to 10400). NT-proBNP values were higher after drainage (p=0.0451). The mean increase after drainage was 8113.2 + 10259.3 ng/L, and the median level was 3290 ng/L (interquartile range 1280 to 9660).</p> <p>Conclusions: NT-proBNP level in patients with cardiac tamponade increases after pericardial effusion drainage. Further prospective studies may be needed to evaluate if lower levels of serum BNP, compared to baseline, can serve as an accurate rule in/out predictor of cardiac tamponade in the shock state.</p>

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Clinical Vignette	Dhruv Vasant	Mathurika Jeyasingam MD, Waheed Abdul MD, Abdul A Ameen MD, Mohammad T. Javed MD	Jersey City Medical Center (Amer Syed)	Tocilizumab related Severe Reversible Cardiomyopathy	<p>Hypersensitivity-associated acute coronary syndrome or more commonly termed Kounis Syndrome is described as coincidental occurrence of acute coronary syndrome in the setting of hypersensitivity anaphylactic insults. Allergic myocardial infarction and angina is called Kounis syndrome. The main pathophysiological mechanism is vasospasm of the epicardial coronary arteries due to increased inflammatory mediators released during a hypersensitivity reaction. Several triggers have been reported capable of inducing this sequale including: certain drugs (antibiotics, antineoplastics, analgesics) chemicals, environmental exposure, diseases, or foreign bodies. We present a case of Kounis syndrome resulting from infusion treatment with Tocilizumab for Rheumatoid Arthritis.</p> <p>A 46-year-old female with a past medical history of sarcoidosis, systemic hypertension, hyperlipidemia, rheumatoid arthritis, admitted for dyspnea nad chest pain after receiving her 4th dose of Tocilizumab (Acterma). Serial EKGs showed: normal sinus rhythm at 95 BPM, normal intervals, T wave inversions in III, aVF, with mild ST depressions in III, aVF, and mild ST depressions in V3. CT Angiography was performed which rule out a pulmonary embolism. Her troponins trended upwards and she was treated for NSTEMI. 2D echo showed severe diffuse systolic dysfunction; cardiac catherization performed showed no abnormalities in the coronary arteries which is consistent with Kounis syndrome.</p> <p>In our case, the patient's initial symptoms during her infusion treatment with Tocilizumab (Acterma) is suggestive of an anaphylactoid reaction as underlined by the established criteria from the Summary report of the Second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. Her initial anaphylactoid reaction, EKG showing an NSTEMI with elevated Troponins and cardiac catherization failing to reveal any underlying coronary artery disease led us to make the diagnosis of Type 1 variant Kounis syndrome.</p> <p>Type I variant defines patients with no previous history of predisposing factors for coronary disease and normal coronary arteries. In these patient's the release of inflammatory mediators can induce either coronary artery spasm without increase of cardiac enzymes and troponins, or coronary artery spasm progressing to acute myocardial infarction with raised cardiac enzymes and troponins.</p> <p>As with our patient, we recommend that clinicians have a high degree of suspicion of Kounis syndrome in patients experiencing drug reaction with signs of ACS. Treatment must be aimed at stopping the insulting agent, providing symptomatic relief with H1/H2 blockers, corticosteroids and evaluating for underlying coronary artery disease. Further research must be conducted to ascertain if it's possible to reinstate the offending drug, especially in such cases where it has been proven that very little treatment options exist.</p>

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Clinical Vignette	Usman Mazahir	Lann Danielle MD, Michael Carson MD	Jersey Shore Medical Center (Mayer Ezer)	Continuous Insulin Infusion for Treatment of Severe Asymptomatic Hypertriglyceridemia	<p>Case: A 66 year old Caucasian male diabetic with coronary artery disease was admitted after presenting to the emergency department after experiencing chest discomfort and diaphoresis while working in his yard, both resolved after rest. He had a history of hypertension, hyperlipidemia, type 2 diabetes mellitus, and coronary artery disease with stents placed 1 and 13 years prior to presentation. His ECG did not demonstrate changes vs. his prior from a year ago and troponin was normal. He reported being noncompliant with his insulin, statin and anti-platelet medications for over 3 months. He also reported consuming an average of 6-8 alcoholic beverages/day. The lab called as his serum was white & opaque. LABS: Sodium 125 mmol/L, Glucose 501 mg/dl, Triglycerides (TG) 13,700 mg/dl, Total cholesterol 1120 mg/dl and LDL 431 mg/dl. Lipase was normal. He denied abdominal pain, nausea, vomiting, diarrhea or any other symptoms of acute pancreatitis. Physical examination revealed obesity, no xanthomas or corneal arcus. He was transferred to the ICU for continuous insulin infusion which effected dramatic improvement of TG with a drop to 1410 mg/dl on Admission Day 6. He was started on fenofibrate, niacin, rosuvastatin 40 mg and orlistat. The cardiac stress test during admission was normal. TG level obtained two months later as an outpatient was 165 md/dl.</p> <p>Discussion: This case illustrates an unusual presentation for a patient with severely elevated TG levels. White, opaque serum, along with the history of poorly controlled diabetes and hyperlipidemia is consistent with severe hypertriglyceridemia which is usually defined as levels exceeding 1000 mg/dl. In severe hypertriglyceridemia rapid lowering of plasma triglycerides has to be achieved as these patients are at increased risk for acute pancreatitis and cardiovascular events. It is quite rare to have values exceeding > 10,000 mg/dl without evidence of pancreatic injury such as our patient. A PubMed search using terms severe, acute, asymptomatic, and hypertriglyceridemia revealed cases of acute pancreatitis associated with levels over 10,000 mg/dl, but no reports of asymptomatic patients. Omega 3 fatty acids and fibrates are used for chronic control, but have no role in the acute setting. Therapeutic options for acute treatment include continuous insulin therapy, heparin or a combination of both, plasmapheresis and octreotide. There are no standard clinical guidelines to approaching severe hypertriglyceridemia, but plasmapheresis is reserved for more emergent symptomatic cases associated with acute pancreatitis. Long term, appropriate diet, lifestyle changes, medications, and close outpatient follow-up to reinforce compliance are essential for patient well-being.</p>

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Clinical Research	Aref Obagi	Shreya Ghetiya MD, Jennifer HeckKannelidis RN, Michael P. Carson MD	Jersey Shore Medical Center (Mayer Ezer)	Compliance and Bleeding Using Updated AHA/ACC Heparin Dosing Guidelines for NSTEMI Patients	<p>Background: In 2015 the AHA/ACC recommended new heparin dosing guidelines for Non-ST elevation myocardial infarction (NSTEMI) patients to decrease the maximum initial bolus from 7500 units to 4000, and the initial infusion rate from 1500 u/hr to 1000. We implemented those guidelines, and as part of the National Cardiovascular Data Registry (NCDR) quality assurance process, compliance and the rates of bleeding were recorded. We analyzed the data to report the rates of compliance with the new recommendations, and determine if the lower heparin doses were associated with a decrease in bleeding events.</p> <p>Methods: The study was conducted in a 546 bed tertiary care center. NCDR defines bleeding events as any of the following within 72 hours of starting heparin: 1) Hemoglobin (Hgb) drop ≥ 3 g/dl; 2) Transfusion of red blood cells with Hgb > 8g/dl; 3) Procedural site intervention to address bleeding.</p> <p>Results: In 2014, 62% (78/125) received a heparin bolus >4000 units vs. 22% (23/105) in 2015. The relative risk (RR) of receiving a bolus >4000 units in 2014 vs. 2015 was 2.8 (95% CI [1.9,4.2]). The average drop in Hgb was similar: -1.9 ± 1.6 in 2014 vs. -1.9 ± 1.7 in 2015. The NCDR bleeding rate in 2014 (2.5%) was not significantly different from 2015 (5%), (Wilcoxon Rank-sum $p=0.2$). With the higher allowable heparin doses in 2014 the risk of a Hgb drop ≥ 3 g/dl for the entire admission was not higher than in 2015 (RR 1.05. 95% CI [0.6,1.9]).</p> <p>Conclusion and Plan: Implementation of the new recommendations was associated with 40% a decline in excessive heparin dosing for NSTEMI patients at our institution, but the rate of NDCR defined bleeding did not change significantly. The stable bleeding rate could be explained by the presence of confounders that were not recorded as part of the nursing quality assurance process, possibly a threshold effect in that the risk of bleeding in these patients does not directly correlate with the degree of PTT elevation, or an effect size too small to be detected by 230 patients. We are now conducting a detailed chart review to describe the demographics of all NSTEMI patients to compare those with an NDCR bleeding event and to patients who had a Hgb drop ≥ 3 during hospitalization but did not meet NDCR criteria.</p>

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Clinical Vignette	Huzaif Qaisar	Xiaoyin Tang MD, and Michael Carson MD	Jersey Shore Medical Center (Mayer Ezer)	Adult Still's Disease with pericarditis	<p>Objective: Diagnose and treat adult Still's disease with pericarditis.</p> <p>CASE: 22 year old female with history of juvenile idiopathic arthritis (JIA) had been treated with abatacept injection, but stopped it a year ago due to loss of insurance. 15 days ago she was treated with a 10 day steroid taper for a flare of her JIA symptoms, but they recurred as the steroids were tapered. She presented with 10 days of diffuse arthralgia and sharp chest pain, aggravated with breathing and recumbency. She had nausea/vomiting, sore throat and subjective fevers for 1 week. EXAM: Temp 101F, HR 132, BP 102/61, RR 18. Distressed, diffuse chest wall tenderness; no murmur/gallop/rub, a few scattered ronchi, right wrist swelling with joint line tenderness, painful passive range of motion of both knees/elbows.</p> <p>Labs: Hb 11gm/dL, PLT 330 K/uL, WBC 37.4 K/uL(4.5-11),Band 7%, neutrophil 88%(50-70%), AST/ALT normal, Bilirubin 2.3mg/dL (0.2-1.3). Ferritin 4820 ng/ml (11-307), ESR 96mm/hr (0-20), CRP 33 mg/dL (<0.744), LDH 275 iU/L (91-200), Mycoplasma IgM 1.12 (<1.10), troponin 0.44, anti CCP IgG 2 units (<19 units) , ANA 0.29(<1.10), hepatitis B, C, EBV, CMV, cocksackie tests negative. . She was started on broad spectrum antibiotics for SIRS, azithromycin due to the elevated Mycoplasma IgM, and ibuprofen for a provisional diagnosis of pericarditis. On Day 2 an evanescent salmon colored rash appeared on her chest wall. After no response for 3 days, she was given 80 mg IV methylprednisolone followed by prednisone 60 mg PO with taper, colchicine 0.6 mg/day for 3 months and celecoxib 200mg/day. Her chest pain, fever and sore throat improved.</p> <p>Discussion: Adult Onset Still's disease (AOSD) is a severe form of JIA (10-15% cases) in patients >16 years of age with at least one systemic presentation. It is characterized by daily fevers, arthritis, evanescent salmon colored rash in 75-95 % cases, and the etiology is unknown. There are three patterns of disease with equal sex distribution: Monophonic (<1 year, 1 episode, presents with fever, rash and serositis), Intermittent (>1 episode), and Chronic (> 1 year, more joint presentation in descending order are knees, wrists, ankles, elbows, PIP and shoulders, fusion of wrist joints is characteristics of ASD). Diagnosis is made by the presence of 2 major Yamaguchi criteria, and five total: Major (fever> 102.2 F > 1week, arthralgia/arthritis >2 weeks, salmon colored non pruritic macular rash, WBC >10k with >80% PMN) and Minor: (sore throat, adenopathy, increased LFT/LDH, Negative RF & ANA, hepatosplenomegaly). Still's disease responds to steroids, and NSAID and DMARDS can be added for moderate to severe disease. In conclusion, AOSD is rare but should be considered in the differential diagnosis when patients >16 years of age present with fever, rash, arthritis, and arthralgias.</p>

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Clinical Vignette	Sarmed Mansur	Ihab Kassab MD, Sarah Abuseif MD, and Michael P. Carson MD	Jersey Shore Medical Center (Mayer Ezer)	NOT "Insulin Neuritis": Acute Neuropathy Precipitated by Rapid Correction of Hyperglycemia	<p>Case: A previously non-diabetic male in his 50's who drank 120 ounces of alcohol daily presented with lethargy and was found to have HHNK with a plasma glucose of 2294 mg/dl. He was admitted to the Intensive Care Unit (ICU) and treated with intravenous fluids and an insulin drip. Day 2: his blood glucose levels were ~200 mg/dl, he was transitioned to subcutaneous insulin glargine 36 units qHS, subcutaneous insulin aspart 12 units TID AC, and transferred to a general medical floor. At that time he noted new numbness and painful paresthesias of both hands, positional dizziness, mild odynophagia, and dysphagia.</p> <p>Exam: Supine BP & HR: 127/89 mmHg, 76 bpm; Standing BP & HR: 95/67mmhg and 91 bpm. Modified barium swallow revealed a single episode of transient penetration with solids with a thin liquid wash. His orthostatic BP improved with midodrine 5mg TID to the point that he could stand and use a walker. Gabapentin was started for the paresthesias. The numbness improved, but the painful paresthesias persisted and were not responsive to opioids. Three weeks later he remained on the midodrine with good effect, but the painful paresthesias persisted.</p> <p>Discussion: Treatment induced neuropathy of diabetes (TIND) is a rare small fiber and autonomic neuropathy that develops after rapid but appropriate correction of severe hyperglycemia. TIND was first described 83 years ago, attributed to the use of insulin, and called inulin neuritis. However, after TIND was reported with oral agents, insulin was absolved of blame. Literature search identified 2 articles including a series of 104 patients encountered over 5 years at a tertiary center in the USA. The reported risk of neuropathy was 20% in those whose absolute drop in HbA1C value was 2% over 3 months, and the risk was 80% in those whose HbA1C dropped 4%. However, those with higher HbA1C values also had a higher rate of diabetic retinopathy. Symptom resolution required 9-18 months in most. The exact pathophysiology of the neuropathy is unclear. It is possible his alcohol intake predisposed him to this condition, so thiamine was continued. There is no evidence to suggest that permissive hyperglycemia and slower reduction in hyperglycemia would prevent this complication. In summary, rapid lowering of HbA1C may predispose patients to TIND, and given the increase in obesity and diabetes, more patients are at risk. However, given the low frequency of TIND, we do not plan to alter management for diabetic patients who present with acute HHNK.</p>

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Clinical Vignette	Saira Chaughtai	Anmol Cheema MD, Daniel Kennedy DO	Jersey Shore Medical Center (Mayer Ezer)	ATYPICAL PRESENTATION OF ANGIOEDEMA IN GUILLAIN-BARRE SYNDROME	<p>Case Summary: A 67 year-old man presented to his primary care physician’s office with diarrhea that improved with antidiarrheal. One week later, he presented again for cold-like symptoms with sinus congestion. The following day, he came to the Emergency Department for worsening dyspnea, muffled voice, facial and tongue swelling consistent with angioedema. PMH: hypertension treated with enalapril, coronary artery disease, hyperlipidemia, DM Type 2, atrial fibrillation, and carcinoid tumor status post right hemicolectomy. His enalapril was held and he received intravenous corticosteroid, H1-antihistamines, and H2-blocker. The following day, he developed excessive oropharyngeal secretions, diplopia, dysphagia, dysarthria, ptosis, and upper extremity weakness of 3/5. Laryngoscopy demonstrated mild edema of the tongue base and a patent airway. His respiratory status rapidly deteriorated requiring endotracheal intubation and intensive care unit admission. Computed tomography scan and magnetic resonance imaging of the head and brain were negative for any pathologic process. The differential at the time included myasthenia gravis due to bulbar weakness; although, acetylcholine antibodies later came back negative. Electromyography of his deltoids and biceps showed low amplitude with reduced recruitment consistent with acute motor axonal neuropathy (AMAN), a GBS variant. He underwent eight sessions of plasmapheresis with minimal improvement initially. He required a tracheostomy, but slowly improved and was later discharged to an acute rehab facility.</p> <p>Conclusion: GBS is an acute polyneuritis believed to be immunologically mediated. It usually presents with an ascending paralysis after an acute viral illness although there are many clinical variants that have been reported in medical literature. We present a patient admitted to the hospital for angioedema who, based on neurological examination and EMG findings, was ultimately diagnosed as the AMAN variant of GBS. There are four subtypes of peripheral neuropathy that can be seen in GBS. These include acute inflammatory demyelinating polyneuropathy (AIDP), AMAN, acute motor and sensory axonal neuropathy (AMSAN), and the Miller Fisher Syndrome (MFS). AIDP is the most common in western countries, whereas AMAN occurs more commonly in Japanese and Chinese youth. In AMAN, sensory nerves are not affected, consistent with our patient. While rare, it is important for physicians to be aware that GBS can present with angioedema, and should be considered the differential of an angioedema not responding to standard treatment.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Tejas Karawadia	Michael Carson MD	Jersey Shore Medical Center (Mayer Ezer)	Hemophagocytic Lymphohistiocytosis (HLH): A cause of fever you shouldn't miss	<p>Case: A male in his 40s with no prior medical issues presented to his PCP with fever for 1 week, was given levofloxacin, but presented to the hospital when the fever did not resolve after a week, and new fatigue developed.</p> <p>Exam: Temp 101F, HR 98, BP 104/54 mmHg. He appeared toxic, with chills, hepatomegaly and papular rash over anterior chest wall.</p> <p>Labs: Hb 8.8 gm/dL (12-16), Plt 22 K/uL (150-450), WBC 2.4 K/uL (4-12), normal electrolytes and creatinine. AST 637 iU/L (10-42), ALT 311 iU/L (10-60), ALKPHOS 110 iU/L(38-136), Bilirubin 2.5 mg/dL (0.2-1.3). Ferritin 27817 ng/ml (10-300), Total iron 78 mcg/dl (60-170), TIBC 212 mcg/dl (230-450), Reticulocyte Count 4.17% (0.5-2%).</p> <p>Treatment with broad spectrum antibiotics (vancomycin and cefepime) and intravenous fluids was initiated. However, blood culture, urine culture and CXR were negative/normal. Bone marrow biopsy to evaluate pancytopenia and liver biopsy on Day #4 both showed increased hemophagocytic histiocytes. A diagnosis of HLH was made. Malignancy evaluation was negative. Positive ANA (1:1280), SSA 68 AU/ml (<40) and SSB 83 AU/ml (<40) suggested Sjogren's syndrome as a trigger. Day #7: weekly etoposide (150mg/m2) and daily dexamethasone (20mg) were started. Over the next few weeks his fever and anemia improved, and he was discharged Day #31 on the same regimen and close outpatient follow up.</p> <p>Discussion: HLH is a rare but aggressive and life threatening syndrome of excessive immune activation. The incidence is 1.2 cases/10x6 persons/ year. Hemophagocytosis describes the classic pathologic finding of activated macrophages engulfing the erythrocytes, platelets, leukocytes and their precursors. HLH can be caused by gene mutation (familial) or sporadic (acquired). The sporadic form has a clear triggering event [viral illness (EBV), autoimmune, malignancy (lymphoma)]. Both forms have excessive activation of macrophages and cytokine production which leads to cytopenias and specific clinical findings. Diagnostic criteria consist of the either presence of a specific HLH gene mutation OR five of the following: Fever; hepatosplenomegaly; two cytopenias (ANC<1000/microL, Hb <9g/dL, platelets <100,000/microL); hypertriglyceridemia and/or hypofibrinogenemia; hemophagocytosis in bone marrow, spleen, lymph node or liver; low NK cell activity; ferritin >500ng/ml, or elevated soluble CD25.</p> <p>Untreated patients have a survival of months and the greatest barrier to therapy is often delayed diagnosis. The case illustrates the importance of bone marrow biopsy in patients with persistent fever and unexplained pancytopenia. Survival in HLH can be dramatically increased via treatment of triggering condition, etoposide and dexamethasone, supportive platelet and PRBC transfusions and in refractory disease, allogenic hematopoietic cell transplant is recommended.</p>

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Clinical Vignette	Tejas Karawadia	Michael Carson MD	Jersey Shore Medical Center (Mayer Ezer)	A Physicians' Dilemma: Acute Intermittent Porphyria in pregnancy	<p>Case Summary: A 26-year-old G2P1 presented at 24 weeks of pregnancy with headache, shortness of breath, lightheadedness, floaters in her vision, dizziness, palpitations and mild abdominal pain for 8 hours. She was recently diagnosed with AIP.</p> <p>EXAM: HR 93, BP 91/57, 96% on Room air. Fetal heart tracing was reassuring. Serum and urine porphyrins were drawn and she was diagnosed with AIP flare. Treatment with carbohydrate loading D10 at 200cc/hr improved her symptoms. By day 3 she was discharged home on carbohydrate rich diet. Lyophilized preparation of hydroxyheme (hematin) was considered, however as her symptoms improved with IV D10 it was deemed unnecessary.</p> <p>Discussion: Porphyrias are inherited disorders caused by decreased enzymatic activity within the heme biosynthetic pathway. AIP is the most common worldwide, with an estimated prevalence of approximately 5 per 100,000, is caused by an inherent deficiency of prophobilinogen deaminase (PBD), which is required for the final steps of heme synthesis. When heme synthesis is triggered, the PBD deficiency leads to a buildup of toxic heme precursors that cause the neurovisceral symptoms such as abdominal pain, vomiting, constipation, extremity weakness, seizures or life threatening respiratory paralysis. Acute attacks can be triggered by factors that increase the heme production pathway (medications, smoking, alcohol, progesterone, reduced caloric or carbohydrate intake, and stress). Seizures may be the initial symptom and it is important to remember that barbiturates, phenytoin and most other antiepileptics can worsen the symptoms by inducing hepatic heme synthesis. Diagnosis of AIP is usually established by urinary porphobilinogen (PBG), which is a highly sensitive and specific test for three most common acute porphyrias and sufficient for initiating treatment. In milder cases initial treatment with D10 carbohydrate loading is sufficient to downregulate ALA synthase (ALAS), the enzyme that controls the rate of heme production. The next treatment option to downregulate ALAS is intravenous hematin. A healthy fetus depends on a healthy mother, therefore the FDA category should NOT preclude using medication when other options have failed and/or the mother's well-being would be compromised. Hematin (Category C) was not necessary given her response to D10. A literature search revealed a retrospective study of 15 women who received hematin during pregnancy, and no adverse pregnancy/neonatal outcomes were noted. During administration of high glucose loads maternal sugars should be monitored, and normoglycemia maintained in order to avoid fetal effects of fetal hyperglycemia.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Shreya Ghetiya	Michael P. Carson, Richard Abramowitz	Jersey Shore Medical Center (Mayer Ezer)	A Rare Case: Granulomatosis with Polyangiitis Presenting Only with Pulmonary Nodules	<p>Lase Summary: A 46-year-old white male with history of hypertension and smoking presented with malaise and pleuritic right lower chest pain. Physical exam was unremarkable except mild tachypnea due to pain. Labs: CBC and CMP normal, serum quantiferon and Cryptococcus antigen negative. CT chest with contrast showed multiple spiculated bilateral lung masses (largest 3.0 x 3.1 x 2.8 cm) with central cavitation and mediastinal lymphadenopathy. Lung nodule biopsy was done. Patient followed up in 10 days at outpatient clinic to review biopsy results that revealed necrotizing granulomatous inflammation suggestive of leucocytoclastic vasculitis. Fungal and AFB stains were negative. At this visit he complained of bilateral ankle arthralgia and hematuria, so he was readmitted and further work up showed elevated ANCA with cytoplasmic staining directed against serine protease 3(PR-3). Other negative studies include HIV Ag/Ab, Hepatitis B/C, and Anti-Nuclear Antibody. He was diagnosed with ANCA associated vasculitis (AAV): Granulomatosis with Polyangiitis (GPA) subtype. He was started on 1g of IV methylprednisolone for 3 days, followed by 60mg oral prednisone daily and oral cyclophosphamide daily. His symptoms including hematuria improved with treatment. He was placed on empiric TMP/SMX for Pneumocystis Jirovecii prophylaxis.</p> <p>Discussion: AAV includes GPA, Microscopic Polyangiitis, Renal Limited Vasculitis and Eosinophilic GPA. All of these entities have overlapping features. Our patient did not have ear, nose and throat manifestations which are typically present in 90% of GPA cases. A “limited” form involving only upper or lower respiratory tract is seen in 25% of cases. However, sole findings of lung parenchymal involvement on initial presentation is extremely rare. C-ANCA is positive in 90% of patients with systemic GPA but only in 60% with limited form. Of those with positive c-ANCA, 80-90% are directed against the PR-3 antigen. ANCA is helpful in diagnosis but does not predict severity or prognosis. Treatment of GPA involves two phases: induction and maintenance. Initial therapy involves high dose steroid and either cyclophosphamide, rituximab, or methotrexate. Plasma exchange is offered in life-threatening illness. 3-6 months of induction therapy is needed to achieve remission after which patients are maintained on low dose cyclophosphamide or methotrexate depending on severity of disease. Prophylaxis for Pneumocystis Jirovecii pneumonia and other opportunistic infections should be given.</p> <p>Conclusion: Although classic presentation of GPA involves Ear/Nose/Throat, it may rarely present as pulmonary nodules, which could be misdiagnosed as infection or malignancy. Early diagnosis and treatment can reduce disease severity, multi-organ involvement and improve overall prognosis.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Shreya Ghetiya	Edward Liu MD, Lito Fune MD.	Jersey Shore Medical Center (Mayer Ezer)	Tap Water- A Risk for Rapidly Growing Non-tuberculous Mycobacteria Blood Stream Infections	<p>Cases Summaries: We present two cases of bacteremia caused by RGM.</p> <p>Case 1: A 20-year-old female with past medical history (PMH) of hepatitis C and injection drug abuse fell and fractured her left clavicle. Several weeks later after using intravenous heroin, she presented with increasing pain and swelling at the fracture site. Her blood cultures grew MRSA. Imaging suggested displaced fracture with osteomyelitis. IV vancomycin was started. She underwent clavicular joint debridement, remained in the hospital for IV antibiotics and developed fever with a PICC line in place. Repeat blood culture grew Mycobacterium mucogenicum. She was initially treated with IV amikacin and cefoxitin, and then amikacin and clarithromycin to complete total 6 wks. It was suspected that she used her PICC line for recreational drug use while in the hospital.</p> <p>Case 2: A 24-year-old male with PMH of end-stage renal disease on hemodialysis via left internal jugular dialysis catheter came in with fevers and malaise. He had fever of 103.5 F. He was treated with IV vancomycin and gentamycin empirically which were changed to IV amikacin and cefoxitin when blood cultures grew M. fortuitum on day 2. He was later switched to oral clarithromycin with IV amikacin to finish a 2 week antibiotic course. The jugular dialysis catheter was removed and temporary left femoral line was placed until his vascular graft matured.</p> <p>Discussion: RGM are mycobacterial species that grow within one week. The primary species are M. fortuitum, M. chelonae, and M. abscessus and include M. mucogenicum. Outbreaks of pulmonary infections and bacteremia have been reported. They are associated with catheter related blood stream infections in patients with hematological malignancies and in those with long term peritoneal dialysis catheters. Pulmonary infections can be very difficult to treat. Primary blood stream infections are uncommon. Good catheter care and sterile flushes are the mainstay of prevention of blood stream infections (BSI). Water used for clinical use such as dialysis needs to be treated and/or filtered to avoid causing BSIs from RGM. Antibiotic choices to treat RGM include amikacin, clarithromycin, fluoroquinolones, doxycycline and imipenem. There are no controlled trials determining which antibiotic regimens are best. Both of our patients had exposure to local water supply (hospital or local dialysis) and this may reflect presence of RGM in local water supply.</p> <p>Conclusions: RGM are emerging causes of blood stream infections and they can cause significant morbidity and mortality in immunocompromised patients. It can be effectively prevented with careful handling of external catheters and proper treatment/filtration of water supply. Treatment of RGM bacteremia is difficult given lack of controlled trials comparing different antibiotic regimens.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Young Kevin xia	Anna Kufelnicka, MD	Jersey Shore Medical Center (Mayer Ezer)	Haemophilus Influenzae meningitis presenting in an adult with Hemoglobin SC	<p>Case Summary: A 72-year-old African American female presented with 4 days of malaise that evolved into headache, neck pain and confusion associated with fever. PMH: mitral valve prolapse, glaucoma and depression. Due to symptoms and signs suggestive of meningitis, lumbar puncture was done and CSF revealed neutrophilic pleocytosis with WBC count 17205 /cc, glucose < than 9mg/dL and protein of 675mg/dL consistent with bacterial meningitis. MRI of the head showed fluid levels within the occipital horns of the lateral ventricles bilaterally suggestive of ventriculitis. CSF culture was negative. She was initially treated with IV ceftriaxone, vancomycin and ampicillin then subsequently narrowed down to ceftriaxone based on FilmArray assay PCR confirming Haemophilus Influenzae. Her mental status improved and she was transferred to rehab to finish ceftriaxone 2 gram IV q. 12 hours for 14 days. Workup for anemia and possible immunodeficiency resulted in positive sickle cell screen (sickledex) and sickle cell Hemoglobin SC was later confirmed by hemoglobin electrophoresis.</p> <p>Discussion: Haemophilus Influenzae (H.flu) are gram negative anaerobic bacteria categorized as encapsulated and un-encapsulated strains. The capsule resists phagocytosis and complement mediated lysis. Clearance of these bacteria requires opsonization assisted by anti-polysaccharide IgM antibodies produced by unique IgM memory B cells found in the marginal zone of the spleen. Therefore opsonized bacteria are only cleared effectively by the spleen. Impaired splenic function due to autosplenectomy is a significant contributor to morbidity and mortality in sickle cell disease. Hemoglobin sickle cell disease (HbSC) is the second most frequent hemoglobinopathy in sickle cell disease and should not be considered a milder form of HbSS. This patient's HbSC may have played a role in her being susceptible to H.flu meningitis which was the leading cause of bacterial meningitis in children before Hib vaccine in 1985. Clinicians should maintain a high degree of suspicion for pathogens that afflict those that may be asplenic or hyposplenic. H.flu has proven difficult to be grown on cultures and is time consuming. Newer PCR assays such as FilmArray allows rapid simultaneous detection 14 bacterial, viral and yeast pathogen with comparable sensitivity and few false negatives in head to head studies with conventional cultures. Rapid detection will allow targeted antibiotic coverage limiting use of unnecessary antimicrobials. Cultures nevertheless should still be obtained to rule out pathogens that are not detected by FilmArray PCR assay and to provide sensitivity testing of isolated organisms.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Young Kevin Xia	Edward W Liu, MD. Anna Kufelnicka, MD	Jersey Shore Medical Center (Mayer Ezer)	Mycobacterium Abscessus presenting in a New England Traveler to Florida	<p>Case Summary: A 75 year old woman presented with 2 months of shortness of breath on exertion, productive cough with copious amounts of white yellow sputum, low grade fevers, and 15 lb unintentional weight loss. PMH significant for pulmonary fibrosis, chronic obstructive pulmonary disease, bronchiectasis and paroxysmal atrial fibrillation. Inpatient chest radiograph showed hyperaeration with fibrotic changes. CT chest noted ground glass opacities with no suspicious nodularities. Outpatient workup by pulmonologist included sputum culture positive for Pseudomonas aeruginosa. She was sent to rehab and treated with ceftazidime for the pseudomonas pneumonia. However, continued dyspnea prompted a return visit to the hospital where a bronchoscopy was done, biopsy was negative for malignancy, but culture from bronchial washings grew pseudomonas, and 4 weeks later AFB culture showed Mycobacterium abscessus. Further inquiry revealed she frequently travels to Florida where M. abscessus is endemic. She was started on IV amikacin 3 times a week, and imipenem and azithromycin daily. Her symptoms improved during inpatient stay, PICC was placed, she was transferred to rehab to complete 1 month of IV antibiotic therapy with close follow up for signs of improvement, to be followed by 12 months of oral/inhaled antibiotic therapy.</p> <p>Conclusions: Mycobacterium abscessus (MA), first described in 1953, is a non-motile, rapidly growing acid-fast bacteria found commonly in water, acidic soils rich in humic & fulvic acids as found in Florida. MA is associated with a wide spectrum of infections; bacteremia, soft tissue & serious nosocomial infections from contaminated medical instruments treated with water not sterilized of MA. Person to person transmission remains rare. Individuals with underlying chronic lung disease and those who are immunocompromised are at higher risk for contracting MA lung infections. Florida leads mycobacterium pulmonary infections by 15% annually. Diagnosis may be difficult to make especially in non-endemic areas where clinicians are not familiar with the pathogen. A high degree of suspicion must be maintained in individuals who are not improving from empiric antibiotic treatment and have chronic progressive symptoms including fevers, night sweats, and weight loss. Appropriate AFB cultures, usually from bronchoscopy BAL specimens, are preferred to isolate these pathogens. Local labs may have difficulty providing in vitro susceptibilities. Reference labs with expertise in mycobacteria may be necessary to provide accurate susceptibilities. Combination antibiotic treatment regimens are prolonged and may have significant toxicities. Expert consultation with Infectious Diseases specialists and/or Pulmonary specialists familiar with this difficult disease should be utilized for optimal outcomes.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Sheila Kalathil	Edward Liu	Jersey Shore Medical Center (Mayer Ezer)	Recurrent Herpes Encephalitis Presenting as Retro-grade Amnesia	<p>Case: A 73 year old woman with a past medical history of herpes encephalitis 29 years ago presented initially to an outside facility with confusion and headache. After a negative neurological workup, she was diagnosed with a transient ischemic attack and discharged home. A few days later, she was re-admitted after being found by her neighbor, confused and wandering in the neighborhood. Upon admission, she was found to be alert and oriented to her place and surroundings, but having no recollection of the preceding events. She could not remember any events in the past 4 or 5 days, or even of the morning itself. She was noted, however, to be full awake, alert, and on physical exam did not have any neurological deficits. A CT head only re-demonstrated left temporal lobe encephalomalacia, from her initial episode of encephalitis. She was diagnosed with amnesia, and transferred to our facility for prolonged EEG-monitoring to rule out seizures. Video EEG determined Right sided frequent sharp wave activity on VEEG, suggestive of possible seizure focus, and she was started on seizure medication. The patient continued to remain confused, and also began having fevers and headache. Lumbar puncture was obtained, and the patient was empirically started on IV acyclovir given high suspicion considering her past history of herpes encephalitis. Lumbar puncture results revealed 206 WBCs, 43% monocytes, and 43 mg/dL glucose. HSV 1 DNA PCR was detected in CSF, confirming the diagnosis. She received a 2-week course of IV acyclovir, continued to struggle with intermittent confusion as well as agitation, but was eventually discharged to rehab in stable condition.</p> <p>Discussion: Herpes Encephalitis is the most common cause of sporadic encephalitis worldwide, and has a bi-modal distribution, with most occurrences either in those younger than 20 or those greater than 50. Nearly 10-20% of the 20,000 viral encephalitis cases are found to be HSV, and the majority of HSV 1. Clinical finding are usually seen in the form of focal neurologic signs, dysphasia, aphasia, or decreased consciousness. Less common syndromes which may be seen are hypomania, Kluver-Bucy syndrome, or amnesic states, as seen with this patient. Herpes encephalitis has high morbidity and mortality, and can result in lasting neurological deficits. Early treatment with IV acyclovir has been shown to reduce lasting neurological deficits. By recognizing uncommon presentations of herpes encephalitis, treatment can be initiated earlier to provide better outcomes.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Sarah Abuseif	Gustavo De La Luz, MD, Mohamed El Sawaf, MD, Michael Carson, MD	Jersey Shore Medical Center (Mayer Ezer)	The Iron Lung: When Iron Pill Aspiration Results in an Emergent Bronchoscopy and A Successful Medical Therapy.	<p>Case: 44 year old woman with a past medical history of Iron Deficiency Anemia on iron supplementation who presented with foreign body aspiration. She reported choking while taking her iron supplement which subsequently caused a severe cough and wheeze. She presented to the ED 12 hours later where a CT scan demonstrated an 8mm foreign body lodged between the right middle and lower lobe. The patient underwent immediate bronchoscopy to attempt retrieval of foreign body. We found severely inflamed, friable, and necrotic mucosa occluding the bronchus intermedius. The usage of the Roth net and forceps collected endobronchial content but the pill begun to disintegrate at the time of bronchoscopy. She tolerate the procedure well. She was started and discharged with prednisone 40mg taper for 12 days, Moxifloxacin 400mg daily for 5 days, an inhaled budesonide and formoterol 160mcg-4.5mcg twice a day and albuterol rescue inhaler. She had an outpatient follow up bronchoscopy at one month and four months demonstrating a significant reduction in necrosis, inflammation, and endobronchial narrowing. Histopathology confirmed iron deposition on endobronchial biopsy. Spirometry one week after pill aspiration showed FEV1 of 2.676L and FEV1/FCV of 77%. After 2 months of medical therapy FEV1 was 2.922L and FEV1/FCV 86%. An 8.42% improvement of lung function was achieved with medical therapy.</p> <p>Discussion: Iron Pill Aspiration was first described in 1988. The syndrome was then described in 2002. In many cases, iron tablet disintegration occurs quickly into the endobronchial tissue, despite early bronchoscopy. The transformation of ferrous to ferric iron is a highly toxic oxidative process resulting in bronchial damage. Mucosal inflammation, ulceration and necrosis is seen. The formation of fibrous tissue causes bronchial narrowing and potential irreversible stenosis. Current literature describes early interventions such as lobectomy, balloon bronchoplasty, bronchial stenting, cryotherapy and mitomycin C. However, in our case systemic steroids, antibiotics and inhaled steroid and beta agonist therapy was successful. Therefore, we report a case study in which medical management was successful in the treatment of iron pill aspiration.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Aref Obagi	Lance Berger MD, Michael P. Carson MD	Jersey Shore Medical Center (Mayer Ezer)	Incidental Finding of Basaloid Squamous Cell Carcinoma of the Thymus During Nuclear Stress Test Using Technetium (99mTc).	<p>Introduction: Nuclear stress test is a test that uses small amounts of radioactive substances and measures how well blood flows into the heart muscle at rest and during activity.</p> <p>CASE SUMMARY: A 55-year-old male with a history of inferior Myocardial infarction, 3 bare metal stents (BMS) in the right coronary artery placed 3 years ago, presented with a warning from his Angelmed device regarding possible ischemia. Angelmed devices are subcutaneous devices that monitor the electrocardiograph (ECG) pattern and alert the patients with a vibratory signal that they might be having active/silent ischemia. He was admitted, had negative troponins, and unchanged ECG. A persantine technetium (99mTc) sestamibi nuclear stress test showed an inferior wall fixed defect consistent with infarction. The nuclear images also detected an incidental mass with increased 99mTc uptake in the anterior mediastinum, raising concerns for malignancy. Cardiac catheterization was delayed to evaluate the mass as he might require resection. CT scan of the chest revealed a right anterior mediastinal mass measuring 4.5 cm x 4.9 cm infiltrating the lung parenchyma. Biopsy revealed poorly-differentiated basaloid squamous cell carcinoma (SCC) of the thymus. Positron Emission Tomography (PET) scan showed a mass with a standardized uptake value (SUV) of 7.2 consistent with malignancy. He then underwent surgical removal of the thymus followed by radiation and chemotherapy.</p> <p>DISCUSSION:Thymus tumors account for less than 1% of all tumors. Basaloid SCC of the thymus is a very rare type of thymic carcinoma. Most cases occur during the 5th decade, are discovered incidentally by routine chest X-ray or thoracotomy for unrelated causes and the size of the tumor usually ranges from 5 to 20 cm. It's considered a low grade malignancy, but 30% of cases are associated with metastasis to lungs and liver. Our literature search regarding tumors detected via Tc99 scans revealed 5 reports of benign Thymoma and one case of invasive epithelial thymoma. The search identified only 36 cases of Basaloid SCC of the thymus worldwide, and none were diagnosed incidentally during a nuclear stress. Treatment options include surgical removal for small size and low stage (stage I and II) and adjuvant therapy with chemotherapy and radiation for large size and high stage (stage III and VI). In summary, we report what to our knowledge is the first case of Basaloid SCC of the thymus incidentally detected during a Tc99 Sestamibi scan ordered to evaluate a patient for suspected coronary artery disease.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Sarah Abuseif	Mayer Ezer, MD	Jersey Shore Medical Center (Mayer Ezer)	Stiff as a Board: The Doxylamine Overdose Causing Severe Acute Rhabdomyolysis.	<p>Case: A 22-year old man with no known past medical history presented to the Emergency Department with severe agitation and bizarre behavior. He was escorted by police after acting strange at a convenience store. At the time of initial assessment, the patient was confused and unable to provide a detailed history including his own name. Physical exam: heart rate 151 bpm, blood pressure 130/71 mmHg, temperature 97.4 F, respiratory rate 18, oxygen saturation 97% on ambient air. He was confused, severely agitated, paranoid and disoriented to person, place and time. Pupils were dilated and minimally reactive, consistent with mydriasis. His posture was of a primitive pose; on all fours and appeared severely constricted. Skin was warm and dry. On palpation, extremities felt stiff as a wooden board. Laboratory values demonstrated a standard urine toxicology screen positive only for opioids. Salicylate and acetaminophen levels were negative. Initial Creatine Phosphokinase (CPK) of 165,400 units/L, BUN 9 and Creatinine 0.98 mg/dL. The urinalysis showed only trace blood. Within hours of his admission, his sensorium returned to normal. It was then he revealed taking “many pills of Doxylamine” with oxycodone. His CPK significantly improved after supportive care with IV fluids to 16,530 units/L; a 10-fold decrease within 48 hours. He was discharged home for outpatient follow up.</p> <p>Discussion: The abuse of OTC medications augmenting the effects of opioids is growing in our communities. While many designer drugs will not test positive in standard urine drug analysis, we need to be aware and monitor for the toxicities associated with anticholinergic medications. Recognizing and treating severe rhabdomyolysis may prevent potential complications including renal failure, DIC, cardiomyopathy, respiratory failure and death from severe metabolic disturbances. A pubmed.org search of rhabdomyolysis and doxylamine failed to show a non-convulsive CPK greater than 160,000 units/L caused by a doxylamine overdose. Therefore, this would be the first reported case to our knowledge of severe rhabdomyolysis with a CPK > 160,000 units/L induced by doxylamine without seizure activity.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Aasems Jacob	Rishi Raj, MD	Monmouth Medical Center (Margaret Eng)	Cardiac Autonomic Neuropathy secondary to systemic amyloidosis	Waldenstrom's macroglobulinemia can often lead to AL amyloidosis. Cardiac manifestations of amyloidosis include heart failure, small vessel disease, conduction system disease, pericardial effusion, and thromboembolism. A 73-year-old male with long-standing Waldenstrom's macroglobulinemia complicated with systemic amyloidosis presented with a witnessed syncopal episode. He had complaints of orthostatic dizziness and palpitations for few months. Orthostatic hypotension and peripheral neuropathy were demonstrated on physical examination. EKG, 24-hour Holter monitoring and 2D echocardiogram were unremarkable. MRI of the brain ruled out stroke. Patients with amyloidosis can develop cardiovascular disease through amyloid cardiomyopathy, small vessel disease, conduction defects, pericardial effusion or autonomic denervation. After ruling out other life-threatening causes, Ewing's battery of tests were done to rule out cardiac autonomic neuropathy. Two heart rate tests and one blood pressure test were abnormal which indicated severe cardiac autonomic neuropathy. Cardiac autonomic neuropathy can mask symptoms of acute coronary syndrome and hence early diagnosis using the simple bedside maneuver is beneficial. The test is also important for prognostication. Absence of augmentation of cardiac output from inadequate autonomic stimulation will lead to postural hypotension, exercise intolerance and tachycardia. There may be no change in heart rate with Valsalva or deep breathing both of which increase parasympathetic tone. As the condition progresses, it may result in cardiac denervation which can result and in silent myocardial infarction, syncope sudden death.

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Quality Improvement/Patient Safety	Aasems Jacob	Sayee Alagusundaramoorthy, Lauren Russo, Patricia Marcelle, Violet Kramer	Monmouth Medical Center (Margaret Eng)	Simulation: The Future is Here	The education of health care providers is an integral part of patient safety. Simulation allows rehearsal of skills in a low risk environment, to engage in repeated, deliberate, and structured practice, and to be assessed and receive timely feedback. Both virtual patients and technology-enhanced simulation are consistently associated with large, statistically significant benefits in the areas of knowledge, skills, and behaviors. For direct patient effects, the benefits are smaller but still significant. Current simulation activities at Monmouth Medical Center include task training for IV and central line placement as well as critical care ultrasound, unannounced in situ mock codes, ICU team training for Rapid Response and ACLS protocols, evening multidisciplinary simulation events, and nursing competency assessment and training using SimMan, SimMan 3G, and multiple task trainers. This cross sectional study assessed differences in outcome with Simulation training. Study questionnaire was answered by 74% Nurses and 19% Medical Residents. 95% had some level of confidence in performing their professional role and 76% felt that practice would help them improve their confidence. 46% felt that simulation would be beneficial. 55% of respondents surveyed have participated in simulation and 90% of them felt their performance had changed after the simulation. 56% of respondents felt that they were better prepared to the task at hand after participating in simulation and also that it was helpful in their professional activity. Simulation of specific tasks on a monthly basis was requested by more than 50% of the respondents.

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Quality Improvement/Patient Safety	Aasems Jacob	Sayee Alagusundaramoorthy, Doantrang Du, Jack Ansell	Monmouth Medical Center (Margaret Eng)	Heparin induced thrombocytopenia diagnosis and management in a community teaching hospital	Heparin exposure can lead to the fatal complication of heparin-induced thrombocytopenia (HIT) in up to 5% of the exposed patients. Unfractionated heparin (UFH) and to a lesser extent, low molecular weight heparins (LMWH) can result in HIT. Autoantibody directed against platelet factor 4 (PF4)- heparin complex is attributed in the pathophysiology of the disease. This can result in platelet destruction and a paradoxical pro-thrombotic state in up to 50% patients resulting in venous and arterial thrombosis. This is a retrospective study assessing the appropriateness in ordering HIT antibody test based on the pretest probability in a community teaching hospital setting. Adults who were admitted in the medical and surgical units of the hospital over 2 years who had an HIT antibody test sent out were included in the study. Majority of the patients were not found to have the pretest probability calculated using 4T score which could have avoided this expensive test. Of the 104 tests sent out during the study period, 43 were positive, but only 8 were true positives as evident by Serotonin Release Assay test. This resulted in patients being treated with alternative anti-coagulation and increased the duration of hospital stay as well as the cost. Half of the patients with HIT antibody test positive were not followed up by Serotonin Release Assay thereby permanently labeling the patients as 'allergic' to heparin. Possible interventions to reduce the inappropriate diagnosis of HIT and ordering of HIT antibody assay would be education of the physicians and surgeons who mostly order these tests, requirement to document 4T score prior to ordering test, review of the lab order by technicians and checklist on the lab order screen in the electronic medical records.

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Muhammad Azharuddin	Dy Prudence, Prem Shukla, Noor Adamo, Steven J. Windsor	Monmouth Medical Center (Margaret Eng)	Chronic Lymphocytic Leukemia with Chylothorax	<p>Chylothorax is lymphatic fluid filled with fat and its digestive products are immersed by intestinal epithelium (chyle) into the pleural space. The main mechanism of chylothorax is chyle leaking into the pleural space due to thoracic duct disruption from mediastinal adenopathy. Lymphoma is the leading cause of chylothorax, followed by lung cancer. Chylothorax is diagnosed when chylomicrons are found in pleural fluid, or the triglyceride levels in the pleural fluid are >110 mg/dL. Chronic lymphocytic leukemia (CLL) is an indolent B-cell non-Hodgkin's lymphoma in the bone marrow, blood, or lymph nodes. It is a chronic lymphoproliferative disorder predominantly in the elderly. Chylothorax commonly occurs in non-Hodgkin's lymphoma, but rarely in CLL. Less than ten cases of CLL-induced chylothorax have been previously reported.</p> <p>An 85 year old female with a history of hypertension, hyperlipidemia, and chronic lymphocytic leukemia (treated by chemotherapy cytoxan, vincristine, obinutuzumab, and prednisone) came with shortness of breath on both exertion and rest with orthopnea and bilateral pedal edema for two weeks. On examination, vitals were normal and she was afebrile. Her chest exam revealed dullness to percussion on bilateral chest with decreased breathing sound. Chest X-ray showed bilateral large pleural effusion. Trials of furosemide were unsuccessful. Echocardiogram confirmed absence of systolic or diastolic dysfunction. Computed tomography (CT) chest displayed large bilateral pleural effusion, with significant lymphadenopathy in the chest and upper abdomen suggestive of lymphoma. CT-guided thoracentesis showed chylous appearing pleural fluid with alkalotic pH of 7.73, lactate-dehydrogenase 128 IU/L, protein 4.3 g/dL, albumin 2.9 g/dL, triglyceride 345 mg/dL, and no malignant cells. Pleural fluid analysis lead to diagnosis of chylothorax. The patient's shortness of breath markedly improved and thoracostomy tube was removed before she was discharged to home.</p> <p>The etiology of chylothorax is divided into four groups: tumor, trauma, idiopathic and miscellaneous. Dyspnea and chest discomfort are the principal complaint. CLL commonly manifests as lymphadenopathy, splenomegaly, hepatomegaly, skin lesions and membranoproliferative glomerulonephritis, sometimes with chylous and hemorrhagic ascites, portal hypertension, and rarely with chylorthorax. CLL-induced chylothorax can be treated initially with pleural drainage and total parenteral nutrition. Treatment options include mediastinal irradiation followed by talc pleurodesis of the pleural space, or surgery through thoracic duct ligation with/without pleurodesis. With mediastinal adenopathy, either chemotherapy or radiotherapy can be done. Thoracic duct ligation is performed in refractory cases, either in the thorax or abdomen, or to create a pleuroperitoneal shunt.</p> <p>CLL-induced chylothorax causes significant increase in morbidity and mortality, since pleural effusion is difficult to maintain despite treatment measures. Repetitive thoracocenteses can be safe with bridging until definitive surgical ligation of the thoracic duct. Clearly, each case of chylothorax must be evaluated on its own to implement a reasonable and effective treatment regimen.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Muhammad Azharuddin	Samrah Zaigham, Wael Ghali	Monmouth Medical Center (Margaret Eng)	Statin-Induced Necrotizing Autoimmune Myopathy	<p>A statin-induced necrotizing myositis (myopathy) is increasingly being recognized. More recently, a rare entity named statin-induced necrotizing autoimmune myopathy (SINAM) was characterized. Symptoms related to statin-use range widely, from common non-specific myalgias to necrotizing myositis, to rare rhabdomyolysis. Risk factors for statin-induced myotoxicity include older age, Asian descent, heavy exercise, excess alcohol consumption, vitamin-D deficiency, diabetes, and thyroid dysfunction. The higher the dose of statin, the more likely myotoxicity occurs.</p> <p>A 57 year old male with a history of diabetes and hyperlipidemia (recently starting on atorvastatin) was hospitalized with progressive weakness of lower extremities for four days, which became so severe he could not walk. On examination, he had symmetric muscle weakness on lower extremities bilaterally. Laboratory studies initially showed creatinine-phosphokinase (CK) of 7061 units/L, which increased to 132000. The patient underwent biopsy of the left quadriceps, which displayed scattered necrotic fibers. Computed tomography of chest, abdomen, and pelvis was unremarkable for malignancy, and magnetic resonance imaging of thoracic/lumbar spine was normal. Diagnosis of statin-associated necrotizing myopathy was made given his history of statin exposure, seropositive anti-HMG-CoA antibodies, and lack of evidence of connective tissue disease, active viral infection, or malignancy. Prednisone 60mg daily was administered with intravenous immunoglobulin 2gm/kg for 5 days. His CK level lowered to 621units/L, and his lower extremity strength returned to normal within a month.</p> <p>Statin-associated myotoxicity can be classified into toxic and autoimmune forms. Early diagnosis of drug-induced myopathy is crucial, since drug withdrawal can lead to full recovery. SINAM is usually categorized by a rapid onset of severe proximal weakness, and CK levels above 6000 IU/L with positive anti-HMG-CoA antibodies. Muscle biopsies can show muscle fiber necrosis, deterioration, and phagocytic penetration. In some cases, lipid filled vacuoles and ragged red fibers were observed.</p> <p>Statin-induced autoimmune myositis is classified in the criteria for adult dermatomyositis and polymyositis, which includes symmetrical muscle weakness, proximal muscle involvement, and elevated serum CK levels. Electromyography is typically normal in SINAM and diagnosis is through detecting anti-HMG-CoA antibodies. Suggested mechanisms include isoprenoid depletion, lower sarcolemmal membrane cholesterol, blockage of ubiquinone or coenzyme Q10 synthesis, or disturbed calcium metabolism.</p> <p>SINAM is treated by discontinuing the offending drug, and performing immunosuppressive therapy. Prednisone is the first-line treatment, and secondary agents include azathioprine, methotrexate, intravenous immunoglobulin, and rituximab. Other treatment options are non-statin lipid lowering agents and modifying lifestyle including stopping smoking, reducing weight, or modifying diet. SINAM is a rare and severe form of muscle toxicity is linked with statin usage. Once diagnosed, early immunosuppressive therapy should be initiated to improve patient outcome. In addition, further study should be done on effective immunosuppressive therapy.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Mridul Gupta	Annam Radhika, Eng Margaret	Monmouth Medical Center (Margaret Eng)	Septic Thrombophlebitis Of Internal Jugular Vein With Septic Lung Emboli As Rare Complication Of Acute Tonsillitis	<p>INTRODUCTION: Lemierre’s syndrome is described as acute oropharyngeal infection with secondary thrombophlebitis of internal jugular vein complicated with metastatic infections. It is also known by the terms such as postanginal septicemia and necrobacillosis. This entity was much more common in pre-antibiotic era and was associated with rapidly fatal disease course within 7 to 15 days. Affected individuals are usually previously young adults. Causative organisms are usually oropharyngeal flora, most common being anaerobe Fusobacterium necrophorum. This infection responds very well to intravenous antibiotics. Treatment regimens also depend upon the pattern of the metastatic infections.</p> <p>CASE DESCRIPTION: A 22-year-old African American male presented to the emergency department with three-day history of fever and sore throat. CT scan of neck with contrast showed acute tonsillitis and he was discharged from emergency department on co-amoxiclav and oral steroids for acute tonsillitis. Patient was called back two days later after his blood cultures showed growth for gram-negative anaerobic bacteria which were later identified as Porphyromonas asaccharolytica. Patient had not taken any medications in these intervening two days. He was started on intravenous ampicillin-sulbactam at the time of admission. Repeat CT Neck with contrast showed rim-enhancing left peritonsillar collections with adjacent thrombophlebitis. Also, multiple new patchy cavitary nodules of the lung suspicious for septic emboli were identified. Diagnosis of Lemierre’s syndrome was made based on radiological findings and metronidazole was added to the treatment regimen. Aspiration of the abscess through multiple passes with 23-gauge needle were unsuccessful. Patient also received anticoagulation with warfarin. Hospital stay was complicated with oral candidiasis, pneumonia, pleural effusion and thrombocytosis. Patient was discharged to home with peripherally inserted central line to complete 5 weeks of antibiotic therapy. Oral anticoagulation was given for three months.</p> <p>DISCUSSION: Lemierre’s syndrome was first reported by Courmont and Cade more than 100 years ago. More detailed description of this syndrome was given by Lemierre in 1938. With use of antibiotics, incidence of this infection has reduced as compared to the pre-antibiotic era. Although clinical picture of this syndrome is characteristic, but lack of awareness among clinicians can lead to delay in initiation of appropriate anti microbial treatment leading to increased morbidity. Common sites for metastatic infections include lungs, bones and joints. CT of neck with contrast is the investigation of choice.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Matthew Norris	Sneha Shredtha	Other	Hypercalcemia in end stage renal failure, a novel finding of Cryptococcus laurentii	<p>Introduction: This case pertains to a woman with end stage renal disease who presented with acute mental status change, diffuse lymphadenopathy, and hypercalcemia. Lymph node biopsy showed non-necrotizing granulomatous lymphadenitis that cultured Cryptococcus laurentii. Although symptoms persisted after correcting electrolyte imbalances, they resolved after three weeks of antifungal therapy. C. laurentii is a lesser known non-neoformans cryptococcus that until recently was considered saprophytic and has been increasingly reported in the literature. Although three previous cases have been reported in patients receiving peritoneal dialysis, we believe this to be the first case associated with hemodialysis.</p> <p>Case Presentation: A 49-year-old African American female with 14-year history of end stage renal disease presented with a three day history of fluctuating altered mental status and low grade fever. History was negative for weight loss, sick contacts, travel, animal exposure, or use of peritoneal dialysis; history was significant for hypertension and cerebrovascular accident one month prior linked to lupus anticoagulant. Patient denied history of smoking, alcohol abuse, and recreational drug use.</p> <p>Her other vitals at admission were normal with the exception of blood pressure measured at 179/90. At admission, the patient was alert and oriented with no new neurological deficits; the only positive finding on exam was axillary lymphadenopathy. CBC showed anemia with mild leukocytosis and HIV screenings were negative. CMP showed calcium 13.5, phosphorous 6.0, sodium 132, potassium 3.1, chloride 86 with other values within normal limits. Bacterial cultures, head CT, X-ray, and EKG showed no relevant findings. However, chest CT showed multiple prominent mediastinal and axillary lymph nodes.</p> <p>Further workup showed PTH was 42.2 whereas PTH-rP and total vitamin D 1,25(OH)₂ were elevated at 65 and 252 respectively. Quantiferon TB gold and flow cytometry were negative. Also, serum ACE and complement levels were within normal limits. Lymph node biopsy showed non-necrotizing granulomatous lymphadenitis that revealed Cryptococcus laurentii during culture. After three weeks of antifungal therapy and resolution of altered mental status, she was discharged back to extended care on antifungal therapy with scheduled follow-up.</p> <p>Discussion: This patient experienced fluctuating mental status that progressively worsened over a one month span. Many differentials were ruled out at this time. One consideration in the setting of diffuse lymphadenopathy, inappropriately high vitamin D 1,25(OH)₂, and suppressed PTH levels (usually high in long-term ESRD patients) was granulomatous disease – which we found. Although we cannot confirm hemodialysis as the vector of transmission, symptom resolution after three weeks of aggressive antifungal therapy supports classifying Cryptococcus laurentii as a pathogen. This case is also the first to link C. laurentii to hemodialysis.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Waqas Jehangir	Bertrand Behm Lopez, MD; Zankhana Mehta, MD; Mellar Davis, MD, FCCP, FAAHPM	Other	Haloperidol as A Treatment of Opioid Induced Hyperalgesia.	<p>Introduction: Opioid-induced hyperalgesia (OIH) is a paradoxical hypersensitization caused by increasing opioid doses. It is often missed or mistaken for progressive disease. The mechanism involves multiple receptors and ion channels. Sigma-1 receptors (S-1R), may be activated by opioids activate in turn various kinases, modulates multiple ion channels, ligand activated ion channels and voltage-gated ion channels and alters monoamine neurotransmission and dampens opioid receptors G-protein activation. This patient history illustrates the challenges to recognition of OIH and suggests a role of haloperidol, potent sigma-1 receptor blocker, in management.</p> <p>Case Description: A 67 years old male with a bone marrow biopsy proven myelofibrosis and myeloproliferative neoplasm was started on azacitidine. After the first does he developed weakness, fever and pancytopenia. He was admitted with a febrile illness and placed on antibiotics. His pancytopenia persisted despite azacitidine. A repeat bone marrow biopsy revealed transformation to AML. He was started in fludarabine, cytarabine and G-CSF which he did not tolerate. Subsequently, the patient's family opted for hospice and comfort measures. He was discharged to a nursing home on hospice care with a hydromorphone PCA 0.3 mg/hr basal and 0.3mg Q30min. During his stay he developed worsening pain which intensified with an increased to 0.6mg/hr basal and PCA bolus 0.6mg Q30min. His pain worsened with 1mg/hr basal and 0.6mg Q10min PRN. Once OIH was considered, he was started on haloperidol 1 mg Q1H PRN which after the first dose relieved his pain.</p> <p>Discussion: S-1R are expressed in the CNS and peripheral nervous system on mitochondrion associated membranes of the endoplasmic reticulum. Haloperidol is the only commercial agent which is a potent S-1R blocker. S-1R activation is coupled to pain facilitation and inhibition of opioid antinociception, antagonists inhibit pain hypersensitivity and "releases the brake" enabling opioids enhanced antinociceptive effects, both at the central nervous system and at the periphery. This patient history illustrates the balance between treating pain with opioids and the adverse effect, opioids induced hyperalgesia. Haloperidol rebalances the equation.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Shaheryar Jafri	Ling Zhang MD, George Everett MD.	Other	PAGET DISEASE OF BONE LEADING TO HYPERCALCEMIA	<p>Introduction: Paget disease of bone is common in old population >55 years.. It is characterized by abnormality in osteoclasts causing increased bone turnover and remodeling. Most often the disease is asymptomatic but sometimes symptoms are mainly due to bone overgrowth causing pain and also sometimes fracture or nerve impingement. Laboratory findings include elevated levels of serum alkaline phosphatase. In rare instances if the people with Paget disease are immobilized due to any reason, they can present with hypercalcemia which can be symptomatic. Common symptoms of hypercalcemia include constipation, polyuria and neuropsychiatric disturbances including anxiety, depression and cognitive dysfunction. We describe an interesting case of Paget disease presenting as hypercalcemia.</p> <p>Case presentation: A 76 years old male with dementia and past history of stage-1 adenocarcinoma of lung status post lobectomy who lives in nursing home was transferred to hospital because of generalized weakness, change in mental status from baseline, increased urinary frequency and constipation for few weeks. It was discovered that he had history of recurrent mechanical falls at nursing home and he remained bedbound most of the times for last few months. Although he had previous history of adenocarcinoma of lung but that was non-metastatic as evident on PET and multiple CT scans and had successful resection. Initial physical examination was remarkable for generalized tenderness mainly in right arm. Initial laboratory findings showed Alkaline phosphatase level elevated at , and serum calcium of 12.2mg/dl and serum albumin of 3.5 g/dl. X-RAY of the right shoulder demonstrated cortical and trabecular thickening in humeral head and proximal shaft suggestive of Paget disease of bone. Workup for hypercalcemia was done including serum intact parathyroid hormone (PTH) level, PTH-related peptide (PTHrp), Vitamin D level, Serum protein electrophoresis (SPEP) and thyroid stimulating hormone (TSH), all of these came back normal. Meanwhile he was managed with isotonic saline hydration and one dose of zolindronic acid. His calcium level gradually improved and his symptoms resolved.</p> <p>Discussion: Hypercalcemia is a common clinical problem which can be due to variety of reasons and most common of those is hyperparathyroidism and malignancy. Other causes include various inherited and acquired conditions including Familial hypocalciuric hypercalcemia, Vitamin D intoxication, Chronic granulomatous disease, certain medications including Thiazide, Lithium, hyperthyroidism, milk alkali syndrome and also immobilization. Paget's disease of bone is not usually associated with hypercalcemia but if those patients become immobilized due to any reason they have increased likelihood to develop symptomatic hypercalcemia which require appropriate management including intravenous hydration and bisphosphonates. Diagnosis of paget disease is manily by characterstic radiographic findings on X-RAY and serum alkaline phosphatase is elevated in most of the patients. Treatment is mainly with oral bisphosphonates but sometimes intravenous zolindronic acid is used.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Caren Chapinski		Overlook Medical Center (Jeff Brensilver)	Neurocysticercosis: A communicable disease?	<p>Introduction: Our understanding of Neurocysticercosis (NCC) has increased over the years as the disease has gained global attention for being the most common cause of acquired epilepsy worldwide. However, there still remains controversy over the mechanisms of disease acquisition.</p> <p>Case: A 24 year old previously healthy female who recently immigrated from Guatemala three years ago with no history of eating undercooked pork presented with headache for two weeks. She described her headaches as bilateral frontotemporal, intermittent, throbbing, with moderate to severe intensity. CT of the head revealed 1.3 cm cyst within the deep left cerebellar hemisphere with evidence of ependymitis. Enzyme-linked immunoelectrotransfer blot (EITB) was positive for anti-cysticercal antibodies. She was diagnosed with NCC thought to secondary to exposure to a tapeworm human carrier. She was started on Albendazole 400mg PO BID and Dexamethasone 2mg PO q6hrs. She was discharged home with Albendazole for 2 more days for a total of 14 days of treatment with a tapering dose of dexamethasone.</p> <p>Discussion: NCC is the most common human helminthic disease of the CNS and a common cause of acquired epilepsy worldwide. It is known that NCC is prevalent in Latin America, sub-saharan Africa, and southern Asia although recently NCC has also been documented in nonendemic countries including the United States.</p> <p>It is common knowledge that the pork tapeworm, <i>Taenia solium</i>, is the main culprit for NCC. Humans become infected with adult tapeworm after they ingest larval cysts contained within undercooked pork. Adult tapeworm produce thousands of eggs. Eggs may appear in human stools within 2-3 months after ingestion of larvae. However, it is the ingestion of <i>T. solium</i> eggs, usually via fecal-oral transmission from an adult tapeworm human carrier that leads to NCC.</p> <p>We speculate that this patient may have developed NCC from initial exposure to an asymptomatic carrier in Guatemala via fecal-oral transmission prior to immigrating to the United States. This patient confirmed that she ate cooked meat but did not eat pork making it less likely that she was initially an adult tapeworm carrier herself but rather was exposed to an asymptomatic carrier. Onset of symptoms of NCC are often delayed with most presenting with symptoms within 5 years but latencies up to 30 years have been reported.</p> <p>Early diagnosis of NCC is important because medical treatment with antiparasitic agents such as albendazole or praziquantel often lead to cyst resolution. Serology with EITB is the most accurate serological test.</p> <p>Fortunately, cases with single parenchymal lesions like this patient generally have a good prognosis with lesions disappearing within 6 months in over 60% cases. If reimaging in six months shows complete resolution of the patient's cystic lesion the patient will no longer need continued antiparasitic therapy.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ayla Gordon	Gina LaCapra, Roberto Roberti	Overlook Medical Center (Jeff Brensilver)	DKA-INDUCED TAKOTSUBO CARDIOMYOPATHY IN PATIENT WITH KNOWN HOCM	<p>The first published case of DKA-induced Takotsubo Cardiomyopathy (TC) was in 2009. Our patient is the 1st reported case of DKA induced TC in a patient with known HOCM in the United States. In the literature, there are only two examples linking DKA to TTC. However, this case report focuses on the biochemical and physiological causes of TC in a patient with known HOCM and new-onset DKA.</p> <p>The most common documented cause of TC is a neurohormonal release of catecholamines. In this clinical vignette of a man presenting with syncope, we describe DKA as the physiological stressor leading to TC. Upon presentation of this patient, initial labs revealed a glucose of 526 and blood gas ph of 7.12. Troponin: 13.8 with EKG showing ST elevations in leads V3-V6. Patient was admitted with st-elevation MI and concomitant DKA. Emergent coronary angiography demonstrated normal coronaries with anterolateral, apical and inferoapical dyskinesis and basal anterior and basal inferior wall hyperkinesis, LVEF was 20%. Clinical picture suggested TC. Transthoracic echo confirmed TC. DKA was controlled by day 4 of hospital stay and repeat echo on day 5 showed documented resolution of apical ballooning. The correlation is explained physiologically: DKA not only increases serum catecholamines, but the metabolic acidosis prevents the healthy myocyte's step-wise chemical channel processes; most notably the sarcoplasmic reticulum's ability to release Ca⁺⁺.</p> <p>Typical treatment of Takotsubo's Cardiomyopathy is supportive in nature and typical resolution is seen within 2 months. However, when there is a physiological underlying cause, the aim is to treat the cause first. In this case, DKA-induced physiological stress on the myocardium lead to apical stunning. When DKA was treated and glucose levels were brought within normal range, repeat ECHO revealed an improved EF and normal ventricular motion, and therefore, overall resolution of TC.</p> <p>TC in previously diagnosed HOCM poses particular complications. With the above patient's baseline outflow tract obstruction due to septal hypertrophy, the acute presentation of dynamic LV outflow tract obstruction and reduced EF due to TC resulted in transient drop in brain perfusion and therefore syncope.</p>

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Clinical Vignette	SO CHUNG		Overlook Medical Center (Jeff Brensilver)	The case of E coli	<p>Introduction: Bloody diarrhea is rare disease, which account for only 3% of stool samples. However, it should receive emergent medical attention, because it often signifies serious or life threatening disorders. The possible causes of bloody diarrhea is very broad. 24.6% of visibly bloody sample were identified the pathogens, and E.coli O157 is most common pathogen. We present a case of a 20 year old female with history of uveitis and positive HLA B27 who presented with bloody diarrhea and abdominal pain, and was diagnosed with E.coli O157:H7 by stool culture.</p> <p>Case Presentation: A 20 year old woman presented to the emergency room with abdominal pain and bloody diarrhea. She had a history of uveitis at the age of 14, with positive HLA B27. Symptom started with bloating and nausea 5 days prior to admission, followed by watery diarrhea and abdominal pain then it progressed to bloody diarrhea. She denied fever, joint pain, vision change, weight loss, tenesmus. On physical exam, she was afebrile, and her vital sign was stable. Her abdomen was soft, not tender, and bowel sound was normal. X-ray of abdomen and chest showed clear lung and no sign of intestinal obstruction. White blood cell count was 10,210, Hemoglobin was 13.4, kidney function was normal. ANCA and Gliadin antibody were negative. CT abdomen and pelvis revealed marked colonic wall thickening from rectum to cecum. Initial differential diagnosis included infectious diarrhea and ulcerative colitis in light of hemorrhagic diarrhea, CT abdomen and pelvis finding, and history of uveitis with positive HLA B27. Patient received one dose of ciprofloxacin and metronidazole at ER, but discontinued to avoid subsequent increasing risk of HUS in case of EHEC infection. Colonoscopy was considered to rule out inflammatory bowel disease, but deferred in setting of acute infectious or inflammatory process. Patient was treated with supportive management as IV hydration and pain medication. On day four of admission, Escherichia coli O157:H7 was isolated from her stool. During her hospitalization, she does not develop the sign of HUS, made complete recovery, then was discharged on day 4 of admission.</p> <p>Discussion: E.coli O157:H7 is most common and important strain of enterohemorrhagic e. coli. However the clinical presentation of E.coli O157 can be confused with other disease such as Ulcerative colitis, Ischemic colitis, Pseudomembranous colitis, Intussusception. In our case, along with CT finding with continuous thickening from rectum to cecum, the history of uveitis and HLA b27 positivity raise possible diagnosis of Ulcerative colitis. In E,coli O157:H7 infection, antibiotic therapy is not beneficial, it elongate the duration of symptom and precipitate progression to Hemolytic Uremic Syndrome. Our patient received empirical antibiotics, however in concerns of HUS development, we stopped antibiotic after one dose.</p>

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Clinical Vignette	Lisbet Suarez		Overlook Medical Center (Jeff Brensilver)	When more than a stone remains: Complications of a retained stent	<p>Case: A 32-year old Spanish speaking man presented to a nearby ED with acute complaints of right upper quadrant abdominal pain and fever. Two years prior he was seen in our hospital with similar complaints. Additional findings included leukocytosis, high AST and ALT, normal amylase and lipase. An abdominal ultrasound showed a distended gallbladder with stones and a dilated common bile duct (CBD). His MRCP was positive for 8-mm obstructing distal CBD stone. He was treated with intravenous antibiotics, underwent ERCP with removal of stone in the CBD, sphincterotomy and placement of a stent; followed by laparoscopic cholecystectomy, which showed an acutely inflamed gall bladder with temporary JP drain placement. He ultimately was discharged on oral antibiotics with instructions to follow up with GI for planned stent removal. The patient was lost to follow up. At this time, in addition to above complaints, he had intractable nausea and vomiting, was tachycardic, with leukocytosis, high AST, ALT, total bilirubin, and lactate. Abdominal ultrasound demonstrated dilated CBD. He was transferred to our hospital 8 hours after initial evaluation, and admitted to the teaching service. He was seen by the resident four hours after arrival, who noted scleral icterus. The case was presented to on-call gastroenterologist; in light of suspected cholangitis, he was continued on IV antibiotics, hydration and narcotics. Overnight he had fevers with rising liver function tests and lactate. Next morning an ERCP was pertinent for retained stent that was removed along with CBD stones and copious pus. His blood grew <i>Enterococcus faecalis</i>. He had improvement of liver function and was discharged home on oral antibiotics with a follow up appointment with medical team.</p> <p>Discussion: Our case demonstrates an example of a preventable adverse outcome, caused by a combination of system and medical errors. The patient needed his stent removal much earlier, however, the dependence on the specialist service for management of his initial illness may have lifted the responsibility off the hospitalist to enforce the importance of his follow up¹. Also a non-effective way of monitoring his outpatient continuity accentuated this failure. On his second admission, there was weak transition of care; initiated by lack of identification of disease severity both from the local ED (e.g no knowledge of prior history), delayed evaluation by resident, and unclear communication with on-call gastroenterologist that his acute cholangitis was caused by retained stent. The development of de novo choledocholithiasis in the setting of retained stents has been discussed in the literature and this knowledge should have been a driving force in his acute management. A good model of patient care with clear communication across professions, explicit patient instructions, and a motivation for continuous learning is one that will minimize errors in medical practice.</p>

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Clinical Vignette	Mohammad Ansari	Sabrina Arshed MD, Abdalla Yousif MD	Raritan Bay Medical Center (Abdalla M Yousif)	REVERSIBLE COCAINE INDUCED ACUTE FULMINANT HEPATIC FAILURE	<p>About 6.4 million people in North America use cocaine, accounting for approximately 45% of its global use. In literature its effects on the cardiac, cerebrovascular, pulmonary and gastrointestinal systems are well known and reported by the medical community. We report a case of a 42 year old male with a history of polysubstance abuse who was brought by EMS to the hospital because of unusual behavior. On initial evaluation, the patient was found to be in acute liver and renal failure. He tested positive for cocaine and THC. He was initially given IV fluids for ARF, but his creatinine progressively got worse and peaked at 8 mg/dl . Dialysis was started on day 3, after which the patient’s mental status improved significantly. The patient’s LFTs progressively got worse, with AST/ALT reaching above 2000 with an INR of 5.1. On day 3 N-acetylcysteine (NAC) was started. On day 4 the patient was transferred to another facility to be managed by a hepatologist. A week later dialysis was no longer required and his liver functions had returned to normal.</p> <p>It is well established that NAC has been proven to decrease the burden of acetaminophen toxicity on the liver by replenishing glutathione which converts toxic metabolites (NAPQI) into benign products. Likewise cocaine also has toxic metabolites (norcocaine and N-hydroxynorcocaine) that cause liver damage. NAC’s role has not been well established as an antidote for non-acetaminophen drug induced (NADI) liver failure. A prospective trial showed significantly improved transplant-free survival at 3 weeks and at 1 year (52% vs. 30%) with the use of NAC for the treatment of NADI liver failure. Another trial showed that NAC was associated with a statistically significant survival benefit (47% vs. 27%). It has antioxidant properties and improves perfusion to the liver. Current guidelines do not include NAC for NADI liver failure. Many physicians will start NAC in acute liver failure because it is benign and has seen to improve patients’ outcome in NADI liver failure based on some studies. We strongly believe NAC should be a part of the treatment for any acute liver failure, at least until, drugs have been ruled out as the possible cause of the original insult.</p> <p>In conclusion, our patient with acute fulminant hepatic failure and made a full recovery. Further research needs to be done on NACs role to neutralize non-acetaminophen drug induced toxicity. NAC is a relatively benign drug which can be given empirically, as it is seen to improve outcome in such cases.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Margaret Omatsone	Kalyani Regeti, Nikunj Kumar Patel, Romana Kanta	Raritan Bay Medical Center (Abdalla M Yousif)	THE IMPACT OF HIGH DOSE FOLATE AND THIAMINE IN ACUTE ALCOHOL WITHDRAWAL IN A COMMUNITY HOSPITAL SETTING.	<p>BACKGROUND: An alcohol dependent patient is at risk of acute withdrawal syndrome (AWS) upon abrupt cessation of alcohol intake. Alcohol dependency predisposes an individual to several nutritional deficiencies, i.e. folate and thiamine deficiencies. Symptoms of AWS range from mild tremors to withdrawal seizures. In addition to relieving the immediate symptoms, supplementation with folate and thiamine replenishes deficient nutrients. The objective of this study is to determine whether administration of higher doses of folate and thiamine during an AWS episode affected patient length of stay.</p> <p>METHODS: We conducted a retrospective chart review comparing the clinical outcomes of 225 hospital admissions for AWS from January 2013-September 2015 at Raritan Bay Medical Center. The actual number of patients was 166, due to patients with multiple admissions. The population was divided into two groups: patients who received standard dose of folate(1mg) and thiamine(100mg) n=188 and patients who received high dose of folate(5mg) and thiamine(300mg) n=34. Exclusion criteria included patients who presented with/had seizures, background mental or behavioral disorders. All patients received scheduled and prn doses of chlordiazepoxide. Patients were monitored daily and discharge from hospital was based on patients being symptom free. Primary outcomes included time to symptom resolution and discharge from hospital, while secondary outcomes included variables affecting hospital stay.</p> <p>RESULTS: The mean age and standard deviation for patients on standard therapy was 50.5±10.4years and on high dose therapy mean age was 50.2±8.2years. Males constituted 74% of the population and females 26%.</p> <p>The data for length of stay was negatively skewed. The patients on standard therapy had a median length of stay of 3 days with an interquartile range of 3 (2-5days) and those on high dose therapy, the median length of stay was 2 days with interquartile range of 1 (2-3days).</p> <p>The distribution of length of stay was statistically significant between patients on standard therapy and those on high dose therapy with a P value =0.002</p> <p>Some variables were found to play a role in determining the length of stay. Linear regression of correlates showed the strongest independent predictor of increased length of stay was age at admission, followed by MCV and blood glucose . High dose folate and thiamine therapy was a significant independent predictor of decreased length of hospital stay.</p> <p>CONCLUSION: Our study demonstrated a possible correlation between administration of high dose of folate and thiamine in an acute alcohol withdrawal episode and the length of stay. Previous studies and case reports have reported the connection between micronutrient deficiencies and the severity of alcohol withdrawal. This study categorically highlights the importance and advantage of high doses of supplementation favorably impacting cost of inpatient stay and clinical outcomes.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Stephen Catalya		Raritan Bay Medical Center (Abdalla M Yousif)	A Rare Case of Pancreatic Tuberculosis	<p>Introduction: Tuberculosis remains a major public health concern, though it has reportedly shown a decline in prevalence both in the US and abroad. Unlike pulmonary tuberculosis, pancreatic tuberculosis remains exceedingly rare, accounting for less than 5% of all tuberculosis cases in the developing world (no US data is available for pancreatic tuberculosis).</p> <p>Case: Here is an 86 year old gentleman who presented with nausea, vomiting, generalized malaise and weakness for 5 days. He had a past history significant for taking cyclosporine for an unspecified skin rash for multiple months and immigrated from the Philippines as a young child. A diagnosis of renal failure was established and was treated successfully. However, a CT scan of his abdomen and pelvis done during the course of his stay showed a 2.2cm x 3.8cm cystic mass in the tail of the pancreas. Initially this cystic mass was perceived to be malignant; however, a biopsy done as an outpatient showed no malignant cells. Ascitic fluid sampled from the site was negative for acid fast bacilli; however, polymerase chain reaction (a highly specific assay that has positive results even when AFB and cultures are negative) revealed the presence of tuberculosis protein. This led instead to a diagnosis of pancreatic tuberculosis. He was started on rifampin, isoniazid, pyrazinamide, and ethambutol and was reportedly compliant as per patient and family. A pleural effusion developed leading to readmission. He went into respiratory failure and was subsequently intubated. Due to worsening renal and subsequent liver failure, anti-TB medications were stopped. Care after 2 weeks in the hospital was withdrawn with the patient subsequently passing away.</p> <p>Discussion: As a clinical entity pancreatic tuberculosis is very rare and the presentation here is atypical compared to the published data. Even in countries endemic with tuberculosis, isolated pancreatic tuberculosis accounts for less than 5% of total cases. A review of case reports shows a dearth of cases in the US, and one study reviewing pancreatic tuberculosis found that such cases are usually young adults who are recent immigrants from countries endemic with tuberculosis. Furthermore, published reports show that the pancreatic masses found to be tuberculous are located in the head, not in the tail of the pancreas.</p> <p>Conclusion: This patient was born in a country endemic with tuberculosis but moved to the US at a young age and then developed symptoms only decades after his last known exposure. The lesion was also located in a location not documented in the literature, being found in the tail. This case does not fit the typical profile outlined in that study and other published case reports raising the absolute need to be aware of both from epidemiological and clinical standpoints.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ali Nadhim	Kanwal Farooq , Bushra Komal , Bhaveshkumar Garsondiya	Raritan Bay Medical Center (Abdalla M Yousif)	Pre-workout supplements safety concern : A case of jaw dyskinesia associated with pre-workout supplements	<p>Introduction: Dietary supplements are used by approximately half of the US population. Most of these supplements are obtained without a prescription, which raises medical concerns regarding safety as these products do not require FDA approval. One of the commonly used supplements are pre-workout supplements which are commonly consumed by athletes to burn fat/increase muscle mass.</p> <p>Case report: A 31 year old male with past medical history only for childhood asthma was brought to the emergency department for involuntary jaw contractions and incoherent speech. He was free of symptoms previously and denied prior episodes. He reported sudden onset intermittent jaw spasms lasting several minutes, with complete recovery of muscular activity. Further questioning revealed that he had been consuming a pre-workout dietary supplement called "C4" at least 4 times/week for more than a year, but had stopped taking the supplement three weeks ago. On physical examination, patient vitals were within normal limits. The Physical exam was significant for jaw spasm and inability to speak fluently. Head CT scan, brain MRI and labs were unremarkable except urine drug screen which was significant for cocaine and marijuana. During hospital course, the patient had two more episodes of jaw muscular contractions, during each episode patient was given lorazepam and diphenhydramine to relieve the contractions.</p> <p>Discussion: Upon further researching the supplement in question, there are two ingredients of interest, the Velvet Bean extract (mucuna pruriens) and N-acetyl-L-tyrosine. The Velvet Bean extract (mucuna pruriens) has been proven through many studies to be as effective as Levodopa in treating and controlling Parkinson disease, while (N-acetyl-L-Tyrosine) is converted into dopamine during endogenous dopamine synthesis process. Both ingredients led to increased dopamine levels, which subsequently decreased when the patient stopped consuming the pre-workout supplement, thus resulting in these motor symptoms. Dyskinesia is rarely described as a result of cocaine use. In patient with parkinsonian symptoms, the use of inhaled cocaine has been reported to ameliorate symptoms without triggering dyskinesia. Chronic cocaine use has been described as a risk factor for acute dystonia in patients using neuroleptic agents, as it leads to an increase in dopamine receptors expression. This increase in expression may act as a synergistic with conditions that decrease dopamine synthesis therefore producing motor symptoms.</p> <p>Conclusion: As most dietary supplements have no FDA approval label, it's very important to list and report such side effects, as well as raise the awareness to the population regarding efficacy and safety of such products. Physicians should be aware of any supplements consumed by their patients and they should advise and educate their patients regarding possible side effects such as jaw dyskinesia.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Pye Oo		Raritan Bay Medical Center (Abdalla M Yousif)	Underlying Osteoarthritis As A Predisposing Condition For Acute Inflammatory Attack In A Patient Anti-coagulated with Apixaban	<p>Background: NOACs have been used increasingly over warfarin for equal efficacy, fewer serious bleeding complications, convenience and less interaction with food and other medications. One of the potential adverse effects which has not been reported from the patients started on Apixaban is the acute worsening of underlying joint disease.</p> <p>Case Presentation : A 78-year-old Hispanic Male with underlying asymptomatic osteoarthritis who was admitted to our hospital with the left gastrocnemius vein thrombosis subsequently confirmed by bilateral lower extremities Doppler Ultrasound was started on Apixaban. After the 4th dose of Apixaban, the patient started complaining of increasing pain and swelling of his bilateral knee joints. Physical Examination showed signs of inflammation with fluid collection in bilateral knee joints. X-ray of bilateral knee joints confirmed the presence of fluid in the joint spaces, moderate effusion on the right and small effusion on the left. Laboratory findings showed ESR 115, CRP 10.1, Uric Acid Level 7.6, Negative Rheumatoid Factor, Antinuclear antibody IgG and hepatitis panel. The synovial fluid examination showed yellow cloudy fluid with RBC 692, WBC 15750, Mononuclear Cell 15%, PMN 85% and Specific Gravity of 1.030. Pathology report of the synovial fluid showed hypo-cellular knee fluid with no polarizable crystals. Both aerobic and anaerobic joint fluid cultures were negative. After excluding the possible causes of acute inflammatory joint conditions and careful revision of newly started medications, Apixaban was discontinued and the patient was started on Warfarin. Patient was also started on prednisone 20 mg twice a day orally for 3 days for inflammation of the joints. On discharge, patient noted significant improvement in pain and swelling.</p> <p>Discussion: After exclusion of common causes, direct inactivation of factor Xa is assumed to be the cause of previously stable underlying osteoarthritis exacerbation. Acute disruption of body homeostasis mechanism in previously damaged joint and synovial microcirculation due to the absent Protein C activation by thrombin which may have led to weaken its anti-inflammatory effect on mononuclear cells and granulocytes. In addition, premature discontinuation of damage repair and restoration along with the body natural anticoagulant mechanism of thrombus resorption which hasten fluid extravasation may have played a role in the development of joint fluid accumulation.</p> <p>Conclusion: Even though the underlying mechanism is not well understood and exacerbation of underlying arthritis hasn't been reported as a possible adverse effect of using Apixaban, the presence of underlying osteoarthritis might have predisposed him to an acute inflammatory attack when started on Apixaban.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Nikunj Kumar Patel	Nikunj Kumar Patel MD, Bhavesh Kumar Garsondiya MD, Margaret Omatone MD, Jamal Ansari MD	Raritan Bay Medical Center (Abdalla M Yousif)	Intra-abdominal abscess in the setting of hypercoagulable state	<p>Introduction : Intra-abdominal abscesses are collections of pus confined within the peritoneal cavity. It can be classified as intraperitoneal, retroperitoneal or visceral. Microbiology includes mixture of aerobic and anaerobic organisms, with most common being E Coli and the anaerobe Bacteroides fragilis.</p> <p>Case Discussion: 38 year old female with a past medical history significant for hypertension and splenic infarction 2 years prior to presentation as well as Protein S deficiency presented with abdominal pain, nausea and vomiting for 2 days. She described periumbilical cramping pain associated with five episodes of bilious vomiting. CBC was significant for WBC count 16.8K per microliter, imaging studies showed 2.5 x 2.9 cm relatively low-attenuation mass with irregular peripheral enhancement located posterior-medially within right lobe of liver, suspected for primary neoplasm. Surgical pathology and cytology shows no evidence of malignant cells, rather suggestive of reactive inflammatory process. Blood cultures showed no growth, however she had persistent leukocytosis with WBC count in the range of 15-17K per microliter and she was discharged from hospital on ciprofloxacin and metronidazole for two weeks. She presented after 5 weeks with abdominal pain, vomiting and bright red blood per rectum. Abdomen-pelvis CT showed multiloculated fluid collection in the left anterior cul-de-sac and right lower quadrant intraabdominal fluid collections consistent with abscess. She underwent CT guided drainage of abscess, fluid culture was positive for Klebsiella pneumonia and Candida albicans. She was treated with Ertapenem and fluconazole; subsequent imaging showed resolution of abscess and blood cultures remained negative.</p> <p>Discussion: Hyper-coagulable states such as Protein S deficiency leading to Splenic infarction predispose to intra-abdominal abscess formation. Conditions that predispose to intra-abdominal abscess include peptic ulcer perforation, perforated appendicitis and diverticulitis. Most common routes are hematogenous spread, lymphatic spread and also via Gastrointestinal tract. Visceral intra-abdominal abscess may be hepatic or splenic in origin and predisposing factors may be traumatic, hematogenous or infarction. Splenic infarction may lead to septic emboli and subsequent formation of intra-abdominal abscess. It can also present with multiple intra-abdominal abscesses and given obscure nature of disease process, it gets missed until at a later stage and hence delayed therapy. It is also important to note the most common organisms shown are Staphylococci, Streptococci, anaerobes and aerobic gram negative bacilli. This case can increase physician awareness with regard to relationship between hypercoagulable state and multiple intra-abdominal abscesses.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Zar Tun	Sameera Syed, Nivarthi Bharadwaj, Claribel Cruz	Raritan Bay Medical Center (Abdalla M Yousif)	Recurrent Meningitis in Adults	<p>Introduction: Community-acquired recurrent bacterial meningitis in adults is a relatively rare disease. It is more common in pediatric population and associated with anatomical abnormalities and immunodeficiency.</p> <p>Case: We report a case of a 42 year old female, with past medical history significant for mild developmental retardation, streptococcus pneumoniae meningitis, and recurrent sinus infection who was brought to emergency department by family members with the complaint of headache, nausea, vomiting, confusion, lethargy, fever, ear pain and neck pain. On examination, she was obtunded with a GCS of 6, tachycardic and had nuchal rigidity. Lumbar puncture was done, CSF culture showed streptococcus pneumoniae. Brain and sinuses MRI showed left posterior ethmoid and sphenoid sinus opacification and mucosal thickening. Facial MRI with and without contrast focus on anterior cranial fossa skull base showed a fluid signal in the left olfactory recess. In the presence of recurrent meningitis and sinonasal inflammatory disease, a fistula with intermittent leakage was possible and was not excluded. CT cisternography and evaluation of nasal secretion for the presence of beta 2 transferrin and beta trace protein were ordered but not done. She was sent to ENT specialist and neurosurgery for further evaluation. For prevention of recurrent meningitis, patient received a vaccination of meningococcus serotype B, haemophilus influenza and pneumococcus. Patient was lost to follow up.</p> <p>Discussion: Recurrent meningitis can occur with bacterial, viral and noninfectious causes. It is more common in the pediatric population who can have congenital bony defects in the cranial vault, basilar skull, ear canal, cribriform plate, and temporal bone. In contrast, the adult population, usually have a post-traumatic or post-surgical defect, which leads to a CSF leak or rhinorrhea. High resolution CT scan can help to detect those defects. Recurrent meningitis is also one of the complications of indwelling devices, like ventricular shunts or cochlear implants. On the other hand, immunocompromised status such as immunoglobulin deficiency, complement deficiency, agammaglobulinemia and abnormalities in opsonizing antibodies can increase the risk of having infections. Neisseria Meningitides meningitis is known as having association with deficiency of terminal complement C5, C6, C7, C8 and C9.</p> <p>Conclusion: Recurrent meningitis is rare in the adult population compared to the pediatric population. Treating patient effectively is vital but gaining an understanding of the causes or risk factors of recurrent meningitis can be immensely beneficial to patients whose defect is correctable by surgery. High resolution CT scan of specific area of skull can help with diagnosing the defect and reduce and prevent the recurrence of meningitis.</p>

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Clinical Vignette	Jaspreet Kaur	Kanwal Farooq, MD; Bushra Komal, MD	Raritan Bay Medical Center (Abdalla M Yousif)	VIRAL MYOPERICARDITIS MASQUERADING AS INFERIOR WALL STEMI: AN INTERESTING CASE REPORT	<p>INTRODUCTION: Myopericarditis usually causes diffuse ST elevation and PR depression which is later followed by normalization of ST and PR segments, and then diffuse T wave inversions. We present to you an interesting case report of viral myopericarditis masquerading as inferior wall STEMI.</p> <p>CASE PRESENTATION: 46 year old male with no past medical history was admitted to the hospital with pleuritic, retrosternal chest pain for one day. It was associated with shortness of breath, cough and flu-like symptoms. Ten days prior to admission, he had returned from Cuba.</p> <p>On exam, the patient looked moderately distressed due to chest pain and labored breathing. Vitals were normal. Physical examination was positive for muffled S1 and S2 and some wheezing on end expiration. EKG showed ST elevation in Leads II, III and aVF. Troponin I was 20.42 µg/L and CK-MB was 124.8 IU/L. The rest of the labs and chest X-Ray were unremarkable.</p> <p>Patient underwent emergent cardiac catheterization and was found to have normal coronary arteries, mid to apical Inferolateral wall hypokinesis and mildly impaired left ventricular ejection fraction (EF) of 45-50%, without evidence of pericardial effusion.</p> <p>Subsequently patient was worked up for non- ischemic causes of myopericarditis, and was found to have Dengue IgM and IgG antibody positive with titers being 3.43 mIU/mL and 8.26 mIU/mL (normal range <1.65 mIU/mL). Coxsackie 1 and 5 were 1:32 (normal 1:8). Confirming it was viral etiology. He was managed conservatively with ibuprofen and his chest pain resolved. With time, he improved clinically and troponins also decreased. On follow up, his echocardiogram showed an EF >55%.</p> <p>DISCUSSION: Myopericarditis is inflammation of mostly myocardium and some pericardial tissue. It is usually caused by viruses, most notably coxsackieviruses, adenoviruses, herpes, echovirus, influenza and parvovirus B19. They cause either direct cytolytic or cytotoxic damage. Clinical presentation depends upon degree of tissue involvement. Some are subclinical and most present with flu like symptoms. In very few cases it can present mimicking myocardial infarction, especially in younger patients, as it did in our patient. MRI and endomyocardial biopsy provides the definitive diagnosis. Treatment is with NSAIDs and colchicine. With time, myocardial damage is usually reversed.</p> <p>CONCLUSION: Myopericarditis is rare cause of focal ST elevation and should be considered in young patients especially with flu like illness.</p>

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Clinical Vignette	Kanwal Farooq	Komal,Bushra MD;Nadhim,Ali MD;Kaur,Jaspreet MD.	Raritan Bay Medical Center (Abdalla M Yousif)	Gastrointestinal Metastases From Breast Cancer: Raising Awareness:A Case Report	<p>Introduction: Breast cancer metastasis to the stomach is rare as the usual sites are lung, bone, liver and brain. It is important to distinguish breast cancer metastasis to the stomach from primary gastric cancer for appropriate treat-ment. Among the breast carcinomas, inva-sive lobular carcinoma has a higher tendency to metastasize to GI, mixed type and infil-trating ductal cancer are rarely the source for GI metastasis.</p> <p>Case presentation: 76 yr. old female with past medical histo-ry of breast cancer s/p modified radical mastectomy and chemotherapy, CAD, DM, HTN, and dyslipidemia presented with c/o generalized weakness and lack of energy. Her initial lab values showed Hb 8.5, Hct 26.5, WBC 8.8, platelet 50,000, BUN 38, Cr. 1.3, Ca 12.4. She was admitted for hypercalcemia and started on IV fluid and Aredia. During the admission, she had an episode of hematemesis on floor so she was transferred to critical care unit and was stabilized with multiple blood transfusions and IV protonix. She underwent EGD and found to have severe gastritis and multiple gastric and duodenal ulcers. Biopsy showed metastatic adenocarcinoma with breast primary, positive for CK 7 and ER and negative for chromogranin and synaptophysin. A decision was made to proceed with radiation therapy as pt. was too week to undergo chemotherapy now.</p> <p>Discussion: GI metastasis is an underdiagnosed complication of breast cancer. The incidence of gastric lesions is about 6-18% versus 8-12% in colon and rectum. The time interval between breast cancer and the diagnosis of GI metastasis is variable ranging from few months to several years. The most useful approach for correct diagnosis is immunohistochemistry. The common positive immunohistochemical markers for breast carcinomas are gross cystic disease fluid protein-15 (GCDFP-15), cytokeratin 7, carcinoembryonic antigen, estrogen receptor, and progesterone receptors, and negative marker is cytokeratin 20. Chemotherapy and/or hormonal therapy are the most commonly used modalities, whereas surgery is indicated mostly in cases of stenosis or complete obstruction.</p> <p>Conclusion: The purpose of this report is to raise awareness of the consideration of GI metastases in patients with history of breast cancer and more attention to be paid towards the patients who present to their primary care physicians with complaints of GI symptoms for appropriate work up for early diagnosis and treatment.</p>

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Clinical Vignette	Bhaveshkumar Garsondiya	Nikunj Kumar Patel, Mohammad Amir Hossain, Lintu Ramachandran	Raritan Bay Medical Center (Abdalla M Yousif)	Pot Paresis: Marijuana as a Cause of Hypokalemic Periodic Paralysis.	<p>Introduction: According to the WHO 147 million people use cannabis, making it the world's most widely cultivated, trafficked, and abused illicit substance. Toxic effect of marijuana impair attention, concentration, short-term memory, executive functioning, nausea, and also hypokalemic periodic paralysis.</p> <p>Case: A 31 year old male with no significant PMH presents to the ED with the chief complaint of "I can't move my arms and legs." The patient was in his usual state of health five hours prior to presentation. Patient denies chest pain, shortness of breath, visual problems, headache, dizziness, loss of consciousness, injury, seizure, bowel or bladder incontinence. Patient smokes one pack per day for more than 15 years, smokes Marijuana 4-5 times weekly and denies drinking alcohol. Vitals were within normal limits. Physical examination was wnl except muscle strength in all extremities was 3/5, DTRs 2+, sensation was intact, CN II to XII were intact and Babinski was negative. Labs were wnl except for potassium 2.1 mmol/L. CT scan of the head showed no acute intracranial hemorrhage or mass. The EKG showed T wave inversion and U wave. Patient was given total 80 mEq of Potassium PO and 40 mEq IV. Repeated potassium level came to 4.4 mmol/L and patient's condition gradually improved, started moving his extremities and was able to ambulate normally.</p> <p>Discussion: A recent study showed that serum sodium and potassium ions levels of marijuana smokers were estimated using atomic emission flame spectrophotometry on hundred (100) subjects comprising sixty marijuana smokers and forty non-marijuana smokers. The mean of serum Na⁺ and K⁺ levels of marijuana smokers were 119±26mmol/l and 2.3±0.7mmol/l respectively, while the controls had 140±6mmol/l and 3.8±0.5mmol/l respectively. The results showed that there is statistical difference in serum sodium and potassium levels of marijuana smokers compared with non-smokers control (P<0.05). Hyponatremia and hypokalemia are the probable causes of these various morbidity and mortality associated with this habit. Hypokalemia due to cannabinoid use may be due to a number of mechanisms including potassium loss via kidneys, potassium loss due to excessive sweating and diarrhea, and potassium shift into cells. One of the mechanism of action of cannabinoid is linked to the inhibition of adenylate cyclase and effect the calcium and potassium channel function to bring inhibition of synaptic transmission. This effect of the cannabinoid receptor on potassium channel was likely the cause of hypokalemia in this patient with subsequent transient paralysis.</p> <p>Conclusion: It thus become very important to keep in consideration the deleterious effects of marijuana leading to electrolyte imbalance with subsequent adverse neurological effects.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Arman Mushtaq	Hiyam Bakhit	Raritan Bay Medical Center (Abdalla M Yousif)	Uremic Encephalopathy Masking Myxedema Coma.	<p>Introduction: Myxedema coma is a rare disastrous presentation that occurs in less than 0.1% of all cases of severe hypothyroidism. Patients present in obtunded state that mimics many serious etiologies leading to unnecessary investigations and interventions causing increased morbidity, length of stay, and cost.</p> <p>Case: A 55-year-old female was brought to ER after she was found confused and desheveled on the floor. In ER she was hypothermic, obtunded and unable to answer any questions. As per medical records she had ESRD on dialysis, diabetes mellitus and hypothyroidism. Lab data revealed Hb. 8.8 g/dl, creatinine 14 mg/dl, BUN120 mg/dl, potassium 8.7mm/L. Chloride 102mm/L, CO2 < 8 mm/L, ABGs : pH of 7.10, pCO2 21, pO2 165, HCO3 6.5. CT of the head was unremarkable. Physical examination: BP 120/70, pulse 55/min, temp. 94.6 F, RR 16/min. Extremities: anasarca and non-pitting edema of lower limbs. Neurologically patient was obtunded. Reflexes were diminished and Babinski was negative. She was admitted to ICU and had two sessions of hemodialysis on consecutive days. Her BUN and creatinine came to base line and acidosis also resolved after first hemodialysis. However her mental status did not improve and she remained hypothermic and bradycardic. She received empiric treatment for herpes and bacterial meningoencephalitis. That was discontinued after negative CSF results. TSH level came back 60. She was started on IV levothyroxine. After receiving two doses of IV levothyroxine her temperature and heart rate normalized and she was completely oriented with normal neurological exam.</p> <p>Discussion: Narrowing the diagnosis to myxedema coma can be challenging due to several chronic diseases presenting in the similar fashion. Conditions like viral or bacterial meningitis, hepatic encephalopathy, sepsis and accidental hypothermia can all present with same spectrum of signs and symptoms. Careful neurological exam in the light of history is pivotal. Brain stem infarct and CO2 narcosis have also been reported where the underlying culprit was myxedema coma induced by severe hypothyroidism. In our case uremic encephalopathy masked under lying myxedema coma. Compliance with thyroid replacement therapy is of utmost importance in patients with multiple co-morbidities and old age and requires stringent follow up at the level of subspecialty and primary care. Further history from our patient revealed that she stopped taking her thyroxine 1 month ago and then started feeling lethargic, depressed and eventually to the extent that she stopped all her daily activities and missed her dialysis sessions, the cascade of events that led to her disastrous presentation.</p> <p>Conclusion: Our case highlights the importance of prompt recognition of underlying hypothyroidism in critically ill patients presenting in obtunded state and to initiate appropriate doses of levothyroxine preferably IV, to halt and reverse the sequela of severe hypothyroidism.</p>

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Clinical Vignette	Romana Kanta	Lintu Ramachandran,Sabrina Arshed,Margaret Omatstone	Raritan Bay Medical Center (Abdalla M Yousif)	Respiratory failure in DKA patient due to hypophosphatemia	<p>Introduction: Initiation of insulin in patients with DKA has been documented to alter phosphate balance and cause hypophosphatemia in a significant number of patients. We report the case of a DKA patient who developed hypophosphatemia during insulin therapy, and proceeded to life threatening respiratory failure despite adequate phosphate replenishment.</p> <p>Case Report: This is a 26-year-old female with PMH of non-compliant diabetes mellitus type 1, admitted due to altered mental status, nausea and vomiting who was subsequently diagnosed with DKA. On inspection, the patient appeared acutely ill with mild respiratory distress. The patient was awake and mildly confused, but followed commands. His vitals were, Temperature: 97.6 F, Pulse: 106 bpm, RR: 20 breaths per minute, BP: 97/64 mm of Hg and O2 sat: 99% on 2L nasal cannula. Physical examination was only remarkable for symmetric weakness of all four limbs. Initial labs: Blood glucose: 907 mg/dL, arterial pH <6.80, PCO2 <15 mmHg, PO2 123 mmHg, bicarbonate <8, ketonemia (+++) and Beta-hydroxybutyrate >8. The effective serum osmolality was 327mOsm/kg.</p> <p>The patient was given insulin, normal saline and bicarbonate. Potassium and phosphorus levels were found to be 3.8 mg/dl and 2.7 mg/dl respectively. KCl was initiated and in the next 24 hours, phosphorus level decreased to 0.6 mg/dl. The patient's respiratory status subsequently deteriorated and patient required intubation as well as ventilator support.</p> <p>On the third day, despite correcting the patient's hyperglycemia and acidosis, generalized weakness persisted. Biochemical investigation revealed severe hypophosphatemia of 0.3 mg/dl. Potassium phosphate was initiated and gradually, the muscle weakness improved over the course of four days. The patient was weaned off the ventilator, and successfully extubated on the 6th hospital day. She eventually recovered and was discharged after 10 days of total hospital stay.</p> <p>Discussion: While extensive guidelines exist to withhold insulin in the case of hypokalemia during DKA, no guidelines currently exist to hold insulin in severe hypophosphatemia. Although, insulin administration has been associated only with small decrease in serum phosphorous concentrations, in patients with DKA, a combination of insulin administration, bicarbonate replacement and fluid resuscitation can cause sudden and severe drop of phosphate levels. In our patient, phosphate level went down to 0.3 mg/dl despite adequate phosphate replacement. The patient developed life threatening respiratory complications due to severe hypophosphatemia, secondary to the DKA treatment. Withholding insulin in this patient could have possibly prevented unwarranted complications.</p> <p>Conclusion: Severe hypophosphatemia and associated complications such as respiratory failure, impaired cardiac output, seizures and rhabdomyolysis can be fatal in the case of DKA. This specific case raises the question of whether establishing specific mandatory guidelines for withholding insulin to prevent hypophosphatemia during DKA cases might not just be beneficial, but essential.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Claribel Cruz	Sabrina Arshed MD, Margaret Omatsone MD	Raritan Bay Medical Center (Abdalla M Yousif)	Case Review: Hyperthyroid Induced Small Bowel Obstruction	<p>Introduction: The thyroid hormones regulate metabolism, secretion of catecholamines and growth hormones. Thyroid dysfunction may lead to systemic signs and symptoms, related to increased metabolism and increased adrenergic activity.</p> <p>Case: A 42 year old Hispanic male with medical history of hyperthyroidism, not currently on treatment, presented to the ED complaining of acute onset abdominal pain, nausea and vomiting. He denied any changes in bowel habits and had a bowel movement on the afternoon of presentation; without any blood, or change in caliber of stool. He states having early satiety in the days leading to presentation.</p> <p>Vital signs were: blood pressure of 160/100mmHg, HR of 130bpm, RR 18bpm and temperature of 99.6F. Physical examination revealed a well-built Hispanic male, with exophthalmos, prominent thyroid with minimal thrill. Cardiovascular examination revealed tachycardia. Abdominal examination showed a soft abdomen with generalized tenderness with minimal guarding, and decreased bowel sounds in all quadrants. CT scan of the abdomen was done which was consistent with small bowel obstruction. Pertinent lab findings included a TSH level of 0.010mU/mL, Free T4 was 7.77ng/dL, total T3 of 286ng/dL, anti-TPO antibody was 525, and anti-TSI antibody was 302. Thyroid ultrasound showed an enlarged thyroid without any masses, and uptake scan revealed diffuse uptake without any masses. Findings were suggestive for Grave’s disease, and patient was subsequently started on Methimazole 20mg every 8 hours and beta-blockade. After initiation of treatment patient’s small bowel obstruction was relieved, without need for surgical intervention.</p> <p>Discussion: Interruption to the normal flow of the intraluminal contents of the bowel leads to bowel obstruction. In turn, this leads to dilatation of the bowel and sequestration of fluids within the lumen. The effects of thyroid hormones on the gut are numerous and spares no portion of the gastrointestinal system. It has been widely studied, but poorly understood. Autonomic nervous system dysfunction leads to a decrease in vagal influence, by impairing the neuro-hormonal regulation of gastric myoelectrical activity, thus leading to decreased gastric emptying. Excess or deficiency of thyroid hormones have multiple effects on the gut, leading to hyperdefecation or constipation. In the setting of hyperthyroidism, thyrotoxicosis leads to decreased small bowel transit time, which is linked to a hypersecretory state in intestinal mucosa. It has been noted to be overcome with propranolol.</p> <p>Conclusion: As clinicians, we are aware of hypothyroidism leading to a decrease in gastrointestinal motility. However, when encountered with common complaints of early satiety, changes in bowel habits, and signs and symptoms of delayed gastric emptying, an endocrine source, including hyperthyroidism, must be part of the differential diagnosis.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Blaine Huss	Elizabeth John MD, Navaneeth Narayanan PharmD, Mary Bridgeman PharmD, Sita Chokhavatia MD	Rutgers - New Brunswick (Ranita Sharma)	Clostridium difficile Infections in the Setting of Chronic Opioid Use: Do Pain Medications Actually Worsen Infections?	<p>Introduction: Patients with a history of chronic opioid use may have worse clinical outcomes related to Clostridium difficile infection (CDI) from alterations in gastrointestinal (GI) function and motility. Understanding the extent of this relationship will allow providers to target at-risk patients and provide prophylaxis and/or alter antibiotic therapies to lower the risk of a serious infection. The intent of this study is to evaluate the relationship between chronic opioid use and CDI disease severity.</p> <p>Methods: A retrospective review of patient medical records was conducted to determine the relationship between chronic opioid use and clinical outcomes related to CDI. All adult inpatients having diagnosis codes for diarrhea and positive C. difficile stool toxin or PCR and chronic opioid use admitted between 1/1/11 and 12/31/15 were evaluated. Patients younger than age 18 years, pregnant, or with inflammatory bowel disease, irritable bowel syndrome, or concurrent etiology of persistent diarrhea (e.g., chronic laxative use) were excluded.</p> <p>Results: Of the 25 cases, the majority occurred in Caucasians (n=18) and males (n=12) with an average age of 52.12±13.29 years. Oxycodone was the most frequently reported chronic opioid used either alone or in combination with other narcotics (n=10), followed by methadone (n=6). The average length of stay (LOS) was 18.44±16.54 days, and an average LOS related to CDI of 14.24±13.24 days. Hospital-acquired (HA) CDI, occurring greater than 72 hours from admission, occurred in 11 (44%) cases. 12 cases (48%) had a readmission within 30 days of hospital discharge, with 10 cases (40%) reportedly using antibiotics prior to admission. 15 patients (60%) were also using acid suppressive medicines during hospitalization. 17 cases (68%) were regarded as mild/moderate CDI, 7 cases (28%) as severe, and 1 (4%) as complicated. Ileus was reported in 6 cases (24%) and toxic megacolon in 3 (12%).</p> <p>Discussion: When compared with previous outcomes studies related to CDI severity, our results suggest that patients on chronic opioids have a younger age of CDI and longer LOS. By demonstrating a qualitative relationship between chronic opioid use and CDI severity, physicians should minimize the amount of narcotics administered to patients being treated for CDI. A follow-up retrospective cohort study is planned.</p>

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Clinical Vignette	Sasha-ann East	Sheetal Patel MD	Rutgers - New Brunswick (Ranita Sharma)	A Case of Post-operative Deep Vein Thrombosis in a Hypercoagulable Patient on Apixaban	<p>Introduction: Hypercoagulable disorders pose an increased lifetime risk of developing venous thromboembolic events (VTE). It is established that patients with these disorders usually need lifelong anticoagulation once they have more than one thromboembolic event. With the emergence of novel oral anticoagulants (NOAC), management of these patients has been streamlined. However, in times of medical illness or surgery, NOACs may not be optimal. These patients may require more aggressive individualized therapy to prevent VTE.</p> <p>Case: This case involves a 48-year-old gentleman with a past medical history of a prothrombin gene mutation complicated by two episodes of provoked VTE four and ten months prior. He had been taking Apixaban, a NOAC, indefinitely as a result. He elected to undergo total hip replacement for osteoarthritis. At the time of the operation, the patient had stopped taking Apixaban for 48 hours under the advisement of his hematologist. The patient tolerated the surgery well without any bleeding complications. Apixaban 5 milligrams (mg) was restarted within six hours after surgery and continued twice daily. The next day, the patient underwent physical therapy and tolerated early ambulation. However, he started to have right lower extremity pain and swelling. An ultrasound of the right lower extremity was significant for an acute proximal deep vein thrombus. Apixaban was then increased to 10mg twice daily. The patient's symptoms and leg swelling improved over the next two days and he was discharged on the higher dose of Apixaban.</p> <p>Discussion: NOACs are a mainstream method of anticoagulation for various types of pro-thrombotic illnesses and predispositions. This is due to their ability to be therapeutic at onset, the convenience of not requiring periodic monitoring of drug levels, shorter half-lives, and ease of compliance. However, hypercoagulable patients may not respond to the standard doses of NOACs during times of stress. Unfortunately, there are not many studies testing the effectiveness of NOACs at different dosage levels in preventing VTE among high risk populations. Furthermore, the standard coagulation assays have not been reliable measurements of NOAC activity.</p> <p>Studies have primarily focused on heparin products and vitamin K antagonists for VTE prevention. Across multiple studies, low-molecular-weight heparin (LMWH) seems to provide better outcomes for high risk surgical and medically ill patients. This is especially true, when therapy is guided by monitoring Factor Xa levels. The patient in this case is a high risk for VTE complications due to his prothrombin gene mutation and the type of surgery he underwent. Despite every precaution to limit the interval off Apixaban and encourage early ambulation, he still developed a DVT in-hospital. Going forward, it may be best to transition high risk patients to LMWH therapy peri-operatively and early post-op until NOACs are proven to be an effective alternative.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ronaldo Ortiz	Ashley Bean, MD, MPH; Michael Marsh, MD; Andrew Berman, MD	Rutgers - Newark (Neil Kothari)	Worsening cough and sputum production in a patient with multiple pulmonary conditions	<p>Pulmonary alveolar proteinosis (PAP) is an uncommon respiratory illness in which protein rich surfactant is deposited within the alveoli. Patients present with dyspnea on exertion, cough, fatigue, and weight loss. Radiography reveals bilateral mid-to-low airspace opacities. Pathologically, the lipoproteinaceous material is periodic acid schiff positive and there is little evidence of an inflammatory process.</p> <p>The patient is a 55-year-old African American man with a 20 pack year smoking history and a past medical history significant for COPD on home O2 plus albuterol and budesonide/formoterol inhalers; chronic systolic heart failure (Left Ventricular Ejection Fraction 30%) and a prior episode of PAP. He presented to the emergency department with complaints of worsening dyspnea of two days duration and orthopnea. He also reported a productive cough, occasionally with blood-tinged sputum, and a weight loss of 20 pounds over 6 months. Oxygen saturation on room air was 88%. Initial arterial blood gas revealed a pH of 7.41, pCO2 of 39 mmHg, and pO2 of 55 mmHg. Physical exam was notable for diffuse rhonchi without wheezing. Clubbing was present. Chest X-ray revealed bilateral patchy opacities; chest CT demonstrated a “crazy paving pattern” with bilateral ground glass opacities.</p> <p>The patient did not improve with nebulized ipratropium-albuterol, steroids or diuretics. Oxygen saturation worsened to 85% with minimal exertion on 4L nasal cannula. The patient was taken to the OR for left lung lavage and thick white liquid was aspirated. The patient clinically improved after the procedure, saturating at 97% via 2L nasal cannula. Right lung lavage was conducted at a later date. the patient was able to saturate at 98% on 2L nasal cannula.</p> <p>The patient had symptoms which overlapped several pulmonary conditions but only improved after whole lung lavage. Radiographically, PAP was suspected though it was unclear if the opacities were chronic. Etiology of the PAP in this case is unknown; serum GM-CSF autoantibody was negative, although significant exposure to environmental factors is suspected due to occupational exposure working on motor vehicles and in building renovation. Diagnosis required the integration of history, exam, radiographic findings, and response to treatment.</p>

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Clinical Vignette	Ali Aziz		Saint Barnabas Medical Center (Sunil Sapru)	Diabetic ketoacidosis as a complication of Myocardial infarction with cardiogenic shock	<p>Introduction: Insulin resistance is defined as a state in which greater than normal amounts of insulin are required to elicit a response. The mechanisms of insulin resistance include: altered insulin receptors, autoantibodies to insulin receptors, increased insulin clearance or excessive counter regulatory hormones and inflammatory cytokines. We present a case of worsened insulin resistance as a consequence of myocardial infarction and cardiogenic shock.</p> <p>Case report: A 55 year old male with a history of type 2 diabetes mellitus on metformin presented with diaphoresis and tachypnea. Blood pressure was labile, respiratory rate was 40/min and oxygen saturation was 88% on room air. Telemetry showed atrial fibrillation with ventricular response of 140/min. He was emergently intubated and sedated. A 12 lead EKG showed 3mm ST elevations in leads II, III, and aVF. Arterial blood gas showed pH 7.18, and pCO2 60. Additional labs showed blood glucose 400, troponin 0.389, BNP 776, anion gap 27, serum bicarbonate 15, lactic acid 6.7, and creatinine 1.49. Urinalysis demonstrated proteinuria, glucosuria and urine ketones. Echocardiogram showed inferior wall hypokinesis with an ejection fraction of 35%. Emergent cardiac catheterization showed total occlusion of the distal RCA and PTCA was performed. Following catheterization the patient developed acute kidney injury (AKI) and did not respond to intravenous diuretics. He developed severe hypotension and required intravenous diltiazem for rate control and norepinephrine for blood pressure support. Insulin drip was started as per DKA protocol and after 3 liters of IV fluids his lactic acid improved to 2.4 from 6.7. Blood pressure remained stable and urine output improved after 48 hours. In the first 24 hours of treatment he received a total of 136 units of insulin. His blood sugars ranged 200-380 and the anion gap improved from 27 to 15. He was switched to subcutaneous insulin but his blood sugars worsened so he was started on insulin drip again. In the 2nd 24-hour period he received total 87 units of insulin and in the 3rd 24-hour period he received a total of 65 units of insulin. On day 4 he received 36U of subcutaneous levemir and the insulin drip was stopped. Blood glucose and insulin requirements gradually decreased with resolution of cardiogenic shock.</p> <p>Discussion: Myocardial infarction with cardiogenic shock causes insulin resistance by elevating catecholamines and cortisol through activation of the sympathetic nervous system. In addition, the inflammatory state accompanying myocardial infarction leads to release of proinflammatory cytokines IL-6 and TNF-alpha that interfere with tyrosine kinase activation in insulin receptor. Insulin drip should be continued for a longer period of time in these patients and early transition from IV to subcutaneous insulin should be avoided</p>

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Clinical Vignette	Sneha Shrestha	BIPLAB SAHA, MD; MIGUEL CONDE, MD	Saint Barnabas Medical Center (Sunil Sapru)	A Rare Case Report of Purpura fulminans in Association with Cryoglobulinemia Secondary to Myelodysplastic Syndrome	<p>INTRODUCTION: Purpura fulminans is a life-threatening condition characterized by acute hemorrhage and skin necrosis due to dermal vascular thrombosis. Cutaneous involvement is the commonest manifestation of cryoglobulinemia but to our knowledge, skin involvement to extent of purpura fulminans in cryoglobulinemia has never been reported. Here we present a rare and challenging case of purpura fulminans due to cryoglobulinemia in the setting of worsening Myelodysplastic syndrome (MDS) who survived in spite of grave prognosis traditionally linked to PF.</p> <p>CASE DESCRIPTION: 70 year old Caucasian female presented with painful, purple, ghastly purpuric rashes over arms and thighs noted when she awoke from sleep. She denied fever, night sweats, chest pain, dyspnea, joint pain, previous history of similar rash and no acute adverse reaction during or immediately after transfusion. She had received 2 units of PRBC 24 hours earlier for hemoglobin of 6.6gm/dl on routine blood work. Anemia was due to myelofibrosis (prefibrotic) with JAK2 mutation. She had hypertension, hyperlipidemia, left breast carcinoma s/p mastectomy with axillary LN dissection in 1989 that recurred in 2008 and treated with chemotherapy. Physical exam revealed HR 100bpm, BP 120/57mmHg, temperature 99.5°F, saturation 100% in room air. There were large tender purple purpuric rashes over arms and thighs with few bullae. Blood work showed hemoglobin 8.8gm%, platelet count 123,000/mm², WBC 120/mm², serum creatinine 1.13mg/dl, serum LDH 458U/ltr and haptoglobin <10mg/dl. Electrolytes and LFT were unremarkable. Direct antibody test (Coomb's titre) was positive even before transfusion. Cold agglutinin test was negative. She was treated with IV immunoglobulin and antibiotics in the setting of neutropenia.</p> <p>Over the next 24 hours, rash worsened to involve reconstructed left breast. Platelet count dropped (66,000 per sqmm). Coagulation studies were not consistent with DIC (PT 13.7sec, INR 1.2, PTT 28.7 sec, fibrinogen 475mg/dl, d-dimer 1,311ng/dl). She was anticoagulated with heparin infusion in spite of thrombocytopenia. At this time, serum cryoglobulin, cryofibrinogen and anti-cardiolipin antibody came back positive. A full thickness skin biopsy from anterior right thigh was performed which on histopathological examination, demonstrated microthrombi in cutaneous capillaries with no vasculitis. Her rash started to clear up and pain improved. Infectious disease work up was negative. She was discharged home on oral warfarin and rituximab. Repeat autoimmune and hypercoagulability work-up confirmed diagnosis of type 2/3 cryoglobulinemia. 5 months later, repeat bone marrow biopsy, FISH and cytogenetics established diagnosis of MDS.</p> <p>CONCLUSION: Purpura fulminans is a rare devastating hematological emergency usually associated with DIC. The reported case is unique as it is the first ever case report to our knowledge of the entity in association with cryoglobulinemia (secondary to MDS). This case also demonstrates that dermal vascular thrombosis improves with anticoagulation with heparin.</p>

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Clinical Vignette	Abhishek Bhurwal	Muhammad Masoodul Haq, Sunil Sapru	Saint Barnabas Medical Center (Sunil Sapru)	Disseminated herpes zoster masquerading as burn injury in an immunocompetent patient	<p>Introduction: Cutaneous dissemination of herpes zoster has been described frequently in immunocompromised individuals but rarely may also occur in immunocompetent patients. We present a case of disseminated herpes zoster mimicking burn injury in an immunocompetent host.</p> <p>Case description: A 66-year-old lady with diabetes mellitus type 2 and hypothyroidism presented with complaints of progressively worsening erythematous vesicular lesions on the face, trunk and extremities for the last 6 days. The lesions were initially diagnosed as burn injury at an urgent care center after she accidentally splashed boiling water on her face and her right hand. She was prescribed topical Neosporin with no improvement over the next four days. Subsequently, she noticed her left eye was becoming increasingly droopy, red, and swollen culminating in it being permanently shut for about twenty-four hours prior to admission. On examination, a vesicular rash was prominent covering her left scalp, forehead, upper cheek. Similar lesions were also appreciated on the back, extensor aspect of both, left forearm and left thigh. The tip of the nose was erythematous and tender. Ophthalmologic exam was significant for left-sided conjunctival irritation with no exudates. The patient had swollen lids on the left side and positive direct and consensual pupillary reflexes. Extra-ocular movements were intact with no diplopia. Fluorescein staining yielded a 5mm branched dendritic corneal lesion. A diagnosis of disseminated herpes zoster infection was made and IV acyclovir started. Blood culture, anti-HIV antibody (ELISA), flow cytometry and chest x-ray, performed to investigate the cause of infection and immune suppression, were all negative. Varicella IgM levels were elevated. The skin lesions improved with treatment and resolved completely in ten days when she was discharged home.</p> <p>Discussion: Diffuse Cutaneous Herpes Zoster is characterized by more than 20 skin lesions located beyond the primary or adjacent dermatomes. The virus can spread through interconnections between several ganglia or hematogenous spreading. It is usually seen in immunocompromised patients such as those with HIV infections, cancer, chemotherapy, and on corticosteroid therapy. It is an extremely rare occurrence in an immunocompetent host. The patients are often treated with intravenous antivirals to prevent cutaneous and visceral dissemination. The possibility of systemic complications in disseminated herpes zoster requires a high index of suspicion to make a diagnosis in immunocompetent individuals. Our patient was particularly challenging as the lesions appeared in the setting of a burn injury. A detailed history and physical examination are vital in making the diagnosis.</p>

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Clinical Vignette	Ali Aziz	Paul Wangenheim, MD	Saint Barnabas Medical Center (Sunil Sapru)	Diabetic ketoacidosis as a complication of Myocardial infarction with cardiogenic shock	<p>Introduction: Insulin resistance is defined as a state in which greater than normal amounts of insulin are required to elicit a response. The mechanisms of insulin resistance include: altered insulin receptors, autoantibodies to insulin receptors, increased insulin clearance or excessive counter regulatory hormones and inflammatory cytokines. We present a case of worsened insulin resistance as a consequence of myocardial infarction and cardiogenic shock.</p> <p>Case report: A 55 year old male with a history of type 2 diabetes mellitus on metformin presented with diaphoresis and tachypnea. Blood pressure was labile, respiratory rate was 40/min and oxygen saturation was 88% on room air. Telemetry showed atrial fibrillation with ventricular response of 140/min. He was emergently intubated and sedated. A 12 lead EKG showed 3mm ST elevations in leads II, III, and aVF. Arterial blood gas showed pH 7.18, and pCO2 60. Additional labs showed blood glucose 400, troponin 0.389, BNP 776, anion gap 27, serum bicarbonate 15, lactic acid 6.7, and creatinine 1.49. Urinalysis demonstrated proteinuria, glucosuria and urine ketones. Echocardiogram showed inferior wall hypokinesis with an ejection fraction of 35%. Emergent cardiac catheterization showed total occlusion of the distal RCA and PTCA was performed. Following catheterization the patient developed acute kidney injury (AKI) and did not respond to intravenous diuretics. He developed severe hypotension and required intravenous diltiazem for rate control and norepinephrine for blood pressure support. Insulin drip was started as per DKA protocol and after 3 liters of IV fluids his lactic acid improved to 2.4 from 6.7. Blood pressure remained stable and urine output improved after 48 hours. In the first 24 hours of treatment he received a total of 136 units of insulin. His blood sugars ranged 200-380 and the anion gap improved from 27 to 15. He was switched to subcutaneous insulin but his blood sugars worsened so he was started on insulin drip again. In the 2nd 24-hour period he received total 87 units of insulin and in the 3rd 24-hour period he received a total of 65 units of insulin. On day 4 he received 36U of subcutaneous levemir and the insulin drip was stopped. Blood glucose and insulin requirements gradually decreased with resolution of cardiogenic shock.</p> <p>Discussion: Myocardial infarction with cardiogenic shock causes insulin resistance by elevating catecholamines and cortisol through activation of the sympathetic nervous system. In addition, the inflammatory state accompanying myocardial infarction leads to release of proinflammatory cytokines IL-6 and TNF-alpha that interfere with tyrosine kinase activation in insulin receptor. Insulin drip should be continued for a longer period of time in these patients and early transition from IV to subcutaneous insulin should be avoided.</p>

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Clinical Vignette	Bushra Syed	Bushra Syed MD, Luigi Bonomini MD	Saint Barnabas Medical Center (Sunil Sapru)	Athroembolic Renal Disease	<p>Introduction: Disruption of renal microcirculation by dislodged atheromatous plaques occurs spontaneously, from plaque erosion following endovascular interventions or with use of anticoagulants and thrombolytics. Resultant ischemia, inflammation and complement activation cause injury. Athroembolic renal disease(AERD) is underdiagnosed, it's suspected in 3% of end stage renal disease in elderly patients. We present a patient with AERD following percutaneous coronary intervention(PCI) and cardiac bypass surgery(CABG).</p> <p>Case: A 77-year-old woman presented with confusion and dyspnea. History included PCI, CABG and mitral valve replacement 3 weeks ago, hypertension, hyperlipidemia, stage 3 CKD with baseline creatinine(Cr) of 1.1mg/dl. Post-operative complication included acute kidney injury attributed to acute tubular necrosis(ATN) and interstitial nephritis from proton pump inhibitor use. Steroids and temporary hemodialysis support was provided. Subsequent improvement in renal function to a Cr of 2.5mg/dl was seen and she was discharged off dialysis. After discharge developed dyspnea, edema and decreased urine output despite adherence to medications which included diuretics. Physical examination showed blood pressure of 187/110, hypoxemia, distended neck veins, rales and bilateral pedal edema. Purple discoloration of toes and livedo reticularis on lower extremities was seen, with intact pedal pulses.</p> <p>Laboratory data showed elevated blood urea(55 mg/dl) and Cr(4.28mg/dl), hyponatremia(124mmol/L), normal potassium(4.5 mmol/L) and bicarbonate(26mmol/L) levels. Eosinophilia(13.9 %) and eosinophiluria were present. Complement levels and CRP were normal, with ESR of 45mm/hr. The BNP was 2700 with normal cardiac enzymes. Urinalysis showed bland sediment, subnephrotic proteinuria and protein to Cr ratio of 325mg/dl.</p> <p>Renal ultrasounds showed no hydronephrosis. Initial management included furosemide and chlorthiazide. Oliguria persisted and Cr up trended to 6mg/dl. Hemodialysis was initiated and she underwent a percutaneous renal biopsy. On biopsy 6 of 29 glomeruli showed global sclerosis, atheroemboli in medium sized vessels with interstitial inflammation and evidence of ATN was present. The patient was discharged on out-patient hemodialysis and remains dialysis dependent.</p> <p>Discussion: Onset of clinical manifestations of AERD, following a vascular intervention ranges from weeks to months. Worsening hypertension and renal impairment is accompanied by embolic manifestations like livedo reticularis, toe gangrene, transient ischemic attacks or hollenhorst plaques. Sixty to 80% cases show rising creatinine and eosinophilia, fifteen percent have hypocomplementemia. Bland urinary sediment, microscopic hematuria, eosinophiluria and variable proteinuria are seen. Definitive diagnosis involves renal biopsy showing occlusion of arcuate, interlobular and glomerular capillaries with cholesterol crystals that leave characteristic biconvex, needle shaped clefts. No effective therapy is available to reverse the existing vascular lesions. Prognosis is poor with mortality rates of 38% after one year. Progressive renal failure requires dialytic support. Statin therapy may improve outcome and cessation of anticoagulation is recommended.</p>

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Clinical Vignette	Randy K Ramcharitar	Paul Ricci, MD, ACP Member, St. George's University School of Medicine, Grenada, West Indies	Saint Barnabas Medical Center (Sunil Sapru)	EMBOLIC STROKE SECONDARY TO HEPATIC HEMANGIOMA WITH AV SHUNT	<p>Introduction: Hepatic (cavernous) hemangiomas are small (<5 cm) benign neoplasms of the liver typically incidentally detected during imaging. Patients are typically asymptomatic, but may present with pain, early satiety, anorexia, nausea, and distension. Complications include Kasabach-Merrit Syndrome, Bornman-Terblanche-Blugart Syndrome, arterioportal shunts in 26% of patients, and very rare AV shunts.</p> <p>Case Description: A 50-year-old non-smoking Caucasian female on treatment with apixaban for DVT and PE presented with blurry vision and difficulty reading the end of words. She denied headache, nausea, vomiting, chest pain, dyspnea, abdominal pain, weight changes, or joint/muscle pain. Physical examination revealed a homonymous blind spot in the right central visual field and right pronator drift. While no defect in language fluency was detected, word repetition was observed in words with more than three syllables. Laboratory findings revealed elevated PT/INR, BUN, cardiolipin IgM and mycoplasma IgM. CT of the head was unremarkable. MRI and MRA of the brain showed multiple acute ischemic foci; the largest within the left parietal and left posterior temporal lobes, consistent with an embolic stroke. Transthoracic echo was unremarkable but transesophageal echo with bubble study was consistent with an extracardiac shunt. Two CTA chest studies showed a 2 cm left lower lobe nodule, left axillary and bilateral supraclavicular lymphadenopathy. Also of note was a 1.3 cm segment V enhancing lesion of the liver consistent with an atypical hemangioma and source of the extracardiac shunt. Pathology report from left lymph node biopsy revealed metastatic pulmonary adenocarcinoma. Additional sources of shunt were ruled out by VQ scan and further imaging of chest, abdomen and pelvis via CT with contrast.</p> <p>Discussion: Malignancy related hypercoagulability can be the etiology for stroke in isolation; however the embolic appearance of the infarcts was strongly suggestive of paradoxical embolism, particularly given the known DVT and PE. Hemangiomas are known to be composed of dilated non-anastomotic vascular spaces that can contain large volumes of blood with very slow flow. This is the first documented case of an atypical hemangioma with arteriovenous shunt presenting with a thromboembolic event. This case represents a rare confluence of factors for this outcome; in particular, treatment with apixaban for DVT and PE, undiagnosed malignancy leading to hypercoagulability and an atypical hemangioma as AV shunt source allowing for an embolic stroke.</p>

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Clinical Vignette	Han Vo	Tracey Li, PharmD; Ahmad Shoubaki; Shih-Fen Chow, MD; Mindy Houg, MD; Su Wang, MD, MPH	Saint Barnabas Medical Center (Sunil Sapru)	Genitourinary Tuberculosis and Pott's Disease	<p>Mycobacterium tuberculosis commonly affects the lungs, but can spread to extrapulmonary organs. Extrapulmonary tuberculosis (EPTB) can present with diverse symptoms that mimic those of other pathologies. Its diagnosis can be elusive and requires a higher sense of suspicion in countries of low TB prevalence. A rare case of simultaneous male genitourinary and spinal tuberculosis (Pott's disease) is reported.</p> <p>A 41 year-old Chinese man initially presented with intermittent lower back pain confirmed by CT scan, which showed lumbar compression fracture, and treated with naproxen. He developed right testicular swelling a few months later. Patient was sexually active and in a monogamous relationship. He denied fever, dysuria, urinary frequency, and urethral discharge. Initial diagnosis was epididymitis, and an empiric antibiotic was started. Urinalysis, chlamydia and gonorrhea tests were negative. Ultrasound of testes confirmed epididymitis and a right hydrocele. Follow-up revealed enlarging, firm, and irregular right testis. Repeat ultrasound revealed heterogeneous testes that increased in size. His complete blood count, prostate specific antigen, and α-fetoprotein were within normal limits. The differential diagnoses were infection versus neoplasm. Urology was consulted; a fine needle aspiration was inconclusive. Upon further investigation, the patient revealed he had active tuberculosis twenty years ago and recalled a six month treatment with medications. Patient had a positive QuantiFERON[®]-TB Gold test and reported no symptoms. His chest x-ray showed a non-specific right upper lobe opacity/density. CT of the chest, abdomen and pelvis confirmed scarring of right upper lobe, cluster nodularity of bilateral apices, 4mm nodules of the right lower lobe, and a 2.7cm hepatic vascular lesion. Tests for HIV and sputum samples for acid-fast bacilli were negative. There was high suspicion for TB orchitis/epididymitis and Pott's disease of the spine. Previous CT of the spine showed T12-L1 compression fracture, and follow-up MRI confirmed multiple areas of abnormal marrow signal and a defect of the bone at L1 extending into soft tissues. Incision and drainage with exploration of the scrotal mass was performed, and the fluid was sent for cultures, acid-fast stain, histopathology and cytology. Urine AFB was positive. Scrotal pathology revealed granulomatous changes with giant cells, consistent with TB. Two years of directly-observed anti-tuberculosis therapy was initiated by the local health department.</p> <p>This case illustrates the challenge of diagnosing EPTB, particularly for physicians practicing in countries of low prevalence. EPTB does not only occur in immunocompromised patients as seen in this case. Barriers to diagnosis included insurance approvals and delays for imaging, communication amongst health care providers, and patient resistance to invasive procedures. Physicians should consider EPTB in patients who originate from areas with high prevalence of tuberculosis.</p>

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Clinical Vignette	Rikitha Menezes	Samar Hameed, MD; Stuart Mendelson, MD; Sunil Sapru, MD	Saint Barnabas Medical Center (Sunil Sapru)	The Hurting Head and The “Sagging” Brain	<p>INTRODUCTION: Cerebrospinal fluid (CSF) provides a supportive cushion for the brain and alterations in the intracranial pressure (ICP) can result in a variety of neurological symptoms including headaches. Although headaches are well known in elevated ICP, rarely, they can also result from reduced ICP. We report an interesting case of a woman who presented with severe positional headaches and imaging studies consistent with spontaneous intracranial hypotension.</p> <p>CASE REPORT: A 23 year-old woman was at work when, while bending over her desk, she experienced what she later described as the worst headache of her life. Her headache was characterized as a stabbing bitemporal pain associated with photophobia and nausea that was relieved significantly by lying supine. She denied recent travel or trauma. Neurological examination did not reveal focal deficits or nuchal rigidity; however, the patient had extensor plantar responses bilaterally. MRI of the brain revealed diffuse meningeal enhancement and low-lying cerebellar tonsils (interpreted by the radiologist as a Type 1 Chiari malformation) not seen on MRI performed 10 years earlier for reasons unrelated to the current admission. The patient’s presentation and imaging findings were consistent with spontaneous intracranial hypotension. She was treated with epidural blood patches, strict bedrest, and caffeine-containing analgesics with improvement of her headache. Upon reexamination, she had flexor plantar responses bilaterally, and resolution of the previous imaging findings on repeated MRI of the brain.</p> <p>DISCUSSION: Our patient presented with a relatively rare syndrome that affects approximately 5 in 100,000 persons annually, women being affected twice as frequently as men. Patients with spontaneous intracranial hypotension typically present with headache, neck pain, nausea, and vomiting. It is suspected that when CSF pressure decreases, the brain “sags” into the intracranial cavity causing traction on the supporting structures, sensory nerves, and bridging veins. When upright, this traction is enhanced by gravity causing the characteristic positional headache.</p> <p>Low CSF pressure results from CSF depletion due to leakage or hyper absorption. CSF leakage may occur due to a tear in the dural nerve sheath or rupture of a previously present spinal epidural or perineural cyst. CSF leaks occur almost exclusively in a spinal location with the majority taking place at the thoracic or cervicothoracic junction.</p> <p>The diagnosis is confirmed by evidence on imaging or a lumbar puncture (LP) demonstrating low CSF opening pressure. Brain MRI classically shows diffuse meningeal enhancement and “sagging” of the brain typically reported as a Type 1 Chiari malformation or cerebellar tonsil herniation as seen in our patient. The mainstay of treatment includes epidural blood patches, generous caffeine intake, and supportive care. Recognition of this syndrome is important in swiftly instituting appropriate therapy and preventing extraneous diagnostic workup and therapeutic measures.</p>

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Clinical Vignette	Yun Li	Mark Goldberg	Saint Barnabas Medical Center (Sunil Sapru)	Two acute myocardial infarctions associated with infectious endocarditis	<p>Septic emboli associated with infectious endocarditis (IE) have been known to cause infarctions in multiple organs, such as the brain, eye, and musculoskeletal system. Rarely do these emboli occlude the coronary arteries and cause myocardial infarction (MI). Here we present a patient of infectious endocarditis with systemic embolization who had two episodes of emboli-associated myocardial infarctions.</p> <p>Patient is a 75 year old African American female with past medical history of hypertension, type II diabetes who presented to the hospital with fever and delirium for one day. She denied chest pain, dyspnea. Upon arrival blood pressure was 170/60, heart rate was 108, and temperature was 102.2F. Her physical exam was unremarkable. Electrocardiogram(EKG) showed ST-elevation in inferior leads. Cardiac enzymes were elevated with CKMB at 48.1 and Troponin I at 1.110. There is an elevated WBC count at 14.1 with bandemia, and worsened serum creatinine at 2.27 from baseline at 1.5. Cardiac catheterization was not performed at the time due to acute kidney injury. Aspirin, metoprolol, atorvastatin, intravenous heparin and antibiotics were initiated. On the next day cardiac enzymes trended down, and EKG ST-changes resolved. Blood culture grew methicillin-sensitive staphylococcus aureus(MSSA). Antibiotics were switched to nafcillin. Transesophageal echocardiogram(TEE) showed vegetations and calcifications of mitral valve anterior and posterior leaflets causing severe mitral regurgitation. Eight days later, patient developed acute respiratory failure. Troponin was again elevated to 4.79 and CKMB remained normal. EKG shows normal sinus rhythm with no ST changes. Cardiac catheterization indicated 80% stenosis in mid-LAD and none in other coronary arteries. Patient underwent emergent mitral valve replacement surgery. Post-op EKG was unchanged, and echocardiogram showed preserved EF and no wall motion abnormalities. Her entire hospital stay was also complicated by pericardial effusion, embolic stroke, left lower limb ischemia requiring below knee amputation, acute renal failure requiring hemodialysis, respiratory failure requiring mechanical ventilation. Pt was discharged with tracheostomy and gastrostomy to a long-term acute care facility, where she completed six weeks of intravenous nafcillin treatment.</p> <p>According to the proposed National cerebral and cardiovascular center criteria for clinical diagnosis of coronary embolism, this patient meets one major criterion and ≥ 2 minor criteria, and receives definite diagnoses of coronary embolisms for both MI events: an STEMI and a NSTEMI respectively. It is also likely that the NSTEMI event was type II MI secondary to sepsis. Medical treatment for acute coronary syndrome (ACS) successfully preserved myocardial functions from ischemic infarct through both MI events, and should remain standard of care when coronary angioplasty or thrombectomy were considered high risk in IE patients with coronary embolisms.</p>

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Clinical Vignette	Sakthivelavan Duraipandian	Praveen Kandula	Saint Barnabas Medical Center (Sunil Sapru)	Thymoglobulin induced Multi-organ Dysfunction following kidney transplantation	<p>Release of cytokines by activated monocytes and lymphocytes may cause Cytokine Release Syndrome - CRS during administration of antithymocyte globulin. Although a rare complication, clinical presentation of CRS may range from an acute febrile reaction to multi-organ failure.</p> <p>A 42-year-old African American gentleman with non-oliguric ESRD due to lupus nephritis was admitted for a deceased donor kidney transplant (DDKT). Intraoperatively, rabbit anti-thymocyte globulin (rATG) induction at a dose of 2mg/kg was initiated after premedication with acetaminophen, diphenhydramine and methylprednisone. Post operatively he received immunosuppression with tacrolimus, mycophenolate and prednisone. During the course of rATG infusion, he became tachycardic, hypertensive, tachypneic, febrile, hypoxic and reduced urine output. His signs resolved with conservative management. On POD 1, tacrolimus level was elevated at 20.1 ng/ml and further dosing was held. A nuclear renal scan revealed adequate perfusion to allograft. Transplant ultrasound revealed no abnormalities. He received the second dose of rATG on POD1. Twelve hours after infusion patient was noticed to have leukocytosis with bandemia, severe thrombocytopenia (31), hyperkalemia (6.9) and metabolic acidosis. He became unresponsive, hypoglycemic, severely hypoxic and hypotensive. Further investigations revealed coagulopathy, severe lactic acidosis, NSTEMI, hepatocellular injury and hypoxic respiratory failure. He was admitted to ICU for multisystem organ failure. There he was found to have evidence of DIC, worsening liver failure, marked right ventricular dilation, and respiratory failure. Thymoglobulin infusion was discontinued. He required emergent hemodialysis, endotracheal intubation and respiratory support. Work up for sepsis, drug induced thrombotic microangiopathy (TMA), pulmonary embolism and hypercoagulability was negative. He required multiple blood and platelet transfusions, empiric antimicrobials and supportive care. His hospital course was complicated by perinephric hematoma after transplant kidney biopsy (which showed ATN and no TMA), kidney laceration, transplant failure which ultimately led to transplant nephrectomy. Patient was transferred out of ICU on day 22, and was discharged from hospital after 43 days of hospitalization.</p> <p>Thymoglobulin reactions documented from literature include fever (46%), nausea (29-37%), vomiting (20%), myalgia (11-20%), hypertension (27-37%), tachycardia (23%), hypotension (10-16%), hyperkalemia (17-27%), acidosis (9%), thrombocytopenia (29-37%), leukocytosis (13%), anemia (12%), dyspnea (15-28%), pulmonary disease (12%), DIC (<1%), Liver failure (<1%), elevated transaminases (<1%).</p> <p>Thymoglobulin binds to T cell receptors causing destruction of T cells and release of cytokines which leads to severe SIRS as discussed above. This Cytokine release syndrome is known to have features including hypotension, tachycardia, MI, ARDS, and pulmonary edema. Our patient, interestingly had almost all the features of Thymoglobulin reaction with CRS and no alternative etiology. Early recognition and aggressive supportive care are critical in the management of this potentially catastrophic condition.</p>

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Clinical Vignette	Rahul Thampi	Sunil Sapru MD, Aimen Liaqat MD	Saint Barnabas Medical Center (Sunil Sapru)	Chronic Intestinal Pseudo Obstruction - A possible initial manifestation of Systemic Lupus Erythematosus	<p>INTRODUCTION: Chronic intestinal pseudo obstruction (CIPO) is uncommon in Systemic Lupus Erythematosus (SLE) but it may be the presenting feature in a significant proportion of those who manifest it. This entity responds well to immunosuppressant treatment but can be life threatening if not treated promptly. A high level of clinical suspicion is vital and can prevent unnecessary invasive diagnostic procedures and surgical intervention.</p> <p>CASE DESCRIPTION: A twenty five year old lady with no significant medical history presented with a 6 month history of intermittent abdominal pain, nausea and vomiting worsening over 1 week. Physical exam revealed soft, distended abdomen with minimal epigastric tenderness and hypoactive bowel sounds. Abdominal obstructive series showed non-specific bowel distention but no signs of mechanical obstruction. Liver function tests were suggestive of biliary stasis. Ultrasound abdomen and CT abdomen were significant for fatty infiltration of the liver and mild splenomegaly. Endoscopy revealed esophagogastrroduodenitis. MRCP showed no signs of biliary stones or ductal dilatation. During the hospital course, she developed pancytopenia, acute kidney injury and acute respiratory failure. She was noted to have pericardial and bilateral pleural effusions on imaging. These raised concerns for an underlying autoimmune process. ANA, SSA, SSB and anti-dsDNA antibody titers were elevated. She was diagnosed with SLE at this point and was started on high dose prednisone. Her symptoms and pancytopenia improved significantly in the next few days. She was discharged on prednisone taper and subsequently transitioned to plaquenil monotherapy.</p> <p>DISCUSSION: CIPO is characterized by signs and symptoms of bowel obstruction in the absence of mechanical occlusion. It is an uncommon presentation in SLE and was first described as a severe form of SLE in 1998. In the largest retrospective study done to date, 57% of the cases presented with CIPO at the time of diagnosis and 82 out of the 85 cases were female. Most cases of CIPO are initially misdiagnosed resulting in delayed treatment (1). Pathophysiology is linked to smooth muscle dysfunction due to vasculitis. Although there is no specific antibody associated with SLE-CIPO, there is a higher prevalence of positive anti-SSA and anti-SSB antibodies. There is a significant association with smooth muscle involvement of the renal pelvis, ureters and biliary tract and these cases generally have unfavorable outcomes. Lupus related CIPO responds very well to treatment with high dose steroids and immunosuppressants including azathioprine, cyclosporine and cyclophosphamide. When it is the initial presentation of SLE, it can be a challenging diagnosis. It is imperative to have a comprehensive understating of this disease entity to facilitate early diagnosis and treatment.</p>

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Clinical Vignette	Muhammad Masoodul Haq	Abhishek Bhurwal, Keith Hawthorne, Sunil Sapru	Saint Barnabas Medical Center (Sunil Sapru)	Carcinoid tumor and ventricular tachycardia - A true association?	<p>Introduction: Carcinoid tumors are rare neuroendocrine malignancies that secrete multiple bioactive substances. The usual cardiac manifestations of carcinoid heart disease are well described but it is rare to have ventricular arrhythmia as a presenting feature of carcinoid tumors. We report a patient who developed serious ventricular tachycardia in the setting of a carcinoid tumor.</p> <p>Case description: A 61 year old gentleman with hypertension, diabetes mellitus, and bullous lung disease, presented with recurrent episodes of palpitations. He denied chest pain, dyspnea, or diaphoresis. He reported similar episodes two years ago when he was treated for ventricular arrhythmia. Physical examination on admission was unremarkable. Initial EKG showed normal sinus rhythm with no ST segment changes. On telemetry monitoring, recurrent episodes of monomorphic non-sustained ventricular tachycardia were noted and intravenous amiodarone drip was started. Chest radiograph revealed a large right sided hydro-pneumothorax which was treated with a video assisted thoracic surgery (VATS) procedure and resection of the bullous lung lesion. Cardiac catheterization for evaluation of coronary artery disease in the setting of multiple risk factors was normal and cardiac MRI was unremarkable. The resected lung lesion was revealed to be a well differentiated neuroendocrine tumor with the cells being positive for synaptophysin and chromogranin A. The Ki proliferation index of the tumor was less than 5% indicating slow growth. The search for a primary lesion was negative. A 24-hour urinary 5 HIAA study, performed one week after the resection, showed undetectable levels of serotonin. The patient did not get any further episodes of palpitations.</p> <p>Discussion: Carcinoid heart disease occurs in 50 percent of patients with carcinoid syndrome. Right side of the heart is most commonly affected, typically manifested by right sided heart failure and may be the initial presentation of carcinoid disease in as many as 20 percent of patients. Our patient had multiple episodes of ventricular tachycardia giving rise to repeated syncopal episodes. One of the differential diagnoses was carcinoid syndrome associated with tumor causing the arrhythmia. This hypothesis was substantiated by the fact that the episodes of ventricular tachycardia resolved after removal of the carcinoid tumor and he had no further episodes of syncope. This case is unique in that the carcinoid tumor was associated with symptomatic ventricular tachycardia without any evidence of myocardial ischemia or any features of typical carcinoid heart disease. We propose that a further exploration of the association between carcinoid tumor and arrhythmia is warranted to better define the relationship.</p>

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Clinical Vignette	Ciandra D'souza	Michael Loy DO; Benjamin Hixon BS ; Frantz Pierre-Louis MD	Saint Barnabas Medical Center (Sunil Sapru)	DRESS Syndrome: A Rare Reaction to a Beta-Lactam	<p>Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), also known as Drug-Induced Hypersensitivity Syndrome (DIHS) is an adverse drug-induced reaction that occurs in 1 in 10,000 drug exposures. The agents most commonly implicated are aromatic anticonvulsant drugs and allopurinol. Presenting symptoms include skin rash, elevated liver enzymes, hypereosinophilia and lymphadenopathy occurring 2-6 weeks after initiation of the causative drug.</p> <p>A 38-year-old female was admitted one month prior with paranasal sinusitis with cavernous invasions leading to brain abscesses with subsequent meningioencephalitis and acute fronto-temporal infarcts which ultimately culminated in Streptococcus anginosus and Streptococcus viridans bactremia. After stabilization in the hospital, she was discharged to a rehabilitation facility on ceftriaxone. It was noted she was clinically improving, however, she developed a pruritic rash approximately three weeks after the initiation of ceftriaxone. Repeat blood work showed elevation of her liver functions greater than three times the upper limit of normal, and around this time she also began spiking fevers, at which point she was sent to the emergency department. Laboratory work indicated leukocytosis, eosinophilia and transaminitis. Physical exam demonstrated facial and peri-orbital edema and a generalized non-blanching, maculo-papular erythematous rash on her upper chest and bilateral upper and lower extremities. However, no laryngeal edema was noted. At this time, ceftriaxone was discontinued and vancomycin and meropenem were initiated. Her clinical course was complicated by admission to the intensive care unit for worsening clinical condition of the aforementioned signs and symptoms, as well as, hypotension and tachycardia, lactic acidosis and acute renal injury. Systemic steroids were started and antibiotics were changed with subsequent improvement. She was ultimately discharged back to the rehabilitation facility on clindamycin and levofloxacin.</p> <p>DRESS syndrome caused by beta-lactams is extremely rare. A Japanese consensus group established 7 diagnostic criteria for DRESS: 1) maculopapular rash developing greater than 3 weeks after initiating a certain drug, 2) prolonged clinical symptoms 3) fever, 4) leukocyte abnormalities, 5) elevation of liver enzymes, 6) lymphadenopathy and 7) reactivation of HHV 6 in the second to third week of these symptoms. A definitive diagnosis requires all 7 criteria to be met, while a probable diagnosis requires 5 of the 7 criteria to be met. Common differential diagnoses include Steven Johnsons Syndrome, Toxic Epidermal Necrolysis, Hypereosinophilic Syndrome, Kawasaki disease and Still's disease. Management of DRESS is primarily by removal of the inciting agent and skin care. The use systemic steroids in the case of end organ damage, while utilized, remains controversial.</p>

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Clinical Vignette	Rachna Kapoor	Hersh Shah MD, Shanojan Thiyagalingam MD, Padmaja Kodali MD	Saint Barnabas Medical Center (Sunil Sapru)	IgA myeloma with increased anion gap, pseudohyperphosphatemia and pseudohyponatremia	<p>Introduction: Multiple myeloma (MM) is a hematopoietic malignancy characterized by proliferation of a single clone of plasma cells resulting in production of monoclonal immunoglobulins or paraproteins (IgG, IgA or IgM). With elevation in serum paraprotein levels the potential for erroneous laboratory results increases secondary to interference of paraproteins with colorimetric assays. Paraproteins can alter anion gap (AG) depending on their cationic or anionic properties. A decreased AG is usually observed in MM, however an increased AG is a rare presentation. We report an unusual presentation of IgA myeloma with an increased anion gap and spurious laboratory findings that included pseudohyperphosphatemia and pseudohyponatremia.</p> <p>Case Presentation: Fifty-seven-year-old female with a history of hypertension presented with progressively worsening shoulder and back pain. Physical exam was significant for diffuse bone tenderness. Bone skeletal survey showed multiple punched out lesions, diffuse demineralization and compression fracture in T12 and L1. Laboratory findings were significant for anemia (hemoglobin 6.2 g/dL) with peripheral blood smear revealing rouleaux formation, hyponatremia 126 mmol/L, elevated creatinine 3.23 mg/dL with blood urea nitrogen 45 mg/dL, albumin of 3.1 g/dL, hypercalcemia 15.5 mg/dL (corrected calcium 16.2) and marked hyperphosphatemia 20.5 mg/dL. Patient had high serum viscosity 3.1 centipoises, elevated total protein levels 11.9 g/dL, elevated IgA 4598 mg/dL, low IgG 580 mg/dL and low IgM 24 mg/dL. Inorganic phosphorous measurements on deproteinized serum samples revealed normal phosphorous levels. Parathyroid hormone and 25-OH vitamin D levels were normal. An increased AG of 24 was observed after correcting for albumin. An electrocardiogram revealed Osborn waves in the setting of severe hypercalcemia. Serum, urine protein electrophoresis and bone marrow biopsy were performed and she was diagnosed as IgA kappa type multiple myeloma. MRI spine revealed rostral-caudal cord edema spanning T10-T12 secondary to significant cord compression requiring emergent intravenous steroids. Patient was managed with intravenous fluids and pamidronate for hypercalcemia. Due to refractory nature of hypercalcemia patient required urgent hemodialysis.</p> <p>Discussion: Anion gap gets significantly altered depending on type of MM. Low AG is commonly observed in IgG myeloma due to its cationic property. In contrast, IgA myeloma can have an increased or a normal AG attributed to its anionic property. Pseudohyperphosphatemia, not a widely known phenomenon, can result from laboratory error. Paraproteinemias can also result in pseudohyponatremia due to reduction in plasma water fraction secondary to increased total protein concentration. Our case not only demonstrates an unusual presentation of MM with increased AG but also highlights the importance of recognizing paraproteinemias as a potential source of discrepant laboratory results before instituting inappropriate investigation and therapy for hyperphosphatemia or hyponatremia.</p>

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Clinical Vignette	Rahul Thampi	Matthew Bushman MD, Lauren McFadden and Anthony Carlino, MD, PhD	Saint Barnabas Medical Center (Sunil Sapru)	Unilateral Painful Ophthalmoplegia: A Case of Tolosa-Hunt Syndrome	<p>Introduction: Unilateral painful ophthalmoplegia is a common symptom that can be caused by the very uncommon Tolosa-Hunt Syndrome. It's a rare disorder first described in 1954, characterized by granulomatous inflammation within the cavernous sinus or superior orbital fissure resulting in orbital pain and paralysis of cranial nerves III, IV, and VI. It is a diagnosis of exclusion; ischemic, infectious, inflammatory, autoimmune and neoplastic conditions must be considered. Increased awareness of Tolosa-Hunt Syndrome can facilitate early identification and treatment.</p> <p>Case Description: A 28-year-old Hispanic male with no medical history presented with sudden onset ptosis of the left eyelid associated with mild swelling and pain. He reported a left sided retro-orbital headache and epiphora of one week duration. He denied any loss of vision. Physical examination was significant for mild conjunctival injection, left eye ptosis without proptosis. Pupils were equal and reactive to light bilaterally and visual acuity was uncompromised. Left extraocular movements were limited in all directions. Other cranial nerves were grossly intact. Laboratory data was significant for mildly elevated ESR. MRI brain and orbits were unrevealing. MRA head showed no evidence of aneurysm and MRV did not display any sinus thrombosis. He was treated for possible cellulitis without any improvement. The possibility of unilateral ocular myasthenia gravis was treated with pyridostigmine with no effect. Comprehensive CSF evaluation including studies for Lyme, Cryptococcus, VDRL, angiotensin converting enzyme, oligoclonal bands and viral antigens was negative. Lack of a concrete diagnosis and failure to improve with multiple treatments prompted further review of previous imaging. Upon review, a homogeneously enhancing left cavernous sinus with a slightly convex lateral margin extending to the left orbital apex was noted consistent with a granulomatous process. On this basis, he was started on high dose prednisone and began to show improvement of ptosis and ocular movements within two days.</p> <p>Discussion: Painful ophthalmoplegia is caused by a variety of conditions including cavernous sinus thrombosis, internal carotid aneurysm, primary and metastatic tumors, lymphoma, sarcoidosis, ophthalmoplegic migraine, vasculitis, or infectious processes. Tolosa-Hunt syndrome is a rare cause with an annual incidence of one in a million. Cases have been reported across age groups; it affects men and women equally. While few cases have been mentioned in conjunction with systemic inflammatory conditions such as SLE, there is no associated systemic inflammatory response. The etiology remains unknown. MRI findings include enhancement and convexity of the wall of the cavernous sinus, however, these MRI findings are nonspecific. Despite that, direct tissue biopsy is seldom obtained as risks often outweigh the benefits. Ultimately, glucocorticoids have both diagnostic and therapeutic potential. Close follow up and repeat imaging is crucial to determine treatment is working effectively and other etiologies are ruled out.</p>

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Clinical Vignette	Najaf Asrar		Saint Barnabas Medical Center (Sunil Sapru)	Iced In: A Rare Case Of Focal Neurological Deficits In The Setting Of Hypoglycemia	<p>Hypoglycemia can present with an array of signs and symptoms, most common of which are diaphoresis, anxiety, palpitations, confusion and occasionally hemiplegia and aphasia. The most feared outcome, brain death, is caused by brain fuel deprivation leading to functional brain failure. If not treated, hypoglycemia may lead to irreversible changes. This case demonstrated a less common presentation of severe hypoglycemia mimicking a cerebral stroke.</p> <p>The patient is a 64 year old African-American woman with a history of steroid induced hyperglycemia; she was taking prednisone for four years following a renal transplant owing to hypertensive kidney disease. At home she uses insulin and nebulizer treatments for her COPD. Due to her failing transplant, hemodialysis was re-initiated; she was often non-compliant with the dialysis schedule and was admitted multiple times with fluid overload. During her hospital stay, she was found to be acutely nonverbal and with a complete left sided hemiparesis. Moreover, she was able to follow commands and was aphasic. Her finger stick glucose level was 43 mg/dL. She was then given glucose 25 g iv; within five minutes an immediate transition was noted as her left upper extremity began to twitch. This twitching soon led to a return of coordinated movements with full strength and the aphasia resolved. Repeat finger stick glucose level was 173 mg/dL one hour later. Due to her rapid improvement no imaging was done. Throughout her hospital course she had no evidence of infection, nor was she on any insulin products which could have led to this hypoglycemia.</p> <p>Hyperglycemia and hypoglycemia can both cause symptomatic neurological status changes. Therefore, changes in blood glucose concentrations should always be considered when evaluating patients with altered mental status or suspected strokes. Hypoglycemia can mimic middle cerebral artery strokes as we have seen in this case. It is important to note that the possibility of a transient ischemic attack in this case cannot be excluded. Failure to check blood glucose levels in the setting of an apparent stroke may lead to the unnecessary and potentially life threatening administration of tissue plasminogen activator. This case demonstrated a rare cause of severe hypoglycemia mimicking a cerebral stroke that resolved completely after the administration of glucose.</p>

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Clinical Vignette	Rachna Kapoor	Mark Jay Zucker MD, Indu Sabnani MD	Saint Barnabas Medical Center (Sunil Sapru)	A Rare Disorder with Rare Presentation EBV negative Post Transplant Lymphoproliferative Disorder Presenting as Cold Agglutinin D	<p>Introduction: Post-Transplant Lymphoproliferative Disorder (PTLD) is a serious complication occurring in the setting of solid organ or stem cell transplantation as a result of immunosuppression. Epstein-Barr Virus (EBV) negative PTLD is a rare but distinct entity. Cold agglutinin disease (CAD) is a rare autoimmune hemolytic anemia (AIHA) due to antibodies against erythrocytes that react at low temperatures. We present the first known adult case of severe AIHA due to cold agglutinins associated with EBV negative PTLD following orthotopic heart transplantation (OHT).</p> <p>Case Presentation: A 56 year-old female with a history of anti-cardiolipin antibody syndrome and OHT in 2012 presented with marked weakness and wide spread livido reticularis across trunk and extremities. Laboratory findings were significant for severe hemolytic anemia with a hemoglobin of 4.5 g/dl, reticulocytes of 7.6%, haptoglobin of less than 8 mg/dl, LDH of 247 U/L, total bilirubin of 3.8 mg/dl and direct bilirubin of 0.3 mg/dl. A peripheral smear showed marked cell agglutination and serology was strongly positive for cold agglutinins. A CT scan of the abdomen revealed thickening of the ileum, cecum and mesenteric lymphadenopathy. A laparoscopic biopsy of mesenteric lymph nodes revealed EBV negative monomorphic PTLD - diffuse large B cell lymphoma (DLBCL). The patient was staged as stage II E monomorphic PTLD with negative bone marrow. EBV IgM and quantitative PCR were negative. Direct antiglobulin test (DAT) was positive and no cryoglobulins were detected. She was managed with steroids, blood transfusions with blood warmer and eventually treated with six cycles of R-CHOP chemotherapy. The patient has been in complete remission with complete disappearance of cold agglutinin antibody.</p> <p>Discussion: To our knowledge this is the first documented case of an adult EBV negative PTLD patient associated with cold agglutinin hemolytic anemia. EBV negative monomorphic PTLD and CAD both are challenging disorders for management purpose. However, both diseases can be treated with Rituximab based immune chemotherapy. Long term complete and sustained remission of CAD is uncommon but our patient has remained in remission for last four years. High vigilance and a low threshold for diagnosis of PTLD should be maintained as it is necessary to initiate prompt therapy in a disorder that can be potentially fatal.</p>

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Clinical Vignette	Chun Tu	Kristen Fless, Ashraf Raslan	Saint Barnabas Medical Center (Sunil Sapru)	Bloody cough! Rheumatoid vasculitis manifesting as Diffuse Alveolar Hemorrhage	<p>Introduction: Rheumatoid arthritis (RA) is a systemic autoimmune disease most prominently affecting synovial joints. Vascular involvement in RA (also called rheumatoid vasculitis RV), although relatively rare, has diverse clinical manifestations and high mortality and morbidity.</p> <p>Case: 63 year old African American non-smoking female with history of seropositive RA for more than 30 years was admitted to the hospital in 2014 with acute renal failure. Her RA was poorly controlled as the patient was not on any treatment for the past 3 years due to poor response to disease modifying anti-rheumatic drugs (DMARDs) and biologic agents. She had classic rheumatoid deformities of her hands and rheumatoid nodules on physical examination. Renal biopsy revealed pauci-immune crescentic glomerulonephritis (PICG). The laboratory examination showed high titer rheumatoid factor (RF), weak positive perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA) antibody and negative cytoplasmic anti-neutrophil cytoplasmic antibodies (c-ANCA) and anti-glomerular basement membrane (GBM) antibodies. Systemic lupus was excluded with biopsy result and normal levels of complement C3 and C4. Patient was treated with high dose intravenous steroid and cyclophosphamide, but her renal function failed to improve and she was started on hemodialysis. Later in the hospitalization, patient complicated with pneumonia and hemoptysis which improved with addition of antibiotics. In September 2016, she presented to the hospital with shortness of breath and frank hemoptysis progressing to respiratory failure requiring intubation. Her RA was still poorly controlled with evidence of synovitis. Auto-immune antibody profile was similar to 2014 hospitalization. Chest X-ray showed new bilateral fine reticular opacity and CT scan showed diffuse interstitial opacities. Bronchoscopy revealed large volume of progressive bloody lavage, consistent with diagnosis of diffuse alveolar hemorrhage (DAH). Patient's hypoxia and hemoptysis gradually improved after initiation of high dose steroid treatment. Other causes of DAH such as coagulopathy, heart failure, or adult respiratory distress syndrome were excluded. Since discharged home, patient has been on low dose steroid with resolution of hemoptysis. She does not require oxygen whereas she required home oxygen before treatment with prednisone. Of note, patient had several additional hospitalizations for pneumonia with hemoptysis. Three out of her four chest CT reports showed bilateral ground glass changes consistent with changes in acute phase of rheumatoid vasculitis . It is possible that the patient had ongoing pulmonary rheumatoid vasculitis that has not been recognized nor treated until her presentation with DAH.</p> <p>Conclusion: We report this rare case of rheumatoid vasculitis presenting as DAH in a patient with history of pauci-immune glomerulonephritis and recurrent pneumonia.</p>

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Clinical Vignette	Nilofer Khan	Ramsey Asmar MD, Sunil Sapru MD	Saint Barnabas Medical Center (Sunil Sapru)	Thyroid metastasis in triple-negative breast cancer: an unusual presentation	<p>Triple-negative breast cancers (TNBC) are defined by the absence of estrogen and progesterone receptors, as well as the absence of human epidermal growth factor receptor 2 (HER2) overexpression. It is characterized by a more aggressive clinical course, higher risk of local and distant relapse, and shorter overall survival when compared to other types of breast cancer (1). Data regarding specific sites of metastasis in patients with TNBC are very limited (2). Here, we describe a patient who presented with highly aggressive TNBC with metastatic involvement of the thyroid.</p> <p>A 46-year-old African American woman with no medical history discovered a rapidly growing neck mass in early August, 2016. By October patient presented to her local hospital with progressive dyspnea. CT scan demonstrated an exophytic thyroid mass with significant tracheal narrowing. She was being transported to our hospital for an elective thyroidectomy. En route, she developed respiratory distress and was emergently intubated. Further imaging confirmed a necrotic mass replacing the entire thyroid gland, as well as a necrotic right supraclavicular lymphadenopathy and multiple bilateral pulmonary nodules consistent with metastatic disease. CT also revealed a 4.3 cm heterogenous left breast mass. Ultrasound-guided aspiration of the thyroid and core biopsy of the breast was performed. She subsequently underwent total thyroidectomy, extensive tumor debulking and tracheostomy for airway protection. Of note, about 2 weeks prior to her admission she had an excisional biopsy of a suspicious right temporal scalp lesion.</p> <p>The patient's breast biopsy revealed a diagnosis of poorly-differentiated TNBC with myoepithelioid characteristics. To our surprise, immunohistochemical analysis of the thyroid and scalp lesions demonstrated metastatic breast cancer. Confirming the tissue diagnosis proved challenging and multiple pathologists reviewed the biopsy. Anaplastic and medullary thyroid cancer were initial considerations based on the clinical presentation. Dermal clear cell mesenchymal neoplasm was also considered based on the histologic appearance of her scalp lesion but the very high mitotic activity/Ki-67 and visceral metastases that was seen in this case were not in favor of this diagnosis. Pulmonary nodule biopsy was deferred. Staging was completed with a CT of the abdomen/pelvis and bone scan and brain MRI. Both negative for evidence of further spread. The patient was promptly initiated on carboplatin and paclitaxel chemotherapy, given her visceral crisis and need for rapid disease control.</p> <p>Metastasis to the thyroid gland is rare, but has been reported (3). While bone is the most common site of metastasis in breast cancer overall, basal-like breast tumors (including TNBC) are associated with significantly higher rates of brain, lung and distant nodal metastasis, and relatively lower rates of bony involvement (4). Cases such as this may incline clinicians to assess the possibility of metastatic disease when encountering new lesions at a site rarely associated with metastases.</p>

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Clinical Vignette	Zinashbezu Gizaw		Saint Barnabas Medical Center (Sunil Sapru)	Chronic tophaceous gout involving the spine	<p>Introduction: Gout is a relatively common condition and its prevalence in the United States is increasing (i.e, 2.9/1000 in 1990 to 5.2/1000). Although there are documented cases of tophus formation about the spine, this is rare. Moreover, there is no non-invasive method of definitively identifying spinal tophi. Since the first case of gout in the spine was published in the 1950's , a series of cases has been reported.</p> <p>Case report: A 44 year old man with a history of hypertension, spinal stenosis and poorly controlled tophaceous gout presented to the emergency department with worsening back pain, fever and difficulty ambulating owing to swelling and pain at the left ankle joint. He had tophaceous gout for more than ten years and was not on any uric acid lowering therapy. The patient was admitted twice within the previous two years with similar symptoms that never fully resolved necessitating that he use a cane to ambulate. Additionally, he noted recent urinary incontinence. On physical examination, he had multiple tophi of the hands, feet, elbow and left ear as well as a tender, swollen and warm left ankle joint. Cranial nerves were intact and no visual abnormality was noted. There was bilateral lower extremity muscle spasm and rigidity with decreased power distally and positive Babinski sign. Laboratory data showed leukocytosis, elevated ESR, CRP, serum creatinine and an elevated uric acid level of 8.5 mg/dl. All other investigations for infectious disease, autoimmune processes and para-neoplastic syndromes were negative. CSF analysis was negative for oligoclonal bands and NMO antibody was negative as well. MRI of the lumbar spine showed straightening of the lumbar lordosis and epidural lipomatosis producing deformity of the thecal sac and moderate degeneration of the facets on the left side at L4-L5. There was also a small focal gadolinium enhancing intramedullary lesion. The patient was treated with colchicine and high dose steroids for four days with significant improvement of spasms and pain. There was partial resolution of urinary incontinence.</p> <p>Discussion: Gouty involvement is more common in the lumbar spine as opposed to other areas of the spine and could present as spinal stenosis, lumbar radiculopathy, spondylolisthesis or cauda equina syndrome. There are some reported cases of spinal gout with symptoms mimicking spinal infection. Although imaging studies are non-specific, a high index of suspicion in the presence of a history of gout along with clinical signs and symptoms and an elevated uric acid level can suggest the diagnosis. Invasive studies can be avoided especially in patients with tophi.</p>

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Clinical Vignette	Sagar Harwani	Arun Minupuri, Anthony Carlino MD	Saint Barnabas Medical Center (Sunil Sapru)	PPI Induced Hypomagnesemia Causing Seizure	<p>Introduction: Proton pump inhibitors (PPIs) are the second most commonly prescribed class of medications globally with annual estimated health-related costs of >11 billion dollars in the United States alone. They are prescribed as the mainstay of therapy for many gastric related disorders, including dyspepsia, GERD, peptic ulcer disease, and H. Pylori. PPIs are generally well-tolerated and have relatively few adverse effects. Complications arise with the long term-usage of PPIs, and include an increased risk of infections (Clostridium Difficile and pneumonia), bone fractures, and electrolyte abnormalities. There is a paucity of literature on the long-term effects of PPIs; however, emerging case reports point towards a relationship between PPIs and clinically significant hypomagnesemia.</p> <p>Case Presentation: A 90-year-old male with a past medical history of GERD, hypertension, hyperlipidemia, and hypothyroidism presented to the emergency department with new-onset witnessed tonic-clonic seizure with subsequent seizure episodes noted while in the emergency department. On initial exam, he was afebrile, hemodynamically stable, with resolving post-ictal lethargy and confusion. Lab work revealed macrocytic anemia (hemoglobin 8.8), hypocalcemia (corrected calcium 7.7 mg/dL), and significant hypomagnesemia (magnesium 0.6 mg/dL). 24-hour urinary magnesium was 8.0 mg/dL. EKG showed normal sinus rhythm with prolonged QTc of 515 milliseconds. CT Head was unremarkable for age. It was noted that the patient was taking both Famotidine 20mg twice daily for over three years and Pantoprazole 40mg daily for an unknown period of time. Electrolytes were aggressively repleted, all proton pump inhibitors were discontinued, and he was started on Lamictal. VEEG monitoring showed no further seizure activity. Over the course of admission, magnesium and other electrolytes stabilized, and patient was seizure free.</p> <p>Discussion: We present a patient with new-onset seizures due to hypomagnesemia and secondary hypocalcemia while on high dose PPI therapy for over three years with more recent dual PPI therapy, most likely due to medical error. PPI-induced hypomagnesemia has become an increasingly recognized phenomenon over the past decade.</p> <p>Currently, there is no accepted mechanism behind PPI induced hypomagnesemia. Early reports suggested that PPI induced hypomagnesemia was not due to renal magnesium wasting, but rather decreased gastrointestinal absorption. Our patient's 24-hour urinary magnesium excretion was low, suggestive of inadequate magnesium intake and/or gastrointestinal losses. More recent studies have focused on PPI use on the TRMP6 transporter, the major pathway for intestinal magnesium absorption. Intracellular magnesium and pH regulate TRPM6 activity. Since PPI therapy decreases gastric hydrogen proton secretion, thereby increasing lumen pH, PPI use could potentially decrease TRPM6 activity, resulting in decreased magnesium absorption.</p> <p>In March 2011, the FDA issued a safety alert warning providers of the risk of hypomagnesemia in patients who have been on long-term PPIs. Clinicians should regularly reassess the need of PPI therapy to prevent clinically significant adverse events.</p>

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Clinical Vignette	Meha Bhargava	Robert Zenenberg, DO	Saint Barnabas Medical Center (Sunil Sapru)	Glomerulopathy With Monoclonal Immune Deposits in a Patient With Underlying Autoimmune Disease	<p>Membranous nephropathy with monoclonal IgG deposits is an incredibly rare disease only described in 14 patients. Here we describe a case of monoclonal IgG1-kappa deposition associated with membranous morphology.</p> <p>A 54 year-old woman with past medical history of Sjogren’s syndrome presented with worsening renal function (proteinuria). She was treated for 6 years with Plaquenil and immunosuppressive agents including Prednisone, Methotrexate, Rituximab (last treatment September 2015) and Imuran. On initial presentation, physical examination was unremarkable and blood pressures were well controlled. Lab work from December 2015 showed a creatinine of 0.79 mg/dl; urinalysis showed 2+ protein by dipstick with no blood. There was no urine protein quantification. She had positive anti-histone antibodies, strongly positive anti- dsDNA (432), and an ANA titer of 1:320. Serum IgG and IgM were increased (20 and >100 U/ml, respectively). ESR was 70. C3 was normal with a depressed C4. Rheumatoid factor (RF) and CCP were both negative.</p> <p>Urinalysis in August 2016 showed 3+ protein, 2+ occult blood with a urine protein to creatinine ratio of 710 mg/g. Serum creatinine was 0.71 mg/dl. Serum C3 levels were normal with a depressed C4. Anti-dsDNA was now negative. Both RF and cryoglobulin were negative. ESR was 36 and CRP was 0.6. Serum light chain and immunofixation were negative.</p> <p>A renal biopsy was performed. Glomeruli basement membranes (GBM) were unremarkable. No clear cut mesangial hypercellularity, endocapillary proliferation, or neutrophil infiltrates were seen. There was moderate patchy tubular atrophy and interstitial fibrosis, accompanied by patchy mononuclear inflammatory cell infiltrate. Mild focal lymphocytic tubulitis was identified. Tubules contained PAS-positive casts. Congo red stain was negative.</p> <p>Immunofluorescence studies showed granular staining 3+ for IgG, 3+ for IgM, 3+ for k light chain, and 3+ for C3 in the GBM, but no significant staining for λ light chain, IgA, or C1. Immunofluorescence staining for γ-heavy chain subclasses showed a dominant 3+ granular staining for IgG1, with no staining for IgG2, IgG3, or IgG4. Electron microscopy revealed diffuse thickening of GBMs with numerous subepithelial and intramembranous electron-dense deposits. Diffuse mesangial electron dense deposits were seen which had a randomly oriented fibrillar substructure (average thickness 30nm) and lacked a central lumen. No extra-glomerular fibrillar deposits were seen. Podocytes showed 90% foot process effacement. Tubular BMs showed moderate diffuse thickening.</p> <p>Therefore, we present a case of Membranous Glomerulonephritis pattern with monoclonal IgG1-kappa organized fibrillar deposition in a patient with longstanding underlying autoimmune disease. This has been described in patients with underlying lymphoproliferative disorders (most commonly Chronic Lymphocytic Leukemia), however there is currently no evidence of overt lymphoproliferative disease in our patient. It is thus important to keep in mind the possibility of monoclonal deposition disease in a patient with underlying autoimmune disease and new kidney dysfunction.</p>

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Clinical Vignette	Ambreesh Chawla	Justin Mottaghi, MD, Austin Healy, Minha Kim, Srihdar Nambi, MD, Sunil Sapru, MD	Saint Barnabas Medical Center (Sunil Sapru)	A Case of SGLT-2 Inhibitor Induced Euglycemic Diabetic Ketoacidosis	<p>Diabetic Ketoacidosis (DKA) is a life-threatening complication of diabetes mellitus. While most cases of DKA present with hyperglycemia coupled with anion-gap metabolic acidosis and ketonemia, some cases, referred to as “Euglycemic DKA”, may present with normal blood glucose levels. Sodium-glucose cotransporter 2 (SGLT-2) inhibitors are a class of oral hypoglycemic agents which target the SGLT-2 transporter in renal proximal convoluted tubules, preventing reabsorption of glucose from urine into the bloodstream. Recent FDA warnings have directed attention to the potential risk of DKA in patients treated with these agents. We present a patient of Type 2 Diabetes Mellitus (T2DM) who was taking a SGLT-2 inhibitor and developed euglycemic ketoacidosis.</p> <p>A 38 year old male with a past medical history of T2DM accompanied by neuropathy, peripheral vascular disease and hypertension, presented to the ED with a three-day history of nausea and vomiting. His home medications included empagliflozin, metformin and insulin detemir. Our patient reported medication compliance and denied recent alcohol consumption. His temperature was 99.2F, BP 159/71 mmHg and pulse 118 BPM. On exam, patient appeared in mild distress with abdominal tenderness. Labs revealed an anion-gap of 24, blood glucose of 176 g/dL with a normal BUN, creatinine and lactic acid. Urinalysis revealed ketones and glycosuria. At this time, euglycemic DKA was highly suspected and patient was admitted to the MICU. DKA protocol was initiated including normal saline, dextrose 5% in water, and IV insulin drip at 0.1U/kg/hr with concurrent potassium supplementation. After his anion gap closed within 24 hours, our patient was switched to a basal and pre-meal insulin regimen and was downgraded to the medical ward.</p> <p>Euglycemic DKA is characterized by normal to mildly elevated blood glucose levels and can be missed due to the absence of profound hyperglycemia that is typically seen in DKA. SGLT-2 inhibitors have gained popularity in the management of T2DM as they provide potential added benefits in patients with dyslipidemia and hypertension. These agents lower glucose levels by increasing urinary glucose excretion, thus reducing the stimulus for pancreatic β-cell insulin secretion. Insulin normally acts to inhibit hormone-sensitive lipase, thereby decreasing the generation of free-fatty acids that serve as a substrate for β-oxidation and ketogenesis. With SGLT-2 inhibitors, marked glycosuria and resultant decreased insulin secretion leads to uninhibited lipolysis, increased β-oxidation and ketogenesis. As a consequence of decreased blood glucose levels, pancreatic α-cells are stimulated to increase glucagon secretion, further promoting lipolysis and ketogenesis. Management of SGLT-2 inhibitor euglycemic DKA follows the American Diabetes Association recommendations with focus on intravenous fluids, insulin and electrolyte management. However, initiation of dextrose infusion is imperative considering the euglycemic nature of the pathogenesis. Upon stabilization and subsequent discharge, patients should discontinue SGLT-2 inhibitors and be advised to maintain adequate hydration.</p>

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Clinical Vignette	Ambreesh Chawla	Justin Mottaghi, MD, Sunil Sapru, MD	Saint Barnabas Medical Center (Sunil Sapru)	The Hidden Challenges of Pyelonephritis in the Setting of ADPKD	<p>It has been well-documented that antimicrobial therapy in acute pyelonephritis requires careful consideration for patients with congenital urinary tract abnormalities. However, in patients with autosomal dominant polycystic kidney disease (ADPKD), there are unique challenges that clinicians must confront in the treatment of pyelonephritis, predominantly the issue of cyst penetration by antibiotics. We describe an interesting vignette of a patient with acute pyelonephritis for which diagnosing ADPKD was crucial to managing what was otherwise considered a routine case.</p> <p>Our patient is a 31-year old female with no significant past medical history who presented to the ED with a three-day history of severe abdominal pain, nausea and vomiting. Additionally, she complained of urinary frequency, dysuria, fevers and chills. On presentation, she was febrile at Tmax 102.8F with tachycardia of 140 BPM and hypotension of BP 92/66 mmHg. Right costovertebral angle and suprapubic tenderness were present on exam. WBC count was 16,600 g/dL with 31% bands. Urinalysis demonstrated nitrites, leukocyte esterase, and >10,000 bacteria. Cefepime and aggressive IV fluid resuscitation were initiated. Due to initial suspicion of intra-abdominal pathology, a CT-abdomen and pelvis was obtained which surprisingly revealed multiple renal cysts with internal septations, consistent with cystic kidney disease. Upon further questioning, a history of ADPKD in multiple first degree relatives was revealed. Urine culture was positive for pan-sensitive E. coli, however both sets of blood cultures were negative. Upon review of the literature, ciprofloxacin was initiated given its more effective cyst penetration. With continued antibiotic therapy and IV fluid resuscitation, our patient's fever resolved and hemodynamic instability markedly improved by hospitalization day two. On day three, patient was discharged home with a twelve day course of PO ciprofloxacin.</p> <p>ADPKD is a hereditary condition in which fluid-filled cysts form in the kidneys, often in association with a variety of extrarenal manifestations. Inherent to this condition is a natural decline in GFR, which is accelerated by genitourinary infection, uncontrolled hypertension and volume depletion. It has been well-established that patients with ADPKD have a high predisposition to urinary tract infections, with a lifetime incidence of 30-50%. Effective infection control has a beneficial role in decreasing the rate of progression to ESRD in such patients. Pyelonephritis in ADPKD patients can present unforeseen challenges to the clinician, in particular the selection of antibiotics with the ability to penetrate cyst membranes. Studies have demonstrated that fluoroquinolones, TMP-SMX and chloramphenicol are most effective in cyst penetration (with the latter rarely used in clinical practice). Our patient initially presented with what appeared to be a routine case of pyelonephritis. However, establishing her underlying diagnosis of ADPKD dramatically altered the course of her hospitalization, facilitating a rapid recovery and early discharge on PO antibiotics.</p>

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Clinical Vignette	Justin Mottaghi	Ambreesh Chawla, MD, Samar Hameed, MD, Sunil Sapru, MD, Frantz Pierre-Louis, MD	Saint Barnabas Medical Center (Sunil Sapru)	Gram Negative Sepsis and ARDS: Is the Bark Worse than the Bite?	<p>In spite of recent strides made in the diagnosis and management of sepsis via the Surviving Sepsis Campaign and other initiatives, there remains a wide spectrum of complications that present a significant challenge to clinicians. One of the most feared sequelae, of gram negative sepsis in particular, is acute respiratory distress syndrome (ARDS), a condition traditionally thought to portend a grave prognosis. However, there may be a subset of patients in which prompt identification of the causative microorganism and initiation of highly targeted antibiotics is associated with improved clinical outcomes. We present an interesting case of a patient with ARDS secondary to ESBL E. coli sepsis who went on to have an unusually rapid recovery.</p> <p>Our patient is a 47-year-old female with congenital urinary tract abnormalities, who presented to the ED complaining of fevers, dysuria and flank pain for three days. Tmax was 102.6 oF and pulse was 110 BPM with left costovertebral angle tenderness. Urinalysis demonstrated nitrites, leukocyte esterase, and >10,000 bacteria. WBC count was 18,700 g/dL with 27% bands. Cefepime and aggressive IV fluid resuscitation were initiated. Pre-admission blood cultures grew gram-negative rods, later identified as ESBL E. coli. On hospitalization day two, a recent outpatient urine culture revealed E. coli with only intermediate susceptibility to cefepime but very high susceptibility to carbapenems. Cefepime was then discontinued and meropenem was started. On the following day, the patient abruptly desaturated to 79% on ambient air. ABG analysis on non-rebreather mask showed 7.41/29/153/18.4 and CT of the chest revealed bilateral alveolar infiltrates with pulmonary edema. The patient's respiratory status continued to decline, ultimately requiring MICU admission. The patient had a MICU stay of only three days, never requiring intubation, and was weaned off high-flow. She was later discharged home with two weeks of ertapenem via PICC and instructed to follow-up with pulmonology and infectious disease.</p> <p>In gram negative sepsis, endotoxin causes pulmonary endothelial injury and subsequent diffuse alveolar damage by initiating a cascade of potent inflammatory mediators. This catastrophic process leads to alveolar leak, hypoxemia, and reduced compliance, all of which are hallmarks of ARDS. Our patient developed features of ARDS in the setting of ESBL E. Coli sepsis, with PaO2/FiO2 ratio of 255 and characteristic radiographic findings. While the overall mortality of ARDS is 26-58%, ARDS with diffuse alveolar damage (as in sepsis) is known to carry an even greater mortality risk. However, our patient had a short MICU stay, never requiring intubation or even the use of NIPPV. On discharge, our patient was ambulatory and sent home without supplemental oxygen. Thus, it is remarkable that our patient had such a favorable clinical outcome which, in practice, may allow clinicians to have a more optimistic outlook on such patients.</p>

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Clinical Vignette	Hamad Ashraf	Sasha Ivanovic MD, Andreas Savopoulos MD	Saint Barnabas Medical Center (Sunil Sapru)	Hematologic Manifestation of Sarcoidosis	<p>Sarcoidosis is a disease of unknown etiology characterized by noncaseating granulomas which cause a wide spectrum of variable clinical manifestations. Sarcoidosis has been associated with various autoimmune diseases but hematologic phenomenon is uncommonly seen.</p> <p>50-year-old African American female presented to the hospital for a bruise after a large object had fell on her and scattered petechial rash in both lower extremities. She also reported shortness of breath on exertion and recent cold like symptoms. During the hospital admission platelet count was found to be 3000. She underwent CT scan of chest and abdomen that found pulmonary nodules throughout both lungs largest being 7mm, bilateral hilar lymphadenopathy and mild hepatoplenomegaly. She was treated with prednisone therapy which was ineffective in treating her thrombocytopenia and subsequently placed on IVIG that raised her platelet count. Bone marrow biopsy at the time was negative. She was lost to follow up thereafter without being treated. Patient presents to the hospital 4 years later with 2 large areas lower extremity ecchymosis located in the left leg, knee and calf. She had symptoms of shortness of breath on exertion that is worsening, chronic cough and 30-pound weight loss. Upon examination petechia were found palate and were also found on the right arm on the location of the blood pressure cuff. Evidence of clots were evident in the nares. Abdominal examination found hepatosplenomegaly. Platelet count was 6000 without schistocytes formation. Microcytic anemia was found on CBC. She had increase in LDH 267, ferritin 156 and ACE 357. Chest x ray found bilateral apical reticulonodular opacities and bilateral hilar lymphadenopathy. CT scan chest revealed cystic lung changes with honeycomb appearance in lung apices and bilateral septal thickening. Lymphadenopathy located in the cervical, supraclavicular, mediastinal, subcarinal areas. Worsening of bilateral hilar adenopathy since previous admission. Increase in size of splenomegaly. Blood flow cytometry for lymphoproliferative disorder was negative. PFT found a restrictive pattern and decreased DLCO. She was treated with 2 units of platelets and IV methylprednisolone for 7 days. Her platelet did not respond to the treatment and she was subsequently started on 30 mg IVIG therapy for 5 days. Her platelet count increased to 86,000. Biopsy of lymph nodes could not be performed due to thrombocytopenia. She is being treated with long term oral prednisone for sarcoidosis.</p> <p>This case illustrates the multitude of presentations sarcoidosis that physicians should be aware of. Three potential mechanism of thrombocytopenia in sarcoidosis patients includes autoantibody platelet destruction, hypersplenism, or bone marrow infiltration. Previous case report associating thrombocytopenia and sarcoidosis found prednisone to be an effective therapy but this cases teaches us that IVIG should also be considered in the management.</p>

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Clinical Vignette	Justin Mottaghi	Ambreesh Chawla, MD, Sunil Sapru, MD	Saint Barnabas Medical Center (Sunil Sapru)	Cellulitis and Hyperthyroidism: The Perfect Storm	<p>Thyroid storm is a life-threatening medical condition characterized by a wide spectrum of clinical manifestations of thyrotoxicosis such as fever, restlessness, tachycardia, and altered mentation, among other signs and symptoms. Well-known differentials which may mimic a thyroid storm include sepsis, pheochromocytoma, drug toxicity, and anxiety disorders. We describe an unusual case of cellulitis precipitating a thyroid storm in a patient with no known history of thyroid disease.</p> <p>Our patient is a 63-year-old female with a past medical history of DVT (on anticoagulation), mitral valve prolapse, and diverticulosis, with no known history of thyroid disease. She presented to the ED with a four-day history of generalized fatigue and fever accompanied by erythema, pain and edema in her left lower extremity. On presentation, our patient was febrile to Tmax of 100.9F with a blood pressure of 114/55 mmHg and a heart rate of 110 BPM. EKG initially showed sinus tachycardia. Laboratory studies were significant for WBC count of 14,700 g/dL with 77% neutrophils. Initially thought to be a simple case of cellulitis, our patient was started on cefazolin and clindamycin with IV fluid resuscitation. However, on hospitalization day three, her clinical condition unexpectedly deteriorated, with a new onset of acute delirium, diaphoresis, temperature of 102.7F, and tachycardia to 210 BPM. A repeat EKG showed atrial fibrillation with rapid ventricular response. These findings prompted the evaluation of thyroid function tests which revealed a TSH of less than 0.005 UIU/mL, fT4 of 4.90 ng/dL, and fT3 of 283.9 ng/dL. Upon clinical suspicion of thyroid storm, propranolol was initiated with propylthiouracil, Lugol's Iodine solution (potassium iodide) and dexamethasone. With continued therapy, our patient's thyroid function studies normalized and she converted back to normal sinus rhythm. Her mental status improved markedly as she was subsequently transferred to the general medical ward.</p> <p>Thyroid storm is a rare, life-threatening complication of thyrotoxicosis, typically triggered by an acute stressor, such as surgery (thyroidal or non-thyroidal), trauma or other acute medical illness. In the setting of an acute infection, the increase in metabolic demand may predispose and exacerbate an existing overproduction of thyroxine (T4) and triiodothyronine (T3). As a result of increased adrenergic tone and vasomotor instability, thyroid storm has the potential to cause multi-organ system failure, which carries an exceptionally high mortality rate. Thus, early and aggressive intervention is vital. This includes supportive care, correction of the underlying infection and reversal of the hyperthyroid state (via blocking the synthesis, release and peripheral conversion of T4 to T3). Our case demonstrates that thyroid storm can arise even in the setting of a seemingly mild superficial skin infection. It is thus imperative clinicians be aware of and prepared to manage this potentially life threatening condition.</p>

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Clinical Research	Ahmed Dirweesh	Sara Wallach, Michael J. Smith, Ritika Zijo, Muhammad Khan, Mohamed Ijaz, Afolarin Amodu, Mohamed Ibrahim, Ambreen Bushra	Saint Francis Medical Center (Sara Wallach)	The Effect of Severely Reduced Kidney Function on Symptomatic Diverticular Disease	<p>Introduction: The prevalence of diverticulosis is increasing with 5-10 percent of patients developing diverticulitis and 5-15 percent developing symptomatic bleed. Diverticulitis can result in abscess, perforation, fistula, or obstruction. Bleeding has combined morbidity and mortality rates of 10-20 percent. The purpose of this study was to compare diverticulitis related complications and transfusion requirements for diverticular bleeding in patients with normal to moderately reduced kidney function (GFR \geq30 mL/min/1.73 m²) and patients with severe renal impairment (GFR <30 mL/min/1.73 m²), and identify factors associated with these outcomes.</p> <p>Methods: We retrospectively reviewed records of all patients with diverticulitis or diverticular bleed treated at our hospital from January 1, 2011 to July 31, 2016. Patients were evaluated to determine baseline GFR, age, gender, race, medications, comorbidities, length of stay (LOS), presence of perforations or abscesses and the need for transfusion.</p> <p>Results: Of the 291 patients included, males were 167(58%). Mean age and LOS for patients with non-complicated diverticulitis was 59\pm1.5 years and 2.9\pm1.9 days in the GFR \geq30 group, and 65\pm4.5 years, 5.8\pm2.7 days in the GFR <30 group. Perforations or abscesses complicating diverticulitis developed in 31/136(23%) of patients with GFR \geq30, and in 13/26(50%) of patients with GFR <30 (odds ratio, 3.4; 95% confidence interval, 1.423-8.06; p-value=0.0073). Mean age and LOS for patients with perforations/abscess was 55\pm3 years, 8.3\pm4 days in the GFR \geq30 group and 65\pm4 years, 8.5\pm4.4 days in GFR <30 group (p-values=0.24, 0.0001). Perforations or abscess occurred in 5/28 Blacks, 3/12 Hispanics and 23/94 Whites with GFR \geq30 (p-value=0.76) and in 3/6 Blacks, 2/3 Hispanics and 8/17 Whites with GFR <30 (p-value=0.72). Blood transfusion for diverticular bleed was required in 11/78(14%) of patients with GFR \geq30 and in 22/51(43%) of patients with GFR <30 (odds ratio, 4.6; 95% confidence interval, 1.99-10.76, p-value=0.0004). Mean age and LOS for patients who were not transfused was 71\pm2 years, 3.1\pm1.5 days in GFR \geq30 group, and 76\pm2.3 years, 4.5\pm3.6 days in GFR <30 group. Among patients who needed transfusion, mean age and LOS was 71\pm3.4 years, 8.5\pm2.5 days in GFR \geq30 group and 71\pm2.4 years, 9\pm5 days in those with GFR <30 (p-values=0.2, 0.04). Transfusion needed in 3/23 Blacks, 2/8 Hispanics and 6/47 Whites with GFR \geq30 (p-value=0.79) and in 6/14 Blacks, and 13/28 Whites with GFR <30 (p-value=0.09).</p> <p>Conclusion: There was significant increase in complicated diverticulitis cases; transfusion rates for diverticular bleeding and LOS in patients with severely reduced kidney function compared to patients with normal-moderately reduced renal function. The study did not detect any differences in age, gender or race between the study groups.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ahmed Dirweesh	Afolarin Amodu, Sumera Bukhari	Saint Francis Medical Center (Sara Wallach)	A False Positive HIV Antibody Test in a Patient with Mediastinal Hodgkin Lymphoma	<p>Introduction: Fourth generation antigen-antibody assays and HIV antibody differentiation assays have high reported sensitivity and specificity. However, up to 0.5% of patients found to be seropositive for HIV are false positives. We report a case of Hodgkin lymphoma with a false positive HIV testing.</p> <p>Case Presentation: A 38-year-old Hispanic male presented with 5 months history of cough with occasional bloody sputum. He also reported intermittent right sided chest pain, fever and night sweating for few weeks, and he has lost 15 lb. unintentionally in the last 3 months. He denied shortness of breath, contact with sick patients, travel or incarceration history. He works as a car-washer, and on monogamous relationship. He denied smoking, alcohol or drug use. On examination, vitals were normal, BMI was 19.1, and had no palpable lymphadenopathy. Dull percussion noted on right middle lung zones. The rest of exam was normal. He had WBCs of 7800/mm³, Hemoglobin of 11.3 g/dl, and normal platelets count. Chest radiograph showed a right mediastinal, hilar and infra-hilar masses, and computed tomography (CT) revealed the mass encasing the bronchi and major vessels, with mediastinal extension. Mass biopsy showed Hodgkin lymphoma (mixed cellularity type) with positive CD30 and CD15. Abdominal CT showed mesenteric and retroperitoneal lymphadenopathy with splenic lesions. A patient-confirmed HIV-1 and 2 Antibody with P24 antigen was first reactive, with an undetectable viral load. A repeat antigen-antibody test came back negative!</p> <p>Discussion: Hodgkin Lymphoma has aggressive presentation with systemic B symptoms, widespread extra-nodal lesions and frequent bone-marrow involvement. Reed–Sternberg cells typically express CD15 and CD30, rarely express CD20, and lack CD45 expression. HIV testing is part of routine testing because of common anticipated association. As false positive HIV screening have been rarely linked to pregnancy, autoimmune disorders, some viral infections and hematologic malignancies, a confirmatory test should be done. There are several speculations about the mechanism of cross-reactivity that is believed to be responsible for the false HIV positivity. Antigenic mimicry between the individual’s own epitopes and retroviral antigens is the likely mechanism for the reactivity with HIV P24 antigen. Muta et al reported false-positive HIV serology in angioimmunoblastic T-cell lymphoma patients and speculated that this may be due to coincidental cross-reaction of subtypes of polyclonal gamma globulin with the HIV P24 antigen. The exact mechanism for false positive antibody reaction in this case may also be related to the HIV P24 antigen cross-reactivity as speculated in other reported cases.</p> <p>Conclusion: Although false-positive HIV testing appears to be rarely described in lymphoma patients, it is vital to appreciate that the cross-reaction with HIV can be a potential complication.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Muhammad Khan	Ahmed Dirweesh, Herbert Conaway, Robert Moser	Saint Francis Medical Center (Sara Wallach)	Anal Neuroendocrine Tumor: An Aggressive Malignancy Masquerading As External Hemorrhoids	<p>Introduction: Neuroendocrine tumors (NETs) arise from neuroendocrine cells throughout the body. The reported incidence of NETs varies from 0.1% and 3.9% of all colorectal malignancies. The rectum is most commonly involved followed by the cecum and sigmoid colon. Anal NET is a devastating growth easily confused with benign hemorrhoids. Physician unfamiliarity with this rare rectal lesion can result in delays in diagnosis and therapy.</p> <p>Case Presentation: A 60-year-old black male presented with a two-week history of intermittent rectal bleeding and anal pain along with tenesmus and constipation. He denied abdominal pain, vomiting, anal or perianal discharge or sinuses, fever, chills, anorexia or weight loss. He had a past medical history of hypertension, coronary artery disease status-post coronary artery bypass surgery, and peripheral vascular disease. Examination was significant for a small perianal lesion located that mimicked a thrombosed external hemorrhoid. It was slightly painful, and partially covered with anal skin. The rest of the anorectal examination was normal and inguinal lymph nodes were not palpable. There were no significant laboratory abnormalities. Excisional biopsy of the anal lesion revealed an invasive poorly differentiated high-grade neuroendocrine carcinoma of a large cell type. Immunohistochemical studies were positive for cytokeratin, chromogranin, and synaptophysin. There was a high Ki-67 proliferation index of >90%. A contrast-enhanced CT scan of the chest, abdomen and pelvis showed multiple large hepatic mass lesions, consistent with hepatic metastatic disease. The patient started a chemotherapy-based regimen and is currently getting his scheduled cycles with obvious clinical improvement.</p> <p>Discussion: Anal involvement by this heterogeneous group of tumors is very rare; involving 1% of cases. NETs show immunoreactivity to Chromogranin and Synaptophysin; which are diagnostic for NET. Neuron-specific enolase (NSE) and CD56 are often positive in NETs, but are not specific. According to WHO classification, NETs are classified into typical carcinoids (low-grade NET), atypical carcinoids (intermediate-grade NET) and small cell and large cell carcinomas (high-grade NET). Immunohistochemistry for Ki-67 (MIB-1) is mandatory to grade the tumor. High Ki-67 proliferative index points towards aggressiveness and probably the potential of metastatic dissemination. NETs carry a poor prognosis with one-third of patients having distant metastasis at the time of presentation most commonly involving lymph nodes, liver and bones.² Large tumor size, lymphovascular invasion, and high mitotic rate are poor prognostic markers.</p> <p>Although the standard therapy for patients with neuroendocrine carcinoma of the anal canal is still unclear, treatment approach should be directed by age, stage, nodal involvement, and tumor histopathology. Surgery with or without adjuvant treatment is the preferred therapy for localized disease, while chemotherapy regimens combining etoposide plus cisplatin or carboplatin are currently considered useful for poorly differentiated tumors and have shown reasonable results.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Rabia soomro	A. Dirweesh MD, I. Acharya MD, M. Ibrahim MD, S. Bukhari MD, S.Wallach MD	Saint Francis Medical Center (Sara Wallach)	Challenging Cases In Lung Cancer : Expect The Unusual	<p>Introduction: Lung cancer is the leading cause of cancer related deaths in the U.S (27 % of all deaths). The awareness and prompt recognition of atypical presentations is critical to the management of the underlying malignancy. We report a series of 3 cases, each with an uncommon initial presentation masking underlying lung cancer.</p> <p>Case Descriptions:</p> <p>Case 1: Thrombotic Thrombocytopenic Purpura A 56 year old African American male with no past medical history, presented with altered mental status. He was found to have anemia, thrombocytopenia, elevated creatinine, and schistocytes on peripheral smear. Imaging showed a 3cm left upper lobe lung mass. Biopsy confirmed adenocarcinoma. Plasmapheresis was started with remissions and replases. Eventually he underwent lobectomy and achieved remission.</p> <p>Case 2: Atrial Fibrillation A 59 year old male with history of COPD, presented with new onset palpitations and unintentional weight loss of 20 lbs in 3 months. EKG demonstrated atrial fibrillation with rapid ventricular rate. Echocardiogram demonstrated pericardial effusion. Imaging of the chest revealed a left hilar and posterior mediastinal mass encasing pulmonary veins with extrinsic mass effect on the left atrium. CT guided FNAC revealed squamous cell carcinoma.</p> <p>Case 3: Lambert Eaton-Myasthenia Gravis (LEMG) A 78 year old female with history of hypertension presented with progressive weakness. Physical exam revealed proximal lower extremity weakness and incoordination. TSH was normal. Imaging of chest showed a large right lobe mass found to be a small cell tumor on biopsy.</p> <p>Discussion: The incidence of Lung cancer is increasing and due to the greater number of cases, atypical presentations appear. Several cases of lung cancer with atypical presentations have been reported and this poses a challenge to the diagnosis of lung cancer.</p> <p>Cancer related thrombocytopenia can be a result of either bone marrow metastasis or a paraneoplastic process. The presence of a primary malignancy is an essential concern in the differential diagnosis of TTP. Prompt recognition of atypical presentation is important as treating the underlying malignancy may be the best therapeutic decision in paraneoplastic TTP given its poor response to standard treatment (as in our patient).</p> <p>Few cases have documented the occurrence of atrial fibrillation as the first manifestation of lung cancer.</p>

					<p>We propose that this presentation can be explained by re-entry mechanism as a result of tumor cells invading the myocardium and leading to conduction abnormalities, from pericardial effusions or a result of mass effect of the growing tumor on the heart.</p> <p>LEMS can be an initial presentation of lung cancer. Few cases of lung cancer associated acute Guillain-Barre like syndrome and also cases of LEMS have been reported. Our case highlights the necessity of a complex differential diagnosis of severe progressive neuropathies.</p>
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Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Muhammad Khan	Ahmed Dirweesh, Shaikh Hamiz, Nigahus Karabulut	Saint Francis Medical Center (Sara Wallach)	Pulmonary Kaposi's sarcoma with Bony Metastases in an HIV Patient:A Remarkable Response to Highly Active Antiretroviral Therapy	<p>Introduction: Kaposi sarcoma (KS) is known to involve the mucocutaneous tissues and the aero-digestive tracts. In AIDS patients, KS has an aggressive course and carries poor prognosis. We present a case of pulmonary KS involving mediastinum with osseous metastases as the first presentation of Human Immunodeficiency Virus (HIV) infection in a young male. The lesions impressively decreased in size and numbers following initiation of highly active antiretroviral therapy (HAART).</p> <p>Case Presentation: A 34 year-old male presented with a two month history of cough productive of whitish sputum. He had 15-20 pounds weight loss in the last 6 months but denied fever, chills, night sweats, hemoptysis, travel or sick contacts. Patient had unprotected sex with multiple partners in past. Examination showed oral thrush, decreased breath sounds and crackles on the right lower lung base. The rest of examination was unremarkable. Chest radiograph showed extensive bilateral peri-hilar opacities more on the right side. Computed tomography (CT) Scan Chest showed right sided large peri-hilar mass mass with multiple lytic lesions involving thoracic and lumber vertebrae, ribs, sternum and clavicles. Blood and sputum cultures were negative, Tuberculosis was ruled out with negative acid fast bacilli smears and a negative Quantiferon gold test. He tested positive for HIV and CD4 count was 7cells/ul. Bronchoscopy with biopsy was unrevealing. Video assisted mediastinoscopy with biopsies of right hilar mass showed spindle cells positive for CD34, BCL2, Vimentin and HHV-8 with diffuse positivity for CD 31 diagnostic of KS. Following initiation of anti-retroviral therapy his condition improved with resolution of symptoms, weight gain and repeat chest CT scan showed marked regression of the disease.</p> <p>Discussion: KS was first described as multifocal angioproliferative lesions that eventually develop into a true sarcoma. Hong and Lee in 2002 found that extra cutaneous or visceral Kaposi's sarcoma lesions were more common in HIV infected patients. Visceral involvement without skin manifestations is rare. Few cases have been reported with sites involving gastrointestinal tract, lungs, liver and spleen as isolated visceral manifestations of KS. The advent of HAART has led to a substantial reduction in morbidity and mortality in KS associated with AIDS.</p> <p>Generally, more widespread disease, or disease affecting internal organs, is treated with systemic therapy directed towards KS (including interferon alpha, liposomal anthracyclines or paclitaxel). AIDS-related KS mainly involves the axial skeleton and/or maxillofacial bones, osseous lesions are commonly osteolytic. CT scan and MRI are superior to plain radiographs for the detection of bony lesions. This case highlights the dual action of HAART regimen on the immune system as well as against human herpes virus 8 (HHV-8) in regressing KS lesions signifying that the recovery of the immune system is the cornerstone in the management of this condition.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ritika Zijoo	Whitney Reid Fink, Abdelhameed E. Nawwar MBChB, Sara Wallach MD	Saint Francis Medical Center (Sara Wallach)	ACUTE PANCREATITIS ASSOCIATED WITH BILIARY STRICTURES	Acute pancreatitis is an inflammatory condition characterized by abdominal pain and elevated pancreatic enzymes. Common etiologies include gallstones and alcohol abuse. Less common causes include biliary strictures. If etiology is uncertain imaging tests may be performed, including endoscopic ultrasound, magnetic retrograde cholangiopancreatography (MRCP) or endoscopic retrograde cholangiopancreatography. Treatment of pancreatitis is supportive however strictures require further intervention. Interventions for treatment of strictures range from stents to surgery. Malignancy must be ruled out, whenever a stricture is detected. Our patient is a 56 year old male with past medical history of recurrent acute pancreatitis admitted to the hospital with epigastric pain, nausea and vomiting. He had an elevated lipase, a normal ultrasound of the abdomen. Lipid panel did not demonstrate hypertriglyceridemia and he denied ethanol abuse. Supportive care led to significant improvement. He was discharged two days after admission as his symptoms resolved, and he tolerated regular diet. He returned to the emergency room the next day with similar symptoms. On the second admission his bilirubin was newly elevated and had a newly developed transaminitis. The patient received a MRCP which demonstrated a mildly dilated common bile duct and nonspecific stricture of distal common bile duct. The patient underwent an Endoscopic Retrograde Cholangiopancreatography (ERCP) for further assessment. The ERCP confirmed the presence of a stricture; biopsy and brush cytology samples were collected. A stent was placed in the distal common bile duct and prophylactic stent was placed in the pancreatic duct. The patient will obtain repeat ERCP and cholecystectomy is under consideration.

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ritika Zijoo	Ahmed Dirweesh MD, Nigahus Karabulut MD	Saint Francis Medical Center (Sara Wallach)	RHODOCOCCUS EQUI PNEUMONIA AND PARADOXICAL IRIS IN A PATIENT WITH AIDS	<p>Introduction: Pulmonary infections are a major cause of mortality and morbidity in Human Immunodeficiency Virus (HIV) infected patients and can progress rapidly to respiratory failure and death without appropriate therapy.</p> <p>Rhodococcus equi (R.Equi) is a facultative, intracellular, gram positive, pleomorphic bacterium which is primarily infects immunocompromised, especially HIV infected individuals. Herein, we present a rare case of an advanced HIV infection and R.Equi pneumonia in a young male that had paradoxical immune reconstitution inflammatory syndrome (IRIS).</p> <p>Case Report: A 47 year old nonsmoker Hispanic man with advanced HIV infection presented with severe necrotizing pneumonia secondary to R.Equi. The patient was initially placed on intravenous Vancomycin, Ceftriaxone, Azithromycin and Clindamycin. After the results of cultures were obtained the antibiotics were changed to Linezolid, Levofloxacin and Azithromycin. The patient showed significant clinical improvement. He was discharged on Azithromycin and Levofloxacin for a total 8 weeks of therapy. His Highly Active Anti-Retroviral Therapy (HAART) regimen was changed to Ritonavir, Darunavir, Emtricitabine-Tenofovir at an outpatient HIV clinic. 3 weeks after switch to new HAART and while on week 4 of Levofloxacin and Azithromycin, patient started having right sided chest pain and cough with low grade temperature. A repeat CT scan of the chest demonstrated increased mass like consolidative opacity measuring 9.7 x 7.9 cm compared to consolidation measuring 7.8 x 6.5 cm in the initial CT scan. Linezolid was added and all three antibiotics were continued for 2 weeks, then Azithromycin and Levofloxacin were discontinued. Linezolid was continued for a total of 4 weeks to complete a total course of 8 weeks therapy. The CT scan findings were concerning, however given the patients improved virological and immunological status and negative work up for other potential differential diagnoses patient was treated symptomatically with biweekly follow ups. His symptoms continued to improve with HIV RNA becoming less than 20 copies and CD4 count of 236 cells /ml.</p> <p>Conclusions: Our findings demonstrate that our patient was experiencing paradoxical IRIS and that the ultimate clearance of R.Equi infection requires robust response from cellular immune system. In our case, the reappearance of clinical symptoms and enlarging infiltrates in CT of the chest while his immunological and virological recovery on the 3rd week of HAART was managed by careful monitoring. In spite of its rarity as an opportunistic pathogen, we recommend R.Equi, an intracellular pathogen, be included in the list of pathogens of IRIS.</p>

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Clinical Vignette	Rishitha Yelisetti	Dr. Areig Ali Awad, Dr. Anand Kaji MD	Saint Francis Medical Center (Sara Wallach)	UNUSUAL CONSEQUENCE OF POLYPHARMACY IN THE ELDERLY	<p>INTRODUCTION: Polypharmacy in elderly not only lead to drug interactions, but can also result in bowel obstruction due to large pills that may remain undissolved. While this is a rare presentation, physicians must be mindful of the number and size of pills patients are consuming daily to avoid this rare complication.</p> <p>CASE PRESENTATION: Patient is an 81-year-old Caucasian male with a past medical history of COPD, coronary artery disease, benign prostatic hyperplasia, atrial fibrillation, hypertension, and GERD. Past surgical history of open cholecystectomy. He presented to the emergency department with complaints of generalized abdominal pain, distension, one episode of non-bloody vomitus, and obstipation for 2 days. He also complained of unquantified weight loss within the last 2 months. The patient had normal colonoscopy 8 Years Ago. Home Medications Included Fluticasone-salmeterol Diskus, Amiodarone, Warfarin, Pantoprazole, Escitalopram, Ferrous Sulfate, Potassium Chloride, Guaifenesin, Montelukast, Multi-vitamin, and Vitamin C Capsule Daily.</p> <p>Physical examination revealed distended belly, hyperactive bowel sounds in all four quadrants. X-ray showed massive distention of colon, favoring marked functional abnormality of the colon, however distal obstruction could not be excluded. CAT scan demonstrated transition zone at rectosigmoid junction with distention of proximal bowel. Colon cancer was high on the differential. Colonoscopy revealed partially dissolved pills clumped together causing the obstruction with questionable component of sigmoid volvulus. The colonic mucosa was intact and no other colonic pathology was observed. The obstruction resolved after pills were removed. The patient tolerated diet and was discharged with appropriate changes made to his medications.</p> <p>DISCUSSION: Common causes of large bowel obstruction in elderly are colon cancer, volvulus, diverticulitis, and uncommon causes like hernia, intussusception, and foreign bodies. Presented is an unusual case of large bowel obstruction. We postulate that the pills in this patient remained partly undissolved because he was also taking a Proton Pump Inhibitor which interfered with the acid secretion in the stomach. Physicians should be conscientious about this rare occurrence especially when patients are taking multiple medications along with proton pump inhibitors</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Rishitha Yeliseti	Dr. Areig Ali Awad, Dr. Anand Kaji, MD	Saint Francis Medical Center (Sara Wallach)	ORGNAIZING PNEUMONIA SECONDARY TO HIATAL HERNIA	<p>INTRODUCTION: Organizing pneumonia secondary to a hiatal hernia is a specific type of inflammatory and fibroproliferative lung reaction due to micro-inhalation of the digestive contents. The current testing for gastroesophageal reflux poorly detects these microaspirations and therefore, may go unrecognized. Awareness of this complication may improve diagnosis, prompt treatment, and further complications.</p> <p>CASE PRESENTATION: 55-year-old Caucasian female with a past medical history of gastroesophageal reflux disease, mild intermittent asthma, moderately large hiatal hernia, and primary hypothyroidism presented to the emergency department with increasing dyspnea and nonproductive cough within the last six month period. She denied fevers, chills, weight loss, chest pain, seizures, skin rash, travel history, animal contact, occupational exposure, or joint pain. She had multiple admissions in the past in which she received extensive work up regarding her dyspnea. Past surgical history and family history were non-contributory. Social history included thirty-pack year smoking history which she quit a year ago and also, recreational drug use. Physical examination is notable for an obese female with dry crackles, Velcro rales at bases, and bilateral expiratory wheeze with decreased diaphragmatic excursion secondary to obesity. The working diagnoses during her admissions were either bronchitis or pneumonia and she was treated with antibiotics resulting in minimal improvement of her symptoms. Review of her computerized axial tomography (CAT scan) from 2013 showed mild patchy bilateral ground-glass infiltrates more prominent on the right and borderline mediastinal adenopathy. Repeat CAT scan showed the progression of interstitial lung disease with mild increase in mediastinal lymphadenopathy. These imaging studies also demonstrated a hiatal hernia. Angiotensin-converting enzyme level was normal. HIV, tuberculosis, streptococcal, and legionella pneumonia were negative. The workup was negative for pulmonary embolism. A video-assisted thoracoscopic surgery and biopsy were conducted that revealed patchy organizing fibrosis with acute on chronic inflammation, along with microabscesses, foreign body granuloma, and foreign material (vegetable matter) with acute bronchiolitis. Acid fast bacilli and Grocott's methenamine silver stain were negative. Findings were compatible with micro-aspirations likely secondary to a hiatal hernia. The patient was referred for surgical repair of the hiatal hernia.</p> <p>DISCUSSION: Chronic aspiration of small volumes of oral and gastric contents results in lung disease. This process is unfamiliar. Chronic occult aspiration causes foreign body giant cells and peribronchiolar granulomas, exogenous lipid pneumonia, organizing pneumonia, or fibrotic interstitial lung disease. Radiologically, the most typical finding is bronchiolitis, with centrilobular nodules, bronchial wall thickening, and tree-in-bud opacities. Progression to frank bronchiectasis and fibrosis occurs in severe and more chronic cases. Fibrosis is likely due to the aspiration and recurrent inflammation.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Areig Awad	Dr. Rishtiha Yelisetti, Dr. Donald Christmas MD	Saint Francis Medical Center (Sara Wallach)	MASSIVE PULMONARY EMBOLISM WITH RIGHT VENTRICULAR STRAIN AND NEGATIVE TROPONINS	<p>INTRODUCTION: Pulmonary embolism is one of the leading causes of cardiovascular mortality. In both submassive and massive pulmonary embolism, elevated pulmonary artery pressure can lead to progressive right ventricular dysfunction. Many clinical studies have demonstrated the presence of ischemia, right ventricular infarction, leading to elevation of cardiac enzymes. We describe a case of massive pulmonary embolism with negative cardiac enzyme.</p> <p>CASE PRESENTATION: A 59 year old Hispanic female with past medical history of asthma, obesity, and degenerative joint disease presented with shortness of breath and productive cough for two days. Associated with progressively worsening pressure-like chest pain, pedal edema, and paroxysmal nocturnal dyspnea for two months.</p> <p>On examination vital signs were temperature 98.2 Fahrenheit, heart rate 88 beats/min, respiratory rate 18 breaths/min, oxygen saturation of 95% on room air, and blood pressure 119/67 mmHg. Physical exam was unremarkable apart from chest wall tenderness. Laboratory workup was unremarkable except for creatinine 1.02 mg/dl (elevated), pro-BNP 2368 pg/ml (elevated), white cell count 12,200 cells/mcl (elevated). Electrocardiogram showed normal sinus rhythm with heart rate of 75 beats per minute, prolonged PR interval, left axis deviation, and S1Q3T3 pattern. Chest x-ray showed mild cardiomegaly. Patient was admitted for ongoing chest pain. On second day, patient had a cardiac arrest and was successfully resuscitated after two minutes of chest compressions. She remained hypotensive; intravenous fluids and vasopressor were instituted.</p> <p>CT angiogram of chest demonstrated severe extensive bilateral proximal segmental pulmonary emboli. Right sided heart enlargement with reflux of contrast into the hepatic veins and inferior vena cava, all compatible with right heart strain. 2 D-Echo revealed severe right ventricular (RV) dilation, RV global hypokinesis, ejection fraction of 55-60%, severe pulmonary hypertension, and right ventricular enlargement with pressure overload suggestive of acute pulmonary embolism. Right ventricular and pulmonary artery systolic pressure of 55 mmhg. Intravenous thrombolytic therapy with rt-TPA was administered. Six sets of troponins remained negative throughout events.</p> <p>DISCUSSION: Cardiac troponins have been shown to identify patients with high risk of mortality and an unfavorable prognosis during acute phase of pulmonary embolism. Cardiac troponin T (cTnT) is a sensitive and specific marker, allowing the detection of even minor myocardial cell injury. In patients with severe pulmonary embolism, myocardial ischemia may lead to progressive right ventricular dysfunction. Meta-analyses of cohort studies suggest that imaging of the right ventricle or biomarkers of myocardial injury alone may be insufficient for guiding therapeutic decisions. Our case justifies the importance of combining clinical judgment, imaging, and cardiac biomarkers when evaluating for a suspicion of right ventricular strain. Right ventricular strain should be suspected despite the absence of cardiac biomarkers.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ishan Acharya	M, Tahir MD; M, Ibrahim MD; K, Gala MD	Saint Francis Medical Center (Sara Wallach)	Rare case report of 3 foot long thrombus extending from the right femoral vein to the right ventricle	<p>Introduction: Transvenous temporary cardiac pacing is used to treat certain medically refractory arrhythmias. Transfemoral route is frequently utilized for its general safety, however severe thrombo-embolic complications are reported in approximately 0.6-3.5% of cases. We report a rare case of transvenous temporary pacing wire-associated clot extending from the right femoral vein to the right ventricle.</p> <p>Case Description: A 73 year old Caucasian male with past medical history of smoking and heavy alcohol use in the past presented with one day history of chest pain. He was found to have an inferior wall ST elevation myocardial infarction and was taken to cardiac catheterization lab urgently. However, the intervention was unsuccessful due to difficult coronary anatomy. During the same time, he developed high-degree atrioventricular block for which he required temporary transvenous pacing. Seventy two hours later, pacing wires were removed as his heart block had resolved. Eight hours after removal of the pacing wires, the patient decompensated and developed sudden hemodynamic instability. An urgent echocardiogram revealed right ventricular failure with a thrombus in the right atrium and the right ventricle. The patient was heparinized and urgently taken to the operating room by the cardiothoracic surgeon for atriotomy and clot excision. An approximately 3 foot long clot was excised from the right atrium which appeared to be originating from the right femoral vein. Subsequently, the patient developed cardiogenic shock due to right ventricular failure and was managed with fluids and multiple pressors. After aggressive measures, his shock resolved and the patient was stabilized. A few weeks later, he was successfully discharged from the hospital on lifelong anticoagulation. Coagulation work up for factor V Leiden deficiency, prothrombin gene mutation, lupus anticoagulant, antithrombin III, protein C and S was negative.</p> <p>Discussion: In this case, we suspect that the thrombosis of the pacing wires occurred despite of prophylaxis against deep vein thrombosis. Interestingly, patient was found to be negative for any underlying coagulopathy although protein C and S deficiency could be a possibility as patient was on anticoagulation when the tests were performed. This case brings to attention that despite the appropriate use of the transvenous pacer in this particular clinical scenario, we must be aware of these rare complications that can occur with the device and they should be timely removed once the need subsides. In our literature search, this is the first case report to describe a clot of this length with full recovery of the patient after urgent removal.</p>

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Clinical Vignette	Ishan Acharya	M, Tahir MD; M, Ibrahim MD; K, Gala MD	Saint Francis Medical Center (Sara Wallach)	Rare case report of 3 foot long thrombus extending from the right femoral vein to the right ventricle	<p>Introduction: Transvenous temporary cardiac pacing is used to treat certain medically refractory arrhythmias. Transfemoral route is frequently utilized for its general safety, however severe thrombo-embolic complications are reported in approximately 0.6-3.5% of cases. We report a rare case of transvenous temporary pacing wire-associated clot extending from the right femoral vein to the right ventricle.</p> <p>Case Description: A 73 year old Caucasian male with past medical history of smoking and heavy alcohol use in the past presented with one day history of chest pain. He was found to have an inferior wall ST elevation myocardial infarction and was taken to cardiac catheterization lab urgently. However, the intervention was unsuccessful due to difficult coronary anatomy. During the same time, he developed high-degree atrioventricular block for which he required temporary transvenous pacing. Seventy two hours later, pacing wires were removed as his heart block had resolved. Eight hours after removal of the pacing wires, the patient decompensated and developed sudden hemodynamic instability. An urgent echocardiogram revealed right ventricular failure with a thrombus in the right atrium and the right ventricle. The patient was heparinized and urgently taken to the operating room by the cardiothoracic surgeon for atriotomy and clot excision. An approximately 3 foot long clot was excised from the right atrium which appeared to be originating from the right femoral vein. Subsequently, the patient developed cardiogenic shock due to right ventricular failure and was managed with fluids and multiple pressors. After aggressive measures, his shock resolved and the patient was stabilized. A few weeks later, he was successfully discharged from the hospital on lifelong anticoagulation. Coagulation work up for factor V Leiden deficiency, prothrombin gene mutation, lupus anticoagulant, antithrombin III, protein C and S was negative.</p> <p>Discussion: In this case, we suspect that the thrombosis of the pacing wires occurred despite of prophylaxis against deep vein thrombosis. Interestingly, patient was found to be negative for any underlying coagulopathy although protein C and S deficiency could be a possibility as patient was on anticoagulation when the tests were performed. This case brings to attention that despite the appropriate use of the transvenous pacer in this particular clinical scenario, we must be aware of these rare complications that can occur with the device and they should be timely removed once the need subsides. In our literature search, this is the first case report to describe a clot of this length with full recovery of the patient after urgent removal.</p>

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Clinical Vignette	Bushra Ambreen	Muhammad Yasir Khan , MD. Nigahus Karabulut, MD	Saint Francis Medical Center (Sara Wallach)	A RARE CASE OF PRIMARY HODGKINS LYMPHOMA	<p>INTRODUCTION: Hodgkin Lymphoma (HL) is a B-Cell neoplasm characterized by Reed-Sternberg (RS) cells or one of the variants, typically present as lymph node enlargement in the neck and mediastinum, and B symptoms (fever, drenching sweats and weight loss). Primary extra nodal lymphomas of the gastrointestinal (GI) tract are extremely rare.</p> <p>CASE PRESENTATION: A 31 year old Hispanic male with past medical history of newly diagnosed HIV presented with rectal bleeding for 3 months. Examination under anesthesia and colonoscopy revealed rectal mass in anterior and lateral margin starting from anal verge with active bleeding. Biopsy of mass was compatible with Cytomegalovirus (CMV) colitis. Patient was started on HAART therapy and treated with ganciclovir for CMV infection. Four months after first encounter patient again presented with bright red bleeding per rectum, fecal incontinence, malaise and 10 lbs. weight loss in last few months but denied fevers, abdominal pain, night sweats, diarrhea or constipation. Abdominal examination did not reveal any masses or hepatosplenomegaly. Rectal examination was significant for ulcerated, tender, mass. Repeat biopsy of the rectal mass revealed lymphoid infiltrate, composed predominantly of small mature lymphocytes with scattered large atypical and mono and bi nucleated cells with abundant eosinophilia cytoplasm consistent with RS and Hodgkin cells. Immunohistochemical stains including LCA, CD30, CD15, PAX5, CD3, CD20 were consistent with HL. Latent membrane protein 1 immunostain was positive for Epstein Barr Virus (EBV). These findings were consistent with classical HL of rectum. Unfortunately patient did not comply with the treatment and went back to home country where he died.</p> <p>DISCUSSION: Primary HL involving the GI tract was first described by Schlagenhauer. Thomas et al in their extensive literature review found 26 cases of primary HD involving the colon with 5 cases of rectal involvement. Primary extra nodal HL most commonly involves the GI tract with stomach involvement in 67% of cases. Esophagus is the most infrequent site of involvement followed by colon. Clinically these tumors can manifest with crampy abdominal pain, weight loss, melena, change in bowel habits, obstruction and bleeding. HIV-related HL presents with advanced disease, stronger association with EBV, and poorer survival compared with HL in the absence of HIV infection. There is no uniform treatment for primary extra nodal GI HL. Case reports have shown that most of the primary extra nodal GI HL without any nodal involvement can be managed with surgery alone. Chemotherapy regimen involving ABVD has been used. This case highlights extremely rare manifestation of Hodgkins Lymphoma presenting as a rectal mass in an HIV patient.</p>

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Clinical Vignette	Shaikh Fawwad	Muhammad Khan, Dirwesh Ahmed, Karabulut Nigahus	Saint Francis Medical Center (Sara Wallach)	Superior mesenteric artery aneurysm in a patient with bacterial endocarditis	<p>Introduction: Mycotic aneurysm, is a result of septic embolization or bacteremia, from a distant source of infection. Prevalence of mycotic aneurysms is not clearly known as related symptoms are present in only 1-5% cases of mycotic aneurysm. Complete resolution with antibiotic therapy is rare and requires surgery in most cases. We present a case of 44-year-old man with bacterial endocarditis who developed mycotic aneurysm of the superior mesenteric artery (SMA).</p> <p>Case Presentation: A 44-year old Caucasian male presented with nonspecific chest pain along with subjective fever with chills. Review of system was otherwise negative. Medical history was significant for mechanical aortic valve replacement 8 years ago, infective endocarditis and hypertension. Patient admitted to intravenous heroin use in last six months. On examination, he was afebrile with normal vital signs. Cardiac auscultation revealed a systolic murmur throughout the precordium and a mechanical aortic click. Lungs were clear to auscultation with decreased air entry in the left lower lung. Rest of the examination was unremarkable.</p> <p>Considering his history of IV drug abuse and mechanical valve, he was started on intravenous vancomycin, rifampin and gentamycin for infective endocarditis and later switched to ceftriaxone and gentamycin as microbiology report grew gram positive cocci. Transthoracic echocardiogram showed possible vegetation around tricuspid valve which was confirmed with trans-esophageal echocardiogram. Patient developed upper abdominal with radiation to back during the hospital stay. CT scan of the abdomen and pelvis was negative for abscess, diskitis or kidney stones but showed a possible mycotic aneurysm of SMA which was not present in the previous CT scan. CTA Abdomen/pelvis confirmed saccular appearing mycotic aneurysm approximately 12x11x15 mm in the mid portion of SMA. Vascular surgery recommended medical management with antibiotics and follow up CTA abdomen in 3 months' time as aneurysm was too small for surgical intervention. Unfortunately patient was non-complaint and did not follow up.</p> <p>Discussion: Visceral artery aneurysms are rare and most cases are of degenerative in origin. Even rarer is the involvement of superior mesenteric artery SMA with an incidence ranging from 0.1 to 2%. Staphylococcus Aureus and Salmonella are the ones usually involved. Our patient grew Group A streptococcus, not usually associated with mycotic aneurysm. CTA is the investigation of the choice and should be used when there is a high degree of suspicion. Management of mycotic aneurysms requires a multidisciplinary approach. Broad spectrum antibiotics should be initiated as soon as possible. Surgical intervention is the mainstay of treatment with no difference in mortality and morbidity between open and endovascular approach.</p> <p>Our case reminds physicians to consider mycotic aneurysm among differentials in patients with IE who present with vague symptoms secondary to compressive effect despite the wide spread use of antibiotics.</p>

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Clinical Vignette	Sumera Bukhari	Seema Niphadkar, MD ; Kristine Ward, MD ; Maneesh Jain, MD ; Michael Styler, MD ; Sara Wallach, MD	Saint Francis Medical Center (Sara Wallach)	Hepatocellular Carcinoma: Nothing "Humerus" about it	<p>Introduction: Hepatocellular carcinoma (HCC) is the most common primary liver malignancy and the fifth most common cancer in men worldwide. Major risk factors include Hepatitis B, hepatitis C, alcoholic liver disease and nonalcoholic steatohepatitis. An extensive literature review revealed only a few case reports describing cases of solitary metastasis to the appendicular skeleton from an unknown primary HCC. We describe a case of HCC, in which a humeral fracture is an initial manifestation.</p> <p>Case Presentation: A 59 year old male with a history of chronic alcoholism presented to orthopedic surgery for further evaluation of a fractured right humerus discovered on X-ray in the emergency department after light gardening. During the exam, he reported dyspnea on exertion and increasing abdominal distention. These alarming symptoms prompted a referral to cardiology for pre-operative clearance. During the evaluation, he was noted to have elevated transaminases, a positive hepatitis C antibody, and IV/IV systolic murmur. He was admitted to the hospital for a complete workup. During the hospitalization, he was found to have hypoalbuminemia, transaminitis, anemia and moderate aortic stenosis. MRI of the shoulder revealed a displaced and angulated fracture of the proximal humerus diaphysis with cortical thinning and endosteal scalloping. No discrete tumor or soft tissue mass was seen but was likely obscured by hemorrhage from the fracture. Further imaging of abdomen/pelvis revealed liver nodularity concerning for cirrhosis and four lesions concerning for HCC and one lesion atypical for HCC in the liver. Staging scans did not reveal metastatic disease in the chest. He underwent a bone biopsy of the right humerus at the fracture site, which revealed the presence of HCC. He underwent open reduction and internal fixation of the right humerus after being medically optimized with plans to start radiation and sorafenib after recovery from surgery.</p> <p>Discussion: The majority of patients with HCC present with right upper quadrant abdominal pain or sequelae of the liver disease. Rarely, patients may have initial symptoms exclusively related to extrahepatic metastases. The most common sites of metastases include the lung, lymph nodes, and portal vein, whereas involvement of the bone is rare. The most common sites of bone metastasis are vertebra followed by the pelvis, rib, and skull. Metastatic involvement is rarely found in the extremities, especially the humerus. Most reported cases of bone metastasis are accompanied by either multiple metastatic spreads elsewhere in the body or previously known HCC. Katyal et al. reported a case series where isolated bone metastasis as the initial manifestation was only seen in 9.5% of patients with only two cases out of 148 patients having lytic lesions of the humerus. These unusual presentations raise the importance of a thorough pre-operative evaluation for pathological fractures in otherwise asymptomatic patients.</p>

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Clinical Vignette	Sumera Bukhari	Ahmed Dirweesh, MD ; Herbert Conaway, MD	Saint Francis Medical Center (Sara Wallach)	Angioedema: Don't Stress the Rare Tumor	<p>Introduction: Angioedema is self-limited, localized swelling of the skin or mucosal tissues, often involve the lips, eyes, face, respiratory and gastrointestinal mucosa. It can be classified as allergic, drug induced, hereditary or idiopathic by etiology. In few instances, Angioedema has been reported with well differentiated Neuroendocrine tumors (NETs) such as Carcinoids. We write a case of angioedema in a patient with poorly differentiated - large cell neuroendocrine tumor (LCNETs) of the rectum after undergoing surgical stress.</p> <p>Case Presentation: A 60-year-old man with LCNETs of the rectum on chemotherapy was transferred to our ICU after failed post-op extubation trial and fluctuating blood pressure. The patient underwent right carotid endarterectomy and was successfully extubated. In the recovery room, he had stridor with hypoxia and got re-intubated with some difficulty by anesthesia. The patient was started on steroids and epinephrine and was hemodynamically stabilized. ENT evaluation showed significant swelling of his tongue, lips and face with laryngeal swelling. He was successfully extubated the next day with significant improvement in his swelling. The patient underwent allergy screen to determine possible triggers like an anesthetic agent, ACEIs/ARBs, latex or silicon use. Hereditary angioedema was excluded by negative workup and ARBs were discontinued. However, no apparent cause was found through workup other than the probable correlation with the underlying neuroendocrine tumor, proposed by the explanation that NETs might have released a surge of hormones due to surgical stress leading to the manifestation of angioedema and episodes of fluctuating blood pressure.</p> <p>Discussion: LCNETs are a rare subtype of NETs with an aggressive nature and a poor prognosis due to its tendency for early metastasis. NETs can arise in different organs, but colonic NETs are exceptionally rare. A study by Bernick et al. showed that 0.6% of patients with colorectal cancer had NETs and only 0.2% of those were LCNETs. Colorectal LCNETs usually present with abdominal pain, hematochezia, constipation, or tenesmus. Symptoms associated with excessive hormone production such as paraneoplastic and carcinoid syndromes may rarely be a presenting feature. Saclarides et al. proposed that although neuroendocrine differentiation and hormone secretion occur at the cellular level, LCNETs may produce an insufficient amount of biologically active compounds. Thus none of the patients with LCNETs displayed clinical manifestations of the carcinoid syndrome in that study. To the best of our knowledge, there are only a few reported cases of angioedema related to carcinoid tumors but no published case with colorectal LCNETs with Liver metastasis presented as angioedema. This case emphasizes the importance of asymptomatic neuroendocrine tumors undergoing surgeries, should be observed post-operatively for a longer time or given prophylaxis as angioedema presenting with Laryngeal swelling can be life-threatening, requiring re-intubations or emergent/urgent tracheostomy.</p>

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Clinical Research	Nilay Patel	Vinaykumar Panchal, Kanishk Agnihotri, Abhishek Deshmukh, Apurva Badheka.	Saint Peter's University Hospital (Nayan Kothari)	Trends of complications of cardiac resynchronization therapy in the United States from 2003 to 2011	<p>BACKGROUND: The utilization of cardiac resynchronization therapy defibrillator (CRT-D) has increased significantly since its initial approval in 2001 for use in patients with advanced heart failure. Limited data exists regarding trends in complications associated with it.</p> <p>METHODS: Using the Nationwide Inpatient Sample, we identified estimated 378,248 CRT-D procedures from 2003 to 2011. We investigated common complications including mechanical, cardiovascular, pericardial complications (hemopericardium, cardiac tamponade), pneumothorax, stroke, vascular complications (consisting of hemorrhage/hematoma, incidents requiring surgical repair, and accidental arterial puncture), and in-hospital deaths described with CRT, defining them by the validated International Classification of Diseases, Ninth Revision, Clinical Modification diagnosis code. Hierarchical two level logistic regression models were built to evaluate outcomes of mortality/complications. Results: We identified estimated 3,78,247 CRT-D procedures from 2003 to 2011. Most common complications included mechanical (5.9%) and cardiovascular complications (3.61%). While frequency of complications rose from 15.88% in 2003 to 17.12% in 2012, the in-hospital mortality has decreased. Age >80 (OR, CI, p-value: 1.38, 1.25-1.53, <0.01), Female sex (1.07, 1.02-1.12, <0.01), increased burden of comorbidities (CCI>=2 1.12, 1.02-1.24, 0.003) were associated with higher mortality/complications.</p> <p>CONCLUSIONS: The overall complication rate in patients undergoing CRT-D has been increasing. Mechanical and cardiac complications were most common complications. Age(>50), female sex and Charlson score ≥2 were associated with higher complications.</p>

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Clinical Research	Naveen Iajpatrai Gupta	S Nayak, A Rastogi – SPUH Rutgers, IIT karagpur, ILBS	Saint Peter's University Hospital (Nayan Kothari)	Immunoblot development for diagnosis of autoimmune hepatitis	<p>Background: Type 1 Autoimmune hepatitis constitutes more than 90 % cases of autoimmune hepatitis, is characterized by ANA and/or SMA positivity detected by operator dependent indirect immunofluorescence with lymphoplasmacytic infiltrate in liver biopsy . No single AIH-1 specific nuclear antigen has been identified , with ANA directed against chromatin, single or double stranded DNA, tRNA, SSA-Ro, snRNPs, ribosomal P protein, cyclic A or histones. No single pattern has been established in indirect immunofluorescence at present, characteristic of AIH.</p> <p>The aim of the current study was to exploit the database in Indian setting to determine nuclear antigens as targets for antinuclear antibodies in patients of autoimmune hepatitis type 1 to develop autoimmune hepatitis specific immunoblots. The study was done at Institute of Liver and Biliary Sciences, New Delhi, India, tertiary care center for liver disorders.</p> <p>Methodology: 75 patients, >18 years who were antinuclear antibody positive with indirect immunofluorescence titre $\geq 1:80$, ALT >40 U/L, serum IgG (>16g/L), nonreactive viral hepatitis markers and within normal metabolic parameters confirmed with liver biopsy were evaluated with various types of immunoblots used for diagnosis of rheumatologic diseases and elisa coated with nRNP/Sm, Sm, SS-A (SSA native and Ro-52), SS-B, Scl-70, PM-Scl, Jo-1, CENP B, PCNA, dsDNA, Nucleosome, Histones, ribosomal P protein, AMA-M2, Sp100, LKM1, gp210, LC1 and SLA antigens.</p> <p>Results: Antibodies to Ro-52 were present in 35 of 75 ANA+ sera (17+ alone, 18 with SS-A). Antibodies to histones positive in 8 of 75 ANA + sera (4 histones + alone, 4 with antibodies to nRNP, SS-A, Ro-52, nucleosome). Antibodies to nucleosome were present in 8 of 75 sera (4 alone, 4 with antibodies to nRNP, SS-A, Ro-52, histones) Antibodies to n RNP were detected with other antibodies in 4 of 75 sera. Antibodies to centromere (Cenp-b) were found alone in 3 of 75 sera. Antibodies to PCNA were detected with anti SS-A in 4 of 75 sera. Antibodies to ds-DNA, Sm, Scl-70, PM-Scl, Jo-1 AMA-M2, ribosomal P-protein, Sp100, LKM-1, gp-120, LC1, and SLA were not detected in any of the 75 AIH patients' sera. 13 sera were non reactive to immunoblot.</p> <p>Conclusion: Immunoblots development with antigens Ro52, SSA, PCNA, nRNP, histones and nucleosome, can detect antibodies in 85% of patients ruling out detection by immunofluorescence microscopy which is highly subjective , operator dependent technique or other nonspecific immunoblots , and also with no established criteria of ANA titres particularly in autoimmune hepatitis patients.</p>

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Clinical Research	Aakash Garg	Amit Rout, Sahil Agrawal, Abhishek Sharma, John B. Kostis	Saint Peter's University Hospital (Nayan Kothari)	Routine Invasive versus Conservative strategy for Elderly patients aged > 75 years with Non-ST Elevation Acute Coronary Syndrome	<p>Background: Current guidelines recommend a routine invasive strategy for patients with non-ST elevation acute coronary syndrome (NSTEMI-ACS) [1]. However, elderly patients with NSTEMI-ACS are under-represented in randomized controlled trials (RCTs) and less likely receive invasive treatment [2]. Therefore, it remains unclear if elderly patients frequently admitted with NSTEMI-ACS benefit from an invasive strategy compared to a conservative strategy .</p> <p>Objective: We conducted a meta-analysis to compare routine invasive and conservative strategies in elderly patients with NSTEMI-ACS.</p> <p>Methods: A systematic review of RCTs in MEDLINE, EMBASE, CINAHL, and Cochrane databases comparing routine invasive with conservative strategy for elderly patients (age> 75 years) with NSTEMI-ACS was performed. The primary composite end-point was death or myocardial infarction (MI). Random effects meta-analysis was conducted to estimate odds ratio (OR) with 95% confidence intervals (CI) for individual end-points.</p> <p>Results: A total of 6 studies with 1,887 patients were included in the final analysis. Compared to a conservative strategy, routine invasive strategy was associated with significantly decreased risk of the primary composite of death or MI [OR 0.65; 95% CI 0.51-0.83]. There were no significant differences between conservative and routine invasive strategies in terms of all-cause death [OR 0.85; 95% CI 0.63-1.20] or cardiovascular death [OR 0.84; 95% CI 0.61-1.15]. A routine invasive strategy led to significant reduction in risk of MI [OR 0.51; 95% CI 0.40-0.66] and need for revascularization [OR 0.31; 95% CI 0.11-0.91] compared to conservative strategy.</p> <p>Conclusion: In elderly patients > 75 years with NSTEMI-ACS, routine invasive strategy is superior to conservative strategy for the composite end-point (death or MI), primarily driven by reduced risk of MI.</p>

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Clinical Research	Kavya Patel	Nilay Patel, Kartik Kalra, Vinay Panchal, Apurva Badheka	Saint Peter's University Hospital (Nayan Kothari)	Atrial Fibrillation In Patients With Non-STEMI – Insights From A National Database.	<p>Background: Atrial fibrillation is associated with an increased risk of developing coronary artery disease subsequently leading to major adverse cardiac events. However, there is paucity of data comparing outcomes in non STEMI patients with and without atrial fibrillation.</p> <p>Methods: We queried the Healthcare Cost and Utilization Project’s Nationwide Inpatient Sample data between 2004-2013 using ICD 9 cm code 410.7 for NSTEMI in the principal diagnosis field. The study cohort was then divided into two comparison groups based on presence of Atrial Fibrillation (427.31) in secondary diagnosis field. Hierarchical two level logistic regression models were built to evaluate outcomes of mortality and major adverse cardiac events (composite of mortality, re-infarction and stroke).</p> <p>Results: During the study period, 827729(weighted 4,082,908) patients were hospitalized with a principal diagnosis of NSTEMI (62.66% age ≥65 years, 42.64% females). Among these, 18.27% patients had a diagnosis of atrial fibrillation. Univariate analysis revealed decreased utilization of PCI in Atrial Fibrillation patients (17.25% vs. 33.05%, p<0.001). After adjusting for confounders, Atrial Fibrillation was a significant independent predictor of increased mortality (OR, CI, P value; 1.27, 1.24-1.31, <0.001) or major adverse cardiac events (1.23, 1.21-1.26, <0.001). Furthermore, in subgroup analysis , NSTEMI patients undergoing PCI were likely to have lesser reduction of odds of mortality if they also had Atrial Fibrillation.</p> <p>Conclusion: We report a nationwide data on outcomes in NSTEMI patient with concomitant Atrial Fibrillation, a combination that portends relatively poor prognosis even when patients undergo PCI.</p>

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Clinical Vignette	Paris Charilaou	Febin John MD, Balaji Yegneswaran MD	Saint Peter's University Hospital (Nayan Kothari)	A rare case of hemidiaphragmatic hernia and volvulus without respiratory compromise in a 94-year-old female	<p>Introduction: Diaphragmatic hernias have been well-documented in the literature especially in the neonatal and adolescent populations, however their incidence in the elderly is rare. In this case report we present a rather unusual incident of a large left hemidiaphragmatic hernia leading to an almost-total collapse of the left lung.</p> <p>Case Presentation: A 94-year-old female presented with chief complaint of acute onset lower abdominal pain associated with hematemesis. Vitals on admission were: BP 164/76 mmHg, HR 104/min, SaO2 100% at room air, RR 17/min, afebrile (98.0°F). Physical exam was only positive for epigastric tenderness and decreased left lung sounds without obvious respiratory distress. Laboratory studies showed leukocytosis with elevated lactic acid and acute kidney injury. Initial imaging revealed a large left diaphragmatic hernia into the left hemithorax with near-total collapse of the left lung (Figure). Imaging done a month prior had revealed mild eventration of the left hemidiaphragm (Figure), for which no intervention was done as she was asymptomatic. The patient underwent a successful exploratory laparotomy, resecting 6 inches of small bowel due to ischemic volvulus. The patient required only 1 day of mechanical ventilation and discharged home 5 days later with fully resolved symptoms.</p> <p>Discussion: Even though this patient had no previous iatrogenic thoraco-abdominal trauma, she had history of falls, which could act as blunt injury and predispose to this condition, with a possible delayed presentation. This can serve as basis for such an elderly patient to present as above, with an eventration acting as a probable precursor. Additionally, increased intra-abdominal pressure from presumed constipation, most likely attributed to her post-operative opiate use (hip fracture 1 month ago), can exacerbate the status of a weak diaphragmatic wall. The absence of respiratory symptoms in this case, complicated the diagnostic thinking in the early stages, especially before any clinical picture of acute abdomen developed to trigger the ordering of imaging studies. Given the absence of any previously documented trauma or thoraco-abdominal surgical history, the diagnosis was not initially suspected. Diaphragmatic hernia in this setting can be easily missed and detected late, even when there is clear history of trauma.</p> <p>Conclusions: It is conceivable that elderly patients with history of diaphragmatic eventration who are at risk of increased abdominal pressure and blunt trauma or falls, may present with diaphragmatic hernias associated with acute abdomen. One should therefore consider further evaluation with diagnostic imaging studies of the thorax/abdomen for a well-timed management.</p>

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Clinical Vignette	Navya Kuchipudi		Saint Peter's University Hospital (Nayan Kothari)	Isolated Vasculitic Neuropathy: A Rare Presentation of ANCA Vasculitis	<p>INTRODUCTION: Isolated vasculitic neuropathy is a rare presentation of ANCA associated vasculitis. It can present in three forms: Mononeuritis multiplex, Distal symmetric polyneuropathy, and Radiculopathy/Plexopathy. ANCA associated vasculitis is usually a part of a systemic disease such as GPA, EGPA, MPA, and PAN. They typically have skin and respiratory tract involvement or intestinal angina in the case of PAN. Isolated neuropathic involvement warrants a close watch for further symptoms as they may present as a full blown systemic syndrome at a later date.</p> <p>CASE: This is a 56 year old female from Mexico who presented with tingling, numbness and mild weakness of both legs of 3 weeks duration to her PCP. At week 5, she saw a neurologist as referred by her PCP who ordered blood work including A1c, TSH, RPR, B12, ANA which were all normal. RF was positive at that time, but she did not have any joint pains. At week 7, her lower extremity symptoms have worsened with bilateral foot drop causing difficulty in walking. At the same time, she experienced bilateral hand weakness. She saw her PCP who advised her to go to SPUH ER.</p> <p>At SPUH, she had bilateral upper and lower extremity distal strength of 3/5 with sensory loss and proximal strength of 5/5. She did not have any joint, respiratory, abdominal or ENT complaints. Repeat blood work was positive for P-ANCA and RF, while remaining were negative. Chest x-ray was normal. EMG showed severe symmetric distal sensorimotor polyneuropathy.</p> <p>She proceeded with a sural nerve biopsy which showed necrotizing arteritis of vasa vasorum. She subsequently was started on pulse dose steroids at 1000mg/day for 5 days and oral Cyclophosphamide. After one week of immunosuppressants, there was no improvement, but she will follow up with outpatient rheumatology.</p> <p>DISCUSSION: Although our patient's P-ANCA was positive, she did not meet any criteria for systemic ANCA associated vasculitis. She may develop into a clinical syndrome at a later time, and there by close monitoring of her symptoms is needed. Vasculitic neuropathy indicates a severe inflammatory response with high morbidity and mortality. High dose steroids with Cyclophosphamide or Rituximab(alternative) is considered first line with slow taper of prednisone over 24 weeks.</p> <p>CONCLUSION: Early diagnosis and treatment of vasculitic neuropathy is needed to prevent long term damage and allow slow regeneration of the nerve. Supportive care such as physical and occupation therapy, foot orthosis or wrist splints, and pain medications are crucial to decrease morbidity.</p> <p>ABBREVIATIONS: GPA: Granulomatosis with Polyangiitis; EGPA: Eosinophilic Granulomatosis with Polyangiitis; PAN: Polyarteritis Nodosa; MPA: Microscopic Polyangiitis</p>

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Clinical Vignette	Sonmoon Mohapatra	Dovid Moradi, Arkady Broder	Saint Peter's University Hospital (Nayan Kothari)	A failed IUD in the Sigmoid Colon? A Case Report	<p>Introduction: Intrauterine devices (IUD) use in women of all ages continues to rise due to its typically high reliability and safety profile.¹ IUD migration to the sigmoid colon after uterine perforation is a rare but serious event. We report a case of an IUD migration into the sigmoid colon in an asymptomatic 30 year old female who subsequently underwent laparotomy and sigmoid resection.</p> <p>Case: We describe a case of a 30 year old female who presented to her OB/GYN for missed periods. Two months prior, she had an uncomplicated Paraguard copper intra uterine device (IUD) insertion. On physical examination the IUD string was not visible at the cervical mouth and further testing confirmed a positive pregnancy. Transvaginal ultrasound showed a single intrauterine pregnancy but did not detect the IUD. As the patient was asymptomatic further workup was delayed until after delivery. After delivery of a healthy baby at 39 weeks, a KUB was performed and revealed the IUD to be intra-abdominal in the left upper pelvis projecting over the sacroiliac joint. Follow-up CT Abdomen and Pelvis showed the IUD anterior to the uterus within surrounding soft tissue. Six months post-partum, laparoscopy was performed and found the IUD strings abutting the colonic wall; however the device was still not entirely visible. The decision was made to refer the patient for colonoscopy prior to further surgical intervention. Upon Gastroenterology consult, the patient stated she felt well overall, but had intermittent abdominal pain at the site of laparoscopy with mild constipation. Colonoscopy revealed thickened mucosa in the sigmoid, and endoscopic ultrasound at that site confirmed metallic structure within the colonic wall invading through and into the submucosal plains. The patient was subsequently referred to general surgery for sigmoid resection.</p> <p>Discussion: Spontaneous migration of an IUD can occur rarely and it is unpredictable. When an IUD string is not visible during pelvic examination, appropriate workup, including radiographs such as ultrasound and CT scans should be obtained to confirm the position of the IUD.² Practitioners who place the IUD's must continue to have a high suspicion for migration in instances of unexpected pregnancy. Early involvement of consulting physicians is often necessary to diagnose and manage IUD related complications.</p>

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Clinical Vignette	Kavya Patel		Saint Peter's University Hospital (Nayan Kothari)	Pulmonary Histiocytosis X, A rare entity	<p>Introduction: Pulmonary Histiocytosis X is a rare ILD with unknown etiology but epidemiologically related to tobacco smoking and mainly affects young adults primarily in the 3rd and 4th decades of life. Common presenting symptoms are non productive cough, dyspnea, weight loss, chest pain, spontaneous pneumothorax, fever, cystic bone disease and diabetes insipidus. Diagnosis is based on history and imaging tests and sometimes on bronchoalveolar lavage and biopsy findings. The initial focus of any therapeutic regimen in patients with pulmonary Langerhans cell histiocytosis (PLCH) is cessation of smoking. Immunosuppressive therapies, such as glucocorticoids and cytotoxic agents, are of limited value as no studies comparing different treatments in adults with lung disease have been performed.</p> <p>Case description: Here, we report a case of a 38 years old male with 20 PPY smoking history who came to Pulmonary Clinic for worsening SOB on exertion, chronic dry cough, significant weight loss and polyuria. He has been diagnosed with COPD for 3 years. He had frequent exacerbation of COPD in last 3 years and every time he was treated with a course of antibiotics and steroids. He did not have abdominal pain, diarrhea, sinusitis, fever, rash, joint pain. On clinical examination, he looked very cachectic. His vitals were normal, except Respiratory rate which was 22. On examination of chest, he had emphysematous chest, bilateral wheezing was noted in the mid lung fields. Cardiovascular and abdomen examination was normal. Our differentials were tuberculosis, malignancy, connective tissue disorders, Alpha fetoprotein deficiency. His routine blood tests were normal except sodium was on higher side, HIV test was non-reactive. Other laboratory studies included ANCA, qauntiferron gold, immunoglobulin level, alpha-antiprotein and ESR which were normal. His Pulmonary function tests showed reduced FEV1 and reduced FEV1/FVC ratio which was consistent with obstructive pattern. He underwent CT thorax which showed severe emphysema, reticulonodular opacities and cysts. Later on, patient was scheduled for lung biopsy to establish a diagnosis. Histologic examination of lung tissue demonstrated PLCH. He was advised to abstain from both active and passive smoking. He was referred to Oncologist and to the expert for a trial of chemotherapy.</p> <p>Discussion and Conclusion: Pulmonary Langerhans' Cell Histiocytosis (PLCH) is an uncommon lung disease that occurs predominantly in adult cigarette smokers. It could have obstructive or restrictive pattern on PFTS and imaging. Lung biopsy plays important role in establishing diagnosis. Clinical course is variable and there is no definitive medical treatment for a progressive disease.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Brittany Fiorello	C. Pitchumoni, MD	Saint Peter's University Hospital (Nayan Kothari)	Thymoma Presenting as Intestinal Pseudo-Obstruction	<p>Background: Intestinal pseudo-obstruction causes symptoms of bowel obstruction in the absence of mechanical obstruction and rarely can present as a paraneoplastic phenomenon. Tumors that can cause intestinal pseudo-obstruction include breast cancer, small-cell lung cancer, gynecologic tumors, and hematologic malignancies.</p> <p>Case: A 53-year-old man with a history of well-controlled essential hypertension presented to the emergency department with 1-month history of abdominal bloating associated with dry mouth and constipation, decreased appetite, 20-lb weight loss, and muscle weakness. He was otherwise healthy, a non-smoker and exercised regularly.</p> <p>On examination, weight was 80.9 kg, BMI 24.969 kg/m², heart rate 65, blood pressure 136/85. Complete eye movements were intact without diplopia, patient had facial symmetry, elevation of the soft palate was symmetrical, tongue protrusion midline, remaining neurological exam was normal. Cardiovascular and respiratory examinations were within normal limits. Abdomen was soft and slightly distended, bowel sounds diminished.</p> <p>Investigation: Hemoglobin 15.7 g/dL, WBC count 7.8 x 10⁵/μL. Urine examination, renal profile, and liver profile were also normal. In paraneoplastic work-up, antibodies to skeletal muscle acetylcholine receptors were <0.30, within normal limits. EMG study was normal.</p> <p>In the diagnostic process, CT of the abdomen and pelvis showed marked gastric dilation. CT of the chest revealed 6.4 x 5.8 x 4.7 lobulated well marginalized mass in the anterior mediastinum. Mass was surgically excised and histology showed lymphocyte predominant thymoma without capsular invasion, type B1.</p> <p>Two weeks after surgical excision of the thymoma, the patient had drooping of his left eyelid in addition to his GI symptoms. Diagnoses of intestinal pseudo-obstruction and dysautonomia of possible paraneoplastic origin were considered. A trial of pyrostigmine showed no improvement, however, the patient began to improve immediately after starting corticosteroids.</p> <p>Conclusion: Intestinal pseudo-obstruction is a classic paraneoplastic syndrome, however, its occurrence in thymoma is rare. The commonly reported paraneoplastic syndromes associated with thymoma include Myasthenia Gravis, Lambert-Eaton Syndrome, neuromyotonia, and autonomic neuropathy. There have been 9 cases in the literature of intestinal pseudo-obstruction as a paraneoplastic syndrome, all of which were associated with Myasthenia Gravis. Symptoms in these cases ranged from isolated gastroparesis to pan-autonomic dysfunction. Antibodies to nicotinic acetylcholine receptors on skeletal muscle results in myasthenia gravis; antibodies to neuronal nicotinic acetylcholine receptors results in paraneoplastic autonomic neuropathy. We hypothesize that our patient would have tested positive for the latter. In this case the antibodies against nicotinic acetylcholine receptors on skeletal muscle were</p>

					within normal limits; however, the antibodies for neuronal acetylcholine antibodies were not tested. These antibodies can impair transmission at autonomic ganglia and antibodies interfering with transmission would lead to dysautonomia presenting as intestinal pseudo-obstruction.
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Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Maanit Kohli	Dr. C.S Pitchumoni	Saint Peter's University Hospital (Nayan Kothari)	Double Trouble; Concomitant EBV & Acute Hepatitis B	<p>Acute viral hepatitis is a systemic infection affecting the liver and is most commonly caused by the Hepatitis A-E viruses and rarely EBV. In cases of Acute Hepatitis B the first virology marker is HBsAg, which becomes undetectable 1–2 months after the onset of jaundice and during the window period, anti-HBc IgM may represent the only serologic evidence recent HBV infection. EBV diagnosis is confirmed with presence of EBV VCA IgM in the appropriate clinical setting.</p> <p>23 year old male with PMH of IBS, Cobalamin C defect with recent diagnosis of Infectious Mononucleosis (2 weeks ago) presented to the ER with left lower quadrant abdominal pain, unremitting fevers ranging between 101-103F despite NSAID use and sore throat for the last 2-3 weeks. Social history was significant for unprotected sexual intercourse and a new tattoo placed, both within the last 3 months. On examination patient found to be febrile, tachycardic, having scleral icterus with a papular rash present over chest and arms, enlarged tonsils with exudates, cervical lymphadenopathy, palpable spleen and abdominal tenderness. Labs were significant for thrombocytopenia 41, positive EBV screen / EBV VCA IGM elevated, with transaminitis with a cholestatic picture AST/ALT 231/431 ALP 436, T.bilirubin of 9.3, INR 1.00. Imaging; Splenomegaly with perisplenic fluid collection seen on CT abdomen.</p> <p>Patient was admitted with a working diagnosis of concomitant EBV and Acute viral hepatitis B. Other causes of viral hepatitis such as HIV, HSV, HCV, and CMV were ruled out with serological testing. NSAIDs were avoided in this setting to prevent further hepatotoxicity. Fevers and abdominal pain subsided with supportive care initiated during the hospitalization A fall in hepatic transaminases was observed with levels AST 99 ALT 177 ALP 267 T-Bill 3.8 at the time of discharge. Patient was discharged home with instructions to avoid heavy physical activity, counselling on safe sex practices and follow up appointments with GI and ID departments.</p> <p>Though Hepatitis A-E viruses cause most cases of viral hepatitis, EBV infections can affect any organ system and should remain in the differential of viral hepatitis. The clinical course is usually self-remitting and managed with supportive care measures of hydration, adequate rest, and antipyretics with minimal role of steroids and antivirals in treatment.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Shweta Kumar	Yegneswaran Balaji	Saint Peter's University Hospital (Nayan Kothari)	Immunosuppressive drugs predisposing to pulmonary infections by Streptococcus milleri group	<p>Introduction: The Streptococcus milleri group (also known as the S. anginosus group) is a subgroup of viridans streptococci that consists of three distinct species: S. anginosus, S. intermedius, and S. constellatus. The organisms were subsequently recognized as normal flora of the human oral cavity and gastrointestinal tract with the ability to cause abscesses and systemic infections. Predisposing risk factors include recent surgery, dental procedures and use of chronic immunosuppressive medications(1).</p> <p>Cases: We present two cases who developed invasive pulmonary infection with S.anginosus while on long term immunosuppressive therapy. First patient, a 65-year old male with history of stroke with residual aphasia and cerebral vasculitis taking mycophenolate and steroids presented with shortness of breath and altered sensorium. Physical examination revealed temperature 102.3 F, HR 100/minute, RR 18/minute, BP 86/45mmHg and decreased breath sound with crackles at right lower lung fields. Chest x-ray showed large right lower lobe infiltrate with effusion. The patient was mechanically ventilated and started on pressors. The loculated empyema was drained with VATS procedure and the abscess sample was sent for culture which grew S.constellatus. The patient was treated with Ceftriaxone and was later extubated and came off pressors. Another patient, a 74-year old female with past history of diabetes, hypertension, rheumatoid arthritis taking infliximab presented with coughing and shortness of breath. On examination, T 101 F, HR 98/minute, RR 17/minute, BP 110/85mmHg, reduced breath sounds at right lung base. Chest x-ray showed right lower lobe infiltrate and CT-scan showed right lower lobe abscess. The culture revealed abscess caused by S.intermedius. She was treated with Unasyn and the abscess was drained under CT-guidance.</p> <p>Discussion: The main safety concern with the use of immunosuppressive agents is the risk of infections. Physicians must adopt preventative strategies and should have a high degree of suspicion to recognize infections early and treat appropriately. Pulmonary infection with S. milleri may result in considerable morbidity and mortality(2). Despite attempts at non-operative management, the majority of patients with a S. milleri pleural space infection require operative intervention for definitive therapy.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Lehaz Kaker		Saint Peter's University Hospital (Nayan Kothari)	Abdominal Pain: a rare case of double acute abdomen.	<p>Introduction: Co-occurrence of acute cholangitis and acute appendicitis is rare. Here we present a case of 48 years old Caucasian male who presented with abdominal pain and was found to have double acute abdomen.</p> <p>Case: A 48 year old Caucasian male with no significant past medical history, presented with two days history of constant dull peri-umbilical pain on the right side that did not radiate or migrate. It was associated with chills and two episodes of vomit. Patient did not report any change in bowel or bladder habit. On examination patient was febrile and hypotensive but alert and oriented. There was no scleral icterus. Abdomen was soft but tender with guarding over LLQ. Murphy's was negative. There was no hepato-splenomegaly, no shifting dullness. Hernial orifices were intact. Initial blood tests revealed bandemia (WBC10.3; Band 10.0, N 82.0, L 7.0, M 1.0), transaminitis (AST 108, ALT 192, Alk Phos 96) and Hyperbilirubinemia (Total 3.9, Direct 1.6, Indirect 2.3). Serum Lipase level was 12. Ultrasound of the abdomen showed cholelithiasis and abdominal CT findings were consistent with acute appendicitis. Vital signs improved with fluid resuscitation and IV antibiotics. He underwent emergent laparoscopic appendectomy followed by ERCP the following day. Pathology of the specimen showed acute appendicitis and cholecystitis with reactive lymph nodes.</p> <p>Discussion: Acute abdomen due to more than one etiology is rare in a young healthy person. It often occurs in patients with underlying chronic intra-abdominal processes, such as, Primary Sclerosing Cholangitis or Ulcerative colitis, in immunocompromised patients related to CMV infection and Geriatric population. However, even in such at risk population co-occurrence at the same time is uncommon. There is no proven link between the acute appendicitis leading to acute cholangitis. It is postulated that intra-abdominal inflammation can lead to cholestasis if not curtailed adequately by Omentum. Second theory implicate hematogenous and lymphatic spread of infective agents via portal system. Notwithstanding the flow of lymph from hepatobiliary and portal areas is in opposite direction toward para-aortic lymph nodes.</p> <p>Conclusion: Acute cholangitis and acute appendicitis rarely occurs together in a young healthy person. Apart from history and physical examination, meticulous attention to investigations is important in order to consider every possible diagnosis, as it is well summed up by Dr John Bamber Hickam; "Patients can have as many diseases as they damn well please."</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Nissy Philip	Shounak Majumder, MD, Santhi Swaroop Vege, MD	Saint Peter's University Hospital (Nayan Kothari)	High-Grade B-cell Lymphoma mimicking an unresectable pancreatic carcinoma	<p>Introduction: Diffuse large B-cell lymphoma (DLBCL) is the most common histologic subtype of non-Hodgkin's lymphoma (NHL). Advances have further subcategorized DLBCL. Although extranodal and extramedullary disease frequently accompanies the initial diagnosis, pancreatic involvement is distinctly uncommon.</p> <p>Case Report: A 58-year-old male presented with the chief complaint of left upper quadrant abdominal pain of 6 weeks duration. An abdominal CT scan revealed a poorly enhancing heterogeneous mass in the tail of the pancreas extending to the splenic hilum. Multiple splenic masses and enlarged upper abdominal lymph nodes were noted suggesting metastatic pancreatic adenocarcinoma. The patient had a long-standing history of cigarette smoking and alcohol use. He denied weight loss, fever or night sweats. He was referred for further management. Endoscopic ultrasound (EUS) demonstrated a 3 cm hypoechoic mass in the pancreatic tail with well-defined margins extending to the splenic hilum, enlarged peripancreatic lymph nodes and multiple discrete hypoechoic splenic lesions. EUS-guided fine needle aspiration and core biopsies of the pancreatic tail mass revealed an atypical lymphocytic infiltrate associated with areas of necrosis suspicious for large B-cell lymphoma. The diagnosis of high-grade B-cell lymphoma, which is similar to DLBCL but cytologically and clinically more aggressive, was subsequently confirmed on a CT-guided core biopsy from enlarged retroperitoneal lymph nodes. The lymphoma lacked a MYC rearrangement by interphase fluorescent in-situ hybridization (FISH) analysis, supporting the diagnosis of high-grade B-cell lymphoma, not otherwise specified, per the 2016 WHO Classification of Lymphoid Neoplasms.</p> <p>Conclusion: Aggressive NHL's such as DLBCL and high-grade B-cell lymphoma should be considered in the differential diagnosis of a pancreatic mass and may be associated with a better prognosis than pancreatic adenocarcinoma. The presence of extensive intra-abdominal lymphadenopathy and concomitant splenic lesions are important diagnostic clues.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Skand Shekhar	David Alcid, MD	Saint Peter's University Hospital (Nayan Kothari)	Lemierre's syndrome presenting as a hepatic metastatic lesion: A case report	<p>Introduction: Lemierre's syndrome (LS) was first described as a clinical entity as a secondary septic thrombophlebitis after a preceding oropharyngeal infection, most commonly by <i>Fusobacterium necrophorum</i> a common component of the oral flora affecting otherwise healthy teenagers and young adults after a short course of oropharyngeal infection.</p> <p>Case Description: We are reporting the case of a 62 year-old male who had presented with fever and chills for three days prior to presentation. He reported soreness of throat for three days prior to presentation. On arrival, the patient was in shock and his hypotension responded to intravenous fluid boluses. His physical exam was normal except for dry mucous membranes. Labs revealed: Hemoglobin= 12.3g/dl, Hematocrit= 39.0, platelet count= 1,41,000/mm³ leukocytosis of 15,000/mm³ with predominant neutrophils and a metabolic panel showed BUN= 38/ serum creatinine =2.02mg/dl with a lactic acid of 4.2mmol/L. A chest x-ray was normal. A CT abdomen initially showed left lobe liver lesion 6cm in diameter, with a suspicion of malignancy . He was treated with broad-spectrum antibiotics for a superimposed infection. An abdominal ultrasound was consistent with a necrotic liver mass strengthening the empirical diagnosis of malignancy with a paraneoplastic leukocytosis. On day four of hospital stay his blood culture x 1 came back positive for gram-negative bacilli and his white count increased. An ultrasound guided liver tap drained pus and blood cultures reported <i>Fusobacterium nucleatum</i>. A CT of the neck revealed a right internal jugular venous thrombus (septic) confirming the diagnosis of Lemierre's syndrome. He was discharged on a six-week course of IV antibiotics and did well subsequently.</p> <p>Discussion: A review of literature done by Iwasaki et al revealed only one case of liver abscess in a person above the age of sixty years out of a total of 13 cases with a mean age of affection being 21.7 years. It also showed that majority of the cases had involvement of other organ systems with sole liver involvement being relatively infrequent. When liver was affected 8 out of 13 cases had multiple liver abscesses like this case as opposed to 5 out of 13 having a solitary abscess. Riordan T et al reviewed Lemierre's syndrome and found that only 4% of patients had hepatic metastatic manifestations. However liver enzyme elevations were as common as 49% in all cases of Lemierre's syndrome. This was seen in this case as well in addition to subsequent findings of a liver abscess. This case exemplifies how a relatively uncommon infectious syndrome can present with variable manifestations including those mimicking a neoplastic process. A high index of suspicion for this syndrome will help in early accurate diagnosis and successful therapy.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Kartik Kalra	Greg Matthew Teo	Saint Peter's University Hospital (Nayan Kothari)	Hypertriglyceridemia – A Neglected Cause For Acute Pancreatitis	<p>Introduction: Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis (AP) after alcohol and gall stones. It is reported to cause 1-4 % of all cases of AP and 56% of pancreatitis during pregnancy. A Triglyceride (TG) level of more than 1,000 mg/dL is needed to cause an episode of pancreatitis. Here we present 4 patients diagnosed with hypertriglyceridemic pancreatitis.</p> <p>Cases:</p> <p>Case 1 – 33 year old Filipino male with history of diabetes and obesity, was admitted for AP. APACHE-II score did not improve despite adequate IV fluids. TG levels were found to be at 2556 mg/dL 48 hours after admission. He was started on regular insulin drip and gemfibrozil. He gradually improved and was discharged with TG level at 617 mg/dL.</p> <p>Case 2 – 49 year old Caucasian male with a history of neuroendocrine tumor was admitted for AP. Patient was undergoing chemotherapy with Everolimus. His TG levels were at 9550 mg/dL on admission. He was started on regular insulin drip, Everolimus was discontinued and gemfibrozil was started. Patient improved and was discharged after 9 days with TG level at 421 mg/dL.</p> <p>Case 3 – 41 year old Sri Lankan male with HTG who was non-compliant to gemfibrozil, was admitted for AP. TG level on admission was 2219 mg/dL. Gemfibrozil was resumed but patient was not started on Insulin drip. Patient had gradual improvement of pain and discharged on a subcutaneous insulin regimen.</p> <p>Case 4 – 31 year old Indian male with history of heavy alcohol use, was initially admitted for alcohol-induced pancreatitis. Patient did not improve in the next 48 hours after admission. TG levels were then found to be at 1870 mg/dL. Patient was then started on Insulin drip and fenofibrate. TG level on discharge was 401 mg/dL.</p> <p>Discussion: The typical clinical profile of hyperlipidemic pancreatitis is a patient with a preexisting lipid abnormality along with the presence of a secondary factor (e.g., poorly controlled diabetes, excessive alcohol use, or a medication) that can induce HTG. The authors believe that a lipid profile should be part of standard work up when admitting patients with AP so that patients with HTG can be identified early and be started on the appropriate therapy. Regular insulin infusions are already appropriate in decreasing triglyceride levels as demonstrated in all 4 cases. Plasmapheresis is not necessary for all patients with HTG pancreatitis and should be reserved for patients who do not have clinical improvement with insulin drip or who already have severe presentation upon admission. Maintenance treatment to prevent recurrent HLP include high-dose fibrates or a combination of fibrate plus niacin. The mainstay of treatment still includes dietary restriction of fat in addition to managing the secondary or precipitating causes.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Alvaro Cornejo Cobo		Saint Peter's University Hospital (Nayan Kothari)	Surgical management for non-resolving pulmonary abscess	<p>Background: Aspiration pneumonia can be complicated with lung abscess, and its management can be a challenge. When lung abscess fails common antibiotic therapy, we need to decide if per-cutaneous drainage or surgical procedure should be performed.</p> <p>Case Report: A 78-year-old gentleman with a past medical history of Parkinson disease presented with chronic nonproductive cough going on for the last month. On physical examination, he was found to be tachypneic, with low O2 Sat, but no added sounds on the lung fields. Lab results showed leukocytosis and a chest x ray showed a cavitary lesion in the right lung field, which was later found to be an abscess on CAT scan. He was started on antibiotic therapy with Zosyn and Vancomycin, was admitted to ICU and had to be intubated for respiratory failure. After 5 days of antibiotic therapy there was no clinical improvement. Patient underwent bronchoscopy with bronchoalveolar lavage which grew Klebsiella. Clindamycin was started for aspiration pneumonia, but there was no improvement over the next two days. Right middle lobectomy was performed, antibiotic therapy was continued, with marked improvement of symptoms.</p> <p>Discussion: Lung abscess is a complication of aspiration pneumonia. Decreased consciousness, alcoholism, drug abuse, anesthesia, dysphagia and chronic diseases like Parkinson or late Alzheimer highly predispose for aspiration pneumonia. Symptoms characteristic of lung abscess are similar to CAP. Chest X-ray will show a cavitary lesion with fluid-air level, but CT can be necessary to confirm diagnosis. Most common pathogens are anaerobes present in the mouth like Peptostreptococcus, Prevotella, Bacteroides and Fusobacterium, but infection can also be caused by S. Aureus and gramm negative bacilli like Klebsiella pneumoniae. Less common bacteria are Streptococcus, Haemophilus Influenza and Moraxella catharrallis which are common pathogens for CAP. Clindamycin is the best initial therapy, but because most lung abscess are polymicrobial, addition of broad spectrum beta lactam with beta lactamase inhibitor is mandatory. Vancomycin should be added if MRSA is suspected. Duration of therapy is controversial, but it's usually several months. Approximately one third of patients dies, one third recovers and one third develops complications like empyema, bronchiectasis, recurrent abscess or other sequelae. Surgical intervention is rarely required for uncomplicated lung abscess. Indications are neoplasm, hemorrhage, empyema of failure to respond to medical management which is usually subjective. Bronchoscopy can be used as diagnostic procedure and if blockage of a bronchioli is suspected as the probable cause of the abscess it may be used for treatment. Percutaneous procedure is another option, but is rarely used because of complications. When surgical intervention is preferred, lobectomy or pneumonectomy is usually performed. There are some clear-cut predictors suggest that surgery should be performed; obstructed bronchus, resistant organism such as Pseudomona Aeruginosa, and extremely large abscess >6cm.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Girija Phadke		Saint Peter's University Hospital (Nayan Kothari)	Unusual Presentation of Non-Hodgkin's Lymphoma with Decompensated Systolic Heart Failure.	<p>INTRODUCTION: Cardiac dysfunction heralding as malignant lymphoma is rare.¹ We present a case of a patient presenting with decompensated systolic heart failure who was found to have stage IV non-Hodgkin's lymphoma (NHL).</p> <p>CASE: 51 year old male with no past medical history presented to the emergency room with several weeks history of swelling over his body which started around his eyes which then progressed to swelling in his legs. He also reported worsening exertional dyspnea. On exam he was tachycardic with HR 104/min, BP was 95/57 mm Hg. He had crackles on lung exam and significant periorbital swelling. He had 3+ pitting edema to his thighs. Generalized lymphadenopathy in bilateral inguinal, axillary and cervical regions was noted. EKG showed sinus tachycardia with incomplete right bundle branch block. Metabolic panel showed elevated total protein of 11.4 g/dl with low albumin of 1.7. He had no proteinuria and normal creatinine levels. He also had anemia with hemoglobin 7 mg/dl.</p> <p>Transthoracic echocardiogram showed concentric LVH and diastolic dysfunction with EF of 20%, with hypokinetic right ventricle. Mild septal hypertrophy of 13 mm and RA enlargement were seen. CT thorax, abdomen and pelvis was done which showed confluent lymphadenopathy in mediastinal, peri-aortic and inguinal regions. Lymph node biopsy was performed showing low grade B-cell follicular lymphoma. Bone marrow aspiration showed extensive involvement.</p> <p>Work-up for paraproteinemia was done which showed elevated serum IgG and IgM levels and beta-2-microglobulin levels. Elevated kappa and lambda light chains and a grossly abnormal kappa/lambda ratio were found raising possibility of myeloma associated amyloidosis.</p> <p>Patient was diuresed and was started on medical management of heart failure with Lisinopril, and Carvedilol on discharge. He is supposed to have chemotherapy with Rituximab. He also needs further further work-up such as cardiac MRI and catheterization as an outpatient.</p> <p>DISCUSSION: This is an interesting case as cardiac involvement by NHL is usually an autopsy finding as most patients do not present with clinically significant cardiac manifestations.² It is also unique as patient was additionally found to have myeloma associated amyloidosis- which may also be a possible etiology of cardiomyopathy. As cardiac involvement is known to be a poor prognostic sign, it is paramount that such involvement be detected and aggressively managed to improve outcomes.</p> <p>1Hafeez I, Alai MS, Iqbal K, et al. Lymphoma Presenting as Severe Left Ventricular Systolic Dysfunction: A Case Report. Oman Medical Journal. 2014;29(3):223-225. doi:10.5001/omj.2014.55.</p> <p>2Mioulet D, Braem L, Heno P, Paule P, et al. Cardiac extension of a non-Hodgkin lymphoma revealed by an atrial flutter. Ann Cardiol Angeiol (Paris) 2009. Apr;58(2):117-121 10.1016/j.ancard.2008.05.007</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Shivam Mital	Arun Mattappallil PharmD, David Alcid MD	Saint Peter's University Hospital (Nayan Kothari)	Penicillin Allergic Patients and Therapeutic Challenges – An Inpatient Solution?	<p>INTRODUCTION: Various penicillin “allergies” are reported by up to ten percent of individuals. This represents a challenge for inpatient treatments, particularly for those who require intravenous antibiotics. These patients experience fewer treatment options, and may be at an increased risk of side effects from less-targeted therapy. Moreover, there are often increased healthcare costs associated with administering certain alternate antibiotics.</p> <p>Another concern is whether these individuals can safely receive antibiotic classes that are structurally related to penicillins – such as cephalosporins, carbapenems, and monobactams – since the beta-lactam structure of penicillins is also found in those three classes. Among patients with true penicillin reactions, up to 8.4 percent may react if given a cephalosporin; Amoxicillin and Ampicillin each have R-group side chains identical to the side chains of certain cephalosporins.</p> <p>The World Allergy Organization divides immunologic drug reactions into two categories:</p> <ol style="list-style-type: none"> I. Immediate reactions (Type I, IgE-mediated): Usually occurring within the first hour of an administered dose. II. Delayed Reactions (Types II-IV, non-IgE-mediated, or other mechanisms): Usually occurring at least six hours after exposure. <p>Studies performed on patients with various drug reactions have shown that less than twenty percent react to the offending agent upon direct challenge. Likewise, more than three-fourths of patients with IgE-mediated penicillin allergies will have lost their sensitivity after ten years. Notably, a drug may never have caused an adverse event, but was implicated due to a temporal association.</p> <p>METHODS: Our outpatient pilot study will enroll ten patients before advancing to the inpatient setting. We are utilizing the FDA-approved PRE-PEN® (benzylpenicilloyl polylysine injection USP) penicillin allergy skin test kit. When combined with Penicillin G and a Histamine control, this (a) “scratch” and (b) “intradermal” testing protocol has a negative predictive value for IgE-mediated penicillin allergies of almost ninety-nine percent. Negative results can be further substantiated with an oral penicillin challenge test, which may become a component of inpatient testing.</p> <p>Pilot Study Inclusion Criteria:</p> <ol style="list-style-type: none"> I. Indeterminate allergy to penicillins or aminopenicillins, such as (a) drug intolerance [e.g. nausea, vomiting, GI discomfort], (b) drug idiosyncrasy [e.g. arthralgias], or (c) anaphylactoid reactions [e.g. rash]. II. No histamine antagonists or leukotriene receptor antagonists used in the prior 24 hours. III. Amenable skin surface is available on the arms or back to perform the test accurately.

					<p>Pilot Study Exclusion Criteria:</p> <ul style="list-style-type: none">I. Recent (ten years or less) anaphylaxis or any history of severe reaction (Stevens-Johnson syndrome, Toxic Epidermal Necrolysis, Interstitial Nephritis) to penicillin or aminopenicillins.II. Patient is or may be pregnant.III. Under the age of eighteen. <p>RESULTS: To date, four patients have been enrolled in our pilot study. Two patients exhibited no signs / symptoms of a localized or generalized adverse reaction to the penicillin components.</p>
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Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Osakpolor Ogbemor	Ruwaa Samarra BA, Devin Walford Godwin BA, Sonmoon Mohapatra, MD.	Saint Peter's University Hospital (Nayan Kothari)	Headache as a presentation of IgG4 related disease	<p>Introduction: IgG4 related disease (igG4-RD) affects multiple systems and patients often present with a myriad of symptoms. A high index of suspicion is needed in making a diagnosis and guiding the course of treatment.</p> <p>Case Description: A 53-year-old lady was admitted into the hospital with headache and altered mental status. Headache was described as frontal and constant with an onset about one week prior to presenting to the hospital. She presented to the emergency department (ED) twice and each time she was commenced on simple analgesia and migraine medications following unremarkable blood tests and CT scan. She presented to the ED a third time because at this point she had developed altered mental status as reported by her husband. Her medical history is significant for Primary biliary cholangitis (PBC), hypothyroidism, and hypertension.</p> <p>In view of her symptoms, the likely hood of meningoencephalitis was considered. She was started on empiric treatment which included ceftriaxone, vancomycin, ampicillin, and acyclovir. A repeat CT Head without contrast was normal. On MRI Brain, there was diffuse flair signal abnormality throughout the sulci bihemispheric with mild leptomeningeal enhancement.</p> <p>CSF analysis confirmed a diagnosis of aseptic meningitis with white cell count of 564, of which 100% were lymphocytes, elevated protein of 144 mg/dL, and a normal glucose level. Her empirical treatment was deescalated to only acyclovir and following a negative HSVPCR screen this was stopped. Viral panel, Lyme screen, gram stain and bacterial culture were negative. The patient remained symptomatic after 7 days on admission. On review of the patients history it was noted that, besides PBC, a diagnosis of IgG4 related cholangitis was also made a year prior to her current presentation. Histology at the time showed fibrosis of the large bile duct. IgG4 serology levels were also elevated. The patient was started on empirical glucocorticoids with dramatic clinical improvement observed in her symptoms. She was discharged on a taper regimen of steroids to follow up in one week. CSF results returned positive for elevated IgG levels supporting the diagnosis of IgG4 mediated pachymeningitis.</p> <p>Discussion: IgG4- RD remains as systematic disease with a wide range of pathology involvement. Dramatic initial response to therapeutic glucocorticoids is a characteristic feature of the disease. Other features include lymphoplasmacytic infiltrate and fibrosis. . This case offers another instance of extra-gastrointestinal manifestations of this autoimmune condition and highlights the importance of clinicians to be aware of the many different manifestations of IgG4-RD. Headache is a common presentation of IgG4 mediated pachymeningitis and elevated CSF IgG levels is also seen. This case also shows the significance of detailed medical history in broadening the differential diagnosis especially when the patient does not respond to conventional treatment.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Surya prakash		Saint Peter's University Hospital (Nayan Kothari)	Water is toxic: Case of psychogenic polydipsia with hyponatremia	<p>Abstract: Psychogenic polydipsia (PPD) is a rare condition characterized by polydipsia and polyuria. It sometimes leads to severe life threatening hyponatremia. I report a young male admitted for syncope secondary to hyponatremia from primary polydipsia.</p> <p>Introduction: Water intoxication can occur in a variety of clinical settings but is generally not well recognized. It is more commonly associated with chronic schizophrenics. The condition may go unrecognized in the early stages when the patient may have symptoms of confusion, syncope and changes in psychotic symptoms. Early detection is crucial to prevent severe hyponatremia, which can be life-threatening. This is a case of young schizophrenic male who had hyponatremia secondary to PPD.</p> <p>Case Report: This is a 38 yr old male with hx of seizure disorder (5 yrs) on Keppra/trileptal/citalopram , hx of schizophrenia in past (currently not on rx) admitted after syncope while micturition and some mild confusion. Patient on arrival in ER was asymptomatic and had no confusion. Vitals and Physical examination were within normal limits. Careful questioning revealed patient had polydipsia/polyuria and had been drinking>5 liters/day for a week.</p> <p>Labs revealed serum sodium 123, creatinine 0.7, serum osm 275, urine osm 106, and urine sodium 18 consistent with Primary polydipsia. Head CT and rest of labs wnl. Water restriction was started and sodium corrected to 134 within 8 hrs. Pt given DDAVP and D5W to reverse rapid correction. By day 2 sodium was 134, and pt discharged home with education about need to avoid excess water.</p> <p>Discussion: PPD occurs in 25% of patients with schizophrenia. Pathophysiology is complex and multifactorial - malfunction of the hypothalamic thirst center is seen as likely cause. It usually occurs in three phases- beginning with polydipsia and polyuria, followed by hyponatremia (water is retained as the kidneys fail to excrete the excess fluid) and finally water intoxication leading to symptoms secondary to movement of fluid into brain cells. Symptoms range from confusion, vomiting, syncope and can eventually lead to seizures, coma and death.</p> <p>Differentials include other cause of hyponatremia especially SIADH. Careful history along with urine osmolality helps us to differentiate these conditions. PPD usually has low urine osmolality in contrast to SIADH (urine osm>100). Water restriction remains main stay of treatment. In severe cases, however, hypertonic saline solution is recommended. Sodium should be closely followed and rapid correction may have to be reversed with DDAVP to prevent CPM.</p> <p>Conclusion: Psychogenic Polydipsia is an unusual cause of hyponatremia that may go unnoticed without an appropriate clinical history that carries high mortality if not promptly recognized. Patient education and water restriction remains cornerstone in preventing future episodes of water intoxication.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Swati Chopra		Saint Peter's University Hospital (Nayan Kothari)	Lithium toxicity in Elderly: Role of ACEI- case report	<p>Introduction: Lithium is commonly used for unipolar and bipolar affective disorder. Lithium has a narrow therapeutic range (1–1.5 mEq/L during acute episode therapy and 0.6–1.2 mEq/L during maintenance therapy). The low therapeutic index and significant toxicity limits its clinical utility. Elderly patients are at particularly high risk of lithium toxicity because of altered pharmacokinetics, polypharmacy and renal impairment.</p> <p>Case Description: 77 year old female with medical history significant for hypothyroidism, atrial fibrillation, hypertension, bipolar disorder presented with lethargy and confusion for 2 weeks. The patient had diarrhea for 3 days before admission. Her medications included : levothyroxine 200mcg once daily, lisinopril 40mg once daily, lithium 300mg twice a day, rivaroxaban 5mg twice a day. Her vitals were normal except for hypotension with negative orthostatics. Her physical examination was positive for coarse tremors . Laboratory studies revealed elevated white cell count of 19.7, hyponatremia of 128mEq/L with fraction excretion of sodium of 0.2%, elevated creatinine of 3.19mg/dl, urine specific gravity of 1.018, elevated lithium level of 2.6meq/L , CT scan of the head was within normal limits. The patient was started on IV fluids, lisinopril was discontinued and daily lithium levels were followed which decreased to 0.70meq/L in six days, along with decreasing creatinine levels. Despite being on isotonic intravenous fluids, the patient developed hypernatremia secondary to transient nephrogenic diabetes insipidus which improved after initiation of chlorthalidone.</p> <p>Discussion: Lithium toxicity can be life threatening and elderly patients are especially at risk for lithium toxicity due to both a lower glomerular filtration rate and reduced volume of distribution. Lithium toxicity can occur with minor declines in renal function or from dehydration. The concomitant use of loop diuretics and ACEI in elderly population increases the risk of hospitalization for lithium toxicity.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Pablo Garcia	Pablo Garcia MD, Felix Renneberg, Steve Bibu MD, Anne Van Hoven MD	Saint Peter's University Hospital (Nayan Kothari)	DIAGNOSIS AND TREATMENT OF RENAL VEIN THROMBOSIS IN THE SETTING OF LUPUS NEPHRITIS AND ANTIPHOSPHOLIPID SYNDROME	<p>Introduction: Renal vein thrombosis (RVT) is a presence of thrombus in the major renal veins. May present with acute symptoms or go unnoticed. The mechanism of RVT in nephrotic syndrome is still unclear. However, alterations in plasma levels of proteins involved in coagulation and fibrinolysis are considered predisposing factors for its development. Antiphospholipid Syndrome (APS) is a systemic autoimmune disorder characterized by venous or arterial thrombosis in the presence of evidence of antiphospholipid antibodies (aPL).</p> <p>Case description: A 29-year-old Puerto Rican female with Systemic Lupus Erythematosus (SLE) for 3 years presented with excruciating left flank pain and tenderness. The initial diagnosis of RVT was made clinically and confirmed with a CT scan of the abdomen. Heparin and warfarin was started. We obtained aPL and was positive for Lupus Anticoagulant. Creatinine 0.59 mg/dl, CRP 42.0 mg/L and ESR 105 mm/hr. Diminished complement proteins C3 (54mg/dL), and C4 (9mg/dL). In addition to the RVT the initial urinalysis showed marked proteinuria therefore we obtained a protein/creatinine ratio which showed marked proteinuria 2.6 g/g. We performed a kidney biopsy for diagnosis and establishing optimal therapy which showed diffuse segmental endocapillary and focal extracapillary proliferative and membranous glomerulonephritis, consistent with lupus nephritis Class IV-S and Class V. Therefore we started her on methylprednisolone 500 mg IV for three days. Posteriorly the patient was discharged with marked clinical improvement, on warfarin indefinitely, prednisone 1mg/kg daily, hydroxychloroquine, lisinopril and mycophenolate.</p> <p>Discussion: We describe the diagnosis and management of renal vein thrombosis (RVT) in a female with Systemic Lupus Erythematosus (SLE), lupus nephritis and APS. Diagnostically, this represented a challenge because of the clinical presentation leading us to a broad spectrum of differential diagnosis. But was facilitated by using imaging test (CT scan of the abdomen). Furthermore, the management represented a challenge due to the concomitant lupus nephritis and APS. The treatment of lupus nephritis Class IV-S and Class V requires prompt intervention with steroid pulses for three days and addition of an immunosuppressant drug like mycophenolate or cyclophosphamide and long term prednisone. Patients with SLE and APS have an increased risk of thromboembolic disease. Anticoagulation with warfarin in symptomatic patients should be initiated to prevent thromboembolism. In this case with hypoalbuminuria due to proteinuria and APS we opt for lifelong anticoagulation. In addition to the treatment these patients require close follow up to assess response to treatment measuring protein in urine, complement levels, kidney function and inflammatory markers ESR and CRP.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Prachi Pophali	Paris Charilaou, David Alcid	Saint Peter's University Hospital (Nayan Kothari)	Elevated B-type Natriuretic Peptide (BNP): Look beyond what is envisaged.	<p>Case: A 54-year old Caucasian woman was sent by her primary care provider for evaluation of raised BNP levels. She had high-grade fevers with chills and generalized malaise four week ago when was treated with antibiotics for asymptomatic urinary tract infection; her fevers resolved but generalized body aches worsened to a point she could not get out of bed. She reported occasional chest tightness at presentation. She recalled an insect bite behind her right knee few weeks earlier and described an erythematous ~5x5cm diffuse rash with blackish center on back of right knee which was almost resolved at admission. Physical exam showed multiple tender areas over chest, abdomen and limbs, diffuse fading rash over right lower thigh and temperature of 100.1F. EKG was normal sinus rhythm with normal intervals, chest X-ray and 2D-Echocardiogram was normal. Laboratory work-up was significant for hyponatremia, raised BNP (3130pg/ml), elevated troponin (0.14ng/ml) and urine positive for leukocyte esterase. Serology for Lyme antibody was positive and western blot showed positive Lyme IgM in 23KD and 41KD bands. Patient was treated with ceftriaxone 2gm iv daily in hospital and discharged on doxycycline 100mg for total of 28 days for Lyme carditis; at two week follow-up her symptoms had completely resolved.</p> <p>Discussion: Elevated BNP is a common laboratory abnormality that challenges internists. Usual causes include Congestive Heart Failure, Chronic Obstructive Pulmonary Disease etc. Presence of fever and generalized body pain with elevated BNP points towards an infectious cause, diagnosis of Lyme disease in such patients would be challenging especially in absence history of tick bite and/or absence of classic rash indicative of source.</p> <p>Lyme disease is the most common vector-borne infection in USA. It is caused by spirochete <i>Borrelia burgdorferi</i> and transmitted by Ixodes tick. The characteristic “bull’s-eye” rash ‘Erythema Migrans’ which has uniform erythema with central clearing is not seen in 20% cases. In our patient, the atypical rash and vague symptoms could have been wrongly attributed to UTI with a possible drug eruption, should her BNP not have been elevated.</p> <p>Lyme is diagnosed by a two-tier testing- screening is done with enzyme immunoassay or immunofluorescence assay which is confirmed with presence of IgM & IgG bands on western blot. First line therapy for carditis is I.V. ceftriaxone in acute phase with transition to oral doxycycline for total of 28 days. Complete resolution of symptoms is seen in majority of treated cases with no residual effect. Repetition of BNP levels to check for resolution of disease is not recommended.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Sonmoon mohapatra	Balaji Yegneswaran	Saint Peter's University Hospital (Nayan Kothari)	An unusual cause of dysphagia secondary to Gerbode ventriculo-atrial defect	<p>Introduction: Cardiovascular dysphagia is a rare clinical entity that is often unrecognized. One of the causes of cardiovascular dysphagia is giant left atrium and commonly seen in patients with mitral valve disease. The Gerbode defect is a ventriculo-atrial defect which can lead to volume overload and left atrial chamber enlargement if the defect is large. We describe a case of an elderly gentleman with a history of acquired Gerbode ventriculo-atrial defect who presented with complain of dysphagia due to enlarged left atrium.</p> <p>Case: An 89-year-old gentleman presented to the emergency for the evaluation of dysphagia and weight loss. He reported dysphagia for both solid and liquids, which had become worse over the past 6 months. He also had noticed 20 kg weight loss in last two years. His past medical history was significant for mitral regurgitation and acquired Gerbode ventriculo-atrial defect after an aortic valve surgery. Three year ago, patient was referred to cardiac surgery for repair of both mitral regurgitation and the ventriculo-atrial defect; however, he could only have mitral valve replacement with bioprosthesis due to surgical difficulties. Physical examination revealed a cachectic gentleman with normal vital signs. Cardiovascular examination revealed loud P2 with a 3/6 blowing systolic murmur at the left sternal border. Breath sounds were reduced at the base of the right lung. Electrocardiogram showed sinus rhythm with a non-specific intraventricular block. Chest X-ray demonstrated an enlarged cardiac silhouette with a small right pleural effusion. Barium esophagogram was done which showed compression of the mid esophagus from an enlarged left atrium. A computed tomogram (CT) confirmed the finding and revealed multi-chamber cardiac enlargement and evidence of mass effect on the esophagus. Echocardiography revealed an enlarged left atrium (6.1cm), enlarged right atrium and hypertrophic left ventricle. Color doppler studies showed presence of a perimembranous subaortic ventricular septal defect with left to right shunt but the jet was directed into the right atrium. Esophagogastroduodenoscopy revealed normal esophageal mucosa with narrowing of mid-esophagus. The patient was not considered a candidate for surgical closure due to multiple comorbidities. Hence, he was managed conservatively and started on mechanical soft diet which he tolerated well. The final diagnosis of cardiovascular dysphagia was made.</p> <p>Conclusion: Gerbode ventriculo-atrial defect is a rare defect that permits shunting from the left ventricle to the right atrium. The diagnosis of an LV-RA shunt is challenging, especially in the context of coexisting abnormalities including an additional intracardiac shunt, enlarged left atrium, tricuspid regurgitation, pulmonary artery hypertension and infective endocarditis. Early closures of these defects are recommended to avoid further complications and delayed management.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Nabil Ghani	Nazir Ahmed, Mary Tobiasson	Saint Peter's University Hospital (Nayan Kothari)	Pernicious Anemia with hemolysis in a young patient - a double rarity in a common disease	<p>Introduction: Pernicious anemia is a common cause of Vitamin B12 deficiency in US, usually occurring after age of 60 years. However, in African-Americans and Latin Americans it can occur at a lesser age. We describe a case of pancytopenia secondary to pernicious anemia in a 29-year-old African American woman with concomitant hemolysis.</p> <p>Case Report: A 29 year-old woman with no significant past medical history presented to the ER for evaluation of abnormal lab work results drawn at an Urgent Care Facility at which she presented 3 days prior. She complained of significant dizziness and fatigue. She had a similar episode 2 months back. At the ER her vitals were normal except for a heart rate of 129 per minute. She had sub-conjunctival pallor but rest of her physical exam was normal. Review of her labs showed pancytopenia with a hemoglobin of 3.1 g/dL (normal range: 12.1–15.9), MCV of 99.7 fL(normal range: 77.8–98), white blood cell count of 2500/dL (normal: 3540-9060) and platelet count of 57,000/dL (normal range: 177000–406000). Her reticulocyte was 1.1%. Iron studies were normal. Additional labs revealed haptoglobin <30 mg/dL (normal range: 36–195), LDH 3026 IU/L (normal range: 140–271), urobilinogen 8.0 mg/dL (normal: 0.2-1.0) and indirect hyperbilirubinemia, all indicating hemolysis. Other liver function and renal function tests were normal. Peripheral smear showed marked anisopoikilocytosis and hypersegmented neutrophils. Subsequently, work-up for pancytopenia was ordered. Pregnancy test, HIV antigen and antibody were all negative. TSH was normal. Vitamin B12 however, was found to be <50 pg/ml (normal is 180-914), folate was normal. Direct antiglobin (Coomb's) test was positive, suggestive of autoimmune hemolytic anemia. We ordered an intrinsic factor antibody test to look for a cause of Vitamin B12 deficiency; it came back positive.</p> <p>After daily parenteral Vitamin B12, patient's hemoglobin increased to 6.8 g/dL on fourth day of therapy, reticulocyte to 11.4% and WBC to 4200/dL. The patient was discharged on indefinite Vitamin B12 supplementation.</p> <p>Discussion: Vitamin B12 deficiency can present with hemolytic anemia in 1.5% of patients due to intramedullary megaloblast destruction. Though uncommon, Coomb's test can be positive in early pernicious anemia, making differentiation from AIHA difficult. Patients with true AIHA do not respond to vitamin B12 supplementation alone, needing steroids for resolution of anemia. Our patient however, responded remarkably early to vitamin B12 therapy. Importantly, though rarely, pernicious anemia can occur with AIHA, as both are primarily autoimmune disorders. Also, prevalence of vitamin B12 deficiency is 3% in patients aged 20 to 39 years. So, it is important for a physician to keep this differential diagnosis in mind while working up a case of pancytopenia or even hemolytic anemia with B12 deficiency in a young individual, especially if African-American or Latin American.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Matthew Tam	Sarah Kwan	Saint Peter's University Hospital (Nayan Kothari)	SGLT2 inhibitor induced diabetic ketoacidosis: two case reports	<p>Sodium-glucose cotransporter 2 (SGLT2) inhibitors are approved for use with diet and exercise by the Food and Drug Administration to lower blood sugar in adults with type 2 diabetes mellitus. SGLT2 inhibitors have been associated with increased risk of diabetic ketoacidosis (DKA). Ketoacidosis may not be recognized immediately due to blood glucose levels lower than expected for DKA which can result in the delay of recognition and treatment.</p> <p>Case 1: A 29-year-old male presented to the emergency department with abdominal pain and vomiting. Medical history was significant for diabetes mellitus with a recent hospitalization for first episode of DKA and hypertriglyceridemia 18 months ago. Medications included dapagliflozin, metformin, lisinopril, and simvastatin, started over 4 weeks prior. Physical examination revealed elbow xanthomas. Relevant laboratory studies included serum glucose 304mg/dL, anion gap 29, venous pH 7.14, and HbA1c 15.7%. Ketones were positive in serum and urine. A lipid profile revealed cholesterol of 1037mg/dL and triglycerides greater than 3000mg/dL. Abdominal CT was normal with no evidence of pancreatitis. He was treated for DKA which resolved the symptoms. The patient was advised to discontinue the SGLT2 inhibitor and was discharged on an insulin regimen.</p> <p>Case 2: A 47-year-old man presented to the emergency department with nausea, vomiting, and abdominal pain, associated with a two-day history of poor appetite. Past medical history was significant for type 2 diabetes mellitus and recent hospitalization 9 days prior for euglycemic DKA. Medications included canagliflozin started approximately 15 months ago, metformin, insulin glargine 29 units bid, and regular insulin sliding scale. On admission, serum glucose was 184mg/dL, anion gap 21, venous pH 7.32, and HbA1c 9.5%. Serum and urinary ketones were elevated. Abdominal ultrasound and CT did not show evidence of an acute process. He was diagnosed with and treated for recurrent euglycemic DKA, attributed to SGLT2 inhibitor use. Symptoms and laboratory findings resolved and he was discharged with canagliflozin cessation.</p> <p>Discussion: In Case 1, the patient's previous DKA and poor glycemic control together likely contributed to the presentation. It was also unknown whether the patient had Type 1 or Type 2 diabetes mellitus. These factors would be considered contraindications for SGLT2 inhibitor use and thus a preventable event. Case 2 exemplifies recurrent euglycemic DKA in the setting of SGLT2 inhibitor use and contributes to existing literature supporting the causal role for these medications in the pathogenesis of DKA. These cases together demonstrate observations under which circumstances SGLT2 inhibitors should be avoided. Clinicians should be aware of this risk and closely monitor their patients on these medications.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Andrew Novick		Saint Peter's University Hospital (Nayan Kothari)	Association between new onset of testicular microlithiasis and new extragonadal metastasis	<p>Introduction: In patients with a history of a testicular germ cell tumor, presence of existing testicular microlithiasis in the healthy contralateral testicle is likely associated with a higher risk of developing a new germ cell tumor in the healthy testicle. Furthermore, a possible link exists between newly diagnosed testicular microlithiasis and the risk of developing an extragonadal germ cell tumor likely of testicular origin. In a case report we document the presence of a newly developed testicular microlithiasis with the presence of newly developed extragonadal germ cell tumor, and in the absence of a new testicular tumor. Investigating this potential correlation could lead to new screening/monitoring guidelines for men at high risk for testicular cancer.</p> <p>Case: A 27 year old male presented to the ED with right testicular pain of 12 weeks duration. Physical examination revealed an enlarged, hard, painful mass in his right testicle which could not be transilluminated by light. U/S of the right testicle showed an 8.6x6.1x7.8 cm heterogenous vascular mass consistent with tumor. U/S of the left testicle showed a homogenous 2.1x5.2x3.6cm testicle with normal tissue. Follow-up CT scan revealed what was likely a right testicular neoplasm, with no other abnormalities and no retroperitoneal findings. The patient underwent right radical testicular orchiectomy, and pathological examination showed a 9cm pure seminomatous tumor with a TNM of PT2. Adjuvant radiation therapy was recommended but patient was lost to follow-up. One and a half years later, the patient showed up again to the ED with left flank pain. Physical exam of the remaining testicle was normal, but U/S showed a normal testicle with new microcalcifications that were not previously present. CT abdomen and pelvis showed a 12.5 x 10.2 x 12.5cm retroperitoneal mass with bHCG positivity, which also was not previously present.</p> <p>Discussion: The purpose of our discussion is to investigate whether or not new onset microlithiasis in high risk patients should be screened for, and if the new onset correlates with extragonadal spread of tumor. Upon initial investigation of the right testicular tumor, microlithiasis in the healthy contralateral testicle was not present, nor was there any retroperitoneal mass present. One year later, newly identified testicular microlithiasis and a newly discovered retroperitoneal mass were present. This observation can potentially guide us to more aggressive monitoring of extragonadal tumor spread based upon the presence or absence of newly formed microcalcifications in the healthy contralateral testicle. Current guidelines instruct high risk patients to go for an annual U/S of the testicle if microlithiasis has already been incidentally found. However, room may exist to routinely explore for the development of new microlithiasis in high risk patients rather than only monitoring these patients when existing incidental microcalcifications are found.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	jagroop khaira	Dr Girija Phadke, Dr Apurva, Dr Shweta kumar	Saint Peter's University Hospital (Nayan Kothari)	Acute Pulmonary Embolism with Right Heart Thrombus	<p>Introduction: Venous thrombo embolism (VTE) affect as many as 900,000 people each year in the United States. The number of deaths resulting from VTE are usually reported from 60,000 to 100,000 with 25% deaths reported to be sudden cardiac death. The prevalence of a right heart thrombus in the setting of an acute PE is 4% to 18%. Three types of Right heart thrombus have been described: 1) Type A are the serpiginous, highly mobile and associated with DVT/PE. They are also the ones associated with high short term mortality of 44%. 2) Type B are non mobile and are thought to be formed in heart itself due to underlying cardiac abnormalities. They are associated with low mortality of 4%. 3) Type C are very rare and have similar appearance to myxomas and are highly mobile.</p> <p>Case: 66 y/o male came to the emergency room with dyspnea for 4 days associated with right leg swelling three weeks ago. On arrival, his vitals were significant for tachycardia and tachypnea. His EKG showed incomplete RBBB, S1Q3T3 pattern and inverted T waves in V1-V3. His CT angiography showed large bilateral pulmonary emboli with right heart strain. He was started on heparin drip and admitted to Intensive care. He wasn't given TPA as he was hemodynamically stable. Venous duplex showed acute deep vein thrombosis involving the femoral, popliteal, gastrocnemius, posterior tibial and peroneal veins. His 2D echo showed findings of right heart strain and a large right atrial thrombus extending into the right ventricle. He was given TPA the following day and continued on heparin drip. He also got the retrievable IVC filter and his right heart thrombus was monitored with serial 2D echoes which resolved completely on 3rd Echo. HE was discharged home on Xarelto.</p> <p>Discussion: Our patient presented with massive PE with right heart thrombus type A. There are no guidelines for treatment of right heart thrombus in setting of PE but consensus is on anticoagulation and thrombolytics typically with type A clots because they have high chances of dislodgement. Without treatment mortality is 100% and survival is 70% with heparin and 62 % with thrombolytics. Given no contraindications in our patient, we used both.</p> <p>Conclusion: Right heart thrombus are diagnosed with 2D echo but is often underdiagnosed condition due to lack of sensitivity of 2D echo. There is no consensus on how to treat this condition. The use of thrombolytics in submassive PE is debated. The use can be verified with large thrombus at risk of dislodging and causing decompensation if there are no contraindications. Surgery embolectomy is another option.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Febin John		Saint Peter's University Hospital (Nayan Kothari)	Silver sign-A forgotten radiological marker of pneumoperitoneum	<p>Introduction: Pneumoperitoneum is a life-threatening condition where early diagnosis and prompt treatment can improve outcomes. Radiological diagnosis is confirmatory and surgical intervention is often required. Supine radiographs of the abdomen in pneumoperitoneum can demonstrate large oval radiolucency which is named as “football sign”. “Cupola sign” is the presence of air under the diaphragm which is a delayed radiological sign in pneumoperitoneum classically visualized on erect abdomen X-rays. “Rigler's sign”, also known as the double wall sign, is another radiologic sign in pneumoperitoneum seen on abdomen radiographs when air is present on both sides of the intestine. There are other subtle signs which are highly indicative of air in the peritoneal cavity- ligamentum teres sign and falciform ligament sign. We describe a case of pneumoperitoneum with falciform ligament sign.</p> <p>Case: An 83-year-old Caucasian male with alcoholic liver disease, decompensated cirrhosis [Child-Turcotte-Pugh Class C], chronic kidney disease-stage 2 and pancytopenia secondary to hypersplenism was admitted to hospital for evaluation of dizziness. His BP was 87/49mmHg, not orthostatic; regular pulse- 53/min and had ascites. Dizziness improved after discontinuing nadalol prescribed for portal hypertension. Therapeutic paracentesis was performed (about 7 liters) after intravenous albumin replacement. About 12 hours post paracentesis, he became hypotensive (BP-49/29 mmHg, pulse rate- 72/minute)with adistended andhyper-resonant abdomen. EKG wasnormal.X ray of the abdomen [Fig 1] revealed prominence of the falciform ligament (FL) [A]positive Rigler’s sign [B] with pneumatosis of the cecum and ascending colon[C]. He underwent subtotal colectomy with ileostomy for a perforated ascending colon. Comfort care measures were initiated as he deteriorated clinically despite all supportive measures.</p> <p>Discussion: FL, a remnant of the umbilical vein, is a double layer of peritoneum. It functions as a lymphatic channel from umbilicus to liver. FL is visualized in the presence of ascites¹ or pneumoperitoneum². Visualization of FL in the setting of pneumoperitoneum is named silver sign or falciform ligament sign. Silver sign is specific for gastroduodenal perforation. In about 7% of large bowel perforation. It is seen about 16-20 hours post trauma.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ayan De	Pitchumoni CS	Saint Peter's University Hospital (Nayan Kothari)	B12 Deficiency after Gastric Sleeve Surgery	<p>Introduction: Obesity and its complications are becoming a major worldwide problem. In the USA, 16.9% of children and adolescents are obese. In response, surgical options such as bariatric sleeve operations are now an important form of management to combat this growing epidemic. However, despite its growing success, there have been studies showing the importance of supplementation to prevent nutritional deficiencies, including B12.</p> <p>Case Report: A 24 year old male law student presented to the Emergency Department for surgical complications of his gastric sleeve surgery three months prior. During his admission he developed weakness and decreased sensation in the legs. Clinically he had decreased sensation from the soles distally to the knees proximally bilaterally and there was decreased power in the proximal muscles. His coordination was otherwise intact, and his reflexes were equal on both sides but diminished in the ankles. His gait was unsteady and showed some “stamping” features, but it was not broad based. His cranial nerves and upper limb examination were normal, as was the rest of his physical exam. The patient had an MRI of the spine which showed no abnormalities. A serum copper, folate and vitamins B1, 6 and 12 levels were ordered. The patient’s vitamin B12 level came back as low at 189 pg/ml. The patient was started on vitamin B12 in the meantime, which showed some improvement in his symptoms. He was then discharged with outpatient follow up.</p> <p>Discussion: Vitamin B12, or cobalamin, is a water soluble vitamin essential for neurological function as well as DNA synthesis and red blood cell production. It is not intrinsic to the human body and is therefore an essential requirement in our diets, from such foods like meat and fortified cereals. Once in the stomach, it binds to intrinsic factor in order to be absorbed in the terminal ileum. Any pathology to this pathway can cause a deficiency, whether it is the lack of intrinsic factor, inadequate intake or decreased absorption. Gastric sleeve surgery is an operation which involves creating a small stomach “sleeve” and then removing the rest of the stomach. This contributes to B12 deficiency in multiple ways including reduced mechanical digestion, acid secretion to release the vitamin from animal proteins and decreased intrinsic factor. There are also suggestions that very overweight patients prior to surgery can be vitamin B12 deficient, with a prevalence of up to 18%. These factors have laid the foundation for suggestions that post bariatric surgery; patients should be started on vitamin B12 supplements to reduce this complication.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Steve Bibu	Ngoc Ha, Lihua Yao, and Kimberly J. Dougherty	Saint Peter's University Hospital (Nayan Kothari)	Plasticity of spinal rhythm generating interneurons after spinal cord injury in a mouse model	<p>Introduction: Neural circuitry generating rhythm and pattern components of locomotion is located in the thoracolumbar spinal cord. Although this locomotor circuitry is not fully understood, most spinal cord injury (SCI) occurs above it so it may be harnessed for improving motor function after SCI. The SCI-induced loss of voluntary control is likely due to both a reduction in descending controls and spinal plasticity, involving alterations in network excitability and/or defects in neural processing. Locomotor rhythm generating interneurons (INs) are an obvious entry point for studying SCI and treatment. Elucidating the alterations to these functional circuits, following SCI, will enhance our understanding of the beneficial as well as the maladaptive plasticity that occurs in the spinal cord. The resultant insights will provide pathways for a more targeted and refined approach to treatment. The main objective of the present study is to identify targets for enhancing locomotor function, focusing on Shox2 rhythm generating INs. A subpopulation of Shox2 interneurons have been implicated in locomotor rhythm generation.</p> <p>Methods: Shox2Cre; Rosa26tdTomato or Shox2Cre; Chx10GFP; Rosa26tdTomato transgenic mice were used for all experiments. Complete spinal cord transections were performed at mid-thoracic level (T8-9) when mice were 5-8 weeks of age. Tissue collection for recordings took place either 3-6 weeks post-injury (mean=5±1) or 8-12 weeks post-injury (mean=10±1), when the mice were 2.5-4 months old. Spinal cords from spinal cord injured mice and uninjured controls were rapidly isolated in a glycerol-based artificial cerebrospinal fluid (ACSF). Transverse (250-350µm) sections were made from the lumbar spinal cord using a vibratome. Whole cell patch clamp recordings targeted Shox2 INs in spinal slices from uninjured and chronic SCI adult mice based on fluorescence expression. Various voltage clamp and current clamp step protocols were run to measure voltage-dependent currents and various firing properties. For afferent fiber stimulation experiments, slices were cut in the parasagittal plane preserving multiple dorsal root entry points in the lumbar region.</p> <p>Results: Shox2 INs displayed a wide repertoire of firing properties which can be linked to underlying voltage-gated currents. There were no major differences in excitability properties between control and SCI Shox2 INs. A fraction of Shox2 INs from both uninjured and SCI mice showed plateau properties, persistent inward currents, and displayed spontaneous bursting properties which were enhanced in the presence of serotonin. Additionally, a subset of Shox2 INs displayed monosynaptic excitatory postsynaptic potentials in response to low threshold afferent fiber stimulation.</p> <p>Conclusion: The intrinsic properties of adult Shox2 INs are relatively resilient to SCI-induced plasticity, leaving them as robust targets for post-injury therapies. Current focus is on how the fidelity of afferent-evoked responses in Shox2 INs changes post-injury.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	FNU Apurva		Saint Peter's University Hospital (Nayan Kothari)	Eythema Ab Igne – An old sign for a new disease	<p>Introduction – Erythema ab igne (EAI) also known as hot water bottle rash/toasted skin syndrome is characterized by localized area of erythema, blisters and/or hyperpigmentation due to application of hot pads or infrared radiation. Historically reported in temperate climates where open fires were used for heating purposes. There is a growing epidemic of Cannabinoid Hyperemesis Syndrome (CHS) characterized by cyclical episodes of uncontrollable vomiting and compulsive bathing in hot water.</p> <p>Case Description - A 22 year old male with chronic cannabinoid dependency was admitted to the hospital for abdominal pain and intractable vomiting for 1 week. The pain was acute onset, in the periumbilical area, severe in intensity and cramping in nature associated with no radiation. It was associated with intractable vomiting episodes and relieved by taking hot showers. He had been applying hot-pads on his abdomen to alleviate pain. He had similar episodes in the past that required frequent hospital admissions. On examination, the patient appeared dehydrated and cachectic. A reticular, reddish-brown, pruritic, non-tender, non-blanching, maculo-papular rash with a few superficial blisters and erosions was observed over the abdomen. This was consistent with Erythema Ab Igne (EAI) or thermal keratosis that occurs after repeated exposure to heat or infra-red radiation. All routine laboratory tests were normal.</p> <p>Discussion – EAI was historically reported in cold climates where open fires were used for heating purposes. Recently EAI is observed in patients with chronic pain exposed to heating pads and on thighs of the portable computer users (1,2). A similar case was reported where EAI was associated with compulsive bathing for nausea relief (3). EAI lesion is generally asymptomatic, but may be pruritic in some cases. CHS has been associated with hot showers which ameliorates the symptoms of pain and nausea. Application of hot-pads to ameliorate the abdominal pain in principle is an extension of the classic “hot shower” therapy. EAI is a poorly recognized physical sign of Cannabinoid dependency due to repeated hot-pad exposure.</p> <p>Conclusion – CHS has been well known to be associated with compulsive hot showers that ameliorate the symptoms of pain and nausea. EAI should be considered a marker of CHS in young adults.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Yuvraj Chowdhury		Saint Peter's University Hospital (Nayan Kothari)	Lymphocytic Interstitial Pneumonia: A rare cause of chest pain	<p>Introduction: Described in 1966 by Carrington and Liebow, Lymphoid Interstitial Pneumonia (LIP) is an uncommon form of interstitial lung disease, characterised by infiltration of the lung interstitium and alveoli by lymphocytes, plasma cell and other elements of the lympho-reticular system. LIP represents a benign polyclonal proliferation of mature B and T lymphocytes which can have either diffuse or focal involvement. Patients typically present with cough (71%) and/or dyspnea (61%) and rarely, with chest pain (6%). It presents with basilar reticular opacities or nodular densities progressing to ground glass opacification and consolidation with air bronchograms on chest x-ray, restrictive pattern on PFT's, and hypoxemia.</p> <p>Case Report: A 79-year-old lady with a past history of chronic non-productive cough, anxiety and hypertension presented with a new onset persistent left sided chest pain. The pain increased with inspiration and was localised. She had no complaints of subjective fever, wheezing, allergies, or heartburn. She denied any weakness, myalgia, joint pain, photosensitivity and eye or mouth dryness. She is a non-smoker and works at McDonalds.</p> <p>On examination she had point tenderness over the 7th left rib and bibasilar crackles on auscultation. Chest x-ray revealed diffuse interstitial markings. CT scan confirmed an increase in bibasilar interstitial fibrotic changes and prominent pulmonary arteries. PFT's revealed restrictive pulmonary dysfunction. Bronchoscopy did not reveal any malignant cells but the biopsy showed focal dense aggregates of mature lymphoid cells which included lymphocytes, plasma cells and histiocytes. Biopsy specimens were negative for acid-fast-bacilli or fungus.</p> <p>The ANA, anti-ssA/Ro and anti-ssb/La titres were normal. HIV viral titres were non-reactive. Serum protein electrophoresis showed an elevation in the M-Spike with a decrease in other classes of Immunoglobulins.</p> <p>Discussion: LIP remains a poorly understood entity with an unknown pathogenesis. On rare occasions, it can present with a sharp pleuritic chest pain. It is seen as the 2nd most common cause of lung involvement in children with AIDS after Pneumocystis, but remains uncommon in adults. EBV and the HIV virus itself have been implicated in having a causal role in AIDS with LIP. When seen in non-HIV patients, it has often been seen in association with Hyper-gammaglobulinemia and Sjogren's syndrome. In about 20% patients, the aetiology remains unclear.</p> <p>It is important to establish the benign nature of the disease. Mono-clonality, hilar or pleural involvement, lymphangitic or broncho-vascular invasion and infiltration of the bronchial wall representing a malignant transformation. Steroid therapy remains the treatment of choice with a scope for immunosuppressive drugs in case of failure of response to steroids or development of side effects.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Mrinali Shetty		Saint Peter's University Hospital (Nayan Kothari)	Neuroborreliosis: Triple Whammy in a Single Patient	<p>Introduction: Lyme's disease is often referred to as, "the great imitator" due to its ingenuity with producing a vast number of manifestations spanning almost every organ system. The neurological manifestations of Lyme's disease are usually seen in the second stage of the illness and on average within 4 weeks of the appearance of erythema chronicum migrans. It extends across the anatomy of the central and peripheral nervous systems and the common manifestations include: meningitis, cranial neuritis (the commonest being involvement of the facial nerve), peripheral neuritis and encephalitis. Though neurological involvement per se is a frequent accompaniment of the disease, it is usually limited to 1 or sometimes 2 abnormalities. Here, I report an atypical case of 3 neurological manifestations in the same patient.</p> <p>Case Report: A 70 year old Caucasian lady presented with chief complaint of sharp stabbing pain down her left arm for the past two weeks. The pain was associated with subjective fevers, an erythematous non itchy rash and intermittent headaches. On examination, the patient was tachycardic and neck stiffness was present, though Brudzinski and Kernig signs were negative. There was no focal neurological deficit and gait was intact. The next day, the patient reported food pocketing in the left side of her mouth and a hemifacial drooping of the left angle of the mouth with flattening of the nasolabial fold was observed on that side. Both eyes shut tightly and couldn't be opened with the examiner's fingers. There was no associated hearing loss or hyperacusis. Lumbar puncture revealed elevated proteins but normal glucose. Lyme serology of CSF fluid was negative but of blood was positive. MRI showed non-specific T2 white matter changes of the brain and multiple degenerative changes of the cervical spine, unlikely to be acute. The patient was put on a IV Ceftriaxone 2 gm once a day for 4 weeks and discharged home.</p> <p>Discussion: Neuroborreliosis warrants the use of intravenous antibiotics. The duration of neurological abnormalities depends on treatment, including time of initiation and antibiotic used. Agents preferred include Ceftriaxone, Cefotaxime or Penicillin G for a length of 10 to 28 days. The CDC estimates that there are about 329,000 Lyme disease occurrences in the U.S. annually. The numbers are staggering, but even more so the disability caused to a human life due to its sequelae as illustrated above, is immeasurable. This puts an impetus on us healthcare providers to better manage this disease. A step in that direction is by learning to diagnose it irrespective of the shroud it carries itself in. My objective was to highlight an interesting manifestation of this common disease thus helping to easier recognize its various forms.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Research	Abhishek kumar Mariswamy Arun Kumar	Dr Kanishk Agnihotri	Saint Peter's University Hospital (Nayan Kothari)	Seasonal trends in Sudden cardiac deaths - A Descriptive study	<p>Background: Sudden cardiac deaths (SCD) are important clinical end points to assess the outcomes of many cardiovascular conditions. Examination of seasonal variation of certain diseases has led to new insights into pathophysiologic triggers. We assessed the seasonal variations in SCD.</p> <p>Methods: The NIS database was used to estimate the annual number of admissions with primary diagnosis of SCD (427.5) from 2006-2012 using the International Classification of Diseases-9-CM diagnosis codes as the principal discharge diagnosis. The frequency of SCD for each month cumulative over 7 years was calculated. It was divided by number of days in that month to obtain the mean SCDs per day for each month. All calculations were carried out using the weighted estimates approximating nationwide population estimates. Results: A total of 953,871 patients had SCD 2006 to 2012. SCD were maximum in the winter months and lesser in the summer months. The mean number of SCD each day (averaged over 7 years) was least in July. The average number of SCD was highest in February; thereafter, dropped to a nadir in July and again rose thereafter to reach a peak in February.</p> <p>Conclusions: We identified an impressive pattern of seasonal variation in SCD. This pattern was seen in all subgroups analyzed as well as in different geographic areas. Documentation of such a pattern may foster investigation of new pathophysiologic determinants</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Sanketkumar Dalwadi		Saint Peter's University Hospital (Nayan Kothari)	Anthracycline Induced Cardiomyopathy	<p>Anthracyclines are anti-cancer drugs which are associated with cardiotoxicity. Among the serious clinical cardiac complications of anthracyclines are Arrhythmias, Myocardial necrosis causing a dilated cardiomyopathy and Vasooclusion or vasospasm causing angina or Myocardial Infarction.</p> <p>A 73 year old woman with past medical history of hypothyroidism, Hypertension and Chronic Obstructive Pulmonary Disease and presented with feeling of tires and weak. She was recently diagnosed with acute leukemia in Poland and had two units of blood transfusion and then she came to United States for further treatment. Patient didn't had any history of weight loss, cough, fever or chills. She was diagnosed Acute Myeloid Leukemia in poland but type of AML was not known. So patient had bone marrow biopsy and diagnosed with Acute Myeloblastic Leukemia with cell maturation (type M2). Patient received induction therapy with duanorubicin and Ara-C (Cytarabine). After completion of induction, patient had complete response as there was no evidence of leukemia after that. Patient was admitted after one month of induction treatment for the first cycle of the consolidation with high dose of Ara-C. Patient was admitted again after one month with complain of worsening of fatigue. Patient had white count elevated in 300s. During admission, patient had respiratory distress and transferred to Intensive Care Unit. Patient had persistent tachycardia (HR 110-130), tachypnea (RR 24-28) and hypoxia. On physical examination, patient had bilateral crackles on auscultation and S3 gallop on cardiac examination. Patient was kept on bipap support. 2D echo showed severe global left and right ventricular systolic dysfunction with LVEF of 35%. Patient had normal LV and RV function and size with EF of 65% 3 months back before receiving treatment of Acute myeloblastic Leukemia. Patient was continued with supportive treatment for congestive heart failure secondary to Dilated Cardiomyopathy as patient has poor prognosis in light of the rapidly progressing refractory acute myeloblastic leukemia.</p> <p>This case illustrated potential for severe dilated cardiomyopathy secondary to use of anthracycline. Myocardial damage secondary to anthracycline is dose dependent and irreversible. It can be reduced by using low dose or using liposomal form.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Vedavyas Gannamani	Sonmoon Mohapatra, Navya Kuchipudi	Saint Peter's University Hospital (Nayan Kothari)	Life Threatening Anemia and Extremely High Ferritin Levels in an Elderly Male with Rosai Dorfman Disease	<p>Introduction: Rosai Dorfman Disease (RDD) is a disorder of histiocytes characterized by their proliferation in various organs and phagocytosis of lymphocytes and neutrophils. RDD commonly presents with lymphadenopathy, usually cervical region, however extra nodal involvement is also known. Skin, soft tissues, upper respiratory tract and central nervous system are frequent extra nodal sites of involvement. It is mostly seen in children and young adults. Here we describe our experience of a case of RDD in elderly male with anemia, thrombocytopenia and liver mass.</p> <p>Case Description: A 61 years old male presented to hospital complaining of extreme fatigue of 3 weeks' duration. He did not complain of fever, night sweats, cough, spontaneous bleeding, jaundice or change in bowel habits. Associated with weakness, extreme loss of appetite was reported. A month prior to this admission patient was diagnosed with Rosai Dorfman disease using biopsies of inguinal, cervical lymph nodes and bone marrow. Home medications include prednisone 40mg daily. At initial presentation, vital signs included blood pressure 69/46mm hg, heart rate 109/min, temperature 97.4F. Examination showed lethargic male with shotty cervical, supraclavicular and inguinal lymphadenopathy and swelling of right lower extremity. Laboratory studies at admission included hemoglobin 5.1g/dl, MCV 75.7fl, WBC 25.6 x 103/cumm, neutrophils 98%, bands 1%, lymphocytes 0.0%, platelet count 53 x 103/cumm. Other serum tests included total bilirubin 0.6mg/dl, lactate dehydrogenase 252U/L, haptoglobin 295mg/dl, ferritin 11102ng/ml and reticulocyte count of 2.6%. A CT thorax, abdomen and pelvis showed multiple lesions in liver, lungs and spleen which were all new compared to imaging done a month ago. In addition, it showed extensive lymphadenopathy involving cervical, supraclavicular, mediastinal, retroperitoneal and inguinal regions. An ultrasound guided biopsy of a liver lesion showed mixed inflammatory infiltrate with histiocytes, neutrophils, and necrosis, compatible with liver involvement by Rosai-Dorfman disease. Immuno-histochemical stains revealed that the histiocytes were positive for s100 and cd68 and negative for cd1a. Patient was later transferred to cancer institute for escalation of care.</p> <p>Discussion: Rosai Dorfman disease is an uncommon cause of massive and/or extensive lymphadenopathy among other causes. RDD is not malignant, however the disease course is highly variable often taking months to years for complete resolution. Definitive diagnosis is by histology and immunohistochemical methods. Systemic spread involving multiple organs and immune dysregulation are indicative of poor prognosis. There are no established management guidelines and treatment approach is tailored case by case basis. Surgical debulking is effective when required and usually does not result in recurrence. Result with radiotherapy are variable. Steroids, among all therapies are the most commonly used for treatment of extensive RDD. RDD is a self-limiting disease, however other differential diagnoses causing massive and/or extensive lymphadenopathy like tuberculosis, acquired immunodeficiency and autoimmune lymphoproliferative syndrome need to be considered.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Abhishek Dave	Dr. Rajat Kumar	Saint Peter's University Hospital (Nayan Kothari)	Isolated 3rd cranial nerve palsy & peripheral radiculo-neuropathy: An uncommon manifestation of Early Lyme Neuroborreliosis.	<p>Introduction: Lyme disease, caused by the <i>Borrelia burgdorferi</i> bacterium, is the most common vector-borne illness in USA with 25,359 confirmed cases in 2014. Approximately 12% patients manifest neurological complications of Lyme disease, known as Lyme Neuroborreliosis (LNB) which may include, a painful meningo-radiculitis with or without paresis, cranial neuropathies and meningitis/encephalitis. Here, we are reporting a case of isolated Oculomotor nerve palsy as a rare manifestation of LNB.</p> <p>Case Description: A 72 year old male without any past medical history visits ED in last week of July with following chronological health events: One month ago patient had fever for 4 days that resolved, and 2 weeks post fever, he developed flu like symptoms of generalized malaise, muscle pain and joint pains without any improvement. Four weeks post fever, patient developed diplopia and drooping of right eyelid. His serum Lyme IgM titer changed to positive that was negative a month ago during febrile episode. Two days after diplopia, patient developed right upper extremity (UE) numbness and altered sensations progressing to bilateral UE weakness. On admission, neurological exam showed right upper eyelid drooping, medial and downward gaze palsy of right eye, right hand dysesthesia(feeling like holding tissue paper), decreased UE muscle strength(right 2/5, left 3/5) with diminished right brachial reflex. Rest of the neurological exam including other cranial nerves and head & neck imaging studies were normal. Patient was exposed to garden 1 month back, but did not remember tick bite or target sign rash. CSF analysis demonstrated: (a).Elevated proteins-251, (b).WBC-634(84% lymphocytes), (c).Xanthochromia, (d).Oligoclonal bands >5, all consistent with aseptic meningitis of LNB. Lyme PCR, Ehrlichia, WNV, HSV, AFB, VDRL were not detected in CSF. On 7th day of Ceftriaxone treatment, diplopia and drooping resolved completely and patient was able to abduct bilateral UE with limited extension. He was discharged with 21 days of 2gm/day Ceftriaxone to rehabilitation center for physical therapy.</p> <p>Discussion: As per European Journal of Neurology, isolated cranial nerve palsy other than facial nerve is rare in American LNB and there is only 1 case reported in 5 year old patient and 1 in 38 year old having 3rd nerve palsy concomitantly with other cranial nerve palsies. This patient demonstrated unique triad of manifestations of LNB- meningitis (lymphocytic), cranial neuritis (3rd nerve) and radiculoneuritis(UE weakness, dysesthesia). Patient fulfilled 2 of 3 criteria for definite LNB as per EFNS guidelines: (i)Neurological symptoms; (ii)CSF pleocytosis; (iii)Bb-specific antibodies produced intrathecally. Therefore, absence of Bb antibodies in CSF as in our case and negative serum PCR does not exclude diagnosis of LNB because spirochete rapidly distributes in connective tissue and may not be detected by PCR. Thus, enhanced level of suspicion is required for diagnosing LNB in Northeastern US especially in summer months.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Ashley Sarasan	Megan Mahon	Saint Peter's University Hospital (Nayan Kothari)	AN UNCOMMON CAUSE OF A COMMON CONDITION	<p>INTRODUCTION: May-Thurner Syndrome (MTS) was first recognized by Virchow who recognized that ileofemoral deep vein thrombosis was five times more likely to occur in the left leg than the right as a result of left common iliac vein compression against the fifth lumbar vertebrae. It has been shown that because of luminal narrowing, MTS patients can present with leg pain, DVT, venous claudication, and venous insufficiencies. In our clinical case, a 41-year-old female presented with recurrent left leg DVT with failure of warfarin and eliquis.</p> <p>CASE DESCRIPTION: A 41 year-old lady presented with left thigh pain for 12 hours. Prior to presentation, she experienced a sharp, stabbing pain on her left thigh and woke up the next morning with severe pain and swelling of the entire thigh up to the knee. She reported no history of chest pain, SOB or palpitations. This patient was previously admitted twice with similar symptoms and was found to have thrombophlebitis of left sided external iliac vein with thrombus visualized in left common femoral vein. Initially discharged on Apixiban, she returned with DVT involving the left common femoral vein and great saphenous vein. At this point was discharged on Coumadin and an IVC filter. Ten days following that admission she presented with the current presentation. She does not have any family history of bleeding disorders, anemia, cancer, or early deaths. She does not smoke and was not on Oral contraceptive pills. Her vitals on arrival were within normal limits. Left lower extremity exam revealed left thigh looking more swollen than the right with tenderness to palpation. Circumferential measurements of her thighs revealed right thigh of 50cm and left thigh of 58cm. Basic workup including a coagulation profile was normal. The INR on arrival was 4.20. Duplex of bilateral lower extremities revealed DVT with distal progression of disease in comparison to previous scans. She was switched to heparin drip and a repeat duplex venous revealed further distal progression involving deep and great saphenous veins. A magnetic resonance venography was then performed which showed proximal left common iliac vein collapsed secondary to compression by the right common iliac artery, consistent with May Thurner Syndrome. She was given 18 hours of EKOS thrombolysis of the left lower extremity before attempting stent placement.</p> <p>DISCUSSION: In conclusion, May-Thurner syndrome should be taken into consideration if there is persistent edema of the left leg, especially in young Women. 2-3% of all lower extremity DVTs are associated with MTS. Severe May–Thurner syndrome is unresponsive to anticoagulation therapy and require thrombolysis if there is a recent onset of thrombosis, followed by angioplasty and stenting of the iliac vein after confirming the diagnosis with a venogram or an intravascular ultrasound.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Yesenia Galan	Garcia, Pablo and Hersh, Joshua.	Saint Peter's University Hospital (Nayan Kothari)	The pharyngeal-cervical-brachial variant of Guillain-Barré syndrome: clinical diagnosis and exclusion of mimics.	<p>Introduction: Guillain-Barré syndrome (GBS) encompasses a spectrum of related neuropathies. While limb areflexia or hyporeflexia is characteristic, variants of GBS described in the literature are often overlapping or incomplete. There are, however, clinical features that distinguish these variants phenotypically. Recognition of variant core patterns permits anticipatory monitoring for complications and identifies therapeutic options early.</p> <p>Case Report: We report a case of right arm weakness which progressed to include weakness of the neck, involvement of the left arm, dysarthria, and dysphagia in a 52-year old male with past medical history of coronary artery disease recovering from an upper respiratory illness one week earlier. Physical examination revealed the diagnostic criteria of ascending oropharyngeal, neck, and arm weakness as compatible with the pharyngeal-cervical-brachial variant of variant of GBS (PCB). Biceps and triceps reflexes were reduced as well as lateral rotation and flexion of the neck. Ability to chew, blink, or swallow was lost. Nerve conduction velocities showed evidence of demyelination, conduction block, and absent F waves. Routine biochemical tests, cerebrospinal fluid results, and brain and vascular imaging were normal. Sputum cultures grew due to Haemophilus influenza and serology showed anti-GT1a IgG reactivity. Treatment with intravenous immunoglobulin was started. By day 4, the patient regained flexion at the elbow of the left upper extremity and right hand grasp function, yet by day 23 hyporeflexia and bulbar deficit were not significantly improved. After six sessions of plasmapheresis, the patient had only minimally recovered strength in both arms by the day of discharge.</p> <p>Discussion: This case illustrates the challenges of approaching the patient with symptoms of bulbar palsy and upper extremity involvement. PCB, previously described as rare, has since been typified in numerous case reports by electrophysiological investigation, serological relationship, and antecedent infection etiology. As immunological and laboratory assays may be inconclusive during the early stages of PCB, an initial evaluation based on careful observation leads to accurate diagnosis and avoids the delay of appropriate management due to misdiagnosis. Several PCB mimics can be excluded by careful observation of the pace and course of symptoms. Botulism is typically associated with symmetric weakness that is descending while fluctuation of ocular and bulbar symptoms is the clinical key that distinguishes myasthenia gravis. Scrutiny of tempo and clearance of signs and symptoms are the clue to differentiating stroke from PCB's monophasic disease course. Pattern recognition remains an important consideration when making a diagnosis based on clinical criteria. As a diagnosis of PCB can hinge on history and neurological physical exam alone, mimics can be excluded early on and unnecessary investigations will not delay management. We conclude that PCB should remain high on the differential when approaching the patient who presents with symptoms of ascending oropharyngeal, neck, and arm weakness.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	SUDARSAN kollimuttathuillam	Lewis Musoke	Trinitas Regional Medical Center	Paraneoplastic Polymyositis as an unusual presentation of colon carcinoma	<p>Colorectal cancer most often presents with a change in bowel habit, weight loss or with bleeding per rectum. Much less commonly, colorectal cancer may present as a paraneoplastic syndrome. Polymyositis is a complement-mediated idiopathic inflammatory myopathy manifested by proximal muscle weakness. Polymyositis may be part of a paraneoplastic syndrome associated with an underlying malignancy, but it has been suggested that tumors of the large bowel are rarely complicated by myositis. Here we present a case of colon carcinoma with muscle weakness being the presenting feature.</p> <p>A 51-year-old Puerto-Rican female with a past medical history of COPD presented with a 2-week history of pain of bilateral shoulders and thighs associated with weakness. Review of systems were positive for loss of appetite, constipation, fatigue and low grade fever 100.2 which resolved with Advil. She denied any loss of weight, melena, nausea or vomiting. She had a 17 pack year smoking history but quit 13 years ago, and denied alcohol or recreational drug use. Family history was significant for breast cancer in two maternal aunts and lung cancer in a son diagnosed at age 29. Physical examination was significant for obese habitus and significant proximal muscle tenderness associated with tenderness. Labs showed mild anemia with hemoglobin of 11.9 with MCV of 80.9 and normal chemistry. CPK, myoglobin, LDH and aldolase levels were elevated indicating myositis. TSH was within normal limits, however thyroid peroxidase antibody was positive. ANA, Anti-Jo antibody, SCL-70 antibody, anti-RNP antibodies, SS-A, SS-B were all negative. MRI of hip showed muscle edema of spinal, pelvic and thigh muscles. The patient was started on treatment with steroids with significant symptomatic improvement and lowering of enzymes. CT scan of chest to rule out malignancy showed severe bullous disease. CT abdomen/pelvis showed thickening of wall of descending colon associated with enlarged pericolic lymph nodes. Enlarged para aortic lymph nodes and multiple hepatic hypodensities were concerning for primary colonic carcinoma with metastasis. MRI done later confirmed the malignant nature of liver masses which were 3 in number and were biopsied. Colonoscopy was performed which showed poorly differentiated carcinoma of colon with metastasis showing mutations in P53 and KRAS, confirmed also by liver biopsy. Muscle biopsy showed scattered fascicular infiltration with inflammatory cells confirming polymyositis. The patient was discharged home on tapering dose of steroid and was started on neoadjuvant therapy with Oxaliplatin/5-FU with leucovorin and Bevacizumab with plan for partial hepatectomy and colectomy after chemotherapy.</p> <p>This case shows the importance of considering neoplasm as a differential for patients presenting with symptoms of myopathy, as although rare, it can be the first symptom at presentation. Although no guidelines exist, numerous case reports suggest a diagnosis of polymyositis warrants a search for occult malignancy.</p>

Category	Name	Additional Authors	Program	Abstract Title	Abstract
Clinical Vignette	Lewis Musoke	Thamer Sartawi MD, Sudarsan Kollimuttathuillam MD, Clark Sherer MD.	Trinitas Regional Medical Center	A Rare Instance of Pneumococcal Bacteremia Involving a Pacemaker Lead	<p>Pacemaker lead infection is an often indolent but serious complication of cardiac pacing. The absence of local signs of infection over the pulse generator pocket is common particularly in late presentation, making the diagnosis all the more difficult. Streptococcus pneumoniae is an unusual cause of pacemaker lead vegetation. Two cases have described disseminated Streptococcus pneumoniae involving a left ventricular assist device and a pocket generator infection. This case emphasizes the significance of considering atypical organisms causing pacemaker lead infections, such as that in a patient with persistent pneumococcal bacteremia.</p> <p>An 87 yr old Cuban male, with a past medical history of sick sinus syndrome and AICD placement in 2012, presented with a 3-day history of productive cough, fever and dyspnea. Preliminary investigations revealed a fever of 101.2 F and leukocytosis of 12,700/μL cells predominantly neutrophils with no bands. There were no signs of localized infection on the pacemaker pocket. Although a chest radiograph showed no clear infiltrate, the suspicion for pneumonia was significant enough to initiate empiric IV antibiotic therapy with ceftriaxone 1g and azithromycin 500mg.</p> <p>Two sets of blood cultures drawn on admission yielded Streptococcus pneumoniae sensitive to ceftriaxone (MIC, of 0μg/ml) after 48hrs of incubation. Repeat blood cultures drawn on the same day yielded the same organism. Ceftriaxone, was subsequently altered to 2g q24hrs and the azithromycin was discontinued. A CT of the chest without contrast ruled out pneumonia and a trans-thoracic echocardiogram showed no vegetation. A Trans-esophageal echocardiogram was performed revealing right atrial lead vegetation. Within 1 week, the patient was transferred to a specialized facility where he underwent successful laser lead extraction. Final cultures for the extracted leads revealed no growth. Postoperatively, the patient completed 3 weeks of IV antibiotic therapy at a subacute rehab. The incidence of permanent pacemaker related infections caused by gram positive bacilli like Streptococcus pneumoniae are low at 4%. Pacemaker lead infections caused by streptococcus pneumoniae have been reported. One such case involved pneumococcal bacteremia triggered by periodontitis. In our reported case, the patient had recurrent persistent pneumococcal bacteremia with a clinical presentation of pneumonia. A retrospective review of the medical records was inconclusive as to whether our patient received pneumococcal vaccination. As further evaluation revealed no clear source of infection, it necessitated evaluation for infective endocarditis and possible lead infection using trans-esophageal echocardiography. The initiation of early treatment with IV antibiotics and complete pacemaker lead extraction is important in the prevention of catastrophic outcomes. The absence of pneumococcal vaccination may well have predisposed our patient to invasive pneumococcal infections. In our case a high index of clinical suspicion, timely diagnosis and intervention were crucial in the management of a cardiac device related infection, particularly in the absence of local findings.</p>

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Clinical Vignette	Lewis Musoke	Alberto Gavilanes MD, William Farrer MD,	Trinitas Regional Medical Center	Raoultella planticola tracheobronchitis in the setting of a large duodenal bleeding ulcer	<p>Raoultella planticola is a gram-negative, non-motile, aerobic bacillus that is primarily considered as environmental bacteria. Although rare, R. planticola has mainly been associated with invasive human infections in immunocompromised states. As of 2016, the clinical significance of its isolation from human specimens is not fully elucidated. To our knowledge, our case constitutes the twentieth report in the world of R. planticola human infection; yet the first report in the setting of a bleeding duodenal ulcer. A 73 year-old Chilean male with a medical history of obstructive coronary artery disease, end-stage renal disease on renal replacement therapy presented after a 1 day history of hematochezia. Initial physical examination was only significant for bright red blood per rectum. Preliminary investigations revealed a hemoglobin of 7.9 g/dL, which represented a 3 g drop from the patient's baseline hemoglobin. An emergent esophagoduodenoscopy revealed a 2 cm duodenal ulcer which was clipped and injected with epinephrine. The patient was continued on pantoprazole by intravenous infusion for 72 hours before starting intravenous bolus of pantoprazole. His hospital course was complicated by Non-ST elevation myocardial infarction and ventilator-dependent respiratory failure. These were precipitated by a persistent bradyarrhythmia leading to asystole that necessitated trans venous pacing. A triple lumen femoral catheter was placed for intravenous access.</p> <p>On day 3 of admission purulent endotracheal secretions with coarse breath sounds were noted with concomitant high grade fevers up to 103.2 F and significant leukocytosis of 22,800/μL cells. Chest radiograph revealed no clear infiltrates or opacities. Endotracheal aspirate cultures, however, grew R. planticola and R. ornitholytica sensitive to ciprofloxacin (MIC \leq 0.25μg/ml) and vancomycin (MIC 2μg/ml). Therefore, a transient course of ciprofloxacin and vancomycin before starting Meropenem, was given as coverage for a suspected respiratory tract infection in the form of tracheobronchitis. Repeat comparative blood cultures obtained from central and peripheral veins were negative for catheter associated blood stream infection. Repeat endotracheal culture, on day 7, was negative. Both femoral catheter and temporary trans venous pacemaker were also removed. Despite these interventions the patient succumbed to septic shock refractory to vasopressor and antibiotic therapies. R. planticola isolated from an endotracheal secretion may suggest that the patient developed sepsis related to R. planticola tracheobronchitis as one of the complications leading to his demise. Our patient possessed many risk factors previously associated to Raoultella human infection including his advanced renal disease and possibly an enhanced alteration of the gut flora, with subsequent bacterial translocation induced by proton pump inhibitors. Understanding the circumstances and the medium where Raoultella is pathogenic to humans becomes important to characterize its associated disease process. We describe the first association of R. planticola tracheobronchitis with a duodenal bleeding ulcer treated with an endoscopic approach and proton pump inhibitors.</p>