TEAM
7
CU5
AN UNUSUAL CASE OF DIFFUSE SEROSITIS

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Granulomatosis with polyangiitis (GPA) is a necrotizing small and medium vessel vasculitis that classically presents with upper and lower airway involvement and glomerulonephritis. Pulmonary nodules and masses, ground glass opacities, and airway involvement make up most of the thoracic findings. Pleural involvement is less common, and cardiac involvement is rare.¹

Mr. CH is a 60-year-old man who presented to an outside hospital with dyspnea, cough with hemoptysis, and fatigue. He was found to have a large left exudative pleural effusion with negative cytology. His past medical history was notable for prior membranous glomerulonephritis (MGN). Over 3 months he developed acute renal failure requiring hemodialysis, loculated bilateral pleural effusions requiring chest tubes, and a hemorrhagic pericardial effusion with tamponade requiring pericardiocentesis. Evaluation was notable for a positive C-ANCA (PR3), low C3, CRP 10 mg/dL (reference range 0-1 mg/dL), UA with microscopic hematuria, and a positive IgA anti-B2 glycoprotein. A renal biopsy showed ischemia and his known MGN, but no features consistent with GPA. A bronchoscopy was normal. A CTA of the abdomen showed splenic and renal infarcts and renal artery stenosis. He was treated with antibiotics, 1g methylprednisolone for 3 days with maintenance 60 mg prednisone, and one dose of rituximab prior to transfer to Johns Hopkins Hospital (JHH).

His course at JHH was complicated by recurrent bilateral pleural and pericardial effusions requiring a pericardial window and lung decortication. Cyclophosphamide was started while prednisone was tapered to 30 mg daily. An extensive workup was notable for continued PR3 positivity, + RF, and high IgG anti-B2-glycoprotein. Abdominal angiography showed numerous arteries with tapering and beaded appearance, including bilateral renal arteries, consistent with medium or small vessel vasculitis. Pathology of his pericardium showed chronic inflammation and his left lower lobe biopsy and pleural rind were notable for obliterated vessels and vascular necrosis highly suggestive of ANCA-related vasculitis. Importantly, pathology was negative for infection or malignancy. The patient was stabilized and discharged with the diagnosis of ANCA vasculitis manifesting as a pleural-pericardial syndrome.

Recognition of the pleiotropic presentations of GPA is important for early diagnosis and treatment to prevent complications. 10-20% have pleural effusions, but pleural thickening is rare and more associated with cancer. Approximately 6% of cases have pericarditis, and tamponade is case reportable.¹² This is a rare presentation of an uncommon illness and thus required an exhaustive workup to rule out more common etiologies of serositis including infection and malignancy.

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Ace-Inhibitor Induced Intestinal Angioedema Presenting with Spontaneous Bacterial Peritonitis (SBP)

Introduction
ACE-i are commonly used medications for the treatment of congestive heart failure and hypertension. Unfortunately, they are also the leading cause of drug-induced angioedema (AE). Angioedema (AE) can present as peripheral or visceral AE

Case description
A 32-year-old Caucasian female with newly diagnosed hypertension started on Lisinopril/HCTZ two weeks prior to presentation, presented to the emergency department with 1 week history of abdominal pain, nausea and vomiting. Physical exam on admission were unremarkable except tachycardia (120 beats per minute), tenderness on epigastric and right upper quadrant area and positive shifting dullness. Laboratories data revealed leukocytosis (WBC 17000), AST/ALT/ALP, PT/PTT/INR, lipase, amylase, urinalysis were normal. Beta HCG was also negative. CT abdomen showed marked ascites with non-specific thickening of jejunum area. Hepatic venous duplex US was also negative for thrombosis of portal hepatic vein. A paracentesis revealed low SAAG (<1.1) with WBC 1064, 85% neutrophil. Patient was treated as sepsis secondary to SBP and blood pressure medication was held during her stay. Her condition improved and discharged home with 7 days ciprofloxacin with instruction to resume lisinopril/HCTZ. One week later she came back with abdominal pain, nausea, vomiting and repeat CT abdomen showed diminished ascites but persistent thickening of loops of jejunum area. Patient underwent diagnostic laparascopy with jejunum biopsy which later on showed submucosal edema without any evidence of malignancy, IBD, granuloma, vasculitis. Patient symptoms resolved completely and she had no recurrence of abdominal pain since the lisinopril was stopped.

Discussion/Conclusion
Isolated intestinal angioedema secondary to ACE inhibitors is a rare side effect of ACEi. Patients typically present with abdominal pain, nausea, vomiting and diarrhea within days to weeks of initiation of the ACEI and generally improve within 24 - 48 hours after cessation of the medication. Supportive care and cessation of ACE-inhibitor usage are the cornerstones of treatment. This is the first case to our knowledge ACEi-induced visceral angioedema with SBP. Awareness is important for early suspicion as it will help in avoiding delays in diagnosis, unnecessary invasive testing, and considerable morbidity.
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A CURE FOR DIABETES: HEPATITIS C TREATMENT
Justin Berk, MD and TingJia Lorigiano, MD

Introduction
The relationship between Hepatitis C Virus (HCV) and Type 2 Diabetes Mellitus (T2DM) has been well documented in the literature. Among patients with HCV, there is a higher incidence of T2DM and HCV viremia is associated with insulin resistance. Sparse case reports demonstrate potential improvement in insulin resistance with direct antiviral HCV treatment.

Clinical Case
We discuss a case of a 38 year old woman with a history of insulin-dependent T2DM that was effectively cured of diabetes after treatment of HCV infection with 3 months of elbasvir and grazoprevir. Her HgA1c prior to the treatment was 13.2 mg/dl (12/2017). One month after HCV treatment, her random glucose was 126 mg/dL (not taking any treatment) and she remained euglycemic on blood sugars and had an A1c of 5.7 at next check in early 2019). This was in the context of weight gain, no changes to diet or exercise, non-adherence to insulin and being lost to follow-up.

Discussion
Improvements in glycemic control have been previously demonstrated in previous case reports though there is minimal discussion of complete resolution of insulin-dependent diabetes mellitus from direct antiviral treatment. Mechanisms for this proposed treatment pathway includes HCV-protein related upregulation of insulin receptor degradation and increased liver inflammation via TNF-α causing dysregulation of glycemic pathways. Our case demonstrates a role for HCV treatment in better control of diabetes and could argue for early treatment of HCV to potentially avoid diabetes-related health complications.
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### WHEN LUPUS HUGS YOUR MUSCLES
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Early detection and aggressive treatment in SLE-nephritis overlap syndrome is required to ensure clinical resolution of symptoms. SLE associated with myositis occurs in 2.6% of cases. In the literature, and because of the diversity of clinical symptoms, reliable data concerning the prevalence of overlap syndromes are not available. Below, we describe a case of myositis as a first presentation of systemic lupus erythematosus that required pulse steroids, IVIG and immunosuppressive therapy.

A 57-year-old man presented with severe bilateral proximal upper and lower extremity weakness for a few weeks associated with dark tea-colored urine. His physical exam revealed markedly decreased strength in bilateral upper and lower extremities, swollen bilateral upper extremities left more than right, and bilateral tender thighs with no evidence of cellulitis or compartment syndrome. Lab workup showed K: 3.8, proteinuria >500 with gross hematuria, CPK 24000, CK-MB 1405, aldolase 38. No appreciable decrease was observed in CPK levels despite aggressive IV fluids and adequate urine output. Therefore, an extensive work up was initiated which revealed positive ANA, anti ds-DNA, anti-cardiolipin antibody, and negative anti Jo and anti-smith antibodies. EMG and muscle biopsy of the right rectus femoris showed inflammatory myopathy and polymyositis.

Our patient was diagnosed with polymyositis based on Peter and Bohan Criteria, and SLE after an immunological work up was positive for some of the lupus serologies. Treatment was initiated with pulse dose steroids, IVIG, and plasmapheresis, and the patient was then discharged on Azathioprine. His weakness considerably improved with immunosuppressive therapy along with a simultaneous decrease in CPK to 309 over the span of 2 weeks.

This case illustrates the need for broad differentials, thorough history taking, and recognition of myositis as a possible initial presentation of SLE. It also demonstrates the importance of aggressive therapy with IVIG and immunosuppression to ensure rapid resolution and improvement of symptoms.

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SUDDEN UNEXPECTED TORSADES IN EPILEPSY
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Sudden unexpected death in epilepsy (SUDEP) is the leading cause of mortality in people with uncontrolled seizures. Though pathophysiology of SUDEP remains unclear, the literature suggests that cardiac arrhythmia is a leading factor in the process. We report an unusual case of sudden-onset Torsades de Pointes (TdP) arrhythmia followed by ventricular fibrillation in a middle-aged woman with recurrent generalized tonic-clonic seizure (GTCS).

A 55-year-old woman with a known 3-month history of seizures was found unconscious with snoring and urinary incontinence at work. Following admission and workup, she experienced a 45-second long GTCS with incontinence while walking to the restroom. Lorazepam and levetiracetam were administered, but within 3 hours there was an episode of unresponsiveness and momentary cessation of breathing. Later that night, she exhibited sudden episodes of nonsustained ventricular tachycardia and received 2 gm of intravenous magnesium sulfate. Despite these efforts, she exhibited polymorphic sustained ventricular tachycardia followed by ventricular fibrillation with loss of consciousness and decreased extremity perfusion. Ultimately, the patient survived and was found to have moderate stenosis within the proximal and mid left anterior descending artery for which she received coronary stenting.

Etiologies of TdP and acquired QT prolongation include an array of medications, congenital abnormalities, genetic polymorphisms, underlying heart disease, old age, and female sex. Our patient suffered from recurrent seizures before arrhythmia, proved negative when worked up for TdP risk factors, and had no prior or family history of heart disease. However, her epilepsy may have contributed to the onset of arrhythmia including TdP. Generalized tonic-clonic seizures should be worked up and closely monitored for cardiac arrhythmias and their often-fatal consequences.

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CATHETER-RELATED RIGHT ATRIUM THROMBUS IN A PATIENT WITH HODGKIN LYMPHOMA.
Amit Rout MD, Abigail Chan MD, Amentshaw Singh MD

Introduction:
Central venous catheters (CVC) can be complicated with venous thrombosis in 40% of cancer patients. Although catheter-related right atrial thrombosis (CRAT) is uncommon, its presence carries significant mortality. The optimal treatment regimen has not been established. We present the unfortunate case of CRAT in a Hodgkin lymphoma (HL) patient.

Case:
A 56-year-old man with stage IV Hodgkin lymphoma presented to hospital for management of an incidental right atrial (RA) thrombus. A year prior, he was diagnosed with metastatic nodular sclerosing HL. A power-injectable port was inserted for chemotherapy administration. He underwent multiple cycles of chemotherapy with poor clinical response. He was being evaluated for hematopoietic stem cell transplant when the pretransplant transthoracic echocardiogram (TTE) revealed a 2.2 x 1.8 cm RA mass and questionable inferior vena cava (IVC) thrombosis. He was sent to the emergency department, initially he was asymptomatic and hemodynamically stable. Intravenous unfractionated heparin was started. Laboratory data showed a slight leukocytosis and anemia. Imaging confirmed the RA mass. Transesophageal echocardiogram (TEE) and cardiac MRI were also done, giving the tentative diagnosis of CRAT. He was planned for possible thrombectomy, however, during the course, he developed severe thrombocytopenia, fever of unknown origin, shock, and later died from these complications.

Discussion:
CRAT remains largely underdiagnosed due to its lack of symptoms. Potential complications include pulmonary embolism, septic emboli, cardiac arrhythmias and dysfunction. Diagnosis can be made using TTE, cardiac MRI, and TEE, with TEE being the most specific. Once diagnosed, anticoagulation remains the mainstay of treatment, while thrombolysis or surgical thrombectomy is reserved for complicated cases or after the failure of anticoagulation. As the use of CVC continues to be widely used, prevention of thrombus formation by avoiding catheter tip in the RA, using routine TTE in chemotherapy patients, shorter duration of placement, and continuously reassessing the need of CVC becomes a necessity.
Bilateral occipital Lobe Infarct Neglect Deficit (BLIND) Syndrome

Introduction: Cortical blindness is loss of vision due to dysfunction of the visual cortices. Patients who suffer from cortical blindness, usually due to bilateral ischemic infarcts of the occipital lobes, tend to lack insight of their visual deficit. This phenomenon of unawareness, or denial of vision loss, is called visual anosognosia. Further, patients often claim to be able to see and will confabulate visual perceptions despite their lack of sight. Historically, visual anosognosia in cortical blindness has carried the eponym, Anton syndrome, named after the Austrian neurologist and psychiatrist, Gabriel Anton (1858-1933), who first described the syndrome in 1899.

Case presentation: A 76-year-old man was traveling to visit his daughter during the holidays when he suddenly experienced blurred vision while driving his car. Subsequently, he stayed in his car for three days because his visual deficit prevented him from using his phone to call for assistance. The police eventually found him, and brought him to medical attention. The patient reported several weeks of increasingly frequent “lightning-like” bright flashes of green and yellow in his right eye, but denied the presence of floaters, curtain loss of vision, or diplopia. He reported that his last eye examination was one year prior, at which time his visual acuity was 20/30 bilaterally. Physical examination was significant for irregular rhythm and tachycardia. EKG revealed atrial flutter with 2 to 1 block and pulse of 150 beats per minute. Ophthalmologic exam demonstrated significantly diminished visual acuity - less than 20/200 bilaterally with 360-degree constriction of confrontational visual fields. Pupillary reflexes and ocular movements remained intact bilaterally. Dilated fundus exam revealed no evidence of retinal, macular, or choroidal pathology to explain the visual loss. Therefore, the patient was deemed cortically blind, prompting further evaluation for suspected stroke. CT brain without contrast revealed bilateral subacute parieto-occipital infarcts with local sulcal effacement. MRI of the brain without contrast showed acute to subacute bilateral PCA territory infarcts and additional small subacute infarcts in the bilateral cerebellar hemispheres. MRA of the head/neck without contrast showed no evidence of intracranial occlusion, stenosis, or aneurysm.

Conclusion: Here, we propose using the term, Bilateral occipital Lobe Infarct Neglect Deficit (BLIND) syndrome, to replace Anton syndrome for patients who develop cortical blindness with visual anosognosia associated with bilateral occipital lobe ischemic infarcts. In addition, consider BLIND syndrome in patients who present with visual deficits and denial of blindness in the setting of cardioembolism due to new onset atrial arrhythmia.

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**MULTIPLE BRAIN ABSCESSES IN THE SETTING OF NEWLY DIAGNOSED LUNG ADENOCARCINOMA.** Garcia, GE, MD; Rittaccio, G, MD. The University of Maryland Medical Center, Baltimore, MD.

In patients with lung cancer the incidence of brain metastases over the course of their disease can be up to 50-60% depending on tumor mutations. However, it is also important to also consider lesser-known diagnoses such as brain abscesses in the appropriate clinical context.

A 63 year-old man with a recent diagnosis of stage IIIC lung adenocarcinoma presented for his first chemotherapy treatment with a 2-day history of fatigue and short term memory loss. Fewer than 24 hours after admission, he became acutely altered, agitated, and non-verbal, but with an otherwise non-focal neurological exam. Magnetic resonance imaging (MRI) brain demonstrated multiple ring-enhancing lesions in the cortex, basal ganglia, and cerebellum. These findings were concerning for metastases vs. abscesses, though the former was prioritized given the patient’s cancer. Of note, an MRI brain 1-month prior did not show any abnormalities. He was started on broad-spectrum antibiotics due to concern for possible abscess. Infectious work-up, including blood and urine cultures, was negative. His clinical status gradually worsened and repeat neuroimaging demonstrated increased hydrocephalus with new midline shift. An external ventricular drain (EVD) was subsequently placed, with the initial cerebrospinal fluid (CSF) sample notable for elevated protein of 203 mg/dL. Within 24 hours, the EVD drained frank pus with repeat CSF studies showing WBC 34,500 K/mcL, lactate 18 mg/dL, protein 569 mg/dL, and glucose <20 mg/dL. CSF 16S ribosomal molecular testing eventually resulted in positive organism identification of *Streptococcus intermedius*. Subsequent transthoracic echocardiogram was negative for valvular vegetations. Antibiotics were narrowed and the patient’s clinical status improved markedly; he was discharged to a rehabilitation unit.

This case highlights the importance of maintaining a broad differential that prioritizes both clinical presentation and data on statistical likelihood of disease.

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SEPTIC METASTATIC ENDOPTHALMITIS DUE TO PYOGENIC LIVER ABSCESS

JP AbiMansour, MD, Jacquelyn Zimmerman MD, PhD, The Johns Hopkins Hospital, Baltimore, MD.

Pyogenic liver abscess is typically due to polymicrobial infection of hepatic parenchyma, however monomicrobial Klebsiella pneumoniae liver abscess (KLA) is a rare cause with increasing global incidence. We present the case of a 60-year-old Filipino female with poorly controlled type 2 diabetes mellitus who initially presented to an emergency room with abdominal pain. Computed tomography (CT) of the abdomen without intravenous contrast showed no acute luminal process but did note vague 1.4 cm hypodensity in the right hepatic lobe that was difficult to assess given lack of contrast. Her symptoms were felt to be secondary to a urinary tract infection and she was discharged on nitrofurantoin. Three days later, she returned to the ED with acute vision loss, pain and erythema of the right eye and a diagnosis of anterior endophthalmitis was made.

Aqueous and vitreous humor cultures grew pansensitive Klebsiella pneumoniae. CT scan of the abdomen with intravenous contrast was performed to evaluate for endogenous sources which showed a 6.7cm thick-walled complex hepatic lesion consistent with abscess. She was initiated on broad spectrum antibiotics and a drain was placed under CT guidance. Drain cultures also grew pansensitive Klebsiella. Blood cultures remained negative for the duration of her hospitalization. Ocular infection progressed despite aggressive intravitreal injections, washouts and vitrectomy. The patient ultimately required complete evisceration of the right eye. She was discharged on a prolonged course of aztreonam due to adverse reactions to other antibiotics administered during her hospitalization. She completed a prolonged 12-week course with complete resolution of the abscess.

As seen in this patient, metastatic eye infection is often catastrophic and has been reported in up to 7.8% of KLA cases. Other sites of involvement include the central nervous system, lungs, and muscle tissue. Diabetic patients are thought to be especially susceptible due to impaired phagocytosis. Patients should also undergo screening colonoscopy after resolution of acute infection given an association with colorectal cancer. Awareness of KLA syndrome and the aforementioned risk factors should prompt providers to seek pathogen-specific diagnoses and closely monitor for potential multi-organ complications.

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Severe Shock as the Initial Presentation of Inflammatory Bowel Disease Unclassified

Background: Age dependent immune modulation plays a major role in the late uncovering of inflammatory bowel disease (IBD). 1/3 of IBD occurs in elderly, yet the diagnosis is delayed by 6 years on average when compared to younger peers who have a 2-year delay. Subtle and unconventional symptoms plus comorbidities play a major role in this.

Case: A 61-year-old veterinarian female with a long-standing history of constipation presented with acute sharp abdominal pain associated with bloating, nausea and non-bilious vomiting. Vital signs were within the normal range. Physical examination showed a slender female in mild distress, with a soft but distended abdomen that was tender to palpation mainly in the left lower quadrant. Pertinent laboratory tests showed bands 24%, WBC 7x10^9/L, platelets 471x10^9/L, lactate 4.6mmol/L, and alkaline phosphatase 216 IU/L. A computed tomography (CT) of the abdomen showed segmental narrowing of the body of the stomach and stool impaction. She was admitted for observation for possible early sepsis; over the course of the next 6 hours she developed severe diarrhea and shock and required admission to the ICU where she was treated empirically for a gastrointestinal infection. She was worked up extensively, but the only significant finding was a positive serum Salmonella IgM. She improved with medical management and eventually antibiotics were deescalated, she completed a 10-day course; however, diarrhea persisted and eventually became bloody. Given her history of chronic constipation the possibility of Celiac disease was entertained but work up was negative. Sigmoidoscopy with biopsy was suggestive of ischemic colitis. The hematocrit resolved by itself and apart from the ongoing diarrhea she was doing well; she was discharged home. At follow up 2 months later she was back to her baseline constipation, her calprotectin level was found to be high and upper and lower endoscopy with biopsies showed minimal active chronic colitis concerning for idiopathic IBD.

Discussion: IBD was originally subclassified into ulcerative colitis (UC) and Crohn disease (CD), however, these two entities are at times indistinguishable and may mimic each other. This led to the introduction of the IBD unclassified (IBD-U) concept which now accounts for 5.7% of IBD. It is usually defined as colitis without an identifiable cause with clinical, endoscopic and pathological features of UC and CD. Miscategorization is very common in the elderly and a careful work up to rule out other causes is necessary. Over the course of 8 years only 20% of patients will still carry this diagnosis, the remaining will have been further subclassified with a ratio of 2:1 in favor of UC.

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TUBERCULOUS MENINGITIS
Adam J. Brownstein, MD and David Furfaro, MD
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Tuberculous meningitis, which represents 1% of all cases of tuberculosis (TB), has the potential to be a devastating complication of extra pulmonary TB with a mortality rate of 15-40%. A 34 year old woman from El Salvador with a past medical history notable for peripartum cardiomyopathy with left ventricular ejection fraction of 45%, type 2 diabetes, and stage IV chronic kidney disease due to focal segmental glomerulosclerosis, presented to the hospital with 1 week of subjective fevers, night sweats, nausea, vomiting, dry cough, and a headache. Upon presentation, her vital signs were within normal limits and initial neurologic exam was non-focal but concerning for meningitis given neck stiffness, headache, and photophobia. She underwent a lumbar puncture (LP), which showed cerebral spinal fluid (CSF) white blood cell (WBC) count of 163/mm³ (41% neutrophils, 59% lymphocytes), protein of 76.1 mg/dL (15-45 mg/dL), and glucose 46 mg/dL (50-75 mg/dL) with negative gram stain and fungal culture. She was empirically started on broad spectrum antibiotics. Computed tomography of the chest revealed innumerable nodules predominately involving the left upper and posterior right middle lobes concerning for an atypical infection. Repeat LP two days later revealed an opening pressure of 36 cm H2O and worsening pleocytosis (WBC 256/mm³). Over the course of several days, she gradually became more lethargic and was empirically started on TB therapy with RIPE (rifampin, isoniazid, pyrazinamide, ethambutol) and prednisone. The following day she became increasingly somnolent and required intubation for airway protection. Magnetic resonance imaging had no evidence of hydrocephalus. She underwent urgent lumbar drain placement. Mycobacterial cultures from her CSF and bronchoalveolar lavage (BAL) were ultimately negative but mycobacterial TB DNA was detected in both, confirming the diagnosis of active TB. The patient had a significant improvement in her clinical condition and returned to her baseline mental status after 5 days of TB directed therapy and CSF drainage. However, she had persistent weakness and paresthesias in her left foot possibly secondary to arachnoiditis. This case illustrates that patients with tuberculous meningitis can rapidly progress from Stage I, without any signs of hydrocephalus, to Stage III disease with stupor and dense hemiplegia, potentially requiring surgical intervention to relieve elevated intracranial pressure. As a result, this diagnosis requires a high degree of clinical suspicion in those at risk for TB reactivation and warrants empiric treatment prior to the return of confirmatory testing.

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Always Be ready: Resolved Lactic Acidosis Following Thiamine Bagley, Patrick DO. Barelski, Adam MD.
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Introduction:
Thiamine (Vitamin B1) deficiencies are associated with cardiac diseases (beriberi) and neurologic diseases (Wernicke-Korsakoff syndrome), however its role in sepsis is emerging in “metabolic resuscitation.”

Case:
A 72-year-old alcoholic man presented with avolition. Vitals were normal and exam revealed euvolemia, normal mentation, and a non-focal neurologic exam. He had a lactate of 6.7 nmol/L and ethanol level of 224 mg/dL. He was treated for alcohol withdrawal, receiving thiamine 100mg IV and crystalloids. Lactate five hours later was 6.2 nmol/L. Sepsis, alcoholic ketoacidosis, and adrenal insufficiency were excluded. No cause of poor lactate clearance was found. Given undetectable folate with a normal albumin, acute nutritional deficiency was presumed. 200mg of thiamine with given, resulting lactate of 1.4 nmol/L 16 hours after presentation.

Discussion:
We show a relationship of thiamine resuscitation and resolution of elevated lactate after excluding more common causes of lactic acidosis. Thiamine is a co-factor for pyruvate dehydrogenase which allows entry the Krebs cycle thereby avoiding lactate production from glycolysis. Total body stores of thiamine are small and easily depleted. In a pilot study of 30 patients with lactate greater than 4nmol/L, thiamine levels were significantly negatively correlated with lactate levels. Another study revealed thiamine-deficient patients with sepsis had significantly improved lactate clearance at 24 hours after receiving thiamine. Experts on sepsis now add ascorbic acid and thiamine to standard treatment.

Conclusions:
Our patient appeared to have accelerated lactate clearance when treated with aggressive thiamine repletion. Metabolic resuscitation is a promising approach to expedite clearance of lactate with an evolving evidence base and potential clinical application in the treatment of sepsis.

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Systemic EBV-Positive T Cell Lymphoproliferative Disorder of Childhood

Introduction: Systemic EBV-positive T cell lymphoproliferative disorder of childhood (STLPD) is a rare complication of primary Epstein-Barr virus (EBV) infection. It has lot of clinical and histologic features that overlap with EBV associated hemophagocytic lymphohistiocytosis (HLH); however, in HLH the patient will not have evidence of malignancy.

Case: A 20 year-old man was admitted to the hospital for an elevated creatinine that was discovered at an urgent care center where he had initially presented due to a 1 week history of diarrhea. He reported that 5 months before presentation he had a sore throat and was treated with amoxicillin; the antibiotic was stopped after he developed a rash. Physical examination was significant for cervical lymphadenopathy. Significant laboratory findings included elevated BUN, serum creatinine, nephrotic range proteinuria, elevated liver enzymes, coagulopathy and elevated ferritin. Imaging studies showed enlarged kidneys, splenomegaly, ascites and diffuse anasarca. Preliminary kidney biopsy was consistent with minimal change disease. He was discharged on prednisone. EBV viral load was measured as being one million copies and left cervical lymph node biopsy was consistent with systemic EBV-positive T cell lymphoproliferative disorder of childhood with distorted architecture and marked paracortical expansion by histocytes and lymphocytes but no hemophagocytic activity. In situ hybridization for EBV encoded RNA (EBV-ER) was positive in the atypical T cell population found on flow cytometry. Ten days later he was readmitted due to worsening renal failure with hyperuricemia, hyperkalemia and volume overload. Unfortunately he died after suffering a cardiac arrest while undergoing a central line placement.

Discussion: Most cases of EBV infections are asymptomatic while some cases are complicated by the development of HLH or STLPD of childhood which is defined as a monoclonal disease of EBV infected T cells with an activated cytotoxic phenotype. STLPD has variable histological changes. Usually the patient follows a fulminant clinical course arising from the hemophagocytic syndrome and multiorgan failure. In terms of management, the clinician may use chemotherapy at the time of diagnosis, but it usually does not respond to treatment.

Conclusion: EBV positive HLH and STLPD have similar clinicopathological findings and can arise from the same disease process. It is hard to distinguish between these due to the technical limitations of clonality studies which have unclear impact clinically with an overall poor prognosis.

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A BURNING QUESTION: IS IT ACTUALLY POLYMYOSITIS?
Rohit Aloor, MD; Raman Bharaj, MD; Medstar Health Internal Medicine, Baltimore, Maryland

Polymyositis, a diagnosis of exclusion, is a commonly misdiagnosed disease. Instead, patients often have inclusion-body myositis, necrotizing autoimmune myositis, or another inflammatory pathology. Scrutinious analysis of the timeline of disease progression, in addition to muscle pattern involvement, enzyme levels, autoantibodies, and biopsy interpretation, is critical for a proper diagnosis and to prevent unnecessary intervention.

A 48-year-old woman, with history of polymyositis and motor vehicle accident (MVA) with resultant neuropathy, presented to the hospital with a three-week history of generalized weakness and fatigue. Polymyositis was diagnosed 18 years prior to admission. The patient had been taking pravastatin for five years and one month prior to admission, she was treated for a urinary tract infection with a three-day course of ciprofloxacin. Since then, her proximal muscle weakness resurfaced after having been relatively dormant since initially diagnosis. Upon presentation, CK levels were elevated to 11,706 and AST/ALT were 414/246. She was started on high dose steroids for three days. Her CK levels started to down trend to 8,458 but after de-escalating to 110mg methylprednisolone, patient's CK and LFTs rose to 19,955 and 513/392, respectively. MRI of the legs revealed fatty replacement throughout the muscles. Her proximal muscle weakness gradually improved after nearly two weeks. Electromyography was limited, but showed evidence of myopathy, ruling out a neurologic cause. Muscle biopsy was performed once CK levels were under 3,600, revealing findings consistent with necrotizing autoimmune myositis. Further, the signal recognition particle (SRP) antibody, a marker for necrotizing myositis, was positive while HMG coA reductase antibody was negative.

Our patient's hospital course likely reflects a diagnosis of statin-induced necrotizing autoimmune myopathy precipitated by recent ciprofloxacin use. The weak inhibition of CYP3A4 by ciprofloxacin inhibits the metabolism of the statin, leading to increased toxicity. Focusing on the timeline of muscle weakness development and clinical progression with steroids, and a broad workup including autoantibodies, EMG, and muscle biopsy proved essential for a comprehensive diagnosis.

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A YOUNG ADULT WITH ACUTE KIDNEY INJURY PRESENTING WITH MIXED NEPHRITIC-NEPHROTIC SYNDROME
Sumit Chhetri, MD; Nabin Karki, MD; Gerard M. Lowder, MD; Jennifer N. Broussard
MedStar Harbor Hospital, Baltimore, MD

Introduction
Acute kidney injury (AKI) is a sudden decrease in kidney function with or without kidney damage, occurring over a few hours or days. Full recovery of kidney function is uncommon, which leaves the risk of long-term morbidity and death — estimates of AKI prevalence range from <1% to 66%. Timely evaluation and management is the critical approach. We present a patient who presented with AKI and steps we performed to find the cause of AKI.

Case report
A 34-year-old Caucasian man with a history of intravenous heroin use presented with diffuse abdominal pain for two weeks associated with intractable nausea and vomiting. Urine volume had been decreased for the past couple of weeks and severely reduced in the last two days. Physical examination revealed the presence of diffuse tenderness to palpation of the abdomen and non-pitting edema of both lower extremities. Blood pressure was 156/94 mm Hg. Hemoglobin was 5.7 gm/dL, so three units of packed RBCs were transfused. Serum potassium was increased at 7.7 mg/dL. Creatinine 14.8 mg/dL and BUN 123 mg/dL, baseline creatinine 1.6 mg/dL. Emergent dialysis was performed for oliguric renal failure and hyperkalemia. Urinalysis showed red colored urine, proteinuria of 100 mg/dL and 578 RBCs/hpf. Urine toxicology screened positive for cocaine and opiates. FeNa was 1.7%. Urine protein to creatinine ratio was 6.4 mg/g present in addition to hypoalbuminemia while rest of liver function tests was normal indicating nephrotic range proteinuria. Hepatitis C was negative, ANA and ANCA IgG antibody titers were not clinically significant. Glomerular Basement membrane antibody IgG was negative. Low C3 and normal C4 pointed towards activation of the alternative complement pathway. Renal biopsy showed membranoproliferative glomerulonephritis (MPGN) with crescents and C3 deposits.

Discussion
Heroin has been associated with the occurrence of renal disease. Morphine, the active metabolite of heroin, is known to have a direct cytotoxic effect on the renal parenchyma including mesangial cell and glomerular epithelial cell hyperplasia. This is thought to be mediated by activated macrophages that ultimately cause mesangial matrix synthesis and injury from oxidative stress.

Conclusion
In new onset AKI, it is always important to find the cause as many of the common etiologies are treatable. If not, AKI is more likely to develop into chronic kidney disease, end-stage kidney disease, and even premature death.
INFECTIVE SUBACUTE BACTERIAL ENDOCARDITIS OF MITRAL VALVE DUE TO ABIOOTROPHIA DEFECTIVA. Patel S, MD, The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Abiotrophia defectiva related endocarditis accounts for 6% of streptococcal endocarditis and has been identified as part of the normal human oral flora. The organism often infects native heart valves in immuno-competent individuals. It is associated with severe presentations including severe heart failure, renal failure and arrhythmias. It is most often associated with poor clinical outcomes.

A 62-year-old male with history of hypertension and mild to moderate mitral regurgitation presented to the emergency department with 6 months of generalized weakness, night sweats, rash, and fevers. One month prior to symptom onset, he underwent routine dental cleaning without any invasive procedures or history of IV drug use. He underwent outpatient workup for hematologic malignancy due to anemia and thrombocytopenia which yielded a normal bone marrow biopsy, EGD, cystoscopy, and PET/CT. Transesophageal echocardiogram two months prior to presentation was notable for normal LVEF with thickened mitral valve leaflets, mild to moderate mitral regurgitation and no vegetations.

During the hospitalization, the patient was noted to have a petechial rash, hepatosplenomegaly, shortness of breath and lower extremity swelling. He was started on IV vancomycin and ceftriaxone for concern of endocarditis. Transesophageal echocardiogram (TEE) was notable for reduced EF of 20% with multiple echo densities notable on the anterior and posterior mitral leaflets with an anteriorly directed jet. Blood cultures were positive for Abiotrophia defectiva. Hospital course was otherwise complicated by renal failure due to embolic phenomena in the setting of endocarditis. Therefore, he was transitioned from IV vancomycin to daptomycin. All remaining rheumatologic workup was negative. He underwent mitral valve replacement surgery ten days after presentation. Intraoperative TEE was notable for new aortic valve vegetations with regurgitation and he underwent both mitral and aortic valve replacements. Post-operative course was complicated by cardiogenic and hemorrhagic shock resulting in death.

Subacute bacterial endocarditis in immuno-competent individuals is rare but should be considered in patients with gradually worsening fatigue and mild valvular abnormalities. Early referral of patients for cardiac surgery is crucial for survival.
INSIDIOUS SEPSIS FROM CANCER TREATMENT
Saif Almushhadani, MD; Himanshu Rawal, MD; George Pyrgos, MD; Louis Saade, MD; MedStar Health Internal Medicine, Baltimore, Maryland

Intra-vesical Bacillus Calmette-Guérin (IV-BCG) is the treatment of choice for non-invasive bladder cancer. The associated complications are local and benign. However, we present a case of IV-BCG induced disseminated TB causing multi-organ system failure and death.

A 77-year-old man with high-grade non-invasive bladder cancer status post trans-urethral resection of bladder tumor and induction and maintenance IV-BCG presented with complaints of progressive weight loss, fatigue, abdominal distention, and dizziness during defecating. Patient had several hospitalizations for pleural effusion, anorexia, and ascites over the course of two months. Upon presentation, laboratory tests were remarkable for hemoglobin of 7.5 g/dL requiring multiple transfusions. Endoscopy revealed duodenal ulcer s/p clipping and CT scan of the abdomen showed extensive ascites and duodenitis. Paracentesis demonstrated transudative fluid consistent with portal hypertension which was not confirmed by portal venous dopplers. Hepatitis panel and ascitic fluid cytology and AFB were negative. Subsequently, the patient became hypothermic to 35.2° C and hypotensive. Laboratory tests were consistent with DIC and he progressively became hypoxic with new diffuse alveolar infiltrates, requiring intubation. Bronchoalveolar lavage (BAL) was consistent with diffuse alveolar hemorrhage. Subsequently, he developed multi-organ failure and family decided to withdraw care. Two weeks later, the BAL and blood cultures isolated Mycobacterium bovis.

Systemic complications due to IV-BCG are rare but associated with high morbidity and mortality which can occur months after the last dose of IV-BCG. The exact mechanism of the systemic complications of IV-BCG is unknown but most likely requires disruption of the urothelial barrier. Isolation of MB in the BAL and blood signifies hematogenous spread and not a hypersensitivity reaction. This rare complication of IV-BCG therapy requires a high level of clinical suspicion and coordination of care between multiple subspecialists. Empiric treatment should be considered until the diagnosis can be either confirmed or ruled out. The symptoms and clinical signs are non-specific and can lead to misdiagnosis. Appropriate blood cultures, bronchoscopy, and PCR in bodily fluids may lead to an accurate and more timely diagnosis.
A CASE OF HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS DUE TO SEPTIC SHOCK SECONDARY TO EHRLICHIOSIS IN A PATIENT WITH RHEUMATOID ARTHRITIS. Norcross G, MD, Strumpf Z, MD, Chen M, MS4, Wilding E, MD, Singh Z, MBBS. University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Hemophagocytic Lymphohistiocytosis (HLH) is a disease of over-activation of the immune system that is more frequently seen in children. However, it can occur in severely ill or immunosuppressed adults. Secondary HLH, or HLH not due to a genetic cause, is the predominant cause in adults. Disease states more commonly associated with secondary HLH are rheumatologic (SLE, adult-onset Still's), malignant (NHL, ALL), and infectious (viral, bacterial, fungal). These etiologies affect leukocytes (typically macrophages, NK cells, or T cells), which are activated and inappropriately target host tissue and cells.

The patient is a 44-year-old man with history of rheumatoid arthritis on weekly methotrexate who presented with fever, headaches, myalgias and sore-throat. He was found to have transaminitis, lactic acidosis, and thrombocytopenia. Shortly after presentation, the patient developed altered mental status, hypotension, and was intubated for airway protection. Broad spectrum antibiotics were started for presumed septic shock. Meningoencephalitis was presumed given altered mental status and physical exam signs of stiffness. A lumbar puncture was performed and showed low glucose, high protein, and leukocytosis with a lymphocyte predominance. Gram stain and culture were negative. A worsening anemia and thrombocytopenia were noted.

The patient was observed to have hemoptysis, prompting a bronchoscopy. The ferritin result sent at OSH was found to be >7500mg/mL. Hematology was consulted for high suspicion of HLH. A bone-marrow biopsy was performed after calculating the H-score. The H-score is a diagnostic score based on labs, imaging, and vital signs. The biopsy results showed “multiple foci and clusters of histiocytes are seen, many with engulfed hematopoietic cells,” and etoposide with high-dose steroids were started. APCR returned positive for Ehrlichiosis. The proper antibiotic regimen was started, and the patient slowly recovered.

In the critical care setting, there are many etiologies for anemia in the setting of septic shock. Further work up which includes an iron panel could suggest alternate causes. Given high mortality of untreated HLH, the diagnosis should be considered when more likely causes are ruled out.

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Splenic Artery Pseudoaneurysm

Introduction: Splenic artery pseudoaneurysm is a rare disease and its diagnosis is challenging due to its variable presentation.

Case Description: A 53-year-old woman presented complaining of constant abdominal pain and distention for two weeks. Her medical history was positive for chronic hepatitis C, chronic alcoholic abuse, cirrhosis, and chronic pancreatitis. Physical examination was positive for icterus and generalized abdominal distention with tenderness to palpation. Initial ascitic fluid analysis did not suggest spontaneous bacterial peritonitis (SBP). Abdominal ultrasound revealed an enlarged fatty liver and a pancreatic pseudocyst. After two days of supportive treatment, there was no improvement in her symptoms or leukocytosis. On repeat paracentesis ascitic fluid analysis was consistent with SBP and antibiotic therapy was initiated. After 48hrs on adequate antibiotic therapy, the patient continued to complain of significant abdominal pain. Abdominal computed tomography (CT) scan showed an enhancing density in the splenic artery suspicious for a contained rupture, an aneurysm or a pseudoaneurysm of the splenic artery. Splenic artery angiogram demonstrated a ruptured pseudoaneurysm with active extravasation of contrast into the inferior polar splenic artery. The patient underwent successfully embolization of this vessel and no further filling of the ruptured pseudoaneurysm was identified on post embolization imaging.

Discussion: The pathophysiology behind splenic artery pseudo aneurysm formation in cases with cirrhosis and portal hypertension is thought to be increased wall stress from portal congestion, medial hyperplasia and fragmentation of the elastic lamina. The diagnosis of pseudoaneurysms is not always easy in cirrhotic patients given its variable presentation and potentially being obscured by other more common manifestation of cirrhosis. Diagnosis is often made with CT or CT angiography while digital angiography remains the gold standard. While limited data are available, risk of rupture in pseudoaneurysms has been reported to be higher than in true aneurysms with approximately 90% mortality if untreated. Therefore, treatment of pseudoaneurysm should be initiated quickly, independent of size and symptoms.

Conclusions: This case exemplifies the importance of keeping a broad differential diagnosis when assessing patients with chronic abdominal conditions like cirrhosis with ascites and chronic pancreatitis presenting with abdominal pain. Splenic pseudoaneurysm should be considered in the differential diagnosis of cirrhotic patients thought to have SBP when they fail to respond to appropriate antibiotic therapy.

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Approaching Anomalous Coronaries
Aparna Sajja MD and David Furfaro MD
Johns Hopkins Hospital, Baltimore MD

Introduction - Anomalous origin of the coronary artery is rare, with an
incidence of about 1%. In most cases, it is not clinically significant and
identified incidentally on imaging. However, some patients may present
with syncope, angina, myocardial infarction, or sudden cardiac death.
Herein, we present two cases that demonstrate the spectrum of disease.

Patient 1 – A 50 year old female with history of iron deficiency anemia
presented after a first episode of syncope on exertion. She endorsed a
transient procrone of lightheadedness, with no chest pain or
neurological symptoms. Vitals and exam were normal.

Electrocardiogram (EKG) showed SIQ3T3. Labs were notable for an
elevated D-dimer (1.10) and microcytic anemia. Computed tomography
pulmonary angiography revealed no pulmonary embolus, but
demonstrated an inter-arterial right coronary artery (RCA). Coronary
computed tomography angiography (CCTA) showed a malignant,
anomalous variant of the RCA originating from the left coronary sinus,
traveling between the aorta and pulmonary artery. This can lead to
transient compression of the RCA and ischemia. Echocardiography
showed left ventricular ejection fraction of 60-65%. No coronary artery
disease (CAD) was noted on left heart catheterization. Cardiology and
cardiothoracic surgery recommended surgery and she subsequently
underwent unroofing of RCA.

Patient 2 – A 71 year old female with heart failure with preserved
ejection fraction, prior ventricular tachycardia status post ablation and
defibrillator placement, presented with dyspnea. EKG showed normal
sinus rhythm. Echo showed normal left ventricular function, but a newly
dilated right ventricle. She underwent left heart catheterization that
showed an anomalous coronary artery originating from the right
coronary cusp without significant CAD. CCTA demonstrated a single
coronary artery rising from the right sinus of Valsalva, with no inter-
arterial course, and with two left anterior descending arteries.

Ventilation/perfusion scan showed no evidence of pulmonary embolism
or chronic embolic disease and she was ultimately found to have
pulmonary hypertension related to obstructive sleep apnea.

Conclusions - Here we present two types and presentations of coronary
artery anomalies, with divergent management decisions. In the first case
with the inter-arterial type, patients are at greater risk of sudden death
especially during exertion. While the second patient had a unique
combination of an anomalous coronary artery with dual LADs, no
intervention was required since the course was not inter-arterial
TWENTY-SIX YEAR OLD MALE WITH END STAGE LIVER DISEASE FROM ALCOHOL. Kraslow M, MD. University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

End stage liver disease (ESLD) due to alcohol classically presents in 40 to 50 year olds with an estimated average daily consumption of over 100 grams of alcohol for 20 years. However, from 1999-2016, the incidence of deaths from ESLD increased three fold in 25 to 34 year olds. This 10.5% annual rise, the most of all ages, is mainly attributable to alcohol. The symptoms of ESLD are abdominal distention from ascites, coagulopathies, muscle wasting, jaundice, encephalopathy, and hepatorenal syndrome (HRS). ESLD ultimately necessitates a liver transplant.

A 26-year-old male without history of liver disease initially presented with abdominal distention and acute shortness of breath. He reported two years of fatigue, loss of muscle tone, and anorexia. He drank 24 ounces of wine or beer daily for the last 8 years. He denied tobacco or illicit drug use. On initial assessment, the patient had anasarca, jaundice, and polybacterial peritonitis. Imaging revealed an enlarged, fatty liver with portal hypertension, and work up ensued for a second contributor to his ESLD. Infectious work up for Epstein-Barr virus, human immunodeficiency virus, cytomegalovirus, syphilis, and Hepatitis A, B, and C was negative and Herpes Simplex 1 and Hepatitis E were positive. Testing for interleukin 2 receptor as well as the antinuclear, liver kidney microsomal, and mitochondrial M2 antibodies were all negative, effectively ruling out autoimmune causes. While the smooth muscle antibody titer displayed weak positivity, it was from an icteric specimen and deemed unnecessary to repeat. Systemic diseases such as Wilson’s disease and alpha 1 antitrypsin deficiency were also ruled out.

Ultimately, testing did not determine an etiology other than alcohol for the patient’s ESLD and he continued to clinically decline. Complications included hepatic encephalopathy and HRS necessitating continuous renal replacement therapy. He received a liver transplant one month after initial presentation.

This case illustrates how the demographic of ESLD is shifting toward a younger population. As physicians, we can work to halt this current trend by screening for alcohol use at a younger age and educating patients earlier about the dangers of alcoholism. It is important to keep liver damage secondary to alcohol in the differential of a younger person with abdominal pain as well as nonspecific complaints.
A RARE CAUSE OF STROKE

Introduction: The artery of Perchon (AOP) is a rare variation of the brain circulation, in which a single artery that arises from the posterior circulation supplies the thalamus bilaterally. This leads to paramedian thalamic infarcts. Studies have found that AOP infarction is involved in 0.1% to 0.3% of all ischemic strokes. The three cardinal features of infarct in this territory are vertical gaze palsy, memory impairment and coma.

Case Report: A 64-year-old male with two prior ischemic strokes, with residual left sided hemiparesis, presented with an acute change in mental status. He was found to have hypercapnic respiratory failure and required intubation and admission to the ICU. On physical examination pupils were not reactive to light, eyes were dysconjugate, with the left eye deviated laterally and left sided medial gaze palsy. Computed tomography (CT) of the head did not show any acute pathology, however, magnetic resonance imaging (MRI) of the brain revealed acute to subacute infarctions within both thalami compatible with infarctions within the territory of the AOP. Patient regained consciousness in less than 24 hours, mental status returned to normal, and he did not have new focal weakness.

Discussion: The thalami and midbrain have a complex blood supply with a vast number of feeding arteries. The thalamic vascular supply is divided in four territories: anterior, paramedian, inferolateral and posterior. The AOP is a rare variant of the paramedian circulation, which is characterized by a single dominant thalamoperforating artery that arises from the proximal segment of the posterior cerebral artery and bifurcates to supply both paramedian thalami. Bilateral thalamic infarctions are rare and account for 0.6% to 2% of cases of ischemic stroke, however, it can represent between 4 to 35% of thalamic strokes. Occlusion of the AOP, is a rare cause of ischemic stroke and gives rise to the characteristic pattern of bilateral paramedian thalamic infarcts.

Conclusion: AOP occlusion is a rare cause of ischemic stroke. It is difficult to initially suspect bithalamic paramedian infarcts because of the complex anatomy, which causes large clinical variability and radiological challenges, thus leading to misdiagnosis. Clinicians should consider AOP stroke in patients presenting with suddenly altered mental status.

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A CASE OF THYROTOXICOSIS PRESENTING AS THIRD DEGREE AV BLOCK. Asiri F, MBBS. University of Maryland Medical Center and VA Medical Center, Baltimore, MD.

Thyrotoxicosis can present in several different ways, making it a challenging diagnosis without biochemical confirmation. Symptoms of thyrotoxicosis include heat intolerance, palpitations, anxiety, fatigue, weight loss, muscle weakness, and irregular menses. Cardiac manifestations include sinus tachycardia, supraventricular tachycardia including atrial fibrillation and atrial flutter with or without cardiac failure.

A 31-year-old-man with history of Loewy-Dietz syndrome, bicuspid aortic valve and ascending aorta aneurysm/enlargement presented to a hospital with worsening palpitations, shortness of breath, and chest pressure. He was also anxious and complaining of lightheadedness. He reported diarrhea, diaphoresis, and unintentional weight loss of 20 lbs over the past 6 months. He was found to have thyrotoxicosis and treated with propranolol and steroids. He then developed third degree heart block with ventricular rate of 30 beats per minute (bpm) with associated hypotension. He was taken to catherization lab for transvenous pacing (TVP) and started on dopamine. During TVP placement, he became asystolic followed by runs of polymorphic ventricular tachycardia. His heart rate decreased with pacing set at 120 bpm. He was intubated for airway protection and transferred to a university hospital. Thyroid stimulating hormone was <0.01 mU/L and free T4 was 6.29 ng/dL. He had positive thyroid peroxidase antibodies and thyroid ultrasound demonstrated evidence of multiple hypoechogenic nodules and increased vascularity suggestive of Hashimoto's. Lyme titer was negative. He was started on propylthiouracil, cholestyramine, Lugol's iodine, and continued on hydrocortisone. He was evaluated by electrophysiology and was noted to have Junctional/first degree AV block. The patient did not require immediate placement of a permanent pacemaker.

The patient had no further arrhythmias and he remained hemodynamically stable. His TVP was removed two days after admission, and his dopamine was weaned off. Upon discharge, free T4 was 3.5 ng/dL and patient was switched to methimazole.

Complete AV block can occur with thyrotoxicosis but remains a rare occasion. Identifying underlying thyroid disease in patients who present with AV block is crucial, as treating the underlying thyroid dysfunction can help remove the stimulus of the arrhythmia. Furthermore, treatment for hyperthyroidism includes beta blockers, which can be deleterious in patients with AV block.
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NOTHING WRONG TO SEE HERE
Kristi Ngo, MD, Resident, National Capital Consortium, Bethesda, MD

Introduction: The broad, sweeping full “review of systems” is often cumbersome and too vague to apply to targeted appointments or admissions, especially in specialties where patients present for one specific complaint and may be hesitant to verbalize ‘unrelated’ problems. This effect can be heightened in geriatric or military populations where patients may perceive a power differential.

Case: A 68 year old, otherwise healthy gentleman was directly admitted to the inpatient Cardiology service for a planned atrial fibrillation cardioversion. He had distressing fatigue for several years that had been attributed to his arrhythmia. In his eyes, the atrial fibrillation was the root of all his complaints and he was eager to go through with his cardioversion. When asked broadly “is there anything wrong you would like to talk about”, he perseverated on his fatigue but ultimately reported “I feel totally fine, doc!” Other open ended invitations to discuss other complaints were met by the same reassurance. Targeted review of symptoms of the heart and lungs were negative. However, when asked directly ‘have you had any vision changes’, he admitted to being partially blind in his right eye since yesterday morning. Physical exam confirmed total vision loss in the lower visual field of the right eye. Urgent Ophthalmology consult revealed bullous retinal detachment sparing the macula. He underwent retinal surgery the next day. Upon later questioning, he reported wanting to get his conversion done first and had planned to ask his primary physician about his vision next week. He ultimately received his conversion that admission, after being cleared for anticoagulation by Ophthalmology. On his one month follow up, he had recovery of his visual field, though with persistent mild metamorphopsia.

Conclusion: Geriatric patients may be accustomed to taking a passive role in their care. They may also see a range of specialists for their comorbidities, and feel that specific complaints are only appropriate for those specialists. This particular gentleman was a perfect storm of the above, with the additional fear that an ‘unrelated’ complaint would prevent him from a desired intervention. This case illustrates the importance of completing a generalized review of systems with direct questions, as opposed to limited or open ended questioning that places the onus on patients to volunteer complaints.

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