A NEW LOOK AT RENAL FAILURE IN THE HEART FAILURE PATIENT. Sahbaz J, MD, Kastner E, MD, Ramani G, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Acute kidney injury (AKI) often complicates acute decompensated heart failure (ADHF). Although cardiorenal syndrome (CRS), alterations in volume status, and nephrotoxic medications are common causes for AKI in patients with ADHF, other causes deserve consideration.

A 76-year-old woman with chronic kidney disease (CKD), heart failure with reduced ejection fraction, and atrial fibrillation, not on anticoagulation, presented with confusion. On physical examination, she was altered, oriented only to self with active hallucinations but appeared comfortable. Physical examination was notable only for bilateral lower extremity edema and an elevated jugular venous pressure.

Lab work revealed an elevated creatinine to 6.1 mg/dL and potassium to 5.4 mmol/L. The patient’s kidney function continued to deteriorate along with her mental status, and hemodialysis was initiated. The patient’s renal failure was initially believed to be due to CRS. However, despite volume optimization, blood pressure optimization, and avoidance of nephrotoxic drugs, her kidney function did not improve. Abdominal ultrasound and CT abdomen were relatively unremarkable for potential causes of AKI. Subsequent renal artery (RA) Doppler showed 60-99% right RA stenosis and an atrophied left kidney. RA angiography revealed a right renal artery thrombosis (RAT). The patient underwent renal angioplasty with stenting and, 4-6 hours postoperatively, began to produce copious urine with creatinine returning to baseline and no further need for dialysis.

Preserving renal function is critical in ADHF management. When AKI develops, mortality rises significantly. Traditionally, evaluation focuses on volume status optimization, meticulous blood pressure management, and careful medication review. If renal function does not improve, a thorough evaluation must be undertaken before conceding irreversible renal failure. Traditionally, RA Doppler is not part of the diagnostic workup of AKI in ADHF. In this case, given the history of atrial fibrillation without anticoagulation, it was crucial in establishing the correct diagnosis and preventing long-term dialysis dependence. RA Doppler is a useful preliminary, non-invasive study to identify stenotic lesions, and angiography is confirmatory. Treatment options for renal ischemia due to a RAT include but are not limited to systemic anticoagulation, local thrombolytic agents, balloononing, stenting, and thrombectomy.
Prophylaxis Turned Poise: A Lesson in Electrolyte Repletion
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Asymptomatic electrolyte derangements are exceedingly common in hospitalized patients and are routinely corrected without delay for evaluation. The underlying etiology is typically multifactorial and associated with the presenting illness. However, infrequently, we must exclude rarer etiologies that are potentially lethal. In the following case, we present a rare, but important side-effect of a common medication.

A 56-year-old man with chronic myelomonocytic leukemia presented for his fourth cycle of hyper-CVAD (Cyclophosphamide-Vincristine-Adriamycin-Dexamethasone). This aggressive regimen was chosen after two cycles of dose-adjusted EPOCH-R (Etoposide-Prednisone-Vincristine-Cyclophosphamide-Doxorubicin-Rituximab) failed to show a satisfactory response. Prior to chemotherapy, the patient tested positive for hepatitis B surface antigen, and was thereafter prophylaxed with tenofovir to prevent reactivation.

His fourth cycle of hyper-CVAD was complicated by tumor lysis syndrome, which was diagnosed after laboratory evaluation showed hyperphosphatemia, hyperuricemia and hypocalcemia. Aggressive intravenous fluids and rasburicase were given with gradual resolution. The hospital stay was further complicated by neutropenic fever, along with severe hypophosphatemia, hypokalemia and hypomagnesemia despite scheduled oral and intravenous electrolyte repletion. Urine studies showed marked glucosuria, increased fractional excretion of phosphorous and magnesium. Given the known association of Fanconi syndrome and tenofovir, this medication was immediately discontinued. Diagnosis was confirmed when the electrolyte abnormalities had resolved prior to discharge.

Tenofovir is a commonly encountered medication for general internists, as it forms the backbone of HIV/HBV treatments. This rare diagnosis was initially obscured by co-existing tumor lysis syndrome, but became evident with refractory electrolyte deficiencies. This case illustrates the utility of pattern recognition, and to consider a rare toxicity of a common medication.
BILATERAL BASAL GANGLIA INFARCTION IN THE SETTING OF DRUG-INDUCED HYPOXIA

Introduction:
Patients can suffer a hypoxic-ischemic brain injury after events such as head trauma, vascular accidents, cardiac arrest, poisoning, or overdose. They usually present in an obtunded or comatose state making it difficult to obtain an accurate history or perform a constructive physical exam. Correctly identifying the extent of the injury through radiologic findings can give insight into the nature of the injury which may aid in acute resuscitation, sub-acute treatment of a patient, and clinical prognosis. Once a patient is stabilized, MRI is the preferred modality to further assess the extent of damage done to the brain after an insult has occurred. Herein, a case presentation of bilateral basal ganglia infarctions in the setting of hypoxia will be discussed.

Case Presentation:
A 54-year-old Caucasian male with a past medical history of polysubstance abuse presented with altered mental status and unresponsiveness. At that time, the patient was found to be obtunded by opiate and benzodiazepine overdose. The patient received narcan and atropine in the emergency room and became responsive. Physical findings included lethargy, but arousable to verbal stimuli, significant psychomotor slowing, asterixis, and decreased strength (4/5) in all extremities. Laboratory values showed elevated creatine kinase max of 9,406 and lactic acid of 3.8. US carotid duplex showed less than 50% stenosis on the right and left carotid arteries. MRI Brain/MRA Head without contrast showed bilateral basal ganglia subacute infarctions, most likely related to hypoxemic state with gross patency of the great vessels of the brain. The patient was treated with aspirin, atorvastatin, intravenous fluids, physical therapy, and supportive care. The patient experienced no focal deficits.

Discussion:
Bilateral basal ganglia damage is a nonspecific finding; so correlating the radiologic findings to the clinical situation and physical exam findings is important. The MRI can show specifically where the brain is damaged, the extent of injury, and provide some information about the nature of the insult. The basal ganglia have high metabolic activity and are often affected first and symmetrically in systemic processes such as hypoxemia, as seen in this patient’s MRI. Other processes such as osmotic myelinolysis, Wernicke encephalopathy, and cerebral deep vein thrombosis can cause bilateral lesions seen on MRI; however, these processes usually affect more inferior structures within the midbrain. Hence, it is important to look at imaging and the clinical picture of the patient simultaneously and to keep a wide differential in lesions with nonspecific etiologies to avoid false attribution and misdiagnosis.

Conclusion:
A majority of brain injuries require a radiological work-up; however, because of the unpredictable nature of the organ it is critically important to clinically correlate findings to imaging results and keep a wide differential diagnosis as the patient’s course progresses.
WOE UNTO ME IF I FORGET EXTRAPULMONARY TB – A TALE OF TWO PATIENTS

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Extrapulmonary tuberculosis (EPTB) is defined as tuberculosis (TB) disease outside of the lung parenchyma. Virtually any organ system can be involved; cutaneous, ocular, lymphatic, CNS, pleural, pericardial, genitourinary, and GI manifestations have been reported. Globally, EPTB accounts for approximately 15% of new TB cases, but may be underreported due to diagnostic difficulty. Diagnosis of EPTB is challenged by a low tuberculosis incidence, relative unawareness among clinicians, and the variable presence of constitutional and organ-specific symptoms. Even when symptoms are present, they often mimic presentations of more common diseases, making it difficult for clinicians to hone in on diagnosis.

Case 1: An 84-year-old Indian female presented after 3 months of fatigue, early satiety, weight loss and progressive abdominal fullness. Exam showed abdominal distension with dullness in the flanks. Imaging demonstrated ascites with peritoneal nodularity. Labs were notable for an elevated CEA-125 and CA-19-9, hypercalcemia, and ascitic fluid with 834 leukocytes per microliter with a lymphocytic predominance. Laparoscopy revealed diffuse peritoneal studding. Biopsy of the omentum was notable for caseating granulomas and a positive direct TB DNA probe test. RIPE therapy was initiated. She was discharged home but she presented 2 days later with new abdominal pain and died from a suspected bowel perforation.

Peritoneal TB is a result of reactivation of bacilli in the peritoneal space or mesenteric lymph nodes. The typical presentation is subacute abdominal pain, constitutional symptoms, diarrhea, and ascites with 80% lymphocytic predominance. Bleeding, obstruction, and perforation are possible complications. Our case highlights: 1) peritoneal TB mimics malignancy; 2) hypercalcemia may complicate TB; and 3) complications such as perforation may be rapidly fatal.

Case 2: A healthy 29-year-old female nursing aide presented to clinic with a painful lump on her back. It was felt to be a lipoma and observed. Over the next two months, it increased in size and pain, it was incised and drained, and treated with a short course of antibiotics. She developed progressive pain and night sweats, and presented to our hospital for evaluation. On exam she was afebrile and her right flank was tender. Imaging revealed T10-L3 vertebral and right rib osteomyelitis with right paraspinal abscesses. She developed fevers while on broad spectrum antibiotics and after adequate drainage of the fluid collections. AFB stains were negative, but her abscess cultures were positive for TB after 16 days. She was discharged home on RIPE therapy.

Spinal TB (Pott’s disease) involving thoracic vertebral bodies comprises up to 50% of skeletal manifestations of TB. The typical presentation is subacute local pain, lower thoracic to upper lumbar involvement, muscle spasms and rigidity, constitutional symptoms, and adjacent abscess formation. In contrast to pyogenic spondylitis, disc space involvement in spinal TB is a late finding. Our case highlights that low thoracic osteomyelitis sparing the disc spaces associated with paraspinal and psoas abscesses should raise concern for Pott’s disease.

The above two cases along with a rising proportion of EPTB among all TB cases highlight the need to raise awareness so that clinician0073 maintain a high index of suspicion for EPTB to provide for timely diagnosis and treatment.


NOT OUR GRANDMOTHERS' SPIROCHETES
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Human Intestinal Spirochetosis (HIS) is an unusual infection that tends to have significant prevalence in children in developing countries and homosexual males, particularly those with HIV infection in developed countries, for unknown reasons.

A 17-year old African American man presented to the emergency department complaining of watery diarrhea and hematochezia of 5 days duration. He admitted to having unprotected anal intercourse two months prior. Vital signs were unremarkable and physical exam findings included submandibular and posterior cervical lymphadenopathy with oropharyngeal ulcerations. Laboratory studies showed leukopenia (3,100), thrombocytopenia (54,000), elevated liver enzymes, and a positive fecal occult blood test. A fourth generation HIV assay was positive. Ultrasound showed only gallbladder sludge. Colonoscopy was scheduled for the next day. Initial clinical impression was acute HIV infection with diarrhea being related to infectious proctitis. The following morning, the patient had a temperature of 38.1°C. Colonoscopy was notable for scattered erythematous mucosa with nodular lesions in the cecum and rectum. Biopsy results demonstrated lymphoid aggregates, reactive parakeratosis, and prominent spirochetosis. Serologic testing for syphilis was negative. Blood culture, stool culture, and ova and parasite testing were negative. After discharge, HIV confirmatory testing was positive with a CD4 count of 346 and viral load of 2.6 million and the patient was notified to follow-up as an outpatient.

HIS is associated with the overgrowth of spirochetes in the genus Brachyspira. Originally linked to animal diarrheal disease, it has been linked to human colonic adherence for lesser-known reasons. The mode of transmission is unknown and it is unclear if the presence marks colonization or an actual disease process. It is usually an incidental finding diagnosed histologically, which is significant for a false brush border appearance. HIS is also found in developed countries, particularly homosexuals regardless of HIV infection or immune status. Since HIS is an entity distinct from syphilis proctitis, it is important to keep it in the differential diagnosis. Antibiotic efficacy is variable in HIS and the use of metronidazole may be more appropriate when there is spirochete invasion deep into the epithelium, which usually correlates with more severe symptoms.
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ROLE OF FIBRINOLYTIC/ MUCOLYTIC THERAPY IN TREATING COMPLEX LOCULATED EMPYEMA
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Parapneumonic effusions are a common complication of bacterial pneumonia that can occur independent of any other factors. Fibrinolytic/mucolytic injections, such as tPA and DNase, into the pleural space are superior to fibrinolytic injection alone. They are associated with decreased surgical referrals and hospital stay.

A 60-year-old Caucasian man with no significant past medical history presented with gradually increasing cough with dyspnea, occasional green sputum production, and left-sided flank pain. He was vitally stable and physical exam was significant for absent breath sounds on the left side of the chest. CXR and CT chest revealed left lung consolidation with an extensive loculated effusion. Upon pigtail catheter placement by IR, viscous yellow fluid was aspirated and sent for microbiologic analysis. There was no spontaneous drainage of the effusion and IR recommended an alteplase lock due to extensive loculations. Culture grew Strept intermedius and patient was started on IV ceftriaxone. Due to inadequate drainage, a decision was made to initiate fibrinolytic therapy, which involved injection, clamping, and drainage of tPA and DNAase for an hour each, twice daily for three days. The patient reported significant improvement in dyspnea. A repeat chest CT showed dissolution of the loculations and expansion of the left lung.

Parapneumonic effusions are pleural effusions that form in the pleural space adjacent to a bacterial pneumonia. Found in at least 40 percent of bacterial pneumonias, the usual parapneumonic effusion is small and resolves with appropriate antibiotic therapy. However, if bacteria invade the pleural space, a complicated parapneumonic effusion or empyema may result, which will require antibiotic therapy plus additional intervention. Isolated fibrinolytic therapy in the past was not associated with improved outcomes in many studies. Recent trials of thrombolysis with tPA combined with DNase to reduce viscosity of infective effusions have resulted in lower referral rate to surgery and shorter hospital stays.

(Indicating review of abstract)
SCARRED FOR LIFE: A RARE CASE OF CONCOMITANT PNEUMOTHORAX AND BILATERAL PNEUMOMEDIASTINUM IN HERMANSKY PUDLAK SYNDROME

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Hermansky-Pudlak syndrome (HPS) is an autosomal recessive disorder that is associated with oculocutaneous albinism, bleeding diatheses, granulomatous colitis, and highly penetrant pulmonary fibrosis especially with HPS-1, HPS-2, and HPS-4 subtypes. It is an extraordinarily rare disorder, occurring at a prevalence of 1:500,000 to 1:1,000,000 worldwide. Pulmonary fibrosis in HPS shows many of the clinical, radiologic, and histologic features found in chronic interstitial pneumonia usually occurring at a younger age. Although pulmonary fibrosis has occurred in numerous patients suffering HPS, there are no reports of coexisting bilateral pneumomediastinum and pneumothorax as a complication of the progressive fibrosis. There are currently no definitive therapeutic or preventive approaches for HPS pulmonary fibrosis other than lung transplant.

A 47-year-old woman of Puerto-Rican descent with a known history of HPS and significant interstitial lung disease on 5L home O2 presented with constant sharp chest pain that started a few days prior to admission. She had grade IV dyspnea on admission with an oxygen saturation of 94% accompanied by a constant dry cough. She was worked up for infection; however, remained afebrile throughout. Blood and sputum cultures were negative and WBC was 15.4 on admission. Computed tomography showed a small right pneumothorax and bilateral pneumomediastinum which was managed conservatively. Eventually, she was transferred to a higher level of care due to her increasing oxygen needs. Her lung findings found in the setting of HPS and pulmonary fibrosis was secondary to the increased intrathoracic pressure from coughing. She was managed with analgesia, anti-tussive, Pirfenidone at her home dose and avoidance of activities that increased intrathoracic pressure.

Pulmonary fibrosis is the most serious complication and the main cause of mortality within a decade of diagnosis in patients with HPS. This case presents a rare event of pneumothorax and bilateral pneumomediastinum occurring together in HPS as an end-stage progression of the pulmonary fibrosis despite being on antifibrotic medication.
EVANS SYNDROME
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Evans Syndrome is a rare variant of autoimmune hemolytic anemia (AIHA) that does not fit the criteria of other recognized immune disorders. Evans Syndrome is characterized by the combination Immune Thrombocytopenia (ITP) and Autoimmune Hemolytic Anemia (AIHA) with a positive direct coombs test. The rarity of Evan’s syndrome makes it a formidable diagnostic challenge.

This patient is a 37-year-old African American woman who presented from a nursing home with complaints of lethargy and weakness for the past 2 weeks. Her past medical history is significant for hypertension, type 2 diabetes mellitus, hyperlipidemia, CKD and CVA with residual left sided hemiparesis in 2010. The patient presented with 3 episodes of emesis each day for 3 days prior to admission and anorexia. She reported mild epigastric pain. She denied hematuria, dysuria, changes in urinary frequency, hemoptysis, oral or anal bleeding. On presentation, the patient’s vitals signs were within normal limits. Physical exam was significant for mild left sided facial droop, 0/5 strength in left upper and lower extremities, 5/5 strength on right side, and mild tenderness in mid-epigastrum. Initial laboratory results revealed hematocrit of 29.5%, platelet count of 28,000, elevated LDH, low haptoglobin, creatinine of 3.21 (baseline of 2.0). Peripheral blood smear revealed schistocytes, elevated reticulocyte count and spherocytes. Further testing revealed ANA (1:160), borderline elevated anti-cardiolipin antibody and positive Coombs test with negative C3 IgG, suggesting warm autoimmune hemolytic anemia with thrombocytopenia. A working diagnosis of AIHA was established and the patient was started on dexamethasone 40 mg for 4 days upon which her platelets stabilized around 28,000. The patient was then started on IVIG upon which her platelets improved to 140,000 and hemoglobin/hematocrit increased to 8/24.3%. Renal function also improved with creatinine of 1.44. Given the autoimmune nature with positive coombs test and rapid improvement with steroids and IVIG without requiring plasmapheresis, the diagnosis of Evans Syndrome was established.

As mentioned, Evans Syndrome is a rare hematologic condition, which must be kept in mind when evaluating patients with positive coombs test, anemia, and thrombocytopenia as early treatment leads to better outcomes. This patient responded well to corticosteroids and IVIG, which is the first line treatment for Evans Syndrome.
Beatriz Wills MD, Richa Gupta MD. Isolated cardiac sarcoidosis. Johns Hopkins Hospital. Baltimore, MD. Case: A 25-year-old male with history of bipolar disorder on numerous psychotropics, CKD and asymptomatic 1st degree AVB and RBBB presented with syncope and collapse. Family history was significant for pulmonary sarcoidosis in his father. Vitals were notable for HR 50s but otherwise within normal limits. Cardiopulmonary exam was normal. Troponin was 0.45. EKGs 2:1 AVB alternating right and left bundle branch block. TTE: EF 45-50%, mildly dilated RV, hypokinesis of the anterior and septal walls suggestive of abnormal conduction. Heart block resolved after cessation of home clonipramine and ECGs subsequently showed a consistent LBBB. Chest CT was unremarkable. Coronary angiography demonstrated no obstructive coronary disease. FDG-PET showed intense FDG uptake of the basal and mid segments of the inferolateral wall (Fig 1) compatible with active-inflammatory myopathy, likely cardiac sarcoidosis. Prednisone 30 mg daily was started. Single chamber PPM (VVI) was placed. At four month follow up, there were no new signs or symptoms suggestive of disease progression. Discussion: Sarcoidosis is a systemic granulomatous disease of unknown etiology. Approximately 25% of patients with systemic sarcoidosis have asymptomatic cardiac involvement, while 5% have isolated clinical cardiac manifestations. While the constellation of cardiomyopathy with conduction disease and a family history of sarcoid makes this diagnosis likely, the lack of extracardiac manifestations and the presence of medications associated with conduction abnormalities provides a degree of uncertainty. EMB is the gold standard for cardiac sarcoidosis, however, it is poorly sensitive in detecting disease due to the patchy nature of cardiac infiltration. The constellation of heart block and non-ischemic cardiomyopathy raised suspicion for infiltrative cardiomyopathy, which triggered further evaluation with PET-CT. On the contrary, PET-CT has a reported sensitivity and specificity of 100% and 95.5%. VVI backup pacing was chosen as the electrical therapy in this instance. The decision to forgo cardiac resynchronization therapy due to the presence of a left bundle was not pursued given the potential for improvement in conduction after treatment of inflammatory sarcoid as well as the withdrawal of offending medications. Further, the decision to not provide tachycardia therapy was also similar lines – hope for EF recovery and decrease in inflammation. Conclusion. Cardiac sarcoidosis is challenging in the absence of systemic manifestations. Varying degrees of cardiac conduction delay and syncope can be the first manifestations. Abbreviations: CKD: chronic kidney disease; AVB: atrioventricular block; RBBB: right bundle branch block; TTE: Transthoracic echocardiogram; EF: ejection fraction; LBBB: left bundle branch block; FDG-PET-CT: Fluorine-2-fluoro-2-Deoxy-d-glucose (18F-FDG) positron emission tomography/computerized tomography;
IRON-DEFICIENCY ANEMIA IN CEREBRAL SINUS THROMBOSIS: CHARACTERISTIC OF A PATIENT POPULATION OR RISK FACTOR?

Introduction: Cerebral sinus thrombosis (CST), while rare, is an important diagnosis, given the associated morbidity and mortality. It is important to obtain a thorough history to determine which patients warrant further investigation for CST. Further, while oral contraceptive (OCP) use is a known risk factor for CST, there may be other factors.

Case Presentation: An 18-year-old female with history of migraines and menorrhagia on OCPs, presented with severe intermittent headache, involving left cranial for 3 weeks. It was associated with nausea, and relieved with naproxen. The patient denied visual changes. Her prior migraines were in the right frontal area, less severe, without visual aura, and responded well to oral analgesics. Physical examination was benign. Laboratory values showed hemoglobin of 8.7 gm/dL, hematocrit 31.9%, MCV 64.6 fl, platelet count of 411 K/ul, iron level 17 mcg/dL, TIBC 517 mcg/dL, iron saturation 3%, ferritin 3.1 ng/mL. MRI with MRA/MRV brain showed complete occlusion of the left sigmoid sinus and transverse sinus. The patient was started on therapeutic enoxaparin and OCP discontinued. She was transitioned to warfarin and was advised follow-up with hematology for hypercoagulable work-up after warfarin treatment. Patient was treated for iron-deficiency anemia (IDA).

Discussion: Aspects of history may suggest CST as the cause of headache. For example, new headache pattern, signs of increased intracranial pressure, seizures or focal neurologic deficits warrant imaging for CST. CST risk factors include females, OCPs, pregnancy, peripartum, and hypercoagulability. Reactive thrombocytosis, associated with IDA, may lead to thrombotic complications. Yakota et al. found reactive Factor VIII activity associated with IDA in a CST patient. Other authors think platelet hyper-responsiveness due to erythropoietin, and secondary hypercoagulability due to red cell deformity in IDA may contribute to CST. However, there may be a confounder, because young females with significant menorrhagia (with co-existing IDA), are the same patients who are prescribed OCPs, which place them at risk for CST. One could study the incidence of CST in females placed on OCPs, with one group rigorously treated for IDA compared to standard treatment. The other possibility could be to study the incidence of CST in males with IDA, compared to males without. Yet, because CST is such a rare disorder, such studies may not have enough power.

Conclusion: It may be beneficial to rigorously treat iron-deficiency anemia in patients started on oral contraceptives for menorrhagia to reduce the incidence of cerebral sinus thrombosis, due to procoagulant associations of iron-deficiency anemia. However, more study is needed.
Not so Fast!
F Ahmed M.D., B Waseem, M.D., D Pradhan M.D., M Lamonte M.D., M Allison, M.D.

**Introduction:** Post-traumatic intracranial pseudoaneurysm is an uncommon cause of neurological deterioration after traumatic brain injury. We present a rare case of delayed post-traumatic intracranial pseudoaneurysm secondary to carotid artery dissection leading to thromboembolism and stroke in a young adult.

**Case description:** A 30-year-old male presented with acute onset dysarthria, aphasia and right sided weakness for 1 hour. Four months before presentation he had sustained extensive head and facial trauma, including fractures of the right orbital floor and medial wall, in a motor vehicle accident. Computed tomography (CT) of the head and CT angiography at the time of the accident had not revealed any abnormalities. Physical examination was remarkable for severe dysarthria and aphasia (NIHSS score 4). Repeat CT head was unremarkable, given the absence of contraindications, alteplase was administrated. Magnetic resonance imaging (MRI) of the brain showed an acute stroke in left temporoparietal region. Magnetic resonance angiography (MRA) of the neck revealed a dissecting pseudoaneurysm in the mid-to-distal left internal carotid artery measuring approximately 2 cm at the level of C1 with a mural thrombus. Conventional cerebral angiography confirmed the presence of the pseudoaneurysm with a distal intimal flap, moderate luminal compromise, and mild-to-moderate embolic occlusion of an insular branch. He was successfully treated with placement of a 5 x 40 mm Acculink stent across the neck of the pseudoaneurysm. Progressive improvement of his symptoms was noted during hospital stay. At follow up patient had complete neurological recovery.

**Discussion:** Extracranial internal carotid artery (EICA) aneurysm is a rare entity accounting for 0.8% to 1% of all arterial aneurysms.¹ Pseudoaneurysms of the internal carotid artery (ICAP) have a disruption across all layers of the arterial wall, unlike true aneurysms, and are less common comprising 14% of all cases of aneurysms.² The major cause of ICAP is previous endarterectomy, other causes include vasculitides, infections and arterial dissection. Traumatic ICAP is most commonly due to motor vehicle accidents (69%) followed by stab-wounds, iatrogenic central venous cannulation, sport accidents, fights, falls, and cervical manipulation. The incidence of EICA injury in patients with blunt trauma is about 0.08%.³ The diagnosis is often delayed due to other associated injuries and delayed manifestation of clinical signs.³ Diagnostic modalities include color Doppler sonography, contrast-enhanced CT or MRI, and cerebral angiography. Although rare, in some cases the initial imaging studies may be normal and the time from the development of the aneurysm to presentation varies widely. EICA pseudoaneurysms (EICAP) may be asymptomatic in 30% – 60% of cases. EICAP can be partially or completely thrombosed and may lead to embolization with cerebral infarction. The enlarging pseudoaneurysm may present as a pulsating neck mass with mass effect, it can cause cranial nerve palsies and hemorrhage. The management options for ICAP include conservative management in young asymptomatic patients, ultrasound guided compression, percutaneous thrombin injection, coil embolization, endovascular stent graft insertion, and surgery.

**Conclusions:** Traumatic aneurysms (TA) are a rare cause of late neurological deterioration in patients with traumatic head and neck injury and should be suspected in all patients who sustain severe injury, especially those presenting with delayed neurologic deterioration. There could be role for delayed repeat imaging in such patients; however, specific guidelines are yet to be established.
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NITROFURANTOIN-INDUCED INTERSTITIAL PNEUMONITIS
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Nitrofurantoin is an antibiotic frequently prescribed for treatment and prophylaxis of urinary tract infections. It is typically well-tolerated with the most common adverse reactions being nausea and headache. Nitrofurantoin-induced interstitial pneumonitis is a relatively rare but serious reaction that should be considered in patients presenting with pulmonary symptoms who have recently taken this medication. Fortunately, with rapid identification and cessation of therapy followed by administration of steroids, the condition is reversible.

A 76-year-old female presented with progressively worsening dyspnea on exertion, cough, and a new oxygen requirement. Her medical history was significant for coronary artery disease and type 2 diabetes. Ten days prior to admission, she was diagnosed with a urinary tract infection and prescribed nitrofurantoin (100 mg by mouth twice daily). During the five days prior to admission, she became increasingly dyspneic while ambulating and she developed a paroxysmal non-productive cough. At the time of presentation, she had four to five word dyspnea and required supplemental oxygen via nasal cannula. While in the emergency department, she received albuterol via nebulizer and IV methylprednisolone, after which she noted significant improvement in her dyspnea. Her CBC showed leukocytosis and eosinophilia, and a chest CT demonstrated a diffuse mosaic attenuation pattern. She was admitted and started on empiric treatment for atypical pneumonia. Based on the eosinophilia and CT findings in the setting of recent nitrofurantoin use, concern arose for possible nitrofurantoin-induced interstitial pneumonitis. A bronchoscopy was performed and leukocytosis with eosinophilia was seen in the bronchoalveolar lavage. At this point, her symptoms began improving, and additional steroids were deferred. She continued to improve and was subsequently discharged without a supplemental oxygen requirement.

Nitrofurantoin-induced lung disease is relatively rare, and initial presentation can be similar to pneumonia or an exacerbation of chronic lung disease. As a recommended first-line therapy for uncomplicated cystitis, nitrofurantoin use will likely expand requiring clinicians to be aware of this adverse reaction. An evaluation with bronchoscopy and BAL can greatly assist in the diagnosis of this process and guide further therapy, as well as help dictate treatment options for future cystitis.

Program Director’s Name: William Shimeall

(indicating review of abstract)
A RARE CASE OF STRONGYLOIDES HYPERINFECTION DIAGNOSED WITH BRONCHOSCOPY. Minkove S, MD, Glick D, MD. University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

*S. stercoralis* is an intestinal nematode endemic in rural areas of subtropical regions that typically leads to mild waxing and waning of gastrointestinal, cutaneous, and pulmonary symptoms. Infection typically occurs after skin penetration by filariform larvae in soil, which then penetrate the skin and migrate hematogenously to the lungs, penetrate the alveolar sacs, ascend the transbronchial tree, and are swallowed. Asymptomatic gastrointestinal infection can persist for years, but in the setting of immunosuppression, can provoke a life-threatening hyperinfection syndrome. Typically, diagnosis is made through stool analysis for larvae, ova, or adult worms, though sampling of other body fluids or organ biopsy can also be diagnostic.

A 37-year-old Honduran man recently diagnosed with brainstem glioma presented with shortness of breath, generalized weakness and non-radiating chest pain. Five months prior to presentation, the patient developed headaches and blurred vision. Two months later, MRI revealed a brainstem mass. He was started on radiation followed by chemotherapy with bevacizumab, temozolide and pulse dose steroids. Ten days prior to admission, the patient underwent his second cycle of this regimen. Subsequently, he developed progressive dyspnea and pleuritic chest pain. On presentation, he was found to be hypoxic to 90% on ambient air with diffuse expiratory wheezes. He was afebrile, with leukopenia but not neutropenia. Chest imaging revealed diffuse ground glass opacities with septal thickening. Despite conservative therapy and broad spectrum antibiotics, his respiratory status worsened. Given his immunosuppression, there was concern for Pneumocystis pneumonia and a bronchoscopy was performed, revealing thin secretions throughout the bronchial tree. Washings from bronchoalveolar lavage showed parasite larval forms morphologically consistent with Strongyloides, as well as rare fungal yeast consistent with Cryptococcus. The patient was started on ivermectin and fluconazole. The patient’s absolute eosinophil count was 0.0 (K/mcl). Four days after the bronchoscopy, the patient decompensated with hypoxic respiratory failure requiring intubation. Ultimately, he was liberated from the ventilator, but decision was made for hospice care given poor prognosis.

This case illustrates an unusual identification of disseminated Strongyloides by diagnostic bronchoscopy. Infected patients placed on immunosuppressive drugs or corticosteroids are at risk for deadly disseminated disease.
Acidemia as a Unique Cause of Cardiomyopathy
Ghazi Rizvi, Asif Surani, Akash Shah, Solomon Seifu, Ricardo Conti

Introduction:
De novo heart failure is a well recognized entity with multiple etiologies. The most common precipitating factors include arrhythmias, valvular dysfunction, and acute cardiac ischemia. Less common etiologies include stress, sepsis, peripartum, and hypertension. In contrast to the well known factors mentioned above we hereby present a case of cardiomyopathy due to prolonged acidemia in a patient with severe diabetic ketoacidosis (DKA).

Case description:
A 39-year-old Black female with latent autoimmune diabetes in adults (LADA) and Graves’ disease presented complaining of protracted nausea and vomiting for one day. The patient reported missing several doses of insulin over the past several weeks due to forgetfulness; she also admitted to non-compliance with diabetic diet. Additionally, she reported polyuria and polydipsia but denied any palpitations or chest pain. She was diagnosed as having severe DKA and thyrotoxicosis. Appropriate management with intravenous fluids, insulin and electrolyte replacement was initiated. She remained severely acidic in spite of the aforementioned measures. Six hours from the time of admission she developed pulmonary edema due to aggressive intravenous fluid hydration. Echocardiography at that time demonstrated an ejection fraction (EF) of 15-20% and severe diffuse hypokinesis. Electrocardiogram (ECG) was negative for signs of ischemia and only demonstrated sinus tachycardia. Eight hours from the time of admission she required intubation due to muscle fatigue and hypoxia. Over the remainder of the day her pH improved with a combination of intravenous insulin, mechanical ventilation, dexamethasone, propylthiouracil, and a sodium bicarbonate infusion. With the resolution of DKA and thyrotoxic state her EF normalized over the next four days. A stress test done on day three did not show evidence of inducible ischemia.

Discussion & conclusions:
Many of the metabolic and vital sign derangements in DKA can contribute to the development of de novo cardiomyopathy including tachycardia, stress and acidemia. Underlying illnesses such as cardiac ischemia or sepsis can also cause cardiomyopathy. We postulate that this patient’s cardiomyopathy was caused by her severe acidosis given the rapid improvement in her systolic function – as determined by EF and fractional shortening - with correction of the acidosis. Other potential causes of cardiomyopathy were effectively ruled out with ancillary testing as follows: wall motion abnormalities seen on echocardiogram were not typical for stress/Takotsubo or sepsis mediated cardiomyopathy. EF was not elevated ruling out high output heart failure, full recovery was observed over a period of 4 days which argues against tachycardia/hyperthyroid mediated cardiomyopathy, and stress test was negative which argues against ischemia mediated cardiomyopathy. Cases like this represent a unique therapeutic challenge as the concurrent cardiomyopathy limits the administration of fluids which is a component of the standard treatment of DKA and as demonstrated by this case, fluid administration can unmask the cardiomyopathy. To identify patients like these we suggest using bedside ultrasounds as a useful addition to the physical exam.
LEVERTICULAR NON COMPACTATION UNMASKS ITSELF AFTER A PNEUMONIA
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INTRODUCTION
Left Ventricular Non Compaction (LVNC) is a rare congenital cardiomyopathy with high morbidity and mortality. Previously diagnosed mainly in pediatric patients, it is emerging as a cause of new heart failure in adults.

CASE
A 35-year old man with known history of asthma, previously employed in the military, presented to the Emergency Department with worsening nonproductive cough, fatigue, pleuritic chest pain, and generalized weakness. On exam he was hemodynamically stable, with bilateral rales on chest auscultation, but no crackles, JVD or peripheral edema. Workup done showed left ventricular hypertrophy on EKG and infiltrates on CXR confirmed with CAT scan consistent with pneumonia alongside cardiomegaly. An echocardiogram showed significant decrease in LV function, EF 35-40%, along with the presence of trabeculae in the left ventricle, most prominent in the apex. The patient denied having had any symptoms of heart failure. The hospital course was further complicated by TIA and the patient was discharged on anticoagulation for lifetime, only to return within a month with symptoms of florid heart failure, after he ran out of medications. He had no significant coronary artery disease on cardiac cath.

CONCLUSION
Despite increasing recognition of LVNC in adults, there is still a significant lag in diagnosis from the time of symptom onset. Our patient had no symptoms for the first 35 years of his life, with florid heart failure set off by a stressor, which was pneumonia. Timely recognition and initiation of a cardiomyopathy treatment regimen can potentially cause early reversal of cardiac remodeling.
WHAT TICKLES THE NOSE CAN BREAK A HEART: STACHYBOTrys, ANTEROGRADE AMNESIA, AND TAKOTSUBO'S MYOCARDITIS

Darish Liske-Doorandish, MD; Sean Shieh, MD; Lyn Camire, MA
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Eosinophilic myocarditis is a significant morbidity associated with hypereosinophilic syndromes (HES), myeloproliferative disorders that promote eosinophil or mediator infiltration of tissues, organs and systems. Though rare, HES can lead to life threatening organ/system collapse, and can remain hidden behind many guises.

Five months after moving into an older home, a 38-year-old woman with asthma and chronic sinusitis stopped her car after leaving work early because she was feeling unwell, unaware of where she was or how she got there. She called her husband who found her parked along her commute route, confused. He took her to the ED. Anxious and tearful, she exhibited acute retrograde and anterograde amnesia and was admitted for altered mental status. Her initial ECGs suggested remote septal infarct but CRP/troponins were elevated. Head CT and MRI excluded acute pathology but revealed extensive sinusitis; CBC showed eosinophil-rich leukocytosis to 16K; LP was negative. She continued to be amnesic and anxious, with chest pain/tightness for 2 days. Repeat ECG revealed septal infarct with troponemia, rising from initial 7.950 to a peak of 11.200 on day 2. TTE revealed an ejection fraction ~60%, absent wall motion abnormality, with mild valvular incompetence. The patient received cardiac catheterization to rule out NSTEMI. Coronary Artery Disease was excluded but findings were consistent with a variant of Takotsubo's cardiomyopathy. Prednisone was initiated. By day 3 her eosinophilia, troponemia, and memory improved. Cardiac MRI characterized “subendocardial hypoperfusion and associated delayed hyperenhancement in anterior and inferior regions... with [apical/septal] thrombus.” Testing showed mold allergy and tests of the home identified Stachybotrys species. Diagnosed with hypereosinophilic myocarditis, the patient remains largely asymptomatic but on ongoing prednisone, and anxious about its long term use and side effects.

Hyperesinophilic inflammatory responses often remain hidden in the open. Given initially mild and varied presentations, they can go long undiagnosed while producing aggregate systemic damage. Awareness of sentinel findings as well as constellations of symptoms may help speed diagnosis and forestall downstream morbidity/mortality.
GASTRIC PLASMACYTOMA AS UNUSUAL CAUSE OF UPPER GI BLEED

Introduction: Plasma cell dyscrasias encompass a spectrum of monoclonal plasma cell neoplastic proliferation including multiple myeloma (MM), monoclonal gammopathy of undetermined significance, & plasmacytomas (bony & extramedullary). Diagnostic criteria help to distinguish each entity. Among plasmacytomas, gastrointestinal (GI) tumors are less common, making GI bleeding an unusual complication.

Case Presentation: 67 year-old Caucasian female presents with abdominal pain for one week associated with nausea & vomiting as well as loss of appetite with 20-lb weight loss over six months. No history of external bleeding. Patient developed severe iron deficiency anemia (IDA, FOBT+) requiring multiple transfusions. Imaging findings included a soft tissue mass right chest wall (CW). Elevated serum total protein levels with an SPEP positive for IgA kappa monoclonal gammopathy. Bone survey negative for lytic lesions with no elevations in serum creatinine or calcium levels. Bone marrow (BM) biopsy positive for 40%-50% plasma cells without any evidence of increased blasts. Work-up for severe iron deficiency anemia revealed two gastric masses & one duodenal ulcer with stigmata of recent bleed & biopsies showing IgA kappa malignant cell proliferation. Biopsy of CW mass revealed atypical plasma cells. At this point, the diagnosis of plasmacytoma was also made. The patient developed overt upper GIB not amenable to endoscopic intervention, so started on palliative radiation. After finishing one course, she decided to stop treatment opting for hospice care. The patient passed away 5 days later.

Discussion: Extramedullary plasmacytoma (EMP) is an uncommon presentation of plasma cell neoplasms. Majority are located in the upper aerodigestive system & less commonly GI tract. Plasma cell neoplasms have potential to progress to MM. Symptoms often depend on tumor location & location along spectrum of disease progression. Our patient did not meet diagnostic criteria for MM as myeloma-defining events (MDE) were absent. Plasma cell proliferation occupying <60% of BM in absence of MDE is more consistent with smoldering myeloma.

Conclusion: It is important to consider GI plasmacytoma in the differential of upper GIB. In patients with monoclonal gammopathy, it is also important to distinguish disease entity & investigate anemia causes in absence of myeloma-defining events & overt GIB. In our patient with FOBT + & severe IDA prompting further work-up, which revealed GI tract EMP as the etiology.

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PNEUMORRHACHIS AS COMPLICATION OF MYCOBACTERIUM KANSASII PNEUMONIA
Joseph Meyer, MD, Member, Johns Hopkins Hospital, Baltimore, MD
Pneumorrhachis, or the accumulation of air in the spinal canal, is an uncommon finding often associated with pneumomediastium. While radiographically alarming, subjects with pneumorrhachis are usually asymptomatic with resolution occurring with treatment of the underlying disease process.
A 52-year-old man with history of HIV/AIDS presented with three months of progressive dyspnea on exertion, productive cough, and 32 kg weight loss. Further review of symptoms revealed one month of odynophagia, pleuritic chest pain, and night sweats. Admission CD4+ lymphocyte count was 2 per mL with HIV viral load was 153,000. Dry chest CT demonstrated multifocal alveolar infiltrates and pneumomediastinum. Given concern of disseminated opportunistic infection, expectorated sputum samples were collected. Antibiotics and antiretroviral therapy were started. By day five, Bactrim was started as empiric Pneumocystis jiroveci therapy given ambulatory hypoxia, beta-D-glucan >500 g/mL, and LDH 275 U/L. As acid-fast stains were negative, bronchoscopic alveolar lavage (BAL) was completed on day 8.
Several hours post-BAL he was emergently intubated for respiratory failure due to worsening right pneumothorax. Repeat chest CT on day 11 showed interval resolution of pneumomediastinum with chest tube in an appropriate position. On day 13, exam revealed increased left subcutaneous air and subsequent chest x-ray showed a new left pneumothorax. A left chest tube was placed. Also on day 13, sputum culture grew Mycobacteria. He was started on RIPE and azithromycin therapy, narrowed to rifampin, ethambutol, and isoniazid on day 15 after his culture grew Mycobacterial kansasii. Given failure to wean the ventilator, repeat dry chest CT on day 25 showed interval enlargement of bilateral pneumothoraces, extensive subcutaneous emphysema, recurrence of pneumomediastinum, pneumoperitoneum, and extraluminal pneumorrhachis. No neurological deficits, headache, or worsened back pain were noted. After discussion of these results, the patient requested to be made comfortable and removed from the ventilator. He passed comfortably the following day.
This case illustrates complications of disseminated Mycobacterial kansasii exacerbated by positive-pressure ventilation resulting in diffuse extra-pulmonic air, most notably in the spinal canal. Despite extensive air in the spinal canal, he remained neurologically asymptomatic. Although rare, it can be seen in patients with extensive subcutaneous or mediastinal air, after trauma (e.g. CPR), or after violent coughing spells.
A THERAPEUTIC CHALLENGE: CONCOMITANT PROLIFERATIVE LUPUS NEPHRITIS AND HEPATITIS C VIRUS INFECTION.
Nehna Abdul Majeed, MBBS; Athmananda Nanjundappa MBBS;
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Membranous nephropathy (MN) is the most common cause of nephrotic syndrome in non-diabetic adults. MN is generally idiopathic, although it has been associated with systemic lupus erythematosus (SLE), hepatitis B virus (HBV) infection, syphilis, hepatitis C virus (HCV) infection, solid tumors, and certain drugs such as nonsteroidal anti-inflammatory drugs.

A 63-year old African American woman with history of chronic HCV infection presented with a one-month history of progressive shortness of breath that worsened with minimal exertion associated with facial and leg swelling. On admission, she was hypertensive and physical exam revealed facial puffiness and bilateral pedal edema. Initial lab work showed elevated creatinine of 1.98 mg/dL (baseline Cr 0.8-1 mg/dL), nephrotic range proteinuria (24 hr urine creatinine was 4.4 gram), and RBCs on urine microscopy. Serological workup showed HCV reactive status with a viral load of 7.3 million IU/mL, serum and urine electrophoreses were negative for monoclonal gammopathy, and anti-nuclear antibody was positive with a speckled pattern and titer of 1:2560. Ultrasound guided renal biopsy and following pathology report showed immune-complex mediated glomerulonephritis with focal proliferation, crescents, diffuse membranous deposits and near full-house immunofluorescence. She was treated with oral prednisone and mycophenolate mofetil. The patient was discharged with close follow up with rheumatology and nephrology as outpatient.

Lupus membranous nephropathy (LMN) typically presents with proteinuria and a normal or slightly elevated serum creatinine. LMN is the form of lupus nephritis (LN) that may present with few or no clinical or serologic manifestations of SLE. Treatment involves immunosuppression with glucocorticoids combined with either cyclophosphamide or mycophenolate mofetil and is usually reserved for patients with poor prognostic factors like persistent proteinuria >3.5 g/day, progressive rise in serum creatinine, and mixed membranous and proliferative lesions on biopsy. Concomitant HCV infection in our patient was a treatment dilemma as liver disease in an immune suppressed patient can rapidly progress to cirrhosis and lack of safety and efficacy studies in treating HCV in patients with severe renal impairment.
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NPPE: A POTENTIALLY FATAL MISSED CONNECTION
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In young healthy patients with no cardiac or pulmonary comorbidities, causes of post-operative pulmonary edema can be challenging to determine. Negative pressure pulmonary edema (NPPE) is an important, but often missed, complication post-extubation with a prevalence of only 0.05%-1%. Common causes include laryngospasm and upper airway obstruction, as can be seen in patients with obstructive sleep apnea or obesity. Additional cases have been reported in patients undergoing facial surgery or young athletic males.

A 24-year-old, thinly-built, previously healthy male with a history of tobacco and marijuana use, presented with gastroenteritis and leukocytosis. He was found to have acute appendicitis per CT imaging and underwent uncomplicated laparoscopic appendectomy. Post-procedure extubation was complicated by laryngeal spasms, hemoptysis, and respiratory distress along with difficulty maintaining adequate oxygen saturation on room air. He had desaturations in the mid 80s on room air requiring placement of 6 liters nasal cannula with improvement in saturation up to low 90’s. Arterial blood gas measurement was unremarkable; however, chest X-ray revealed evidence of bilateral pulmonary congestion. Additional workup including labs, history and examination failed to provide evidence of cardiac disease, pulmonary embolism, or infectious pulmonary disease as an explanation for his symptoms. He was given one dose of IV furosemide and subsequent chest x-ray revealed improvement of pulmonary edema. In the setting of post-operative laryngospasm, chest x-ray findings, and improvement of symptoms with diuresis, the patient was diagnosed with NPPE. His saturations improved, hemoptysis resolved, and, by the following morning, he maintained high oxygen saturation up to 98-100% on room air. He was discharged home the next day without further pulmonary complication.

This case illustrates the importance of recognizing post-extubation NPPE in young patients who lack the typical risk factors or comorbidities. Although a common cause of NPPE is laryngospasm, this diagnostic process can be challenging, as it remains a diagnosis of exclusion. It is important; however, to be aware of this potential outcome in order to implement appropriate management and preventative strategies.
STAPHYLOCOCCUS-ASSOCIATED GLOMERULONEPHRITIS
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Staphylococcus-associated glomerulonephritis (SAG) is an IgA-predominant immune complex-mediated disease that presents concurrently with an active infection and has a relatively poor prognosis. Without proper identification, this disease process may prove to have irreversible effects on renal function.

A 70-year-old man with past medical history of smoking, COPD, and laryngeal cancer status-post radiation and laryngectomy, presented with one week of fever, chills, rigors, worsening shortness of breath, and dry cough. On admission, he was afebrile, tachypneic, tachycardic (100), BP 89/51, with oxygen saturation of 91% on RA. On physical exam he had dry mucous membranes, a 3/6 systolic murmur, bilateral coarse breath sounds, and mild wheezing. Initial labs showed a normal CBC, hyponatremia (127), creatinine 4.9, and BUN 43. Chest X-ray showed no infiltrates or effusions. Blood cultures were sent and he was started on antibiotics for presumptive community-acquired pneumonia, along with IV fluids for AKI. Blood cultures grew methicillin sensitive staphylococcus aureus (MSSA) and antibiotics were switched to cefazolin. By day 5, the patient’s symptoms had improved and the AKI resolved; however, he had another episode of shortness of breath. CT chest done on day 12 showed bilateral infiltrates with a cavitating appearance consistent with septic pulmonary emboli from MSSA bacteremia. With a short course of steroids, his breathing improved; however, kidney function started to decline again. UA showed large blood, RBCs, and RBC casts. C3, C4 and ANA levels were found normal. The patient underwent a renal biopsy, which demonstrated glomerulonephritis. Electron microscopy showed subendothelial hump-like deposits and immunofluorescence microscopy showed predominantly IgA deposits, consistent with SAG. The patient was continued on antibiotics for 6 weeks, after which his kidney function stabilized, but unfortunately never returned to his previous baseline.

SAG presents with elevated creatinine, hematuria, RBC casts, proteinuria and/or edema. Predisposing factors include diabetes, malignancy, alcoholism, and intravenous drug abuse. Renal biopsy proves to be helpful to rule out other differentials. Treating the underlying infection helps in resolution of the disease; however, some patients may never return to their baseline kidney function and may progress to end stage renal disease without prompt detection.

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A CASE OF INFECTIVE ENDOCARDITIS PRESENTING AS RENAL INFARCTION

Introduction: Infective endocarditis (IE) typically presents with highly variable clinical manifestations. At times, it may present with systemic complications due to septic embolization. This case report illustrates a patient who presented with non-specific constitutional symptoms as well as flank pain and urinary symptoms who was subsequently diagnosed with renal infarction secondary to infective endocarditis.

Case Presentation: A 61-year-old Caucasian male with a past medical history of rheumatic fever (RF) presented with complaints of weakness and generalized fatigue associated with fevers, chills, night sweats, and forty-five pound weight loss. He also had increased urinary frequency, incontinence, hesitancy, nocturia and constant severe left flank pain for about three weeks. Physical exam was significant for a cachectic appearing gentleman with tachycardia and irregular heart rhythm, grade 3/6 holosystolic murmur in mitral area as well as severe right CVA tenderness. Laboratory work-up revealed leukocytosis (WBC count of 15.2), elevated NT-proBNP (14626), rising troponin (as high as 1.010), and hematuria but no UTI on urine analysis. EKG revealed atrial fibrillation with rapid ventricular rate. CT Abdomen and Pelvis with IV contrast was performed to investigate patient’s left flank pain with hematuria. This revealed two wedge-shaped left renal lesions highly suggestive of renal infarct. Considering his history of RF and the murmur heard on physical exam, an echocardiogram was obtained which revealed a mobile 1.2cm x 0.6cm vegetation on the right coronary cusp of the aortic valve along with severe aortic regurgitation and rheumatic changes of the mitral valve with severe mitral regurgitation. The patient was subsequently diagnosed with bacterial endocarditis and treated with Vancomycin and Piperacillin-Tazobactam. Blood cultures grew streptococcus mutans. The patient was later transferred to University of Maryland for cardiothoracic surgical evaluation of his valvular lesion.

Discussion: IE typically presents with highly variable clinical manifestations and is at times associated with systemic complications due to septic embolization which may be present in up to 25 percent of patients. This typically presents as infarction of lung parenchyma, kidneys, spleen, and other organs. This case presents a patient with constitutional symptoms as well as flank pain, and urinary symptoms who was found to have wedge-shaped renal lesions suggestive renal infarction secondary to IE. Renal infarction is rare and occurs mostly in 3 settings: cardioembolic disease, renal arterial injury, and hypercoagulability. Hence, when it is found in association with non-specific constitutional symptoms, one should consider IE as its cause.

Conclusion: Cardioembolic disease is the most common cause for renal infarction and signs, symptoms and radiologic findings of renal infarction along with constitutional symptoms should prompt physicians to consider the important diagnosis of IE.

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PERI-PROSTHETIC PSEUDOTUMOR

Peri-prosthetic pseudotumor is a process where pseudotumors are generated by metal ion debris from wear of a metal-on-metal hip prosthetic. Recent case studies have reported this in non-metal-on metal prostheses as well.

A 64-year-old physically active man presented to the ER with left leg swelling and discomfort for several weeks. His past medical history included resurfacing in February 2014 of a prior left total hip arthroplasty. Several weeks of a non-healing wound was treated with antibiotics for suspected cellulitis. Initial work-up was negative for deep vein thrombosis. There was minimal improvement after a course of Bactrim and oral steroids. After 2 weeks he presented back to the ER, denying any recent illness and having maintained his daily gym cycling.

On examination, there was mild left groin and femoral edema, a dusky color, slight warmth, and normal pulses. He had normal passive range of motion with a subtle click with left hip flexion and extension. Labs, including inflammatory markers, were unremarkable except for a mild elevation of creatinine of 1.7 that was attributed to the recent Bactrim use. X-rays were negative. CT Angiogram showed a 12x8x8 cm multi-loculated complex fluid collection in the left iliopsoas. Abscess was suspected, and orthopedics was consulted. Patient underwent interventional radiology drainage of the left hip joint and fluid collection. Synovial fluid was bloody with a white blood cell count of 532.

The patient was transferred urgently to a tertiary orthopedics center for further treatment. Synovial fluid and fluid collection sample analysis was done using a novel assay that determines the amount of alpha-defensin. Infectious workup was negative, including synovial fluid, fluid collection, alpha-defensin, and Lyme testing. Serum cobalt and chromium levels, checked by the specialist at the tertiary center, were normal. Our patient is followed closely to determine the need for eventual replacement. It was concluded that his sterile fluid collection was from a peri-prosthetic pseudotumor caused from irritation of soft tissue from metal ions from his left hip replacement resurfacing.

This case illustrates how symptoms of peri-prosthetic pseudotumor mirror those of an infected abscess. Recognition of this diagnosis is important to facilitate rapid treatment of an affected joint. Early recognition allows definitive treatment which consists of total hip arthroplasty and hopefully limits unnecessary long term antibiotics. As more of the newer, hip resurfacing type implants are used, awareness of this diagnosis is beneficial for medical professionals.
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STROKE MIMIC OR MIGRAINOUS INFARCTION?

A 65-year-old female presented to the Emergency Department with a headache associated with visual disturbance. The patient reports a throbbing, pounding left-sided headache, spanning the left forehead to left mastoid, and rated it as 10/10 in severity. Patient reports that bright lights, motion, and cold temperature worsen the headache. Patient also endorses a vague red haziness to objects in her right visual field, as well as mild nausea. Patient denies dysarthria, facial weakness, dysphagia, extremity weakness, difficulty ambulating, and aura. 10 days prior to her visit, patient visited the ED for similar complaints, but was discharged after the headache improved with analgesics. She reports experiencing these headaches every day since, with today’s headache being the most severe. Upon further questioning, patient recalls similar headaches spanning back 30 years ago, but has never received a diagnosis. Her past medical history is significant for well-controlled hypertension, poorly-controlled type 2 diabetes mellitus (A1c > 14%), poorly-controlled hypercholesterolemia & hypertriglyceridemia, and anemia. She smokes ½ packs per day. Physical examination was notable only for a right hemianopia on visual field testing. Laboratory and ECG tests were normal. Head CT did not reveal any acute bleeding or edema. A complex migraine was suspected, and she was treated with IV magnesium sulfate, IV ketorolac, topiramate, and metoclopramide. Her symptoms improved. Given her visual findings, she was admitted to observation for further evaluation with MRI.

The next day, the patient underwent MRI scan of her brain, which demonstrated hyperintensity on DWI imaging and hypointensity on ADC mapping in her left posterior occipital lobe, consistent with acute cerebrovascular accident. She was started on aspirin and statin, as she was outside the window for tPA. Additionally, she was started on topiramate and magnesium oxide, received diabetes education and smoking cessation counseling, and discharged home.

Stroke can present like a migraine, migraine can present like a stroke, migraine can cause stroke, and both can happen independently. Additionally, imaging can be equivocal. This patient had undiagnosed migraine headache as well as multiple risk factors for atherosclerotic stroke: hypertension, smoking, diabetes, and dyslipidemia. These risk factors also increase her risk for infarct during acute migraine. The clinician is cautioned to consider stroke in patients with migraine and multiple atherosclerotic risk factors.
Hypoglycemia: A diagnostic dilemma in the elderly
R Jagata M.D., H Nguyen, M.D.

Introduction: 90% Insulinomas are solitary benign tumors; they are more common in women and the highest incidence is in middle-aged adults. Symptoms attributable to hypoglycemia due to insulinoma may occur over a period of 25 years or more before the offending lesion is identified.

Case: An 88-year-old male, with multiple comorbidities, was found unconscious at an outside facility, blood glucose at the time was 32 mg/dL. He was treated with two glucagon injections which raised blood glucose to 102 mg/dL. The night before presentation the patient had eaten a full meal and had an uneventful evening. Medical history was significant for multiple previous episodes of hypoglycemia that required hospitalization and were attributed to different etiologies over the years such as sepsis and levofloxacin. Review of systems was significant for fatigue, weakness, dizziness upon standing, frequent falls, and unexplained syncopal episodes. Physical examination was unremarkable. Laboratory investigations revealed HbA1c 3.6%, random cortisol 23.5 mg/dL, random insulin 264 uU/ml and 345 uU/ml ( ), C-peptide 25300pmol/L and 20700pmol/L (80-350pmol/L), proinsulin 498pmol/L (<22pmol/L), insulin antibodies <0.04u/ml. Computed tomography (CT) of the abdomen was remarkable only for a stable small right posterior hepatic lesion. Endoscopic ultrasound (EUS) of the pancreas was recommended to evaluate for lesions that could cause hypoglycemia. EUS revealed a 1.2cm round hypoechogenic lesion between the head and uncinate process. Cytology from fine needle aspiration revealed cells positive for the neuroendocrine markers synaptophysin and chromogranin. The clinical picture, serum markers, and pathologic findings were consistent with a diagnosis of insulinoma. Unfortunately, he was deemed to be a poor surgical candidate given his age and comorbidities. He was treated medically with diazoxide. He responded well to treatment.

Discussion: Hypoglycemia induced by insulinomas is usually episodic and provoked by fasting; snacking helps to avoid hypoglycemia. Although rare if the patient presents with symptoms consistent with Whipple’s triad (fasting hypoglycemia [<50 mg/dL], symptoms of hypoglycemia, and immediate relief of symptoms after the administration of glucose) work up for insulinoma should be pursued. Clinically significant insulinomas can be 10 to 20mm in size at the time of diagnosis and can be easily missed by CT scan. Endoscopic ultrasonography has 89.5% sensitivity and 83.7% accuracy for the detection of insulinomas not localized with noninvasive tests; it is especially helpful in diagnosing insulinomas of the head and body of the pancreas. The combination of clinical, biochemical and imaging tests is required to confirm the diagnosis. Staining with immunohistological reagents like synaptophysin, neuron-specific enolases, and chromogranin, have been used as general markers of neuroendocrine tumors. Insulinomas are best treated surgically. Medical management is with diazoxide plus a thiazide diuretic; long-acting and synthetic somatostatin preparations can be used as well.

Conclusions: Clinicians should have a high suspicion for insulinoma in patients presenting with recurrent hypoglycemia. Delays in diagnosis are common because there is often misattribution of symptoms to psychiatric, cardiac, infectious or neurological disorders. Diagnosis and can be easily missed by CT scan due to the small size of the tumors. EUS if the follow up imaging of choice when noninvasive tests are negative.
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HEYDE’S SYNDROME AND RECURRENT GASTROINTESTINAL BLEEDING
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INTRODUCTION: Gastrointestinal bleeding due to angiodysplasias in patients with aortic stenosis is termed as “Heyde’s syndrome”. We are reporting a case of recurrent gastrointestinal bleeding, in which capsule endoscopy played a key role in the identification of angiodysplasias and diagnosis of Heyde’s syndrome.

CASE REPORT: An 85-year-old male with history of duodenal diverticulum, presented to the hospital with 1-day history of melena. On week prior the index admission, he was admitted for hematemesis and underwent prophylactic embolization of the gastroduodenal artery, as upper endoscopy (EGD) and colonoscopy were negative. On admission, he was hemodynamically stable. He was noted to have an ejection systolic murmur (III/VI) in the aortic area with radiation to the carotids and epigastric tenderness. Lab results: hemoglobin - 8.2 g/dl, hematocrit - 25.9% with normal platelets and coagulation studies. He underwent EGD and colonoscopy during the index admission and the source of bleeding could not be identified. With the history of recurrent gastrointestinal bleeding and presence of an ejection systolic murmur in the aortic area, Heyde’s syndrome was suspected. Echocardiogram showed severe aortic stenosis with valve area of 0.6 square centimeter and mean aortic valve gradient of 37 mmHg. Capsule endoscopy revealed multiple arteriovenous malformations in the proximal to mid jejunum, consistent with the diagnosis of Heyde’s syndrome. After several blood transfusions, his hemoglobin stabilized and he was discharged home with outpatient cardiology follow-up for aortic valve replacement, as he had New York Heart Association (NYHA) class III symptoms, in addition to the recurrent gastrointestinal bleeding. He underwent trans-catheter aortic valve replacement (TAVR) and has not had any further episodes of gastrointestinal bleeding.

DISCUSSION: Angiodysplasias are dilated, thin-walled tortuous vessels in the mucosa and submucosa of the gastrointestinal tract. They are the second leading cause of lower gastrointestinal bleeding in elderly patients. Heyde’s syndrome should be suspected in patients with recurrent gastrointestinal bleeding and aortic stenosis.
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STICKY PSEUDOMONAS STRIKES
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_Pseudomonas aeruginosa_ is an uncommon pathogen of community-acquired pneumonia in immunocompetent patients. However, infections with this organism are associated with a high mortality rate. An 83 year-old female with a remote history of breast cancer, hypertension, hypothyroidism, and osteoporosis, presented with a 20-pound unintentional weight loss over four months, fatigue, and cough. On presentation to the ED, she was afebrile, tachycardic (104 bpm), mildly hypertensive (BP106/62), and saturating 92% on room air. Labs were significant for a neutrophilic-predominant leukocytosis of 32.8 with an ANC of 28, normocytic anemia (Hg 9.1), hyponatremia (Na 127) and an elevated creatinine of 1.2. Outpatient records demonstrated a recent bronchoscopic culture that grew ciprofloxacin-sensitive _Pseudomonas putida_ and prior to admission, she had completed an 8-day course of Ciprofloxacin with mild improvement of her symptoms. Her chest CT on admission showed interstitial fibrosis, as well as new cavitory lesions, as compared to a previous scan four months prior. She was empirically treated with vancomycin, piperacillin-tazobactam, and cefepime. To further confirm the diagnosis and to ensure no underlying malignant process was apparent, a CT-guided fine-needle aspiration and later open wedge biopsy were obtained, which demonstrated no signs of malignancy. While blood cultures were negative, sputum cultures consistently grew _Pseudomonas putida_, which did not match the clinical course. Therefore, a sample was sent out for further testing, which demonstrated growth of a mucoid-type _Pseudomonas aeruginosa_. In follow-up three months after discharge, on a prolonged antibiotic course with inhaled tobramycin and IV aztreonam, her chest CT demonstrated significant improvement of both the cavitory lesions as well as the diffuse infiltrates, although she continued to have bronchiectasis and bilateral nodularity.

This case illustrates an unusual subacute presentation of mucoid _Pseudomonas aeruginosa_ organizing pneumonia in an immunocompetent patient, which could have easily been missed due to the misidentification of the original cultures. While this patient had mild bronchiectasis prior to this infection, she had no other significant risk factors for a pseudomonal infection. As _Pseudomonas aeruginosa_ pneumonias carry a high mortality rate, it is important to maintain a high level of clinical suspicion.
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An Unusual Case of Calciphylaxis Presenting as Tender Indurated Rash

A 44-year-old woman was admitted to the hospital for altered mental status after missing dialysis sessions and, once stabilized, she reported several months of a waxing and waning chronic, tender indurated rash on her abdomen that had now spread to her bilateral inner thighs. She had a past medical history of end stage renal disease secondary to biopsy-proven AA amyloidosis and is dependent on hemodialysis, AIDS (recent CD4 count of 27) not on anti-retroviral therapy, systolic heart failure, untreated Hepatitis C, and active methadone maintenance therapy for opioid addiction. She noted acute tenderness to palpation on her abdomen over two distinct areas of dusky induration overlying the right and left lower quadrants. The next morning, the induration had worsened and extended over the entire abdomen, her flanks, and her bilateral thighs. Over the next few hospital days, these areas worsened from erythematous induration to becoming violaceous with areas of skin breakdown around a skin biopsy site.

Nephrology, infectious disease, and dermatology were consulted and nephrology suggested early stage calciphylaxis, and indeed her phosphate was elevated to 9 mmol/L on admission. Dermatologic biopsy and histologic examination confirmed a diagnosis of calciphylaxis. She was started on high-dose non-calcium based phosphate binders three times per day and IV sodium thiosulfate administered with hemodialysis.

Calciphylaxis, or calcific uremic, arteriolopathy is a rare and highly morbid syndrome characterized by painful, indurated nodules appearing on adipose-rich tissue. The lesions can vary from violaceous mottling, erythematous papules or plaques, or livedo reticularis to more advanced stage lesions with stellate purpuric configuration with central areas of cutaneous necrosis. Prevalence is between 1-4% of dialysis patients and is associated with up to 80% mortality, most often from sepsis from super-infection of skin ulcerations. Current therapeutic modalities include cessation of steroid administration or warfarin therapy if either are present, excellent wound care, surgical debridement, normalization of calcium and phosphate levels, and sodium thiosulfate.

This case demonstrates the importance of having a high index of suspicion for calciphylaxis in patients with end stage renal disease as the lesions can appear rapidly and be variable in appearance. In addition, the diagnosis and therapy benefits from an interdisciplinary approach such as nephrology, infectious disease, and dermatology to aid in optimizing wound care, balancing calcium and phosphate levels and initiating therapy with sodium thiosulfate and/or oxygen therapy.
BIG MUSCLES, DILATED HEART: EFFECTS OF ANABOLIC STEROIDS ON A BODYBUILDER

Competitive bodybuilders are known to use anabolic steroids (AS) to gain a competitive edge. Reported complications of AS include hepatic adenomas and dilated cardiomyopathy. We report the case of an AS user with right upper quadrant (RUQ) pain who was found to have dilated cardiomyopathy with reduced ejection fraction (EF).

Our patient is a 28 year old competitive bodybuilder with a past medical history of sleep apnea who presented with acute pain, nausea, and emesis during a weight-lifting workout. He reported 2-3 years of AS use without tobacco or illicit drug use. Family history was negative for sudden cardiac death, coronary disease, stroke, or other cardiomyopathy. On arrival, exam was notable for sinus tachycardia, a tense abdomen, and RUQ pain. Labs showed leukocytosis and elevated transaminases. Abdominal imaging demonstrated a large subcapsular and intraparenchymal hematoma. He was diagnosed with a ruptured hepatic adenoma, likely secondary to AS use and underwent two embolization procedures.

During his stay he required isotonic fluid resuscitation and red blood cell transfusions, after which he developed an oxygen requirement and noninvasive ventilation. Radiography demonstrated bilateral patchy infiltrates and cardiomegaly concerning for transfusion associated circulatory overload. Transthoracic echocardiography (TTE) demonstrated an EF=30-35%, moderate-severe global hypokinesis of the LV, dilated right ventricle (RV), and moderately dilated left atrium (LA) and right atrium (RA). Evaluation of the etiology was negative for Hepatitis B, Hepatitis C, HIV, and thyroid disease. There was no evidence for inducible ischemia by PET/CT and MRI was negative for abnormal morphology or scar/fibrosis. It was thought his dilated cardiomyopathy was due to AS use. He was diuresed and started on a heart failure medication regimen. Repeat TTE after 5 months demonstrated persistent LV and RV dilation, mild global hypokinesis of the RV, and moderately decreased LV systolic function with an EF=35-40%.

Our patient presented with subcapsular hematoma and hepatic adenomas. His dilated cardiomyopathy was only uncovered after acute respiratory symptoms in response to transfusions. There have been a number of separate case reports of hepatic adenomas or dilated cardiomyopathy and systolic heart failure in those taking AS. With discontinued use of AS, EF has been reported to recover, but often does not improve fully. Although rare, our case suggests that it is important to consider and possibly screen for other organ complications from AS use in a patient presenting with one clinical scenario. Doing so can prevent
HEMODYNAMIC MANAGEMENT IN A PATIENT WITH PENTOLENOLOGY OF FALLOT AND SEPSIS. Saeed F, MD, Sahbaz J, MD, Huang S, MD, Fisher S, MD. University of Maryland School of Medicine and Baltimore VA Medical Center, Baltimore, MD.

Tetralogy of Fallot is a congenital heart disease that is characterized by four anatomic abnormalities in the heart: pulmonary artery stenosis, overriding aorta, right ventricular (RV) hypertrophy, and ventricular septal defect (VSD). When a fifth abnormality, such as an atrial septal defect (ASD), is present the condition is called Pentoleology of Fallot. These anatomic abnormalities cause significant changes in normal physiology: RV outflow obstruction leads to right-to-left shunting across the VSD and subsequent hypoxia and cyanosis. Definitive treatment is with surgical repair although prior to surgical repair, medical therapy can be used. Oxygen may be given to increase pulmonary vasodilatation and systemic vasoconstriction. Intravenous beta blockers are given to relax the RV outflow tract and promote forward pulmonary blood flow, thus reducing shunting. Intravenous phenylephrine is given to reduce systemic afterload, further reducing right-to-left shunting.

A 52-year-old man with no known past medical history presented with right thigh necrotizing fasciitis. He was found to be hypoxicemic and tachycardic and an echocardiogram revealed a large secundum ASD, VSD, overriding aorta, right ventricular outflow tract (RVOT) muscle bundles with obstruction, severe pulmonary stenosis and RV hypertrophy with preserved RV function. Following surgical debridement, he became febrile with worsened hypoxemia and tachycardia that improved on esmolol, phenylephrine, and vasopressin. The patient underwent multiple surgical debridements and had similar changes in clinical status following each procedure, thought secondary to peripheral vasodilation caused by transient bacteremia and cytokine release. Phenylephrine was used post-operatively to increase peripheral vasoconstriction and reduce right-to-left shunting. Rather than titrating to blood pressure, phenylephrine was titrated to oxygen saturation of >85%. Esmolol was simultaneoulsy titrated to control tachycardia. The patient was weaned from the above medications shortly after each surgical procedure.

This case illustrates the abnormal hemodynamics and oxygenation that are present in a patient with Pentoleology of Fallot. Such a condition presents unusual challenges in the treatment and management of other medical conditions such as infection and sepsis.
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HIV-ASSOCIATED PRIMARY ARTERIAL THROMBOSIS
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A variety of vasculitic pathologies have been associated with HIV. The
majority of reports describe pathology of the small to medium-sized
arteries; however, cases of large vessel arterial thrombosis have also
been described. We present a case of suspected HIV-associated primary
arterial thrombosis.

A 61-year-old non-smoking male with newly diagnosed HIV/AIDS
(CD4 count of 59 cells mm⁻³) presented with severe left foot pain. The
patient was noted to have sensory loss, a cool foot, and a non-
palpable dorsalis pedis pulse. Angiography revealed a totally occluded
proximal left superficial femoral artery extending to the popliteal artery.

Maximal medical therapy was unsuccessful and a left femoral artery to
peroneal artery bypass with a right saphenous vein graft was performed.
Thereafter, dry gangrene of multiple toes was noted and transmetatarsal
amputation was performed followed by multiple revisions. Five days
following the initial bypass, the graft was found to be completely
thrombosed necessitating a below the knee amputation. A trans-
esophageal echocardiogram revealed lack of intracardiac thrombus, no
patent foramen ovale, and normal cardiac valves. A MRI of the thoracic
aorta demonstrated no intraluminal thrombus, hematoma, or dissection.
Furthermore, the results of an extensive hypercoagulable workup did not
suggest a cause of thrombosis and the pathologic specimen was notable
for absence of significant arterial calcification.

Primary large vessel arterial thrombosis associated with HIV is thought
to represent a distinct subset of vasculopathy in HIV-positive patients.
The most striking feature in these often young patients is the normality
of the arterial tree proximal to the thrombosed arteries. Patients also
tend to have low CD4 counts, an average of 255 cells mm⁻³ in one case
series. Most patients present with advanced tissue necrosis and non-
viable limbs. While aggressive surgical therapy is recommended, limb
salvage is frequently unsuccessful in this subset of patients.

This case describes a patient with HIV/AIDS who presented with acute
limb ischemia not likely attributable to other etiologies. This patient's
vasculopathy is suspected to represent the distinct clinicopathologic
entity of HIV-associated primary arterial thrombosis.

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Simultaneous Presence of Angioedema and Dystonia Associated with Risperidone Use

Dystonia and angioedema are both associated with significant morbidity and mortality, especially if not recognized and treated early. We herewith report a case in which both phenomena occurred concurrently with administration of risperidone, a simultaneity that has not been heretofore reported.

A 40 year old paranoid schizophrenic male presented to the emergency department with progressively worsening facial stiffness, lip swelling, inability to open jaw with resultant drooling for a duration of 2 days. This was associated with increasing stiffness in all limbs, which precluded ambulation, and restricted movements of the upper extremities. Of note, he was recently discharged from the psychiatry inpatient service after treatment of suicidal ideation with associated psychotic features. His regimen of antipsychotic medication was changed to risperidone during his psychiatric inpatient stay.

His airway was never compromised and vital signs remained stable. The risperidone was stopped, and he was treated with intravenous diphenhydramine (antihistamine), which is utilized for its high grade anticholinergic activity to counteract the dystonia. His levels of complement activity (C3, C4), C1 esterase, and CPK were all normal. The stiffness and angioedema progressively improved over the course of several days, and the patient was managed on an alternate antipsychotic without any complications.

Acute dystonic reactions associated with antipsychotics shows a greater preponderance in young male patients. This is particularly true of the newer atypical antipsychotics, as was the case in our patient. With respect to the association of angioedema with atypical antipsychotics, rare cases have been reported in the medical literature. As risperidone is a widely prescribed antipsychotic, angioedema appears to be a rare adverse event. The biochemical mechanisms for these two phenomena are distinct, and there does not appear to be a plausible biochemical basis for the occurrence of the two reactions simultaneously.

This case highlights a novel simultaneous occurrence of two potentially ominous clinical entities associated with the institution of risperidone, the prompt recognition of which is central to precluding significant morbidity.
THE RARE DIAGNOSIS OF CHOLECYSTO-BILIARY AND CHOLECYSTO-
DUODENAL FISTULA IN ONE: MIRIZZI SYNDROME TYPE VA
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Introduction: Mirizzi Syndrome (MS) is a rare complication of cholelithiasis. It is characterized by gallstone impaction of the infundibulum of the gallbladder (GB) or cystic duct, causing extrinsic compression of the common bile duct (CBD) and obstruction of the hepatic duct. We describe here an exceptionally rare case of Type V MS with cholecysto-biliary fistula, cholecysto-duodenal fistula, and painless jaundice.

Case Presentation: A 50-year-old Caucasian female with a past medical history of GERD presented with three days of yellowing skin and darkened urine. Prior to this, she endorsed two weeks of occasional postprandial nausea, bilious emesis and fatigue. She reported no abdominal pain, fever, pruritus, or change in bowel or bladder habits. Physical examination found notably icteric skin, sclera and mucous membranes in the absence of abdominal tenderness or hepatomegaly. Work up revealed elevated total bilirubin 7.3 mg/dL, alkaline phosphatase 1,343 U/L, AST 413 U/L, and ALT 450 U/L. Abdominal ultrasound and CT visualized a 3 mm GB wall, 1.2 cm dilated CBD, hepatic steatosis, gallstones, and biliary tree obstruction at the proximal extrahepatic bile duct level. ERCP displayed a possible cholecysto-duodenal fistula, and a filling defect seen in the distal CBD suggesting extrinsic compression from the cystic duct. A CBD and pancreatic duct stent were placed at this time. Laparoscopy revealed a fibrosed GB with dense subhepatic adhesions. The cholecysto-duodenal fistula was identified and divided using an endoscopic stapler. The cystic duct was unable to be clearly identified and therefore a laparotomy was performed. Upon visual and tactile inspection, the cystic duct was found to be obliterated with a cholecysto-biliary fistula clearly identified. Subtotal cholecystectomy was performed using an endoscopic stapler, leaving behind a portion of the GB wall as replacement for the defect in the CBD. Inspection of the GB revealed five gallstones, the largest measuring 3 cm. A drain was left behind in the GB fossa. The patient’s hyperbilirubinemia and transaminitis gradually resolved and imaging revealed no evidence of biliary leak.

Discussion: As it affects less than 0.3% of patients with cholelithiasis, signs of obstructive, painless jaundice do not typically lead to a diagnosis of MS V. This case demonstrates the need for multi-modality workup in order to properly identify these patients. Imaging in this case was able to preoperatively suggest the diagnosis, but ultimate classification often requires laparotomy with careful visual and tactile inspection of the involved anatomy.

Conclusion: Mindful interdisciplinary collaboration is crucial to recognize and appropriately treat the various types and complications of MS, especially in rare cases like MS V. Laparoscopic surgery can be attempted in these patients, but if there is any question about aberrant anatomy, open surgery should be performed.
Atypical presentation of ascites in AL Amyloidosis
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Introduction:
AL Amyloidosis is a systemic disease characterized by the deposition of amyloid fibrils which are misfolded aggregates of monoclonal immunoglobulin light chains in the heart, kidneys, GI tract, skin, peripheral nervous system and liver. Symptoms may differ based on the type of protein and also the organ involved.

Case Description: A 66 yo M presented with a seven week history of recurrent abdominal distention and marked weight loss. On examination his abdomen was non-tender, moderately distended with hepatomegaly (mass in the RUQ and epigastric area 3-4 inches below the xiphisternum) and positive shifting dullness. No stigmata of liver failure or scleral icterus were noticed. Labs were significant for polycythemia, mildly elevated transaminases, elevated GGT and ALP (>700 IU/L). Normal Bilirubin. CEA was mildly elevated to 5.5. CT abdomen showed moderate ascites and mesenteric edema. No evidence of bile duct dilation or splenomegaly was noticed. Ascitic fluid cytology was negative for malignancy. Echocardiogram showed moderate concentric left ventricular hypertrophy and EKG showed low voltage which raised the suspicion of infiltrative cardiomyopathy. 24-hour urine collection showed nephrotic range proteinuria. SPEP showed a M spike of monoclonal IgG lambda. Bone marrow biopsy showed 15% plasma cells. Liver biopsy confirmed extensive involvement with amyloidosis on congo red staining. Chemotherapy was initiated. Unfortunately, he survived only for a month after the diagnosis and died of massive GI bleed and hemoptysis.

Discussion: Although ascites presents in about 10-20% cases of systemic amyloidosis, it is rarely reported as the primary presenting sign. Our patient did not have any typical features of chronic liver disease like jaundice or impaired synthetic function. This could be most likely due to sinusoidal type of portal hypertension caused by the reduction of vascular space of hepatic sinusoids by massive perisinusoidal amyloid deposits. It also illustrates that diagnosis of AL amyloidosis requires a high degree of suspicion as the presenting clinical features can be subtle and nonspecific causing delayed diagnosis.
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WHEN A GI BLEED WON’T STOP
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Sinistral (left-sided) portal hypertension is responsible for less than 1% of upper gastrointestinal (GI) hemorrhages. The mechanism of action is typically venous compression in the left portal venous system. This causes increased back pressure in the portal system resulting in the formation of gastric varices raising the risk of bleeding.

A 53-year-old man with history of alcoholism, early cirrhosis (MELD score of 11), non-bleeding esophageal varices, and recurrent alcoholic pancreatitis, presented with 2 days of hematemesis and 1 day of bright red blood per rectum. He denied any recent NSAID or alcohol use. The patient looked pale, but was normotensive in the ED. Labs revealed hemoglobin of 6.0, prompting treatment with pantoprazole and octreotide, and transfusion of 1 unit of PRBCs. The patient was transferred to the ICU where he continued to have intermittent hematemesis with hemoglobin continually below 7.0, requiring an additional 12 units of PRBC over the next 2 days. EGD performed on hospital day 2 demonstrated stable non-bleeding esophageal varices and a large blood clot in the body of the stomach that was assumed to be covering the source of the bleed. No active bleeding was identified and no interventions were performed. The patient continued to have hematemesis and a subsequent EGD was performed 1 day later that showed small gastric varices with bleeding at the location of the previously visualized clot. CT of the abdomen showed splenomegaly and possible splenic vein thrombosis for which the patient underwent a successful splenectomy on hospital day 5. Post-operatively, the patient’s hemoglobin stabilized and no further blood transfusions were required.

Sinistral portal hypertension usually results in reversal of flow throughout the gastric veins, creating varices. The majority of cases involve some type of pathology in the pancreas given the close proximity to the splenic vein. Treatment typically involves treating the underlying cause along with a splenectomy, which decreases inflow into the portal system. The overall prognosis is dependent on the underlying pathology, with malignancies presenting a poorer prognosis. Despite representing less than 1% of upper gastrointestinal bleeds, it is critical to note that conventional methods of treatment for GI bleed (EGD, banding, PPI’s) are not effective treatments for sinistral portal hypertension and that this condition will require surgery to correct.
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Introduction: Distal (type 1) renal tubular acidosis (RTA) is an uncommon disorder; it results from a defect in urine acidification in the distal tubule due to impaired excretion of hydrogen ions.\textsuperscript{1,2} Symptoms are usually nonspecific. Early diagnosis and appropriate treatment are important as the resulting electrolyte and acid-base imbalances can be life threatening.\textsuperscript{2}

Case: A 44-year-old female with type 2 diabetes presented complaining of severe generalized weakness for 3-4 days. She was supposed to be on insulin; however, since she was feeling unwell she had not been taking any of her medications. She denied nausea, vomiting, and diarrhea. Physical examination was remarkable for tachycardia, frail appearance, drowsiness (although she was able to converse), and dry oral mucosa. Laboratory investigations showed: WBC 13,800/microL, sodium 123 mEq/L, blood glucose 1200 mg/dL, potassium 1.5 mEq/L, bicarbonate 4.4 mEq/L, anion gap (AG) 16 mEq/L, lactic acid 2.40 mmol/L, serum creatinine 1.4 mg/dL, serum osmolality 338 mOsm/kg, and beta-hydroxybutyrate 0.14 mM. She was diagnosed as having hyperosmolar hyperglycemia without ketoacidosis, mild acute kidney injury (AKI), metabolic acidosis, and pseudohypokalemia. The etiology of her metabolic acidosis was unclear; she denied alcohol use. She did not have significant uremia, and was not taking any medications that could cause metabolic acidosis. Patient was aggressively treated with intravenous potassium replacement, hydration, bicarbonate drip, and subcutaneous insulin. Intravenous insulin was not used given her profound hypokalemia. Over the next 24-48hrs symptoms started to improve and lactic acid level normalized; however, bicarbonate level continue to be low (8–10 mEq/L) despite normalization of the AG (12 mEq/L). At this point the possibility of an RTA was entertained. Urine studies revealed: pH 6.0 and urine anion gap 11.5 mEq/L. Additional investigations ruled out several potential etiologies including autoimmune disorders (SLE, Sjögren’s syndrome), nephrocalcinosis/hypercalcuiuria, obstructive uropathy, and medications. Patient was diagnosed with idiopathic distal renal tubular acidosis. She was treated with bicarbonate citrate and ACE-I for her acidosis and to support her potassium level respectively. Hyperglycemia and AKI resolved; potassium and bicarbonate levels normalized. She was eventually discharged and scheduled to follow up with PCP, nephrologist, and endocrinologist.

Discussion: Distal (type 1) RTA is an uncommon disorder. Besides idiopathic, major causes are autoimmune diseases and hypercalciuria. Clinical manifestations are sometimes nonspecific and diagnosis can be challenging. The presence of distal RTA should be considered in any patient with unexplained normal AG metabolic acidosis as illustrated in this case. Urine pH and ammonium excretion (represented by urine AG) play crucial role in the diagnosis of this disorder.\textsuperscript{3,4} Electrolyte imbalances, especially hypokalemia, should be recognized and appropriately corrected as these could be life threatening. This case highlights the importance of obtaining a thorough history and investigations for normal AG metabolic acidosis, identifying early electrolyte imbalances (especially hypokalemia), doing diligent explorations to look for the cause of the RTA, and in certain situations (such as in this case) avoiding the use of intravenous insulin drip to manage severe hyperglycemia in the setting of severe hypokalemia.
DROWNING IN BLOOD, NOT WATER
Himanshu Rawal, MD; Maimoona Inayat, MD
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Mucus plugging due to lung infection is a common cause of lung collapse, which is treated both conservatively and invasively aimed at mobilization of the plug. Lung collapse secondary to Clot-Plugging is rare, and demands early diagnosis to expedite management. A 91-year-old woman was transferred from a nursing home for hemoptysis for 2 days. She was admitted to the Intermediate Care Unit for given acute hypoxemic respiratory failure after pulmonary edema and pulmonary embolism were ruled out. CT scan with contrast showed bilaterally diffuse, scattered, faint, ground glass opacities, thought to be due to an infectious or inflammatory etiology. Initial suspicion was bronchiectasis or possible aspiration. Her respiratory failure rapidly improved with supplemental oxygen; however, she desaturated again. Chest X-ray showed left hemithorax opacification with leftward mediastinal shift suggestive of lung collapse, that was presumed to be caused by mucous plugging. The patient received incentive spirometry and aggressive chest physiotherapy aimed at mucus plug mobilization since bronchoscopy was considered to be high risk given the patient’s age and nutritional status. No improvement in ventilation was noted on follow up X ray and her oxygen demands remained high. Repeat CT scan showed near complete opacification of the left hemithorax with fluid in the left lower lobe bronchi and in the peripheral portion of the left mainstem bronchus. A fiberoptic bronchoscopy was eventually performed which showed total left lung atelectasis from multiple large retained blood clots. Chest X-ray after bronchoscopy showed resolution of lung collapse and improved ventilation in the left upper lobe. She was successfully discharged to a rehabilitation center after successful return to room air oxygen.

Pulmonary atelectasis leading to lung collapse secondary to bronchial occlusion by a mucus plug is often treated by chest physiotherapy and antibiotics. This case illustrates the importance of recognizing clot-plugging as an important cause of lung collapse in patients with idiopathic diffuse alveolar hemorrhage. Respiratory failure from lung collapse following an episode of airway bleeding should arouse suspicion of an obstructing blood clot, which is not amenable to the conservative management strategy of mobilizing mucus plugs. Early use of fiberoptic bronchoscopy for removal of the offending clot with re-expansion of the affected lung segments is an effective and safe treatment.
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TO HYDRATE OR DIURESE...THAT IS THE QUESTION
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Transposition of the great arteries (TGA) is a ventriculoarterial lesion and a cyanotic congenital heart disease, characterized by the aorta arising from right ventricle and the pulmonary artery originating from left ventricle, resulting in the creation of two parallel circulations. The prevalence is 2.3-4.7/100,000 live births in USA. Thirty percent of patients with uncorrected lesions will die in the 1st week of life, 50% in the 1st month, and 90% within the 1st year. For those that survive, maintaining the correct fluid balance can be challenging.

A 60 year-old female with a childhood history of TGA and VSD, which were never corrected and complicated by Eisenmenger Syndrome, presented to the ED for pleuritic chest pain and confusion associated with worsening dyspnea and sweating. At baseline, the patient had dyspnea with walking more than 1 block, normal mobility, and was able to perform instrumental ADLs. Her last known echocardiogram showed an EF of 45%-50%. Pertinent vital signs included tachypnea (RR 22-24), tachycardia (HR 110-120) and hypoxia (O2 sat 61%). On physical exam, a thrill was felt over the tricuspid area with a loud S1/S2, kyphosis, and poor air entry bilaterally with crackles, clubbing, and peripheral cyanosis. On lab work, Hgb was 19 and an ABG showed chronic hypoxic hypercapnic respiratory acidosis. MRI showed compression fracture and kyphoscoliosis. The clinical diagnosis of acute on chronic respiratory failure secondary to underlying community-acquired pneumonia was established. Management was initiated with HFNC at FiO2 of 86% at 40L/minute (with which her O2 sat improved to 70%). The patient was discharged after having improved with antibiotics. Shortly after, the patient was readmitted for pneumonia which was complicated by vascular congestion and AKI after receiving 1L of fluids. A furosemide drip was started at 5mg/hr and the patient was subsequently transferred to John’s Hopkins Hospital for a right-heart catheterization and Swan Ganz. However, the patient developed worsening pneumonia and was found to have influenza A, after which she subsequently passed away.

It is important to mention that patients with TGA and Eisenmenger’s can poorly tolerate rapid fluid shifts and hypovolemia, which makes it important to maintain adequate systemic vascular resistance (SVR). Low SVR can exacerbate the right-to-left shunt causing a sudden cardiovascular collapse and death, especially in the setting of sepsis/severe infection, as these patients are highly preload-dependent.
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DIFFUSE LARGE B-CELL LYMPHOMA PRESENTING AS CAVITARY PULMONARY NODULES
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A 52-year-old man with a past medical history of GERD and depression presented to the emergency department after experiencing two months of back pain and shortness of breath. Associated symptoms included productive cough, chills, fever, and 20 kilograms of unintentional weight loss over two years. He had no smoking or significant travel history. His exam was notable for intermittent fever on arrival (maximum temperature of 38.2 C), mild crackles at the left lower lung base, and cough with non-bloody yellow sputum.

His prior workup at outside facilities included CT of the chest showing multiple cavitory lung masses and three negative sputum AFB cultures. He had non-diagnostic fine needle and core biopsies of the lung as well as a non-diagnostic biopsy of a posterior mediastinal lymph node. Pertinent lab results at admission included a primarily neutrophilic leukocytosis of 16.33 K/mcL. Workup included a CT angiogram of the chest that demonstrated innumerable lung nodules, several of which were cavitary, as well as mediastinal and abdominal lymphadenopathy.

He underwent a biopsy of a retrocrural mass which was again non-diagnostic, and ultimately proceeded to a left VATS lung wedge resection. Pathology was consistent with diffuse large B-cell lymphoma (DLBCL), non-germinal center. He was started on chemotherapy (R-CHOP) with overall improvement in symptoms over six months. DLBCL is the most common histologic subtype of non-Hodgkin’s lymphomas and causes approximately 10% of primary pulmonary lymphomas. Though extranodal involvement occurs in approximately one third of patients with DLBCL, pulmonary involvement is atypical (<2%). The differential diagnosis broadly includes infection (tuberculosis, necrotizing bacterial pneumonia, fungal pneumonia), immune process (granulomatosis with polyangiitis, rheumatoid arthritis nodule, sarcoidosis, pulmonary Langerhans cell histiocytosis), neoplasm (metastases, primary lung malignancies), and embolic phenomenon (septic, thromboembolic).

In summary, we describe multiple cavitary pulmonary nodules secondary to DLBCL, an atypical presentation for this disease. Workup should be directed to exclude other more common etiologies and histopathology can confirm the diagnosis.
A RARE LYMPHOMA UNDERLYING MULTI-ORGAN SYSTEM FAILURE. Todd S, MD, Glick D, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore.

Hepatosplenic T-cell lymphoma (HSTL) is a rare but aggressive extra-nodal and systemic neoplasm originating from splenic gamma-delta cytotoxic T-cells. It most commonly occurs in young adult males and typically presents with cytopenias, liver failure, and hepatosplenomegaly. Liver biopsy and flow cytometry with immunohistochemistry on peripheral blood are accurate and minimally invasive techniques for diagnosis. Median survival is roughly four months and treatment is complicated by marked chemo-resistance and blast transformation. Given the high rate of mortality, HSTL should be considered in any young male patient presenting with systemic symptoms and splenomegaly.

A 57-year-old man was transferred to the University of Maryland for a liver transplant evaluation. Past history was significant for asplenia after a motor vehicle collision and biopsy-confirmed chronic hepatitis due to a remote history of alcohol abuse. During a routine hepatology appointment, it was found to have hypoxia, pancytopenia, and worsening liver function. He was admitted and a liver biopsy and flow cytometry were performed. His mental and respiratory status declined and eventually required broad spectrum antibiotics and diuresis.

He suffered a pulseless electrical activity (PEA) arrest. Return of spontaneous circulation (ROSC) was achieved and he was treated for presumed Klebsiella pneumoniae bacteremia secondary to cholangitis. The patient’s intensive care unit (ICU) course was complicated by worsening multi-organ failure including disseminated intravascular coagulation and pancytopenia. Eventually, the results of the flow cytometry revealed HSTL. Given the patient’s excellent functional status on admission, palliative chemotherapy with cladribine and methylprednisone was initiated. However, his clinical status continued to decline with the development of Vancomycin-Resistant Enterococcus bacteremia and refractory shock requiring vasopressors. Ultimately, the decision was made to terminally extubate the patient.

This case underscores the importance of early detection and high index of suspicion for HSTL. Given the rarity and severity of HSTL, there is little data available to guide staging and treatment of these patients. With newer, less invasive screening tests performed on peripheral blood, early diagnosis is possible. Still, even with prompt diagnosis, this disease is frequently fatal and more research is needed to improve speed of diagnosis and treatment options for these young patients.
NATIVE TRICUSPID VALVE ENDOCARDITIS AS A SEQUELE FROM A MEMBRANOUS VENTRICULAR SEPTAL DEFECT.
Lingel J, MD, Khural J, M.D. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Infective Endocarditis (IE) affecting the tricuspid valve is most commonly seen in the setting of intravenous (IV) drug abuse. The mechanism for this involves the tricuspid valve being damaged from solid particles that are injected along with the illicit drugs and ultimately can result in infection. Additionally, there are cardiac factors that increase the risk for IE. These include rheumatic heart disease, artificial heart valves, unrepaired congenital heart defects, or implanted cardiac devices such as defibrillators or pacemakers. *Staphylococcus aureus* is commonly an isolated organism. Viridans streptococci can also lead to infective endocarditis; however, this organism grouping is less commonly seen in the setting of right-sided endocarditis. This case highlights right sided IE caused by viridans streptococci secondary to congenital heart defects.

A 27-year-old female with a history of polycystic ovarian syndrome (PCOS) and report of a “hole in her heart” at birth, presented to the hospital with 1 month of fevers, chills, night sweats, fatigue, body aches, and vaginal bleeding. She was initially treated for adenomyosis for her vaginal bleeding, however she was also found to have 4/4 bottles of positive blood cultures for alpha hemolytic viridans streptococci. She denied history of IV drug use, as well as recent dental procedures. Examination findings were notable for a temperature of 39.2°C and a 3/6 systolic ejection murmur heard best at the left sternal border. There were no Janeway lesions, Osler nodes, or splinter hemorrhages on exam. A transthoracic echocardiogram (TTE) was performed and revealed a small membranous ventricular septal defect (VSD) and a thickened septal leaflet of the tricuspid valve with minimal prolapse and mild to moderate regurgitation. There were no vegetations on the tricuspid valve. Subsequently, a transesophageal echocardiogram (TEE) was performed which revealed prolapse and thickening of the tricuspid valve with severe regurgitation without any evidence of vegetation. The VSD is noted to have left-to-right shunting on color doppler with a jet peak velocity of 5.6 cm/s. Based on 2 major criteria of positive blood cultures for typical microorganisms and new valvular regurgitation, the patient is diagnosed with IE and treated with 6 weeks of IV antibiotics.

Though viridans streptococci less commonly causes tricuspid valve endocarditis, it is believed that the jet from the VSD led to damage of the tricuspid valve, creating a nidus for infection. The patient was ultimately referred to cardiac surgery for repair of her VSD.

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A CASE OF NON-INFECTIONOUS ENCEPHALITIS IN A YOUNG MALE
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MedStar Good Samaritan Hospital, Baltimore, Maryland

Anti-NMDA receptor encephalitis is an autoimmune disorder that occurs four times more commonly in females and is frequently associated with a paraneoplastic syndrome.

A 33-year old African American man presented with altered mental status and agitation for 1 day. On physical examination, he was afebrile but was agitated and had incoherent speech without focal neurological deficits. There was no left shift or leukocytosis and he had negative urine toxicology results. An MRI of the brain was normal. He was started on empiric treatment for bacterial and viral meningitis/encephalitis. His mental status continued waxing and waning with intermittent seizure-like activities involving muscle stiffness, ankle clonus, and episodes of repetitive orofacial movements and memory impairment. Multiple EEGs were negative for epileptiform activity. His CSF cultures and viral NAAT’s were negative. When infection was ruled out, he was empirically started on steroids for the possibility of autoimmune encephalitis. The patient’s symptoms improved transiently but relapsed after 2 days. Finally, results for anti-NMDA receptor antibody from his CSF was positive with titers of 1:32 (ref value 1:2) with negative known paraneoplastic auto-antibodies. He was started on IVIG; however, he did not improve so was started on rituximab after which he gradually improved clinically and was discharged to a sub acute rehabilitation facility.

Anti-NMDA receptor encephalitis can present with seizure-like symptoms and is not always associated with a paraneoplastic syndrome. Recovery is good but, slow. Symptoms may relapse, especially in patients without tumors and in patients with undetected or recurrent tumors.
HOW SWEET IS TOO SWEET: AN ATYPICAL CASE OF PANCREATITIS INDUCED HYPERGLYCEMIC HYPEROSMOLAR STATE
Rameez Jafri, MD; Nicholas Bedard, MD; Abhilasha Singh, MD; Usman Sagheer, MD; Sadaf Mustafa, MD; Maimoona Inayat, MD

Introduction:
Hyperglycemic hyperosmolar state (HHS) is a serious, life-threatening medical emergency that usually afflicts elderly patients with type 2 diabetes. It is characterized by hyperosmolality, insulinopenia and severe hyperglycemia. Early diagnosis and management are paramount to reinstate hemodynamic stability since the case fatality rate for HHS is exceptionally high associated with multiple complications.

Case:
A 42-year-old non-diabetic unresponsive African American woman with a BMI of 35.0 was brought to our facility with altered mental status and an elevated blood glucose level of more than 2000 mg/dL. Review of systems included a one-week history of nausea and vomiting. On admission, her sodium was 126 mEq/L, bicarbonate was 12 mEq/L, BUN was 80 mg/dL, creatinine was 4.95 mg/dL, lipase of 11,000 U/L, and triglycerides were 586 mg/dL. Her arterial blood gas showed pH of 7.12, a pCO2 of 37, a pO2 of 104. Due to her elevated lipase, a CT scan of the abdomen was obtained and demonstrated findings consistent with acute pancreatitis. This was thought to be the underlying cause of her HHS. Her hospital course was complicated by acute encephalopathy secondary to gross metabolic derangements, respiratory failure requiring intubation, and fluid refractory hypotension necessitating vasopressors and ICU admission. The patient eventually recovered from HHS and was discharged in a stable condition.

Discussion:
This case is an important review, as it underscores an atypical presentation of HHS due to relatively young age and irregular degree of extreme hyperglycemia. While pancreatitis has been described as an underlying cause of HHS, it is only seen in a minority of cases. This case study postulates that the patient’s excessive nausea and vomiting lead to pancreatitis secondary to ischemia, which induced an HHS. This is vital as hyperglycemia superimposed on any ischemic insult can increase the risk of multi-organ failure, as with our patient. Hence, recognition and management of resulting complications needs to be expedient.
EXHAUSTED AND BLUE IN THE FACE: AN APPROACH TO CHROMHIDROSIS

Introduction: Chromhidrosis, or abnormal colored sweat, is a rare condition that often confuses clinicians and presents diagnostic challenges. Guidance on how to approach a patient with colored sweat is not well synthesized many cases are diagnosed clinically without confirmation of the causative agent. Here we describe a case of a 21-year-old male with atypical phenylketonuria (PKU) who presented with an acute onset of blue-green colored sweat.

Case Presentation: A 21-year-old male presented to the children’s hospital emergency room due to concern for a flare of his atypical PKU, dihydropteridine reductase deficiency (DHPR), based on a two-day history of progressive fatigue and blue-green skin discoloration. At presentation, he was found to have blue-green skin discoloration on his eyebrows, ears, chin, neck and fingers with spread proximally in the subsequent hours up his forearms. The pigment could be incompletely and temporarily wiped off with an alcohol swab or paper towel, with return after 30-60 minutes. The alcohol swab retained a distinct blue-green color. He denied similar discoloration in the past and had no recent changes in medications. For 5 years, he had been taking L-5-hydroxytryptophan (5-HTP) for neurotransmitter level augmentation in a bright blue-green capsule (25mg capsule, 4 capsules 4 times daily) from the current pharmacist who denied known compounding or capsule changes.

Discussion: In this case we discuss the challenges encountered while attempting to confirm a suspected diagnosis of chromhidrosis. The literature provides limited guidance on how to approach a patient with colored sweat. Unfortunately in our case, the underlying etiology of the patient’s skin discoloration (e.g., lipofuscin, melanin or other pigment) could not be ascertained but we strongly believe it was due to the dye in his medication capsule. We further discuss a reasonable and succinct diagnostic approach to the patient with suspected chromhidrosis.

Conclusion: Based on the clinical history and findings in our patient, we concluded that our patient had a diagnosis of true eccrine chromhidrosis caused by dye in his medication capsule. Chromhidrosis and pseudochromhidrosis do not cause any medical harm to patients but they can cause psychological stress and embarrassment. The causative substance, once identified, should be eliminated and goal of treatment is to reduce sweating to improve patient’s symptoms and quality of life.
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Indicate your participation

in research process (4 sentences or less): I was the admitting provider for this patient and cared for her throughout her hospital stay.

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END STAGE LUNG DISEASE AND HEART FAILURE AS A PRESENTATION FOR PULMONARY LANGERHANS CELL HISTIOCYTOSIS. Green S, MD, The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Pulmonary Langerhans cell histiocytosis (PLCH) is a rare interstitial lung disease of unknown pathogenesis characterized by infiltration of Langerhans cells into the lungs. It is a smoking-related disease and clinical presentation varies widely from dyspnea on exertion to cough, chest pain, fatigue, and spontaneous pneumothorax.

A 39-year-old man presented with a ten-year history of dyspnea on exertion with rapid worsening in the past 18 months, now requiring rest after two steps. He previously smoked one pack of cigarettes per week for twelve years, but quit two years prior to presentation. He was placed on home oxygen one month prior to presentation when he was discharged from an outside hospital where he was treated for the same dyspnea. On examination, he was afebrile with pulse oximetry 92-94 percent on 4 liters nasal cannula oxygen. His exam was notable for clear lungs, clubbing of his digits, jugular venous distention to the angle of the mandible, and bilateral lower extremity pitting edema.

Computed tomography (CT) chest showed severe diffuse emphysematous changes and five distinct pulmonary nodules, largest measuring 1cm. Alpha-1 antitrypsin genotype was normal. Pulmonary function tests showed a diffusing capacity of the lungs for carbon monoxide (DLCO) that was 29% of predicted. Electrocardiogram showed evidence of right heart strain with right ventricular hypertrophy and right axis deviation. A right heart catheterization showed pulmonary artery pressures of 51 mm Hg. Transthoracic echocardiogram demonstrated an ejection fraction of 40%, down from 55% one month prior.

During his hospitalization, the patient underwent thorascopic biopsies of the right lung. Immunostain for CD1a and S100 were performed and highlighted patchy aggregates of Langerhans cells on all tissue samples, confirming the diagnosis of PLCH. A chest tube was placed after the procedure and the patient continued to have a high oxygen requirement, requiring humidified high flow nasal cannula (HFNC). The patient is a Jehovah’s Witness and does not accept blood products. He is currently awaiting transfer to Houston Methodist Hospital for a bloodless lung transplant.

The primary treatment for PLCH is smoking cessation, though natural history of the disease is variable ranging from spontaneous remission to respiratory failure and death. Overall five-year survival for patients with PLCH is greater than 75%.
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LYMHPOPROLIFERATIVE DISORDER SECONDARY TO EBV IN A PATIENT WITH HYPOGAMMAGLOBULINEMIA
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Introduction: Common variable immunodeficiency disorders (CVID) are a heterogeneous group of syndromes associated with hypogammaglobulinemia and a defect in specific antibody production. These defects increase the susceptibility to pathogens. This susceptibility, along with other factors, appears to increase the risk of malignancy, especially lymphomas.

Case: We present a 52 year old female with a history of atypical CVID with EBV initiated lymphoproliferative disorder with evidence of disease progression on recent PET/CT. Initially she presented to her rheumatologist with a 2 month history of aching and bilateral swelling in her MCP’s, PIP’s, and dry eyes. She was diagnosed with rheumatoid arthritis and treated with adalimumab, azathioprine, and a long prednisone taper. During her prednisone taper she began to experience a flu-like illness with associated painful right inguinal lymphadenopathy and night sweats. FNA of lymph node was initially non-diagnostic and an excisional biopsy was performed. The pathology from the biopsy showed EBV associated lymphoproliferative disorder without concern for high grade Non-Hodgkins lymphoma or Hodgkins lymphoma. The patient was started on a course of IVIG in October but the response with mixed and some radiographic progression of her lymphoproliferative disease was noted. Given her current condition, immune reconstitution is the likely next step in treatment.

Discussion: Treatment of underlying autoimmune disorders in patients with CVID is often complicated by numerous factors. The disease itself places that patient at risk for lymphoid malignancies. Additionally, novel medications such as TNF blocking monoclonal antibodies suppress the already weakened immune system placing the patient at further risk for chronic EBV infections. These patients are often given the difficult choice of increased risk of malignancy or poorly controlled autoimmune disorders. Unfortunately, this patient will likely require allogenic stem cell transplant to halt her lymphoproliferative disorder.

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Title: Reassessing the Diagnostic Criteria for Acute Pancreatitis

Background: The American College of Gastroenterology recommends the presence of two of the following criteria to diagnose acute pancreatitis (AP): (i) abdominal pain consistent with the disease, (ii) serum amylase/lipase 3x the upper limit of normal, and (iii) characteristic findings from abdominal imaging. Imaging should be performed when the diagnosis is in doubt or in refractory cases. We herewith report a case of suspected AP diagnosed without imaging. However, critical findings that were later evident on imaging studies suggest an alternative diagnosis.

Case Description: A 50-year-old woman with past medical history of alcohol abuse and prior episodes of pancreatitis presented to the Emergency Department (ED) with a witnessed seizure. She also complained of epigastric pain and dyspnea. Initial laboratory tests showed potassium 2.2, creatinine 1.54, magnesium 1.5, WBC 2.3, and alcohol level 269. A second seizure occurred in the ED. She received lorazepam, electrolyte replacement, and intravenous fluids. Because of her abdominal pain, a lipase level was obtained. It was 2775 u/L, with a lactate of 7.7 mmol/L. Based on fulfillment of 2 of 3 of the AP criteria, as well as the compelling clinical history of prior episodes of pancreatitis with an elevated alcohol level, the patient was diagnosed with AP. No abdominal imaging was performed.

A chest X-Ray was obtained to evaluate the dyspnea. This incidentally showed free air under the right diaphragm. A CT then confirmed the presence of critically large amounts of free air and fluid in the abdomen, obscuring visualization of the pancreas. The patient then became hypotensive despite aggressive fluid resuscitation, with a repeat lactate of 9.4. The patient underwent emergent surgery, in which a perforated anterior duodenal ulcer was surgically corrected.

Discussion: Peptic ulcer disease (PUD) is a well-documented cause of lipase elevation. As both AP and PUD can cause abdominal pain and lipase elevation, imaging can prove helpful, and indeed critical, in differentiating between these two important life-threatening diagnoses.

Conclusion: Despite a verified chronic history, a high index of suspicion for imaging must be maintained in patients presenting with suspected AP. Consideration should be given to modifying the diagnostic criteria to allow for imaging if PUD or similarly concerning diagnoses are a possibility.

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