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2019 Mulholland Mohler Resident Meeting

A congenital cause for chest pain
Alan P Jacobsen MBChB, Susan M Lin MD, Department of Medicine,
The Johns Hopkins Hospital, Baltimore, MD

Congenital anomalies involving the coronary arteries are common. Within this, anomalous aortic origin of coronary artery (AAOCA) is uncommon but may increase the risk of sudden death and 2018 ACC/AHA guidelines for management of this disorder exist. A 40-year-old male with no prior medical history presented to the ED following one month of progressive exertional chest pain. His vital signs were normal and physical exam unremarkable. Normal sinus rhythm on EKG and serial troponins were negative. Transthoracic echo showed an ejection fraction of 65% without valvulopathy or regional wall motion abnormality. CTA chest demonstrated a right coronary artery which arose from the left coronary sinus and was severely narrowed as it coursed between the pulmonary trunk and ascending aorta. Findings confirmed on coronary angiogram; no evidence of myocardial bridging or atherosclerosis. The patient achieved 13.4 METS on an echo exercise stress test with exacerbation of his chest pain but without electrocardiographic or echocardiographic evidence of ischemia. He proceeded to fractional flow reserve (FFR) with intravenous adenosine. The FFR ratio of 0.91 at maximum hyperemia was not consistent with an obstructive physiology. However, intravenous ultrasound (IVUS) showed systolic compression of the ostium, which in some views appeared nearly obliterated. The cardiothoracic surgery team were consulted. Following lengthy multidisciplinary discussions, the patient proceeded to unroofing of the RCA given his persistent chest pain. The perioperative period was unremarkable and the patient was remained chest pain free one month after his procedure.

The patient presented with the most common variant of AAOCA, a RCA arising from the left coronary sinus (79%). The 2018 AHA/ACC adult congenital heart disease guideline recommends evaluation to assess for high risk features for sudden cardiac death such as age <30, evidence of ischemia, symptoms or relation to exercise. Features of the coronary ostium and proximal coronary artery course (slit-like/fish-mouth-shaped orifice, acute angle takeoff, intramural course, interarterial course and hypoplasia of the proximal coronary artery) also confer increased risk. In the presence of electrical evidence of ischemia or ischemic symptoms, it is a Class I recommendation to proceed to surgical management. Weaker recommendations for surgical management include a left coronary artery arising from right coronary sinus regardless of the presence of ischemia and either AAOCA if a ventricular arrhythmia is present.
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A Rare Presentation of Daptomycin Induced Eosinophilic Pneumonitis

Introduction: Eosinophilic pneumonia is a rare respiratory distress syndrome characterized by accumulation of eosinophils in the lung parenchyma. The pathophysiology is thought to be secondary to antigen detection by alveolar macrophages, which recruit T2 Helper cells that release interleukin 5, causing eosinophil proliferation and recruitment to the lungs. We herewith report a case of eosinophilic pneumonitis due to daptomycin in a patient with pre-existing pneumonia.

Case Presentation: A 27 y.o. female with past medical history of poorly controlled type I Diabetes Mellitus, Charcot arthropathy of the left ankle, and multiple foot surgeries was admitted with osteomyelitis of the left ankle. She underwent a left talotibial calcaneal arthodesis. On postoperative day (POD) one, the patient was febrile, tachycardic, tachypneic, and hypoxic, requiring 2 liters of nasal cannula. CT Angiography showed bilateral dependent consolidations, likely atelectasis or aspiration pneumonia. She was started on piperacillin-tazobactam and azithromycin, which was changed to ampicillin-sulbactam on POD three.

Bone cultures from surgery returned later that day, however, and were positive for staphylococcus capitis. Thus, the antibiotic regimen was changed to daptomycin, which the patient had tolerated several times in the past without issue. On POD four, the patient became tachycardic to 120-130 beats per minute, febrile to 103.4, tachypneic, and hypoxic, saturating at 94-96% on 3L of nasal cannula. The patient was transferred to the ICU for high flow nasal cannula. At this time, new onset peripheral eosinophilia of 8.7% was noted on complete blood count and confirmed with a manual differential. The daptomycin was discontinued on POD six. The peripheral eosinophilia peaked at 12.1 on POD nine and then began to trend downward. By POD 11, the patient was weaned off high flow nasal cannula and subsequently to room air. She was discharged with six weeks of oxacillin for osteomyelitis suppression.

Discussion: This case highlights the need for clinicians to be mindful of unusual presentations of daptomycin induced eosinophilic pneumonitis, especially in patients with pulmonary complications prior to daptomycin administration. In this case, possible aspiration pneumonia delayed the recognition of daptomycin induced eosinophilic pneumonitis.
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**ABSTRACT FORM:** Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and **STAY WITHIN THE BORDERS!**
Sepsis from *Candida glabrata* urinary tract infection requiring amphotericin intravenous and bladder irrigation: a case report

**INTRODUCTION**
While Candiduria is a common event in hospitalized patient, *Candida glabrata* is a rare species to be isolated. This organism is challenging to treat given its higher dose-dependent susceptibilities. Amphotericin B, a drug of last-resort given its toxicities, may well be needed.

**CASE DESCRIPTION**
There was an 83-year-old man with extensive past urological history including prostate cancer who was found to have severe sepsis with acute kidney injury and bilateral hydronephrosis secondary to urinary tract infection. He was initially treated with antibiotics and the switched to fluconazole when *C. glabrata* was isolated from urine culture. However, given lack of improvement, azole resistance was suspected; therapy was transitioned to IV amphotericin and even amphotericin bladder irrigation. After urine culture cleared, he was discharged on PO fluconazole to elder rehab, where he tolerated therapy well before being discharged to SAR.

**DISCUSSION**
While this patients was already at increased risk for urinary tract infection, given his history of prostate cancer, *C. glabrata* is not a common organism. Kauffman et al. (2000) studied 861 patients with funguria and only 15.6% of them had this species. Rather than increased resistance, Hii et al. (2019) demonstrated that this microorganism has increased dose-dependent susceptibility. Conventional amphotericin B has systemic adverse reactions in >10%, including renal dysfunction, which was of special concern in this patient given his kidney injury and hydronephrosis.

**CONCLUSION**
Antimicrobial choices are limited when facing genitourinary fungal infection, and there should be suspicion for drug resistance when facing *C. glabrata*. While amphotericin is not to be used lightly, there is a not-insignificant chance that it is required for such an infection.
A Rare Cause of Takotsubo Cardiomyopathy
Alicia Liendo, MD, Kellen Mulhern, DO, Ravitej Khunkhun, MD
Sinai Hospital of Baltimore- Baltimore, MD

Introduction: Takotsubo Cardiomyopathy is commonly associated with stressful events leading to a catecholamine excess which causes coronary vasospasm. Stressful conditions are the most widely known risk factor however, there have been case reports of patients with a stress induced cardiomyopathy without a stressful predisposition and an anatomic abnormality of their coronary arteries. Investigation of coronary abnormalities may be justified on a case by case basis, as this could also be a potential risk factor, albeit rare.

Case: Patient is a 57-year-old woman with a past medical history of hypertension, dyslipidemia, 20 pack year tobacco user who presented to the hospital with crushing chest pain and dyspnea. It started at church but denied any stressful events. Her overall physical exam was unremarkable. She was found to have a troponin elevation and her EKG showed T wave inversions in multiple leads. Cardiac catheterization revealed no obstructive coronary artery disease and an anomalous right coronary artery (RCA) from the left coronary cusp. Echocardiogram revealed severely reduced left ventricular systolic function with an ejection fraction of 15-25% and apical ballooning. A CT angiogram confirmed that the RCA coursed between the pulmonary artery and aorta with a slit like configuration, which predisposed the artery to compression. She subjectively improved with a beta blocker and diuretic while she was admitted, and was discharged with beta blocker and angiotensin receptor blocker with outpatient cardiology follow up.

Discussion: In this case, we observed a woman without a stressful predisposing event who developed Takotsubo Cardiomyopathy due to an anomalous right coronary artery. In angiographic studies, the estimated incidence of anomalous right coronary arteries is 0.019% to 0.49%. Symptoms range from asymptomatic to sudden cardiac death. This likely occurs due to the systolic compression of the RCA from the aorta and pulmonary infundibulum during a catecholamine surge. There are no definitive recommendations towards management, but current practices involve surgical intervention in patients with ongoing cardiac complications or medical management in patients who demonstrate recovery with medications alone. In patients with stress induced cardiomyopathies, congenital coronary anomalies present a unique risk factor with highly variable treatment courses. Screening for this phenomenon should be considered on a case by case basis.
Lipase Negative Hypertriglyceridemia Induced Acute Pancreatitis

Introduction: Acute pancreatitis is a common inflammatory disorder with an incidence of 4.9 to 35/100,000. The diagnosis is made by meeting two out of three diagnostic criteria which typically include positive serum markers. We hereby present an unusual case of pancreatitis with negative serum markers.

Case: A 40-year-old woman with a history of severe gallstone pancreatitis 4 years prior, status post cholecystectomy and a life-long non-drinker, presented with 10/10 left sided and upper abdominal pain that radiated to the back, and was aggravated by oral intake and deep breathing. Physical examination was significant for tachycardia, left upper quadrant, flank and costovertebral angle tenderness. Laboratory tests showed lipase 39 U/L, AST 18 U/L, ALT U/L, total bilirubin 0.5 mg/dL, and triglycerides (TG) 1550 mg/dL. A computed tomography scan of the abdomen was compatible with acute pancreatitis. No evidence of an obstructive processes was seen on laboratory tests or imaging. A diagnosis of pancreatitis, possibly induced by hypertriglyceridemia, was made. She was treated with analgesia and fluids. Over the next 48hrs her symptoms failed to improve, repeat lipase levels were persistently normal but triglyceride levels were on the rise. She was started on an insulin drip, gemfibrozil and omega-3. After 5 days her symptoms finally started to improve, goal TG level of 500mg/dL was achieved after 11 days of therapy.

Discussion: Acute pancreatitis has numerous etiologies but by far the most common ones are alcohol and gallstones. Hypertriglyceridemia induced pancreatitis occurs in 1 to 14 percent cases. False negative amylase level can be caused by hypertriglyceridemia greater than 500 mg/dL due to interference with calorimetric reading, but this has not been reported to be an issue with lipase levels. Acute on chronic pancreatitis could present with normal lipase and amylase levels but there was no evidence of such in this case. A normal lipase level has a very high negative predictive value (94-100%) which usually suffices to rule out acute pancreatitis, however, as demonstrated by this case pancreatitis can occasionally occur with normal lipase levels. The cornerstones of treatment are analgesia and fluid resuscitation, however, when hypertriglyceridemia is the cause an insulin drip or plasmapheresis may be required. Insulin works by inhibiting lipase in adipocytes which reduces triglyceride degradation and the release of fatty acids into the circulation. In a literature review 17 published cases of lipase negative pancreatitis were identified; of these only 2 were due to hypertriglyceridemia. We believe this is the third ever reported case of acute pancreatitis from hypertriglyceridemia with normal lipase levels.
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AEROMONAS JANDAE CELLULITIS DUE TO SALT WATER EXPOSURE. Alnabusi M, MD; Foppiano Palacios C, MD. University of Maryland Medical Center and Baltimore VA Medical Center, Baltimore, MD.

Aeromonas species are non-spore forming Gram negative rods that are facultatively anaerobic bacteria mainly found in aquatic environments. Aeromonas species are most commonly associated with gastroenteritis but can also cause skin and soft tissue infections (STIs) including cellulitis, myonecrosis, ecdyshyma gangrenosum, pustules, ulcers, and abscesses.

A 70-year-old male presented to the emergency department with fevers, diaphoresis, rigors, nausea, and a rash that started 3 days ago. The patient recently returned from North Carolina, and while on the beach, he stepped on an oyster shell lacerating his left great toe. Examination revealed a 6 x 12 cm erythematous, violaceous, blanchable patch with evidence of excoriation on the left anterior shin in comparison to the right. This was consistent with cellulitis of the left lower extremity. A computer tomography (CT) scan was performed due to concern for necrotizing fasciitis, and it revealed mild generalized soft tissue infiltration and overlying skin thickening most prominent in the anterolateral aspect of the leg suggestive of cellulitis. There were no notable fluid collections or soft tissue gas. The patient was initially started on vancomycin and piperacillin/tazobactam, and then doxycycline was added for empiric coverage of vibrio species given his saltwater exposure and concomitant wound. Blood cultures grew Aeromonas jandaei in both bacterial bottles, and the patient was subsequently switched to oral ciprofloxacin. He was discharged on ciprofloxacin with a plan to complete 2-week course of antibiotics and have outpatient follow up with his primary care physician.

Aeromonas species are enteric pathogens of serious public health concern as they have numerous virulence determinants that are linked to human diseases, such as gastroenteritis, soft-tissue and muscle infections, and septicemia. STIs typically occur after freshwater traumatic injuries, including alligator, fish, snake, and leech bites. A high degree of clinical suspicion for Aeromonas species is warranted when treating fresh water injuries, along with a low threshold to consult surgery due to necrotizing fasciitis. Aeromonas tends to be susceptible to fluoroquinolones, tetracyclines, aminoglycosides, carbepenems, monobactams, and third and fourth generation cephalosporins.

Initial treatment of STIs related to salt water trauma must be promptly treated with broad-spectrum antibiotics to avoid significant morbidity and mortality.
METFORMIN INDUCED LACTIC ACIDOSIS. Alotaibi M, MBBS, Ali F, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Metformin is the first-line treatment for patients with type 2 diabetes mellitus (DM). Metformin-induced lactic acidosis (MALA) is a rare and serious complications of metformin. We report two cases in which hemodynamic stability returned within 12 hours after the initiation of renal replacement therapy (RRT) in patients with MALA induced by acute kidney injury.

A 58-year-old man with a history of DM was admitted for generalized weakness. On admission, his vitals were stable. He was oliguric. Initial laboratory result showed Sodium 136 mEq/L, Potassium 7.5 mEq/L, Chloride 92 mEq/L, Bicarbonate 9 mEq/L, BUN 92 mg/dl, Creatinine 14.2mg/dl, from a baseline of 1.3 mg/dl, PH 7.00 and lactate 17mmol/L. The patient's medications included metformin. One hour after arrival to the intensive care unit, the patient developed profound hypotension and hypothermia (92F) which required vasopressors. PH was unmeasurable (i.e., less than 6.85), lactate 22 mmol/l; serum potassium 8.0 mEq/L and bicarbonate less than 6 mEq/L. Sustained low-efficiency dialysis (SLED) was initiated. The patient’s hemodynamics dramatically improved within the first 12 hours. The metformin level prior to SLED was 9.9 mcg/ml and was 6.1 mcg/ml 10 hours afterward (normal range:1-2 mcg/ml).

A 78-year-old woman with a history of DM was found unresponsive. Initial blood pressure was 40/20 mm Hg; pulse was 26 beats/min. Temperatures were unmeasurable. Laboratory result showed Sodium 139 mEq/L, Potassium 6.5 mEq/L, Chloride 97 mEq/L, Bicarbonate less than 6 mEq/L, BUN 104 mg/dl, Creatinine 5.7 mg/dl from baseline 1.0, pH 6.68 and lactate 11.1mmol/l. Vasopressors were required. Continuous venovenous hemofiltration (CVVH) was initiated. The patient’s hemodynamics dramatically improved within the first 12 hours. Serum metformin level on admission was 19 mcg/ml and 1 day after initialing CVVH was 3.0 mcg/ml.

Our cases demonstrated prompt efforts to initiate RRT can not only dramatically correct acidosis but restore hemodynamic stability in patients with MALA.
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QUINIDINE CALLS IT QUITS: THE CHANGING FACE OF SEVERE MALARIA TREATMENT
Tijana Tuhy, Johns Hopkins Hospital, Baltimore, MD
Severe malaria, presenting as end-organ dysfunction or parasitemia >10% in the majority of cases due to Plasmodium falciparum, is seen in the United States predominantly as a traveler’s illness and is a medical emergency. Prompt diagnosis and treatment are crucial as mortality can approach 30% in the first 24 hours after presentation. Quinine-derived therapy has been used for treatment since its discovery in the 1630s. It is poorly tolerated in its intravenous (IV) form, associated with hypoglycemia, deafness, and blindness. Dose-dependent cardiac toxicity (particularly torsades de pointes, TdP) is among its most lethal side effects. In contrast, artemisinin derivatives are the most potent antimalarials, clearing parasitemia faster than quinine and associated with lower mortality rates in both adults and children (15% vs 22%). Intravenous artesunate is the first-line, WHO-recommended treatment for severe malaria but it is neither FDA-approved nor commercially available in the United States. Due to ongoing research by Walter Reed Army Institute of Research, it is available as part of a compassionate use protocol for patients who fail treatment with IV quinidine. Recently, the manufacturer of IV quinidine in the US halted production, and the entire US supply of quinidine expired on April 1, 2019.

In this case, we present Mr. D, a 65 y.o gentleman from the Democratic Republic of the Congo who presented with abdominal pain, fevers, confusion, and headache shortly after arriving in the United States. He developed abdominal pain, fevers, and headaches, thus presented to a local ED where a peripheral blood smear demonstrated malarial parasites with 9.3% of cells infected. He was transferred to Johns Hopkins Hospital where treatment was initiated with IV quinidine until QRS was noted to be prolonged from 474 ms to 554 ms after loading and continued drip. Infusion was halted and the CDC was contacted for IV artesunate, which was delivered by courier from the nearest depot in New York City. With treatment, his parasitemia percentage became undetectable, mental status improved, and QRS normalized.

While malaria remains rare, initiating treatment rapidly is the greatest predictor of mortality. Due to pharmacologic/economic pressures, changes in treatment schema may be unfamiliar to physicians when they encounter this disease. With IV quinidine no longer commercially available, physicians must contact the CDC for the release of artesunate expediently to prevent delay in treatment. This will present unique challenges for the treatment of the critically ill patient while awaiting delivery of a limited resource.

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
3-2-1 Blast-Off: An aggressive case of Acute Promyelocytic Leukemia, Zachary Reed, National Capital Consortium, Jeremy Perkins, Department of Hematology/Oncology, Walter Reed National Military Medical Center

Introduction: This case report represents the presentation of a rare and aggressive type of Acute Promyelocytic Leukemia (APL) associated with diffuse intravascular coagulation (DIC) in a young patient. It reinforces the necessity of starting all-trans-retinoic-acid (ATRA) at first suspicion of APL. This patient required correction of fibrinogen and platelets more aggressively than typical acute leukemia patients. She presented as high-risk leukemia (WBC >10K) which necessitated additional therapy for leukoreduction. This uncommon subtype of a rare disease demonstrates the necessity of early diagnosis and aggressive treatment to avoid poor outcomes in patients with acute leukemia.

Case: A 29 year old woman with history of melanoma s/p excision 17 years ago presents with 1 week of bruising of her extremities and frequent nose bleeds. Initial blood work revealed elevated WBC count to 32K with 44% blasts as well as thrombocytopenia. A repeat CBC showed WBC of 38K with 70% blasts and platelets of 12K. Fibrinogen was low and D-dimer was significantly elevated.

Differential included acute myeloid leukemia (AML) vs APL, favoring APL due to the presence of DIC. The patient was started on ATRA and given transfusions of platelets and cryoprecipitate. Peripheral smear showed bi-lobed "butterfly" blasts without Auer rods and only a few small granules - concerning for microgranular APL. A bone marrow biopsy was performed and sent for evaluation by pathology. While the flow cytometry from the bone marrow biopsy was pending, the patient's WBC count continued to rise rapidly, necessitating treatment with Hydroxyurea for leukoreduction. Her peripheral WBC count reached a maximum of 87K. The bone marrow cellularity showed 82% blasts or blast equivalents and flow cytometry demonstrated abnormal myeloid blast population. Fluorescence in situ hybridization (FISH) was positive for promyelocytic leukemia retinoic-acid-receptor-alpha (PML-RARA) translocation consistent with a diagnosis of APL.

Once this diagnosis was made, the patient was started on arsenic trioxide (ATO) therapy with the addition of gemtuzumab-ozogamicin (GO). The patient responded appropriately to treatment and her WBC downtrended rapidly over the first week of induction chemotherapy with ATO/ATRA/GO.

Discussion: APL is a rare but aggressive form of AML with only 600 new cases per year reported in the United States. It is important to initiate therapy with ATRA at the first sign of possible APL even before a confirmed diagnosis is made. A peripheral smear alone in a new APL case may not always show Auer rods (a classic sign of APL) as 25% of new APL cases are a microgranular variant such as this one. Once a diagnosis of APL is made, initiation of proper therapy in a timely manner is critical for patient survival. If the patient's APL is an aggressive type, therapy with ATRA/ATO may not be enough, and an additional agent, such as GO, may be necessary for rapid cytoreduction.
Nitroglycerin-Induced Hypotensive Bradycardia
Fatimah Alkhunaizi, MD, and David Furfaro, MD, Osler Medical Residency

Hypotension following the administration of nitroglycerin is a common and well-described side effect, and is typically associated with a reflex tachycardia. A less frequently encountered side effect is hypotensive bradycardia.

A 90-year-old man in overall good health and no known prior coronary artery disease presented with one month of crescendo angina and dyspnea on exertion. He had been trialed on a long acting nitrate without relief. He then had a stress test that was reportedly positive and was referred to the emergency department. On arrival he was hypertensive to 160/75mmHg. Physical exam was notable for a 2/6 holosystolic murmur at the apex, clear lung fields, and jugular venous pressure of 8 cm H2O above the right atrium. Electrocardiogram (EKG) was notable for an old left bundle branch block but no other abnormalities. Troponin-I peaked at 0.21 ng/mL and NT-pro-BNP was elevated at 8000 pg/mL. Chest radiograph showed pulmonary vascular congestion and interstitial edema. He received diuretics, which led to improvement in his dyspnea and his hypertension. However, he continued to have chest pain at rest. In this setting 0.4mg of sublingual nitroglycerin was administered. Within one minute, his blood pressure decreased from 130/60 to 50/30mmHg and he became severely bradycardic to 30bpm. He was symptomatic with facial flushing, nausea, vomiting, and severe anxiety. A code was called and defibrillator pads were placed given concern for impending arrest. Intravenous fluids were infused rapidly and within two minutes his blood pressure had recovered and his bradycardia resolved. No atropine was administered. EKG and troponin following this episode were unchanged. Transthoracic echocardiogram revealed global left ventricular hypokinesia with a severely reduced ejection fraction of 20-25%. His right ventricular (RV) function was intact and he had no aortic stenosis. He underwent urgent coronary angiography, which was notable for severe diffuse triple vessel disease not amenable to percutaneous intervention. An intra-aortic balloon pump was placed and he underwent bypass surgery with excellent recovery. This case illustrates the potential for life-threatening hypotensive-bradycardia with the administration of sublingual nitroglycerin in a patient with intact RV function. Possible mechanisms for the bradycardia include the Bezold-Jarisch reflex, vasovagal effect, and global ischemia. Overall, the response to nitroglycerin cannot always be predicted. Caution should be exercised with its administration, especially in those with an unclear degree of underlying cardiac dysfunction and in elderly patients who are more...
PRIMARY SPLENIC DIFFUSE LARGE B-CELL LYMPHOMA - TRULY HIDDEN CASE OF FEVER OF UNKNOWN ORIGIN.
Bathini S, MD, Chekka P, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Diffuse large B-cell lymphoma (DLBCL) is a proliferation of malignant B-cells that can present with various B-symptoms including night sweats, weight loss, and fevers. Primary splenic B-cell lymphoma is a very rare type of non-Hodgkin Lymphoma (NHL) constituting less than 1% of all NHL. While these patients often present with abdominal pain and splenic masses, they can also present with more subtle symptoms.

A 46-year-old Filipino male with a history of diabetes mellitus presented to the emergency room with 3 months of fevers. He reported daily temperatures up to 40 °C that were associated with rigors, diaphoresis, 5 lb weight loss over 8 weeks, and occasional right upper quadrant discomfort. He also recently traveled to California. His infectious workup was negative for bacteremia, parasites, human immunodeficiency virus, hepatitis B and C, Epstein-barr virus, Q Fever and Coccidiomycosis. On presentation, patient had a fever to 39.2 °C. Physical exam was positive for a 2/6 systolic murmur at left lower sternal border. Laboratory studies at that time were significant for elevated inflammatory markers with C-reactive protein of 7.7 mg/L, ferritin greater than 4000ng/mL, sedimentation rate greater than 120 mm/hr, negative antinuclear antibody, rheumatoid factor, and M-spike on serum protein electrophoresis. He was presumptively treated for a rheumatologic condition with high dose steroids; however, patient continued to have breakthrough fevers.

A positron emission tomography/computed tomography noted a mildly enlarged spleen with diffusely increased uptake. His initial bone marrow biopsy was initially negative for malignancy, however further staining raised concern for possible B-cell lymphoma with uncertain clonality. He underwent a splenectomy, which was complicated by hemoperitonium secondary to a ruptured spleen. Spleen pathology confirmed the diagnosis of DLBCL with minimal bone marrow involvement. Patient was treated with with rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone, along with adjunctive intrathecal chemotherapy and his fevers resolved.

Primary splenic DLBCL is a true “zebra” which can present with rather vague symptoms. Fortunately, definitive therapy with splenectomy in early stages has shown to decrease mortality. Consideration of oncologic causes of fever of unknown origin such as primary splenic DLBCL can lead to early treatment and improve overall mortality.
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Ziprasidone-Induced Quincke’s Disease

Introduction:
Quincke’s disease is a rare, localized form of angioneurotic edema with isolated uvular swelling. Ziprasidone is an atypical antipsychotic medication that has been associated with IgE-related pedal edema, life threatening hypersensitivity syndrome, and angioedema. We herewith report a patient who developed localized uvular edema upon starting ziprasidone treatment.

Case presentation:
A 41 year old man with a past medical history of schizophrenia was brought to the Emergency Department (ED) for disorganized aggressive behavior. The primary clinical impression was acute psychosis. He was also diagnosed with rhabdomyolysis based on elevated creatine phosphokinase (CPK) levels and complaints of body aches. Intramuscular ziprasidone and intravenous (IV) fluids were administered. Several hours later, while still in the ED, the patient began to experience globus sensation in his throat with increased oral secretions.

Examination at that time revealed severe edema of the uvula, with the uvula measuring 5-6 cm in length and 2-3 cm in diameter, without any swelling of the face, lips, tongue, or pharynx. The patient was breathing comfortably with adequate oxygenation. There was no wheezing or stridor. The patient was treated with IV epinephrine, methylprednisolone, and famotidine, with gradual but full resolution of his edema.

Discussion:
Quincke’s disease is a well demarcated, localized uvular angioedema. It can potentially cause laryngeal obstruction and may be life threatening. To our knowledge, this is the second known reported case of ziprasidone-induced angioedema. Clinicians should be aware of this potentially serious allergic reaction when treating patients with ziprasidone.

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
THE CONCURRENCY CONUNDRUM: A CASE OF CHRONIC LYMPHOCYTIC ANEMIA AND MULTIPLE MYELOMA IN ONE
Abigail Chan MD, Roberto Martinez MD
Sinai Hospital of Baltimore, Baltimore, Maryland.

Introduction: Multiple myeloma (MM) and chronic lymphocytic leukemia (CLL) are lymphoproliferative diseases from B-cell progenitors. The presence of both malignancies in a single patient is rare and can complicate management. We present a case of a newly diagnosed concurrent CLL and MM, resulting in untimely death.

Case: A 70-year-old woman presented with a 6 weeks history of chest pain, diaphoresis, and dyspnea. Her vital signs and physical exam were normal. An ischemia workup was unrevealing. Her CBC showed leukocytosis, thrombocytopenia, and anemia. Further workup was notable for acute kidney injury (AKI), hypercalcemia, and a gamma gap. The 24-hour urine protein electrophoresis and urine immunofixation detected a monoclonal spike. Her free light chain ratio (FLC) was 281.60, with kappa FLC measuring 3829.7 mg/dL. Imaging revealed hepatomegaly, splenomegaly, mild lymphadenopathy, without lytic lesions. Bone marrow biopsy showed kappa-restricted CLL and MM with plasmacytosis of >95%, confirmed by immunostaining. A complex karyotype involving 17p deletions and mutated IgVH were observed. She had high risk Rai stage IV CLL and stage III MM with high risk features. Her AKI worsened despite hydration, and plasmapheresis was started for cast nephropathy. The hospital course worsened after she developed sepsis and transfusion-dependent anemia. She was started on dexamethasone and hemodialysis, but eventually succumbed to her disease prior to chemotherapy initiation.

Discussion: CLL originates from minimally proliferative mature B cells with the inability to undergo apoptosis, while MM results from the rapid accumulation of plasma cells. Causes of CLL-related MM were thought to be due to clonal evolution, plasma cell differentiation within a population of CLL, or as an effect of previous treatments. In spite of the difference in pathogenesis and natural history, the overlapping clinical syndromes make the diagnosis difficult. Glucocorticoid-containing regimens are effective in MM and most cases of CLL, but CLL with 17p deletion require targeted therapy as they poorly respond to immunotherapy. The accurate staging and risk stratification impact overall treatment choice and prognosis.
CRUSTED SCABIES MASQUERADING AS LEUKEMIA CUTIS

Introduction: Crusted scabies usually presents in patients with poor cellular immunity (AIDS, Leukemia, Lymphoma, Steroid/Chemotherapy, solid organ transplant), the elderly and disabled patients. We present a case of Norwegian (crusted) scabies in a patient with leukemia cutis. The diagnosis can be delayed in cases when it occurs with another pre-existing skin condition.

Case: Our patient is a 62 male who presents with a diffuse maculopapular rash of one month duration. The initial rash was pruritic and started on his thighs and then spread to his lower abdomen and progressed to involve his back and arms as well. Labs were significant for persistent lymphocyte predominant leukocytosis and peripheral eosinophilia. Bone marrow biopsy was consistent with CLL. His skin punch biopsy then revealed spongiform dermatitis with a brisk superficial and deep dermal mononuclear cell infiltrate including eosinophils and an atypical lymphocytic component, consistent with involvement by CLL/SLL. Given that the rash was severe and was the only manifestation of his CLL, he was treated with chemotherapy which resulted in the resolution of the rash. However, the rash reappeared some months later and was similar in appearance to the leukemia cutis. The rash did not resolve with the second cycle of chemotherapy. The rash eventually progressed into an intensely pruritic rash with diffuse desquamation and crusting of the palms, finger webs and shins. Subsequent biopsy confirmed crusted (Norwegian) scabies and improved with treatment.

Discussion: Norwegian scabies is rare and is notorious for masquerading as other dermatological conditions like contact dermatitis, psoriasis and in this case leukemia cutis. It is most commonly seen in immunocompromised patients and caused by Sarcoptes Scabiei. Duration of treatment is longer than normal scabies given the thick layer of crusting and plaques formation.

Conclusion: Crusted Scabies should be suspected in patients with immunocompromise. Diagnosis can be delayed as it can masquerade as other dermatological conditions.
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AUTOIMMUNE ENCEPHALITIS REVERSED BY RITUXIMAB IN A PATIENT WITH RELAPSED CLASSICAL HODGKIN LYMPHOMA TREATED WITH IMMUNE CHECKPOINT BLOCKADE PRIOR TO BONE MARROW TRANSPLANT

The adoption of immune checkpoint blockade (ICB)-based approaches in relapsed/refractory Hodgkin lymphoma prior to allogeneic bone marrow transplant (allo-BMT) has been met with concern over the potential for increased graft-versus-host disease (GVHD) or immune-related adverse events (IRAEs). Recent case series within our group have suggested that post-transplant cyclophosphamide (PTCy) may limit the incidence of immune-mediated toxicity, even in the mismatched donor setting, after ICB. Here, we report the development of severe autoimmune encephalitis after ICB followed by allo-BMT with PTCy which was successfully reversed with rituximab.

Four months after anti-PD-1/LAG3 immunotherapy and three months after allo-BMT for relapsed/refractory Hodgkin lymphoma, a 20 year male presented to our service with altered mental status, agitation, paranoia, and suicidal/homicidal ideation. Exam revealed pressured speech with a tangential thought process, but was without focal neurologic deficits. MRI brain and spot EEG were unremarkable. CSF revealed 100 WBCs per cubic mm with a lymphocytic predominance (88%), total protein of 113.1 mg/dl, and glucose of 54 mg/dl, suggestive of an inflammatory process. CSF infectious workup including bacterial culture/smear, fungal culture, and viral studies (CMV, EBV, HHV6, enterovirus, West Nile virus, JCV, HHV6, HSV, and VZV) was negative, and serum infectious workup was also negative. A diagnosis of autoimmune encephalitis was established; NMDA receptor encephalitis was suspected due to prominent psychiatric disturbance. His condition was refractory to steroids and plasma exchange but dramatically reversed with initiation of rituximab, suggesting an antibody-mediated process. In addition, rituximab led to full RBC engraftment, suggesting benefit of rituximab in diminishing anti-ABO antibody titers.

To our knowledge, this is the first incidence of autoimmune encephalitis associated with ICB and post-allo-HSCT cyclophosphamide. In addition, autoimmune encephalitis after ICB is typically steroid-responsive. Given that the PD-1 checkpoint has been shown to be a regulator of B cell activation in addition to its classically defined role in T cell function, our patient’s response to rituximab and resistance to steroids may imply a novel mechanism of ICB-mediated toxicity secondary to upregulation of humoral immunity. Overall, this could suggest that post transplant...
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CLOT-22—A DYNAMIC THERAPEUTIC DILEMMA
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Introduction: Active hemoptysis with concomitant pulmonary embolism (PE) is a therapeutic dilemma. Treatment of PE risks perpetuating a potentially uncontrollable bronchopulmonary bleed which can become more acutely demanding when hemodynamics are threatened. We describe a case of a patient with hemoptysis, subsequently found to have a cavitory lesion and massive PE.

Case: 65-year-old male, with history of heart failure and 50 pack-year smoking history, presents with three weeks of worsening intermittent hemoptysis with several ounces of “bright red and bloody sputum.” He denies fevers and night sweats but reports a 40 lbs. weight loss over three months in setting of stable, chronic lower extremity edema. He had no recent travel. He was afebrile, normotensive but tachycardic and requiring oxygen. He had bibasilar crackles, wheezing in the left middle lung field and mild bilateral lower extremity pitting edema. Hgb was 12.1g/dL. No leukocytosis. Chest radiograph revealed pulmonary edema and bibasilar consolidations. Chest CT revealed bibasilar pneumonia with a right middle lobe cavitory lesion (22mm). He was placed in isolation and started on empiric antibiotics. BAL was performed, after which he developed respiratory failure, necessitating transfer to the ICU and intubation. He became hypotensive requiring pressors, and TTE revealed a LVEF of 20% with right ventricular enlargement. A CT of the chest, abdomen, pelvis revealed bilateral PE and enlarging right middle lobe cavitory lesion (35mm). A mediastinal lymph node was sampled by EUS, confirming SCLC. Given the risk of significant clot burden with PE in newly diagnosed malignancy in the setting of stable hemoptysis, the patient was started on a heparin drip. A bronchial blocker set was placed at bedside should the hemoptysis worsen and require tamponade.

Discussion: Massive hemoptysis can be defined as bloody expectorant ≥ 500 mL/day or ≥ 100 mL/hr, which this patient does not have. Massive PE is defined by hypotension, which conferred a higher risk of mortality. Thus, management of massive PE was prioritized in the setting of hemodynamically stable hemoptysis. Had massive hemoptysis been present, bronchial artery embolization would have been required. Understanding risk, in this case, simplifies the therapeutic dilemma.
A 41 year-old man with past medical history of hypertension who presented with acute-onset fevers, myalgias, abdominal pain and dark stools. He was in his usual state of health until one week prior to presentation when he developed fever to 104 F, body aches and dark stools. Of note he had recently traveled to Las Vegas, though denied spending any time in forest or woody areas. His only medication was amiodipine, and he denied illicit drug use. On admission, his vitals were notable for a low-grade fever of 38.1 C, heart rate 107, blood pressure 178/79, SpO2 97% on room air. Labs were notable for WBC 11.7, hemoglobin 16.2, platelets 60, creatinine 1.2, AST 550, ALT 317, ALP 99, INR 1.1, D-dimer 4.13, ESR 27, CRP 11.3. Ferritin was 29,996, with 33% transferrin saturation. Viral studies were sent, including a respiratory viral panel, HIV, HCV, EBV, CMV, HHV6, and HHV8.

ANA, ANCA, and C3/C4 were sent to rule out autoimmune or vasculitis etiologies. Blood cultures were obtained and tazobactam-piperacillin was started. By day three, his fevers and myalgias had resolved, his abdominal pain was improved, and his liver enzymes downtrended. His platelets improved to 100s and peripheral blood smear showed atypical lymphocytes with no schistocytes. Blood cultures were negative and antibiotics were stopped. However, his creatinine continued to rise to 4.9, and urine studies showed proteinuria and hematuria with granular casts. He underwent kidney biopsy which revealed severe acute tubular injury without significant tubulointerstitial fibrosis or inflammation. The patient continued to improve with supportive measures, with eventual downtrend in creatinine, and was discharged with presumed diagnosis of viral infection. However, one month later, all diagnostic workup has returned negative, creatinine has returned to baseline 1.2 with resolution of proteinuria, and patient remains symptom free.

In patients with extreme ferritinemia (>10,000), important diagnoses to rule out include hemophagocytic lymphohistiocytosis, iron overload, hematologic malignancy, and adult-onset Still’s disease when appropriate. More commonly seen clinical diagnoses such as bacterial, viral and fungal infections have also been associated with extreme hyperferritinemia in case reports, and in this case the patient was presumed to have a viral infection despite negative workup for common etiologies.
DIFFERENTIATING MYOCARDITIS FROM SARCOIDOSIS IN CARDIOMYOPATHY. Pudalov D, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Myocarditis and sarcoidosis are diagnosed more frequently given the widespread use of cardiac MRI and PET.

The patient is a 32 year old African American man with a history of hypertension (HTN) who presented to the hospital with fever and shortness of breath. He was in his usual state of health until one week prior to presentation, when he experienced worsening dyspnea on exertion, paroxysmal nocturnal dyspnea with chest pain, fevers, chills and muscle aches. He denied significant family history of cardiac disease, recent travel, or use of alcohol, tobacco, illicit substances, anabolic steroid or stimulants. He did start taking a multivitamin one month prior to presentation, but otherwise was not on medication.

Upon presentation, temperature was 37 degrees C (Tmax 38.8), blood pressure was 129/94, heart rate was 112, respiratory rate was 28, oxygen saturation was 96% on room air. Physical exam was notable for jugular venous distension to 15 cm level, tachycardia with regular rhythm, tachypnea, bibasilar crackles, 1+ bilateral pitting edema with warm and well perfused extremities.

Labs were notable for WBC 7.9, BUN 15, Cr 0.81, glucose 101, total protein 7.1, albumin 4.0, AST 44, ALT 94, T Bili 1.0. Initial troponin was 4.38 and peaked at 11. His BNP was 2,200. RVP, HIV, malaria, hepatitis A/B/C all negative. TSH 1.57, A1C 5.9. EKG showed sinus tachycardia, left axis deviation, poor R-wave progression with dominant R waves throughout the precordium, biventricular enlargement. TTE demonstrated dilated LV (7.3 cm) with low LVEF 10-15% with a mildly dilated and dysfunctional RV but was otherwise without valvular dysfunction. LHC negative for coronary disease. Cardiac MRI showed moderately dilated LV and multifocal areas of transmural delayed gadolinium enhancement involving the mid to basal inferior lateral wall consistent with myocarditis.

His hospital course was complicated by NSVT and bradycardia including sinus arrest; the patient underwent a cardiac PET scan to evaluate for sarcoidosis, which demonstrated an active granuloma. The most likely unifying diagnosis presumed to be cardiac sarcoidosis.

Pathology revealed absence of non-caseating granulomas or myocarditis. A dual chamber AICD was placed and the patient was discharged on a heart failure regimen. The case illustrated the challenges in differentiating myocarditis from sarcoidosis given the various diagnostic modalities available.

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CORONARY ARTERY ANEURYSM IN A PATIENT WITH ACUTE LATERAL WALL MYOCARDIAL INFARCTION.
Csehak K, MD, Cardona S, MD. University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Coronary artery aneurysm is an uncommon condition with multiple studies suggesting an incidence of 1 to 5% among patients receiving coronary angiography. An aneurysm is commonly defined as a localized dilation exceeding the diameter of adjacent normal segments by 50%.

Atherosclerosis is the most common cause of coronary artery aneurysms, followed by vasculitides including Kawasaki disease and polyarteritis nodosa. Systemic lupus erythematosus, infection, trauma, congenital malformations and iatrogenic causes represent additional etiologies. Coronary artery aneurysms are clinically important as they can cause death when they thrombose or rupture.

A 54-year-old woman presented to the Emergency Department at an outside hospital with acute onset, left-sided chest pain that awoke her from sleep, with associated nausea and vomiting. Initial electrocardiogram demonstrated 3mm ST segment elevations in leads I and aVL with reciprocal ST segment depressions in leads III and aVF, consistent with acute lateral wall myocardial infarction. The patient received aspirin, clopidogrel, was started on a heparin infusion, and transferred to the University of Maryland Medical Center for emergent coronary angiography. Angiography revealed a large, ectatic left main coronary artery and a large aneurysm of the proximal left anterior descending (LAD) coronary artery with patent flow. The second diagonal branch appeared occluded, however, because this vessel arose from the aneurysmal segment, no clear origin could be seen and no interventions were attempted. Subsequent computed tomography angiography of the coronary arteries again revealed an 18mm partially thrombosed aneurysm involving the proximal LAD and proximal first diagonal branch. During admission, the patient was continued on dual anti-platelet therapy with aspirin and clopidogrel, along with a heparin infusion while warfarin was initiated to reach a therapeutic International Normalized Ratio of >2.0. Due to the location of the aneurysm, surgical intervention was deemed high-risk and the patient was discharged on clopidogrel and warfarin for continued medical management of her coronary artery aneurysm. She was asymptomatic at the time of her discharge.

While coronary artery atherosclerosis is a ubiquitous disease of adults, coronary artery aneurysm is a much less common condition but can have similar clinical implications.
# SWEET SYNDROME: A CLASSIC CASE OF A CLASSICAL VARIANT

*Mark Marchitto, MD; Sauradeep Sarkar, MD; Gregory Vo, MD; Mitchell Klapper, MD; Sinai Hospital of Baltimore*

Sweet syndrome (SS), or acute febrile neutrophilic dermatosis, is a rare inflammatory disorder associated with fever and painful skin lesions. Three variants have been described: malignancy-associated, drug-induced, and classical. Classical, or idiopathic SS, typically affects females aged 30 to 50, and is associated with infections (notably upper respiratory tract or gastrointestinal), inflammatory bowel disease, or pregnancy. Steroids are the mainstay treatment and often result in dramatic symptom improvement. SS is diagnosed using a specific criteria that must include both major criteria (abrupt onset of painful plaques and nodules, and histopathologic evidence of neutrophilic infiltration), and at least two minor criteria (pyrexia, response to steroids, associated or underlying process, and elevated inflammatory markers and/or neutrophil-predominant leukocytosis).

A 31-year-old female with no medical history presented with painful skin lesions involving her extremities and back, she also reported a flu-like illness 2-weeks prior with subjective fever, sore throat, and myalgia. She had no prior history of similar symptoms, nor family history of autoimmune, rheumatic, or connective tissue disease. On admission, vital signs were normal. Skin examination revealed multiple indurated and erythematous plaques involving the bilateral upper and lower extremities and back. Given the unknown etiology of the patient’s symptoms, a broad workup was ordered and was notable for a neutrophil-predominant leukocytosis, elevated inflammatory markers, and a markedly elevated antistreptolysin-O antibody, however strep culture was negative. On evaluation by dermatology, there was high suspicion for SS as the patient fulfilled the majority of the diagnostic criteria, so the patient underwent a bedside skin biopsy which revealed a neutrophilic dermatosis, confirming SS. The patient was started on steroids and her symptoms improved rapidly.

This case is a “classic” presentation of an uncommon condition, classical variant SS, as all of the criteria were fulfilled for diagnosis. The precipitating event was likely a resolved streptococcal or viral upper respiratory tract infection given the patient’s preceding symptoms. Despite the classic presentation, SS still remains uncommon with a constellation of seemingly nonspecific symptoms. When approaching similar cases, particularly in the setting of an unrevealing workup, it is important to maintain a broad-differential to reduce the risk of premature closure and overlooking a rare diagnosis.

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