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<th>Medical Students</th>
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**Quality Improvement - Medical Students**

**Nathan Stratton**  
**Michael Perlmutter**

*Protocolized Fluid De-Resuscitation of Patients Admitted to ICU with Sepsis and Septic Shock using an EHR Best Practice Alert*

Objective: Increase the use of IV furosemide to reduce iatrogenic harm and re-admission in patients receiving ≥6 liters of crystalloid in the course of resuscitation for treatment of sepsis or septic shock in the ICU.

Background: There is extensive evidence to guide care during initial ED and ICU resuscitation. Protocolized resuscitation of adults admitted to an intensive care unit with suspected sepsis is characterized by the initial administration of large volumes of IV fluids and ongoing infusion of maintenance fluids, often beginning during acute resuscitation in the Emergency Department and continuing during ICU admission and stay. However, there is a paucity of evidence to guide de-resuscitation following the acute phase of illness. Recent studies suggest that outcomes are improved in this population from protocolized diuresis to reduce the incidence of iatrogenic complications secondary to fluid overload. A review of patient data in our healthcare system demonstrated that approximately 50% of patients hospitalized with a diagnosis of sepsis or septic shock were discharged without diuresis following fluid resuscitation.

Method: A quality improvement initiative at one facility used an electronic health record (EHR) Best Practice Alert (BPA) notify physicians of potential patients who were candidates for diuresis during the course of admission for sepsis or septic shock. The BPA was activated for patients who had a net fluid input above 6L since admission and were without previous diuresis during the hospital course. It excluded those requiring vasopressor support or who had evidence of renal insufficiency. Physicians received an alert regarding patients meeting criteria and were able to order a 10mg dose of furosemide from the alert window if desired.

Results: Following a three month pre-implementation surveillance period, BPA testing and physician education, the tool was made visible to clinicians for the next 5 months. After implementation, 73% of the patients who were identified by the process received furosemide prior to discharge compared to an estimated 25% during the surveillance period. A chart review of patients triggering the alert who did not subsequently receive diuresis revealed that many had contraindications to furosemide which were too complex to account for in the design of this QI process.

Conclusion: The use of a Best Practice Alert in the EHR to notify physicians of patients who could benefit from diuresis resulted in an increase in the use of pre-discharge IV diuretics for patients with high net-positive fluid input status during an admission for sepsis or septic shock.
Minnesota Physicians’ Familiarity and Use of Provider Order for Life Sustaining Treatments (POLST)

Introduction: Provider Orders for Life-Sustaining Treatment (POLST) forms are a means of aligning end of life care with a patient’s wishes to avoid unwanted and unnecessary medical intervention. Currently, there is no data on how the POLST system is being utilized and its impact on Minnesota medical care. The purpose of this study was to survey Minnesota physicians to determine the utilization, benefits, and concerns associated with the POLST form in Minnesota.

Methods: A total of 6,526 physicians identified from the Minnesota Medical Association’s physician registry were given the opportunity to participate in the study. A survey was generated to determine familiarity, utilization, perceived benefits, and concerns with the POLST (Figure 1). Demographic variables for the physicians were also collected including specialty, practice location, and years since medical school graduation. Practice location was defined as Twin Cities, and non-Twin Cities. Results were analyzed using Student’s T-test. All analysis was performed using Stata (College Station, Texas).

Results: A total of 656 physicians completed the survey (10%). There were 363 physicians (55%) who practice at least partially in the Twin Cities and 293 (45%) outside the Twin Cities. The most common specialties were family medicine (n=157, 24%) and internal medicine (n=127, 19%). Overall, 401 physicians (61%) were familiar with the POLST. There was no significant difference in familiarity between practice locations (n=222, 61% for Twin Cities versus n=179, 61% for non-Twin Cities, p=0.988). Of physicians who were familiar with the POLST, 295 (74%) use the POLST in their practice. The most commonly endorsed problems with the POLST were patients being unaware of the form (n=140, 47%) and patients not understanding the purpose of the form (n=127, 43%). The most common endorsed benefits of the POLST were that it allows patients to express wishes about care (n=250, 85%), avoids unnecessary treatment (n=253, 86%), and facilitates end of life planning (n=251, 85%).

Discussion: The results of this study suggest that physicians in Minnesota are equally familiar with the POLST in the Twin Cities and outside the Twin Cities. Many of the physicians who are aware of the form are using it in their practice. The benefits the respondents indicated align with the stated objectives of the POLST, demonstrating that the form is having the desired effect on patient care. The most common concerns with the POLST were related to patient awareness surrounding the form, indicating a potential need for further public outreach and education about the POLST.

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This study is the first to report on the utilization, benefits, and concerns associated with the POLST form in Minnesota. These results will guide future
Interventions to improve the POLST as well as future studies to determine additional information surrounding POLST use in Minnesota. Twenty-one residents completed the post-intervention survey. The high intensity group (n=12) reported greater interest in similar curricula in the future (50% vs 22%), and rated the SP encounters as beneficial to their communication skills (83% vs 44%). They were more likely to report incorporating feedback from SPs (83% vs 44%) and faculty observers (75% vs 55%) into subsequent patient encounters. After a year of interventions, SPs evaluated residents with the CARE empathy checklist following face-to-face encounters. SPs rated the high intensity group higher in: making the patient feel at ease, letting patients tell their story, providing clear explanations, and helping take control of the patient encounter. There was no difference in overall performance between the two groups on the CARE empathy checklist (high intensity mean 27 vs 28 for standard). Our intervention provided focused training on the communication of value-based decision making. Learners reported benefits in their communication skills and increased likelihood of incorporating skills into future patient encounters. In patient simulations they utilized more patient-centric methods. However, there was no difference in overall performance on an empathy checklist. Future work will focus on validation of a four-habits based behavioral check list, training of video raters, and review and scoring of a second video encounters to assess difference in skills in the high intensity versus the standard curriculum groups.

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<th>Hana Manzoor</th>
<th>Differences in Clinical, Diagnostic and Treatment Characteristics of Hyperthyroidism in Immigrant Population from Iodine Deficient and Sufficient Countries</th>
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<td>Dr. Zaigham Rana</td>
<td>Introduction: Hyperthyroidism is a common global endocrine disorder with varying prevalence estimated to be from 1.6 % - 8 %. Among genetic and autoimmune factors, exposure to iodine (or lack thereof) may be responsible for such variabilty. Furthermore, severity of iodine deficiency leads to compensatory thyroid hyperplasia and possible underlying autonomy which may lead to development of hyperthyroidism upon exposure to sufficient or excessive iodine. We studied the differences in clinical, diagnostic and treatment characteristics of diffuse hyperthyroidism in immigrant population from iodine sufficient and iodine deficient countries.</td>
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<td>Dr. Warda Niaz</td>
<td>Methods: Retrospective observational study conducted at the center for Diabetes, Endocrinology and Metabolism at Hennepin Healthcare System (HHS), Minneapolis, MN. Electronic medical records of patients underwent I 123 thyroid uptake and scan with uptake of 10% or more at 4 hours or 30% or more at 24 hours and diffuse scan distribution between January 1, 2004 and December 31, 2014 were reviewed. Patients were then categorized into either from iodine sufficient (IS) or deficient (ID) countries. Clinical, biochemical, imaging and treatment variables and responses were analyzed. Results:A total of 235 patients (178 [75.7%] from IS and 57 [24.2%] from ID countries) were included. Mean age of patients in IS group was lower compared to ID group [38.1 vs. 46.4 respectively, (95 % CI for difference -13.36 to – 3.123), p-value: 0.002]. Higher proportion of female patients were in ID group [86% vs. 69.7% respectively, (95% CI for difference 0.08 to 0.24), p-value: 0.015]. Smoking and eye disease were noted to be dependent on each other in both groups (chi-square test for independence, p-value: &lt;0.0001). High titer s of thyroid peroxidase (TPO) antibody were noted in IS group [72.2% vs. 37.1% (95% CI for difference 0.19 to 0.51), p-value: &lt;0.0001] whereas high titer s of thyroid stimulating immunoglobulin (TSI) level were not significantly different between the groups. Patients in ID group received higher dose of I 131 therapy compared to IS group [16.49 vs. 11.56, (95% CI for difference -7.55 to -2.32), p – value: &lt;0.0001. Conclusion: Immigrant patients with hyperthyroidism demonstrate difference in terms of age of onset, gender, autoimmunity and dose of I 131 treatment depending upon their country of origin in terms of iodine exposure.</td>
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Correlation of Exhaled Nasal Nitric Oxide to Sinus CT and Sinonasal Outcome Test Scores: A Prospective Study

Introduction: Pancreatic cancer remains among the most aggressive cancers due to the unknowns regarding its physiologic characteristics. Patients diagnosed with pancreatic cancer exhibit significant mortality rates, even in cases of resectable disease and approximately 10% of patients with pancreatic cancer don’t produce the classical marker, CA19-9. Significant effort has been dedicated to identifying more reliable tumor markers for screening and assessment of treatment response. Detection and quantification of ctDNA in patients with pancreatic cancer remains a novel and unexplored avenue as its usefulness and reliability are unknown, making it the subject of ongoing research. In this study, researchers examined correlations between ctDNA levels, CA19-9 levels, and tumor size changes in 17 patients receiving treatment at Banner MD Anderson Cancer Center in Gilbert, AZ.

Methods: Present study is approved by our local IRB. Research was conducted in accordance with HIPAA regulation. Subjects on the present study were obtained from patients participating in ctDNA trial. 17 subjects qualified based on data available. The ctDNA index levels were obtained from the sponsor, Chronyx, baseline and post-treatment cycle values. These ctDNA levels were correlated to CT scans performed. CT scan and ctDNA were considered at equal time points if they were obtained less than or equal to 4 weeks apart. The sizes of the primary pancreatic tumor and size of the largest metastatic lesion were also used for analysis. Size was measured using transverse images with perpendicular measurements of each lesion used to calculate the area. Data was analyzed with Spearman’s correlation.

Results: Baseline ctDNA levels did not correlate with patient demographic data (N=17; gender, p=0.63; age, p=0.82), baseline size of primary mass on CT scan (N=16; p=0.85), baseline vessel involvement on CT Scan (N=17; p=0.58), presence of metastasis on CT scan (N=17; p=0.78), size of largest metastasis on CT scan (N=17; p=0.85), presence of peripancreatic lymph nodes on CT scan (N=17; p=0.45), or overall survival (N=8; p=0.6). However, there appears to be a correlation regarding a decrease in ctDNA and decrease in overall tumor area as calculated from CT scans following treatment (N=7; p=0.12).

Conclusion: Based on statistical analysis, ctDNA appears to be secreted independent of the primary tumor size, location, or presence of metastasis. However, individual analysis does show that changes in ctDNA seem to correlate with changes in tumor size following treatment. The low sample size of this study is likely the reason for statistically insignificant results. A larger sample is needed to thoroughly analyze the correlation and relationship between pancreatic cancer progression and ctDNA.

Pulseless Legs and Acute Respiratory Failure: An Unusual Presentation

Introduction: Peripheral artery disease (PAD) is a common condition affecting 3 to 12% of individuals worldwide, with 413,000 inpatient admissions yearly in the United States and Europe. Alternate diagnoses are often overlooked when peripheral pulses are weak. It is imperative that physicians be aware of rare and potentially life-threatening conditions that can mimic PAD, especially in patients with atypical presentations.

Case: A 33-year-old Micronesian female presented to the emergency department with one week of worsening dyspnea. Her past medical history was significant for hypertension and infertility. On admission, she was tachycardic
and tachypneic with a blood pressure of 219/175 mmHg. Her oxygen saturation was 68% on room air and she required intubation. She developed two episodes of pulseless electrical activity requiring CPR. A chest X-ray revealed diffuse bilateral ground glass opacities. Acute respiratory distress syndrome (ARDS) from an infectious or inflammatory cause was high on the differential. She was treated with empiric antimicrobials. Cultures of blood, bronchial washings, and urine were unremarkable. ANA, p-ANCA, c-ANCA, coccidioides PCR, and antiphospholipid antibodies were also negative. Shortly after admission, she developed anuric acute kidney injury (AKI) requiring intermittent hemodialysis. As her blood pressure control improved, lower extremity pulses were lost. Doppler showed absent flow in the lower extremities below the groin. CT chest revealed severe focal mural and intraluminal calcification/stenosis of the distal descending aorta. CT angiography revealed an acute thrombotic occlusion at the site of stenosis. It became evident that her presentation was consistent with flash pulmonary edema secondary to severe hypertension above the level of occlusion. This suprarenal occlusion also explained her AKI. She successfully underwent an axillary to right common femoral artery bypass to re-establish perfusion, with plan for future definitive repair. She improved significantly post-operatively, and was discharged home with no sequelae.

Conclusion: This patient’s CT findings are consistent with a rare yet well-defined disease entity by the name of Coral Reef Aorta. This phenomenon is characterized by the presence of internally protruding calcifications involving the suprarenal or juxtarenal aorta, resulting in significant obstruction of the lumen. Only about 50 cases have been reported in the literature thus far. Chronic manifestations include renovascular hypertension, intermittent claudication, and visceral ischemia. Patients with this disease may develop acute complications including upstream hypertensive emergency, downstream embolic events, and multiple organ failure. It is a life threatening condition with in-hospital mortality rate of 13.3%, requiring emergent revascularization. Early CT in patients with suspected coral reef aorta is critical for timely surgical management. During critical illness, mean arterial pressure targets may need to be considerably higher than normal to maintain lower limb perfusion. Familiarizing physicians with this rare entity is important, as it could be rapidly fatal in the absence of prompt recognition and management.

Corey Babcock

**When Tissue is not the Issue, but Culture is the Vulture: Invasive Actinomyces Causing Incomplete Paraplegia**

Case: A previously healthy 66-year-old man presented with a several-week history of progressive right upper chest pain, dyspnea, and hemoptysis. He also noted odynophagia and a 15-pound weight loss. History was only notable for a 30 pack-year smoking history (quit six months ago) and recent tooth extraction a few months prior. A chest CT revealed a “very large malignant-appearing right apical mass with both mediastinal and chest wall invasion” with significant mediastinal lymphadenopathy. Labs were notable for a leukocytosis of 18.7 with neutrophilic predominance, thrombocytosis of 725, and a microcytic anemia. An IR-guided lung biopsy was obtained and revealed only chronic inflammation with no definitive diagnosis.

Approximately one month later the patient presented with two days of worsening right greater than left lower extremity weakness and numbness, with lower extremity exam revealing 3/5 strength, decreased muscle tone, and clonus. Leukocytosis and thrombocytosis had worsened to 28 and 1336, respectively. Thoracic spine MRI showed an extensive lung mass that had now invaded into the thoracic spine with spinal cord compression, and repeat physical exam revealed 1/5 hip flexor strength bilaterally. He was given a fraction of radiation to reduce cord compression. A CT-guided chest wall biopsy was obtained, again negative for malignancy, but anaerobic culture of this biopsy came back positive for gram-positive filamentous bacteria
compatible with Actinomyces meyeri. He was started on dexamethasone taper and IV meropenem, subsequently narrowed to penicillin G. Neurosurgery performed multi-level thoracic laminectomy with epidural abscess incision and drainage. At the time of submission, the patient has undergone around one month of spinal cord rehabilitation with minimal improvement in lower extremity strength.

**Conclusion:** This case highlights an extremely rare presentation of Actinomyces infection. The delay in proper treatment for this patient may in part be related to the significant degree of persistent paraparesis. Therefore, it is important to keep a broad differential diagnosis in the context of a new mass crossing tissue planes.

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<th>Baril Jackson</th>
<th>Fever of Unknown Origin After Treatment for Sepsis</th>
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<td>Dr. Sam Ives</td>
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Introduction: Fevers of unknown origin (FUO) are defined as fevers > 38.3°C in an immunocompetent patient on multiple occasions over at least a 3 week period without explanation. The diagnosis is usually made after extensive infectious workup. We report a case of FUO with a rheumatologic cause after a diagnosis and treatment of sepsis did not eliminate fevers.

Case: A 69-year-old Hmong woman with a history of GERD, diabetes mellitus type 2, peptic ulcer disease, intermittent asthma, anemia, paranasal sinusitis, hypertension and recent hospital stay for pyelonephritis and a peroneal DVT presented to the hospital after recurrent falls and acute shortness of breath. She was found to have sepsis secondary to a proteus mirabilis UTI, elevated INR secondary to warfarin, and patellar fracture. Treatment included IV antibiotics, vitamin K, and EGD for upper GI bleed. Chest x-ray was concerning for aspiration post-EGD which was covered by antibiotics. The morning of anticipated discharge to a SAR, she was vomiting, febrile to 104 F, tachycardic to 130s, and hypotensive with SBPs between 80 and 90. On exam she was confused, tremulous, wheezing and toxic appearing.

The patient was transferred to the ICU and treated with antibiotics, vasopressors, and ventilator support. She was found to have adrenal insufficiency, elevated inflammatory markers, eosinophilia, fevers, encephalopathy, and no focal neurological deficits. Upon transfer to the floor a week later, further infectious workup was negative, and antibiotics were stopped. Nightly fevers to 102 F persisted with day spikes to 104 F for another week. Urine culture was positive for enterobacter aerogenes. She required a second transfer to ICU. Hematology-oncology and rheumatology were consulted. Studies included a positive PR3 titer but negative MPO titer and normal bone marrow biopsy. With the history of worsening asthma in the last year, prior paranasal sinusitis, current FUO, and intermittent peripheral eosinophilia up to 12% (1.1 of total WBC 8.8) during the hospitalization, the diagnosis of eosinophilic granulomatosis with polyangiitis or other steroid-responsive eosinophilic syndrome was made.

The patient was treated with 500 mg IV methylprednisolone daily for 3 days followed by oral steroids resulting in marked clinical improvement. After significant deprescribing on the floor, the patient was discharged to a rehab center. She is followed by rheumatology as an outpatient on a steroid taper with plans to switch to a steroid free regimen.

Discussion: Fever of unknown origin is of rheumatologic etiology in approximately 30% of cases. Although patients may have signs and symptoms of infection, they can also meet criteria for fever of unknown origin when they do not improve with treatment of infections. In these cases, care teams should consider other reasons for fever, including rheumatologic and oncologic causes, and pursue appropriate work-up and consultation.
**A Simpler Answer – After All This Time**

Introduction: There is an allure associated with rare diagnoses in medicine. Diagnosis momentum refers to the “stickiness” of a diagnosis once attached to the patient, and is one example of cognitive bias that can lead to diagnostic errors. This can lead to misattribution of symptoms to a patient’s existing (even incorrect) diagnosis if clinicians are unaware of this tendency and do not keep their initial approach adequately broad.

Case: A 55-year-old woman with history of rheumatoid arthritis and longstanding diagnosis of acute intermittent porphyria (AIP) presented to the emergency department with acute recurrence of abdominal pain. Since her AIP diagnosis 20 years ago, she has had intermittent episodes of abdominal pain attributed to porphyria. Each episode was like the original one, she never experienced any neurological symptoms. These episodes increased in frequency and intensity, and she sought a hematology consult for worsening AIP. While workup was ongoing, she had three emergency room visits in which episodes were treated with fluids, analgesics, and anti-emetics. Finally, at the third visit her pain was noted to be localized to the right upper quadrant and evaluation revealed transaminitis and hyperbilirubemia. Abdominal ultrasound showed evidence of cholecystitis.

Urine porphobilinogen collected during this episode was normal, effectively eliminating the possibility that AIP was the cause of her abdominal pain. She was admitted and underwent endoscopic retrograde cholangiopancreatography, which demonstrated filling defects consistent with choledocolithiasis. After undergoing cholecystectomy during hospitalization, her bilirubin and transaminases normalized. At follow-up, there was no remaining evidence of inflammation and laboratory work remained within normal limits.

Discussion: This case is important because it highlights the potential negative impacts of cognitive biases on diagnosis. Clinicians frequently use shortcuts when making clinical decisions, and usually these shortcuts serve them well. However, it is important that the clinician is aware of the shortcuts that impact decisions and has practical means of correcting errors that may arise as a result. Diagnosis momentum probably had a large role in the long-term misdiagnosis in this case. In particular, when a disease is rare there is a tendency to attribute all symptoms to that disease and not look for alternative explanations. If the disease is infrequently encountered, it may also be difficult to identify when a symptom does not fit and alternative explanations are needed. It is therefore important to have robust illness scripts even for rare conditions.

Internists make diagnoses every day, and shortcuts make that job quicker and easier. However, as this case demonstrates, a potential pitfall is misdiagnosis and possible long-term delay in treatment. It is therefore in every clinician’s interest to be aware of such errors so the impact can be minimized and if errors occur they can be addressed as quickly as possible.

**Elusive Etiology of Paraneoplastic Hypereosinophilic Syndrome**

Introduction: Eosinophilia is a common hematological finding and is associated with several disease states. It is most commonly reactive, but a wide range of less common and potentially fatal etiologies exist, including paraneoplastic syndromes. Regardless of the etiology, eosinophilia can progress to hypereosinophilic syndrome (HES), in which eosinophils persist in tissue, directly causing organ dysfunction. Paraneoplastic-induced HES is rarely reported, thus a case is presented of non-small cell lung cancer (NSCLC) leading to paraneoplastic HES mediated by GM-CSF.

Case: A 70-year-old with prostate cancer and a 50 pack-year smoking history...
presented to the ED with two weeks of non-productive cough and fatigue. Physical exam was unremarkable. The patient had mild leukocytosis (15.3; abs Eos: 0.6) and a chest x-ray suggestive of emphysema. He was treated for viral bronchitis and referred for COPD work-up.

At a routine oncology appointment three months after his ED presentation, his white count was 47.1 (abs Eos: 22). Further evaluation revealed elevated inflammatory markers, IgE, GM-CSF, and alkaline phosphatase. IL-5 was normal. His work-up was negative for infectious/autoimmune processes, myeloproliferative malignancy, and karyotype/cytogenetic abnormalities. CT chest/abdomen/pelvis without contrast (Cr: 1.4) was notable for gastric wall thickening and spiculated lung nodules (2-4mm).

Over the next 6 weeks, the patients’ abs Eos rose to 70. He was treated for HES, and two weeks later, he was admitted to the hospital (abs Eso: 18; platelet count: 33). EGD and stomach biopsy were negative. Cardiac MRI showed a left ventricular thrombus and multifocal hepatic masses, prompting a CT chest/abdomen/pelvis with contrast, which revealed multiple hepatic masses, osseous metastases, and pulmonary nodules. Poorly differentiated metastatic carcinoma consistent with lung adenocarcinoma was observed on subsequent liver biopsy.

Six months following the patients’ initial presentation, he was diagnosed with paraneoplastic GM-CSF mediated eosinophilia leading to HES secondary to metastatic NSCLC (stage IVb adenocarcinoma, T1aNxM1c; PDL-1 100% positive). The patient received chemotherapy with Pembrolizumab and Vinorelbine. To date, the patient’s cell counts have stabilized, and there has been an interval reduction in the size of hepatic metastases. However, potential peritoneal carcinomatosis as well as escalating pulmonary involvement have been reported. At the present time, the patient is in modified hospice care.

Discussion: Accurate diagnosis of non-reactive eosinophilia and HES can prove to be a challenging task. This case illustrates an elusive diagnosis of HES etiology, in which certain test results did not add up, including early elevated alkaline phosphatase, prompting extensive evaluation. The diagnosis of paraneoplastic-induced HES is a rare subset of eosinophilia, but similar to other less common causes of HES, it requires treatment targeting the underlying etiology. Thus, in cases of eosinophilia with unclear etiology, intentional evaluation for the underlying causative pathologic process is necessary to provide appropriate and effective treatment.

Christopher Dinh
Aunika Swenson
Dr. Deanne Kashiwagi

Mitochondrial Neurogastrointestinal Encephalopathy: A Rare Cause of Gastroparesis

Case Presentation: A previously healthy 24-year-old female from Saudi Arabia presented to Mayo Clinic for evaluation of five years of severe progressive idiopathic gastroparesis. Her symptoms began with right lower quadrant pain, diarrhea, vomiting, and weight loss. These progressed to the inability to tolerate oral intake and her BMI decreased to 9. She had extensive outside testing including two diagnostic laparoscopies, two colonoscopies, and CT enterography which showed thickening of the distal ileum. Upper endoscopy, celiac panel, autoimmune testing, and vasculitis workup were all unrevealing. Barium study showed massive gastric dilation, and MRI and gastric emptying study confirmed severe gastroparesis. Capsule endoscopy was attempted but required surgical intervention to advance the capsule to the colon with intraoperative findings of an underdeveloped bowel. She attempted multiple empiric treatments without improvement including antibiotics, budesonide, azathioprine, and domperidone. She had no family history of gastrointestinal disease, though her parents were cousins.

At our institution, autoimmune, endocrinologic, hematologic, and infectious workup were negative with the exception of C. difficile, which was treated.
Further testing revealed elevated urine organic acids, uracil, and thymine, suggestive of an inborn error of metabolism. TYMP gene sequencing found a previously unreported homozygous variant c. 1048C>T, predicted to be pathogenic due to premature protein termination. MRI brain revealed leukoencephalopathy. These findings were consistent with Mitochondrial Neurogastrointestinal Encephalopathy.

Discussion: Mitochondrial Neurogastrointestinal Encephalopathy (MNGIE) is a rare, autosomal recessive genetic disease caused by mutations in the TYMP gene which encodes for thymidine phosphorylase. Fewer than 200 cases have been reported in the medical literature as of 2011. Loss of this enzyme results in accumulation of thymidine and deoxyuridine and causes an imbalance in mitochondrial (mtDNA) deoxyribonucleoside triphosphates leading to mtDNA mutations and reduced mtDNA synthesis. Impaired mitochondrial function leads to the gastrointestinal and neurological problems characteristic of this disease.

Many patients present with nonspecific gastrointestinal symptoms such as abdominal pain, early satiety, nausea, dysphagia, GERD, diarrhea, and weight loss. The differential diagnosis includes IBD/IBS, intestinal pseudo-obstruction, celiac disease, and anorexia nervosa. As the disease progresses, symptoms become more severe and include gastroparesis, bacterial overgrowth, malnutrition, cachexia. Neurologic manifestations include peripheral neuropathy, ophthalmoplegia, ptosis, lower extremity weakness, and sensorineural hearing loss. Leukoencephalopathy is a classic imaging finding.

Currently there are no curative treatments for MNGIE. Management is mainly supportive and involves nutritional support, airway protection, avoiding medications that interfere with mitochondrial function, neuropathic pain treatment, and physical/occupational therapy to preserve mobility and independence. Experimental therapies include hemodialysis and peritoneal dialysis to remove excess nucleosides, and liver and hematopoietic stem cell transplantation to restore thymidine phosphorylase levels.
chemotherapy for his new diagnosis of metastatic mesothelioma and since starting treatment has not had any recurrent joint symptoms.

Discussion: Acute-onset arthritis is a rare medical emergency that requires rapid evaluation and treatment with empiric antibiotics since septic arthritis is often rapidly destructive. Prompt synovial fluid analysis is crucial to determine etiology. Despite this, only 50% of patients with polyarticular arthritis receive a definitive diagnosis. Pattern of joint involvement and clinical course can assist with correct diagnosis. Our patient had an acute-onset asymmetric, migratory, polyarticular arthritis of medium and large joints lasting 1-2 days in each location. The differential diagnosis for synovial fluid with a white blood cell count greater than 100,000 cells/mm3 is septic arthritis, crystal-induced arthritis, or leukemic infiltration. Synovial fluid showed classic findings of septic arthritis with significantly elevated WBCs, neutrophil predominance and no crystals, but when gram stain and culture showed no organism his history and clinical course helped refine the differential diagnosis. His continued clinical improvement despite discontinuing antibiotics and lack of recurrence is suggestive of inflammatory arthritis secondary to paraneoplastic disease.

The Milky Way to My Heart

Introduction: Amyloid cardiomyopathy is an uncommon disorder with high mortality rates, particularly in light-chain amyloidosis. Its non-specific symptoms and uncommon incidence makes for a unique diagnostic challenge.

Case: A 70-year-old man was referred to the Mayo Clinic with 5 months of anasarca and recurrent chylous ascites and left chylothorax. He had symptoms of dyspnea, orthostatic hypotension, bloating, early satiety, 20 pound unintentional weight loss, anasarca, and left periorbital purpura. He was a 25 pack-year current smoker with a past medical history including type 2 diabetes mellitus, hypothyroidism, and coronary artery disease status-post coronary artery bypass graft 2 years prior. Physical exam revealed left periorbital purpura, decreased breath sounds in left lower lobe, protuberant abdomen without tenderness, thickening of abdominal wall and skin of extremities with 3+ pitting edema to pelvis, with normal cardiac exam and no appreciable lymphadenopathy. Significant labs included hemoglobin 12.7, serum albumin 2.3, alkaline phosphatase 271, NT-proBNP 23,354 pg/mL, and high sensitivity troponin 189 ng/L. The total ascitic protein was 2.5, ascites albumin 1.0, with a serum-ascites albumin gradient (SAAG) of 1.3 The elevated SAAG and total ascetic protein of 2.5 suggested a cardiovascular etiology. Subsequent transthoracic echocardiogram revealed increased LV and RV wall thickness, biventricular systolic dysfunction, and strain pattern consistent with amyloidosis. Fat pad biopsy found a peptide profile consistent with AL (kappa)-type amyloid deposition, and liver biopsy showed amyloid deposition in portal tracts and vessel walls consistent with hepatic amyloidosis, but not meeting criteria for cirrhosis. He was ultimately diagnosed with AL (kappa light chain) cardiac and hepatic amyloidosis. The patient was prescribed furosemide, spironolactone, midodrine, and cyclophosphamide-bortezomib-dexamethasone therapy, with a recommendation to follow a medium chain triglyceride diet and to take octreotide as temporizing measures to manage the ascites while awaiting the effects of treatment.

This case illustrates the utility of SAAG and total ascitic protein concentration to narrow the differential when complex patients present with chylous ascites and pleural effusion. Although uncommon, systemic amyloidosis is an important disease to consider with the presenting symptoms of this case, as recognition of this syndrome is critical to make a timely diagnosis and provide disease-directed therapy.
Andrew Hughes  
Dr. Chetan Shenoy  

A Tale of Three Stress Tests: Making the Right Choices in Diagnostic Testing

Learning Objective: Cardiac stress tests are frequently used. There are many options for stress testing. Physicians must understand the pros and cons of the various stress tests in order to make personalized choices for each patient.

Case Presentation: A 46-year-old morbidly obese (BMI 55) male had atrial fibrillation incidentally noted on a DOT physical exam. In retrospect, he experienced exertional dyspnea for 6 months prior to the diagnosis. He was otherwise asymptomatic from a cardiovascular standpoint, and he was physically active. His family history was significant for premature coronary artery disease but the remainder of his risk factor profile was unremarkable. An exercise stress test was ordered at a community hospital. At the stress test, his heart rate was in the low 100s due to atrial fibrillation. Therefore, the exercise stress test was stopped and switched to a pharmacological nuclear stress test. The nuclear stress test was a “very suboptimal study due to the patient’s large body habitus, suboptimal normalization, and significant soft tissue attenuation.” Myocardial perfusion imaging identified two abnormalities: a small non-transmural infarct and a small reversible defect consistent with moderate ischemia. The left ventricular ejection fraction (LVEF) was 39%. Based on the nuclear stress test, he was referred to the University of Minnesota for further evaluation. Coronary angiography was considered, but given the very suboptimal nuclear stress test, a stress cardiovascular magnetic resonance imaging (CMR) was performed instead. It demonstrated a LVEF of 52%, no ischemia, and no infarction. Despite his large body habitus and atrial fibrillation, the CMR was of optimal image quality. After 3 years of follow up, he is alive without experiencing any adverse cardiovascular events.

Discussion: There are several choices for stress testing including: exercise ECG, stress echocardiography, nuclear perfusion imaging, and stress CMR. Each of these options has pros and cons, which should be considered in the context of the individual patient. Physicians should select the appropriate stress test based on clinical features (i.e. obesity or ability to exercise), and potential risks (i.e. radiation exposure with nuclear stress test). Stress testing in obese patients is challenging. Stress echocardiography has poor acoustic windows. Nuclear stress tests have suboptimal image quality due to attenuation artifacts and decreased signal-to-noise ratio. In contrast, the diagnostic and prognostic value of vasodilator stress CMR is not impacted by obesity. As illustrated in this case, physicians are often faced with choices regarding which diagnostic approach to pursue for their patients. By understanding the pros and cons of various diagnostic options, physicians can appropriately utilize healthcare resources, obtain more efficient diagnoses, and expose their patients to fewer risks.

Joshua Labott  
Dr. Park Zheng  
Dr. Bassim El Sabawi  
Ashley A. Peterson  
Annabelle K. Soares  
Dr. Jaime de la Fuente  
Virginia Dines

Painless Jaundice: The Sudden Onset of Jaundice, Coagulopathy, and Worsening Diabetes

Introduction: Jaundice is the second most common presenting symptom of pancreatic adenocarcinoma in the head of the pancreas, with 82% of patients presenting with jaundice.1 For the general internist, it is important to recognize jaundice as a possible presenting sign of pancreatic cancer.

Case: We present a case of an 82-year-old Caucasian female who presented with painless jaundice. She had a past medical history of metformin controlled type 2 diabetes and atrial fibrillation on warfarin. The patient initially presented to her local emergency department (ED) with 24-hours of painless jaundice and 2 weeks of fatigue. Initial labs were significant for an INR >8.0, glucose 497mg/dl, total bilirubin 17.9mg/dl with direct bilirubin 13.6mg/ml, AST 143U/L, ALT 132U/L, alkaline phosphatase 1339U/L, and lipase 74U/L. The patient was admitted after administration of intramuscular vitamin K.
On admission, physical exam was remarkable for jaundice, scleral icterus, and hepatomegaly without tenderness or guarding. CT abdomen and pelvis revealed a pancreatic head mass with marked biliary and pancreatic duct dilatation. Further work up, including ERCP and FNA, allowed for both diagnostic and therapeutic processes. A biopsy was taken and a covered metal stent was placed in the common bile duct, relieving the bile duct obstruction. The patient’s supratherapeutic INR resolved after the stent was placed, warfarin was held, and vitamin K was given.

The patient’s jaundice began to resolve drastically 24 hours after ERCP stenting with new total bilirubin of 7.2mg/dl. FNA biopsy resulted in pancreatic adenocarcinoma. She was discharged with oncology and palliative care follow-up.

Discussion: This case illustrates the importance of being familiar with the evaluation of painless jaundice as well as the potential presenting symptoms of pancreatic adenocarcinoma located at the pancreatic head. The delayed presentation and diagnosis is associated with a very poor prognosis with a 5-year survival of 7% with all stage survival rates combined. As in this case, tumors located in the head and neck of the pancreas can obstruct pancreatic and biliary ducts. This slow obstruction causes jaundice with no associated pain. Supratherapeutic INR can be present if there is a significant obstruction, as vitamin K is a fat soluble vitamin and requires bile acid secretion for proper absorption. This is exacerbated even further if patient is taking vitamin K antagonists. Also of note, new onset of diabetes, or worsening diabetes, has been associated with development of pancreatic cancer thought to be due to tumor secreted products.

James McCluskey
Dr. Katarina Wrzos
Dr. Mouaffa Tello
Dr. Oana Dickinson

A Pernicious Case of Anemia

Introduction: Pernicious anemia (PA) is rare in the general population (<0.1%), but it is the most common cause of vitamin B12 deficiency. The typical finding associated with PA and vitamin B12 deficiency is megaloblastic anemia. This is often accompanied by several other characteristic findings, such as neurologic manifestations (peripheral neuropathy, cognitive impairment, weakness), psychiatric/mood changes, and gastrointestinal symptoms. The case at hand is an uncommon presentation of B12 deficiency with hematologic, neurologic, and cardiovascular consequences that led to the diagnosis of PA.

Case Description: Our case begins with a 54-year-old black woman with a past medical history notable for coronary artery disease, paranoid schizophrenia, depression, and hypothyroidism. She had been seen in various clinical settings over the previous two weeks for vague complaints of abdominal pain, worsening weakness, fatigue, and presyncope. Over this time period, her labs had demonstrated worsening pancytopenia, and she was found to be severely deficient in vitamin B12 with undetectable levels. No further workup had been initiated. During an outpatient visit with her primary care provider for her first injection of B12, she became hypotensive and presyncopal and was thus transferred to the emergency department where she was eventually admitted to the medicine ward. A peripheral smear collected at the time of admission showed macrocytic anemia, leukoerythroblastosis with myeloblasts, dacrocytes, and evidence of intramedullary hemolysis, concerning for an acute malignancy. Physical examination revealed autonomic dysfunction with orthostatic hypotension, and bradycardia. A transthoracic echocardiogram was unremarkable. Due to worsening pancytopenia and the presence of blasts on the peripheral smear, a bone marrow biopsy was performed. The biopsy showed severe megaloblastic anemia, but no increase in blasts. While labs to evaluate for PA were pending, and esophagogastroduodenoscopy was completed. This
showed antralised stomach mucosa with intestinal metaplasia consistent with chronic atrophic gastritis. On hospital day three, testing for anti-intrinsic factor antibodies returned positive, confirming the diagnosis of PA. Anti-parietal cell antibodies also returned positive later. Additional studies revealed low folate and methylmalonic acid, with elevated homocysteine. With the guidance of hematology, treatment with intramuscular vitamin B12 injections were initiated, and gradual improvement in all cell lines and mean corpuscular volume was observed prior to discharge. Besides improvements in laboratory studies, the improvement in our patient’s symptoms were dramatic; she was no longer orthostatic, and felt the strongest and most energetic she had in quite some time.

Discussion: Vitamin B12 deficiency is a known cause of megaloblastic anemia and pancytopenia; it is however a less well-known cause of leukoerythroblastosis with myeloblasts and dancrocytes. These findings are rarely reported in the literature, and this case can serve as a reminder of an uncommon presentation of vitamin B12 deficiency and PA.

Michael Michalik

**GATA2 Deficiency and The Lupus Masquerade**

Introduction: GATA2 deficiency is a hereditary, autosomal dominant bone marrow failure disorder characterized by hematologic and immunologic problems throughout life with a high risk of evolution to acute myelogenous leukemia (AML) or chronic myelomonocytic leukemia (CMML). This heterogeneous disorder produces a broad spectrum of disease including cytopenias, severe viral and bacterial infections, neoplastic disorders, and even venous thrombosis associated with positive Lupus anticoagulant. Making a correct diagnosis is critical, as immunosuppression can lead to worsening infections and accelerate the risk of developing neoplastic disorders.

Case: A 35-year-old woman with a longstanding history of an ill-defined immunodeficiency characterized by chronic leukopenia, anemia, and thrombocytopenia presented to clinic for further evaluation of recurrent sinopulmonary infections. Her history of recurrent severe bacterial infections began at age 2 and had resulted in multiple hospitalizations and prolonged courses of antibiotics. In addition, she was noted to have chronic warts since age 12, often hundreds at a time, unresponsive to topical medications and surgical removal. 7 years ago, she was diagnosed with Systemic Lupus Erythematosus (SLE) after developing a DVT and PE and being noted to have a positive ANA, antiphospholipid antibody and Lupus-like anticoagulant. She was started on Prednisone and Hydroxychloroquine. In May of 2017, she was re-evaluated by a new rheumatologist who questioned the SLE diagnosis as all of the Lupus markers were not positive.

She was subsequently referred to immunology where a Next-generation sequencing (NGS) panel was obtained and revealed a GATA2 variant (chr3:128202711, pR337) with an allele burden of 46.82%, suggesting GATA2 haploinsufficiency. She is currently in the process of tapering off Prednisone and Hydroxychloroquine. In addition, she is being evaluated for possible hematopoietic stem cell transplant (HSCT) due to the presence of bands on differential and night sweats, raising the suspicion that she may be developing myelodysplastic syndrome (MDS) or AML.

Discussion: GATA2 deficiency is a heterozygous germ line mutation which leads to aberrant hematopoiesis resulting in a constellation of diseases and a predisposition to developing myeloid neoplasia. Thrombotic events and transiently positive Lupus anticoagulant can be seen in up to 25% of patients often leading to an incorrect diagnosis of SLE. Diagnosis of GATA2 deficiency in such patients is imperative to reduce the use of immunosuppression, which
can lead to both worsening infections and increased neoplastic transformation. Proper diagnosis is also important to direct clinicians towards HSCT, which is the only curative therapy for GATA2 deficiency.

<table>
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<tr>
<th>Kevin Miller</th>
<th>I Will Live in Thy Heart</th>
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<td>Dr. Ashish V. Chintakunlawar</td>
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Case report: A 74-year-old woman presented to the emergency department with a four-day history of marked fatigue and heart palpitations.

Eight years prior, she was diagnosed with Grade 2 papillary thyroid carcinoma, which was surgically excised; 2/10 neck lymph nodes were positive, and there was extrathyroidal extension. She was treated with adjuvant radioactive iodine therapy totaling 162 millicuries (mCi). Her post-therapy scan was negative. Five years prior to the acute presentation, she developed a neck recurrence. Repeat surgical resection was performed; 5/25 lymph nodes were positive. She received adjuvant radioactive iodine totaling 165 mCi. Again, her post-therapy scan was negative. She was disease-free until two years prior to the acute presentation, when her thyroglobulin increased and a CT scan revealed pulmonary nodules. These were 18FDG-PET-positive, but observed as they were slowly progressive. Eighteen months prior to the acute presentation, she underwent further bronchoscopic evaluation for dyspnea, which ended up being inconclusive. However, the procedure was complicated by atrial fibrillation with rapid ventricular response; she was discharged home on metoprolol, diltiazem and apixaban. She continued to be observed without treatment until the acute presentation.

After arriving to the emergency room, an EKG was performed which showed significant ST elevation in the anterolateral precordial leads. However, coronary angiography did not show any arterial obstruction. Echocardiogram revealed a left ventricular ejection fraction of 50%, but with an enlarged, akinetic septal wall measuring 27 mm in width. There were also questionable bi-ventricular apical thrombi. She was started on heparin and observed.

During the next two weeks, her energy returned to normal. However, further work-up revealed a single 6 x 4 cm peripherally enhancing, centrally necrotic mass in the cardiac septum extending from the apex to the mid-ventricle encasing the distal left anterior descending artery. She underwent cardiac biopsy. This revealed atypical epithelioid cells in insufficient numbers for complete immunohistochemical characterization. In addition, there were significant lymphocytic infiltrates and necrosis which met the Dallas criteria for acute myocarditis. After the procedure, she developed symptomatic tamponade necessitating urgent pericardiocentesis. Although the biopsy was inconclusive, she went on to receive 20 Grays of palliative radiotherapy to the mass in 5 fractions. She developed some symptomatic heart failure afterwards, but remains alive three months later.

Discussion: Papillary carcinoma is the most common type of thyroid cancer. Distant metastases are rare and confer markedly worse survival, but tend to occur in the lungs or skeleton. The heart is an incredibly uncommon site for metastatic spread and extremely complex to manage, especially given the indolent nature of the disease. Herein, the results from the cardiac biopsy remain mysterious, with the concurrent lymphocytic infiltrate either representing a second independent process, or perhaps a vigorous inflammatory reaction to the tumor itself.
### Sepsis and a Fuzzy Prosthetic Valve: A Striking Case that is NOT Infective Endocarditis

**Introduction:** We present a case that illustrates a complication of transcatheter aortic valve replacement (TAVR) that can mimic other potentially life-threatening conditions.

**Case Presentation:** This is a case of an 83-year-old male with a past medical history of aortic stenosis status-post TAVR two years prior, post-TAVR perivalvular leak plug, recurrent thromboembolism, and Waldenstrom gammaglobulinemia on immunosuppression with Rituximab. He presented with one day of fever, rigors, left leg swelling, and redness. In the emergency department, he was tachycardic, febrile, and hypotensive. Physical exam was significant for left leg erythema, tenderness to palpation, and 1+ pitting edema below the knee. The initial differential diagnosis included infection (skin or another source) and deep vein thrombosis/pulmonary embolism. Blood cultures were drawn and cefepime initiated. Leg U/S was negative for DVT. During admission blood cultures grew methicillin resistant coagulase-negative Staphylococcus. Given his history of TAVR, infective endocarditis (IE) was a concern and vancomycin was added to his antibiotic regimen. TEE was performed and revealed a mobile echodensity on the aortic side of the prosthetic valve measuring 5x2mm, possibly attached to the perivalvular leak plug—suggestive of either vegetation or thrombus. As teams were preparing to prolong antibiotic treatment for 6 weeks and possible surgical intervention in the setting of prosthetic valve IE, his symptoms resolved and blood cultures cleared within 48 hours. When his TEE was compared with an immediate post-TAVR TEE, it became evident that this echodensity was previously present, consistent with native valve leaflet overhang post-TAVR. Antibiotic treatment was de-escalated, and continued for fourteen days for cellulitis and community acquired bacteremia. The patient fully recovered and is now well.

**Discussion:** Native leaflet overhanging (NLO) is a common finding after TAVR due to the mechanism of deployment of the transcatheter heart valve, especially in first generation short stents (THV) (5–7). NLO is thought to be relatively benign if there is no interaction between the THV cusps and the native valve (5). However, it might be a risk factor for complications after TAVR such as THV thrombosis secondary to blood stagnation and decreased flow (3,8,9). NLO likely results from low implantation in the ventricle. Consequently, overhanging leaflets may exert downward pressure during diastole leading to central aortic regurgitation (10), early degeneration, migration, and embolization towards the ventricle (3,5,7,11,12).

We recommend, first, to correctly measure the aortic valve annulus to avoid undersizing or oversizing the THV. Second, when suspecting TAVR-related complications we suggest comparing new echo findings with previous studies, as missing diagnosis can lead to unnecessary prolonged antibiotic regimens and invasive interventions. Finally, in patients with previous TAVR and TEE findings consistent with IE, NLO should be considered as a differential when the patient does not follow the expected clinical course.

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### A Rare Case of Pituitary Adenoma: The mysterious Crooke’s Cell Adenoma

**Introduction:** Cushing’s disease is defined by excess cortisol and can be caused by endocrinologically functional, adrenocorticotropic hormone (ACTH)-producing pituitary corticotroph adenomas[1]. Excess cortisol leads to a cytoplasmic accumulation of cytokeratin filaments and hyalinization which characterizes Crooke cell tumors, a rare variant of corticotroph adenoma[2]. Crooke’s cell tumors are very rare (largest case series of 36) reported by George et al., aggressive, and prone to recurrence after resection which makes identification and distinction from other adenomas crucial[2].
Case: A 42-year-old Caucasian male, with a history of obesity, HTN, DM2, and refractory hypokalemia presented to the hospital as a direct admit with symptoms of headache, eye pressure, pedal edema, and weight gain for 4 weeks. Despite interventions, his symptoms didn’t improve and suspicion for Cushing’s syndrome increased.

The patient had a cushingoid appearance with positive parinaud sign (limited upward eye deviation) both suggestive of possible pituitary mass. CT head revealed a 1.2cm right pituitary macroadenoma with remodeling of sella turcica. Random cortisol 39.6 and ACTH 140. Low and high dose dexamethasone suppression test failed to suppress cortisol levels which suggested ectopic hypercortisolism. Inferior petrosal sinus sampling confirmed right sided pituitary Cushings as the source of hypercortisolemia. The patient underwent endoscopic endonasal transsphenoidal (EET) resection of pituitary macroadenoma with limited visualization secondary to cavernous sinus bleeding. Initially postoperative cortisol levels decreased but then started to rise increasing suspicion for residual sellar tumor. The patient underwent repeat EET resection after which cortisol levels dropped and reached a nadir of 3.0 with ACTH 13. Pathology results revealed Crooke cell variant adenoma.

Discussion: Crooke’s cell tumors are a rare variant of pituitary adenoma and are important to distinguish from other adenomas due to their unique cellular behavior. The cause of hyalinization within these tumors is unknown. It is also unclear as to why this hyalinization results in increased ACTH production as hyalinization typically results in loss of function[3]. These tumors are also very aggressive and prone to recurrence even after resection. Some patients with cushing’s syndrome and macroadenoma may fail high dose dexamethasone suppression testing and behave as ectopic cushing’s syndrome[4]. If patients with evidence of pituitary tumor fail dexamethasone suppression test it is important for clinicians to maintain suspicion of aggressive tumor variants such as Crooke’s tumor which require close follow up and management including possible radiation therapy[5]. In conclusion, this case highlights a cushing’s syndrome caused by a rare variant of pituitary adenoma, a Crooke’s variant tumor.

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Jessica Padniewski  
Dr. Mohammed Horani

Hypokalemia-Induced Rhabdomyolysis: A Rare Manifestation of this Electrolyte Disturbance

Introduction: Rhabdomyolysis is a condition defined by muscle necrosis with subsequent release of muscle cell contents into circulation[1]. These cellular components are measured and used to monitor resolution. Rhabdomyolysis is commonly caused by drugs, toxins, crush injury, overexertion, infection, extreme temperature change, or hypoxia [2]. Severe long-term hypokalemia has been recognized as a rare cause of rhabdomyolysis due to reduced potassium release from contracting skeletal muscle cells. Low potassium with lack of release during contraction leads to diminished blood flow, resulting in ischemia with muscle injury as well as reduced skeletal muscle integrity[3]. Muscle weakness is classically associated with severe hypokalemia, but few cases of rhabdomyolysis with elevated CK have been attributed to low potassium.

Case: A 58-year-old male with a history of BPH, medicated hypothyroidism, and neuropathy who was unable to move for 10 hours after a fall occurring because his “legs gave out.” This fall occurred in the setting of worsening generalized weakness for 2.5 weeks and was brought to the Emergency Department with dizziness and shortness of breath. He was found to have severe hypokalemia(1.8), CK (9000), and leukocytosis (20,000). Serial BUN/Cr values ranged from 8-12/0.9-1.2 respectively. Patient had insignificant findings on CXR, ECG, and CT abd/pelvis, and normal vitals. Nephrology found no evidence of renal potassium losses with a urinary potassium level of 13.1mmol/L (<25 mmol/L) and suspected decreased intake of potassium.
Subsequent labs revealed potassium (2.2) with CK was now at (10,900). Admission day 3, he developed postobstructive diuresis with 260cc output over two hours. Following foley placement, K increased (3.0) and CK continued to rise (11,050). Over the subsequent days, K continued to rise (5.3) and CK(7,100). Over the next 4 days CK and creatinine were down trending, and he was discharged with resolution of his presenting diagnoses. Close outpatient follow up was recommended for electrolyte and CK monitoring.

Discussion: This case demonstrates that an elevation of CK can occur due to extended duration of hypokalemia. This gentleman continued to have worsening of serum CK despite resolution of his infection, repositioning, and appropriately controlled hypothyroid. Although the loss of free water could elevate serum CK levels, this patient’s CK levels increased before patient had a severe increase in urinary output. Only with normalization of K did his CK improve. Also, drastic elevation of his serum CK to 11,000 was not likely to be found after being down for only 10 hours, and he possessed no other identifiable risk factors or kidney dysfunction to account for this level of CK elevation.

Arshia Sandozi
Dr. Mark Leo

Primary Neuroendocrine Tumor of the Testis

Introduction: Neuroendocrine, also known as carcinoid, tumors, are rare neoplasms that present most commonly within the gastrointestinal tract. They are characterized by the ability to produce peptides that may lead to development of endocrine syndromes. Most neuroendocrine tumors are well-differentiated and slow growing, however, the less common moderate and poorly-differentiated tumor can be aggressive and contribute to earlier mortality. We present a rare presentation of primary neuroendocrine tumor of the testis.

Clinical presentation: A 49 year old male presented for evaluation of painless left testicular mass. He first noticed the mass 15 months ago. He denied any trauma, pain, discomfort, flushing, abdominal pain, nausea, diarrhea, dysuria or hematuria. Urinalysis revealed no sign of infection and no hernia was evident. Left testicle was noted to be twice as large as the right, and firm and smooth.

Scrotal ultrasound at that time had revealed a heterogeneous left testicular mass suspicious for malignancy. Alpha fetoprotein and beta human chorionic gonadotropin were within normal limits. He was referred for surgical evaluation but did not return for follow up for 15 months, at which time he underwent left radical orchiectomy. Abdominal computed tomography showed no evidence of other primary or metastatic disease.

On gross examination, the tumor had almost entirely replaced the left testis. No hemorrhage or necrosis was evident. Microscopic sections of the left testis revealed involvement of tumor, demonstrating nests and cords of cells with uniform appearance. The cells had round nuclei with granular chromatin. Rare mitotic figures are seen and immunohistochemically stained images were positive for chromogranin, synaptophysin, and CDX2, consistent with the diagnosis of neuroendocrine tumor. No significant cytological atypia or necrosis was identified. Tunica, epididymis, and spermatic cord remained negative for tumor involvement, and there was no evidence of lymphovascular invasion. Patient was referred for DOTATATE scan to assess for nontesticular primary source of tumor, but elected not to proceed with testing.

Discussion: There have been just over 100 documented cases of primary neuroendocrine tumors of the testis between 1930 and 2015. Ten percent of these cases presented with symptoms of carcinoid syndrome due to peptide secretion. This case highlights the importance of evaluating for presence of primary tumors in less common sites. Unexplained symptoms of carcinoid syndrome without evidence of gastrointestinal site warrants examination and evaluation for nongastrointestinal sites of primary neuroendocrine tumor.
Casey Smith  
Dr. David Ewart

**Pleural Plaques Underneath the Red Shawl**

Case: A 70 year old man was evaluated by his PCP for a rash 6 weeks post-total knee arthroplasty. The rash was progressive and covering his head, arm, upper chest and back. On examination he had decreased breath sounds and dullness to percussion at his right base. Laboratory findings reveal elevated CRP and ESR. Chest x-ray revealed a large right pleural effusion with right perihilar soft tissue fullness. Chest CT revealed a large, partially loculated, right pleural effusion with bilateral calcified plaques, and irregular thickening of right anterior pleura. The rash was in a shawl and heliotrope distribution. Gottron's papules were present. Strength was 5/5 throughout all proximal muscle groups. The patient was admitted and a thoracentesis performed. Pleural fluid was exudative and cytology was negative for malignant cells. Because of a self-reported history of asbestos exposure and the CT findings of pleural plaques, suspicion for mesothelioma was high. A CT-guided pleural biopsy revealed malignant mesothelioma. The day of discharge the patient reported myalgia. CK was rechecked and found to be elevated (770). MRI of the right deltoid showed intramuscular edema. The patient was started on prednisone 80 mg daily and underwent a surgical muscle biopsy, the results of which are pending. TIF1-gamma antibody, which is associated with dermatomyositis, was positive on a myositis antibody panel.

Discussion: Dermatomyositis, in particular amyopathic dermatomyositis, is associated with malignancy but has never been described in the context of mesothelioma. While initially amyopathic, this patient ultimately developed muscular involvement and diagnosis was made by identifying weakness with evidence of muscle breakdown, identification of the involved muscles by MRI, and ultimately pathologic diagnosis by surgical muscle biopsy. The treatment in most cases is immunosuppression with corticosteroids being the primary component. Further immunosuppression is often required and in cases associated with malignancy, treatment of the underlying cancer. The case demonstrates that although most cases of dermatomyositis present with myopathy it is important to recognize there are a subset of patients that present amyopathic. Amyopathic dermatomyositis has been found to be associated with interstitial lung disease and malignancy. Inflammatory myopathies have a variety of associated antibodies and can be identified using the standardized antibody panels. Anti-TIF1-gamma antibodies have been linked to cancer-associated-myositis. The presence of TIF1-gamma antibodies in this patient suggests the dermatomyositis and malignant mesothelioma are linked and further illustrates that patients with myositis should have a thorough, detailed workup for malignancy.

Leah Soderberg

**Pointing Toward a New Diagnosis: A Case of ANCA Vasculitis in the Setting of HfPEF**

Introduction: Dyspnea and shortness of breath in heart failure patients is most often initially attributed to heart failure exacerbation, particularly in the presence of pulmonary edema and improvement following diuresis. Despite this, the full differential diagnosis for dyspnea remains expansive and in the presence of findings that do not obviously match a simpler and more straightforward clinical picture, high suspicion for rarer etiologies can lead to improved clinical decision making and identification of underlying disease processes that may result in severe debilitation and even fatality if not promptly treated.

Case: A 62-year-old man with heart failure with preserved ejection fraction, Type 2 diabetes mellitus, hypertension, and obesity was hospitalized for dyspnea and hypoxia attributed to an exacerbation of his heart failure and fluid overload. His symptoms resolved with inpatient diuresis, but despite diligent weight monitoring, fluid restriction, and compliance with medications at home,
He continued to have respiratory failure and hypoxia requiring repeated hospital admissions for aggressive diuresis.

Simultaneously during this period, he also developed spontaneous, non-healing, dusky violaceous ulcerations with necrotic centers on multiple fingertips and complained of new arthralgias and swelling of his finger and knee joints bilaterally. Labs ordered by the man’s primary care physician for evaluation of this growing list of symptoms were revealing: a C-reactive protein of 15.7 mg/L, sedimentation rate of 20 mm/hr, and positive anti-nuclear antibody (1:320 speckled pattern)—findings supportive of an underlying autoimmune process further complicating his course. Additional work-up was positive for p-ANCA antibodies and myeloperoxidase antibodies consistent with ANCA vasculitis, and follow-up chest CT showed subpleural reticular markings, ground glass opacities, bronchiectasis, and early honeycombing bilaterally in the lung bases, all consistent with interstitial lung disease, most likely due to ANCA vasculitis lung involvement. Spirometry was remarkable for restriction and reduced DLCO, also consistent with rheumatoid lung and persistent dyspnea and hypoxia refractory to measures aimed solely at stringent management of the man’s heart failure and cardiac risk factors. He was subsequently treated with rituximab and prednisone in addition to routine management of his comorbid chronic conditions, and eventually his respiratory symptoms stabilized.

Discussion: Although ANCA vasculitis is uncommon and rarely appears on an initial differential diagnosis for dyspnea—particularly when other more obvious etiologies are present—the best outcomes for the disease result from early identification and treatment, particularly with regards to its pulmonary and renal complications. This case serves as a strong reminder of the value in closely considering symptoms that do not fit a patient’s clinical picture, as they may offer hints at an underlying etiology that, if promptly diagnosed and treated, can greatly improve a patient’s outcome and offer the best chance for a more stable medical course.

McKenzie Tolan

Contaminate or Culprit: A Case of Cutaneous Malakoplakia

Introduction: Malakoplakia is a rare granulomatous disease caused by the overgrowth of gram negative organisms secondary to impaired macrophage/histiocyte function. It generally infects the genitourinary tract and can present as single or multiple tumors. There are few case reports of cutaneous involvement in the head and neck.

Case description: A 92 year-old man presented with a one-month history of a fleshy, exophytic right-sided neck lesion and no systemic symptoms. The lesion was tender, bled easily, with frequent serous drainage, and had increased in size within this timeframe. With concern for possible malignancy the patient underwent a CT scan demonstrating a 4.2 x 2.1 x 2.0 cm mass of the right neck involving the skin and invading underlying muscle along with 2 enlarged lymph nodes in the contralateral neck. A probable diagnosis of malignancy was made. The patient was seen by dermatology and ENT, where a shave biopsy, punch biopsy, and two FNA samples were obtained. Shave biopsy demonstrated histiocytic reaction with coccobacillary organisms, and no evidence of malignancy in the sample. Punch biopsy and FNA sample findings were consistent with shave biopsy results. With no clear evidence of malignancy, an infectious etiology was pursued and the patient was started on a 14-day empiric trial of ciprofloxacin in attempt to diminish any possible infectious influence. Repeated cultures were drawn, consistently demonstrating 3+ E.coli along with PMNs and RBCs. The E.coli was initially considered a contaminant. The lack of presumed diagnostic findings led to an excisional biopsy of the deep tissue. The resultant pathology was consistent with cutaneous malakoplakia. Treatment plan included a 3 month course of ciprofloxacin.
Discussion: Malakoplakia is a rare disease primarily found in the genitourinary tract. It is considered the result of an acquired defect in macrophage/histiocyte function that subsequently enables bacterial overgrowth. The species most commonly implicated is E.coli. Patients with malakoplakia are often older and may be immunosuppressed. Cutaneous malakoplakia, particularly in the head and neck, clinically mimics malignancy and this case highlights the importance of maintaining a broad differential when evaluating neck masses in the elderly population. Additionally, it is important to think twice about what originally might be considered contaminate.

Grant Wintheiser
Dr. Gregory Pajot
Dr. Wil Santivasi

Disseminated Histoplasmosis Masquerading as a Hematologic Malignancy in an Immunocompetent Adult

Introduction: Histoplasmosis is an opportunistic fungus found ubiquitously in the Ohio and Mississippi River Valleys in the United States. In immunocompetent patients, infections generally remain asymptomatic and confined to the lungs, while in immunocompromised patients, dissemination can occur and involve the reticuloendothelial system, pharynx, GI tract, skin, adrenal glands, and CNS. However, disseminated disease has been documented in patients with no known cause for immunocompromise. Diagnosis in these cases can be challenging given the non-specific signs and symptoms.

Case Presentation: A 74-year-old woman from Iowa presented to an outside emergency department with severe fatigue, left-upper quadrant abdominal pain, and dizziness. She was found to be hypotensive with initial labs notable for acute kidney injury, elevated uric acid level (17), thrombocytopenia (30,000) and neutropenia (460) with monocytosis (1,310). CT abdomen showed adenopathy and marked splenomegaly. A bone marrow biopsy was obtained with findings suspicious for a clonal NK-cell lymphoproliferative disorder. She was sent to Mayo Clinic for further workup. Prior to transfer she developed worsening pharyngitis prompting a CT scan of the neck, which showed extensive cervical lymphadenopathy as well.

Given her bone marrow findings and nasopharyngeal symptoms, serum EBV DNA testing was performed to evaluate for possible extranodal natural killer/T cell lymphoma. EBV DNA was positive. However, further examination of the biopsy slides by our pathologists favored the NK cells to be reactive. Instead, there was concern for CML given a distinct population of atypical monocytes, although repeat bone marrow suggested against this. PET-CT imaging findings were consistent with probable lymphoma involving the bone marrow, spleen, tonsils, and lymph nodes diffusely. For definitive diagnosis, she underwent excisional biopsy of a left inguinal node. Pathology showed necrotizing granulomatous lymphadenitis and fungal organisms most consistent with histoplasmosis, as well as EBV-positive lymphoproliferation. Subsequent serum histoplasmosis antibody testing was positive, although urine antigen testing was negative. She was referred to infectious diseases and is currently on itraconazole for treatment of disseminated histoplasmosis.

Discussion: This case illustrates the uncommon finding of disseminated histoplasmosis in an individual with no known cause for immunocompromise. Although rare, chronic progressive disseminated histoplasmosis has been noted to occur in apparently immunocompetent older adults. The suspected cause is an unidentified deficiency in cellular immunity, which is plausible in this patient given her concurrent EBV reactivation. Hepatosplenomegaly, lymphadenopathy, oropharyngeal and GI lesions, pancytopenia and transaminitis are common findings in disseminated histoplasmosis, but are non-specific and found in many other diseases, including hematologic malignancies. Thus, it is important to maintain a broad differential and have a high index of suspicion for fungal infection in patients from endemic areas.
Residents

Quality Improvement- Residents

Bjorn Bakken
Dr. Sarah Prebil

*Drop Me a Line: Improvement in Central Line Monitoring in a Hospital Setting*

Introduction: Central Line Associated Blood Stream Infections (CLABSI) represent a major complication of hospitalizations and are associated with significant rates of mortality. There are an estimated 250,000 catheter associated bloodstream infections that occur in the United States annually. In the year 2017 Abbott Northwestern Hospital experienced 20 such infections.

Case Description: A 47 year old male was admitted to Abbott Northwestern Hospital on December 2017 with shortness of breath and 20 pound weight loss. Labs demonstrated severe pancytopenia and a bone marrow biopsy later confirmed acute myeloid leukemia. A port was placed and he was started on chemotherapy the following day. He was hospitalized for the duration of his chemotherapy. He became febrile and tachycardic two weeks later. Blood cultures ultimately grew Bacillus cereus, a common organism associated with port infections in immunocompromised hosts. He was treated with vancomycin, and his blood cultures ultimately remained negative for the following 2 weeks. He was discharged with plans to pursue stem cell transplant.

Discussion: At Abbott Northwestern Hospital, there were 20 CLABSI cases in 2017 compared with 27 in 2016, which represented a 26% decrease in overall cases. Comparatively, the rates decreased from 1.05 cases per 100,000 line days in 2016 and 0.84 cases per 100,000 line days in 2017, a decrease of 19%. Arguably the largest contributing factor was the number of overall line days per year. In 2016, there were 20,862 line days compared with 20,179 in 2017. This highlights the importance of removing central lines as soon as they are no longer indicated as this intervention remains the most effective way to mitigate CLABSI rates. There are several other important interventions that an internal medicine physician can take to help decrease overall CLABSI rates, including visualizing the dressing during the daily physical exam, communicating with the nursing team if the dressing is not intact and reinforcing patient education about proper care and maintenance of their central line.

Laura McCarthy
Dr. Nasreen Quadri
Dr. Jaya Dharvasala

*Assessing and Addressing Mental Health Burden in Newly Arrived Refugees to the US: A Multi-stage Quality Improvement Study*

Background: Refugees are a unique population at risk for mental health disease. Histories of trauma, detention, as well as loss of culture and social support systems may contribute. Barriers exist to identifying their mental health needs and aligning them with treatment. Inadequate identification may occur due to lack of self-referral, poor application of standard screening questions, language differences, and provider time constraints. At a primary clinic in Saint Paul Minnesota, there was concern that patients were being inadequately screened for mental health burden. A quality improvement study investigating this clinical question was initiated.

Objective: To identify appropriate methods of screening for mental health disorders in newly arrived refugees to the United States within one urban clinic's health system in Saint Paul, Minnesota serving a primarily refugee population.

Methods: Following a clinic-wide distribution of a two-question mental health screening tool, we performed a retrospective chart review from August 1, 2014 to July 31, 2015 of adult primary refugee patients presenting for new arrival screening to assess provider screening uptake. Based on the results, the 2
question tool was expanded to 5 questions. Recommendations by an expert workgroup including members from the Minnesota Department of Health, Center for Victims of Torture, and University of Minnesota School of Social Work were incorporated. Focused mental health screenings trainings with providers were completed. The second phase of the study involved a subsequent chart review from January 1, 2016 to December 31, 2017 analyzing the results of the new five-question screening tool to assess changes in the uptake of screening, burden of illness identified, and number of mental health referrals.

Results: Initially using the 2 question screening tool, only 25.6% (86/336) of all adult refugees were screened with mental health questions at their arrival visit. Of those, 18.6% (16/86) screened positive. Only half (8/16) were referred for mental health services. All who were referred (8/8) completed a mental health visit. Using the five-question screening tool, 63% (382/605) were screened, 10% (37/382) screened positive, and 68% (25/37) were referred.

Conclusion: Initial results from 2014-2015 showed significant barriers to achieving universal mental health screening and referral despite recommendations to do so. With the expanded five-question screening tool and focused provider training, more patients were screened and referred from 2016-2017, but fewer individuals screened positive. The differences may have occurred due to increased staff training and awareness as well as integration of the questions into the electronic medical record.

**Research- Residents**

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<th>Jonathan Bjork</th>
<th>Characteristics of Gout and HLA-B*58:01 Genotype Frequency in a Minnesota Hmong Population</th>
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<td>Morgan Brown</td>
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<td>Dr. Kathleen Culhane-Pera</td>
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**Characteristics of Gout and HLA-B*58:01 Genotype Frequency in a Minnesota Hmong Population**

Background: The Hmong community has a severe form of gout with earlier symptoms, more tophi, and higher uric acid levels. Allopurinol is the most common anti-hyperuricemic treatment but may cause severe cutaneous adverse reactions (SCARs) with mortality rates approaching 25%.

The HLA-B*58:01 allele is strongly associated with allopurinol-associated SCARs. HLA-B*58:01 frequency varies widely across populations, from 20% in Taiwanese to 2% in Caucasians. In high frequency populations screening has been shown to be cost effective. Current guidelines suggest screening for those of Korean descent with CKD stage 3 or worse, Han Chinese, and Thai. With roots in southern China, we investigated basic clinical characteristics of gout and HLA-B*58:01 frequency in Hmong patients.

Methods: Data was collected from two sources: Hmong patients with gout presenting to Regions Hospital starting 1/1/2016 – 7/1/2018 and Hmong volunteers with and without gout (recruited at a local health fair). HLA-B*58:01 genotype frequency was calculated for a pooled allopurinol-naïve Hmong cohort (total n=72) and compared to prevalence data for other populations (Han Chinese Korean, Caucasian, African, and Indian) using a two-proportions test.

Results: Hmong patients with gout (n=30) tended to be male with polyarticular, tophaceous gout (approaching 50%), higher rates of obesity, hypertension (HTN), hyperlipidemia (HLD), diabetes (DM), CKD, and elevated uric acid levels. HLA-B*58:01 frequency among the pooled cohort of allopurinol-naïve Hmong was 2 of 72 (2.8%).

Conclusion: Hmong with gout have more severe disease, high rates of obesity, HTN, HLD, DM, CKD, and elevated uric acid levels. Allopurinol-naïve Hmong have a statistically significantly lower prevalence of HLA-B*58:01 than Han Chinese and Koreans. There is no significant difference between Caucasian, African, and Indian populations. Although with roots in southern China, these
Data suggest that screening may not be necessary in Hmong with gout before starting allopurinol.

| Elliot Graziano  
| Dr. Byron Vaughn  
| Dr. James Campbell  
| Dr. Andrew Reinink | Successful Implementation of a Therapeutic Drug Monitoring Program and its Impact on Patient Management and Outcomes |

**Introduction:** Therapeutic drug monitoring (TDM) of infliximab (IFX) is an important clinical tool in the management of inflammatory bowel disease (IBD), both in the reactive and proactive setting. We conducted a practice improvement project utilizing a commercially available drug concentration assay through a national program that provides two free tests per year for patients on IFX.

**Methods:** Patients with IBD treated with IFX were enrolled in TDM starting in September 2016 when the program became available. The infusion center, consisting of patients and staff, completed and signed necessary paperwork to enroll in the program. Samples were drawn at the time of infusion, paperwork was attached, and samples were sent to a commercial lab. Electronic medical records were retrospectively assessed to determine if use of the assay impacted clinical decision making and outcomes.

**Results:** Fifty-eight patients were enrolled in TDM (55.2% Crohn’s, 43.1% Ulcerative Colitis and 1.7% indeterminate colitis) with 81 unique IFX levels from 10/6/16 to 9/1/17. Forty-three percent (35/81) of TDM was performed in the proactive setting (no evidence of clinical, serologic or endoscopic disease activity at the time of testing). TDM resulted in changes of IFX therapy in 49% instances (37.5% following proactive TDM, 62.5% following reactive TDM). Of the changes, 31 were therapy escalation, 5 were de-escalation and 4 were discontinuation of IFX. Three levels (3.7%) in two patients had associated antibodies (one patient stopped IFX (antibody level >200), one remained on IFX (antibody level 10)). Of the 24 reactive TDM levels resulting in dose escalation, 19 (79.2%) improved in at least one clinically relevant area with symptoms being the most common area of improvement (15 of 19 levels). Of the 35 proactive IFX levels, 15 resulted in a dose change, 11 escalations and 4 de-escalations.

**Discussion:** Successful implementation of a TDM program into practice is possible through identification of key stakeholders from the local clinic, clinical laboratory, and infusion center, along with the development of a standardized workflow for send out labs. Barriers to implementation included multiple clinic sites and outside infusion centers. Using a readily available program, it is possible to develop and implement an institutional system for TDM that can result in clinically meaningful therapy changes, improved outcomes and increased flexibility in optimizing therapy.
<table>
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<th>Christine Gruessner</th>
<th>Edema on Bone Marrow Biopsies: An Early Warning Sign of Ankylosing Spondylitis?</th>
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<td>Ankylosing spondylitis (AS) is an inflammatory autoimmune disorder that primarily affects the spine. If untreated it causes significant morbidity. AS often presents with symptoms that are both articular and extraspinal, including enthesitis, synovitis, and dactylitis. It may also be associated with nonarticular disease, such as uveitis, psoriasis, and IBD. Patients frequently carry the gene for human leukocyte antigen HLA-B27; 90-95% of patients with AS are positive for HLA-B27. Prevalence estimates of AS vary considerably, and has been shown to be higher than the national average in Minnesota. Aggressive intervention in the early stages of AS has been shown to have better outcomes in terms of symptom management, long term pain relief, prevention of bone loss, and suppression of inflammatory effects on bone. AS is often accompanied with the finding of bone marrow edema; in a recent study, bone marrow edema was present in 70% of AS patients. Radiographic structural changes are seen on average 7-10 years prior to the clinical diagnosis of AS. The following is a study of 10 patients who have undergone bone marrow biopsy for abnormal cell counts, who several years later were diagnosed with AS. In each of these patients, bone marrow edema (ranging from mild to marked) was seen on their biopsies. The finding of edema is not customarily reported in bone marrow biopsy pathology reports; however, reporting the finding of edema on a biopsy may have important clinical implications, and may prompt screening for early signs and symptoms of AS.</td>
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<th>Kristopher Holaday</th>
<th>Minnesota Detoxification Scale (MINDS) Assessment Protocol for Treatment of Alcohol Withdrawal, Pre and Post Comparison</th>
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<td>Dr. Steve Hanovich</td>
<td>Background: Acute alcohol withdrawal is commonly encountered in general hospital settings and can result in serious consequences. Symptoms and complications of withdrawal represent medical emergencies that carry significant clinical risk and requires attentive medical management. Although there is no national guideline for the assessment and treatment of acute alcohol withdrawal, the current standard of care favors symptom triggered therapy with use of assessment scoring system (CIWA is the most common) and the drug of choice is the benzodiazepine group. In the spring of 2014, a pilot was implemented at Unity Hospital (part of Allina Health, MN) with the intent of systematizing treatment of alcohol withdrawal at Allina Health using the Minnesota Detoxification Scale (MINDS) assessment for diagnosis with diazepam as a drug of choice to treat acute alcohol withdrawal. By direct comparison MINDS assessment includes fewer screening domains than CIWA and less subjective variation. Treatment for a positive screening utilizes a longer-acting benzodiazepine with a set dosing schedule administered earlier upon recognition of withdrawal symptoms. By the end of 2016, all Allina hospitals had implemented the use of MINDS protocol for patients presenting with alcohol withdrawal symptoms. This study aims to address whether implementation of the MINDS protocol order set for diagnosis and treatment of acute alcohol withdrawal results in a meaningful, measurable improvement in Allina hospital’s patient outcomes.</td>
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Hypothesis / Objectives: Allina Health hospital patients who were treated for alcohol withdrawal after the implementation of the MINDS protocol experienced better outcomes compared to patients who were treated for alcohol withdrawal before the implementation of the MINDS protocol. The study’s primary outcome will be hospital length of stay (LOS) and mortality. Secondary outcomes include: readmissions (ED or all-cause readmission within 30 days), calendar days receiving benzodiazepines and total dose administered, and number of ICU stays.

Methods: This study will use data from patients who have been treated for alcohol withdrawal at Allina Health hospitals both before implementation of the MINDS protocol and after implementation of the MINDS protocol, between January 2013 and December 2017. The analytic approach will involve comparing the “pre-MINDS” and “post-MINDS” groups of patients with multiple linear and logistic regression analyses. Based on existing data, an estimated 25,000 patients will contribute data to the various analyses of outcomes and complications under this study.

Results / Conclusions: Review of patient data pre-implementation, 03/01/2013 - 01/26/2014, and post implementation, 03/10/2014 - 11/05/2014, of the MINDS alcohol withdrawal protocol at Unity Hospital demonstrated a pre-MINDS LOS of 113.05 hours/4.71 days, and a post-MINDS LOS of 76.5 hours/3.2 days. The average total diazepam equivalents administered pre-MINDS was 102.4 mg, and post-MINDS 87.3 mg. Preliminary data suggests reduced hospital LOS and benzodiazepine total dose administered.

Brynn Stenehjem
Matthew Lillyblad
Dr. Catherine St. Hill
Dr. Vincent Agboto
David M. Tie

Effect of Patient’s Body Weight and Blood Volume on the Incidence of Coagulopathy and Complications in Patients with Pulmonary E

Background: Treatment with tissue plasminogen activator (tPA) prevents clinical deterioration and reduces mortality in high-risk pulmonary embolism (PE), but may be associated with post-thrombolysis coagulopathy (PTC) and hemorrhagic complications. Most regimens employ fixed-dose tPA irrespective of patient size. The associations between the incidence and magnitude of PTC and tPA dose adjusted for weight and estimated blood volume (EBV) are unclear.

Methods: A retrospective cohort of 91 patients with PE received fixed-dose (50 or 100 mg) IV tPA based on a clinical algorithm (Table 1). Pre- and post-tPA fibrinogen, PTT, INR, and platelet (PLT) values were obtained. Variations in these tests were analyzed with respect to tPA dose, actual body weight (BW), ideal body weight (IBW), and EBV using Pearson correlations, two sample independent t-tests, and linear regression analyses.

Results: Twenty-nine (31.9%) patients had massive (13 with cardiac arrest), and 62 (68.1%) had high-risk submassive PE. Sixty-two (68.1%) patients received 50 mg, and 29 (31.9%) received 100 mg IV tPA. In non-cardiac arrest patients, 93.6% had SPESI ≥ 1, and mean shock index was 0.997. On average, fibrinogen decreased by 162 mg/dl (14 to -427, 38.6% decrease), PLT count decreased by 43 thou/mm3, (37 to -254, 16.8% decrease), and INR increased by 0.38 (-0.1 to 2.8). Post-tPA fibrinogen values were < 170 mg/dl in 33.8% and < 100 mg/dl in 18.5%; INR > 1.5 in 34.4%; platelets < 140 thou/mm3 in 31.9% and < 100 thou/mm3 in 8.3%; and PTT > 35 s in 60.3%. There was significant correlation between higher tPA dose normalized to: 1) BW, IBW, and EBV and magnitude of post-tPA fibrinogen decrease (r = -0.51, -0.44, and -0.51 respectively) (Fig. 1), 2) BW and EBV and higher post-tPA PTT (r = 0.42 and 0.31 respectively). Dose normalized to BW and EBV predicted post-tPA INR > 1.5 (r2= 0.48 and 0.51 respectively). Dose normalized to EBV was consistently associated with most markers of PTC. There were 10 (11.1%) minor bleed complications, seemingly unrelated to PTC,
most following surgical procedure, vascular intervention, or tPA with simultaneous full heparin anticoagulation.

Conclusions: Post-thrombolysis coagulopathy is common, and its degree is affected by tPA dose and the patient’s weight and EBV. Larger studies of weight- and EBV-adjusted tPA regimens in PE are warranted.

**Eric Wilkerson**  
**Dr. Jonathon Kirsch**  
**Diabetes Mellitus in the Ngabe Population Seen by Floating Doctors Outreach Clinics in Bocas del Toro, Panama**

Background: Floating Doctors outreach clinics visit Ngabe communities in the Bocas del Toro region of Panama to provide primary care services. During these clinic visits Finger Stick Blood Glucoses are performed for diagnosis of Diabetes Mellitus (DM).

Methods: This was a retrospective observational study of the clinic visits performed by analyzing the electronic medical records of Floating Doctors mobile clinics for the Ngabe population from March 2015-July 2017.

Results: DM was diagnosed in 3.9 % of patients seen at Floating Doctors outreach clinics using a FSGS ≥ 126 mg/dL. An additional 2.1% of patients were found to have hyperglycemia in a nonfasting blood glucose ≥ 200 mg/dL, but symptoms for the hyperglycemic patients were not reported

Conclusion: Diagnosed or probable DM has a disease burden of 6% of the population within the Ngabe population. Floating Doctors mobile clinics are the primary care clinic available for diagnosis and management of all chronic diseases. As such, additional evaluation for DM within the mobile clinic settings with point of care hemoglobin A1c is important to appropriately diagnose and manage DM in the Ngabe population.

**Janewit Wongboonsin**  
**Dr. Paul Drawz**  
**Dr. Joseph Merighi**  
**How to Travel as a Dialysis Patient? A Qualitative Study on Travel Arrangement in Chronic In-Center Hemodialysis Patients**

Background: For people with end-stage renal disease on a renal replacement therapy (RRT), “travel” and “independence” are rated as 2 of the top 5 factors that inform their choice of a treatment modality. While home dialysis modalities offer patients a high degree of independence, the most common RRT in the USA is in-center hemodialysis (IHD). The limits imposed by IHD treatment can present a variety of challenges for patients who wish to travel. This exploratory study explored how IHD patients managed their travel and the role of dialysis social workers in executing travel arrangements for patients.

Methods: An interview-based, qualitative study was conducted with IHD patients being treated at a large Midwestern Medical Center and community-based dialysis social workers. Data collection was conducted from August 2017 to September 2018. Patients were screened from an inpatient nephrology consult panel and the patients enrolled in the study provided contact information for their dialysis social workers. Interviews with patients focused on experiences before, during and after travel, and social workers were asked to describe their role in helping patients who wish to travel. Interviews were conducted until saturation of themes was reached. Two coders used a grounded theory (constant comparative) approach to analyze the data from verbatim transcriptions. The study protocol was approved by the University’s IRB.

Results: Sixteen patients and eight social workers were enrolled in the study. The patient sample included an equal number of women and men (n=8), 13 whites (81.3%), a mean dialysis vintage of 5.3 years, and an average of 4.4 domestic trips completed. Only 1 patient reported 2 international trips. Social workers were female (100%), the majority were white (n=7; 87.5%), and they
reported an average of 2.5 travel requests/month. Preliminary findings for patients indicated that limited knowledge of dialysis facility options and uncertainties about staff at host dialysis units were key concerns in preparing for and engaging in travel. Social workers described insurance literacy and confirming "chair times" as key factors in planning out-of-state travel.

Discussion: This study identified multiple concepts and perspectives surrounding travel arrangements in chronic IHD patients. There is limited research on travel issues for chronic IHD patients and this exploratory investigation is among the first to articulate barriers and facilitators associated with travel from the perspective of patients and social workers. Guidelines on travel for IHD patients should be readily available and incorporated into ongoing patient education, especially when patients initiate RRT. Promoting and supporting travel for IHD patients can serve to increase their sense of autonomy and provide opportunities to improve their quality of life.

Clinical Vignette- Residents

Bariituu Adam

Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA): It’s Not Always about Stenosis

Introduction: MINOCA is increasingly recognized and was recently added to the 4th Universal Definition of Myocardial Infarction. 6% of myocardial infarctions occur in patients with non-obstructive coronary arteries (coronary artery stenosis less than 50%). These patients tend to more frequently be younger, have only a slight male predominance, and are less likely to have hyperlipidemia compared to patients with myocardial infarction secondary to obstructive coronary artery disease. It is a working diagnosis. Frequently, a definitive diagnosis can be obtained with the use of multiple imaging modalities including cardiac MRI (cMRI).

Case Presentation: A 49 year old woman with a pmh significant for hypertension and hypothyroidism, presented with sudden substernal chest pressure (8/10) and shoulder pain. Associated symptoms included dyspnea, nausea, and neck tightness. Earlier in the evening she experienced tongue tingling and lip swelling due to possible shrimp exposure, which resolved prior to presentation. In the ER, an EKG demonstrated t-wave flattening in the lateral leads and t-wave inversions in leads III and aVF. Laboratory results were significant for a troponin elevation of 0.156 and a normal d-dimer. She was started on a heparin and nitroglycerin drip, metoprolol tartrate and atorvastatin. A serial EKG demonstrated dynamic changes, t-wave inversions in leads V3-V6. Her troponin continued to rise, peaking at 12.528. Echocardiogram demonstrated new mid-anteroseptal and mid-inferoseptal wall akinesis. Surprisingly, her coronary angiogram demonstrated tortuous coronary arteries without stenosis. The differential diagnosis expanded to include stress cardiomyopathy, microangiopathic disease or myocarditis. All of which could explain the troponin elevation and wall motion abnormality, in the absence of obstructive coronary artery disease. CMRI was obtained to further elucidate the cause of her new cardiomyopathy. CMRI demonstrated anteroseptal wall hypokinesis and late gadolinium enhancement showing subendocardial hyperenhancement of the mid-anteroseptal segment and microvascular obstruction in a septal branch of the LAD coronary artery. This was consistent with an ischemic process, a true myocardial infarction.

Discussion: This case highlights the expanded differential in patients presenting with MINOCA and the utility of cMRI in establishing a definitive diagnosis of true myocardial infarction. Potential causes of MINOCA include plaque disruption, coronary thrombosis, vasospasm, spontaneous coronary artery dissection or type II MI. In a meta-analysis of MINOCA publications, cMRI was able to identify subendocardial infarction in 24% of patients. Other findings
on cMRI included myocarditis (33%), Tako-subo cardiomyopathy (18%), dilated cardiomyopathy (2%), and abnormalities such as pericarditis and amyloidosis (7%). 26% of patients had no evidence of a myocardial abnormality. Additional investigations to determine the etiology of MINOCA that have been used include intracoronary imaging with IVUS or optical coherence tomography, provocative vasospasm testing, as well as evaluating for thrombophilia. Given the various etiologies, it is important to pursue additional testing to guide long term management.

Kenneth Aduba
Dr. Brett Hendel-Paterson

NNO Laughing Matter: A Case of Nitrous Oxide Induced Myeloneuropathy

Nitrous oxide (N2O), also known as “laughing gas”, has long been used as an inhalational anesthetic in surgical and dental procedures; in manufacturing; and in food processing, where it is commonly used as a foaming and mixing agent in whipped cream. In the body, it converts active reduced vitamin B12 to the inactive oxidized form by an irreversible oxidation reaction. Vitamin B12 plays a role in the synthesis of myelin sheaths. Due to its euphoric effect and easy availability, nitrous oxide has gained popularity as an inhalant of abuse by addicts, who conveniently inhale it from whipped cream chargers known as “whippits”. This has led to cases of acute vitamin B12 deficiency with neuropsychiatric presentations. Below is a case of subacute combined degeneration of the spinal cord following chronic abuse of nitrous oxide.

Case description: A 24 year old female presented to the ED with a one week history of progressive bilateral upper and lower extremity tingling, numbness and weakness; gait imbalance and diminished fine motor skills. She gave a one year history of recreationally inhaling “Whippits” every weekend but reported using it every day in the 2 week period before her symptoms appeared, inhaling as much as 50 to 100 canisters one night while partying with friends.

Examination revealed bilaterally absent vibration sensation up to her knees; bilaterally absent proprioception in toes; mild dysmetria and mild sensory ataxia with finger-nose and heel-shin tests; and positive Romberg. Her hemoglobin was 9.9 g/dl and MCV 98 fl. She had low vitamin B12 of 164 pg/ml; elevated homocysteine of 30 µmol/L; and elevated methylmalonic acid of 1.17 µmol/L. Complete metabolic profile; folate level; CSF chemistry, microbiology and immunology were unremarkable. MRI of the spine revealed abnormally hyperintense signal within the dorsal columns of the spinal cord from C1-C6 with possible extension into the cervicothoracic junction compatible with subacute combined degeneration due to acute vitamin B12 deficiency. The patient was treated with weekly vitamin B12 injections for 6 weeks and regular physical and occupational therapy sessions. She made a full recovery after 3 months.

Lessons: It is often easy to forget less common, yet potentially deleterious chemicals of abuse such as nitrous oxide. Physicians need to consider a wide range of differentials when investigating neuropsychiatric presentations. Though neurotoxicity from Vitamin B12 deficiency can potentially be reversed as occurred with our patient, other cases have reported only partial recovery with long term deficits and even death. Prognosis depends on early diagnosis, which usually rests on quick clinical suspicion, cord findings, and exclusion of other causes. Appropriate vitamin B12 supplementation, abstinence from nitrous oxide, as well as physical and occupational therapy are also crucial to recovery.

Hamna Ahmad
Dr. James Leatherman

A Rare Presentation of GPA

Introduction: Granulomatosis with polyangiitis (GPA) is one of the antineutrophil cytoplasmic antibody-associated vasculitides predominantly involving airways, lung parenchyma, and kidneys. Pleural involvement,
however, as the initial presentation is very rare.

Case Presentation: A 37-year-old male was referred to the pulmonary clinic for evaluation of right-sided pleural effusion and a pleural based mass that was found on CT after he presented with chest pain. He had had a biopsy of the mass that was non-diagnostic. On follow-up imaging, he had some improvement in the pleural effusion but persistence of the mass and development of sub-pleural nodules. He developed hemoptysis shortly after. Additional history revealed that he had also had some hearing loss in his right ear and had been seen by an ear nose and throat specialist some months before and had fluid aspirated from his middle ear. His review of systems otherwise was negative. His physical examination was unremarkable. Further work-up included a T spot and fungal serologies that were negative. A PET CT scan was done that revealed very high levels of uptake in the right lower lobe mass and the sub-pleural nodules in addition to the persistent pleural effusion. PET scan was also significant for otitis media and otomastoiditis. He underwent a thoracentesis for the pleural effusion and at that time, one of the sub-pleural nodules was biopsied that showed granulomatous inflammation with necrosis but special stains were negative for microorganisms. The pleural fluid showed predominance of T lymphocytes. Fungal and AFB cultures were negative. Because of the hemoptysis, a bronchoscopy was done that was entirely normal. At this point, the differential included noninfectious etiologies including IgG4 related disorder, granulomatosis with polyangiitis and pulmonary hyalinizing granuloma. His anti-neutrophil cytoplasmic antibody was positive at 1-160 and his anti-APR 3 antibody was markedly elevated at over 2000. Given the markedly positive anti-proteinase 3 antibody titer together with the pulmonary and pleural findings in the absence of positive cultures to suggest an infectious disorder, GPA was the most likely diagnosis. The patient was treated with Prednisone and Rituximab therapy with appropriate response.

Conclusion: This case illustrates the importance of the fact that GPA should be considered when thinking about the differential diagnoses of pleural effusions especially in the absence of other common etiologies.

Hasan Ahmad Hasan Albitar
Dr. Robert Vassallo

Lymphatic Plastic Bronchitis with Chyloptysis Secondary to Thoracic Duct Stenosis

Background: Plastic bronchitis is a condition characterized by expectoration of branching bronchial casts. It is most frequently encountered in children with congenital heart disease following Fontan surgery, and is uncommon in adults. We report a case of lymphatic plastic bronchitis secondary to thoracic duct stenosis in a 42-year-old male.

Case presentation: A 42-year-old previously healthy male presented to the clinic with episodic cough productive of thick secretions that had the appearance of branching airway casts. He noticed that symptoms were worse upon consumption of foods with a high fat content and conversely, improved with a low fat diet. Chest CT scan showed multifocal nearly confluent centrilobular groundglass opacities in the right middle and lower lobes. Bronchoscopy with bronchoalveolar lavage (BAL) revealed milky fluid return from the right middle and lower lobes with bronchial casts in the right middle lobe. Infectious studies performed on the BAL and casts were negative. BAL triglyceride level was 479 mg/dl and histopathologic analysis of the casts with Oil Red O staining showed 76% lipid-laden macrophages consistent with chyloptysis.

A diagnostic lymphangiogram was performed and revealed segmental obstruction of the thoracic duct and diffuse abnormal lymphatic collaterals in the supraclavicular region with back-filling of many small lymphatic vessels towards the upper mediastinum. Subsequently, percutaneous thoracic duct embolization was performed and no more filing of the left upper chest.
collaterals was seen. Following lymphangiography and embolization the patient experienced complete resolution of symptoms. He denied any further episodes of chyloptysis or coughing up casts on follow-up.

Discussion: Plastic bronchitis is uncommon in adults. In the past, it was thought that most cases in adults are idiopathic. However, recent studies have shown that most patients have abnormal pulmonary lymphatic flow. Our case highlights that evaluation of abnormal lymphatic flow in patients with plastic bronchitis is important as interventional procedures may prove curative in such cases.

Tiffany Albrecht  Which worm? Cavitary Lung Lesion in a Young Traveler

Case: An 18-year-old healthy male from Eastern China presented to the hospital with three months of cough progressing to one month of fevers, night sweats, hemoptysis, and weight loss. He had been seen multiple times at an urgent care and was initially diagnosed with allergic rhinitis. One month later, he was seen at a clinic for onset of hemoptysis. A chest radiograph showed a left lower lobe consolidation and Quantiferon Gold was found to be negative. He was diagnosed with community-acquired pneumonia and started on a 5-day course of azithromycin. One week after completing the antibiotics, his symptoms had progressed with new night sweats and 10-kilogram weight loss. He was admitted to the hospital for intravenous antibiotics and further work-up.

On admission, computed tomography of the chest showed left lower lobe consolidation with multiple cystic appearing foci and a 5-centimeter collection of air and fluid. Sputum gram stain and cultures and serial Acid-Fast Bacilli cultures were unrevealing. Additional travel history was significant for a recent trip to China during which patient consumed shellfish, raising concern for parasitic infection. Sputum paragonimus, stool ova and parasite, and strongyloides, echinococcus, and filaria IgG were negative. He was empirically treated with praziquantel and discharged on an empiric 6-week course of augmentin. At follow-up, symptoms had improved, but new peripheral eosinophilia was noted.

Four months following discharge, the patient developed fever and abdominal pain. Repeat computed tomography of the chest showed new scattered pulmonary nodules and large cystic cavities in the left lower lobe. Echinococcus IgG, strongyloides IgG, stool ova and parasite were, again, negative. Symptoms resolved spontaneously without antibiotic therapy. Work-up for vasculitis with myeloperoxidase and proteinase 3 antibodies was negative. Pulmonary cystic echinococcus was thought to be the most likely etiology despite negative serologies given the appearance of cystic lesions and the poor sensitivity of serologic testing in extrahepatic echinococcus.

Conclusion: This case highlights several important points for the general internist. First, as international travel and immigration become more common, parasitic infections should be included in the differential diagnosis of cavitary lung lesions, particularly in the presence of peripheral eosinophilia. Second, definitive identification of an organism can be challenging in these cases as radiologic and laboratory evaluation has limited sensitivity and empiric treatment is often necessary. Finally, close follow-up is warranted in cases of diagnostic uncertainty as non-infectious etiologies such as vasculitis and malignancy can mimic parasitic infections of the lung.

Alexandra Alejos  Broad Differential to Catch Broad Based Buds

Case: An 18-year old male presents to clinic with a two week history of mildly productive cough and four days of penile sores. He notes chest pain with the cough as well as sweats, chills, poor appetite and back pain. He denies fevers
but notes he has been taking acetaminophen and ibuprofen and three days of valacyclovir. He is febrile to 101.3 with an exam notable for clear lungs and four 2mm scabbed penile lesions. He is prescribed azithromycin for bronchitis and lesions are swabbed for HSV. He takes his azithromycin as directed but continues to have worsening symptoms and presents to the emergency department four days later noting persistent cough and night sweats as well as weight loss and sore throat. Social history reveals tobacco and marijuana use and that he spends time in caves near his home. He is afebrile and saturating well on room air. His chest x-ray is remarkable for a right upper lobe pneumonia with a likely cavitation. Labs are notable for a negative monospot and strep test and leukocytosis to 17.1. He is given ceftriaxone and prescribed levofloxacin and close follow up. CT scan the following day shows severe pneumonia with cavitation primarily affecting the right upper lobe. He is switched to piperacillin-tazobactam and vancomycin and admitted for inpatient management of pneumonia. Further testing revealed positive urine EIA for histoplasmosis and he is started on amphotericin. Serum testing is negative for blastomycosis and histoplasmosis. Sputum gram stain on hospital day two shows yeast consistent with candida and occasional broad-based budding yeast. He continues to be febrile and methylprednisolone is added. He has worsening leukocytosis and continuing fever, so a lung biopsy is performed. The biopsy shows diffuse alveolar damage without fungi and was complicated by a pneumothorax.

Conclusion: Blastomycosis is not uncommon in Minnesota. It is worth noting that even with appropriate therapy patients may have ongoing fevers and it can take one to two weeks for symptoms to improve. In this case the positive sputum and urine were consistent with blastomycosis and sufficient for diagnosis without additional information. Urine antigen testing for histoplasmosis is known to cross react with blastomycoses. Blastomycosis is considered a reportable disease in Minnesota, however these guidelines vary from state to state. Amphotericin B is the mainstay of treatment for severe disease and is well known for causing hypokalemia and hypomagnesemia. Hypokalemia in particular is very common due to an increase in membrane permeability and can induce a non-anion gap metabolic acidosis. Amphotericin can also cause acute kidney injury, which is generally reversible when amphotericin is stopped. This mechanism is largely not understood. The main way to lower this risk is by avoiding other nephrotoxic medications.

Kelsey Angell
Dr. Joshua Owen
Dr. Amy Candy

Subcutaneous Nodules Heralding an Aggressive Acute Monocytic Leukemia

Case: A 78-year-old man with a history of diabetes, gout, hyperlipidemia, asthma, hypertension and prostate cancer treated with radiation and chemotherapy presented to the emergency department (ED) with shortness of breath, dry cough and lower extremity weakness. He had seen his primary care provider a week prior for weakness, shoulder pain and dyspnea and was prescribed a short course of prednisone. He subsequently developed acute scrotal swelling and underwent ultrasound demonstrating bilateral testicular masses and was referred to Urology for further outpatient evaluation. On presentation to the ED, his exam was unremarkable except for diffuse, soft, non-tender 1-2 cm subcutaneous nodules over his trunk and extremities. Initial laboratory data was significant for white blood cell (WBC) count 13.2×10^9/L and hemoglobin of 12.8 g/dL. His chemistry panel and platelets were within normal limits. Chest xray showed diffuse interstitial prominence. He was admitted to the hospital and administered levofloxacin for possible community acquired pneumonia. On labs the following day, his WBC rose to 25×10^9/L and subsequently to 45×10^9/L and automated differential showed lymphocyte and monocyte predominance. His creatinine rose from 1.8 mg/dL to 2.4 mg/dL. Hematology/Oncology was consulted, a peripheral smear obtained and bone marrow and subcutaneous nodule biopsies were planned. The following morning, the patient developed acute respiratory distress and somnolence. An arterial blood gas demonstrated respiratory acidosis and the
patient was placed on BiPAP and transferred to the ICU. A CT of the chest and abdomen showed numerous subcutaneous solid nodules, bilateral pleural effusions, extensive lymphadenopathy in the chest, abdomen, and pelvis, mild splenomegaly, mild ascites and hypodensities in the liver and spleen thought to be leukemic deposits. His WBC continued to rise to 74 ×10^9/L. A peripheral smear showed 76% blasts, predominantly monocytic with small percentage of conventional myeloblasts. Findings were consistent with acute myeloid monocytic leukemia with morphologic evidence of active microangiopathy. His uric acid was 11.5 mg/dL, LDH over 4000 U/L and creatinine increased to 4.2 mg/dL. He became more encephalopathic with progressive respiratory distress and oliguria. His respiratory distress was thought to be secondary to leucoagglutination in the lungs and his skin nodules were likely leukemic deposits. The patient’s disease appeared to be progressing rapidly with overall poor prognosis. Oncology recommended hydroxyurea to temporize the disease and did not feel the patient would tolerate the toxicities of induction chemotherapy. A multidisciplinary meeting was held with the patient’s family to discuss his care. Given a lack of curative therapies, his family elected to pursue comfort measures with transition to hospice care and the patient expired the following day.

Subhepatic pseudocyst: a rare cause of septic shock following ERCP

Introduction: Pancreatic pseudocysts are abnormal mature collections of pancreatic fluid that can develop in association with acute or chronic pancreatitis. They can occur anywhere in the abdomen, and only rarely develop in the subcapsular hepatic space. Here, we share the discovery of an infected hepatic subcapsular pseudocyst of the pancreas causing septic shock following endoscopic retrograde cholangiopancreatography (ERCP).

Case Description: A 55-year-old woman with ethanol-related chronic pancreatitis and biliary stricture was transferred to the ICU for hypotension 8 hours following ERCP. She was originally admitted for acute cholangitis and underwent ERCP for an exchange of the occluded stent placed 4 months earlier. After the procedure, her abdominal pain briefly resolved, before worsening with associated diaphoresis and hypotension. Abdominal examination revealed mild right upper quadrant tenderness without sign of peritonitis. Laboratory studies were notable for leukocytosis (14.6 k/L), 1.5 g/l hemoglobin drop, and lactic acidosis (2.6 mmol/L) with slightly elevated serum lipase (489 U/L). Abdominal CT scan demonstrated a previously undescribed subcapsular fluid collection and a large volume of fluid throughout the peritoneum with associated inflammatory changes. The presence of pneumobilia suggested the biliary stent remained patent. The patient was empirically started on vancomycin and piperacillin-tazobactam and ultimately required vasopressor support. She underwent CT-guided percutaneous subcapsular drain placement with immediate return of 1.5 liters of opaque yellowish fluid. Fluid analysis showed elevated lipase of 62,901 U/L with cultures positive for ESBL Escherichia coli, Streptococcus constellatus, and Enterococcus faecium, consistent with infected pancreatic pseudocyst.

Discussion: This case report highlights an atypical presentation of pancreatic pseudocyst as well as a rare septic complication of ERCP. At present, the mechanism by which subcapsular pseudocysts develop is unknown, though it is likely driven by inflammatory pancreatic juices slowly eroding through the hepatic capsule. In a recent review of available case reports, intrahepatic subcapsular pseudocysts were seen most frequently in patients with chronic alcoholic pancreatitis. Patients generally presented with nonspecific abdominal pain and were diagnosed via CT. The management of subcapsular pseudocysts has not been standardized and generally involves placement of a percutaneous drain, operative intervention, or simple aspiration; however, endoscopic strategies have also been described. Generally, operative intervention has been
reserved for severe infection or rupture in patients with intrahepatic pseudocysts. Rarely do subcapsular pseudocysts become infected, with only a few case reports available in the literature. In this case, we postulate the pseudocyst became seeded by bacteria during ERCP resulting in infection and subsequent sepsis.

**William Archibald**  
Dr. Shruti Patel  
Dr. Tariq Azam

**Avoiding IRIS in the Millet Fields**

Introduction: Treating HIV and tuberculosis (TB) simultaneously presents challenges for the treating physicians as underlying infections are associated with the immune reconstitution inflammatory syndrome (IRIS) when beginning antiretroviral treatment for HIV. Here, we present the case of a patient who through careful timing of antituberculosis and antiretroviral therapy, IRIS was avoided.

Case: A 33 year-old man of Vietnamese descent presented to the emergency department with severely altered mental status and diffuse lymphadenopathy. Initial workup consisted of CBC, CMP, and CXR. He was found to have hyponatremia to 125, an elevated white count of 10.2, and his chest x-ray showed diffuse micronodules throughout both lungs. Subsequent CT scan showed centrally necrotic cervical adenopathy and a miliary pattern of pulmonary nodules, suspicious for TB. Antimicrobials were held pending clinical workup. However, on hospital day 2, the patient became septic and he was empirically started on RIPE therapy. HIV testing was positive with a CD4 count of 4 confirming the diagnosis of AIDS. An AFB sputum smear came back positive for TB, confirming the diagnosis of TB. Additionally, cultures of sputum and lymph node biopsy were positive for TB. His altered mental status remained of unknown origin, and two LPs were performed during his hospitalization in an attempt to confirm CNS involvement of TB. Both came back negative for TB involvement of the CNS, but due to his continued altered mental status, moxifloxacin was added empirically to the patient’s treatment regimen given the concern for CNS involvement despite negative LPs. Antiretroviral therapy was held due to the risk of IRIS being particularly severe in patients with TB involving the CNS. However, the patient’s mental status eventually improved and ART was initiated on day 15 of RIPE therapy and was well tolerated. The patient was discharged to home to complete directly observed TB therapy. IRIS occurs as a result of the newly reconstituted immune system from ART therapy causing an excessive inflammatory response due to a preexisting infectious process. Patients with HIV diagnosed with TB are particularly at risk for IRIS.

Conclusion: The risk of IRIS in the HIV patient with TB dictates treatment timing. Infectious disease experts recommend in patients like ours above with HIV and a CD4 count less than 50 that ART be initiated within 2 weeks after starting TB treatment if there is no CNS involvement. For CD4 counts greater than 50, ART can be initiated 8 weeks after antituberculosis therapy begins. For individuals with TB CNS involvement, regardless of CD4 count, ART is held for 8 weeks due to the risk of IRIS. The evidence for these strategies is strong, with the SAPiT, STRIDE, and CAMELIA trials all contributing to the evidence base.

**Jennifer Arnold**  
Dr. Ryan Kelly

**The Journey to Transplant: Addiction as a Speed Bump, not a Roadblock**

Introduction: Opioid dependence and Opioid Use Disorder has risen to epidemic proportions in recent years and this in turn has complicated the medical management of our patient’s other comorbid conditions. Current first line treatment for opioid use disorder (OUD) is Medication Assisted Treatment (MAT) with buprenorphine-naloxone or methadone.
Case Presentation: A 36 year old male with cystic fibrosis who during the work-up for lung transplantation admitted to 1 year of illicit opioid abuse. The patient had been taking over 150mg of oxycodone a day after his brother’s suicide. During an admission for failure to thrive he was successfully initiated on buprenorphine-naloxone and was stable on 24mg a day. The initial transplant plan was to list the patient for transplant only after he had been off of buprenorphine-naloxone for 6 months. However, the likelihood of this patient being able to maintain sobriety off buprenorphine-naloxone without maintenance treatment with buprenorphine-naloxone was highly unlikely, and would likely lead to the patient dying prior to making it to lung transplantation. After multiple interdisciplinary educational meetings regarding opioid abuse disorder and maintenance with MAT, the patient was able to be listed for transplantation while on MAT. Continued multidisciplinary coordination was required for the day of surgery plan which involved holding buprenorphine-naloxone the day of surgery and providing the patient with a hydromorphone PCA. The patient maintained sobriety while on buprenorphine-naloxone and underwent a double lung transplant 8 months after initiation of buprenorphine-naloxone. He was reinitiated on buprenorphine-naloxone several days postoperatively.

Discussion: There is only reported case of organ transplantation while on buprenorphine-naloxone in the literature, a heart transplant, making this the first known case of a patient with OUD on buprenorphine-naloxone receiving a lung transplant. This patient was almost not listed for transplant due to misunderstandings of sobriety while on buprenorphine-naloxone among his care team. Even with the high prevalence of OUD and known treatment success with buprenorphine-naloxone, this case highlights the need for greater understanding of OUD and treatment with buprenorphine-naloxone among physicians.

Tess Baril
Dr. Collin Messerly
Dr. Nicha Wongjarupong
Dr. Wojciech Kraszkiewicz

To Transfuse or Not to Transfuse: A Unique Case of Autoimmune Hemolytic Anemia

Case: A 25-year-old woman presented to the emergency department (ED) with ten days of dyspnea on exertion, headaches, and her heart racing at rest. The patient had previously presented to primary care two weeks ago for similar symptoms where she was treated for possible sinus infection with amoxicillin-clavulanic acid. Since that time, her dyspnea and tachycardia have increased. On arrival to the ED, the patient was markedly pale, weak, and was found to have splenomegaly. Labs were significant for hemoglobin 5.0 g/dL, hematocrit 13.3%, Haptoglobin <8 mg/dL, LDH >1000 U/L, as well as elevated bilirubin with elevated AST. There were no signs of bleeding. Current medications include oral contraceptive pills, naproxen, aspirin, caffeine, and amoxicillin-clavulanic acid. The patient was admitted for further workup which revealed a reticulocyte index of 0.2%, a positive Coombs test, and hypogammaglobulinemia with low IgG and IgA levels suggestive of common variable immunodeficiency (CVID). The patient had no history of recurrent infections. She was started on IV methylprednisolone 250 mg daily for treatment of autoimmune hemolytic anemia (AIHA). Due to her hypogammaglobulinemia, IVIG was initiated. The patient did not respond to steroids or IVIG. She was not transfused until her hemoglobin fell below 4.0 g/dL. She ultimately tolerated transfusions well, but increasing LDH was noted climbing to >5000 U/L. Her reticulocyte index remained low at 0.2%. Parvovirus testing was negative. Her bone marrow biopsy revealed normal production of all cell lines. Due to her failed response to steroids, the patient was offered second line treatment of splenectomy versus rituximab. She was ultimately treated with rituximab and her hemoglobin stabilized around 5.0 g/dL. She was discharged home with plans to complete four cycles of rituximab, continue high dose steroids at 1 mg/kg and have close follow up with hematology. After completing all four doses of rituximab, the patient reports...
improvement in her symptoms, her labs show a hemoglobin of 8.7 g/dL, an appropriate reticulocyte index of 4.3%, and LDH 1772 U/L. This case highlights the treatment options for autoimmune hemolytic anemia and the importance of a complete workup which in this case revealed an underlying immunodeficiency.

Conclusion: Common variable immunodeficiency is a primary immunodeficiency disorder characterized by impaired B cell differentiation with defective immunoglobulin production. It is the most prevalent form of severe antibody deficiency in both children and adults, affecting about 1 in 50,000 individuals. About 20% of patients with CVID develop autoimmune disease. Autoimmune hemolytic anemia is due to anti-red cell antibodies binding to antigens on the surface of red cells causing their rapid destruction. A reticulocyte count takes 7-10 days to peak in response to anemia. This case demonstrates the use of a second line agent, rituximab, when the first line treatment of steroids fails.

Alexander Baxa

Entertain all Possibilities: An Unusual Case of Cardiogenic Syncope

Introduction: Syncope is a concerning clinical picture that can have many etiologies including neurogenic, cardiovascular, neuropsychogenic, orthostatic hypotension, metabolic, medicinal, etc. Ultimately, the cause needs to be determined in a timely, yet thorough manner, ensuring that presumably improbable causes are not neglected.

Case presentation: A 56 year-old male with a history of gout, hypertension, diverticulosis, and nephrolithiasis was admitted to the hospital after a syncopal episode while biking. He describes that while biking he experienced tunnel vision and lightheadedness prior to veering off the road. The patient denied chest pain, shortness of breath, palpitations. He is an avid biker and subjectively has high level of fitness having cycled 100 miles last week – this was just another morning for him.

Recent history was remarkable for mid-back pain for which the patient underwent an exercise stress test which was negative. History of “T-wave things” which he was told were benign, occasional lightheadedness upon standing, a recent viral URI. Patient denies history of syncope, loss of consciousness, heart disease, arrhythmias, and stroke.

Initially, the differential remained broad. CT head was without intracranial pathology, CT neck without significant stenosis of major arteries. EKG with T-wave inversions and q waves that were reported to be chronic. Troponins were noted to be 0.052. Telemetry remarkable for premature atrial and ventricular contractions. Outside stress echo from outside hospital noted to be normal in 2016. Overnight, patient was found to have non-sustained ventricular tachycardia and cardiology was consulted. A stress test was performed and was remarkable for diffuse wall motion abnormalities, severely decreased left ventricular systolic performance with stress, and with remarks of being MARKEDLY abnormal. The patient was without angina. Cath lab was activated and patient found to have severe three vessel disease. PCI completed and patient discharged on dual anti-platelet therapy.

Discussion: Cardiogenic syncope can be diagnosed when a patient loses consciousness secondary to a confirmed cardiac illness. This case reiterates that, as clinicians, it is always necessary to complete a thorough workup for all patients that present with concerning syncope. This workup should include all possibly etiologies, including those that are less likely. In this case, without a complete cardiac workup, it would have been near impossible to make the correct diagnosis. Yet, in a patient with a negative cardiac history, a recent negative cardiac stress test, an unchanged EKG, lack of chest pain, shortness of breath, palpitations, and a very slight elevation of troponins, it would have been
reasonable to forgo further testing in the acute setting. Fortunately for this patient, thorough analysis and workup was completed and significant disease was not missed. If we are presented with concerning syncope, it is essential to entertain all possibilities.

Robertson Bayer

**More Than a Pain Crisis: A Case of Multifocal Acute Stroke in an Adult With Sickle Cell Disease**

Introduction: Sickle Cell Disease (SCD) affects about 100,000 Americans. Patients with this condition are at risk for multiple complications beside simple anemia. Among the most concerning of these are acute vaso-occlusive events, such as Acute Chest Syndrome and acute ischemic stroke. Prompt recognition of these events and rapid initiation of appropriate therapy is critical in preventing significant morbidity and mortality.

Case: We present the case of a middle-aged woman with sickle cell disease who has had frequent pain crises as well as a recent deceased donor kidney transplant who initially presented with one of her typical pain crises but subsequently developed multifocal ischemic strokes. Her initial presentation in the Emergency Department was notable for a hemoglobin above her typical baseline and diffuse pains throughout her lower extremities. She had no respiratory or neurologic symptoms at time of presentation. She was started on fluids, pain management, and supplemental oxygen to maintain supraphysiologic oxygenation. Approximately 36 hours later, she developed acute onset of upper extremity weakness which was followed very shortly thereafter by rapidly decreasing level of consciousness. She had no fever or leukocytosis and had not been complaining of any significant neck pain prior to clinical deterioration. She was transferred to the ICU and a dialysis/apheresis catheter was placed for exchange transfusion. She did not require intubation. While awaiting transfusionist arrival and appropriate blood availability from blood bank, she underwent MRI demonstrating multifocal acute ischemic strokes. Approximately 1 hour into exchange transfusion, she had improvement in her mental status and by the following morning had only minimal residual left upper extremity weakness. She eventually discharged to a Transitional Care Unit and subsequently regained nearly full strength of the affected extremity and had minimal residual deficits.

Discussion: The cause of this patient’s acute decompensation was not fully clear. She had been receiving erythropoietin injections for anemia due to her kidney disease, and there was some concern this may have contributed. Her rapid improvement with exchange transfusion is typical of these events. However, there can be significant logistical difficulties in promptly initiating this treatment at centers that do not perform it frequently. Further, the procedure itself is not without risks, the most common of which are hemodynamic changes from the exchange as well the typical risks associated with blood transfusion. This case can be used to highlight the details of the exchange procedure itself, the typical indications, and discuss some ways to ensure prompt access for those patients who need it most.

Evan Beacom

Dr. Ryan Jelinek

**Granulomatous Mediastinitis Secondary to Histoplasmosis**

Introduction: Histoplasmosis is an endemic fungal infection along the Ohio River Valley, in Minnesota and in Wisconsin. It is the most common of the endemic mycoses to result in hospitalization, with an estimated 500,000 cases in the U.S. annually. The typical port of entry for the pathogen is the lungs. Severity of illness depends on host factors such as underlying lung disease, as well as pathogen factors including size of inoculum. Histoplasmosis often presents as self-limited, subacute pneumonia in the immunocompetent host. Chronic pulmonary infection manifests in those with underlying lung disease. Disseminated disease is considered an AIDS-defining illness. This dimorphic
fungus also causes a myriad of extrapulmonary manifestations in humans about which little is known.

Case Description: MM is a 37 year old female with PMH of GERD taking only occasional H2 blockers who presented with two weeks of substernal chest pain and odynophagia. She endorsed three days of fever up to 102F with shaking chills for 24 hours. She had difficulty swallowing, but no weight loss, regurgitation, or progression of GERD symptoms. Initial laboratory workup showed mild leukocytosis.

CT pulmonary angiogram was obtained for evaluation of chest pain. This revealed a 4.0 x 3.3 cm calcified subcarinal lymph node and thickening of the distal esophagus. Pulmonary findings were consistent with chronic histoplasmosis, but this did not explain the patient’s fever, chills, odynophagia and leukocytosis. Concern for mediastinitis prompted esophagram and EGD. Esophagram was unrevealing. EGD showed a small fistula in the distal third of the esophagus draining purulent material. Diagnosis of acute mediastinitis was made and the patient was started on Augmentin.

Serology for histoplasmosis revealed M-band on immunodiffusion and complement fixation titers of 1:8. Itraconazole was started. Esophageal stenting was considered to prevent further leaking of GI contents into the mediastinum, and the patient was transferred to another hospital where specialty-trained cardiothoracic surgery was available in the event of esophageal rupture.

Discussion: Granulomatous mediastinitis is a known, albeit rare, complication of histoplasmosis. This is a syndrome of enlarging, encased and sometimes necrotic mediastinal lymph nodes in patients with a history of pulmonary histoplasmosis. Calcified masses, as in our patient, may cause bronchial stenosis, esophageal fistulae, among other sequelae. Mediastinal granuloma is a progressive syndrome with a variable natural history, and is generally not amenable to simple cure with antifungals. Itraconazole should be tried, but often treatment is surgical to remove granulomatous tissue or repair damage it causes. This syndrome can threaten the function of vital structures in the mediastinum. Early recognition of chronic histoplasmosis is thus important for patients with lung disease or smoking history in endemic areas, even without clear history of exposure, in order to recognize and treat this uncommon complication of a common endemic mycosis.

Brian Berglund
Dr. Gretchen Rasmusson

Oh, the Irony: A Tale of Iron Infusion Causing Weakness, Fatigue and Profound Hypophosphatemia

Introduction: This case presents an interesting and increasingly recognized incidence of severe hypophosphatemia caused by intravenous iron.

Case: A 56-year-old female presented two weeks after receiving parenteral ferric carboxymaltose for iron deficiency anemia secondary to celiac disease. Her symptoms included extreme fatigue, myalgia, perioral numbness and carpopedal spasm. Pertinent medical history included prior thyroidectomy for papillary thyroid cancer with autotransplantation of her parathyroid gland. She had previously experienced symptomatic episodes of hypocalcemia from surgically-induced hypoparathyroidism, and vitamin D deficiency secondary to celiac disease. Her current medications included 25-dihydroxyvitamin D and calcium carbonate. On initial presentation laboratory analysis revealed decreased calcium at 7.6 mg/dL (normal 8-10), normal 25-dihydroxyvitamin D at 71.3 ng/mL (normal 30-80) and elevated PTH at 135 pg/mL (normal 14-87). She was treated with increased oral calcium supplementation without improvement in her symptoms. At this point, a serum phosphorus level was found to be low at 0.7mg/dL (normal 2.5-4.5). She was hospitalized and a 24-hour urine phosphorus level was elevated at 1.6 g/24H (normal <1.2 g/24H). A
1,25-dihydroxyvitamin D level was low at 14 pg/mL (normal 18-78). She was treated with aggressive oral and parenteral phosphorus, calcium, and calcitriol (1,25 dihydroxycholecalciferol). She was stabilized and discharged on oral medications and eventually achieved normal serum phosphorus, calcium, and 1,25-dihydroxyvitamin D after weeks of supplementation. In retrospect, the delay in diagnosis of hypophosphatemia was likely due to focus on past history of hypoparathyroidism and resultant hypocalcemia. Phosphorus balance is partly controlled by a hormone called Fibroblast Growth Factor 23 (FGF23). Four months after the insult, the patient’s FGF23 level was checked and noted to be inappropriately high given ongoing hypophosphatemia. In this case of intravenous iron-induced hypophosphatemia, the wasting of urinary phosphate is mediated by decreased degradation of FGF23. The resultant increased FGF23 causes urinary phosphate wasting through decreased transcription of sodium phosphate co-transporters in the apical brush border membrane in the proximal tubule. FGF23 also decreases transcription of 1-alpha-hydroxylase, resulting in decreased 1,25-dihydroxyvitamin D. In an effort to increase production of 1,25-dihydroxyvitamin D, PTH is secreted which promotes calcium and phosphorus liberation from bone, as well as renal calcium resorption and phosphate wasting. There have been increasing case reports of severe and prolonged hypophosphatemia and osteomalacia associated with iron infusion. Intravenous iron-induced hypophosphatemia is a complication for physicians to keep in mind given the increased use of parenteral iron. Further studies are needed to clarify which patients are at increased risk.

Aaron Boothby  
Dr. Daniel O’Leary  
Dr. Thenappan Thenappan

AMS in an Immune Suppressed Patient: Avoid Anchoring on Infection

Case: We present the case of a 65 year old male with history of benign prostatic hypertrophy, type 2 diabetes mellitus, and non-ischemic cardiomyopathy s/p heart transplant on tacrolimus, and recently hospitalized with CMV colitis complicated by AKI, who was traveling home following discharge when he developed urinary frequency, progressive confusion, and agitation. On presentation to community ER he was oriented only to person. The patient’s wife reported he had been complaining of headache and had become progressively confused and agitated while continuing to make frequent trips to the bathroom. On physical exam he was noted to be afebrile, tachycardic, hypertensive, alert, and oriented to name only. An extensive workup for altered mental status was notable only for hypomagnesemia. The patient was given magnesium, empiric broad spectrum ABX, fluids, and transferred to University of Minnesota for further evaluation. On presentation to UMN he was incoherent, tachycardic, and hypertensive. On physical exam his abdomen was noted to be distended and diffusely tender to palpation. Bladder scan revealed 1.2L of retained urine which was drained via catheter. Vancomycin and ceftriaxone were initiated for empiric meningitis coverage and a LP was performed; LP results were ultimately unremarkable. An MRI was ordered; however, completion was delayed due to continued agitation. Mental status slowly improved, and MRI was completed on hospital day 4 showing multiple confluent regions of T2 hyperintensity consistent with posterior reversible encephalopathy syndrome (PRES). All infectious workup returned negative and the patient returned to baseline mentation on hospital day 7. Discussion: PRES is thought to be caused by failure of cerebral autoregulatory mechanisms to adapt to changes in blood pressure resulting in vasogenic edema. However, the exact mechanism is not fully understood. It is known to occur more frequently in patients with the following factors; immune suppression, hypomagnesemia, AKI, and hypertension. Altered mental status, headache, seizures, and visual changes are the most common presenting symptoms. Rapid recognition of the trigger and reversal of hypertension are the key elements of treatment. With treatment, symptoms tend to resolve in a week, although deficits may linger. In the immune suppressed patient, it is easy to anchor on infectious etiologies, our case underlines the importance of maintaining a broad differential and immediate recognition of easily reversible inciting factors. In this case, urinary
retention led to painful bladder distention causing hypertension and was the likely triggering event resulting in PRES. Cysto-cerebral syndrome is another described etiology of AMS in which urinary retention leads to altered mental status. It tends to occur in elderly patients and is speculated to result from sympathetic hyperactivity secondary to bladder distention. However, unlike this case, symptoms typically resolve very quickly upon decompression of the bladder.

Thomas Breen  Dr. Tony Chon  Dr. Patrick Hoversten

Hemophagocytic Lymphohistiocytosis: Recognizing a Rare Cause of Febrile Neutropenia

Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening disorder characterized by excessive activation of CD8+ T lymphocytes and macrophages that lead to organ damage. Prompt diagnosis and initiation of treatment is imperative for survival of affected patients; however, the variable clinical presentations and rarity of this syndrome make it challenging to diagnose. Current recommendations for establishing a diagnosis include molecular identification of an HLH-associated gene mutation or meeting five of eight clinical criteria. These include fever ≥38.5°C; splenomegaly; peripheral blood bicytopenia; hypertriglyceridemia and/or hypofibrinogenemia; hemophagocytosis in bone marrow, spleen, lymph node, or liver; low or absent NK cell activity; ferritin >500 ng/mL; and elevated soluble CD25 (soluble IL-2 receptor alpha). Treatment requires immunosuppression, typically with etoposide and high dose steroids.

Case: An 18-year old female with no past medical history developed sepsis following routine wisdom teeth extraction. Subsequent evaluation identified neutropenia with an ANC of 0. Subsequent workup for underlying hematologic disorders led to the diagnosis of HLH after meeting six of eight clinical criteria, including splenomegaly, hyperferritinemia (3,092), hypertriglyceridemia (273), bicytopenia (hemoglobin 8.6, ANC 0), fevers (>38.5°C), and elevated soluble IL-2R (9,790). Bone marrow and liver biopsies were non-diagnostic. Genetic testing was negative for congenital HLH. Work-up for rheumatologic, infectious, and malignant etiologies were non-revealing. Patient was started on high dose prednisone and offered etoposide for presumed HLH. She refused etoposide due to the negative side effect profile and self-discontinued prednisone therapy shortly after discharge.

Patient was admitted several weeks later with fevers, tachycardia, an abscess on the right inner thigh, and sinus pain. The abscess subsequently developed necrosis requiring extensive debridement. Cultures grew Corynebacterium, stenotrophomonas maltophilia, E. faecalis, and E. cloacaee. CT sinus showed pansinus and bilateral mastoid inflammatory opacification with mucosal thickening involving the ostiomeatal complexes. ENT was consulted and performed debridement with pathology that demonstrated invasive fungal sinusitis, thought to be aspergillosis. She was initiated on a broad-spectrum antibiotic and antifungal regimen.

Hematology subsequently started the patient on prednisone and filgastrim. Etoposide was held due to the active infections. Her ANC remained 0. The infections continued to progress, requiring daily nasal debridement and further debridement of the thigh wound. Cultures drawn from the thigh wound subsequently grew fusarium, which carries a 50% rate of mortality. The fusarium was thought to be disseminated. Patient was started on granulocyte transfusions for a 7-day course and the neutrophil count slowly started to rise. She remained hemodynamically stable and her infections slowly started to resolve.

Discussion: HLH is a rapidly progressive and life-threatening syndrome that is challenging to diagnose. It is essential to establish a diagnosis of HLH and
begin treatment with immunosuppressive agents quickly to minimize the progression of the syndrome and avoid some of the grave complications.

Courtney Burnett  
Dr. Susan Burton  
Dr. Bennett Clark

A Case of Diacetyl-Induced Lung Injury Secondary to Banana Flavored E-Cigarettes

Introduction: Since the 1960s, the prevalence of conventional cigarette use among adults in the United States has declined, while the use of electronic cigarettes has increased. We present a case of diacetyl (2,3-butenedione)-induced lung injury secondary to flavored e-cigarette use. Of particular importance to general internists, this case exemplifies the importance of adjusting history taking to include questions on e-cigarette use specifically.

Case: A 43-year-old female with a history of interstitial lung disease presented with one day of cough and shortness of breath. She had been admitted to the hospital ten times in the preceding twenty-one months for similar episodes. Upon arrival to the emergency department, her oxygen saturation was 65% on room air. She was tachycardic and tachypneic. Laboratory evaluation was notable for a white blood cell count of 17.8 k/ul. Rapid influenza test, pneumococcus and legionella urine antigens, polymerase chain reaction respiratory viral panel, and 1,3-β-D-glucan tests were negative. At the time of admission to our service, the patient was briefly treated with empiric antibiotics for presumed bacterial pneumonia. A CT angiogram of the chest revealed no pulmonary embolus but did show stable diffuse centrilobular ground glass opacities on chronic emphysematous changes. Symptoms improved rapidly and the patient maintained SpO2 > 90% on room air by the following morning.

Our patient used banana flavored e-cigarettes, which she started around the time of her first admission for acute hypoxia. She reported worsening of symptoms with the use of flavored e-cigarettes and rapid improvement in symptoms while abstaining. The exposure-related time course of the patient’s acute recurrent respiratory failure and her rapid improvement when abstaining led to a diagnosis of ILD secondary to diacetyl-induced lung injury. Diacetyl-induced lung injury secondary to flavored e-cigarette use has been dubbed “flavor worker’s lung,” as previous studies have demonstrated a high prevalence of obstructive lung-function abnormalities and pulmonary disease among workers with occupational exposures to diacetyl. Studies have postulated a relationship between declining forced expiratory volume in one second (FEV1) and cumulative exposure to diacetyl.

Discussion: Of particular importance to this case, our patient initially denied conventional cigarette use. She endorsed e-cigarette use only when asked, “Do you smoke anything else at all?” suggesting that clinicians must ask explicitly about e-cigarette use to elicit a complete history of exposures. This case demonstrates an under-recognized and potentially life-threatening complication of flavored e-cigarettes and serves as a reminder to physicians that e-cigarette use is an important risk factor for lung injury. Specific, targeted questioning on e-cigarette use is a vital component of the social history and should be incorporated into routine clinical practice. Despite the popular perception that e-cigarettes are safer than traditional cigarettes, they likely carry a risk of acute lung injury due to diacetyl exposure, among other toxins.

Marilia Campos

A Novel Presentation of Neuroendocrine Carcinoma

Introduction: Pancreatic neuroendocrine carcinomas (NEC) are rare, poorly differentiated neoplasms that arise from the endocrine tissues of the pancreas. They have a very low incidence (<1 in 100,000 per year). The majority of NECs are nonfunctioning tumors and present with abdominal pain. With the less common functional NECs, patients can present with symptoms of glucagonoma, insulinoma, VIPoma and/or gastrinoma. NECs often present late, as in our
patient’s case, with metastatic disease present at the time of diagnosis. When metastatic disease is present treatment options are limited to palliative chemotherapy. Our patient’s unusual presentation with melena was a result of the location of his tumor relative to the duodenum and its vascular supply.

Case Presentation: This is a 53-year-old male with a history of multiple gastric ulcers, prior H pylori infection, and hypertension who presented to the GI clinic with chronic abdominal pain, 5 episodes of melena, and anemia. EGD revealed a medium sized ulcerated mass in the third portion of the duodenum. Ct of the abdomen and pelvis confirmed the presence of a mass in the pancreatic head with duodenal invasion and also revealed a second mass in the pancreatic tail as well as an enlarged inguinal lymph node. Pathology from the duodenal biopsy and EUS guided FNA of the pancreatic tail lesion both demonstrated high grade poorly differentiated neuroendocrine carcinoma. FNA from the inguinal node confirmed metastatic disease. Patient completed staging workup with a bone scan, brain MRI and chest CT which did not demonstrate additional metastases. With multifocal, metastatic disease, the patient was offered and undertook palliative chemotherapy, which has been well tolerated. Follow-up imaging with CT and endoscopy show decrease in size of all lesions. Despite this response, life expectancy with this aggressive tumor remains limited.

Conclusion: This case illustrates an atypical presentation for NEC resulting from direct extension into the duodenum, causing the patient to present with melena due to duodenal ulceration. His NEC may also have played a role the patient’s chronic abdominal pain which had previously been attributed to his H. pylori and gastric ulcers (however, gastrin levels were not checked). As in this patient, the non-specific nature of symptoms from non-functional NECs, combined with the low incidence in the general population makes early diagnosis and treatment challenging.

Lindsay Carafone
Dr. William Archibald

Pain After Eating? Check the PAN!

Introduction: Polyarteritis Nodosa (PAN) is not typically first on the differential when a patient presents with abdominal pain. PAN is a medium, and sometimes small, vessel vasculitis affecting muscular arteries. It can present in different ways; signs and symptoms include abdominal pain (bowel angina after meals), livedo reticularis, mono- or polyneuropathy, myalgias with tender leg muscles, new onset hypertension, unexplained weight loss, and testicular pain. This is a case of a man presenting to the hospital with abdominal cramping after eating.

Case: A 64 year old man with a history of hypertension who is otherwise healthy presents with two weeks of new onset abdominal pain and cramping which only occurs after meals. He also notes new onset myalgias, with pain in his calves and thighs when walking. Rest relieves the pain. He endorses left testicular pain and weight loss of about 10 pounds in the last few months. Initial physical exam is largely unremarkable with normal muscle strength and a benign abdominal exam. He is afebrile with blood pressure in the 150s/80s and pulse in the 70s. However, when gait is observed as he ambulates down the hallway, he begins to experience pain in his calves. Laboratory results show normal electrolytes and CBC, but elevated erythrocyte sedimentation rate >100 and elevated C-reactive protein of 37.8. At this point, ANA, ANCA and rheumatoid factor are obtained, however these are all normal. Though the ANCA was negative, there was still suspicion for vasculitis and an angiogram was obtained which showed a beading appearance of the vessels off of the superior mesenteric artery. This was consistent with the rosary bead appearance of polyarteritis nodosa, and he was started on steroids with gradual resolution of his symptoms.

Conclusion: Polyarteritis nodosa should be considered in patients presenting with otherwise unexplained abdominal pain, particularly with the characteristic
“bowel angina” that occurs after meals. A high index of suspicion for PAN is necessary in the setting of other clues to diagnosis such as myalgias, arthralgias, testicular pain, unexplained weight loss and a rash consistent with livedo reticularis. This will prevent delay in diagnosis and initiation of steroid treatment. This case also illustrates the importance of vasculitis work up beyond obtaining ANCA labs. PAN is ANCA negative. Thus even when ANCA work up is negative, further work up including angiography should be obtained if the index of suspicion is high for vasculitis such as PAN. The classic finding on imaging is segmented aneurysms with the appearance of a “rosary sign.” Biopsy of small or medium sized vessels will show polymorphonuclear cells. Treatment is steroids, followed by steroid-sparing agents such as azathioprine or methotrexate.

Supavit Chesdachai
Dr. Janewit Wongboonsin
Dr. Meghan Rothenberg

Non-Resolving Cellulitis

Cellulitis is one of the most common diagnosis, accounting for up to 10% of infectious disease-related hospitalization in the US (1). The challenge in treatment usually stems from failure to respond to empiric antibiotics which should raise the suspicion of resistant organisms, unusual organisms, or underlying pathophysiology that interferes with bacterial clearance (2). We report a case of cutaneous histoplasmosis presented with non-resolving cellulitis.

A 56-year-old male with medical history significant for psoriatic arthritis treated with monthly infliximab and pulmonary histoplasmosis on suppressive oral ketoconazole 200 mg daily since 2007, who was transferred to our facility with non-resolving cellulitis of his right forearm. One week prior to the transfer, he presented to his local clinic with two days of swelling, redness, warmth and pain in his right forearm two weeks after a steroid injection at right wrist due to chronic wrist pain. He was treated with multiple antibiotics including cephalixin, doxycycline, levofloxacin, vancomycin and piperacillin-tazobactam without improvement. He denied preceding trauma, animal contact and recent travel. Physical exam showed ill-defined erythematous plaque with swelling and warmth on the ventral aspect of his right forearm extending to his wrist and the hypothenar area of his right hand. Labs were remarkable for elevated C-reactive protein and procalcitonin. Doppler ultrasound demonstrated no evidence of deep vein thrombosis. Magnetic resonance imaging (MRI) revealed diffuse edema in the flexor musculature of the forearm extending into the carpal tunnel surrounding the flexor tendons and extending into the hand. Given the lack of improvement despite different broad spectrum antibiotics and his history of histoplasmosis, Histoplasma and Blastomyces antigens were sent from blood and urine on hospital day one. While these results were pending, skin biopsy was performed on hospital day three which identified numerous round yeast forms within suppurative inflammation and focally within histiocyte cytoplasm. Subsequently Histoplasma antigen from both urine and blood returned positive and a diagnosis of cutaneous histoplasmosis was made.

Histoplasma is the most common endemic fungi in North America (3). Dermatologic findings of cutaneous histoplasmosis are most commonly ulcerated papules and plaque, although it can also present as pustules, macules and erythema nodosum (4). There has been increasing recognition of histoplasmosis in patients receiving biologic treatments for rheumatologic disease (5). Furthermore, in these patients, histoplasmosis is more likely to present atypically and more severely (6). Our case illustrates Histoplasma as a cause of non-resolving cellulitis in a patient on a TNF alpha inhibitor and highlights the need for clinicians to maintain a high index of suspicion for this infection in patients on biologic therapies.

Christopher Choo
Jafary Fafara

An Upper Endoscopy Autoimmune “Hat-Trick”: Esophagitis Dissecans Superficialis, Autoimmune Gastritis, and Celiac Disease
Kevin Song
Dr. Brian Hanson
Dr. Hector Mesa
Emilie Hudalla

Introduction: Esophagitis dissecans superficialis (EDS) is a rare desquamating condition of the esophagus characterized by the diffuse sloughing of superficial mucosa. While the course of EDS is typically benign, the endoscopic appearance can be frightening to the unsuspecting endoscopist. It is thus important to better understand this elusive pathology. We report a case of EDS and autoimmune gastritis diagnosed in a patient evaluated for refractory celiac disease.

Case Description: A 52-year-old female with celiac disease on a strict gluten-free diet presented to outpatient endoscopy clinic with persistently loose stools, new onset esophageal dysphagia, and persistently elevated tissue transglutaminase. Her medications included meloxicam, oxycodone, and nortriptyline for fibromyalgia. Upper endoscopy revealed non-bleeding sloughing mucosa along the entire esophagus. Interestingly, ink markings were identified upon the sloughing mucosa that matched those of the patient’s nortriptyline capsule. Biopsy results found mucosal splitting and focal inflammation in the squamous epithelium, confirming EDS. Endoscopy also revealed diffuse gastric mucosa atrophy and duodenal mucosa flattening. Biopsies of these areas showed chronic gastritis consistent with autoimmune gastritis and duodenum without microscopic abnormalities.

Discussion: EDS is a rare condition. It has been associated with celiac disease, central nervous system depressants, SSRIs, NSAIDs, endoscopic injury, and bullous dermatoses. The underlying pathophysiology is not known, but is proposed to be a response to physical, toxic, allergic, and even ischemic insults. EDS is mostly a self-limited finding without lasting pathology. It can be reversed by suppressing acid, addressing the precipitating factor, or using glucocorticoids in those with bullous dermatoses. Follow-up endoscopy can be performed to evaluate mucosal healing, though no clear guidelines exist for timing or necessity of this.

Conclusion: To our knowledge, this is the first case report documenting concurrent histologic evidence of EDS and autoimmune gastritis. Prior cases have discussed the potential association between EDS and autoimmune conditions such as celiac disease and bullous dermatoses. Our case adds to a small but growing body of literature suggesting an underlying autoimmune predisposition for EDS. We now propose a link between EDS and autoimmune gastritis.

Grace Choong
Dr. R. Thomas Tilbury

How to Stop a Heart: A Case of Cardiac Noncompaction

Introduction: Noncompaction is a rare condition characterized by increased trabeculations in the ventricle that result from developmental arrest of cardiac compaction in embryogenesis. Arrhythmias, heart failure and embolic events are all clinical signs of cardiac noncompaction.

Case: Mrs. S is a 54 year old woman with a history of hypertension and hyperlipidemia who presented as an outside of hospital cardiac arrest. CPR was started immediately. Upon arrival of paramedics, the initial rhythm strip showed ventricular fibrillation and she was successfully defibrillated to a narrow complex rhythm. In the emergency room, she had a new left bundle branch block with ventricular tachycardia and was cardioverted to a narrow complex rhythm. An arterial blood gas revealed a pH 7.15, PCO2 57, bicarbonate 20. Other blood work was significant for potassium 3.4, bicarbonate 18, white blood cell count 22.6, and initial high-sensitivity troponin 92 (normal ≤ 10). Chest x-ray showed signs of pulmonary vascular congestion. Urgent coronary angiogram showed no significant coronary artery disease. Transthoracic echocardiogram revealed a left ventricular ejection fraction (LVEF) of 25%, moderate-severe left ventricular enlargement, and prominent trabeculations that
did not meet criteria for cardiac noncompaction. The subsequent rhythm showed signs of short PR interval and atrial fibrillation, suggestive of pre-excitation atrial fibrillation. Prior ECG strips redemonstrated a short PR interval without evidence of a delta wave. Electrophysiology was consulted who was concerned for evidence of an accessory pathway. An MRI demonstrated borderline evidence of left ventricular noncompaction and severe left ventricular enlargement without evidence of inflammation or infiltrative disease process. Her LVEF had improved to 50%. An electrophysiology study was subsequently performed, but an accessory pathway could not be demonstrated, suggestive of noncompaction being the primary cause of her cardiac arrest. Due to concerns of recurrent cardiac arrest with noncompaction, an intracardiac defibrillator was placed. She was dismissed on amiodarone, lisinopril, metoprolol and apixaban for new onset atrial fibrillation.

Discussion: Though this case did not meet all criteria for a diagnosis of cardiac noncompaction, it highlights some uncommon causes of cardiac arrest. Common causes of cardiac arrest, including ischemia, electrolyte abnormalities, acidosis, thrombosis should be ruled out first. Shortened PR intervals may indicate the presence of an accessory pathway bypassing the atrioventricular node, which can lead to life-threatening arrhythmias. The first imaging modality to diagnose cardiac noncompaction include echocardiography and possibly cardiac MRI to further characterize the ventricular wall and rule out infiltrative causes of cardiac arrest. Patients with a diagnosis of cardiac noncompaction should be managed with a β-blocker, ACE-inhibitor, and loop diuretic dependent on symptoms.

Conclusion: Noncompaction is a rare structural abnormality that should be on the differential when all other causes of cardiac arrest have been ruled out.

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### Caitrin Coffey

**Dr. Jason Eckmann**  
**Dr. Alberto Rubio-Tapia**  
**Dr. Darrell Pardi**

**Chronic Intestinal Pseudo-Obstruction with Pneumatosis Intestinalis in a Patient with Mixed Connective Tissue Disease**

**Introduction**: Chronic intestinal pseudo-obstruction (CIPO) is a disorder of the gastrointestinal tract causing dilation of the bowels and obstructive symptoms despite no mechanical obstruction. CIPO can be a result of paraneoplasia, visceral neuropathy or myopathy, or infection. It is also a rare complication of several connective tissue diseases and should be recognized in patients with rheumatologic disease and recurrent symptoms of bowel obstruction.

**Case Description**: A 63 year-old woman with a history of mixed connective tissue disease manifesting predominantly as inflammatory arthritis and chronic fatigue, two years of recurrent small bowel obstructions, chronic diarrhea, and malnutrition was admitted to the hospital urgently from a clinic visit due to concern for small bowel obstruction. Her symptoms included nausea, bloating, and constipation, and CT scan showed dilated loops of bowel with extensive pneumatosis intestinalis. Detailed history and review of prior records and imaging showed no substantial change from the patient’s baseline symptoms. Physical examination revealed a distended, soft abdomen with hyperresonance to percussion and no tenderness to palpation. Laboratory workup revealed mild, chronic anemia but was otherwise unremarkable. Review of the CT enterography with enteroclysis showed dilated loops of small bowel with no distinct transition point. A preliminary diagnosis of CIPO was made. The patient’s diet was advanced, constipation resolved without intervention, and she was discharged the following day to pursue continued outpatient workup. Subsequent workup with EGD revealed grade D esophagitis with culture of a small bowel aspirate consistent with small intestinal bacterial overgrowth (SIBO). Colonoscopy was normal. Esophageal manometry revealed aperistalsis of the esophagus. Rheumatology recommended continued treatment with methotrexate for inflammatory arthritis. RNP antibody was positive at >8 (normal <1.0); ds-DNA, anti-Smith, SSA/SSB, Jo-1 and scl-70 antibodies were
Discussion: Recognition of CIP in patients with over six months of nausea, bloating, malnutrition and diarrhea or constipation, particularly in the setting of an underlying connective tissue disorder, may allow prevention of unnecessary hospitalization or surgery. Diagnosis relies on awareness of the condition, detailed history and physical examination, recognition of potential underlying causes, and exclusion of mechanical obstruction with cross-sectional abdominal imaging. Complications of CIP include SIBO, malnourishment, chronic pain and nausea, and impaired quality of life. It is important to recognize that pneumatosis intestinalis and even pneumoperitoneum, which may result from chronic intestinal distension in CIP, do not typically represent acute surgical conditions and generally do not correlate with bowel ischemia. Surgical intervention may be deleterious and should be avoided. Treatment of CIP includes symptom control, dietary modifications and optimizing nutrition, as well as treatment of the underlying systemic condition when present. Limited data supports use of octreotide, and for those with SIBO, antibiotic treatment is indicated to improve symptoms.

**Deirdre Croke**

**Stop Horsing Around: Ask a Better Social History**

Case: A 68 year old man with a history of type II diabetes mellitus, cirrhosis, and left hip arthroplasty presented with acute onset of drenching night sweats, subjective fevers and a several month history of worsening left hip pain, low back pain, and weight loss. He did not have a leukocytosis and comprehensive metabolic panel was within normal limits. ESR and CRP were elevated to 44 and 94, respectively. Blood cultures were drawn and four of four bottles resulted positive for Gram positive cocci in clusters within several hours. He was admitted for further work up. A CT of the abdomen showed a pocket of thick-walled fluid around the left hip prosthesis, and an MRI of the spine was normal. Urinalysis and chest X-ray were unremarkable. The left hip joint fluid was aspirated and the patient was empirically started on cefazolin. The cultures were identified as Streptococcus Equi subspecies zooepidemicus. Upon obtaining further history from the patient, it was discovered that he has frequent contact with horses, the most likely source of this bacteria. He handles, feeds, and cleans horses almost daily and one of them frequently huffs through his nose/mouth onto the patient.

Streptococcus equi subspecies zooepidemicus is an opportunistic pathogen found in horses and as a rare human zoonosis. It is associated with upper respiratory tract infections in horses and causes strangles, a syndrome of lymphadenitis that can lead to suffocation due to compression of the upper airway. In humans Strep equi has been associated with severe and fatal infections including endocarditis, arthritis, meningoencephalitis, and septic shock. The patient’s horses had not been ill, and horses can be asymptomatic colonizers of the bacteria.

Discussion: The fluid aspirated from the patient’s hip was sterile, and transthoracic echocardiogram showed no evidence of vegetations. The patient was transitioned to IV penicillin for a 6 week course with resolution of his symptoms. He was advised to wear a mask around the horses and to practice consistent thorough hand washing after contact with horses. Providers should be aware of this uncommon and potentially life threatening condition, mostly involving persons with close contact with horses. This case demonstrates the importance of a more targeted and thorough social history to inform the source, potential complications, and risk of recurrence in cases of rare infections.

**Elizabeth David**

**Dr. Thomas Freeman**

**Diagnosis of Metastatic Pancreatic Adenocarcinoma After Presentation with Isolated Sclerotic Bone Lesions**
Introduction: Although exocrine pancreatic cancer makes up <5% of new cancer annually in the US, it is the 3rd leading cause of cancer death in women and 4th in men. A large part of this discrepancy is due to the fact that pancreatic cancer is often not diagnosed until late in the course of the disease. Unfortunately, by the time of diagnosis, only 15-20% of patients are candidates for curable surgical resection. Painless jaundice, the classic presentation, occurs in only ~50% of patients while more non-specific symptoms, such as weight loss, asthenia, anorexia, and abdominal pain are much more common. Here, we present a case report of a patient who presented with abdominal pain who was incidentally found to have diffuse sclerotic bone lesions concern for metastatic cancer and was eventually diagnosed with metastatic pancreatic adenocarcinoma.

Case: A 68-year-old male with a history stable lung nodules and a recent diagnosis of celiac vasculitis, presented with R-sided abdominal pain. The symptoms had been present for 4 months and led to a significant functional decline. He also noted a 30lb unintentional weight loss. Extensive outpatient workup included a benign laboratory evaluation and unremarkable EGD. CT imaging of his abdomen and pelvis one month prior to presentation showed enhancement of the celiac artery most consistent with vasculitis. Rheumatology evaluated the patient and initiated empiric high-dose prednisone (60mg) and azathioprine. His symptoms did not improve after two weeks and he presented back at the VA. On admission, his labs were notable for a mild leukocytosis and an isolated elevated alkaline phosphatase. Repeat CT abdomen/pelvis was significant for re-demonstration of the celiac and mesenteric enhancement, but more notable for new sclerotic bony lesions within the pelvis and lumbar spine. The imaging did not identify and obvious primary tumors. A subsequent bone scan showed uptake within the proximal and axial skeleton most concerning for metastatic malignancy.

He subsequently underwent a malignancy workup including PSA (normal, 0.67), TSH and thyroid ultrasound (unremarkable). A PET scan showed avid uptake in the sclerotic bone lesions as well as in the area of the celiac plexus, similar to the CT findings. Again, no primary lesions were identified. He underwent bone biopsy, with pathology confirming metastatic adenocarcinoma with an uncertain primary. The differential included upper GI or pancreaticobiliary. Hematology/oncology and gastroenterology were consulted and recommended that given the rapid progression, the patient should undergo an endoscopic ultrasound. The patient was found to have a 10.3mm x 15.8 mm pancreatic head lesion as well as a liver lesion concerning for metastatic disease. Biopsies confirmed the diagnosis of metastatic pancreatic adenocarcinoma. The patient was discharged from the VAMC with a plan for close follow up for initiation of palliative chemotherapy.

Kathryn del Valle
Dr. M. Caroline Burton

Swelling, Nausea, and Shortness of Breath in a 19 year-old Woman

Introduction: Nephrotic syndrome can have many systemic manifestations in addition to severe proteinuria and anasarca, such as secondary immune compromise and subsequent severe infections.

Case: A 19 year-old woman without significant past medical history was admitted with shortness of breath and hypoxia, nausea, and severe edema. Over the past 2-3 months, she had experienced worsening systemic swelling and associated 30-lb weight gain. In the few days prior to admission, she had increased cough and difficulty breathing as well as nausea, vomiting, abdominal pain, and diarrhea. Upon presentation, she was tachycardic to 122 and hypoxic; she required 4L supplemental O2 via nasal cannula to maintain O2 saturation >88%. Physical exam revealed diminished breath sounds in the bases, 2+ pitting edema in bilateral lower extremities, tender cervical lymphadenopathy, and diffusely tender, mildly distended abdomen. Initial laboratory evaluation
was notable for creatinine 1.24, albumin 1.1, sodium 131, total cholesterol 480, and triglycerides 473. 24-hour urine collection revealed 6.28 g of protein. IgG level was markedly low at 95 (normal 767-1590), with IgA and IgM levels on the low end of their respective normal ranges. Chest CT showed diffuse bilateral tree-in-bud and ground glass opacities. Abdominal x-ray was unremarkable, and renal ultrasound was negative for thrombosis, hydronephrosis, or other structural abnormalities. C.difficile stool testing was also negative.

She was given IV albumin supplementation, diuresed, and started on broad spectrum antibiotics for community acquired pneumonia. Evaluation for secondary etiologies of nephrotic syndrome, including autoimmune panel, HBV/HCV/HIV serologies and other infectious work-up (sputum and blood cultures, fungal serologies, etc.), and monoclonal protein studies, was negative. Renal biopsy was performed, and pathology confirmed minimal change disease. Bronchoscopy showed diffuse bronchitis but no other endobronchial abnormalities; bacterial, viral, and fungal studies from bronchial specimens were negative. Therefore, she was started on high dose prednisone and given IVIG approximately 24 hours later. Of note, several days following admission she underwent neck ultrasound with plans for biopsy given her cervical lymphadenopathy and concern for underlying lymphoma; however, by time of ultrasound her lymphadenopathy had resolved, suggesting it was of primarily infectious etiology.

Over the course of her 5-day admission, she made remarkable clinical improvement. She was successfully weaned off of supplemental oxygen, her gastrointestinal symptoms resolved, and she felt much better overall. She was discharged with plans for close outpatient follow up.

Discussion: Nephrotic syndrome can precipitate severe multisystem organ dysfunction even in previously healthy young adults, such as acute hypoxic respiratory failure secondary to immune compromise and gastrointestinal distress. Diagnostic evaluation should include infectious, autoimmune, hematologic, and structural renal disease assessments. Though minimal change disease more commonly presents in children, it remains an important cause of nephrotic syndrome in adults, and the mainstay of treatment is high-dose systemic steroids.

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**Brinda Desai**

**Can “Intestinal” Behcet’s Disease Completely be Separated from Crohn’s Disease?**

Introduction: Behcet's disease (BD) is a multi-systemic vasculitis characterized by multi-systemic organ involvement. Diagnosis is based on clinical criteria - there are no pathognomonic laboratory tests. Although gastrointestinal (GI) and systemic features of BD and inflammatory bowel disease overlap to a considerable extent, they are generally viewed as two distinct diseases. Oshima et al. reported that over 40% of BD patients had GI complaints. Although GI symptoms are common, the demonstration of GI ulceration is rare in pure BD. This so-called intestinal BD accounts for only 1-2% of cases. In intestinal BD, ulceration of the GI tract can be found throughout the intestine, but the most frequent area of involvement is the ileocecal region. Only 15% of intestinal BD diffusely involve the colon.

Case: A 25-yr-old female was admitted to our hospital because of oral and genital ulcer, lower abdominal pain, and frequent bloody diarrhea. Vitals were significant for temp of 38.8°C, blood pressure of 98/55, Pulse of 142, respiratory rate of 20. Lab work-up was significant for leukocytosis of 13.2, hemoglobin for 7.4, MCV for 67, and platelets for 374. Lactic acid was elevated to 5.8. Patient was fluid resuscitated, blood and urine cultures were collected, and started on empiric antibiotics for sepsis with concern for intra-abdominal
infection. Work-up for infectious etiologies were negative. Colonoscopy showed diffuse involvement of multiple longitudinal ulcers with a cobblestone appearance which are extremely rare in Behcet's disease.

However, there were no granulomas, which is the hallmark of Crohn's colitis. Patient's bowel symptoms, endoscopic appearance, and the response to medical treatment (steroids and infliximab) were compatible with Crohn's colitis. Therefore, BD and Crohn's disease may be closely related and part of a spectrum of disease rather than always existing as two distinct diseases. This then brings to question how we treat a patient who presents with an overlap of these two diseases. Further evaluation should be done to evaluate the efficacy of combination treatment for the overlap presentation of Crohn’s and Behcet’s.

**Samuel Dolezal**

*Follow your heart: Clozapine a Life-Saving or Life-Threatening Treatment for Schizophrenia?*

**Introduction:** Clozapine is an atypical antipsychotic frequently utilized in treatment-resistant schizophrenia. Although effective in managing symptoms of schizophrenia, patients require routine monitoring for potential side effects. Many of the potential side effects can potentially mask as commonly presenting medical conditions offering clinicians a formidable diagnostic challenge.

**Case:** A 45-year old male with history of schizophrenia and marijuana abuse presented to an outside hospital Emergency Department with rash, generalized weakness, and confusion. Symptoms started the evening prior to presentation and rapidly progressed prompting evaluation. On arrival, vitals and labs were consistent with severe sepsis including fever, tachycardia, leukocytosis, and elevated lactate. Chest X-ray showed pulmonary edema without clear infiltrate. Fluids and broad-spectrum antibiotics were started for sepsis of unclear etiology before transfer to a tertiary medical center. Preliminary blood cultures returned positive for Gram-positive cocci in clusters.

Despite appropriate treatment for presumed Gram-positive sepsis, the patient's overall condition rapidly declined, and he suddenly became hypoxic and hypotensive requiring intubation, sedation, and multiple vasopressors. Echocardiogram performed was significant for normal left ventricular size and thickness with severe global hypokinesis with an estimated left ventricle ejection fraction of 15%. Additional history obtained from family revealed that the patient had started clozapine five days prior to the start of presenting symptoms. Given concern for a possible medication-induced side effect, clozapine was withheld, and patient subsequently improved with supportive cares. Blood cultures eventually resulted positive for coagulase negative Staphylococcus, a presumed contaminant. Serial echocardiograms revealed significant improvement with return to a normal ejection fraction approximately one week after initial presentation. The patient was placed on an alternative antipsychotic regimen, discharged from the hospital with close follow up, and has been symptom free since.

**Discussion:** This case illustrates the potential risk for severe cardiomyopathy associated with the use of clozapine. Clinicians should have a low threshold to urgently perform echocardiography in suspected clozapine-induced cardiomyopathy. Clozapine should be withheld in the presence of cardiomyopathy without additional explanation as recognition of this condition is critical in order to prevent a potential life-threatening side effect. Although not well studied, expert opinion recommends that patients with clozapine induced cardiomyopathy also should not be re-challenged with the medication.

**Jason Eckmann**  
**Dr. Caitrin Coffey**  
**Dr. Vandana Nehra**

*Rebel without a Cause: A Case of Localized Gastrointestinal AL Amyloidosis without Underlying Plasma Cell Dyscrasia*
Introduction: Amyloidosis is an uncommon condition caused by the deposition of misfolded serum proteins in extracellular tissue, including in the gastrointestinal (GI) tract. Immunoglobulin light chain associated (AL) amyloidosis, which less commonly involves the GI tract, occurs as a result of underlying clonal plasma cell proliferation. Here, we present a case of symptomatic localized gastrointestinal AL amyloidosis without evidence of underlying plasma cell dyscrasia.

Case: A 75 year-old man with history of type II diabetes mellitus and prostate cancer treated with androgen deprivation therapy and external beam radiation presented with one month of watery diarrhea with intermittent hematochezia and nausea. Medications included aspirin, meloxicam, metformin, and omeprazole. Vital signs and physical examination were normal. Complete blood count demonstrated a mild anemia without leukocytosis. Stool studies including tests for bacterial pathogens, ova, and parasites were negative. Further investigation with colonoscopy was remarkable for erythematous, friable mucosa in the distal sigmoid colon and rectum. Pathology from this area, as well as from random biopsies throughout the colon, demonstrated deposits with apple-green birefringence under polarized light with Congo red staining. Mass spectrometry confirmed the presence of AL (lambda)-type amyloid deposition. Gastric and small bowel transit studies were within normal limits. EGD with duodenal biopsies showed amyloid infiltration of the second part of the duodenum. Serum free light chains were normal, serum and urine protein electrophoresis with immunofixation showed no evidence of monoclonal-protein spike, and bone marrow biopsy was negative for clonal plasma cell proliferation. The patient was diagnosed with localized AL amyloidosis of the GI tract without evidence of underlying plasma cell dyscrasia.

Discussion: Gastrointestinal amyloidosis is most commonly associated with systemic disease, although approximately 20% of cases are limited to the GI tract. Presenting symptoms generally arise from malabsorption or dysmotility, and include diarrhea, weight loss, abdominal pain and gastrointestinal bleeding. Most cases of GI involvement are associated with AA (reactive) amyloidosis, though AL amyloidosis represents approximately 1% of cases. Light chain-related disease is almost always associated with underlying clonal plasma cell proliferation and systemic disease, and only rare cases of localized AL amyloidosis of the GI tract without plasma cell dyscrasia have been reported. Recognition of these cases is crucial, as patients do not require systemic chemotherapy and have a favorable long-term prognosis. Diagnosis of GI amyloidosis is challenging and requires a high degree of clinical suspicion. Importantly, negative serum/urine protein studies or bone marrow biopsy may not be sufficient to exclude underlying amyloidosis. Therefore, in unexplained cases of diarrhea or GI bleeding, patients should undergo endoscopy with tissue biopsies and Congo red staining, the gold standard for diagnosis, to definitively rule out GI amyloidosis.

Jonathan Edmiston
Dr. Emily Hudson
Dr. Kealy Ham

Flaccid Paralysis after Fishing Trip

Introduction: West Nile Virus (WNV) is a mosquito-borne flavivirus causing seasonal outbreaks in the United States and should be considered in febrile patients with acute neurologic illness with possible exposure. 70 - 80% of patients are asymptomatic, but those with symptoms experience headache, weakness, myalgia, arthralgia, and sometimes gastrointestinal symptoms or maculopapular rash. Less than 1% of those infected develop neuro-invasive disease; either meningitis, encephalitis, or acute flaccid paralysis. Respiratory weakness can occur with anterior horn cell involvement, isolated limb paresis can occur with WNV induced poliomyelitis, and an associated Guillain-Barre syndrome can also contribute to weakness. As of September 2018 there has been only 1 neuroinvasive case of WNV in Minnesota.
Case: Our case is a 47 year old male with a past medical history of schizophrenia and distant IV drug use who presented after 1 week of headache, abdominal pain, diarrhea, malaise, and fever. His girlfriend called EMS when she came home to find him sitting on the floor and verbally unresponsive. In the ED he was febrile (105) and unresponsive with flaccid right upper extremity weakness and tonic flexion of left. Labs revealed leukocytosis of 13.1, lactate 2.7, CK 12,414, and CSF with lymphocyte predominant pleocytosis of 510 and protein elevated to 58 and he was initiated on broad spectrum antibiotics.

He developed hypoxic respiratory failure and was intubated. MRI showed enhancement of leptomeninges, left thalamus (subacute infarct vs. cerebritis) and cervical anterior horns. On further investigation his girlfriend remembered he had been on a fishing trip a few weeks prior and an extensive encephalitis panel was sent out which eventually came back positive for WNV and Jamestown Canyon Virus (JCV) IgM. Over the next week he developed generalized weakness, no longer able to move any of his extremities against gravity. Diaphragmatic weakness was suspected as well when he was unable to wean off the ventilator. He was eventually transitioned to a tracheostomy and weaned to trach-dome.

Conclusion: This case demonstrates the complexity of neurologic sequelae resulting from arbo-virus meningoencephalitis. The patient came in with possible seizure, with elevated CK and tonic upper extremity activity. His left upper extremity flaccid paralysis is likely explained by poliomyelitis with the area of enhancement in the central cervical cord. EMG findings and significant generalized weakness which subsequently developed point towards an associated Guillain-Barre syndrome which could also explain his diaphragmatic and respiratory weakness. It is also interesting that he tested positive for WNV and JCV. These can both cause neuroinvasive disease for which the treatment is largely supportive care. There are case reports of treatment with IVIG treatment or treatment with IVIG from populations living in areas endemic with WNV, however there are mixed results and more data is required.

Matthew Eggebrecht

Cough to Cavitary Lesion: Multifactorial Pulmonary Case

Case Description: A 67 year-old male with history of GERD, chronic ischemic heart disease and GERD presents to the hospital with concerns for an atypical respiratory infection. Patient was seen in pulmonary clinic 3 months prior with cough, sputum and dyspnea. Initial chest CT was significant for GGO and GG nodular opacities in the RUL. Treated with a course of Levofoxacin with temporary improvement.

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An Unexpected Cause of Chronic Diarrhea

Introduction: Chronic diarrhea, defined as diarrhea that persists for more than four weeks, is a common (affecting 3-5% of the population) but challenging diagnostic scenario. Given the multitude of possible underlying causes, a thorough history (including medication history), physical examination and cost-efficient diagnostic work-up is paramount.

Case presentation: A 64 year old female with a history of GERD, hypertension and type 2 diabetes mellitus presented to her primary care physician with a 3 month history of intractable, non-bloody diarrhea and steatorrhea (with up to 30 bowel movements per day that occurred during the day and night). This was associated with cramping abdominal pain which was not relieved by defecation, along with an unintentional 20 pound weight loss. Her symptoms were severe enough to result in dehydration and hypotension, necessitating inpatient admission. There was no relief with diphenoxylate-atropine or loperamide. She denied the use of artificial sweeteners, and her symptoms were not relieved by exclusion of dairy and gluten. Home medications included insulin and
olmesartan. Work-up included negative stool pathogen studies (including Clostridium difficile), colonoscopy with random biopsies (negative for microscopic colitis) and negative celiac serology. A CT scan of the abdomen/pelvis showed no acute pathology and CT enterography showed no small bowel changes. An upper endoscopy was grossly normal, with no growth from small bowel aspirates. Duodenal biopsy showed evidence of partial villous atrophy with crypt hyperplasia, chronic inflammation and an increase in intraepithelial lymphocytes consistent with possible drug-related injury. As olmesartan had been reported to cause a similar picture, this medication was discontinued and the patient was prescribed anti-diarrheal medications. The patient reported significant improvement in her symptoms at an outpatient follow-up appointment a few weeks after discharge.

Discussion: Causes of chronic diarrhea include inflammatory bowel disease, microscopic colitis, malabsorption syndromes (including lactose intolerance, chronic pancreatitis, celiac disease and small intestinal bacterial overgrowth), chronic infections, irritable bowel syndrome and medications. While villous atrophy has classically been associated with celiac disease, there has been increased interest in villous atrophy secondary to drug-induced enteropathy. Classic culprit medications include azathioprine, mycophenolate, methotrexate, neomycin, colchicine and the angiotensin receptor blocker, olmesartan. Case series of olmesartan resulting in severe, chronic diarrhea have triggered the FDA to institute label changes warning of this adverse effect in 2013. The enteropathy can develop months to years after medication initiation. Treatment includes discontinuation of the offending medication and supportive anti-diarrheal therapy.

Conclusion: A thorough medication history is a crucial component of evaluating chronic diarrhea, as several medications can result in drug-induced enteropathy resulting in malabsorptive diarrhea. It is important that internists are aware of medications like olmesartan which can result in chronic diarrhea secondary to villous atrophy.

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Arrested Red Blood Cell Maturation

Introduction: Emerging treatments for metastatic melanoma have made dramatic effects on survival for patients; however, their potential toxicities are less well known.

Case Presentation: A 26 year old female with recently diagnosed metastatic anal melanoma was admitted to hospital service for acute anemia with fatigue. She completed her third cycle of nivolumab and ipilimumab after local resection. Three days prior to admission, she had been evaluated at local emergency room; she was transfused with two units of packed red blood cells for hemoglobin 6.9, MCV 77.6, and reticulocytes less than 31%. Her physical exam upon admission was unremarkable with no evidence of jaundice or bleeding. Her laboratory studies showed hemoglobin 7.1, MCV 79.4, indirect bilirubin 1.3, haptoglobin <14, and LDH 877, which was consistent with hemolytic anemia. Additional lab results were negative for blood cultures, hepatitis panel, HIV, herpes simplex virus, parvovirus B19, paroxysmal nocturnal hemoglobinuria, glucose-6-phosphate dehydrogenase deficiency, disseminated intravascular coagulopathy profile (includes platelet count, PT, aPTT, thrombin time, fibrinogen, d-dimer, and soluble fibrin monomer), Coombs test, anti-nuclear antibodies, and thyroid profile. The continued presence of low reticulocytes less than 31% prompted a bone marrow biopsy which showed hypercellular marrow with decreased and left shifted erythropoiesis and increased megakaryocytes and granulocytes. Since there was no maturation of red cells and primarily red cell lineage affected, this was consistent with pure red cell aplasia.
She was treated with blood transfusions as needed to maintain hemoglobin above 7, in addition to intravenous immunoglobulin, methylprednisolone, and cyclosporine. She remained hemodynamically stable during hospitalization. She was discharged with cyclosporine and prednisone. She required frequent blood transfusions on a weekly basis and after a month of treatment, she exhibited clinical improvement with stable hemoglobin and reticulocyte. She was tapered off cyclosporine and methylprednisolone once her hemoglobin normalized. Nivolumab and ipilimumab were discontinued; she continued surveillance for her metastatic melanoma.

Discussion: Pure red cell aplasia (PRCA) is a syndrome defined by a normocytic normochromic anemia with severe reticulocytopenia and marked reduction or absence of erythroid precursors from the bone marrow. There are two main types of PRCA: congenital (Diamond-Blackfan anemia) and acquired. Acquired PRCA can be associated with infections, such as parvovirus B19; drugs such as azathioprine and procainamide; neoplasms such as thymoma, lymphoma, or carcinoma, and autoimmune diseases. Abnormalities from PRCA are limited to the red cell lineage. PRCA in rare instances is associated with cancer therapies such as immune checkpoint inhibitors (ICI).

Conclusion: PRCA should be considered in patients receiving ICIs, especially when no other source for acute anemia is found. Treatment for acquired PRCA includes treatment of underlying cause or cessation of offending drug, as well as supportive care with blood transfusions. Immunosuppressant such as corticosteroids and cyclosporine should be considered.

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Not All Obstruction is Mechanical: A Case of Colonic Pseudo-Obstruction

Introduction: Acute colonic pseudo-obstruction is a disorder characterized by dilation of the colon in the absence of an anatomic obstruction. For the general internist, differentiating severe colonic distension due to pseudo-obstruction from other diseases that can lead to colonic dilation, such as toxic megacolon, volvulus, or a true obstruction, is critical to initiate appropriate treatment, specialist consultation, and avoidance of perforation.

Case: We present a case of a 93-year-old female who was transferred to our institution for intermittent abdominal pain, abdominal distension, and severe colonic distension seen on abdominal plain film. Her past medical history is significant for advanced dementia, hypertension, and immobility due to right hip fracture status post hemi-arthroplasty 3 months prior, complicated by recurrent cellulitis of the surgical site requiring recent admission for intravenous antibiotics.

Upon presentation, further history was notable for intermittent crampy lower abdominal pain with occasional loose stools, but no associated nausea or vomiting. Current medications included IV vancomycin for right hip cellulitis, oxycodone as needed for pain, and amlodipine for hypertension. Exam demonstrated a protuberant abdomen with mild discomfort to palpation without rebound or guarding and decreased bowel sounds. She was afebrile and hemodynamically stable.

Initial work-up yielded a leukocytosis of 13.9 and CRP of 54.5. CT of the abdomen/pelvis demonstrated a markedly distended sigmoid colon, measuring 15 cm in diameter, consistent with toxic megacolon with no evidence of obstruction or volvulus. Clostridium difficile PCR returned negative.

Given reassuring vitals, no peritoneal signs on exam, and negative C. Diff PCR, toxic megacolon was thought to be less likely and she was diagnosed with colonic pseudo-obstruction in the setting of recent operation, use of opioids, and...
calcium channel blocker. Conservative management with tap water enema and nasogastric/rectal tube decompression was initiated but was unsuccessful. A Hypaque enema was then performed, demonstrating no obstruction, but decompression was unsuccessful. Treatment was escalated with IV neostigmine which caused temporary complete heart block, that resolved with glycopyrrolate, but did not lead to decompression. Urgent flex sigmoidal decompression was subsequently performed. On the following day, she became increasingly dyspneic with oxygen desaturations in the 80s. Chest X ray demonstrated bilateral pulmonary congestion and pleural effusions. Following discussion with family, comfort care was initiated and she was discharged to hospice.

Discussion: This case illustrates the several factors that may lead to colonic pseudo-obstruction in susceptible patients including: metabolic derangements, trauma, neurological disease, orthopedic surgery, and the use of offending medications. It highlights the several treatment options ranging from supportive care, including enemas, rectal tube compression, and avoiding anti-motility medications, to more aggressive therapies, including IV neostigmine and flex sigmoidoscopy. Lastly, it demonstrates factors influencing outcomes of colonic pseudo-obstruction including age, severity of illness, and duration of colonic distention.

John Emmel

When Snoring Isn’t Boring: Lessons for Primary Care

Introduction: The primary care clinic is the first place that patients go when they sense that something is wrong. It is important to recognize when a common complaint has red flags that merit further exploration. In this case, a 70 year-old overweight man presented with sudden-onset sleep apnea. His symptoms began four days prior due to pharyngeal swelling from superior vena cava (SVC) syndrome.

Case Presentation: A 70 year-old overweight male with a history of CAD (s/p CABG 2yr prior) and a 45pkyr history of smoking presented to his primary clinic with 4 days of new-onset snoring. He had been “afraid to go to sleep” because he didn’t want to stop breathing. He was reassured that this was “nothing serious” and a sleep study was ordered.

The next day, he presented to the ED for one day of facial swelling. CT PE study revealed a large mediastinal mass compressing the SVC. PET showed a likely primary lung cancer widely metastatic to bones and liver. Biopsy confirmed small cell lung cancer and he was started on palliative chemotherapy with carboplatin and etoposide and is now on third-line chemotherapy.

Discussion: Sleep disordered breathing is a very common complaint in the primary care setting. It can be associated with underlying neurologic, cardiac, or pulmonary disease in cases such as Cheyne-Stokes breathing or central sleep apnea. However, it is much more commonly caused by an obstruction in the upper airway. This obstruction causes varying degrees of increased airway resistance during sleep and leads to a spectrum of pathology from primary snoring to obstructive sleep apnea (OSA). Obstruction is most often attributed to body habitus or oropharyngeal tissue relaxation. In Westernized countries, it is estimated that as many as 15% of middle-aged men and 5% of middle-aged women have OSA. Occasionally, however, snoring and/or the cessation of breathing during sleep can be caused by other underlying pathologies, and these are imperative not to miss. A primary physician should be watchful for red flags in the history or physical exam which can point to these pathologies. Red flags include rapid onset or worsening of snoring, facial swelling, frequent sore throats, mouth breathing, enlargement of oropharyngeal or facial tissues, neurologic deficits, or dysphagia. These can point to underlying diagnoses such as SVC syndrome, adenotonsillar hypertrophy, chronic allergic rhinitis,
| **Erik Engebretson**  
| **Dr. Erin Main**  
| **Gas Where You Least Expect It: A Case of Clostridium Sepsis following Endoscopy**  
| **Introduction:** Clostridium perfringens is a gram-positive rod-shaped spore forming bacteria that is part of the normal flora of the gastrointestinal tract. It is a common cause of food poisoning, but can also cause more serious infections such as clostridial myonecrosis which is commonly known as gas gangrene. The toxin implicated in the process of clostridial myonecrosis is the α-toxin.  
| **Case:** A 70-year-old male presented to the emergency department with a one day history of acute onset abdominal pain. Three days prior to his presentation he had undergone an uneventful upper endoscopy with biopsy for surveillance of Barrett’s esophagus. Additionally, there was an incidental finding of three gastric polyps that were removed. On presentation the patient was afebrile, hypertensive and ventricular paced with a heart rate of 60. Physical exam demonstrated diffuse abdominal pain that was most prominent in the epigastric region with significant guarding. Laboratory studies on initial presentation were notable for a leukocytosis of 12.93 10³/µL and normal lactate of 1.7 mmol/L. Abdominal x-ray and CT scan of the abdomen and pelvis were unable to reveal an etiology for his pain and he was admitted to observation for further monitoring. Over the next 12 hours he continued to have severe abdominal pain. He remained afebrile, but developed worsening hypoxia and hypotension. His mental status started to deteriorate. Repeat labs were obtained and revealed a worsening leukocytosis of 15.6 10³/µL and elevated lactate of 13.2 mmol/L. He was given intravenous fluids and started on broad-spectrum antibiotics. He was transferred to the intensive care unit where he required urgent intubation for airway protection and hypoxic respiratory failure. Shortly after intubation, the patient suffered a PEA arrest and required several rounds of CPR before return of spontaneous circulation was obtained. He was continued on broad spectrum antibiotics given concern for gram negative septic shock and required multiple vasopressors and stress dose steroids for blood pressure support. Unfortunately, despite maximal support, the patient became progressively more bradycardic, culminating in repeat arrest, asystole, and death. Blood cultures subsequently grew Clostridium perfringens. Interestingly, autopsy demonstrated signs of Clostridium perfringens within the stomach, spleen and liver and bilateral bronchopneumonia. It was felt his prior EGD and gastric polyp removal led to translocation of clostridium and eventually overwhelming sepsis.  
| **Conclusion:** This case illustrates the importance of considering clostridial infections in patients that recently had instrumentation of their gastrointestinal tracks. Although rare, the high morbidity and mortality associated with clostridium infections warrants consideration and prompt antibiotic use if clinical suspicion is high.  

| **Robert Fell**  
| **Dr. Armin Rashidi**  
| **Dr. Michael Rhodes**  
| **A Rare Cause of Massive Splenomegaly**  
| **Case Presentation:** A 33 year-old Ethiopian man, and a Jehovah’s Witness, with no significant medical history presented with 1-2 months of left upper abdominal pain, drenching night sweats, early satiety, and >20-lb weight loss. His social, family, and drug histories were unremarkable. Physical examination noted tachycardia and massive tender splenomegaly. Initial work-up showed WBC 9x10⁹/L (normal differentials), Hb 13.8 g/dL, and platelets 101x10⁹/L. INR was 1.34 and PTT was 42. Liver panel was normal. LDH was elevated at 342 U/L. HIV and viral hepatitis serologies showed no active infection. A peripheral blood smear showed atypical lymphocytes. CT scan noted marked splenomegaly (25.7 cm), normal appearing liver, and no lymphadenopathy. Comprehensive infectious and autoimmune work-up’s were negative. Peripheral
blood flow cytometry showed no aberrant immunophenotype on B or T cells, and a bone marrow aspiration/biopsy was unremarkable.

A liver biopsy showed prominent sinusoidal infiltrate of atypical lymphoid cells. Staining was positive for CD2, CD3, CD7, and TIA-1, and negative for CD5, CD8, CD20, and CD56. Findings were consistent with hepatosplenic T-cell lymphoma (HSTCL). Planning chemotherapy was complicated by the patient’s decision against receiving blood products (if needed), but he agreed with thrombopoietin (TPO) receptor agonists for thrombocytopenia and erythropoiesis stimulating agents (ESAs) for anemia if required. He subsequently underwent six uneventful cycles of CHOEP (cyclophosphamide, doxorubicin, vincristine, etoposide, prednisone) and achieved a complete remission. No TPO receptor agonists or ESAs were required. He then underwent an autologous hematopoietic cell transplantation (HCT) using BEC (busulfan, etoposide, cyclophosphamide) conditioning at a center with significant experience in “bloodless” transplantation. Nine months after transplant, the patient relapsed and was entered into a clinical trial to receive nivolumab.

Discussion: Hepatosplenic T-cell lymphoma is a rare, aggressive peripheral T-cell lymphoma. HSTCL manifests as hepatosplenomegaly with cytopenias, systemic symptoms, and typically no lymphadenopathy. It affects males more than females (9:1). Median age of onset is 20 years old. Twenty percent of cases occur in the setting of immunosuppression. Median survival is less than two years. There is no standard therapy. Many patients respond to initial chemotherapy, but relapses are common.

Standard supportive care during autologous HCT (i.e., high-dose chemotherapy with autologous stem cell rescue) is the use of blood products while awaiting engraftment. Jehovah’s Witnesses do not accept major blood products. The great majority of medical centers deny stem cell transplantations in patients who refuse blood products due to high risks of bleeding and profound anemia.

Management of hematologic malignancies with chemotherapy and autologous HCT using alternative strategies for hematopoietic support has been described for patients who refuse blood products. These strategies include minimizing iatrogenic blood loss, priming red cell mass with ESAs and iron, and attentiveness to hemostasis including preventive measures and aggressive management of active bleeding.

Retrieving the anchor on a hidden autoimmunity

Case: A 40-year-old African American female presented with two weeks of hemoptysis and arthralgias. Physical examination was remarkable for tachypnea, coarse breath sounds, violaceous lesions overlying proximal interphalangeal joints, well-demarcated blue discoloration of several distal toes bilaterally and distal left 4th finger. Initial labs showed leukocytosis to 17.1 k/uL, sodium 126 mmol/L, potassium 5.8 mmol/L, creatinine 1.52 mg/dL, ALT 77 U/L, AST 162 U/L. She was started on antibiotics and normal saline but rapidly deteriorated requiring intubation. Computed tomography (CT) pulmonary angiography showed bilateral pulmonary emboli (PE) and extensive airspace consolidation consistent with acute respiratory distress syndrome. Bronchoscopy was unremarkable. Magnetic resonance imaging of the brain showed small acute infarcts. There was concern for anti-phospholipid antibody syndrome (APS) and Rheumatology was consulted. Further labs returned showing C3 of 58 mg/dL, C4 of 12.2 mg/dL, lupus anti-coagulant positive, beta 2 glycoprotein IgG positive, anti-cardiolipin negative, lactate dehydrogenase 845 U/L, haptoglobin 192 mg/dL. Biopsy of her left 4th finger showed vascular thrombi without evidence of vessel wall inflammation. Her PEs, cerebral infarcts, liver and kidney injury, anti-phospholipid antibodies, and biopsy were
consistent with catastrophic anti-phospholipid antibody syndrome.

She was treated with anti-coagulation, plasmapheresis, steroids and discharged home on warfarin with new requirement of supplemental oxygen. Following discharge, the patient presented three times to the emergency department with significant dyspnea and hypoxia with negative infectious work-up. On the third presentation, she was admitted with diffuse arthralgias and dyspnea with tachypnea, 102° fever, and an oxygen saturation of 94% on Oxymask. She had coarse breath sounds, unchanged skin findings and leukocytosis to 18.2. A repeat chest CT revealed extensive bilateral infiltrates, more apparent in lower lobes without definite consolidation. Without infectious etiology or improvement of symptoms after ten days, rheumatology was re-engaged. The chronic relapsing nature of her symptoms was thought not characteristic of APS and the differential was broadened to include collagen vascular disease causes of interstitial lung damage. Lung biopsy revealed non-specific interstitial pneumonia without granulomas or vasculitis. Myositis panel was positive for Anti-Jo-1 antibody. A concomitant diagnosis of pulmonary predominant anti-synthetase syndrome (ASS) was made with APS.

Conclusion: Anchor bias played a significant role in delay and work-up of our patient’s co-morbid ASS as her symptoms were attributed to previously diagnosed APS. Broadening the differential, in this case, was vital. Interestingly, a single case of APS-ASS overlap syndrome exists in the literature.1 Had we continued to attribute her symptoms to APS, she would likely have had progressive and irreversible damaged her remaining lung tissue.

Max Fuller

A Case of Pulmonary Edema and Chorea: Was it the Methadone?

Introduction: Pulmonary edema is a common problem encountered in the inpatient setting. Most commonly, it is due to cardiac causes with non-cardiogenic causes occurring much less often. This is a case of non-cardiogenic pulmonary edema also presenting with choreiform movements thought to be secondary to prescribed methadone use.

Case Presentation: A 31 year old woman with a past medical history of opioid use disorder and pulmonic stenosis repair as teenager presented with 2-3 days of worsening dyspnea. Patient recently incarcerated, released one month before encounter. Denies any recent drug use but was enrolled in methadone clinic 10 days prior to admission. Currently on 40 mg of Methadone without any take home doses.

Exam remarkable for new onset choreiform movements of right upper extremity, bilateral crackles on lung exam, III/VI systolic murmur, no fever.

CXR and chest CT concerning for pulmonary edema. TTE demonstrated normal ejection fraction with no acute valvular abnormalities.

Blood cultures negative, testing for Rheumatic Fever negative and negative autoimmune workup. Urine drug screen with mass spectroscopy only revealed Methadone and Methadone metabolites. Continued to have intermittent hypoxia during hospitalization. Patient did not display infectious symptoms but due to hypoxia and CT findings was treated for possible atypical pneumonia with Ceftriaxone and Azithromycin.

Continued to diurese patient and decreased Methadone dose to 10 mg. Hypoxia and choreiform movements gradually improved.

Discussion: Pulmonary edema is most commonly seen due to cardiogenic causes. However, non-cardiogenic edema has been well described from methadone use among many other causes.
In evaluating non-cardiogenic edema, providers must consider atypical infection, alveolar hemorrhage, medication toxicity, and other causes. Especially in light of opioid crisis, providers must be aware of potential side effects of methadone maintenance therapy given its likely increased use in coming years.

**Tsege Gebreslasse**

*Atypical Radiological Presentation of Transfusion Associated Circulatory Overload (TACO)*

Introduction: Transfusion-associated circulatory overload (TACO) is defined as acute onset or worsening respiratory distress during or up to 12 hours after transfusion. Chest radiologic examinations usually show bilateral pulmonary edema. Risk factors for TACO include cardiac and renal abnormalities. Recognizing transfusion risk factors and understanding TACO could exist even when classic radiological findings are lacking is important and can prevent delay in diagnosis and treatment. This case illustrates a 91 year old male who developed acute hypoxic respiratory failure after receiving FFP.

Case Description: The patient is a 91-year-old male with significant PMH of mechanical MVR, chronic diastolic heart failure and Chronic kidney disease stage III, transferred from Outside Hospital (OSH) for acute dyspnea attributed to heart failure exacerbation. The patient endorsed acute dyspnea that woke him up in the middle of the night with indigestion like epigastric pain. On arrival to our hospital, his dyspnea was improved, vital signs were stable. The patient was saturating well on room air. His physical exam was negative for crackles on lung auscultation, no JVD and no lower extremity edema. His labs were remarkable for troponin of 1.5, INR of 2.6 and BNP of 5,000. Repeat labs however showed rising troponin. ECG showed evolving ST elevation and new left buddele branch. He was diagnosed with ST elevation myocardial infarction (STEMI). Prior to percutaneous coronary intervention for the STEMI, patient received 2 units of FFP to reverse elevated INR. After the patient returned from PCI, he acutely became tachypnea with respiratory rate of 28, tachycardic with heart rate of 120’s, hypertensive with systolic blood pressure of 170s and hypoxic with oxygen saturation in the low 90s. He had diffuse bilateral crackles on lung exam. Laboratory findings were most significant for a BNP of 25,000. Chest x-ray showed acutely evolving bilateral patchy infiltrate (right greater than left). The patient was placed on BIPAP and received IV bumex with clinical improvement.

Discussion: According to the International Society of Blood Transfusion, TACO is defined as acute onset or worsening respiratory distress during or up to 12 hours after transfusion; and 2) two or more of the following:

A. Evidence of acute or worsening pulmonary edema  
B. Evidence for unanticipated cardiovascular system changes including development of tachycardia, hypertension  
C. Evidence of fluid overload  
D. Elevation in natriuretic peptide levels (greater than 1.5 times the pre-transfusion value)

Although our patient met most of the diagnostic criteria, the CXR finding (right greater than left lung opacity) was unusual. Atypical CXR finding then leads to broad differential which can delay the actual diagnosis. Thus it is important to recognizing the diagnosis of TACO, even in the events that classic radiological findings do not occur.

**Christine Gruessner**

*A Case of Hyalinizing Clear-Cell Carcinoma: a Rare, Indolent Tumor with Aggressive Mimics*
### Case

This is the case of a 24-year-old pregnant female who presented with a nodule on the right soft palate of the mouth.

Clinical impression was of a hemangioma. Underwent resection of the lesion. Histology and immunohistochemistry was nonspecific (sheets of basaloid clear cells in a background of mucin, with no ducts or glands, no chondroid, no basal like membrane material). FISH showed EWSR1 rearrangement in 90% of nuclei. This is consistent with a diagnosis of hyalinizing clear-cell carcinoma (HCCC), and helped to distinguish it from aggressive mimics such as mucoepidermoid carcinoma, myoepithelial carcinoma of the salivary gland, epithelial-myoepithelial carcinoma, and soft tissue myoepithelial tumor. HCCC is an indolent tumor which is cured with complete excision, as is the case in this patient. EWSR1 rearrangement is a consistent finding in this rare, low-grade salivary gland tumor.

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### David Harmon

**Near-Fatal, Slow Onset Asthma Exacerbation Secondary to an Abrupt Environmental Change**

**Introduction:** Status asthmaticus is a feared complication of asthma and may have a rapid onset over 2-6 hours, or slow onset with up to 3 weeks of progressive symptoms. Here we describe a unique case of near-fatal, slow onset asthma.

**Case:** A 28 year old man traveling from Saudi Arabia with a history of asthma presented to an outside hospital in respiratory distress. He arrived in Rochester one month prior to support an ill friend. The patient’s initial presentation of severe respiratory distress prevented his ability to provide history. His friend reported increased rescue inhaler usage throughout the month with abrupt worsening a few days prior to his ER presentation. The patient had a history of worsened asthma with previous visits to Rochester, and he is a current smoker.

In the ER, he worsened despite bronchodilator and corticosteroid therapy and was intubated, sedated, and paralyzed prior to transferring to Mayo Clinic, Saint Mary’s Hospital for management of status asthmaticus. Arterial blood gas on arrival revealed significant respiratory acidosis with pH 6.91 and pCO2 >100 mmHg. Chest x-ray demonstrated clear lung fields. Our initial ventilatory strategy utilized an increased expiratory time, reduced respiratory rate, and high inspiratory flow, with a plan to tolerate hypercapnia and high peak pressures. Management included continuous albuterol and ipratropium nebulization, intravenous methylprednisolone, intravenous magnesium, and heliox therapy in addition to atracurium, propofol, and ketamine, the latter of which was selected for its bronchodilatory effects. The patient experienced persistent bronchospasm and worsening hypercapnia the following day, and intravenous aminophylline infusion was started with minimal improvement. The patient was subsequently initiated on inhaled isoflurane with further consideration of extracorporeal membrane oxygenation (ECMO) if no improvement occurred. He responded favorably to isoflurane administered in the ICU with improvement in peak pressures, intrinsic PEEP, and hypercapnia. He was able to be weaned off sedation and was extubated on hospital day 7. He was discharged on hospital day 11 with an asthma action plan in place.

**Discussion:** Our patient encountered multiple potential asthma triggers during his time in Rochester: allergen exposure amidst increased pollen index, inhaled irritant from continued smoking, and emotional stress with his friend’s illness. According to the Global Initiative for Asthma, our patient carried multiple risk factors for asthma-related death including, lack of an asthma action plan, no use of inhaled corticosteroids, and potential overuse of short acting beta-agonist, though this is unclear from the history. An asthma action plan including symptom tracking, peak flow measurements, and instruction of appropriate response, such as seeking medical attention, would have benefited this patient. Smoking cessation would have also been beneficial as it has been associated.
with increased asthma-related hospitalization as well as diminished responsiveness to inhaled and systemic steroids.

### Hiba Hashmi

**Periodic Paralysis - A Graves' Omen**

Introduction: Thyrotoxic hypokalemic periodic paralysis (THPP) is a rare condition which mainly presents in Asian males. Hypokalemia, hyperthyroidism and paralysis of the lower extremities is the characteristic triad of THPP which can be triggered by hyperinsulinemic states. The sudden onset of paralysis however is usually attributed to a neuromuscular or rheumatic cause rather than an endocrine one, particularly in non-characteristic populations. We report a case where acute onset paralysis heralded the diagnosis of Graves' disease and explore management strategies including judicious potassium replacement and optimal treatment of the thyrotoxic state.

Case Description: A 38-year-old Hispanic gentleman presented to the Emergency Department with profound weakness of his lower extremities, associated bilateral hip pain and a waxing and waning malar rash for 3 months. Lab workup was unrevealing with K+ 4.3 and negative ANA. He had presented similarly twice before with an unrevealing workup. A rheumatology referral was made given concern for SLE. Four days later, he presented with a pruritic facial rash and lower lip swelling which improved with steroids. The following day he presented with acute onset progressive paralysis of his upper and lower extremities bilaterally, with greater severity in the lower extremities. CT head was normal. Chemistry panel revealed critically low potassium of 1.5., with prominent U waves on EKG. He was given 10 mEq potassium through a peripheral line, 2mg mag sulphate, 40 mEq PO potassium, and 20 mEq KCl through a central line. He regained full strength of his extremities following potassium replacement to 4.2. TSH was undetectable at <0.1, Free T4 (4.5), Free T3 (10.6), Total T3 (277) and TSI (370) were all elevated. Propranolol 40 mg BID and Methimazole 20 mg BID were initiated. Thyroid scan revealed elevated radioiodine uptake, and diffuse thyromegaly supporting the diagnosis of Graves’ disease. He was treated with I131 treatment. T4 continued to be elevated (2.8) following treatment, therefore methimazole was reinitiated. However, he subsequently gained 20 lbs with associated periorbital puffiness and sluggishness indicative of hypothyroidism (T4 0.1 and TSH 25.9). Methimazole was stopped and levothyroxine continued with achievement of a euthyroid state.

Conclusion: This case illustrates that paralysis can herald Graves’ disease in the absence of textbook features such as exophthalmos or goitre. Furthermore, it can present with a malar rash, mimicking Systemic Lupus Erythematosus. Therefore, a broad differential including endocrine, rheumatic and neuromuscular aetiologies should be considered and a thorough workup including T4 and TSH performed early on. Fatal hyperkalemia from overzealous correction is a grave consequence, yet no clear guidelines exist on replacement. Treatment with propranolol which relieves the thyrotoxic state and prevents the adrenergic intracellular shift of potassium as well as definitive therapy with radioiodine ablation can be expedited with early recognition of THPP.

### Kaitlin Hellie

**A Case of Unintentional Toxic Aspirin Ingestion**

Objective: Recognize the signs and symptoms of acute salicylate toxic ingestion; Understand the pathophysiology behind the classic acid-base shifts that occur in acute salicylate toxicity; Identify the clinical markers for initiating hemodialysis in cases of salicylate toxicity

Case: The patient was a 43 year old male with history of lower back pain status post lumbar decompression three months prior to presentation, which had been
complicated by discitis, who presented to the Emergency Department with altered mental status, chest pain, hallucinations, vomiting, and diarrhea. The patient admitted he had been taking large volumes of aspirin (8-12 325mg tabs for 3 days prior to presentation) and ibuprofen to treat acute lower back pain. In addition, he was a frequent user of wintergreen-flavored chewing tobacco.

On presentation, he was tachycardic to the 140’s, normotensive, and tachypnic with respiratory rate in the 40s. His presenting labs were notable for white blood cell count of 25, potassium of 3.3, bicarbonate of 12 with anion gap of 20, creatinine of 1.46, negative troponin, ETOH <0.010 g/dL, but with an aspirin level of 92.4 mg/dL and <5.0 ug/mL tylenol. Venous blood gas showed a primary respiratory alkalosis with metabolic acidosis (pH 7.48, CO2 20). His lactate was normal at 1.4. Poison control was contacted and the patient was started on sodium bicarbonate infusion and transferred to a higher level of care.

On admission to the ICU, the patient was emergently initiated on hemodialysis. The patient was also intubated due to impending respiratory failure. Metabolic abnormalities and ASA level normalized after hemodialysis and the patient was discharged three days after admission.

Discussion: Aspirin is one of the most commonly used pain medications in the world. Toxic ingestions accounted for nearly 25,000 visits to United States emergency departments in 2014. The classic presentation of salicylate toxicity includes a primary respiratory alkalosis, due to activation of the respiratory center in the medulla in the brain, followed by development of a metabolic acidosis, a result of interference in the citric acid cycle. Acute management of salicylate poisoning is complex due to the multiple acid-base derangements. In general, intubation is not recommended as the risks of hypoventilating the patient or causing a transient respiratory acidosis during intubation both have high risk for increasing mortality. Patients should also receive high volume of sodium bicarbonate containing intravenous fluids with intent of alkalinizing the urine to facilitate excretion. Hemodialysis is recommended for patients exhibiting altered mental status, patients with acute or chronic kidney disease that would impair excretion, salicylate levels >90mg/dL for patients with normal renal function, levels >80mg/dL in patients with impaired function, and severe acidemia with pH <7.20.

Jose Henao

**Disseminated Histoplasmosis Presenting as the First Opportunistic Infection in a Newly Diagnosed AIDS Patient**

Introduction: Histoplasma is endemic in North, South America and Asia. Incidence is under-reported but is one of the most common causes of AIDS-Related deaths by fungal infection. After inhalation the yeast form infects alveolar macrophages; in the immunocompetent host, the infection is contained with formation of granulomas. In immunocompromised hosts the fungus can spread through the blood or lymph leading to disseminated histoplasmosis. Immunocompromised individuals may develop disseminated histoplasmosis with multiple organ involvements.

Case Presentation and Discussion: Clinical presentation is heterogeneous: fever, asthenia, gastrointestinal, neurological and skin manifestations may be present; in untreated patients mortality reaches 39%. This clinical vignette presents a male with AIDS coming in with disseminated Histoplasmosis. Patient is a 35 yo Mexican male with history of MSM and newly diagnosed AIDS who presented to the ED with sub-acute cough and a purpuric rash. He was recently diagnosed with HIV infection (due to unintentional weight loss) and was seen in ID clinic 1 week prior this admission. Labs revealed CD4 count 13, viral load 2,460,000. He was given Genvoya and PCP prophylaxis but the patient did not start these. In the ED he complained of 50 pounds unintended weight loss and extreme fatigue; also 3 months of nonproductive cough and a rash for which he
completed a course of doxycycline without improvement. He was found to be febrile, hypoxic and tachycardic. He denied headache, neck stiffness, chest pain, abdominal pain, diarrhea or urinary symptoms. Work up showed pancytopenia, transaminitis, abnormal CT C/A/P with miliary nodules and splenic lesions. Differential remained broad but concerning for disseminated fungal infection, mycobacterial or bacterial infection. Blood, skin and sputum cultures obtained and grew Histoplasma capsulatum. Mycobacterial infection ruled out with negative quantiferon, sputum and TB PCR; PCP negative, RPR and Cryptococcus serum antigen also negative. Urine and blood histo antigens positive and blasto antibody negative. CSF histoplasma antigen negative. CMV serum PCR positive. Ophthalmology consulted and CMV retinitis ruled out with dilated eye exam. Patient completed 2 weeks of Amphotericin therapy with resolution of fevers pancytopenia, hypoxemia and improvement of his rash, discharged on oral itraconazole with plan to follow up in ID clinic in 2 weeks. Examination of histological or cytological specimens with H&E, PAS and MGS may lead to identification of Histoplasma spp. Serologic tests include immunodiffusion, antigen detection and identification from culture which is the gold standard for diagnosis. Treatment includes intravenous liposomal amphotericin B for at least two weeks, followed by itraconazole.

Conclusion: ART should be started as soon as possible monitoring for risk of drug interactions or IRIS. This case illustrates the importance of having diagnostic suspicion and knowledge of this pathology as mortality can be very high, but with prompt treatment outcomes can be very positive.

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<tr>
<th>Toy Hess</th>
<th>Venous Stenosis, Lymphedema, Sacroiliitis and Hidradenitis Suppurativa in a 31 Year-Old Somali Female: Can it be SAPHO Syndrome?</th>
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<td>Dr. Kayla Lundeen</td>
<td>Introduction: SAPHO syndrome is a rare autoimmune disorder diagnosed clinically by a constellation of symptoms including Synovitis, Acne, Pustulosis, Hyperostosis and Osteitis. This syndrome has been described as most prevalent in Caucasians between the ages of 30 and 50. Venous thromboembolism is listed as a rare complication of SAPHO syndrome, most commonly in the subclavian veins, described in about 25 case reports.</td>
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<td>Case Presentation: This is a case of a 31 year old Somali female with a past medical history of morbid obesity, left sided sacroiliitis and right iliac vein stenosis post angioplasty with chronic right lower extremity lymphedema who was admitted to the hospital with a one month history of progressively worsening right sided low back pain and groin rash. On physical exam the patient was tender over the right SI joint with a positive Faber’s test. The right inguinal area revealed multiple tender, fluctuant subcutaneous nodules draining purulent fluid.</td>
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<td>Methods: Lab workup revealed elevated inflammatory markers, positive beta glycoprotein IgM antibody at 111 (greater than 40) and positive ANA at 1:80 titer with speckled pattern. MRI revealed new right sided sacroiliitis with findings of right SI joint synovitis and osteitis of the right sacral bone. Dermatology was consulted and confirmed the rash diagnosis of hidradenitis suppurativa. Given the sacroiliitis and hidradenitis suppurativa, the patient was given a unifying diagnosis of SAPHO syndrome.</td>
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| | Discussion: The significant right lower extremity lymphedema and previously described right iliac vein stenosis in this young woman was intriguing and puzzling. Further review of previous CT abdomen with contrast and angiography of the right lower extremity with radiology revealed that the iliac vein stenosis was more extensive than initially thought and is consistent with chronic venous thromboembolism burden. The chronic venous thromboembolism is likely secondary to local inflammation due to SAPHO syndrome. However, the positive beta-2 IgM glycoprotein antibody, iliac
venous thrombosis and history of one previous stillbirth also raises the possibility of a concurrent antiphospholipid antibody syndrome.

Conclusion: This case demonstrates the importance of considering autoimmune etiology when a young person, of any ethnicity, presents with sacroiliitis and pustular skin findings. An accurate diagnosis of SAPHO syndrome and prompt, appropriate treatment will aid in both symptomatic relief and prevention of further joint destruction. Additionally, the case highlights the importance of re-evaluating a patient’s medical history when a new diagnosis arises to make sure the pieces all fit together. The patient in this case had carried the diagnosis of iliac vein stenosis for two years with sequelae of significant lymphedema which persisted despite angioplasty. Determination of the etiology of the stenosis being from chronic VTE, likely secondary to inflammation related to SAPHO syndrome or due to an overlapping phospholipid antibody syndrome, allowed for appropriate treatment with long-term anticoagulation.

Ivana Ho
Dr. Robert Myers

Recognizing Kaposi Sarcoma Inflammatory Cytokine Syndrome

Introduction: Rapid start programs for HIV treatment are starting to be instituted across the country. One of the major risks is not recognizing opportunistic infections at diagnosis, resulting in immune reconstitution inflammatory syndrome (IRIS).

Case: Patient is a 26 year old man, who was recently diagnosed AIDS and syphilis when he presented to an outside facility for fever and rash. He was treated with penicillin for syphilis and started on Bictegravir-Emtricitabine-Tenofovir alafenamide (Biktarvy) through the Rapid Start program. He then presented one month later with fevers to 39.2, chills, mild cough, and shortness of breath, and was admitted to medicine service. He was also found to have disseminated Kaposi’s sarcoma on his skin and in his lungs when bronchoscopy was performed. Patient continues to be febrile and mildly tachycardic, so extensive infectious work up was done and was negative except for Human Herpes Virus-8 (HHV-8) Viral Load of 9,040 copies and skin biopsy consistent with Kaposi Sarcoma. He also had CT chest, abdomen, and pelvis showing diffuse lymphadenopathy and splenomegaly. There was concern whether this was purely disseminated Kaposi Sarcoma vs Multicentric Castleman Disease (MCD) vs Lymphoma. Interleukin 6 was elevated. Left axillary lymph node was biopsied and was consistent with Kaposi Sarcoma without features of the other two. Patient was then treated with Pegylated liposomal doxorubicin (Doxil). Biktarvy was then re-initiated three days after with subsequent resolution of fever.

He was readmitted two months later with similar symptoms toward the end of his chemotherapy cycle, and again underwent extensive work up, which was negative for infection. His cutaneous Kaposi Sarcoma had improved significantly at that time, and fever again resolved with continuation of chemotherapy.

Discussion: It is unclear which rash this patient presented with when he was initially diagnosed with HIV and Syphilis, but patient was enrolled into a Rapid Start Program, which can take place in different settings, including Emergency Rooms vs Urgent Care vs Primary Care Centers. Although these programs have shown to decrease time to HIV viral load suppression and improvement in CD4 counts, it is essential that the providers involved in these programs become well versed in recognizing opportunistic infections and delay anti-retroviral therapy for patients with high risk for IRIS.

Conclusion: Kaposi Sarcoma Inflammatory Cytokine Syndrome is a rare and recently recognized entity with a high mortality rate. It can present along the full spectrum of systemic inflammatory response syndrome, sometimes requiring Intensive Care Unit admissions for hypotension and respiratory...
failure. It is therefore important to recognize this syndrome and treat early with chemotherapy. It is also important to recognize other clinical manifestations of HHV-8, as MCD and Lymphoma, which can co-exist with Kaposi, as the chemotherapy regimen would differ significantly.

Katherine Holten  
**Autoimmune toxicity related to the use of Pembrolizumab**

Introduction: Increased use of monoclonal antibodies for targeted cancer treatment has been one of the fastest growing fields in oncology. Pembrolizumab (Keytruda), available since 2014, is an Anti-PD-1 monoclonal antibody that works as an immune checkpoint inhibitor, allowing the body’s immune system to identify and destruct cancer cells that express PD-L1. It is currently FDA approved to treat several cancers, including metastatic non-small cell lung cancer, metastatic melanoma, recurrent or metastatic cervical, gastric, head and neck cancers, and relapsed or refractory Hodgkin’s lymphoma. As immunotherapy gains increased utilization for treatment, it is important to be aware of the potential autoimmune toxicity related to its mechanism of action.

Case: In this case, an 84-year-old gentleman with metastatic melanoma was treated with pembrolizumab and the side effects are highlighted. The patient was initially diagnosed with melanoma on the posterior right shoulder in 2010, which was resected with negative margins. In September 2015, the patient developed a persistent cough, with chest CT demonstrating two large pleural-based masses in the right upper and lower lobes. Therapeutic wedge resections were performed, with pathology confirming BRAF negative metastatic melanoma. Routine follow up imaging in May 2017 showed recurrent disease in the mediastinum and left retroperitoneum. He was started on Pembrolizumab every 3 weeks in June 2017, with favorable radiographic response. The patient was an active octogenarian, playing tennis and traveling to India between his infusions. In October 2017, he was diagnosed with hypothyroidism (TSH found to be 104, after being normal just 6 months prior); he was started on levothyroxine. In November 2017, after his sixth pembrolizumab infusion, he developed severe neuropathic pain. Extensive work-up for etiology was negative, but the patient’s oncologist elected to stop pembrolizumab at that time. He then presented to the hospital in January 2018 for progressive generalized weakness and falls. MRI of the cervical, thoracic and lumbar spine showed multiple spinal cord lesions consistent with demyelinating disease. CSF studies were unrevealing and it was determined that he had multifocal transverse myelitis secondary to an autoimmune side effect of pembrolizumab. His quadripareisis progressed, requiring high dose solumedrol and plasmapheresis. He did not respond significantly, with resulting paraparesis of the lower extremities.

Conclusion: Immunotherapy has shown great promise in the field of cancer treatment. Notably, pembrolizumab has been shown to reduce the risk of disease progression by 50% in patients with advanced NSCLC who were negative for EGFR or ALK gene mutations and had >50% PD-L1 expression. While there has been immense benefit from its use, several immune mediated side effects have been documented: pneumonitis, colitis, hepatitis, hypophysitis, thyroid disorders, and neurotoxicity. With increased use of cancer immunotherapy, it will be important for physicians to recognize potential side effects that our patients may experience.

Marie Hu  
**An Important Cause of Worsening Heart Failure and Myopathy**

Introduction: Acute thyrotoxicosis can present with life-threatening hemodynamic instability and is a medical emergency. The differential diagnosis is broad and includes causes such as Graves’ disease, toxic adenoma, thyroiditis, exogenous, and iodine-induced. Among iodine-induced cases, the antiarrhythmic amiodarone is the most notorious etiology, for which clinicians
Case: A 79-year-old male presented with 2 months of shortness of breath, generalized weakness, falls, diarrhea, palpitations, and 30-lb unintentional weight loss. His past medical history was significant for ischemic cardiomyopathy (EF 42%), ventricular tachycardia s/p ablation (previously on amiodarone, now on sotalol), CAD, atrial fibrillation on warfarin, T2DM, and COPD. He was evaluated by both Cardiology and Neurology in the outpatient setting; work-up included TTE demonstrating worsened EF to 33% with lateral regional wall motion abnormalities and EMG showing a proximal myopathy and length-dependent large fiber peripheral neuropathy. However no clear etiology was identified for these findings. He was then referred back to his PCP, who rechecked thyroid tests (previously normal 2 months ago); these returned markedly abnormal with TSH <0.01 and free T4 >7.8. He was then admitted to inpatient medicine for further management. Endocrinology was consulted, and additional labwork included total T3 319 (nl 80-200), free T3 9.1 (nl 2.8-4.4), negative thyrotropin receptor Ab, and urine iodine/Cr ratio 4686 (nl 70-1150). Thyroid ultrasound showed a diffusely enlarged thyroid with no suspicious nodules, and NM uptake scan showed mild homogenous radiotracer uptake with no ectopic foci and reduced uptake at 4 hours. Given that the patient had been on amiodarone for his VT up until 5 months ago, it was felt that amiodarone-induced thyrotoxicity was the most likely cause of his acute hyperthyroidism. He was initiated on both methimazole and prednisone for possible Type I and Type II amiodarone-induced thyrotoxicity. Repeat thyroid tests 1 week later were significantly improved, and he was discharged to a SNF for short-term rehabilitation.

Discussion: Amiodarone is notorious for its thyrotoxic effects, with the potential to induce both hypothyroidism and hyperthyroidism because of its iodine moieties. There are two mechanisms for amiodarone-induced thyrotoxicity, with Type I due to the excess iodine providing increased substrate for thyroid hormone synthesis and Type II due to direct toxic effects of amiodarone leading to a destructive thyroiditis. Notably, because amiodarone has a half-life of 100 days, up to 20% of amiodarone-induced hyperthyroidism can occur even after it has been discontinued. Oftentimes, the beta-blocking effects of amiodarone can mask typical adrenergic hyperthyroid symptoms, and individuals with underlying heart disease may simply present with worsening heart failure or other nonspecific symptoms. Thus, clinicians should maintain a high index of suspicion for amiodarone-induced thyrotoxicity, and should monitor thyroid tests frequently both during and after use of amiodarone.

Phyllis Huang

**A Curious Case of Takayasu Arteritis in an Ethiopian Female**

Introduction: Takayasu arteritis has worldwide distribution and has been well described in the medical literature in Asians and Caucasians. In recent decades, Takayasu arteritis has been increasingly recognized in African populations with retrospective studies in South Africa, Tunisia and Morocco showing variation in clinical features and presentation between regional and ethnic differences.

Case: This clinical vignette illustrates the unusual presentation and delayed recognition of this disease in an Ethiopian female. A 19 year old woman with one year history of intermittent headaches was admitted after a witnessed tonic-clonic seizure. She was found to have hypertensive emergency, PRES syndrome, elevated inflammatory markers, leptomeningeal enhancement concerning for CNS infection, positive TB gold quantiferon test. Early course was complicated by worsening neurological exam and elevated intracranial pressure requiring External Ventricular Drain placement. MRI brain showed PRES as well as diffuse leptomeningitis which prompted extensive work up for CNS infection. As patient had positive TB gold quantiferon and all infectious work up was unremarkable, patient was initiated
on dexamethasone and four drug TB treatment. Within 24 hours, patient had normal neurological function and was extubated and transferred out of ICU. Six days after initiating TB treatment with steroids, public health records showed that patient had in fact completed treatment for latent TB in the past and treatment was stopped. On hospital day 15, the work up for secondary hypertension revealed 60-70% stenosis of abdominal aorta with significant aortic wall thickening, 80% stenosis of right renal artery, 50% stenosis of left renal artery, and 70% stenosis of SMA. No involvement of aortic arch and its branches noted.

Meanwhile, patient has been on dexamethasone with dramatic improvement in inflammatory markers. She was transitioned to methylprednisolone pulse for 3 days followed by slow oral prednisone taper. Rheumatologic serologic work up was unremarkable including anti-phospholipid antibodies except for the elevated inflammatory markers. Given the radiologic findings of aortitis with secondary stenosis of the aorta, renal arteries and SMA causing secondary hypertension and PRES, elevated inflammatory markers, response to steroids, ethnicity of the patient and overall clinical picture, the diagnosis of Takayasu arteritis was made. Patient was discharged on day 21. On follow up, patient was asymptomatic and inflammatory markers have resolved.

Conclusion: This case illustrates how diagnostic delays can happen in populations previously thought to have low incidence of vasculitis secondary to paucity of high quality epidemiological data. It is possible that vasculitis is not rare in Africa but have hitherto been under-diagnosed. Additionally, it is now more evident that there are ethnic and regional difference in vascular involvement and clinical presentation of patient with Takayasu arteritis [1,2,3]. For that reason, vasculitis should be considered early in clinical course when diagnosis is undifferentiated and regardless of ethnicity of the patient.

**Steven Hwang**
**Dr. Adam Sawatsky**
**Dr. James Lloyd**

**Minimal Change Disease in the Setting of Certolizumab**

Introduction: Minimal change disease (MCD) accounts for approximately 10 percent of cases of nephrotic syndrome in adults. Most cases of MCD are idiopathic, but some are thought to arise secondary to medication exposure, including exposure to sulfasalazine, certain antimicrobials, and immunosuppressive agents. Tumor Necrosis Factor Alpha (TNF-alpha) inhibitors are effective in treating autoimmune inflammatory diseases, but their side effect profiles have not been fully elucidated. Here, we describe a case of biopsy-proven MCD in a patient with rheumatoid arthritis treated with certolizumab.

Case Presentation: A 49-year-old woman presented with a 1-week history of foamy urine, peripheral edema, and 15-kg weight gain. She had a history of rheumatoid arthritis and Sjogren’s syndrome. She was begun on certolizumab 6 months prior to presentation. Physical exam demonstrated anasarca, including prominent bilateral lower extremity edema and ascites. Laboratory evaluation revealed renal impairment of unclear duration (creatinine 2.4 from baseline 1.0) and severe hypoalbuminemia (1.7). Urinalysis showed markedly elevated protein without prominent hematuria, and confirmatory 24-hour urine collection was consistent with nephrotic syndrome (17.5 g protein collected). Renal ultrasound showed echogenic renal parenchyma bilaterally, consistent with intrarenal disease. Renal biopsy was subsequently performed, and electron microscopy revealed slightly thickened glomerular basement membranes and evidence of diffuse (100%) podocyte foot process effacement consistent with MCD. In view of these findings, the patient’s certolizumab was discontinued, and she was initiated on high-dose prednisone and diuretic therapy, leading to marked improvement in her edema and related symptoms.

Discussion: We describe a case of a 49-year-old woman initiated on
certolizumab for treatment of rheumatoid arthritis and who subsequently developed biopsy-proven MCD approximately 6 months later. A few reports in the literature describe associations between certolizumab and focal segmental glomerulosclerosis, and others suggest an association between other TNF-alpha inhibitors and MCD; however, the case presented here is the first report of a potential certolizumab-induced MCD. Prior reports demonstrated similar timing between TNF-alpha inhibitor initiation and the development of nephrotic syndrome (3-6 months after initiation) as in this case.

As TNF-alpha inhibitors become more commonly used to treat rheumatoid arthritis, we may see more reports of medication-induced MCD. There is biological plausibility, as TNF-alpha blockade has been shown to shift T-helper cell activity towards a Th2 response and the promotion of humoral immunity, and MCD has been associated with elevated levels of various Th2 cytokines. This suggests that TNF-alpha inhibition may have made our patient more susceptible to the development of MCD.

Overall, this case underscores the need to have a high suspicion for idiosyncratic and previously undescribed side effects of novel drugs when unanticipated outcomes occur. In this case, the temporally associated onset of this patient’s nephrotic syndrome with the initiation of certolizumab suggests a medication-induced mechanism.

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**Brenden Ingraham**  
**Dr. Marcia Venegas**

**Mixed Connective Tissue Disease with Mixed Etiology ILD**

**Introduction:** Adalimumab is an uncommon cause of interstitial lung disease (ILD). Patients on adalimumab for connective tissue disease can have a mixed ILD with fibrotic changes in addition to the ground glass opacities (GGOs) resulting from adalimumab.

**Case Description:** 64-year-old female is admitted for workup and management of subacute, progressive dyspnea, pleuritic chest pain, dry cough, and new oxygen requirement. History is significant for plaque psoriasis on adalimumab (initiated 3 months prior to presentation), ulcerative colitis status post proctocolectomy, sicca symptoms, Raynaud’s phenomenon, GERD, maternal history of rheumatoid arthritis, and a 50-pack-year smoking history. CT showed moderate, multifocal GGOs consistent with nonspecific interstitial pneumonia (NSIP) or usual interstitial pneumonia (UIP) without honeycombing. Pulmonary function testing showed severely reduced DLCO. Ejection fraction was 60% without significant echocardiographic abnormalities. Labs remarkable for normal leukocyte count; moderately elevated ESR and CRP; and mildly elevated RNP, LDH, and aldolase. Rheumatologic and infectious testing (including bronchoalveolar lavage) was otherwise negative. Transbronchial biopsy showed bronchiolar wall and parenchyma without neoplasm or granuloma. Video capillaroscopy confirmed secondary Raynaud’s. Adalimumab was stopped, and she was started on prednisone 30 mg daily. She improved clinically and was discharged with home oxygen and plans for outpatient rheumatology and pulmonology follow up. Repeat CT chest one month later showed significant improvement in GGOs but persistence of fibrotic ILD. Pulmonology believed the remaining disease represented UIP versus NSIP related to underlying connective tissue disease. Rheumatology felt that the ILD with elevated RNP in the setting of her other rheumatologic symptoms were suggestive of mixed connective tissue disease (MCTD). Azathioprine was started with plans to wean the prednisone. She has since been improving clinically.

**Discussion:** Tumor necrosis factor inhibitors, like adalimumab, have been implicated in rare cases of interstitial pneumonia with a prevalence of 0.5% to 3%. ILD occurs in > 50% of those with MCTD. Careful consideration should be given to the underlying cause as adalimumab-induced ILD is a diagnosis of...
exclusion after ruling out infectious and cardiac etiologies. It is important to recognize fibrotic lung disease independent of the GGOs from adalimumab, especially when ILD persists despite discontinuation of the offending agent. Removing the adalimumab is often sufficient, but corticosteroids can be utilized in more severe or refractory cases. Fibrotic changes in the setting of connective tissue disease may require prednisone or other steroid-sparing agents, like azathioprine.

Sae Jang  
Dr. Harrison Gerdes  
Dr. Shounak Majumder

**Antiphospholipid Syndrome as a Cause of Severe Gastrointestinal Bleed**

**Introduction:** Antiphospholipid syndrome (APS) is an autoimmune hypercoagulable disorder characterized by thromboembolic events. We present a case of severe acute anemia due to gastrointestinal hemorrhage.

**Case Description:** A 55 year-old woman with past history of APS complicated by multiple deep vein thromboses and miscarriages, prior hemorrhagic stroke, chronic slow GI bleed with iron deficiency anemia, thrombocytopenia, and CKD stage 3 presents to the ED with two-month history of progressively worsening shortness of breath, dizziness, and chest discomfort with exertion, which acutely worsened over the last day. Initial evaluation revealed hemoglobin of 3.9 grams/dl and presence of heme-positive stools. Patient denied abdominal pain, hematochezia, or maroon-colored stools. She endorsed black tarry stools which she attributed to her oral iron supplementation.

She was admitted to the hospital and received 5 units of packed red blood cells with increase of Hgb to 9.7. Anticoagulation was held. EGD showed erosive gastropathy and colonoscopy showed non-bleeding plaque type lesions which were treated with argon plasma coagulation. Capsule endoscopy and Meckel’s scan were unremarkable. When patient was trialed back on anticoagulation, her Hgb dropped to 7.4.

Her hospitalization was further complicated by AKI on CKD with a peak creatinine of 2.4 and nephrotic range 24-hour proteinuria of 4.5 grams without casts. Serologic work up included positive ANA 1:160 in a speckled pattern, rheumatoid factor, and low C3 and C4. Antiphospholipid antibodies were positive. Other vasculitis and rheumatologic laboratories were negative.

Kidney biopsy showed acute renal microangiopathy with severe tubular atrophy and interstitial fibrosis. Severe hypertensive arteriosclerosis and acute tubular injury was also present. Complete repeat endoscopic work-up showed gastric antral vascular ectasia and significant segmental mucosal ulceration suggestive of ischemia. CT chest showed a wedge-shaped area of infarct in the right middle lobe.

Given her history of APS, the microangiopathy seen on renal biopsy, wedge-shaped pulmonary infarct, and ischemic-appearing colonic ulcerations, it was felt that patient’s overall presentation and severe GI bleed was due to ischemic colitis from micro-infarcts and APS. Patient received a course of IV methylprednisolone, plasmapheresis, and rituximab infusion. She was bridged to a therapeutic level of warfarin with stable hemoglobin prior to discharge from the hospital.

**Discussion:** Abdominal manifestations of APS are uncommon but can be life threatening. Reported cases of thromboses include Budd-Chiari syndrome, hepatic-veno-occlusive disease and occlusion of small hepatic veins, or hepatic infarction. Intestinal manifestations are even more infrequent. This patient had catastrophic APS defined by 1) Widespread thrombosis in multiple organ systems, 2) development of manifestations simultaneously within a week, 3) confirmation of small-vessel occlusion on histopathology, and 4) laboratory confirmation of antiphospholipid antibodies. This case highlights that
abdominal involvement should be considered in patients with APS.

Laurens Janssens
Dr. Sunanda Kane

The patient who could not eat at AL

Case: A 77 year old non-diabetic man from Mississippi with history of prostate cancer (status post resection and local irradiation) and locally excised melanoma presented with a one year history of intermittent abdominal distention, nausea, bloating and reduced appetite. He endorsed minimal pain and denied any vomiting during these episodes, but reported an approximate 15 lb (6.8 kg) weight loss over the last year.

Outside work-up consisted of multiple imaging studies demonstrating marked gastric distention as well as delayed gastric emptying (96% and 93% retention at 2 and 6 hours, respectively). Outside colonoscopy was negative and EGD was negative except for a distal esophageal ulcer and focal active duodenitis. He was started on a PPI and trialed on an alternating regimen of metoclopramide and erythromycin, without symptomatic relief. Multiple trials of antibiotics for potential bacterial overgrowth did not resolve symptoms. In addition, he had been complaining of increasing shortness of breath on exertion and lower extremity edema.

He was referred to our clinic for further diagnostic work-up. Unfortunately, during his stay he had a syncopal event in his hotel room and the subsequent ED visit revealed a new L1 fracture that required hospitalization. Colonoscopy showed congested distal ileal mucosa and friable ascending colonic mucosa. Biopsies from both sites revealed amyloid deposits by Congo red stain. Simultaneous serum protein electrophoresis noted a monoclonal IgG lambda peak with M-spike of 1.5 g/dL and bone marrow biopsy revealed approximately 70% of marrow cellularity consisting of plasma cells. Echocardiogram confirmed severely increased concentric left ventricular wall thickening with mildly decreased voltage in the limb leads on ECG, consistent with cardiac amyloidosis. Kidney and liver function were normal without morphologic abnormality on CT scan. Interestingly, neither fat pad biopsy nor bone marrow biopsy showed presence of amyloid. The patient was diagnosed with Myeloma and AL Amyloidosis with gastrointestinal and cardiac involvement.

Conclusion: This case highlights the broad differential for gastric distention and dysmotility. When the ‘usual suspects’ such as diabetes, medication, post-surgery and neurological disease have been ruled out, rarer causes like an infiltrative process (scleroderma, amyloidosis) and autoimmune gastrointestinal dysmotility should be considered, especially when there is evidence of multi-organ involvement. Common manifestations of gastrointestinal tract amyloidosis include GI bleeding, malabsorption, protein-losing enteropathy and chronic gastro-intestinal dysmotility. The diagnosis of amyloidosis is based on positive tissue diagnosis. While the combination of a fat pad and a bone marrow biopsy has a high sensitivity (89%) in diagnosing AL amyloidosis, negative results do not rule out amyloid elsewhere and tissue sampling from the affected organ site is essential, as demonstrated in this case.

Joseph Jensen

SBE or not SBE: That is MI Question

Case: A 78 year old man with a past medical history notable for HTN, PVD, and GI bleed was admitted with shortness of breath and chest pain. EKG showed acute ST segment elevation in V1 and diffuse ST segment depression in the inferior and lateral leads. Angiogram revealed 95% stenosis in the proximal LAD which was treated with one drug eluting stent.

His post-procedure course was complicated by upper GI bleeding that was stabilized with five hemostatic clips. On hospital day 3, he developed respiratory failure and hemodynamic compromise that required BIPAP and
transfer to the ICU with initiation of broad spectrum antibiotics. Echocardiogram showed normal global systolic function, moderate MR and mild AI. Blood cultures returned positive for streptococcus mitis thought to be due to gut translocation from a single small non-bleeding angioectasia seen on colonoscopy.

On hospital day 11, the patient developed combined septic and cardiogenic shock with repeat echocardiogram revealing severe MR. He was intubated, a left heart catheterization revealed a patent stent, and an intra-aortic balloon pump was placed. TEE revealed mitral and aortic valvular destruction due to endocarditis. Both Cardiology and Infectious Disease surmised that this occult infectious endocarditis likely caused his initial acute MI presentation. CV surgery deemed the patient high risk, and his family elected to transition to comfort care.

Discussion: Cardiac complications in endocarditis, most commonly heart failure and perivalvular abscesses, occur in up to half of patients. However, forms of metastatic infections are important to note as these can occur in 13 to 44 percent of patients, most often prior to clinical presentation. Common sequelae include stroke, paralysis, blindness, ischemia, splenic or renal infarction, PE, and acute MI. Known risk factors for metastasis include left sided vegetation, specific microbiology, large vegetation size, and the presence of antiphospholipid antibodies.

Intuitively, right sided vegetations metastasize to the lungs whereas left sided vegetations can deposit virtually anywhere in the systemic circulation with the cerebral circulation and femoral artery being the two most common sites. Mitral valve lesions are more likely to spread than aortic valve lesions—25 versus 10 percent. The most common pathogens for endocarditis are Staph aureus, Viridans group strep, enterococcus, coagulase-negative staphylococci and Strep bovis, but the most likely pathogens to embolize are S. Aureus and Streptococcus bovis.

Vegetation size is another important risk for embolic spread and for mortality. Vegetation mobility coupled with size >10mm are predictors of new embolic events after controlling for pathogen type. Vegetation size >15mm is also a predictor of one-year mortality. These risks have been shown to decrease after initiation of appropriate antibiotic therapy as only 24% of emboli occur after antimicrobial initiation.

Community Acquired MRSA: An Increasing Common Skin Infection Presenting as a Red Herring

Introduction: Skin and soft tissue infections are among the most common conditions seen by primary care physicians. Community-acquired methicillin-resistant Staphylococcus Aureus (CA-MRSA) have become more common, and can cause infections in healthy young adults. CA-MRSA is diagnosed based on history, risk factors, physical exam findings, and available cultures. Our case describes a patient who presented with a “spider bite” but upon further evaluation she was diagnosed with CA-MRSA.

Case: A 28-year-old previously healthy female presented to primary care clinic with a new rash on her right medial thigh. She presented to a walk-in clinic three days previously and was given cephalaxin for presumed cellulitis. The lesion initially presented as a small bump with clear drainage but had become painful, red, and increasing in size despite use of antibiotics. Risk factors included exposure to similar lesions on the hands of her husband. She was also concerned about a possible spider bite since she sleeps in a basement bedroom. On exam, she appeared well and was afebrile. Serosanguinous drainage was noted on the dressing. The lesion was a 10 cm X 5 cm erythematous plaque
with a 2 cm X 2 cm purple ulcer in the center, and there was 5 cm X 3 cm underlying induration. Bedside ultrasound showed a 2 cm X 2 cm hypoechoic collection with debris, suggestive of an abscess. She underwent incision and drainage and was treated with oral doxycycline for presumed CA-MRSA infection in the setting of prior allergic reactions to trimethoprim-sulfamethoxazole (TMP-SMX). Fluid culture from her husband’s finger abscess grew MRSA.

Discussion: The prevalence of community acquired MRSA infections has been increasing and whether empiric antibiotic therapy needs to be active against MRSA depends on risk factors, such as failure of previous antibiotic treatment, presence of abscess, and close contact with others who have MRSA colonization or infection. CA-MRSA without abscess often responds to appropriate empiric therapy. However, bedside ultrasound can be helpful in diagnosis of abscess in the absence of a classical fluctuant area. Incision and drainage is the standard of care for abscesses. There is empiric and evidence based reasons for both incision and drainage with and without use of antibiotics. Small, uncomplicated, single abscesses in an immunocompetent patient without systemic symptoms, indwelling devices, extremes of age and other complication risk can be drained without antibiotics in an attempt to spare antibiotics or avoid drug allergies. Co-treatment with antibiotics though has been shown to have better outcomes and less recurrence in some studies. First line oral agents for CA-MRSA infections include doxycycline, TMP-SMX, or clindamycin. Household co-infection and transmission is not uncommon reiterating the importance of history, especially in our case.

### A Deceptive Cause of Chest Pain

Case: A 71-year-old female with a history of coronary artery disease and coronary artery bypass graft (CABG) with left internal mammary artery (LIMA) to the left anterior descending artery (LAD), type 2 diabetes and hypertension presented to the emergency department for exertional chest pain that radiated to the left arm and jaw. The patient was noted to be mildly hypertensive, tachypneic, and hypoxic to 88% on room air. On exam, she had a regular rate and rhythm without extra heart sounds and lungs had diffuse crackles. ECG demonstrated inferior and anterolateral T-wave inversions with mild ST-depressions. Serial troponins trended positively from 0.01 μg/L to 0.09 μg/L. She was diagnosed with a non-ST elevation myocardial infarction and loaded with dual antiplatelet therapy, heparin, and diuresed. She underwent cardiac catheterization which demonstrated total occlusion of her prior saphenous vein graft to the obtuse marginal artery and a 90% stenosis of the proximal left subclavian artery which compromised flow through the LIMA graft. These findings supported a diagnosis of coronary-subclavian steal syndrome. Therefore, the patient underwent percutaneous transluminal angioplasty with bare metal stent placement to the left subclavian artery and her anginal symptoms resolved.

Discussion: This case highlights the broad differential diagnosis in patients that present with chest pain who have a history of CABG with an internal mammary artery graft. In addition to the more common obstructive coronary artery disease, in patients with prior CABG, subclavian steal syndrome must be considered as a potential cause of acute coronary syndrome. Subclavian steal syndrome refers to the retrograde blood flow in the vertebral artery or internal mammary artery due to proximal subclavian stenosis causing a reversal in the pressure gradients and therefore reversal of blood flow. In patients with a history of CABG through the internal mammary artery, this syndrome may cause insufficient coronary circulation, and is referred to as coronary-subclavian steal syndrome. Neurological symptoms can also occur due to insufficient blood flow to the posterior circulation via the vertebral artery. Progressive subclavian stenosis most commonly occurs due to atherosclerosis, as seen in this case, but
may also be due to large artery vasculitis or thoracic outlet syndrome. Symptoms are often exertional. Physical exam can demonstrate a difference in brachial systolic blood pressure between the affected arm and normal arm of ≥15mmHg. Carotid, subclavian, or vertebral artery bruits are also suggestive. Diagnosis can be made with Doppler ultrasound, magnetic resonance, computed tomography angiography, or invasive angiography. In cases such as this, where the stenosis is diagnosed after bypass surgery, percutaneous treatment is recommended.

Anthony Kashou  
Dr. Eileen Russell

Assessing Disease Activity of Takayasu Arteritis in the Setting of Acute Thrombosis

Introduction: Takayasu arteritis (TA) is a large-vessel vasculitis characterized by granulomatous inflammation, most often affecting the aortic arch and its primary branches. It typically affects young females in the 2nd and 3rd decades of life. Prevalence of TA is greatest in Asia with about 150 cases reported annually. In the United States and Europe, the incidence is only one to three per one million. The disease course is often indolent with early disease manifesting as nonspecific constitutional symptoms and late disease marked by symptoms related to progressive arterial involvement.

Case: A 38-year-old Caucasian female presented to the emergency department with a cold, pulseless right arm. She was healthy until three years ago when she developed sudden-onset heart failure thought to be secondary to myocarditis. Two years later, she developed claudication symptoms in her lower extremities. Imaging demonstrated numerous large vessel stenoses, and the diagnosis of TA was made. The patient had been on infliximab and prednisone for five months when she developed claudication and paresthesias in her right arm. CT angiogram revealed stenosis of the right subclavian artery without occlusion. Her rheumatologist increased the prednisone to 60 mg daily for presumed active disease.

Four weeks later in the emergency department, repeat CT angiogram revealed near occlusion of the right subclavian artery and a 10 cm embolic occlusion of the brachial artery. Heparin was immediately initiated. ESR and CRP were normal. PET scan showed no FDG avidity within the vasculature. As such, overall TA activity was presumed to be low, and it was decided that it was safe to proceed with revascularization without antecedent increase in immunosuppression. The patient underwent a limb-preserving common carotid artery to axillary artery bypass and brachial artery thromboembolectomy. She was discharged with recommendations to continue infliximab and antiplatelet therapy and taper prednisone with close follow up.

Discussion: While acute phase reactants (eg, ESR, CRP) can be helpful in monitoring disease activity, they are unreliable in differentiating active from inactive vasculitis when used alone. Symptoms and imaging must be used in conjunction, but imaging such as PET may be unreliable in the setting of high-dose steroid use as demonstrated with this patient. Revascularization procedures can be essential for patients with life-threatening arterial stenosis and occlusion; in non-life-threatening situations, surgery should be avoided in active disease to prevent further aggravation of vessel inflammation.

Unfortunately, there remains little evidence on how to effectively monitor activity and manage TA, especially in patients with acute arterial occlusive disease. Improved methods to assess disease activity and future clinical trials of management strategies will improve the care of these complicated patients.

Hayden Kelly  
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When Screening Becomes Anything but Routine

Introduction: Fifteen million colonoscopies are performed each year for colon
cancer screening. The most common serious adverse events are hemorrhage (1%) and perforation (0.1%). An often under-reported, and rare complication, is splenic injury.

Case Description: A 58 year old woman with history of ankylosing spondylitis and cholecystectomy presented to the emergency department with acute onset diffuse abdominal pain, left shoulder pain and lightheadedness. Patient reported having a routine screening colonoscopy 24 hrs prior to presentation. No immediate complications during procedure were noted, other than poor visualization. Physical exam was significant for hypotension (89/58 mmHg), tachycardia (116 beats per minute), diffuse abdominal tenderness and guarding. Laboratory evaluation showed a white blood count of 25,000, hemoglobin of 8.3 g/dl (baseline 13-15g/dl), platelets of 428,000, creatinine 2.62 mg/dl (baseline within normal limit), and venous lactate of 16.3 mmol/L. Urgent abdominal computed tomography (CT) scan showed an abnormal spleen which was reported as concerning for rupture plus hemorrhage - grade V splenic injury. General surgery was immediately consulted and massive transfusion was started (total of 6 units of packed red blood cells, 4 units of fresh frozen plasma, and 2 units of platelets). Hemodynamics improved and patient was taken urgently to the interventional radiology suite for empiric coil embolization to splenic artery. Post-procedural course was uneventful and she was discharged home on post-procedural day 6. In addition, patient received standard post-splenectomy vaccinations.

Discussion: Colorectal cancer is the second leading cause of cancer deaths. The U.S. preventive services task force (USPSTF) recommends colorectal cancer screening with occult blood testing, sigmoidoscopy or colonoscopies starting at age 50 until age 75. Up to 33% of patients report at least one minor, transient GI symptom after colonoscopy. Serious complications are uncommon. Retrospective studies have found overall serious adverse event rate was 2.8 per 1000 procedures. Splenic injury is rare following colonoscopy, with a reported incidence of 0.00005 - 0.017% but a mortality rate of 5%. The presumed mechanism of injury is by direct trauma to the spleen and excessive spleno-colic ligament traction. Because of its rarity and lack of awareness, this life threatening diagnosis has potential to be delayed. Abdominal pain within 24hr is the most reliable indicator and one must keep this diagnosis in the differential. Abdominal CT is the gold-standard to diagnose splenic rupture. Depending upon patient stability and splenic injury grading, treatment options include observation, embolization, or surgery.

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Dr. Muneeb Rehman  
Dr. Absar Tahir  
Dr. Delamo Bekele  
Dr. Eric Matteson

A Cold Case of Digital Ischemia

Case: A 59 year-old male from Alaska with a history of nicotine dependence, coronary artery disease, hypertension, hyperlipidemia and multiple amputations presented for further evaluation of recurrent digital ischemia. His symptoms began a year prior when began experiencing progressive bluish discoloration of the lower extremities associated with pain, swelling, and eventual digital necrosis resulting in subsequent amputation of 3 digits despite normal vascular studies with pathology revealing pauci-inflammatory small vessel occlusion by predominantly pink-purple homogeneous material that was PAS-stain positive. Ten months later, despite smoking cessation, the patient experienced worsening of his symptoms and developed new digital ischemia with left great toe necrosis bilateral lower extremity ulceration, and intractable pain. Physical exam revealed amputated left 4/5th and right 5th toes, black/blue discoloration of the left great toe, and periungual infarcts most prominent in the fingers without Raynaud’s phenomenon. CT imaging and an echocardiogram were unrevealing for an embolic source of peripheral arterial disease and repeat vascular studies were within normal limits. Autoimmune serologies were negative but further workup revealed an elevated ESR (44) and CRP (9), a SPEP with an increased M-spike in the gamma globulin portion, elevated total protein with elevated
kappa free light chains (2.8) and a 10% Type 1 Cryoglobulin component. Bone marrow aspirate and biopsy revealed a CD-20 negative kappa-expressing monoclonal plasma cell population. The patient ultimately underwent apheresis with resolution of cryoglobulinemia and treatment with prednisone. He was subsequently started on Bortezomib for an underlying lymphoproliferative disorder.

Discussion: Cryoglobulinemia is characterized by the precipitation of proteins including immunoglobulins and complement proteins in the blood below 37°C. Type 1 cryoglobulinemic (10-15% of cases) precipitate tends to be of monoclonal immunoglobulin origin (IgG or IgM), related to an underlying lymphoproliferative disorder whereas a polyclonal cryoprecipitate is implicated in mixed cryoglobulinemia and classically associated with an underlying rheumatologic disorder or viral infection such as hepatitis C and HIV. Clinically, type 1 cryoglobulinemia manifests with signs and symptoms suspicious for vascular insufficiency including digital ischemia, skin necrosis, and livedo reticularis. The diagnosis is established by laboratory isolation of cryoprecipitate and measurement of cryocrit with treatment directed toward the underlying disorder to prevent hyperviscosity and further vascular injury to multiple organs. Overall, it is important to recognize that patients with type 1 cryoglobulinemia often have an underlying lymphoproliferative disorder such as a hematologic malignancy or a monoclonal gammopathy of undetermined significance that warrants further investigation and appropriately targeted therapy. Prognosis and complications in patients with type 1 cryoglobulinemia correlate with the underlying hematologic disorder and its treatment course.

Brittany Kimball
Dr. Karim Sadak

Breast Cancer in the Hodgkin Lymphoma Survivor: Practical Considerations in the Care of Adult Survivors of Childhood Cancer

Case: A 44-year-old premenopausal woman with a history of Hodgkin lymphoma (HL) at age 15, treated with mantle radiation and ABVD chemotherapy, presented to a childhood cancer survivor program for routine follow-up care. She had no acute complaints. Past medical history includes hypothyroidism, multiple radiation-related basal cell carcinomas, chronic bronchitis with evidence of pulmonary fibrosis on chest x-ray, and several benign breast biopsies; all sequelae from her prior therapies. Family history is remarkable for maternal grandmother and two maternal aunts with breast cancer. She is married with no children, does not smoke, and drinks approximately 4 glasses of wine per week. Physical exam was unremarkable.

Routine tests were ordered based on risks related to prior treatments, including TSH/free T4 for hypothyroidism, fasting lipid panel due to increased risk for metabolic syndrome, carotid ultrasound and echocardiogram due to increased cardiovascular risks, and breast MRI for high risk breast cancer screening (she had elected to discontinue recommended yearly mammography). On routine breast MRI she was found to have a 6 mm mass in the left breast. Ultrasound-guided core needle biopsy showed invasive ductal carcinoma. She was treated with bilateral mastectomy and sentinel lymph node biopsy, which showed an 8 mm grade 2 invasive ductal carcinoma and negative sentinel lymph nodes. The tumor was ER/PR positive and HER2 negative. She did not require adjuvant chemotherapy and elected not to pursue endocrine therapy with Tamoxifen. She continues to do well with no evidence of recurrence or additional complications from her prior HL-directed therapies.

Conclusion: This case highlights the risk of secondary breast cancer in survivors of pediatric and young adult HL treated with chest radiation to the developing breast, which has been characterized as a 35% cumulative incidence of breast cancer by age 50. According to the Children’s Oncology Group Long-Term Follow-Up Guidelines, annual screening with mammography and breast MRI is recommended from age 25 or 8 years posttreatment for women treated with ≥20
Gy chest radiation before age 30. The biology of secondary breast cancer tends to be more aggressive than that of primary breast cancer and overall survival is also worse due to comorbid conditions. In addition to counseling on lifestyle measures, including healthy diet and exercise, smoking cessation, and avoidance of heavy alcohol intake, which all may reduce overall cancer risk, internists should be aware of the early breast cancer screening recommendations for these patients. This case also emphasizes the unique treatment decisions faced by this high risk population, including the option of bilateral mastectomy to prevent future malignancy in the contralateral, unaffected breast. Ongoing research seeks to identify additional strategies for reducing breast cancer risk in the HL survivor population, including trials with selective estrogen receptor modulators and prophylactic bilateral mastectomy.

Bradly Kimbrough  
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Dr. Elida Voth

**Prostate Cancer Presenting as Back Pain**

Introduction: Though often an indolent process, prostate cancer once metastasized has high morbidity and mortality. Metastatic prostate cancer is found on initial diagnosis in 4% of patients. A high degree of suspicion is needed in patients presenting with urinary symptoms in conjunction with bone pain.

Case: A 61-year-old-man presented to the emergency department (ED) with hematuria and three months of low back pain, urinary frequency, and lower extremity edema. Over the past 2.5 months, he had been evaluated three times for these symptoms. During these visits he was noted to have left inguinal lymphadenopathy. A urinalysis and ultrasound of his left leg had been unremarkable, and a lumbar x-ray revealed degenerative changes in his spine. His pain had been refractory to acetaminophen, non-steroidal anti-inflammatory drugs, chiropractic intervention, and a transcutaneous electrical nerve stimulation (TENS) unit. One day prior to this ED visit his primary care physician (PCP) had discovered lower thoracic vertebral tenderness and ordered basic labs, a prostate-specific antigen (PSA) and CT abdomen/pelvis.

In the ED, he was found to have significant bilateral pitting edema, inguinal lymphadenopathy, and no neurologic deficits. Labs drawn by his PCP showed a PSA of 184 ng/mL (normal ≤4.5 ng/mL) and an alkaline phosphatase of 543 U/L (normal 45-115 U/L). CT abdomen/pelvis showed innumerable osteoblastic metastases and a destructive lytic lesion spanning the vertebral bodies of T10 and T11. Extensive retroperitoneal, pelvic, and inguinal lymphadenopathy was present.

He was admitted to medicine where an MRI of the thoracic and lumbar spine showed no cord compression. Fine needle aspiration of a left inguinal lymph node confirmed prostate adenocarcinoma. Orthopedic and radiation oncology recommended outpatient radiation and kyphoplasty for pain relief and prevention of vertebral collapse. An ultrasound of the inferior vena cava showed no evidence of thrombosis, and the lower extremity edema was attributed to lymphedema. In the outpatient setting, he began androgen deprivation therapy and chemotherapy with docetaxel.

The majority of metastatic prostate cancers metastasize to either the pelvis or vertebrae. It is important to ensure that patients with vertebrae metastases do not require urgent surgical intervention for spinal instability or cord compression. Palliative external beam radiation, as seen in this patient, can provide significant pain relief from bony metastases. Kyphoplasty can provide structural support in the case of pathologic fractures.

This case also highlights recent advances in prostate cancer management. Prior to 2015, the standard of care for metastatic prostate cancer was continuous androgen deprivation therapy. Recent landmark trials have shown that androgen
therapy combined with either docetaxel or glucocorticoids and abiraterone improve survival.

Conclusion: Clinicians should have a high suspicion for prostate cancer given that screening benefits are unclear, and prostate cancer is the third leading cause of cancer death in men.

Andrew Knutson  
Dr. Peter Lund  
*Music To My Ears: A Case of Chronic Salicylism*

Introduction: Chronic salicylate toxicity has a higher prevalence and mortality rate than acute salicylate toxicity. This is largely due to the difficulty in recognizing chronic toxicity and the delay in treatment.

Case: A 42-year old man with recently diagnosed atrial fibrillation and systolic heart failure, morbid obesity, and major depressive disorder presented to the emergency department with auditory hallucinations and shortness of breath. The patient had called police to his home because he heard burglars in his closet. Police noticed he was short of breath and called EMS. Of note, he had been hospitalized two months prior with new dilated cardiomyopathy and ejection fraction of only 25%. EKG and telemetry revealed atrial fibrillation with rapid ventricular response. Ischemic evaluation was negative and he was eventually diagnosed with tachycardia-induced cardiomyopathy. Since discharge, he had not been compliant with diuresis and his weight had increased 30 lbs. In the emergency department, he described hearing musical tones and noted severe jaw pain. He was tachycardic and tachypneic, but vital signs otherwise were within normal limits. Upon exam, patient showed expiratory wheezing, poor dentition with gross caries and tenderness over right lateral mandible. Admission labs were remarkable for a slightly elevated brain natriuretic peptide, a decreased bicarbonate level, a mildly elevated anion gap, and a normal lactate. He was admitted for acute congestive heart failure exacerbation. Upon further discussion with the patient, he noted ringing in his ears for the last month. He denied alcohol or drug use. He was taking large amounts of naproxen and aspirin for his jaw pain over the last few months. Bedside ultrasound of the heart and lungs showed grossly normal systolic function and no pulmonary edema. Salicylate level obtained and returned elevated confirming the diagnosis of chronic salicylate toxicity. He was treated with IV bicarbonate and did not require hemodialysis. His salicylate levels normalized over the next 24 hours. His hemoglobin level continued to drop and esophagogastroduodenoscopy was performed which showed a duodenal ulcer. He was eventually also diagnosed with osteomyelitis of the jaw requiring antibiotics and multiple tooth extractions.

Discussion: This case highlights some of the difficulty in identifying chronic aspirin toxicity. The presenting symptoms of chronic toxicity can differ from acute in that central nervous system symptoms, such as tinnitus, delirium, and hallucination, hyperventilation, and pulmonary edema are more common, making it more difficult to recognize. Delay in treatment is a major reason for the increased mortality and increased need for dialysis in chronic salicylate toxicity. The mainstay of treatment, however, remains the same in acute and chronic: alkalinization of the blood and urine and hemodialysis for more severe cases.

Christopher Kobe  
Dr. Ben Trappey  
*Systemic Capillary Leak Syndrome set off by Influenza Virus Infection*

Introduction: Monoclonal gammopathy-associated systemic capillary-leak syndrome, also known as Clarkson syndrome, is a rare, potentially life threatening condition characterized by recurrent episodes of capillary hyperpermeability in the context of a monoclonal gammopathy. During the acute episodes, fluid and protein leak from the intravascular compartment into the interstitium causing clinical signs of acute hypovolemia and interstitial
edema. The laboratory work-up is pathognomonic with marked hemoconcentration (Hct > 49% in men and > 43% in women) and paradoxical hypoproteinemia. We present a case of a 57 year old female with a past medical history of complex regional pain syndrome and hypertension diagnosed with Clarkson disease with monoclonal gammopathy IgG lambda type potentially triggered by influenza virus infection.

Case: The patient initially presented to an outside hospital with 5 days of fevers and fatigue and one day of syncope. On presentation, she was found to have influenza B with mixed anion gap and non-anion gap metabolic acidosis, Hbg 18.1, Hct 50.7, Ca 6.3, and hypoalbuminemia. Based on her presentation, she diagnosed with severe dehydration and presumptive sepsis due influenza. She was given a 30 cc/kg bolus of crystalloid fluid as well as started on empiric ceftriaxone with resolution in her lactic acidosis. However, during the evening on day of admission, the patient became unresponsive during repositioning in bed. The patient was found to be hypoxic to the mid 80s and had an unobtainable blood pressure on cuff reading despite some mentation. She was given an additional 2 L of crystalloid fluid as well as started on levophed and broadened to vancomycin and zosyn. Her hemodynamics improved however repeat labs demonstrated worsening lactic acidosis and hypoalbuminemia with an increasing hemoglobin to 19.5 and troponinemia. Given her continued periods of symptomatic hemodynamic instability and worsening lactic acidosis requiring an additional 7 L of crystalloid fluid and intermittent pressor support, she was transferred to the University of Minnesota with the presumed diagnosis of capillary leak syndrome. Throughout her hospitalization, she was monitored closely for common secondary sequelae and continued to receive hemodynamic and respiratory support as needed. She was eventually discharged home in stable condition after 7 total days of hospitalization. Subsequent serum and urine immunofixation confirmed monoclonal gammopathy with IgG lambda chain type. A review of her available medical records demonstrates no subsequent follow up or recurrent episodes.

Conclusion: Since first being described in 1960 by Dr. Clarkson and colleagues, fewer than 250 cases worldwide of systemic capillary leak syndrome (SCLS) have been reported. We will describe this case and highlight aspects of the pathogenesis, common complications, and treatment/prophylaxis of the syndrome.

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Dr. Jaskanwal Deep Sara
Dr. Juan Ripoll Sanz
Dr. Justin Brandler
Dr. Anjali Bhagra

Stevens-Johnson Syndrome: The Danger of Polypharmacy

Background: Stevens-Johnson Syndrome (SJS) is a severe mucocutaneous reaction that causes extensive necrosis and detachment of the epidermis. It is usually caused by medications and incidence ranges to as high as 5.3 cases per million people. Mortality can be as high as 10% in patients suffering from SJS.

Case: A 25-year-old male presented with a 3-day history of painful oral desquamation, odynophagia, diffuse rash, and sloughing lesions on his genitals. Past medical history was significant for mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes and seizure disorder. He was recently admitted for status epilepticus and was started on lacosamide (on chronic lamotrigine) and Bactrim for prostatitis. Physical examination was significant for a diffuse erythematous maculopapular rash, oral candidiasis, and desquamating ulcers of the lips, oropharynx, and genitals. Labs showed a hemoglobin of 9.8 g/dL, leukocytes of 6.5x10^9 cells/L, and lactate of 2.9 mmol/L. A dermatology consult was immediately placed and lesions were biopsied. Workup was negative for blood cultures, HSV, VZV, CMV, Syphilis, Chlamydia, Gonorrhea, Tick borne panel, HIV, and Mycoplasma testing. A fungal culture of his oropharynx revealed candida dubliniensis. Pathology results confirmed Stevens-Johnson syndrome. Due to high suspicion of SJS, lacosamide, lamotrigine, and Bactrim were held immediately on admission and
were the likely cause of his presentation. He was transitioned to Phenytoin for seizure prevention and discharged on fluconazole for oral candidiasis.

Discussion: SJS is characterized as a severe widespread epidermal necrosis causing tissue loss at skin/mucosal surfaces. SJS is defined as skin detachment of <10% of the body surface while Toxic epidermal necrolysis is >30%. Diagnosis is based on clinical presentation and biopsy showing full thickness necrosis of the epidermis and keratinocyte apoptosis. The main cause for SJS is an idiosyncratic reaction to systemic medications, with the most common culprits being sulfonamide antibiotics, anticonvulsants (phenytoin, lamotrigine), beta-lactam antibiotics, allopurinol, nevirapine, and oxicam NSAIDs. Mycoplasma pneumoniae infection has also been reported to cause SJS. Clinical presentation generally consists of a prodrome of fever and upper respiratory symptoms with progression to ulceration of mucosal areas and painful erythematosus vesiculobullous rash. Clinical course of SJS usually lasts about 8-12 days. Treatment relies predominantly on quick recognition, prompt withdrawal of the culprit drug, and supportive care in the burn ICU with fluids, pain control, and treatment of any superimposed infections (staph aureus and pseudomonas). The SCORTEN score is used to determine the clinical setting where the patient would be best managed and prognosis. Adjunctive therapies with corticosteroids, IVIG, and cyclosporine have also shown some promise. Mortality from SJS can range from 4-10% with disease severity being the main risk factor for mortality. By reporting this classic presentation of SJS, we reinforce the typical presentation and steps for diagnosis and treatment.

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Introduction: Amyloid light-chain fibril (AL) amyloidosis is a rare plasma cell dyscrasia, caused by deposition of unstable, misfolded monoclonal light chains into various organs leading to devastating organ dysfunction. Skin involvement is found in about 25% of individuals with AL amyloidosis. Here we present a rare, but classic presentation of AL amyloidosis, in which the diagnosis was made following a unique chief complaint.

Case: A 60 year old male with no significant past medical history presented to Dermatology clinic with a chief complaint of chronic “eye puffiness.” The patient described slowly progressive swelling around his eyes for the last two years. Other associated symptoms included bruising with minimal trauma (especially around his eyes), increasing difficulty in chewing food, and development of tongue ulcers. He also described a shiny, non-pruritic rash over his back, for which he was applying triamcinolone cream.

On skin exam, the patient had yellowish, waxy and translucent periorbital papules and plaques causing distortion of normal eyelid anatomy, along with a faint rim of ecchymosis surrounding his eyes. Waxy, yellowish shiny plaques, along with numerous ecchymoses were observed over his back and neck. Notably, the patient also had mild macroglossia.

Skin biopsy performed of left lower eyelid and his mid back identified diffuse hyaline deposits highlighted with Congo red staining, consistent with amyloidosis.

Complete blood count with differential and comprehensive metabolic panel were within normal limits. Urine protein electrophoresis identified 50% monoclonal peak protein. Serum protein electrophoresis, however, was negative. Free light chain serum assay showed elevation in lambda light chain of 45 mg/dL. Both serum and urine immunofixation electrophoresis identified monoclonal light chain of lambda type. The patient’s presentation was consistent with primary systemic AL amyloidosis and he was referred to Hematology/Oncology for further management. The patient had bone marrow biopsy performed, consistent with plasma cell dyscrasia, showing amyloid deposition and lambda monotypic plasma cells. Further workup did not suggest that the patient had any other evidence of organ involvement.
The patient was found to have good prognosis disease, with a plan to undergo multiple myeloma-like treatment regimen (with bortezomib, cyclophosphamide, and dexamethasone) followed by bone marrow transplant.

Discussion: Primary systemic amyloidosis is a rare disease, which carries a poor prognosis without therapy. Skin is involved in about 25% of individuals with primary systemic amyloidosis. Skin infiltrations by amyloid present as waxy, translucent, or purpuric papules, and commonly manifest with periorbital purpura (“raccoon eyes”), ecchymosis, and macroGLOSSIA. Skin manifestations of primary systemic amyloidosis should be recognized by the general internist, as early detection may bear important prognostic significance for the patient.

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### Aaron Kramer

**Occam's Razor or Hickam's Dictum? A Case Highlighting the Usefulness of Bedside Ultrasound**

Case: A 33 year old, previously healthy male presented to the ED with shortness of breath and hemoptysis. He returned to Minnesota the day prior to admission after a 2 year trip to East Africa. His flight was 12 hours, but states he felt short of breath prior to getting on the plane.

Upon arrival to ED, he was tachycardic with HR 129, but otherwise hemodynamically stable with O2 saturation 97% on RA, afebrile. CXR revealed RLL infiltrate, IV ceftriaxone and azithromycin were initiated in the ED for presumed CAP. Given his recent travel to endemic area, sputum cultures were obtained and he was admitted to the floor under isolation precautions.

What was quickly realized was there was more to the patient’s presentation than pneumonia. Patient was complaining of pleuritic chest pain, exam revealed bilateral leg swelling and lung sounds with bibasilar crackles. Heart sounds revealed no murmurs, but patient remained tachycardic but no murmurs were appreciated. Bedside ultrasound confirmed right lung infiltrate with mild pleural effusion, while heart views showed evidence of right ventricular dilatation and LV EF appeared grossly reduced.

As a result of the findings on bedside ultrasound, Care Everywhere was reviewed after patient consented. Patient received LAD thrombectomy in 2010 after presenting with chest pain. Echo was ordered, revealed severely reduced EF 28%, no obvious wma, and moderately reduced right ventricular function. CT Chest with contrast revealed bilateral pulmonary emboli with evidence of pulmonary infarction. He was started on IV heparin. Cardiology obtained CTCA, which revealed nonobstructive CAD, but with left and right apical thrombi. Lifelong anticoagulation was recommended and medications were initiated for HFrEF.

Furthermore, Quantiferon gold came back positive and Infectious Disease initiated treatment for presumed active TB infection. Ultimately, patient’s sputum cultures grew Mycobacterium intracellulare and TB medications were stopped.

**Conclusion:** This case illustrates how one can be influenced by anchoring, which is a psychological heuristic that influences how physicians assess patients. Specifically in this case, a young patient admitted for “pneumonia” delayed diagnosis and treatment of bilateral PE, bilateral apical thrombi, and MAI infection. Furthermore, this case highlights the importance of utilization of Care Everywhere, bedside ultrasound, and that Occam’s razor does not always apply.

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### Manoj Kumar

**Spotaneous Tumour Lysis in Non Small Cell Lung Carcinoma**

Case: A 56 year old female presented with one month of progressive exertional dyspnea and two days of pleuritic chest pain. Past medical history was significant for 20 pack years of smoking and emphysema. CT angiogram done
to rule out pulmonary embolism revealed a 5.2 x 3.8 cm left infrahilar mass and bilateral supraclavicular, subcarinal, esophageal and celiac axis lymphadenopathy and a moderate pericardial effusion. Ultrasound guided needle biopsy of the left supraclavicular lymph node revealed tissue with fibrosis and necrosis containing small groups of malignant cells with immunostaining positive for CK7 and TTF-1 and negative for CK20, p40, mCEA, CK5/6 and CDX-2, compatible with non small cell lung cancer of adenocarcinoma subtype. Nine days after admission the patient developed the acute onset of altered mental status and respiratory insufficiency. Laboratory investigations revealed a serum potassium of 7.6 mEq/L, uric acid of 10.5 mg/dl, phosphorus of 7.7 mg/dl, creatinine of 2.09 mg/dl (baseline creatinine was 0.9 mg/dl) and calcium of 9.2 mg/dl thus meeting three of the four laboratory criteria for Tumour Lysis Syndrome per the Cairo-Bishop definition. She received a single dose of rasburicase and IV hydration. A follow up uric acid severe hours later was 4.6 mg/dl. Pericardiocentesis was performed for symptomatic relief, with immunohistochemical staining of the fluid showing positivity for MOC31, TTF1, CD68 and negativity of calretinin compatible with a lung adenocarcinoma. In this setting of stage 4 disease and her acute clinical deterioration, her prognosis was deemed poor, and in discussion with the patient and family, was placed on comfort care.

Conclusion: Tumour lysis syndrome (TLS) is an oncologic emergency, resulting in metabolic derangements, notably hyperkalemia, hyperphosphatemia, hyperuricemia, hypocalcemia and potential renal insufficiency due to uric acid crystallization in the renal tubules. Correction of electrolyte abnormalities, aggressive hydration and urate lowering therapy with agents such as allopurinol or rasburicase are mainstays of treatment. TLS most commonly occurs following cytotoxic/radiation therapy in malignancies with high proliferation rates and ‘‘bulky’’ disease; spontaneous tumour lysis occurs much less commonly. TLS is most often seen in hematological malignancies such as high grade lymphomas and acute leukemias. It is much less commonly reported in solid tumours. Only eight cases of TLS have been reported in Non Small Cell Carcinoma of Lung (NSCCL) and they were triggered by cytotoxic and radiation therapy. We report the first case of spontaneous TLS in NSCCL.

A high degree of suspicion and expectant management is needed for the occurrence of TLS, with prompt institution of supportive therapies to decrease morbidity and mortality from this complication.

Vinayak Kumar
Dr. Kimberly Johnson
Dr. Maurice Sarano

A Complicated Case of Hereditary Hemorrhagic Telangiectasia causing High Output Heart Failure

Background: Hereditary hemorrhagic telangiectasia (HHT), formerly called Osler-Weber-Rendu syndrome, is an autosomal dominant disorder that results in vascular defects such as arteriovenous malformations (AVMs) and telangiectasias. Clinical complications include mucocutaneous telangiectasias, epistaxis, gastrointestinal bleeding, and AVMs in pulmonary, hepatic, and cerebral circulation.

Case Description: 71-year-old female with a history of hereditary hemorrhagic telangiectasia (HHT), atrial fibrillation with recent left atrial appendage closure with WATCHMAN device, and severe tricuspid regurgitation (TR) was admitted for decompensated heart failure. On admission, EKG indicated atrial fibrillation (AF) with rapid ventricular response and transthoracic echocardiogram (TTE) showed a growing pericardial effusion compared to the TTE from 2 months prior, which was obtained following the placement of WATCHMAN device. Repeat TTE with bubble study showed moderate to large pericardial effusion without tamponade physiology, severe TR, severe RA enlargement, estimated RVSP 55 mmHg, moderately enlarged RV with reduced systolic function, and multiple large hepatic AVMs. Given her history of HHT
and the hepatic AVMs on TTE, the clinical findings were most consistent with high output cardiac failure due to shunting through the hepatic AVMs (Cardiac Index 5.56 L/min/m2). Her AF was controlled with metoprolol and she was given a 10 g amiodarone loading dose with intention of later cardioversion. She was diuresed heavily with partial improvement of symptoms. She later developed evidence of tamponade physiology and underwent pericardiocentesis with temporary pigtail catheter placement. Pigtail output was hemorrhagic and consistent with microleak from WATCHMAN placement. For her right-heart failure, she underwent bioprosthetic tricuspid valve replacement (without anticoagulation due to bleeding risk). She continued to be diuresed after the surgery.

She is scheduled for triple phase CT chest/abdomen/pelvis once stabilized to perform targeted intervention on hepatic AVMs.

Discussion: High output heart failure is an etiology often overlooked as a cause for heart failure beyond the settings of sepsis, severe anemia, or hyperthyroidism. Our patient had developed significant cardiac remodeling with development of severe TR and AF as a consequence of multiple large untreated hepatic AVMs, which can be a known occurrence in the setting of HHT. This case demonstrates the oversimplification of just characterizing heart failure by ejection fraction, and further highlights the importance of maintaining a broad differential when diagnosing the etiology of new onset heart failure in order to treat all possible reversible causes, especially in the setting of rare conditions like HHT.

Rebecca Kummer

Recognizing Thrombotic Thrombocytopenic Purpura - Thinking Outside the Pentad

Introduction: Thrombotic thrombocytopenic purpura (TTP), a primary thrombotic microangiopathy caused by deficiency of the ADAMTS13 protein, is a rare but clinically significant cause of thrombocytopenia. Without treatment, this condition is highly fatal. Therefore, it is important to recognize this condition early on in its course. The pentad of TTP is well known, but it is rather uncommon for a person to demonstrate all five of these signs and symptoms at presentation, and in fact many do not appear critically ill. This is a case of acquired TTP in an elderly man with vague presenting symptoms and the rapid development of his clinical course.

Case Presentation: The gentleman was an 81-year-old man with diabetes and hypothyroidism who presented to the emergency room by ambulance with one day of nausea, epigastric abdominal pain, and dyspnea. He was vitally stable, afebrile, and in no acute distress on arrival. Work up was significant for elevated troponin without EKG changes, an elevated lipase, and an elevation in bilirubin. The patient was also found to have a Hgb of 8.8 (baseline ~10) and platelets of 11. CT abdomen/pelvis was negative for acute pathology. The patient was admitted to the cardiac floor for further work up of his elevated troponins and suspected pancreatitis. Shortly after arrival to the floor he developed fevers and hypertension. He became altered and aphasic, which was a drastic change from his neurologic examination in the emergency room. A stroke code was called. The patient required sedation for additional imaging and became obtunded, requiring intubation for failure to protect his airway. Upon arrival to the MICU, the patient’s symptoms were reviewed and significant for fever, anemia, severe thrombocytopenia, and neurologic changes highly concerning for TTP. Hemolysis labs and a peripheral smear were obtained, which were consistent with microangiopathic hemolysis. Hematology/Oncology was consulted and recommended initiating plasma exchange. PLEX was started less than 24 hours after presentation. Prednisone 1 mg/kg was added to the treatment regimen the following day. The platelets normalized after the fifth PLEX treatment. The ADAMTS13 activity returned as low (<5%), reflective of high risk of recurrence. Attempted to continue PLEX until platelets were normal.
x2 and LDH had normalized, however the patient and his family requested to discharge prior to treatment completion.

Conclusion: This case highlights the importance of recognizing TTP early on in its clinical course. The classic pentad of TTP is quite rare at time of presentation, with many more cases presenting with abdominal pain and other vague symptoms. Quick recognition of this syndrome is necessary to limit the consequences of this fatal condition.

Erik Kuross

A Unique Case of ITP Secondary to Sarcoidosis

Introduction: ITP is classified as primary or as secondary and is a diagnosis of exclusion. Secondary ITP is most well known for being secondary to medications, autoimmune disorders (SLE and APS,) lymphoproliferative disorders, HIV, and hepatitis C. Here we have an unique case of ITP secondary to Sarcoidosis.

Clinical Presentation: A 63 year old male with a past medical history significant for HTN and DMII presented to the HCMC ED after having an episode transient confusion while at work. He was found to have a platelet count of 17 k/cmm with his last platelet count in 2015 being 278 k/cmm. He had no previous illness, no signs of bleeding, no concerning medications, nor any signs of hemolysis on peripheral smear or labs. Heme/Onc was consulted and patient received two doses of IV steroids with little response, and then 2g/kg IVIG with normalization of platelets. Further lab workup revealed normal LFT’s, and negative hepatitis C, hepatitis B, and HIV. Thrombocytopenia was presumed to be due primary versus secondary ITP on admission with further workup to be completed.

Head CT was done which incidentally found mediastinal lymphadenopathy. Follow up CT CAP showed perihilar/mediastinal LAD and pulmonary nodules < 1 cm. While in the hospital he was also found to have a left temporal enhancing lesion on brain MRI with differential concerning for primary brain neoplasm, lymphoma and sarcoidosis. Further workup of lymphadenopathy included bronchoscopy, EBUS lymph node biopsy, and mediastinoscopy. These were negative for malignancy but did show non necrotizing granulomas as well as necrotizing granulomas with some concern for fungal involvement. While pursuing further fungal workup patients platelets dropped to 12 k/cmm and he was treated with 1g/kg IVIG with appropriate response. Further fungal workup was negative and it the patient was diagnosed with pulmonary and neurological sarcoidosis. The patient was started on treatment for sarcoidosis and has had no further ITP flares.

Discussion: Primary and secondary ITP remain a diagnosis of exclusion, and the response to therapies differ for both. It is important to rule out secondary ITP before assuming thrombocytopenia is from primary ITP. The above patient developed ITP a second time despite proper treatment. Since beginning treatment for sarcoidosis he has had no more episodes of ITP. This is a striking example of the differences in pathophysiology of primary and secondary ITP, and it points out the need for proper diagnosis and treatment of underlying disorder when necessary.

Conor Lane
Dr. Patrick Hoversen
Dr. Tony Chon

Normal Transesophageal Echocardiography and Normal Positron Emission Tomography in MRSA-Related Cardiac Device Infection

Introduction: Staphylococcus aureus remains a leading cause of community acquired bacteremia and is associated mortality rates of 20 to 40 percent. Methicillin-resistant Staphylococcus aureus (MRSA) has shown a dramatic rise in prevalence since it was first described in the 1960s and is associated with even higher mortality rates than methicillin-sensitive Staphylococcus aureus.
Cardiovascular implantable electronic devices (CIEDs) have become an increasingly important treatment modality for a wide array of cardiac arrhythmic disorders and heart failure, but device and lead infection are an important associated cause of morbidity.

Case: A 69-year-old man presented to hospital with fever, chills and persistent diarrhea and stool PCR confirmed community-acquired Clostridium difficile infection. He was treated with a course of oral vancomycin. The patient had a dual-chamber permanent pacemaker inserted 18 years prior for sinus node dysfunction and Mobitz type 2 atrioventricular block. The atrial lead was revised 11 years prior to admission for elevated pacing thresholds and his ventricular lead was revised 4 years prior to admission. On day 6 of hospital admission, he developed high-grade fever to 39°C and blood cultures demonstrated growth of MRSA. The initial suspected source was a peripheral intravenous cannula which developed phlebitis, and the patient was treated with intravenous vancomycin. Daily blood cultures showed persistent MRSA bacteremia despite resolution of right arm phlebitis/cellulitis. Transesophageal echocardiography (TEE) was performed for suspected device/lead infection which did not demonstrate any vegetations. Fluorodeoxyglucose positron emission tomography-computed tomography (F-FDG PET/CT) was performed to evaluate for lead infection and assess for other potential sources for infection. F-FDG PET/CT scanning showed no evidence of lead infection and no occult sources of infection. Due to persistent bacteremia, the patient proceeded to device and lead extraction. Culture of extracted leads showed > 100 colony forming units/10 ml growth of MRSA. The patient’s bacteremia resolved and he completed a course of intravenous vancomycin. His pacemaker was reimplemented and the patient was successfully discharged.

Conclusion: Heart Rhythm Society and Infectious Diseases Society of America practice guidelines for diagnosis of CIED device infection advise that TEE is the key imaging modality for confirming the diagnosis of device or lead infection. The most recent guidelines suggest F-FDG PET/CT scanning is a useful adjunctive test with high sensitivity and specificity for CIED infection, which can aid in diagnosis. This case demonstrates that despite the sensitivity of TEE and F-FDG PET/CT scanning, patients with normal TEE and PET but persistent MRSA bacteremia may still have lead/device infection requiring device extraction.

Daeinelle Lang
Dr. Victoria Walston
Dr. Dame Idossa
Dr. Meltiady Issa

An Unusual Case of Hodgkin Lymphoma Misdiagnosed as Chronic Osteomyelitis

Introduction: Classic Hodgkin Lymphoma is a malignant lymphoma arising from B-cells which accounts for about 10% of all lymphomas. The most common presentation is asymptomatic lymphadenopathy, but presentation can vary depending on the stage and areas involved. Bone involvement and musculoskeletal symptoms can be seen, and this poses a diagnostic challenge which can lead to the misdiagnosis and treatment as chronic osteomyelitis. Advanced imaging and biopsy are often necessary to establish the diagnosis.

Case Presentation: A previously healthy 40-year-old man was evaluated multiple times for several months of atraumatic low back and left leg pain, weight loss, fevers, chills and night sweats. Work up at that time showed leukocytosis and eosinophilia with negative blood cultures. CT scan showed lytic destruction of the left sacrum with iliac and retroperitoneal lymphadenopathy suggestive of osteomyelitis. A bone biopsy showed bony destruction but was inconclusive for an etiology. He was treated with 2 months of broad spectrum IV antibiotics but had progression of symptoms, requiring hospital admission. Physical exam on presentation to the hospital showed an ill-appearing man in moderate distress, unable to ambulate due to pain. No skin rashes or focal points
of infection were noted. Neurologic exam showed no cauda equina and no focal deficits, but some limitation due to pain. Labs showed C-reactive protein of 190, erythrocyte sedimentation rate of 135, leukocytosis at 19K with 21% eosinophilia, and a normocytic anemia with hemoglobin of 8.4. CT showed progression of L5 and sacral destructive lesions and extensive abdominopelvic lymphadenopathy. He underwent CT-guided biopsy of the sacral bone and pelvic lymph nodes. Pathology showed extensive areas of necrosis associated with acute and chronic inflammation. Lymph node pathology showed atypical large CD30+ mononuclear lymphoid cells. PET scan showed diffuse FDG uptake in common iliac chain, retroperitoneal, and pulmonary hilar lymph nodes, along with diffuse bone marrow, spleen, sacral and iliac bone activity. CT-guided core biopsy of a left iliac node confirmed Classic Hodgkin Lymphoma. He was initiated on ABVD chemotherapy and was discharged in stable condition to continue his treatment as an outpatient.

Discussion: This case illustrates an unusual presentation of Classic Hodgkin Lymphoma that was initially missed and treated as osteomyelitis. It is vital to keep a broad differential for patients who present with lytic bone lesions and hematologic abnormalities, including infectious etiologies, rheumatologic disease, and malignancy. Additionally, the diagnosis of chronic osteomyelitis, despite lack of isolated microorganisms and no response to antibiotics, should raise suspicion for another process, including malignancy, which warrants prompt further investigation. Advanced imaging and multiple biopsies might be needed to establish the diagnosis, given that the pathology of Hodgkin Lymphoma involves a small amount of neoplastic cells on predominantly inflammatory background which contributes to the diagnostic difficulty.

**Jeffrey Larson**

**Antiphospholipid Antibody Syndrome as a Cause of Mechanical Mitral Valve Thrombosis and Thromboembolic Myocardial Infarction**

Case Presentation: A 28 year old female presents with atypical chest pain, headache, and dizziness. She has known antiphospholipid antibody syndrome (APS) complicated by left MCA stroke (2013) and nonbacterial thrombotic endocarditis (NBTE) status-post mechanical mitral valve replacement and is on therapeutic warfarin. Admission labs notable for elevations in troponin, NT-ProBNP, CRP ESR, a supratherapeutic INR of 4.2 and a low chromogenic factor X, the latter used to adequately assess plasma thrombin generation in the presence of lupus anticoagulant. TTE showed a decreased LVEF from one month prior with a new inferior wall motion abnormality but was otherwise unchanged and showed no evidence of a thrombus. Anticoagulation was withheld after repeat INR was subtherapeutic, however, TEE thereafter showed a large mobile thrombi of high embolic potential on her mechanical mitral valve annulus. This, along with her new WMA and troponin elevation, suggested a thromboembolic myocardial infarction might be the etiology of her underlying chest pain. Repeat TEE one day later showed an increase in the MV thrombus size despite initiation of therapeutic heparin. After extensive consultation with various specialties a low dose infusion of thrombolytics (alteplase at 1 mg/hr for 25 hours) was started to reduce the patient’s clot burden. She was given a second round of low dose thrombolytics after repeat TEE two days later showed normalized LVEF and decreased clot burden. The patient ultimately underwent a third and fourth round of thrombolytic infusion, the latter at a more aggressive alteplase regimen (4 mg/hr) administered over 35 hours rather then 24 hours which ultimately led to significant improvement of her MV clot burden. Ultimately the decision was made to treat the patient with a “modified” catastrophic APS regimen which included high dose corticosteroids, 5 days of IVIG, resumption of heparin and warfarin following discontinuation of thrombolytics, and a prolonged rituximab regimen. Regular monitoring of her
INR, chromogenic factor X, and cardiolipin Antibody were needed to guide therapy. Plasmapheresis was withheld given absence of classic catastrophic APS. Fortunately, the patient’s symptoms improved during her hospital course with the only complication being significant bruising of her extremities.

Conclusion: Despite the lack of robust literature characterizing the use of continuous thrombolytic therapy for mechanical mitral valve thrombi, the patient’s MV thrombi improved significantly without acute complication using a combination of low dose and high dose continuous alteplase, intermittent therapeutic anticoagulation with heparin drip, and serial TEEs. Management of the patient’s primary issue (APS) is of utmost importance for long-term control of her thrombotic events. Additionally, the patient’s supra-therapeutic INR on admission did not correlate with an appropriate chromogenic factor X levels in the setting of her lupus anticoagulant.

Leo Laub
Dr. Anjum Kaka

An HIV+ Patient with Abdominal Discomfort and Lower Extremity Nodules

Case: A 46 year old male with HIV presented to his primary care physician with growing nodules on his abdomen and shin as well as intermittent epigastric pain. The patient was recently discharged after a 1-month hospitalization for pulmonary tuberculosis (TB) and Candida esophagitis. On admission, he had not been on antiretroviral therapy (ART). He was started on rifampin, isoniazid, pyrazinamide, and ethambutol for TB, and two weeks later on abacavir/dolutegravir/lamivudine (Triumeq) for HIV (viral load 56,703 copies/mL, CD4 381 cells/m3). His course was complicated by fevers ascribed to TB immune reconstitution inflammatory syndrome.

At the PCP office, the patient complained of an abdominal skin nodule that had been present for over a year, but had recently increased in size. He also described 2-3 months of intermittent epigastric pain without obvious precipitating or relieving factors and non-responsive to pantoprazole. On exam, he had tenderness in the epigastrium on deep palpation and a 1x0.5 cm erythematous, firm nodule located in the right upper quadrant of his abdomen. Two additional maroon, 0.5x0.5 cm subcutaneous nodules were noted on his right shin. A PET scan showed FDG-avid lesions in the stomach and liver. He underwent excision of the abdominal nodule and pathology showed Kaposi sarcoma (KS). EGD demonstrated an ulcerated, non-circumferential, bleeding mass in the fundus; pathology confirmed H. pylori-associated gastritis and KS. He received bismuth subsalicylate, omeprazole, amoxicillin, metronidazole, and ART was continued. Within five months of starting ART, he had an undetectable viral load. Clinically, his abdominal pain resolved and the nodules on his leg nearly disappeared. A PET three months later showed improvement of the fundic mass and resolution of the hepatic lesions.

Conclusion: KS is an angioproliferative disease driven by human herpes virus 8 and is seen most commonly in patients with HIV/AIDS. It typically presents as a characteristic-appearing cutaneous disease. In more advanced cases of KS, almost any visceral organ can be involved, including the oral cavity (33% of patients with KS), GI tract (40% of patients with KS) and lungs. A lower CD4 count at HIV diagnosis, history of substance abuse, and mental illness increases the risk of KS. Treatment depends on the extent of tumor burden. ART is generally sufficient for limited disease; systemic chemotherapy is occasionally needed for diffuse disease. In this case, chemotherapy was deferred given how quickly his viral load fell and cutaneous disease improved with ART alone. He was last seen about a year after his initial presentation and has not had recurrent abdominal discomfort.
Introduction: Multidrug resistant tuberculosis (MDR-TB) is an infection caused by Mycobacterium tuberculosis that is resistant to at least both isoniazid and rifampin. Resistance to first-line anti-TB medications poses a therapeutic challenge for providers and patients alike due to the lower efficacy of second line therapies as well as increased side effects, inconvenience of dosing schedules, and prolonged isolation of patients in the hospital.

Case: An 80-year-old Hmong-speaking male from Laos with past medical history of hypertension, hyperlipidemia, opioid dependence, and anxiety was admitted directly to Regions Hospital by the Minnesota Department of Health (MDH) due to a positive acid-fast bacilli (AFB) culture and subsequent PCR sequencing showing MDR-TB. He was asymptomatic prior to admission but was attending an adult daycare center in St. Paul to which multiple cases of MDR-TB had been linked. Admission labs were unremarkable and physical exam revealed bilateral crackles on lung auscultation but was otherwise normal. He was started on a regimen of pyrazinamide, linezolid, moxifloxacin, and amikacin. Ethionamide was added to the regimen once he was tolerating the above medications. Cycloserine was not added due to complications with severe depression, which was evaluated multiple times by inpatient psychiatry. Treatment for his depression was complicated by the possibility of interactions between anti-depressants and anti-TB medications. He also developed severe nausea, GI upset, and poor appetite, causing the patient to frequently refuse doses altogether. Numerous family conferences were convened with the help of Hmong interpreters to discuss the necessity of taking every medication consistently, the importance of nutrition in fighting infection, and the possibility of G-tube feeding. The patient and family eventually agreed to a nasogastric tube placement to facilitate better nutrition, however, the patient pulled out the tube immediately after placement. Given improved nutrition markers and consideration of overall mental health, further tube feeding was avoided. After over 11 weeks of therapy, repeat AFB cultures were negative. He was discharged to home on IV amikacin and moxifloxacin through PICC line and oral pyrazinamide, ethionamide, and linezolid through directly observed treatment by Ramsey County.

Conclusion: This case demonstrates the importance of recognizing a patient population who is vulnerable and at higher risk for MDR-TB as well as familiarizing available treatment options for MDR-TB. It also emphasizes dilemmas that are often associated with the medications used for MDR-TB, in addition to the need for prolonged hospitalization and severe side effects. Lastly, this case demonstrated many of the cultural barriers that can be encountered in medicine, including language barriers, mistrust of the medical system, and traditional beliefs that may be at odds with western medical protocols. This specific case encourages medical providers to consider cultural humility when interacting with patients from different cultures.

Sharon Li

It's Not a Fluke: The Cause of a Hepatic Abscess

Introduction: The internist is no stranger to the constellation of findings that accompany cholecystitis in its multiple forms—indeed, cholecystitis is such a common admission diagnosis that cholecystectomy is the 6th most common operating room procedure performed during hospital admission. Though outcomes of acute cholecystitis are generally good, complications including gangrene, perforation, cholecystoenteric fistula formation, and gallstone ileus increase morbidity and mortality of this otherwise common and treatable condition.

Case: The patient is a 46-year-old female who lives on a farm and has a history of polysubstance abuse who presented to her primary care doctor with right upper quadrant abdominal pain, weakness, subjective fevers, chills, and intermittent drenching sweats for one week. She was transferred for admission
to our facility after right upper quadrant abdominal ultrasound revealed a cystic mass within the gallbladder fossa that on follow up CT abdomen was concerning for perforated cholecystitis and hepatic abscess. Hepatic panel was significant only for a slightly elevated alkaline phosphatase at 181, tumor markers including AFP and CA19-9 were within reference range, and Echinococcus, E. histolytica, and hepatitis serologies were negative. No ova or parasites were isolated in the patient’s feces. On hospital day 3, interventional radiology placed a drain in the fluid collection and sent the fluid for cytology and culture—it grew Streptococcus anginosus, an abscess-forming gram-positive organism.

The patient was discharged with the drain in place and on four weeks of IV ceftriaxone. After documenting resolution of her hepatic abscesses on antibiotics, the patient underwent laparoscopic cholecystectomy without complication.

Discussion: Usually a commensal of the oral mucosa, Streptococcus anginosus is known to be associated with postoperative fluid collection in appendicitis and, in one case, pyogenic liver abscess in a patient with poor oral hygiene and underlying tuberculosis. It has not before been documented to cause cholecystitis. Abscess formation, as in this case, is a proclivity of this organism, and it is therefore considered a particularly virulent member of the viridans streptococcus group which should be considered a true pathogen when isolated.

Conclusion: Clinicians should be aware of hepatic abscess of a rare but important presentation of cholecystitis, as management will likely be complicated and require a multidisciplinary treatment team to minimize the increased morbidity associated with this condition.

Why Does Dura Matter After LP?

Introduction: This case describes the evaluation, differential and necessary diagnostics involving a patient with meningeal carcinomatosis.

Case: A 58 year old woman with recently diagnosed lymphoma presented with fever and confusion. Two months prior she had been diagnosed with stage IVe peripheral T cell lymphoma. She underwent a partial cycle of CHOP and two cycles of EPOCH that finished three weeks prior to admission. She had a disappointing follow-up PET CT just before admission that demonstrated mild partial response to chemotherapy. Over the preceding week, family reported lethargy, dehydration and progressive confusion.

In the ED, she was hemodynamically stable but had a fever (101.5) and was noted to have seizure-like activity for 15-20 seconds. Labs were notable for leukocytosis (11.9), elevated lactate (2.4). She was unable to initially have a CT, MRI or LP due to agitation. She was placed on broad spectrum antibiotics to cover meningitis and admitted to the ICU.

Her hemodynamics decompensated further, requiring intubation and sedation. Once she stabilized, MRI was performed and showed leptomeningeal enhancement of the folia. This was concerning for bacterial meningitis vs meningeal carcinomatosis. LP was required to differentiate and determine treatment, and this confirmed malignant cells and no growth on CSF cultures, consistent with meningeal carcinomatosis. Her antibiotics were stopped and she was started on intrathecal cytarabine chemotherapy. She was extubated after two days and discharged on hospital day 7.

Conclusion: Important in this case was distinguishing the order of diagnostics. An LP will cause leptomeningeal enhancement on MRI, thus had an LP been performed prior to imaging, it would have been more difficult to interpret
results. When able, brain imaging should be performed prior to LP to both assess for increased intracranial pressure as well as preserve pathology. This is contrasted by the need for urgent/emergent LP in cases of bacterial meningitis and risk of delaying antibiotics.

Another aspect to this case is the recognition of CNS spread of lymphoma as part of the differential for confusion, fever and seizure, as well as what assistance a lumbar puncture can provide.

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### Justin Lockrem

**Thyrotoxicosis: A case of Worsening Schizophrenic Psychosis and the Rare Utilization of IV Methimazole as a Bridge to Thyroidectomy**

**Introduction:** Hyperthyroidism due to Grave’s disease and its treatment with methimazole and PTU has been documented numerous times in the literature and with good results. Both medications are administered orally/enterally and present a challenge to a patient who has questionable capacity and severe schizophrenia with worsening psychosis in the setting of thyroid storm.

**Case:** Mr. M is a 65 year old man with Grave’s disease and multiple admissions for hyperthyroidism complicated by tachycardia induced cardiomyopathy, congestive heart failure, atrial fibrillation with RVR, and worsening internal pre-occupation and psychosis on top of underlying schizophrenia. He presented in afib with RVR, acute on chronic psychosis, and systolic heart failure in the setting of weeks without taking his oral methimazole. On presentation, vitals were remarkable for HRs in the 160s with stable soft blood pressures. Physical exam remarkable for defiance, internal pre-occupation, and refusal of multiple interventions. Labs remarkable for T3 >460 and undetectable TSH.

Initially his atrial fibrillation was controlled with esmolol drip due to his known systolic heart failure. The patient continued to decline oral and rectal administration of medications but was amenable to IV medications. This posed a large ethical and medical dilemma as to deliver the safest care and attempting to abide by the patient’s wishes and values despite active thyrotoxicosis, psychosis, and questionable capacity.

A case series from Georgetown was utilized to advocate for the creation of IV methimazole, which to our knowledge, has never been used at this institution prior. Two weeks of IV methimazole allowed for stabilization of thyrotoxicosis and bridged to definitive management with eventual thyroidectomy. IM levothyroxine along with IM Haldol was eventually used for simplicity and to assist with adherence to medication regimen.

**Conclusion:** This case illustrates the difficult ethical, legal, and medical decisions around non-adherence and mental health, and the limiting factor of medication administration.

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### Teija Madhusoodanan

**Pheochromocytoma: When an Incidentaloma Can Cure A Patient's Hypertension**

**Introduction:** Pheochromocytoma is a rare catecholamine-secreting tumor that accounts for less than 0.2 percent of patients with hypertension. Often patients with pheochromocytoma may go undiagnosed and treated simply as essential hypertension. Classic symptoms include sweating, tachycardia, headache, palpitations, and hypertension, which can sometimes be resistant to treatment even with use of multiple anti-hypertensive agents. In many patients, the diagnosis is made only after an adrenal mass is incidentally found on imaging for unrelated issues. The treatment is surgical resection if possible.

**Case Presentation:** The patient is a 73 year-old woman with past medical history of hypertension, chronic kidney disease, and recurrent UTIs. She initially
presented to the Urology clinic for further evaluation of pelviectasis. After undergoing cystoscopy and CT urogram, she was found to have a left adrenal mass. An MRI was performed and characterized the mass as 2.5 x 3.5 x 2.7 cm in size and irregularly shaped with central necrosis concerning for malignancy. On further workup, the patient was found to have high renin (thought to be related to lisinopril use), normal 1mg dexamethasone suppression test, upper limit of normal plasma normetanephrine, and four times the upper limit of plasma free metanephrines. Studies were repeated including plasma and 24-hour urine studies which found persistent elevation of plasma free metanephrines as well as elevated 24-hour urine epinephrine and metanephrine confirming the diagnosis of pheochromocytoma. In the weeks leading to the surgery, the patient was prepped with alpha and beta blockade. When sufficiently blocked, she underwent left adrenalectomy with biopsy confirmed pheochromocytoma. In the initial weeks after the surgery, she was weaned off of alpha and beta blockade and no longer required anti-hypertensive medications. Additionally, her initial symptoms of palpitations and fatigue had completely resolved. Repeat plasma and 24-hour urine studies were within normal limits. Plasma metanephrines decreased from 2.1 nmol/L to <0.2 nmol/L (normal <0.5 nmol/L). Plasma normetanephrines decreased from 0.91 nmol/L to 0.39 nmol/L (normal <0.9 nmol/L). Twenty four hour urine metanephrines decreased from 1307 micrograms/day to <20 micrograms/day (normal 39-143 micrograms/day). Twenty four hour urine epinephrine decreased from 73 micrograms/day to <5 micrograms/day (normal 1-5 micrograms/day).

Discussion: This case illustrates the classic presentation of a rare disorder, pheochromocytoma. With hypertension, palpitations, and the incidental finding of adrenal mass on CT, this patient’s treatment course for her hypertension dramatically changed. Additionally, for this patient, treating the pheochromocytoma prevented progression of her known kidney disease as well as provided protection from the other adverse effects of uncontrolled hypertension.

Dermatomyositis Presenting as Interstitial Lung Disease

Case Presentation: A 41-year-old male presented with a 6-week history of a dry cough and progressive dyspnea with exertion. He denied any systemic symptoms. He had received two courses of antibiotics recently without improvement in symptoms. He did not report any sick contacts. He was a non-smoker, originally from Mexico but had moved to the US about 5 years ago and had not travelled since. He worked as a cook at a noodle shop, did not keep any pets, and denied any occupational exposures. He denied having had similar symptoms previously and denied a family history of autoimmune disease.

On exam he was hypoxic and had diffuse dry crackles bilaterally on auscultation. Skin exam was normal. Chest x-ray revealed bibasilar opacities and CT imaging showed bilateral ground-glass infiltrates with traction bronchiectasis and hilar and mediastinal adenopathy. Broad spectrum antibiotics were initiated but later discontinued after pulmonary consultation. His presentation was felt most likely to be secondary to an interstitial lung disease and a wedge biopsy was obtained which revealed acute organizing fibrinous pneumonia. He was prescribed daily prednisone with a planned taper and serologies for autoimmune diseases associated with lung involvement were sent.

A few weeks after completing the prednisone taper he presented in clinic with recurrence of his cough, progressive fatigue and stiffness and swelling in both hands. Chest auscultation again revealed bilateral crackles. Examination of his hands revealed generalized swelling without synovitis, palmar hyperkeratosis with fissuring of the distal fingers and erythematous scaling papules over the dorsum. Lab tests revealed a positive anti-nuclear antibody, elevated creatine kinase and a positive anti-Mi2 antibody. Daily prednisone was re-initiated but
this time in conjunction with azathioprine. Over the next few months, the patient experienced a significant improvement in his symptoms and repeat imaging showed improvement of the bilateral opacities.

Discussion: It is interesting how this patient initially presented with isolated lung involvement and later on developed clinical and serologic evidence of an inflammatory myopathy. He was also positive for anti-Mi2 antibodies, the presence of which is frequently associated with the development of interstitial lung disease. His clinical phenotype was most representative of dermatomyositis due to the presence of Gottron’s papules and muscle weakness, however the development of mechanic’s hands is very unusual for this condition and would be more consistent with anti-synthetase antibodies which this patient tested negative for. The possibility exists that an as yet unidentified antibody which was not tested for contributed to the development of his disease. He derived substantial benefit from immunomodulatory therapy which is the mainstay of treatment for inflammatory myopathies. Chronic treatment with these regimens mandates monitoring for their long-term adverse effects and potential complications.

Cassedy Mahrer
Dr. Rawad Nasr

Lupus Enteritis: A Case of Difficult to Treat Abdominal Pain in an SLE Patient

Introduction: Lupus enteritis is a rare complication of Systemic Lupus Erythematosus (SLE) and is a source of significant distress and morbidity for patients. Clinical symptoms include severe abdominal pain, vomiting and diarrhea. Diagnosis is contingent upon radiologic evidence of small bowel edema or histologic evidence of lupus vasculitis on tissue biopsy in the absence of other etiologies. Treatment of lupus enteritis is based on anecdotal evidence.

Case Presentation: This case involves a 22 year old female patient with newly diagnosed SLE with the following clinical and serologic features: positive ANA with a titer of 1:1280 with speckled pattern, positive ScL-70, RNP, and SSA antibodies, hypocomplementemia, bitemporal alopecia, leukopenia, anemia, discoid lupus, oral ulcers, sicca syndrome and polyarticular inflammatory arthritis. One week after her initial visit to the rheumatology clinic, the patient presented to the emergency department with subacute progressive abdominal pain in the setting of her active SLE. CT scan of the abdomen revealed the classic target sign and diffusely edematous small bowel and colon. Stool studies, GI-PCR panel, and C-difficile PCR were negative.

Urinalysis showed nephrotic range proteinuria. Renal biopsy was performed and revealed class II lupus nephritis. The patient was initiated on pulse steroid therapy and her abdominal pain significantly improved 48 hours after initiation of glucocorticoids. With the biopsy proven class II lupus nephritis, classic radiologic signs, exquisite response to glucocorticoids and absence of infection, the diagnosis of lupus enteritis was made. However the patient’s abdominal pain deteriorated again and was associated with constipation and decreased oral intake. Further evaluation revealed superimposed Small Bowel Obstruction in the setting of opiates and steroids intake. The patient was treated with supportive therapy and tapering off opiates and steroids. Due to worsening abdominal pain and inability to tolerate oral intake for prolonged time, she was initiated on intravenous cyclophosphamide with resolution of her gastrointestinal symptoms.

Discussion: This case highlights the challenges facing clinicians in treating SLE patients with lupus enteritis: abdominal pain in these patients could be due to lupus enteritis and/or other causes; the choice of therapy is dependent on the nature and extent of internal organ involvement, ability to tolerate oral intake, and based on anecdotal data of case reports and case series (cyclophosphamide in this case was given for the enteritis and not the nephritis and it was given intravenously due to inability of intake by mouth); and managing the potential
side effects of therapies.

Marty Paige
Dr. Eric Olson

*Did He Just Have a STEMI?*

Introduction: Coronary artery bypass graft (CABG) pseudoaneurysm is a rare but potentially fatal condition. The most common presenting symptoms are chest pain, shortness of breath and hemoptysis. Given its low incidence yet serious complications, the diagnosis and management of this condition are challenging.

Case: The patient is a 69-year-old man with a remote history of five-vessel CABG who presented with a several-week history of small volume hemoptysis and typical chest pain. Of note, he was hospitalized one-week prior to presentation for Salmonella enteriditis bacteremia, ultimately treated with levofloxacin.

On initial evaluation, he was hypotensive and tachycardic with a 1/6 systolic murmur. Following a positive D-dimer, chest CT angiogram was obtained to evaluate for pulmonary embolism. This showed a large anterior mediastinal mass, concerning for CABG pseudoaneurysm, thymoma or lymphoma. Initial ECG showed ST-segment elevations in the anteroseptal leads, and troponin trend was significant at six hours. He continued to have active chest pain and was initiated on aspirin and heparin due to ischemic changes. MRI confirmed the CABG abnormality, showing a 7.6 x 5 cm pseudoaneurysm with active, contained bleeding. Coronary angiogram was revealing for pseudoaneurysm of the proximal segment of the saphenous vein to obtuse marginal graft with active flow into a dissection flap. There was mass effect on the vein graft supplying the diagonal branch.

Throughout the hospital course, the patient was treated empirically for recent salmonella bacteremia with ceftriaxone, although blood cultures remained negative. He required nitroglycerin infusion to control chest pain with increasing serial troponins. The patient underwent repeat cardiac catheterization to embolize the vein graft pseudoaneurysm.

Conclusion: This case demonstrates an unusual but life-threatening complication of CABG. It is also important to consider mycotic aneurysm in the setting of recent nontyphoidal Salmonella bacteremia. Further ramifications of unrecognized pseudoaneurysm can lead to ischemic complications due to coronary vessel compression, as seen in this case, as well as rupture, cardiac tamponade, and fistula formation. Additional study is required to determine optimal management and outcomes of CABG pseudoaneurysm.

Nicholas McDonald
Dr. Alex Schwegman
Dr. Rajiv Gulati

*Another Acute Coronary Syndrome Admission?*

Introduction: The differential for chest pain is broad and ranges from benign to life-threatening causes. While acute coronary syndrome (ACS) is often considered, it is imperative to maintain a differential so that ACS mimickers are not overlooked.

Case: An 85-year-old woman presented to an outside emergency department with progressive, substernal chest pain which radiated into her arms. This pain originally started two weeks prior, was worse with exertion and relieved by rest. This was associated with dyspnea on exertion. Her pain improved with standing. Over the past two weeks, even small amounts of physical activity would cause her pain to occur. Her past medical history was notable only for stage I, right-sided breast cancer s/p lumpectomy and radiation 7 years prior.

In the emergency department she was found to be hypertensive with systolic pressures >200 mmHg. EKG was notable for ischemic changes in the anterior leads. Serum Cardiac Troponin-T (4th generation) was elevated at 0.01 ng/mL
(outside lab reference ranges unavailable). A CT angiogram was negative for acute aortic dissection but incidentally revealed a heterogeneously enhancing and partially cystic 4.8 cm right adrenal mass (Figure 1). She was given Metoprolol and Captopril with improvement in her blood pressure to 122/63. Given the concern for acute coronary syndrome, she was commenced on intravenous unfractionated heparin and transferred to our institution.

Upon admission, her troponin (4th generation) was undetectable and remained undetectable at 3 hours and 6 hours. While at rest, she remained symptom free and normotensive. However, after using the restroom and bending over to pick up her underwear, she developed recurrent episodes of tachycardia, chest pain, and hypertension >200 mmHg. This was reproducible on the subsequent day whenever she bent down. Plasma metanephrines initially obtained on admission returned elevated with metanephrine levels of 3.4 nmol/L (reference range <0.50) and normetanephrine levels of 7.0 nmol/L (reference range <0.90 nmol/L). Confirmatory urine metanephrines returned highly elevated. MRI obtained for confirmation and surgical planning was consistent with the diagnosis of pheochromocytoma (Figure 2). She underwent alpha blockade with phenoxybenzamine followed by definitive surgical management. At interval follow-up after surgery, she was doing well without further episodes of chest pain.

Conclusion: This case emphasizes the importance of maintaining a broad differential for patients presenting with chest pain. Given her risk factors, description consistent with typical angina (and admission to an ischemic cardiology service), there was significant potential for anchoring bias on the diagnosis of ACS. This case also highlights the importance of utilizing clinical judgement when portions of the history or exam do not fit with your working diagnosis.

| Charlie Meade |
| Dr. Kaiser Lim |

**Tracheobronchomalacia: An Asthma Mimic Hiding in Plain Sight**

Case: A 49 year old female was admitted to the medicine floors with shortness of breath. She carried a chart history of poorly controlled severe persistent asthma, as well as obesity with a BMI of 38.

Described ongoing shortness of breath following recent hospital discharge. She increased use of her home non-invasive ventilation with incomplete symptomatic relief. Denied infectious symptoms and had no known history of heart failure, appearing euvoletic on exam.

In the 6 months prior to admission she had 10 ED visits for shortness of breath, and 2 additional hospital admissions. Discharge diagnoses included asthma, anxiety, and vocal cord dysfunction. Her pulmonary regimen at admission included prednisone 20 mg daily as well as an inhaled long acting beta agonist, high dose inhaled corticosteroids, and a leukotriene receptor antagonist. She had been on oral corticosteroids for most of the prior 2 years.

Non-hypoxic on presentation and arterial blood gas pH 7.46, pCO2 30, PO2 112. Prior blood gases showed similar respiratory alkalosis suggestive of hyperventilation. PFTs taken prior to admission consistently showed no reduction in FEV1/FEC ratio, no evidence of restrictive disease, no gas exchange abnormality.

Given her non-obstructive PFTs and her apparent lack of response to near-maximal asthma therapies, suspicion was high for an asthma mimic. Review of prior CT imaging was suggestive of dynamic large-airway collapse and during hospital admission she underwent limited bronchoscopy. Significant expiratory airway collapse was observed suggestive of tracheobronchomalacia. Her positive pressure support was uptitrated with good response and she was
discharged with a plan for further pulmonology follow up for procedural consideration and to guide de-escalation of asthma therapies.

Tracheobronchomalacia (TBM) involves weakening of the supporting structures of the large airways. Exhalation results in excessive dynamic airway collapse (EDAC). The epidemiology and underlying causes of TBM are poorly characterized but when significant can result in nonspecific respiratory complaints which often mimic more common pulmonary diseases such as asthma. Direct bronchoscopic observation of airway collapse is the current diagnostic gold standard although dynamic CT imaging during inspiratory and expiratory phase can also be useful.

Conclusion: This case highlights the usefulness of maintaining high clinical suspicion for alternative diagnosis in patients with atypical asthma presentations, patients who appear to have severe disease, and patients who are treatment resistant. Non-obstructive PFTs are generally not consistent with severe asthma and a clue to an alternative diagnosis. TBM/EDAC is a known asthma mimic that can sometimes be identified on prior cross sectional imaging. Informative diagnostic testing is available either via bronchoscopy or dedicated inspiratory and expiratory cross sectional imaging. TBM will not respond to traditional asthma therapies and definitive treatment is often procedural.

Marta Michalska-Smith

Undress and Reassess

Introduction: The skin exam is often overlooked during a clinical encounter, yet often serves as an important diagnostic clue in the evaluation of a patient. This case demonstrates the importance of keeping the skin exam in mind during the development of a differential.

Case: 57yr old male presents with fevers, weakness, dysuria and frequency x3 days, frequent falls over the last two weeks and right knee pain. He has a history of bipolar and schizoaffective disorder—family is reporting mental status and memory changes concerning for manic episode. Physical exam is notable for a swollen and warm right knee with limited ROM due to pain, and a few scaly plaques over the extremities and face. Laboratory studies are remarkable for elevated inflammatory markers and a UA suggesting infection. Arthrocentesis does not meet criteria for septic arthritis and knee swelling is presumed to be due to pre-patellar bursitis. He is started on a seven-day course of Bactrim to treat UTI, and discharged to an inpatient psychiatric facility for rapidly declining mental status. He experiences worsening right knee pain during this stay, and he is transferred back to the medicine ward for presumed septic joint. MRI of the right leg reveals changes out of proportion to what would be expected from a joint effusion. A bone scan reveals lytic lesions suspicious for malignancy. CT chest/abdomen/ pelvis is completed revealing three calcified pulmonary nodules. Bone biopsy pathology returns positive for Blastomycoses. Brain MRI is obtained to evaluate for other areas of involvement, and reveals five small areas of abnormal intensity concerning for cerebral blastomycosis. He experiences worsening knee pain and repeat x-ray shows a pathological fracture with underlying osseous lucency. He completes 4wks of IV liposomal amphotericin and a year of PO Variconazole with complete resolution of the disease.

This patient had a complex course with various working diagnoses, starting with UTI and bursitis, to septic joint, to bone tumor, to metastatic malignancy, and finally to disseminated fungal infection. Looking back, his many presentations can be explained by his diagnosis of disseminated blastomycosis, which can affect almost any organ. On review, this patient’s rash first appeared a month before his initial presentation and can be described as multiple, well-demarcated, thickened, scaly keratotic salmon colored plaques consistent with dermatological blastomycosis. It is reasonable to believe that if this patient’s
skin findings had been taken into consideration during one of his many points of contact with the healthcare system, accurate diagnosis could have been expedited and complications such as his pathological fracture and C. Diff infection could have been prevented.

Conclusion: This presentation will examine the importance of the skin exam as well as the most appropriate diagnostic work-up for suspected disseminated fungal infection.

Daphne Moutsoglou  
Dr. Sally Berryman  

**A Case of Acute Diarrhea Due to Ingestion of Salmonella-Tainted Kratom**

Introduction: Kratom (Mitragyna speciosa), is a tropical tree native to Southeast Asia. It has recently gained popularity as a recreational drug due to its opioid and stimulant properties, occurring mainly through the compounds mitragynine and 7-hydroxymitraginine. Its recreational use is banned in 13 countries and 6 states in the US, and for a brief period in 2016, plans to make it a schedule I controlled substance were considered. From January to May of 2018, an outbreak of Salmonella linked to kratom was reported in 41 states that infected 199 people and resulted in 38 hospitalizations.

Case Presentation: A 24 year old male presented to clinic for five days of diarrhea, without fevers, chills, or blood in his stool. He states that he had been chronically using kratom, and that recently, after onset of his diarrhea, he had been using it more often, due to its constipating side effect. Despite increased kratom use, his diarrhea persisted. The patient was afebrile with normal vitals, and physical exam showed non-distended abdomen that was non-tender. Labs showed normal electrolytes, blood counts, thyroid function, and normal liver function. An enteric pathogen panel was obtained that was positive for Salmonella. Due to severity of symptoms, he was treated with a three-day course of ciprofloxacin, 500 mg BID, and his symptoms resolved. Due to recent announcement from the CDC regarding Salmonella contamination of kratom, the patient was advised of the likely source and to stop using kratom.

Discussion: Kratom’s opioid- and stimulant-like effects and ease-to-obtain have resulted in increased use in the US. Knowledge of the recent outbreak of Salmonella linked to kratom allowed us to target it as the likely cause of this patient’s diarrhea. As always, a thorough review of medications, both prescribed and over-the-counter and recreational, are important as well as updated knowledge on current events in order to ensure proper treatment.

Bryan Neth  
Dr. Michael Richter  
Jose Castellanos  
Marc Greenberg  
Dr. Dennis Regan  

**Chronic Lymphopathy: A Case of Recurrent Pleural Effusions, Lymphedema, and Yellow Nails**

Case Presentation: An 86-year-old man presented to the hospital with increasing bilateral lower extremity edema and generalized weakness. His past medical history was significant for atrial fibrillation, recurrent pleural effusions, and Parkinson's disease (PD). The patient’s weakness and edema progressed over the last several months and had been associated with generalized functional decline and increasing dependence in his ADLs. Moreover, he developed increasing dyspnea on exertion over the last several weeks. He had no changes in medications or recent illnesses over this time period. He denied chest pain, headache, abdominal pain, or changes in bowel or bladder. Physical examination was remarkable for 2+ pitting edema in the bilateral lower extremities extending proximal to the knees, discolored nails on his bilateral hands and feet, heart was regular rate and rhythm, and lungs were clear with diminished breath sounds at the bases. CBC and a metabolic panel were within normal limits. CXR revealed bilateral (L>R) pleural effusion without noticeable pulmonary consolidation.

The patient underwent a thoracentesis, which yielded 825cc of exudative fluid
with elevated triglycerides. We next focused on diuresis (6.5L total) with leg wraps to improve lower extremity edema. The patient’s symptoms were likely related to his underlying Yellow Nail Syndrome. The progressive nature of his symptoms over several months was consistent with prior episodes. His functional decline was thought to be secondary to both pleural effusions and edema complicated by the patient’s PD. After symptomatic management, he was discharged to a skilled nursing facility for acute rehabilitation.

Discussion: Yellow Nail Syndrome is a rare disorder characterized by the constellation of lymphedema, pulmonary pathology, and yellow nails. Less than 400 cases have been reported with an estimated prevalence of <1/100,000. Although the cause is unknown, it has been thought to be related to lymphatic dysfunction. Lymphedema most often occurs in the lower extremities and is indistinguishable from primary lymphedema. The pulmonary findings can be diverse. Commonly associated findings include chronic cough, recurrent pneumonias and pleural effusions (95% exudative), and bronchiectasis. Yellow nails are the main clinical manifestation. Yellow Nail Syndrome has no cure. Pulmonary findings and lymphedema are treated symptomatically.

Our patient’s clinical course began with recurrent pleural effusions about 20 years prior to presentation. This necessitated thoracenteses every 6 months for management of dyspnea. About 3 years after his initial pleural effusion our patient began to develop extensive bilateral lower extremity and groin edema. Finally, about 10 years after initial pleural effusion our patient’s nails began to harden and eventually became discolored to a near-yellowish hue with several of his nails falling off and failing to regrow. Yellow Nail Syndrome should be considered in the setting of lymphedema and recurrent pleural effusion, particularly if characteristic nail changes are evident.

| John Ogden  
Dr. Neel Shah  
Dr. Alina Bridges | Under Your Skin: An Atypical Mycobacterium Infection |
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<td>Introduction: A 47 year old male from Dubai with a PMH of ESRD on dialysis presented to the Emergency Department with left eye blindness and diffuse, tender subcutaneous nodules one month after being appropriately treated for a tunneled catheter-related S. epidermidis associated blood stream infection.</td>
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<td>Investigation: Upon gathering further history, he worked as an administrator at a Seaport in Dubai. He had no recent travel history. He was in a monogamous relationship with his wife of 20 years. He did not use IV drugs. Initial concern was for recurrence of his catheter-associated blood stream infection with subsequent endocarditis. Blood cultures were drawn, broad spectrum antibiotics were started, and a trans-esophageal echocardiogram were obtained in addition to basic blood work. His CBC showed no signs of infection with a normal white blood cell count. Trans-esophageal echocardiogram showed no evidence of thrombus, and blood cultures remained negative throughout hospitalization. HIV, hepatitis B and C, and tuberculosis testing were all negative as well. Leukemia and lymphoma phenotyping showed no evidence of a hematologic malignancy. A skin biopsy was ultimately done, revealing suppurative and granulomatous inflammation with acid fast bacilli. Tissue culture from the skin biopsy grew mycobacterium abscessus.</td>
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<td>Conclusions: Mycobacterium abscessus is a non-tuberculous, rapidly growing mycobacterium (RGM) that is found naturally in soil and water worldwide. It has recently emerged as the cause of an increasing number of infections in humans. It typically affects immunocompromised individuals, but not always, as in this case. It causes pulmonary disease, especially in cystic fibrosis, but can also cause bone, skin and soft tissue, and disseminated disease. It should be considered in the differential diagnosis of patients presenting with sporotrichoid skin lesions.</td>
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Emily Olson  
Dr. Jordan Kautz

**Clostridium Difficile and Alcohol: A Cautionary Tale**

Introduction: Clostridium difficile infection (CDI) in alcoholic hepatitis (AH) increases inpatient mortality, length of stay and cost of care. Clinical challenges include that AH patients are often already malnourished, have underlying cirrhosis, and may receive corticosteroids, worsening immunosuppression and further increasing mortality risk from infection.

Case: A 61-year-old female with chronic alcohol use with underlying compensated cirrhosis, two prior CDI several years ago, and Roux-en-Y gastric bypass was transferred to our hospital with an anion gap lactic acidosis and transaminitis consistent with alcoholic hepatitis (MELD score 30). Based on her Maddrey discriminant function (74.5), prednisolone was initiated. The anion gap improved with hydration.

One day later the patient reported frequent loose bowel movements and C difficile stool PCR testing was positive. Based upon CBC, creatinine, lactate, and albumin, Vancomycin 125mg QID was started for non-severe recurrent CDI. On day 7 the Lille score indicated no significant benefit, thus prednisolone was discontinued. The team chose to continue the patient’s proton pump inhibitor due to symptomatic reflux, known varices, and slowly down-trending hemoglobin.

The patient’s stool frequency continued and on hospital day 9 GI consultation recommended dose escalation of vancomycin to 500mg QID and the addition IV metronidazole 500mg q8h. Unfortunately, stool frequency increased further and she developed an oliguric acute kidney injury. With concern for antibiotic induced acute interstitial nephritis compounding hypovolemia, vancomycin and metronidazole were replaced by fidaxomicin with ultimate plan for fecal transplant. Poor nutritional status, as evidenced by vitamin deficiencies and decreasing albumin, further complicated the patient’s fluid status and subsequent management.

Renal injury worsened, requiring hemodialysis. As her bowel movement frequency decreased, she became encephalopathic in the setting of further down-trending hemoglobin. Lactulose, broad spectrum antibiotics, and an octreotide drip were started. Infectious work-up and fecal occult blood testing were negative, and her encephalopathy slowly improved. The patient began to decline dialysis, citing quality of life and was ultimately discharged home on hospice.

Discussion: Even in asymptomatic patients, because of significant risks in AH patients including mortality, an extensive screen should be done for underlying infection. In the appropriate clinical context, this should include CDI testing. Furthermore, risk stratification for CDI is essential to provide patients with appropriate treatment. Although CDI in patients with concurrent alcoholic hepatitis may be classified as non-severe or non-fulminant based on laboratory criteria, due to their high risk of adverse outcomes, cases should be treated as severe. Careful attention should be given to adjunctive medications including corticosteroids and antibiotics as well as to underlying liver disease and nutritional status.

Eloy Ordaya Espinoza  
Dr. Michael Schnaus  
Dr. Joseph Thurn

**Double Trouble: A Unique Case of Hypercalcemia caused by Two Underlying Etiologies**

Introduction: Hypercalcemia is a common condition frequently presenting with abdominal pain, constipation and altered mental status. Some malignancies and granulomatous diseases can produce non-PTH mediated hypercalcemia from calcitriol synthesis by activated macrophages. Herein, we present a case where hypercalcemia was caused by an abdominal diffuse large B-cell lymphoma and
Actinomyces infection.

Case: A 77 years old male with type 2 diabetes mellitus, hypertension and chronic kidney disease, presented in clinic complaining of abdominal pain, constipation and fatigue for 8 months. Also, narrowing stools, weight loss and confusion were noted during the last 3 weeks. He denied fever, melena or hematochezia. Physical exam revealed abdominal masses and laboratory testing showed calcium of 11.5 mg/dL. Complementary testing showed normal PTH (6.3 pg/mL), PTH-rp (17 pg/mL), 25(OH) vitamin D (22 ng/mL), but high 1,25(OH) vitamin D (91 pg/mL, normal range: 20-60 pg/mL). Abdominal CT scan demonstrated mesenteric, pelvic and retroperitoneal adenitis, and thick cecal wall. Follow up colonoscopy revealed an infiltrative cecal mass, with biopsy showing an ulcerated mucosa with abundant Actinomyces organisms without evidence of malignancy. Patient was hospitalized for expedited management.

On admission, vital signs were stable. Physical exam showed poor oral dentition, tender palpable masses in the mid abdomen and right lower quadrant. Patient was somnolent without other neurologic abnormalities. Laboratory testing showed leukocytes 14.5 K/cmm, hemoglobin 11.9 g/dL, MCV 87.1 fL, creatinine 2.3 mg/dL, urea 39 mg/dL, and calcium 13.4 mg/dL (corrected calcium for albumin 14.4 mg/dL). Other blood tests were unremarkable. Repeat abdominal CT showed enlargement of retroperitoneal lymph nodes, the largest of 11.8 cm x 11.9 cm. Normal saline, calcitonin and penicillin G were initiated. During the following days, leukocytosis resolved, and calcium and creatinine improved. Patient underwent FNA of the mid-abdomen mass revealing a diffuse large B-cell lymphoma (DLBCL). Prednisone was started as a pretreatment for DLBCL and therapy for hypercalcemia. Later, patient developed melena with hemoglobin drop to 6.7 mg/dL. Repeat colonoscopy showed an oozing, ulcerated and partially obstructing cecal mass of 10 cm x 5 cm, requiring ileocecral resection and ileostomy. Biopsy of the mass was consistent with DLBCL without evidence of Actinomyces organisms. Penicillin was subsequently discontinued, and chemotherapy was initiated with rituximab and vincristine. The patient was ultimately discharged from the hospital and has completed six rounds of chemotherapy.

Conclusion: Identifying the etiology of hypercalcemia can be challenging to clinicians. We demonstrated a unique case of lymphoma resulting in hypercalcemia that was complicated by Actinomyces infection. Hypercalcemia treatment is based on energetic hydration, calcitonin, bisphosphonates as well as treatment for the underlying disease. Steroids can be useful for the treatment of non-PTH mediated hypercalcemia in patients with lymphoma and granulomatous diseases by decreasing calcitriol production by activated macrophages.
dressings, and fluids to replace insensible losses. Acitretin 25mg was continued every other day. Over the next several days, her WBC continued to rise with a neutrophil predominance. She did not endorse any localizing signs or symptoms of infection. An infectious workup including blood cultures, urine, and imaging was unrevealing except for asymptomatic bacteriuria which was empirically treated with TMP/SMX due to increased incontinence. WBC continued to rise to 33. Liver function tests (LFT’s) were concurrently elevated and rising (ALT 207, AST 249, ALK 417).

Clinically significant LFT elevation in the setting of diffuse pustular psoriasis raised a broad differential diagnosis including: viral infections, drug-induced liver injury, autoimmune syndromes, and obstructive etiologies. Viral serologies were negative for HAV, HBV, HCV, EBV, CMV, HSV, and varicella zoster. Autoimmune studies and ultrasound with Doppler were also unrevealing.

With persistence in her skin care regimen, she began demonstrating a slow, progressive improvement in skin appearance and labs. Several weeks later, she had complete resolution of her rash and normalization of WBC and LFTs. Negative infectious and hepatic workup, along with progressive clinical improvement with supportive skin care and acitretin favors a unifying diagnosis of severe GPP as the cause of her LFT elevation.

Conclusion: GPP is a pustular puzzle because it can present in many different ways, has a wide range of extra-cutaneous manifestations, and has few distinguishing features from acute generalized exanthematous pustulosis (AGEP) aside from the lack of a suspected offending medication. Rising LFT’s are a common sequelae of pustular psoriasis and thought to be caused by neutrophilic cholangitis. Acitretin is a synthetic retinoid used in maintenance treatment for GPP and can also cause elevation of LFT’s further clouding the clinical picture. However, liver biopsies in patients with GPP taking acitretin have not been associated with worsened liver histology. Fortunately, persistence paid off in treatment of this pustular puzzle.

Paul Park

When Oral Hygiene Causes More than Bad Breath: A Case of HACEK Endocarditis

Introduction: Infective endocarditis (IE) is a condition characterized by infection of the endocardial surface of the heart and often refers to infection of one or more heart valves or an intracardiac device. Fastidious growth and culture-negative tendencies of HACEK-group organisms (Haemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, and Kingella kingae) cause a diagnostic and therapeutic challenge for patients with IE.

Case: A 64-year-old man with previous history of MRSA endocarditis s/p mechanical aortic valve and multiple antibiotic allergies (including Steven-Johnson Syndrome) was admitted to the hospital with a 4-day history of poor oral intake, chest pain, nausea, vomiting, and abdominal pain. EKG was normal and labs were remarkable for elevated troponin, creatinine, ESR, and CRP. In addition to lab findings, his physical exam was notable for poor dentition, a systolic murmur, and diffuse mild abdominal tenderness. Blood cultures were obtained and TTE showed new severe aortic insufficiency. Subsequent TEE was suspicious of aortic valve vegetation and he was started on linezolid based on previous sensitivity. Blood cultures returned positive 2 days later and results initially reported as gram-negative rods. Linezolid was discontinued and meropenem was started. However, a morbilliform rash developed and antibiotic was switched to ceftriaxone and linezolid; rash worsened with ceftriaxone so he was started on gentamicin in addition to linezolid. Meanwhile, the microbiology lab was not able to identify the organism. Minnesota Department of Health (MDH) was contacted for help in identifying the organism via MALDI-TOF and 16S rRNA sequencing. Worsening renal function was a serious concern.
with gentamicin but the choice of antibiotics was limited secondary to patient’s severe allergic reactions to multiple antibiotics including vancomycin, piperacillin/tazobactam, and levofloxacin. With rRNA sequencing, MDH was finally able to identify the organism: Cardiobacterium hominis. The patient underwent levofloxacin desensitization since C. hominis was sensitive to fluoroquinolone, and he was discharged on a 6-week course with a close follow-up with Oral Maxillofacial surgery and Infectious Disease.

Conclusion: This case demonstrates the importance of including the HACEK-group organisms as a possible culprit of IE, especially in the setting of slow-growing blood cultures or negative cultures. It also emphasizes the benefits/risks of antibiotic use and choice of treatment for IE in the setting of serious antibiotic allergy and limited choice of antibiotics.

Shruti Patel
Dr. William Archibald
John Welby
Dr. Tariq Azam

Not Just Corn Dogs - Arboviruses at the State Fair

Introduction: West Nile Encephalitis is a difficult diagnosis as infections are most commonly asymptomatic and testing is not always sensitive to the disease. Here, we report a case of fever of unknown origin where prompt evaluation was conducted based on the patient’s symptoms and recent travel exposure.

Case: A 58-year-old male with type 2 diabetes mellitus presented with fever of unknown origin, headaches, chills, and altered mental status after a 10-day camping trip to the Iowa State Fair. On admission, he was disoriented and noted to have a right-sided abducens palsy. The remainder of his examination was unremarkable. Given the concern for meningoencephalitis, he was started on oral doxycycline, intravenous ceftriaxone, vancomycin, ampicillin, and acyclovir without resolution of his symptoms. Lumbar puncture was performed. CSF showed an elevated cell count at 144 with neutrophil predominance, normal glucose, negative gram stain, and elevated protein at 72mg/dL suggestive for a viral etiology. Notably, his WBC remained within normal ranges with no positive cultures in urine, CSF, or blood. CSF West Nile Virus (WNV) PCR and WNV IgG were negative, but CSF WNV IgM antibody was positive. This laboratory finding, in conjunction with his neurologic symptoms, confirmed the diagnosis of West Nile Virus meningoencephalitis.

Fever of unknown origin is a common complaint encountered by internists, and West Nile infection is a rare but important etiology to consider. Recently, there has been an increased incidence of West Nile infections, particularly in the Midwest. Additionally, rising global temperatures are resulting in longer mosquito seasons and broadened geographic distributions.

While more than 75 percent of West Nile infections are asymptomatic, it is important to accurately diagnose patients that have clinical manifestations. From a public health perspective, this is important as it may prompt more aggressive mosquito control and public safety measures. As West Nile meningoencephalitis has no definitive treatment outside of supportive care, diagnosis can prevent aggressive antibiotic exposure for the patient and guide postinfectious rehabilitation. Up to 40% of patients with West Nile virus infection will experience persistent symptoms such as fatigue, impaired memory/concentration, headaches, and myalgias for months to years after the initial infection.

Conclusion: In patients with fevers, headaches, and altered mental status traveling from endemic areas, it is imperative to draw serum IgM and IgG titers for West Nile Virus in addition to the standard meningoencephalitis workup. A positive IgM titer for West Nile Virus in the CSF is superior to PCR for the diagnosis of West Nile virus given that the sensitivity of PCR detection in patients with known infection is ~55% in cerebrospinal fluid and ~10% in blood. Despite this, nucleic acid amplification testing can be useful in
**Can’t Bear to Pee: Xanthogranulomatous Pyelonephritis**

Introduction: Patients with recurrent kidney stones have increased risk of complicated urinary tract infections (UTIs) and pyelonephritis. We treat patients with recurrent, complicated UTIs with antibiotics and by addressing the underlying urinary tract abnormality. Xanthogranulomatous Pyelonephritis is a rare variant of pyelonephritis that needs to be considered, as it is a life threatening condition that requires more aggressive intervention.

Case: We present a case of a 57 year-old female with a past medical history of multiple sclerosis, breast cancer diagnosed in 2015, unprovoked deep vein thrombosis and massive pulmonary embolus in 2015 on chronic anticoagulation with apixiban, peripheral vascular disease, chronic kidney disease stage III, recurrent urinary tract infections, and recurrent kidney stones, who presented to an outside hospital following a fall that resulted in diffuse, persistent lower back pain. Further evaluation discovered gross hematuria without urinary symptoms and an acute kidney injury. A serum creatinine level was elevated to 2.4 mg/dL from her baseline of 1.6 mg/dL. Ceftriaxone was initiated for likely recurrent UTI; however, urine culture was growing <10k E. Coli CFU and blood cultures were negative. A CT abdomen and pelvis with intravenous contrast showed severe left-sided hydronephrosis, with severe left renal cortical thinning and several non-obstructing left renal calyceal calculi measuring up to 12mm in diameter. It also demonstrated no evidence of renal excretion of intravenous contrast material after 20-minute delay indicative of a non-functioning kidney. The findings were suggestive of Xanthogranulomatosus Pyelonephritis. She underwent a left radical open nephrectomy; however, surgery was complicated by unsuccessful kidney mobilization and the procedure was aborted.

She transferred to our institution for continued care and optimization prior to surgery. Urology performed a left radical open nephrectomy. During surgery, it was noted the kidney was densely scarred and adherent. The patient was admitted to the ICU following surgery. She later recovered and was discharged from the hospital with improving renal function.

Discussion: This illustrates the risk of Xanthogranulomatosus Pyelonephritis developing in the setting of recurrent renal stones and UTIs. Xanthogranulomatosus Pyelonephritis is most prevalent among middle-age women with a history of recurrent UTIs. Presentation typically includes flank pain, fever, malaise, and weight loss. Physical exam is occasionally notable for a unilateral renal mass that is palpable on abdominal exam. Blood tests are nonspecific and CT scan is the preferred diagnostic tool. It characteristically shows replacement of renal tissue by rounded, low density, rim enhancing areas, characteristically nicknamed the “Bear Paw Sign”. It should be differentiated from renal cell carcinoma, which can have a similar presentation, and the patient should undergo nephrectomy for definitive treatment.

**Capecitabine Induced Coronary Vasospasms**

Case: A 63-year-old man with a past medical history of locally advanced rectal adenocarcinoma presented to the emergency department with chest pain concerning for acute coronary syndrome. He had several 10 minute episodes of intermittent chest pain over the last 24 hours. These were categorized as painful substernal pressure sensations that would occur spontaneously, and he would become diaphoretic and dyspneic.

Serial ECGs and troponins were negative. His lipid profile, hemoglobin A1C, blood pressure, and exercise stress test were all normal. However, during one
of the episodes of angina, ST elevation on telemetry was observed and sublingual nitroglycerin relieved the pain. Three days prior he had started taking capecitabine for his rectal adenocarcinoma.

Conclusion: Capecitabine is an oral prodrug of 5-fluorouracil (5-FU) which can be used in the treatment for colorectal cancers. While 5-FU induced vasospasms are well known, capecitabine induced cardiotoxicity is less well documented. He was eventually discharged while taking long acting nitrates which helped prevent him from having further episodes of chest pain. Providers should recognize reversible causes of coronary vasospasm to help provide proper treatment and avoid unnecessary diagnostic testing which could be potentially harmful.

**Jorge Reyes-Castro**  
Dr. Gregory Wieland

*The Paradox of the Induced Right to Left Shunt: Paradoxical Brain Embolism Induced by Massive PE with Underlying PFO*

**Introduction:** Altered mental status is the presenting sign for a wide spectrum of diagnoses with a wide array of clinical severity. This clinical vignette details a 36-year-old female presenting to the emergency department with altered mental status, focal neurologic changes, and hypoxia.

**Case Description:** A 36-year-old female without significant past medical history was brought to the emergency department by EMS with altered mental status. On arrival, vital signs were significant for a HR of 150 bpm and SpO2 of 86% on room air. Neurologic exam revealed obtundation and left lateral inferior gaze deviation without midline crossing. CT head with angiography showed multiple infarcts involving the bilateral cerebellar hemispheres, a small focus of acute intraparenchymal hemorrhage, and ventricular dilation consistent with noncommunicating hydrocephalus. TPA was withheld in case decompression craniectomy was required. The patient was intubated for acute hypoxic respiratory failure. Despite 100% FiO2 and PEEP of 12 cmH2O, PaO2 remained 85%. Given persistent hypoxia there was concern for shunt physiology. Bedside cardiac ultrasound showed an enlarged right ventricle, while CT chest with angiography demonstrated extensive pulmonary emboli with right heart strain and possible patent foramen ovale. TTE showed a large right to left shunt at rest and with Valsalva likely due to a PFO. Given the severe cor pulmonale, right-to-left shunt resulting in stroke, and extensive clot burden in the lower extremities, mechanical pulmonary thrombectomy and IVC filter placement were performed. On hospital day 6, an external ventricular drain was placed for relief of hydrocephalus. Over the next several days, the patient was placed on therapeutic heparin, and with continuation of the EVD, her neurologic status improved. She was extubated and doing well neurologically at the time of discharge. A repeat TTE performed nearly 4 weeks following the initial study did not show evidence of right-to-left shunting.

**Discussion:** Our case demonstrates a right-to-left cardiac shunt induced by a massive PE with an underlying PFO. PFOs are common in the general population with a 25-30% prevalence (1). Under the right physiologic conditions, an increase in right-sided pressures can dramatically increase the degree of right-to-left shunting across the PFO, thereby profoundly increasing the possibility of paradoxical emboli (2). In the presence of massive to submassive PE, PFO should be ruled out. This case also demonstrates the need to consider a PFO when patients at low risk cerebrovascular disease present with ischemic stroke.

**References:**  
**Timothy Rinden**

*C-ANCA Associated Aortitis: A Case of Rare Large-Vessel Vasculitis Responding to Rituximab*

Introduction: Anti-neutrophil cytoplasmic antibody (ANCA) associated vasculitis is typically known for small vessel disease. Large-vessel vasculitis most commonly is comprised of Giant Cell Arteritis and Takayasu’s Arteritis. Large vessel involvement in ANCA-associated vasculitis is rare. When noted, outcomes are often poor and can lead to aortic dissection and rupture.

Case Presentation: This case involves a 52 year old male patient who presents with 2 weeks of fevers, night sweats, and chills. He was initially treated for a dental abscess with Clindamycin. The chills resolved, but the patient developed severe retrosternal chest pain that persisted for another week. He ultimately presented to the hospital and a CT chest was obtained. It was notable for a 6 cm ascending aortic aneurysm. There was also significant ascending, aortic arch, and abdominal aortic wall thickening, consistent with inflammatory aortitis. Initial CRP was 26 and ESR was 98. A large pericardial effusion was also noted. Patient reported a history of temporal headaches without temporal artery tenderness on exam. The patient also had petechial rash. Serologic evaluation showed: positive c-ANCA at 1:160, strongly positive PR-3 antibody, positive ANA at 1:80 with speckled pattern, positive rheumatoid factor at 33.8 with negative CCP antibody, and normal IgG4 level.

Discussion: Patient was started on prednisone therapy by his PCP and appeared to be responding well. Patient continued to have elevated inflammatory markers despite prednisone therapy for one year. Methotrexate was tried, but failed. He finally presented to the rheumatology clinic and tocilizumab therapy was tried for possible giant cell arteritis and failed. During this time, his inflammatory markers did not normalize and prednisone therapy remained above 20 mg daily. In order to receive cardiothoracic surgery for the aneurysm, his disease needed to be in remission with a prednisone dosage of less than 20 mg daily. Decision was then made to try rituximab for possible c-ANCA associated aortitis. Patient received 2 cycles of rituximab infusions and inflammatory markers significantly decreased, symptoms resolved, and chronic prednisone dose decreased to 15 mg daily. Patient successfully underwent thoracic aortic aneurysm repair. The biopsy showed aortitis with eosinophilic and lymphocytic infiltration. Patient continues to do well with resolution of symptoms.

Conclusion: This case represents a rare case of ANCA associated aortitis, which responded to rituximab therapy. It highlights the challenges providers face with diagnosing and treating aortitis. The patient required high doses of steroids to treat his aortitis. However, these high doses were preventing him from the needed thoracic aortic aneurysm repair, hence the need for steroid sparing immunomodulatory therapy. Tocilizumab and methotrexate were prescribed on the basis that the patient had giant cell arteritis without clinical improvement. ANCA associated aortitis was finally suspected due to the ANCA positivity and rituximab was prescribed with exquisite clinical response.

**Eileen Russell**  
Dr. Anthony Kashou  
Dr. Thorvardur Halldanarson

*Maybe It’s Not the Immunotherapy: A Case of Metastasis to the Bile Ducts in Colon Cancer*

Introduction: The most common sites of metastasis in colon cancer include liver, thorax, and peritoneum. Liver metastases can cause intrahepatic obstruction and jaundice; extrahepatic biliary obstruction can be caused by metastases adjacent to biliary structures. Metastasis directly to the bile ducts is a far less common cause of biliary obstruction in colon cancer patients.

Case: A 65-year-old male with previously treated BRAF-mutated metastatic colon adenocarcinoma on a clinical trial of pembrolizumab and an anti-LAG3
agent presented to the clinic complaining of acutely worsening epigastric pain. Prior to this therapy, he had received atezolizumab, bevacizumab, and capecitabine on another trial, but his cancer eventually progressed. He had received his first infusion of pembrolizumab and anti-LAG3 therapy one week prior to presentation. A CT of the abdomen performed before the first infusion showed continued enlargement of known bilateral adrenal metastases, unchanged mild porta hepatitis lymphadenopathy, and no biliary ductal dilatation. On laboratory evaluation at presentation, he was found to have ALT 298, AST 189, total bilirubin 8.6, direct bilirubin 6.9, and alkaline phosphatase 402. A right upper quadrant ultrasound was unremarkable. The initial concern was for immunotherapy-mediated hepatitis, and he was started on methylprednisolone at 1 mg/kg/day for this.

His elevation of transaminases, hyperbilirubinemia, and hyper-alkaline phosphatemia persisted. A repeat CT of the abdomen showed interval development of moderate intrahepatic ductal dilatation and partial extrahepatic ductal dilatation. MRCP was performed one week after presentation and demonstrated marked wall thickening and enhancement of the extrahepatic bile ducts with stricturing at the hepatic hilum, moderate to severe intrahepatic ductal dilatation, and lymphadenopathy at the porta hepatis, suspicious for an infectious or inflammatory etiology of stricture due to its development over a short time period. ERCP with EUS showed a stricture in the common hepatic duct with associated wall thickening up to 4 mm and no associated mass. Brushings and fine needle biopsies were collected, and stents were placed into the left and right main hepatic ducts. Biopsies revealed poorly differentiated adenocarcinoma with signet ring cell features, consistent with metastatic disease to the bile ducts rather than inflammatory biliary stricture as a side effect of immunotherapy.

Discussion: Metastasis was not immediately suspected as the cause of biliary obstruction in this patient. Immunotherapy-mediated hepatitis and autoimmune cholangiopathy were at the top of the differential diagnosis, although this would have been unusual timing as the hepatitis associated with anti-PD-1 agents typically appears seven weeks from initiation. BRAF-mutant colon cancers have a unique pattern of spread, with metastases to the peritoneum and distant lymph nodes being more common compared to BRAF wild-type tumors. It is important to consider unusual sites of metastasis in BRAF-mutant colon cancers when the cause for a patient’s presentation is initially unclear.

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**Alexander Ryu**
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**An Atypical Case of Microscopic Colitis Characterized by Severe Abdominal Pain, Cachexia and Visible Endoscopic Lesions**

Introduction: Microscopic colitis (MC) is a common cause of chronic, watery diarrhea, particularly in the elderly, where it accounts for over 20 percent of such cases. Herein, we present an unusual case of MC notable for severe abdominal pain, cachexia and visible endoscopic lesions.

Case: A 67-year-old woman presented with four months of diarrhea leading to a 20 percent weight loss and generalized weakness. Her medical history was unremarkable except for a 52 pack-year, ongoing, smoking history. She suffered from 10 or more watery, non-bloody stools per day, uncorrelated with any specific ingestions. She endorsed occasional nocturnal diarrhea, and had developed episodic severe abdominal pain within the last month. Prior to presentation, she had undergone upper endoscopy and colonoscopy revealing only mild gastritis; no biopsies were obtained. Because of her abdominal pain and anorexia, she had also undergone abdominal CT arteriography that showed no stenoses. On admission, vital signs were unremarkable. Examination was notable for cachexia with a body mass index of 14.5, and mild epigastric tenderness. Investigations were significant for mild leukocytosis and electrolytes suggesting moderate volume depletion.
The patient was rehydrated and underwent colonoscopy. Visual inspection revealed diffuse mild colonic mucosal changes with congestion, edema and loss of vascularity. The terminal ileum featured similar abnormalities. Colon and, notably, ileum biopsies returned diagnostic for MC, collagenous subtype. Stool infectious studies, fecal leukocytes and celiac serologies all were negative. The patient was started on budesonide therapy with significant symptomatic improvement.

Discussion: Microscopic colitis classically presents with chronic, watery diarrhea in the context of a visually normal colon, but with specific histopathologic changes. Diagnosis is by biopsy in the appropriate clinical context. Two histopathologic subtypes exist: lymphocytic colitis is characterized by the presence of numerous intraepithelial lymphocytes, while collagenous colitis is characterized by the presence of a thick subepithelial fibrous band. Symptoms may be self-limited, chronic or recurrent.

Risk factors for MC include smoking (our patient’s main risk factor) and the use of NSAIDS and PPIs, among others. Discontinuation of offending agents is recommended. Differential diagnoses include celiac disease, bile acid diarrhea and inflammatory bowel disease. Budesonide therapy has the strongest evidence base, and is usually administered until symptom remission. Mild cases may benefit from bismuth subsalicylate, antidiarrheals or cholestyramine.

Our patient’s presentation was atypical in severity. Her diarrhea and abdominal pain had led to profound weight loss. Her colonoscopy was also visually abnormal; some evidence suggests that the presence of visible colonoscopic lesions, while uncommon, correlate with heightened inflammation and symptom burden. However, the most novel feature was her small bowel involvement, described only sparingly in the literature.

Conclusion: Microscopic colitis can present with severe symptoms, visible endoscopic lesions and small bowel involvement. Clinical suspicion and colonoscopic biopsies are critical for prompt diagnosis.

Tyler Schmidt
Dr. Alice Gallo De Moraes
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A Case of PD-L1 and CTLA-4 Inhibitor Associated Hypophysitis

Objectives: Review the mechanism of various cancer immunotherapies. Understand the presentation of toxicities associated with cancer immunotherapies

Introduction: Immunotherapy has revolutionized the way various cancers are treated. Immunotherapy involves activating the immune system to more effectively eliminate cancer cells. These medications have found great promise in the treatment of various malignancies, but they carry a risk of various significant side effects. Immune-related adverse events (iRAEs) are toxicities resulting from non-specific activation of the immune system, and can affect almost any organ system. Here, we demonstrate a patient who was undergoing treatment for his malignancy and developed serious complications.

Case History: A 63 year old male was admitted for acute renal failure and altered mental status. His past medical history was significant for stage IV renal cell carcinoma with subsequent IVC thrombus (on Eliquis), Type II Diabetes Mellitus, Hyperlipidemia, Coronary Artery Disease s/p stent placement, meningioma s/p resection and psoriasis. In the ED, his potassium was 7.5 and he was admitted to Cardiology for management of hyperkalemia and ECG monitoring.

On hospital day 2, due to continued hyperkalemia, elevated BUN and anuria he was transferred to the MICU for dialysis. He underwent dialysis on day 2 and day 3 with no improvement in mental status. In the setting of continued altered
mental status and hyperkalemia, there was concern for adrenal insufficiency. On hospital day 4, morning cortisol was low and with an undetectable ACTH he was diagnosed with adrenal insufficiency secondary to hypophysitis from his immune modifying treatment of the metastatic renal cell carcinoma. The patient was started on IV hydrocortisone management 50 mg q6h and improved drastically after 3 doses. He was discharged to the general medicine floor for further management and was subsequent discharged home 5 days later.

Discussion: Immunotherapy has demonstrated great advances in the treatment of cancer. CTLA4 inhibitors (ex: Ipilmumab) enhance T-cell activation amplify T-cell proliferation, as well as promote the generation of memory T-cells, thereby providing a long-term anti tumor response. PD-1 Inhibitors (ex: Nivolumab) function to inhibit the binding of PD-L1 to PD-1 thereby boosting the immune response to these cancer cells. Meta-analysis has shown that iRAEs occur frequently in the setting of cancer immunotherapies with an overall incidence of < 75% with anti-CTLA-4 monotherapy and < 30% from PD-L1 Inhibitors. The most common side effects from using immunotherapies are gastrointestinal (44%), endocrine (6%) and hepatic (5%). Autoimmune hypophysitis is the most frequent endocrine side effect. Hypophysitis has been found to occur in up to 17% of patients on CTLA-4 Inhibitors, usually within the first 8-9 weeks of induction. The side effects of each of the immunotherapies applicable to a patient’s diagnosis should be discussed with the benefits and risks weighed in each.

Ilya Shadrin
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Pulmonary Hypertension and Pericardial Effusions – To Tap or Not To Tap

Background: Pulmonary hypertension (pHTN) is a disease of the pulmonary vasculature that is classified into one of five categories based on its etiology. When a concomitant pericardial effusion is present, this often presents challenges with regards to diagnosis of cardiac tamponade and need for potential pericardiocentesis.

Case: A 78 year-old man with known pHTN secondary to severe interstitial pulmonary fibrosis was sent to the ED from primary care clinic with worsening shortness of breath. He has a history of severe right-sided heart failure, pericardial effusion, and chronic atrial flutter. On admission, he was hypoxic to 84-87% with activity while on his home regimen of 3-4L of nasal cannula oxygen. Physical exam revealed pitting lower extremities edema up to his thighs bilaterally. CT of the chest/abdomen showed increased pericardial effusion, and new ascites. He received initial diuresis with IV furosemide with mild improvement in his respiratory status. Repeat echocardiogram showed worsened right heart failure with a right ventricular systolic pressure of 87 mmHg, small D-shaped left ventricular cavity with extrinsic compression from a severely enlarged right ventricle with poor systolic function, and a moderate-large circumferential pericardial effusion. Standard echo features of tamponade physiology were absent, although it was noted that this can be masked in severe pHTN. The next morning, he desaturated to 62% following a walk but recovered quickly with rest. At that time, he had a narrow pulse pressure of 100/80 and became bradycardic to the low 50s after receiving his morning dose of metoprolol, which he was apparently not taking at home. A rapid response team was called, bedside echocardiogram remained largely unchanged, pulsus paradoxus was checked and was absent. After discussions with Cardiology, decision was made not to pursue pericardiocentesis due to the risk of hemodynamic collapse. He underwent further careful diuresis with near-resolution of his peripheral edema and improvement in respiratory status prior to discharge.

Discussion: Cardiac tamponade is generally a clinical diagnosis. Echocardiogram supports the diagnosis by often showing collapse of the right sided chambers among other signs. This case illustrates several critical aspects
of managing pericardial effusions in the presence of pHTN. First, standard
echocardiographic findings of tamponade physiology may be absent due to
significantly elevated right ventricular systolic pressures. Secondly, due to
extrinsic compression of the left ventricle, these patients are often dependent on
their heart rate to maintain adequate cardiac output, and aggressive rate control
for any co-existing conditions should be avoided. Lastly, decisions regarding
pericardiocentesis should be discussed with Cardiology, as in the setting of
highly elevated right ventricular systolic pressures, removal of pericardial fluid
could promote further expansion of the right ventricle and lead to a left
ventricular collapse and a sudden drop in the cardiac output.

| Andrea Sitek |
| Dr. Kenneth Warrington |
| A Rare Cause of Testicular Pain |
| Introduction: ANCA-associated vasculitis rarely presents with testicular symptoms. Case Description: A 54-year-old man with no significant past medical history developed testicular pain one month prior to presentation. This was followed by daily fevers, night sweats, myalgias and 9 kg weight loss. He was initially evaluated in the outpatient setting and was prescribed antibiotics for presumed epididymitis with no improvement in symptoms. He was subsequently hospitalized, and an extensive infectious workup, including blood cultures and testing for Lyme disease, syphilis, tuberculosis, Legionella, Brucella, West Nile, and HIV was negative. Additional studies included bone marrow biopsy, cross-sectional chest and abdominal imaging, colonoscopy and tumor markers, all of which were unrevealing. He was ultimately discharged with a course of broad spectrum antibiotics and antifungals with arrangements to continue workup as an outpatient. He presented to the ED one week after discharge with ongoing fevers and progression of testicular pain. Laboratory studies revealed significant anemia, leukocytosis, elevated creatinine, hematuria with renal epithelial cells and proteinuria. Testicular ultrasound showed innumerable, small hypoechoic lesions throughout both testes without Doppler flow. CT abdomen/pelvis showed mildly edematous kidneys with stranding. He was admitted to a tertiary care hospital, and Infectious Disease and Rheumatology were consulted. A thorough infectious workup was negative. Markers of inflammation were elevated and c-ANCA was positive at 1:2048, with PR3 antibody of >8 (normal <0.4). In view of progressive kidney injury, he underwent renal biopsy. This showed pauci-immune necrotizing and crescentic glomerulonephritis, consistent with a diagnosis of granulomatosis with polyangiitis (GPA). Treatment consisted of IV methylprednisolone followed by a prednisone taper and rituximab infusions. His renal function improved, and fevers and testicular pain resolved. Discussion: Small vessel vasculitis, like GPA, is a rare cause of testicular pain. Among patients with vasculitis, testicular pain is more often seen with polyarteritis nodosa. Our patient presented primarily with testicular pain as an initial manifestation of systemic small vessel vasculitis, an atypical presentation which resulted in delayed diagnosis and progression to kidney injury. A final diagnosis was made after identifying PR3-ANCA positivity and pursuing renal biopsy. This case highlights that vasculitis should be considered in patients with constitutional symptoms and a persistent inflammatory state. It is also important to consider that vasculitis can cause testicular pain. This can be a difficult diagnosis given the nonspecific sonographic testicular findings. |

| Kevin Song |
| Dr. Yan Bakman |
| Dr. Brian Hanson |
| A Rare Case of Chronic Pain |
| Introduction: Annular pancreas is a congenital anomaly characterized by a ring
of pancreatic tissue partially or completely encircling the second portion of the
duodenum. Whereas it is commonly described in neonates or infants with
gastrointestinal obstructive symptoms, the prevalence in adults is rare and the
presentation variable. Here we describe a rare case of annular pancreas in an
adult patient with chronic abdominal pain.

Case: A 63 year old male with no significant medical history presented with 5
year history of progressive, post-prandial, burning RUQ and epigastric pain
associated with bloating and nonresponsive to PPI. Patient’s previous workup
included an abdominal ultrasound 5 years ago which showed normal gallbladder
without any gallstones and an esophagogastroduodenoscopy (EGD) 3 years ago
without any significant endoscopic or histopathological findings.
On examination, patient appeared well but has lost 10 lbs in the past 3 years.
The abdomen was soft without any tenderness, palpable masses or
organomegaly. Labs were notable for normal bilirubin, amylase and lipase. A
repeated EGD was performed due to the progressive nature of patient’s
symptoms, which showed severe bleeding erosive gastritis and severe
inflammatory nearly obstructed post bulbar duodenitis. CT abdomen was
obtained and showed a ring of pancreatic tissue completely surrounding the 2nd
part of duodenum. Laparotomy confirmed the diagnosis, and the annular
pancreas was resected. On follow-up visit, patient’s epigastric pain resolved.

Discussion: This case demonstrates a rare case of epigastric abdominal pain. In
a retrospective study of annular pancreas, the median age of presentation is 47
years old. Congenital anomalies are rare in adult patients with the most common
being malrotation, duodenal web, and Shatzki ring. Annular pancreas is
associated with neoplasia, in particular pancreatobiliary malignancies. Patient
most commonly present with abdominal pain, dyspepsia, and rarely with
gastrointestinal obstructive symptoms. The gold standard for diagnosis is
operative exploration of duodenum and pancreatic head, however imaging
modalities have played an increasing role in the diagnosis of annular pancreas
such as in our patient. For patients with duodenal obstruction, the procedure of
choice is duodenal bypass.

Christopher Spoke
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Dr. Amy Holbrook
Dr. Omar Abdullahi

Good Catch: Using Point of Care Ultrasound to Diagnose Pulmonary
Embolism during Code Blue

Case: A 45 year-old woman with a history severe anemia (secondary to alpha
thalassemia and iron deficiency anemia), poorly defined clotting disorder (prior
spontaneous subclavian arterial thromboembolism, with recommended lifelong
anticoagulation), HIT (on Arixtra), and insulin dependent DM2 presented to the
emergency department for evaluation of fatigue. In the ED, patient was found to
be tachycardic to 110’s with normal BP, normal oxygen saturations. Labs were
remarkable for WBC 11.4, HBG 7.2, glucose 546, beta hydroxybutyrate 4.6,
and creatinine 1.46. UA with signs of infection. IV fluids, Ceftriaxone, IV PPI,
and IV insulin per DKA protocol were started. The patient had reported non
adherence to insulin and arixtra for previous few weeks. GI was consulted on
admission for concern for GI bleed as hemoglobin reached nadir of 6.2. She was
given 1 unit RBCs and underwent EGD which showed few non-bleeding ulcers
in the gastric antrum. Her hemoglobin remained stable in low 7’s and her
Arixtra was planned to be restarted.

On hospital day 2, she reported feeling lightheaded, with new chest pain and
shortness of breath while getting up to the bathroom. She became hypoxic to
80% and tachycardia to the 130’s. She complained of increasing chest pain.
Patient became hypotensive, with worsening hypoxia despite supplemental
oxygen. Point of care ultrasound (POCUS) was used and cardiac evaluation
revealed a large right ventricle and hyper dynamic left ventricle. She developed
pulseless electrical activity and CPR was initiated. She was defibrillated x 2
with ROSC. Patient was emergently placed on VA ECMO and proceeded to OR
for direct surgical thromboembolectomy. There was a significant clot burden discovered bilaterally in her pulmonary arteries and thromboembolectomy was performed. Post-operatively she remained on VA ECMO and was decannulated on POD #1. She was extubated and was completely neurologically intact. She was discharged from hospital to TCU on hospital day 24.

Discussion: This case illustrates the utility of POCUS during code blue situations. POCUS helped narrow the differential for the cause of patient’s rapid deterioration, and shortened time to initiation of ECMO and immediate thromboembolectomy. CTPE study was not pursued to confirm the diagnosis, given the clinical picture and POCUS evidence for pulmonary embolism as the diagnosis.

Joseph Steffens
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Bidirectional Ventricular Tachycardia in the Setting of Digoxin Toxicity

Introduction: Bidirectional ventricular tachycardia is an uncommon arrhythmia and can present as a manifestation of cardiac glycoside toxicity (1). We present a complex case that demonstrates the development of this arrhythmia in the setting of digoxin toxicity with reportedly “normal” serum digoxin levels.

Case Description: A 61 year old male with a history of alcohol and cocaine dependence presented to the Emergency Department with two weeks of worsening exertional dyspnea and bilateral ankle swelling. He was found to be in atrial fibrillation with rapid ventricular response (RVR). He initially received rate control management with a diltiazem drip. Formal echocardiogram showed an LVEF of 5-10%. On hospital day 2 he had a ventricular fibrillation cardiac arrest. He was given magnesium, bicarbonate, and was defibrillated once before ROSC was achieved. Coronary angiography revealed an LAD occlusion without significant coronary artery disease suggestive of thromboembolic event. Patient underwent mechanical thrombectomy, balloon angioplasty, and stent placement. Because of difficult to control rate and labile blood pressure on hospital day 5, he was loaded with digoxin followed by a daily maintenance dose. On hospital day 10, he developed new episodes of ventricular tachycardia. At that time he had a serum potassium of 4.5 mEq/L, calcium of 9 mg/dL, and a digoxin level of 1.5 ng/mL (therapeutic range for digoxin is 0.4-2.0 ng/mL). EKG obtained showed bidirectional ventricular tachycardia. Digoxin was discontinued and digifab was given on hospital day 11. Soon afterwards the patient spontaneously converted to sinus rhythm.

Discussion: Various clinical presenting features of cardiac glycosides/digoxin toxicity have been known to medicine for centuries (2). The most common cardiac arrhythmia associated with digoxin toxicity is sinus bradycardia (3). Although a less common arrhythmia, bidirectional ventricular tachycardia is a serious arrhythmia that requires emergent intervention. It is demonstrated on electrocardiography with the appearance of alternating left and right axis deviation, as result of the proximity of the arrhythmia exit to the His bundle region. Note that the patient’s serum digoxin level was within therapeutic range which happens to be the case in majority of cardiac glycoside toxicity cases. The patient’s ventricular tachycardia episodes promptly resolved with the administration of digoxin antibodies. This case illustrates another example that clinical features of cardiac glycoside toxicity do not always correlate with serum levels of digoxin.

Yosuf Subat
Dr. Hilary DuBrock

This Dog is Getting on My Nerves

Case: A 60-year-old male with a history of psoriatic arthritis previously on adalimumab underwent uncomplicated left total knee arthroplasty. Postoperatively, the patient’s dog reportedly licked his surgical wounds frequently, and he subsequently developed erythema, pain, and swelling of the left knee. Arthrocentesis revealed purulent fluid and cultures grew Pasteurella multicoda. The patient underwent incision and drainage with prosthetic exchange and was initiated on ceftriaxone, with subsequent improvement of the left knee. However, he then developed visual impairment, dysarthria, and ataxia. This led to progressive confusion, requiring intubation for airway protection. EEG was negative on two occasions, MRI was unremarkable, and lumbar puncture did not demonstrate any evidence of infection. The patient was then transferred to Mayo Clinic for further evaluation and management.

Upon arrival to the medical ICU, the patient was off all sedation, but obtunded and intubated. Physical examination was notable for fever to 38.4 C, tachycardia, absent oculocephalic reflex, anisocoria, and areflexic quadriparesis. Corneal and pupillary reflexes remained intact and left knee did not demonstrate signs of infection. Lumbar puncture demonstrated albuminocytologic dissociation. EMG was consistent with acute demyelinating polyneuropathy. Serum anti-GQ1b antibody was sent and the patient was initiated on intravenous immunoglobulin (IVIG) and completed a five-day course. He later required tracheostomy and PEG tube placement.

Serum anti-GQ1b antibody returned positive at 1:12,800, consistent with diagnosis of Miller-Fisher syndrome, a variant of Guillain-Barre syndrome. Repeat MRI brain demonstrated focal enhancement of cranial nerves III and V bilaterally, with subcortical and periventricular hyperintensities. The patient’s neurological status improved and he was discharged to inpatient rehabilitation. PEG and tracheostomy were subsequently decannulated. Over the next few months, the patient made an excellent recovery and was ambulating independently without significant difficulty.

Discussion: Miller Fisher syndrome (MFS) is a variant of Guillain-Barre syndrome (GBS) typically presenting with ophthalmoplegia, ataxia, and areflexia. The condition is felt to result from post-infectious autoimmune process, resulting in molecular mimicry, and subsequent cross-reactivity to peripheral nerves. Antibodies against GQ1b (ganglioside component of nerve) are positive in 85-90% of cases of MFS and are strongly associated with oculomotor nerve involvement. Course is typically self-limited, but reports suggest improvement with IVIG.

To our knowledge, this is the second reported case of MFS associated with P. multicoda infection. It is felt that there may be molecular mimicry between the lipopolysaccharide of C. jejuni (a common cause of GBS) and GQ1b ganglioside. Pasteurella multicoda is a gram negative coccobacillus which also contains a lipopolysaccharide capsule. Given the rarity of MFS, there is a paucity of data regarding a possible association with P. multicoda.

Benjamin Sudolcan

No Good Deed Goes Unpunished: When Appropriate Treatment has Unintended Consequences

Introduction: Drug-induced Immune Thrombocytopenia (DITP) is a relatively rare cause of thrombocytopenia, with an annual incidence of 10 people per million affected. It is important to have a high index of suspicion when treating a patient with isolated thrombocytopenia without concomitant coagulopathy however, as treatment differs and clinically significant bleeding is more
Case Description: A 60 year old male was admitted directly from the infectious disease clinic 1 week after discharge for an orthopedic hardware infection with Staphylococcus epidermidis, which required a prolonged course of outpatient IV vancomycin and rifampin. He had no other past medical history. During an ID clinic follow up, the patient reported that he had developed red spots on his legs, sores in his mouth, and blood in his stool. His labs were significant for a mild normocytic anemia and marked thrombocytopenia. On admission, the patient was afebrile with normal vital signs. He appeared comfortable and was found to have large bilateral buccal hematomas and multiple ecchymosis on the tongue. He did not have lymphadenopathy or hepatosplenomegaly, but he did have bilateral lower extremity petechiae. The remainder of his physical exam was unremarkable.

His admission labs were notable for a hemoglobin of 9 g/dL, platelets of 20 k/cmm after a platelet transfusion of 2 units (clinic platelet count was 2 k/cmm), and normal white count. His reticulocytes were mildly elevated, and B12 and RBC folate were normal. BMP, INR, LFTs, haptoglobin, and LDH were within normal limits. Peripheral smear did not have evidence of hemolysis or dysplasia. HIV, Hep B, and Hep C serologies were negative. Hematology was consulted and recommended a platelet transfusion threshold of 20 k/cmm, IVIG for 2 daily doses, and holding current antibiotics. The patient’s platelets remained stable over the next two days, and he was discharged on IV daptomycin. His serum was tested for drug-dependent platelet antibodies and was positive for vancomycin; he did not have antibodies against rifampin.

Conclusion: Clues to suggest DITP over primary ITP include initiation of a new medication (especially a known offender) 7-10 days prior to the onset of platelet fall and a nadir platelet count < 20 k/cmm. In addition to stopping the likely offending agent (lab results are generally not received quickly enough to be helpful in the acute setting), IVIG should be given. Corticosteroids (a common mainstay of ITP treatment) are often not effective in DITP. Patients with bleeding more significant than minor purpura should be hospitalized for observation and likely platelet transfusion.

Terin Sytsma
Dr. Alexandra Wolanski-Spinner

Systemic Mastocytosis with an Unusual triad of Chest Pain, Syncope and Bleeding

Case Description: A 69-year-old man with hyperlipidemia presented with months of recurrent chest pain, dyspnea and diaphoresis followed by syncope. Episodes occurred at rest, 1-2 times per month. Electrocardiograms and stress echocardiogram were negative. Coronary angiogram showed severe myocardial bridging in the left anterior descending coronary artery.

He underwent myocardial bridge unroofing. Intra-operatively, he had marked hypotension and an unusual amount of sternal bleeding. The day after surgery, he became profoundly hypotensive (systolic pressure 20mmHg). Emergent bedside thoracotomy for concern for cardiac tamponade revealed no hematoma. There was a considerable soft tissue bleeding but no surgical site bleeding. Despite receiving no heparin products since the initial operation, aPTT was greater than 300 seconds (normal 25-37 seconds). He was given protamine and multiple units of blood products. After stabilization, he was given recombinant factor VII to stop further bleeding. On post-operative day 3, he again developed significant hypotension (mean arterial pressures 30-40mmHg) and was noted to be flushed and diaphoretic. He received high dose pressors, diphenhydramine and steroids. Tryptase was significantly elevated at 143.0 ng/mL (normal <11.5 ng/mL). Antihistamines were initiated. Repeat tryptase 24 hours after symptom resolution remained elevated at 100.0 ng/mL.
Bone marrow biopsy showed systemic mastocytosis involving 5% of marrow cellularity and no concurrent myeloid neoplasm. KIT D816V mutation was positive. The mast cells were spindled and expressed CD 25. The patient was diagnosed with systemic mastocytosis (SM; likely indolent classification). He was placed on certirizine, montelukast, ranitidine and aspirin and was given an epinephrine pen. He has not experienced any further syncopal or bleeding episodes His significant post-operative bleeding and elevated aPTT were likely from mast cell release of heparin.

Discussion: Mastocytosis is a rare disease resulting from accumulation of abnormal mast cells, occurring in cutaneous, localized and systemic forms. SM can range from indolent to rapidly deteriorating. Persistently elevated tryptase is quite specific for SM, and D816V mutation is detectable in the majority of cases.

Symptoms of SM result from abnormal mast cell infiltration and release of mast cell mediators. Our patient presented with syncope, hypotension and bleeding, likely from release of mast cell mediators such as histamine and heparin. Other signs and symptoms include flushing, pruritus, nausea, abdominal pain, anemia, osteoporosis and cognitive difficulties.

Indolent SM is the most common form of SM and has a good prognosis. Initial treatment is typically based on symptom management with histamine blockers, proton pump inhibitors, steroids and epinephrine. In more aggressive forms, multiple chemotherapy agents and stem cell transplant might be considered.

Conclusion: In the evaluation of unexplained anaphylactic reactions, SM should be excluded to prevent life-threatening complications. Syncope may be a presenting symptom, and non-cardiac causes must be considered.

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**Jeremy Taylor**

**Sugar Momma: A Case of Refractory Diabetic Ketoacidosis in a Pregnant Patient**

Case: A 29-year-old G2P0101 female at 32 weeks gestation with history of endometriosis, hypertension, preterm delivery secondary to severe pre-eclampsia, and uncontrolled type I diabetes presented to an OB/GYN clinic with complaints of nausea, vomiting, and weakness. She reported that she was nonadherent with her insulin regimen and that she had lost 15 pounds over 2 weeks.

She was found to be in diabetic ketoacidosis (DKA) with a blood glucose of 258 mg/dL, anion gap of 20 mmol/L, serum bicarbonate of 7 mmol/L, and serum ketones of 6 mmol/L. No infectious cause of her DKA was found. She was given intravenous (IV) fluids and started on an insulin drip. Her anion gap closed, and serum bicarbonate normalized. Her serum ketones also decreased to 0.7 mmol/L but did not normalize. She was transitioned off the insulin drip and started on subcutaneous insulin. However, she continued to have nausea and vomiting. She went back into DKA with a blood glucose of 187 mg/dL, serum bicarbonate of 18 mmol/L, anion gap of 14 mmol/L, and serum ketones of 4.4 mmol/L. She was restarted on an insulin drip and once again her anion gap and bicarbonate normalized. Her serum ketones decreased to 1.8 mmol/L but again did not normalize. She was transitioned off the insulin drip a second time to subcutaneous insulin. She went back into DKA with a blood glucose of 243 mg/dL, anion gap of 20 mmol/L, serum bicarbonate of 9 mmol/L, and serum ketones of 5.6 mmol/L and pH of 7.04. She was placed on an insulin drip and given supplemental IV glucose to increase the amount of IV insulin required to normalize her blood glucose. Her serum ketones finally normalized to 0.2 mmol/L and DKA resolved. She was started on betamethasone and delivered her baby two days afterwards.
Conclusion: This case highlights that pregnancy alters the physiology of the patient, which made the management of her DKA challenging. Pregnancy is a state of accelerated starvation as the fetus is constantly acquiring glucose and therefore predisposes the patient to DKA. Additionally, metabolic changes such as respiratory alkalosis with compensatory excretion of bicarbonate results in a lower buffering capacity of the serum. Hormonal changes such as increases in progesterone, cortisol, insulinase, and human placental lactogen antagonize the action of insulin. This patient’s presentation of DKA was also precipitated by vomiting, which further contributed to her state of starvation. Emesis from any cause is a common precipitating event for DKA in pregnant women. This case also highlights that DKA in pregnancy can occur at lower blood glucose levels rather than higher blood glucose levels seen in nonpregnant patients.

Lauren Thornton

**Emboli causing STEMI in a Patient with Atrial Fibrillation and Mitral Annular Calcification**

Introduction: ST elevation myocardial infarction has a very specific connotation to a physician, immediately bringing to mind the pathophysiology and treatment plan. While these generally hold, it is important to recognize additional etiologies.

Case: The patient is a 78-year-old female with past medical history of CREST scleroderma, hypertension, paroxysmal atrial fibrillation not on anticoagulation who presented to the emergency department with one day history of new onset, non-radiating chest pain. On exam, pulse was regular rate and rhythm, no murmurs were appreciated, and distal pulses were intact. She had no jugular venous distension, lungs were clear to auscultation, and no pedal edema. Her troponins were elevated to 31µg/L initially. Serial electrocardiograms revealed persistent ST elevations and Q waves in anteroseptal leads. The patient was taken for emergent coronary angiography, which revealed culprit lesion of 100% occlusion to the distal segment of the left anterior descending artery. This was reduced to 35% using thrombectomy and percutaneous transluminal coronary angioplasty. Additionally, solid, ivory colored tissue fragments 3-4mm in length were aspirated from the left anterior descending artery, initially appearing atherosclerotic, and were sent for pathology. No other significant coronary artery disease was noted. There were no post-procedure complications. A trans-thoracic echocardiogram the following day revealed akinetic anterior distal septum and apex, with ejection fraction 45%.

Medical management after the event included aspirin, clopidogrel; beta-blocker and future initiation of an ACE-inhibitor. She also went into atrial fibrillation during hospitalization, and was started on amiodarone. Anticoagulation was not initiated due to history of major gastrointestinal bleeding. Pathology returned revealing fibrin with calcifications and acute inflammation consistent with an embolic etiology, not primary plaque rupture related atherothrombotic material. It was suspected that the emboli originated from caseous mitral annular calcification.

Conclusion: This case illustrates an alternative etiology of acute myocardial infarction, in a patient with no known coronary artery disease. Embolic lesions are less common than atherosclerotic cardiovascular disease. Studies estimate that up to 4-7% of patients with acute myocardial infarction did not have atherosclerotic coronary disease, and that coronary artery embolism is the etiology in about 3%. The etiology of the embolic lesion must be considered as well. While the patient was presumed to have embolus from valve, there is additional research indicating that atrial fibrillation is the most common cause of coronary artery embolism. While acute identification of the myocardial infarction is similar, the management and outcomes may be different. This patient continued on dual anti-platelet therapy, however discussions surrounding the need for this were ongoing.
Saphenous Vein Graft Pseudoaneurysm Presenting as Anterior Mediastinal Mass

Introduction: The classic differential diagnosis for anterior mediastinal masses includes thymoma, teratoma, lymphoma, or thyroid mass. Saphenous vein graft pseudoaneurysm (SVGPsA) is a rare, but serious complication following coronary artery bypass grafting (CABG) that could also present as an anterior mediastinal mass. The diagnosis is clinically challenging and requires a high index of suspicion.

Case: A 69-year-old man with a past medical history of coronary artery disease status post five-vessel bypass in 1996 and placement of two drug-eluting stents into the saphenous vein graft (diagonal/ramus artery) following NSTEMI in 2015, hypertension, hyperlipidemia, and recent diagnosis of salmonella bacteremia on ceftriaxone, presented with hemoptysis, intermittent chest pain, and dyspnea. He had an elevated D-dimer (922 ng/mL), lactate (2.33 mmol/L), leukocytosis (10.8x10^9/L) with neutrophilic predominance, and increasing troponins (92 ng/L). EKG showed ST depression in leads V2-V6. Chest CT revealed a large anterior mediastinal mass measuring 4.5x6.2x6 cm occluding one of the coronary artery grafts. Transthoracic echocardiogram demonstrated an ejection fraction of 48% with regional wall motion abnormalities at the base and mid-inferolateral walls. Chest MRI revealed an aortic pseudoaneurysm measuring 7.6x5 cm at the takeoff of the bypass graft with free communication and active to-and-fro flow between the pseudoaneurysm and aorta. Bleeding was contained within the pseudoaneurysm with no active extravasation into the mediastinum. The pseudoaneurysm was thought to be potentially infectious in nature secondary to salmonella bacteremia. In subsequent days, he developed chest pain with dynamic ST changes in leads V1 and V2, and underwent diagnostic coronary angiogram which noted pseudoaneurysm and dissection flap involving the sequential bypass graft to the first obtuse marginal and ramus. An attempt was made to embolize the pseudoaneurysm, but unfortunately this was complicated by an acute left MCA infarction resulting in right hemiparesis with severe aphasia and dysphagia. Due to poor functional status, surgical intervention was not pursued and the patient received conservative management.

Discussion: SVGPsAs are a rare, but potentially fatal complication following CABG with incidence <1%. SVGPsAs tend to occur at the anastomotic site secondary to technical error, infection, partial tear, or pericardial adhesions. Presentation varies widely from asymptomatic mass to complications such as myocardial infarction, rupture, mass effect, heart failure, or fistula. The average time to presentation is 13 years after CABG. The classic triad of chest pain, mediastinal mass, and history of CABG has been suggested to suspect SVG aneurysms. Cardiac MRI has been reported to be an extremely useful diagnostic tool, but often a multimodality imaging approach is needed as demonstrated in this patient. Treatment options include surgical or percutaneous interventions, offering the outcomes, and conservative management. This case highlights the importance of maintaining a high suspicion for new anterior mediastinal masses in patients with history of prior CABG.

Fever of Unknown Origin with an Unexpected Progression

Introduction: Fever of unknown origin (FUO) is a common and complex medical presentation. Various diagnostic protocols exist to help guide a clinician’s investigation. However, attempting to adapt guidelines in the setting of an immunocompromised patient can create a challenging clinical scenario.

Case: A 77-year-old woman with a history of renal transplantation, taking oral
immunosuppressive agents, was admitted to the hospital for further workup of persistent fevers for six weeks. Extensive workup for an etiology, with highest suspicion for an infectious etiology, was undertaken with the assistance of the consultant services. All initial lab testing and imaging conducted returned negative; thus, fitting the clinical entity of classic FUO after three days of investigation. A positron emission tomography CT scan was performed after one week of investigation, showing extensive porta hepatis radiotracer uptake and concerning for central cholangiocarcinoma. Subsequent magnetic resonance cholangiopancreatography revealed non-specific periportal edema. Endoscopic retrograde cholangiopancreatography was performed, with biopsies of a small intraductal polyp negative for malignancy. Notably, daily fevers ceased after ERCP, but this was confounded by antibiotic initiation before ERCP as pre-procedure prophylaxis and continuation despite an absence of purulent material observed after sphincterotomy. Unfortunately, soon after ERCP, the patient’s clinical condition deteriorated with the development of mild hypothermia, hepatic encephalopathy, severe transaminitis, an elevated INR, and worsening renal function studies. All clinical signs suggested acute liver and renal failure of yet-unknown cause. The patient later required transfer to the medical intensive care unit for vasopressor and continuous renal replacement therapies. A liver biopsy was performed, showing extensive confluent hepatocellular necrosis with positive herpes simplex virus (HSV) staining, consistent with HSV hepatitis. Despite rapid initiation of antiviral therapy, the patient expired due to progressive multi-organ failure.

Conclusion: HSV infection is not a common cause of FUO, and if infection is present, it typically progresses rapidly in an immunocompromised host. While more standard viral testing was performed, HSV testing was not conducted in this patient. The case was additionally complicated by erroneous interpretation of her rapid defervescence after ERCP and on antibiotics as suggestive against persistent infection. In light of the various immunosuppressed states that can occur in the modern medical era, this case demonstrates value to the internist in keeping a broad differential at every stage of patient evaluation for FUO. This case also reflects the importance of developing comprehensive diagnostic tools for FUO in immunocompromised states. While there is no standard approach that could be used for every immunocompromised patient, a stepwise tool for evaluating solid-organ transplant patients could be developed to assist in ruling out opportunistic viral infections that are treatable if detected early – such as in this case.

Shravya Vinnakota
Dr. Korosh Sharain

Hidden in Plain Sight: Vertebral Artery Dissection

Introduction: Vertebrobasilar insufficiency often presents with subtle signs that require a high index of suspicion for accurate and timely diagnosis. Clinical features can include local symptoms like headache, neck pain or focal neurological deficits; however, these are often mild or absent.

Case: A 57-year-old male presented with complaints of progressively worsening nausea and vertigo for 3 weeks. This was associated with intermittent headaches recurring every few days. Prior to symptom onset, he slipped on ice and landed on his back, causing a whiplash injury. He denies direct trauma to the head or loss of consciousness. He had no improvement in symptoms with Ibuprofen. His past medical history is significant for sensory seizures secondary to a benign intracranial dysembryoplastic tumor, well-controlled on Levetiracetam for several years. Physical exam revealed right-sided horizontal nystagmus. A non-contrast head CT did not reveal any acute pathology. His nausea and vomiting improved with conservative measures. However, he continued to have intermittent vertigo, therefore, an MRI with angiography of the head and neck was obtained and revealed a right vertebral artery dissection (VAD) and acute infarction of the right cerebellar hemisphere. He was initiated on daily Aspirin and high-intensity statin therapy. CT Angiography of the abdomen/pelvis was
obtained to look for associated conditions and was negative. The patient was discharged with outpatient neurology follow-up. His symptoms gradually resolved and he underwent surveillance imaging after 3 and 6 months, which demonstrated a stable evolving infarct.

Discussion: Spontaneous artery dissections are uncommon but account for 25% of strokes in the young. Etiology can be varied and difficult to identify. Trauma of varying degree, especially in the setting of underlying risk factors, has been linked to VAD. Subsequent ischemia is more common than hemorrhage as it is thought that emboli from thrombus formation at the dissection site are the cause for the ischemic sequelae. Recent literature suggests a 3-step bedside oculomotor exam (HINTS-head impulse test, nystagmus and test of skew) is more sensitive than early MRI for ischemic strokes. Non-contrast head CT has only 16% sensitivity for detecting acute ischemia. The diagnosis is made by neuroimaging with CT angiography or MRI with angiography of the head and neck. In the setting of ischemia, thrombolytics are indicated in the initial 4.5-6 hours. Additionally, antithrombotic therapy with antiplatelet/anticoagulant drugs is recommended for at least 6 months. Endovascular interventions or surgery are considered for patients with subarachnoid hemorrhage or recurrent ischemic symptoms while on antithrombotic therapy. Secondary preventive measures should target cardiovascular risk factors. For an internist, it is crucial to consider vascular dissections in the differential for headache and vertigo in a young adult, especially with any trauma to the head.

Elida Voth
Dr. Alex Kimbrough
Dr. Jason Szostek

A Case of Abdominal Pain and Worsening Confusion in a 76-Year-Old Female

Introduction: Stercoral colitis is a rare complication of chronic constipation with an estimated 200 cases reported in the literature. The hypothesized pathogenesis is that fecal impaction exerts pressure on the colonic wall and impairs transmural perfusion causing ischemia. This acute condition can rapidly progress to bowel perforation and hemodynamic instability if not promptly recognized. Clinically, stercoral colitis may present as an acute abdomen, however, the variability in physical exam and laboratory findings make it a diagnostic challenge. We describe a case of an elderly woman presenting with abdominal pain and worsening confusion in the setting of chronic constipation.

Case: A 76-year-old female presented to the emergency department (ED) with abdominal pain and worsening confusion. She had a past medical history of dementia, coronary artery disease, paroxysmal atrial fibrillation, and type 2 diabetes mellitus. Two weeks prior, she presented to the ED for worsening confusion, falls and diarrhea. Physical exam revealed lower abdominal tenderness. A CT abdomen/pelvis at that time showed a large amount of stool in the colon and rectal distention. She was discharged from the ED and instructed to increase her home bowel regimen. On the day of admission, which was 11 days later, the patient returned to the ED with copious diarrhea, several days of decreased oral intake, and a fall at her skilled nursing facility.

In the ED, the patient appeared lethargic and disoriented. On physical exam, the abdomen was soft, diffusely tender to palpation in all four quadrants, and without rebound tenderness. Rectal exam revealed copious brown stool from the rectum and soft brown stool in vault. Repeat CT abdomen/pelvis demonstrated large volume stool in the rectosigmoid colon with diffuse circumferential wall thickening, new surrounding fat stranding and vascular engorgement, and new mild mesenteric edema, all of which supported the diagnosis of stercoral colitis. Labs were significant for leukocytosis with neutrophilic shift, elevated lactate, and acute kidney injury.

Several hours after admission to medicine, there was an acute change in the patient’s abdominal exam with exquisite tenderness to palpation and new rebound tenderness. General surgery was consulted, and the patient was taken
to the operating room. Abdominal exploration revealed a retroperitoneal bowel perforation due to stercoral ulcer. Unfortunately, the patient passed away several days later due to complications of the perforation.

Conclusion: We report the case of a patient with chronic constipation which progressed to stercoral colitis and eventually bowel perforation. This case highlights the importance of a rectal exam and close outpatient follow-up in a patient that presents with lower abdominal tenderness and excessive stool burden on imaging. Lastly, this case reinforces the utility of CT scan and the high degree of clinical suspicion necessary in order to diagnosis stercoral colitis.

Jessalyn Weaver  
Dr. Lynn Weber  
Dr. Katie Won

An Unusual Skin Reaction in a Patient with Non-Small Cell Lung Cancer

Introduction: Systemic chemotherapy is associated with numerous and potentially severe adverse effects. Taxanes are a class of chemotherapeutic agents commonly used to treat a wide variety of cancers including breast cancer, non-small cell lung cancer, and ovarian cancer, among others. There are numerous well-described side effects associated with taxane chemotherapy including hypersensitivity reactions, neutropenia, alopecia, gastrointestinal upset, and neuropathy. This case illustrates an unusual reaction to paclitaxel chemotherapy.

Case Description: Mrs. B is a 78-year-old female with past medical history significant for hypertension, type II diabetes mellitus, and Stage IIB non-small cell lung cancer with prior left lower lobectomy in 2005. In 2016, she presented with mediastinal and hilar lymphadenopathy and a new right infrahilar soft tissue mass on imaging concerning for recurrent disease. Fine needle aspiration of the mass showed adenocarcinoma consistent with a lung primary. She received radiation therapy with concurrent weekly low dose carboplatin/paclitaxel chemotherapy. Her first cycle was given through a peripheral IV in the right antecubital fossa, with subsequent cycles given through a port-a-cath. Shortly after receiving the second dose of her chemotherapy, she returned to clinic with throbbing pain in her right arm with associated skin discoloration and itching.

On physical exam there was erythema with scattered ecchymoses extending from the wrist to the mid upper arm. There were scattered excoriations from scratching without open or draining lesions. An ultrasound of the right upper extremity showed no evidence of deep vein thrombosis. Benadryl and hydrocortisone cream were prescribed to help alleviate symptoms, but were not effective. The reaction was initially thought to be a chemical phlebitis related to taxane infiltration. The localized painful “cellulitis”, skin changes, and associated severe local pain eventually improved over several months with supportive therapies alone. Single agent carboplatin was subsequently used to complete her chemotherapy.

Discussion: There are many well-described adverse effects of taxane chemotherapy. However, classification of these agents as either vesicants or irritants is poorly defined. The most common dermatologic side effects associated with taxane chemotherapy include hypersensitivity reactions (manifested by urticarial, morbilliform rash, flushing and angioedema); Dorsal Hand-foot syndrome (scaly erythematous lesions located on the dorsal aspects of hands and feet); nail toxicities; and chemotherapy-induced alopecia. This case illustrates a relatively uncommon cutaneous reaction to paclitaxel infusion, namely a delayed recall hypersensitivity reaction, potentially related to prior extravasation of this agent into the soft tissues. Most of these reactions occur on the first or second exposure to these agents. The mechanism of taxane-induced hypersensitivity reactions has not been well defined, although an IgE-mediated mechanism involving mast cell/basophil activation has been postulated. Despite this uncommon complication of taxane administration, there are presently no data to support taxane administration through central lines versus peripheral administration.

Jason Wiederin

Malignant Pleural Effusion in Multiple Myeloma; A Rare Presentation with
### Grim Prognosis

**Introduction:** Malignant pleural effusions in multiple myeloma are uncommon. Even rarer are primary myelomatous pleural effusions which result from pleural invasion by plasmacytomas via the pleura, chest wall, or lungs. These are associated with high-risk disease and a poor prognosis.

**Case Presentation:** Patient is a 61 y.o. male with history of multiple myeloma with metastasis to the bone and brain complicated by anemia, thrombocytopenia, and renal failure. Presenting symptoms improved after therapeutic thoracentesis with removal of 1.6 L of bloody/cloudy fluid. Pleural fluid analysis revealed plasma cell dyscrasia-malignancy with kappa restricted atypical plasma cells consistent with myelomatous pleural effusion.

**Discussion:** While the incidence of pleural effusion in multiple myeloma is as high as 6%, the incidence of a primary malignant myelomatous effusion is much less common, <1%. Typically, the pleural effusions are from typical offenders such as decompensated heart failure, renal failure, and hypoalbuminemia. A primary malignant effusion in the setting of multiple myeloma is due to direct extension of myelomatous disease into the pleura. It is important to differentiate primary malignant effusions from secondary pleural effusion as the former are associated with worse prognosis and poor overall survival.

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### Kathleen Wilcox

**A "Minor" Illness, a Major Complication: Acute Rheumatic Fever**

**Introduction:** Though uncommon in the United States, recognition of Acute Rheumatic Fever is essential; if left untreated, it can progress to rheumatic heart disease. Worldwide, rheumatic heart disease is the leading cause of cardiovascular death during the first 5 decades of life.

**Case:** The patient is a young, previously healthy man who presented to the ED with one day of chest pain, after three days of fever, fatigue, nausea, and sore throat. On physical exam the patient had a low fever, tachycardia, and tachypnea, and was found to have erythematous and enlarged tonsils with exudate. Lab work revealed elevated and rising troponins, an elevated sedimentation rate and C-reactive protein. A transthoracic echocardiogram showed decreased left ventricular ejection fraction. The patient was taken to the cath lab, and found to have normal coronary arteries. Subsequently on admission, a urine drug screen was negative, and a rapid strep test was positive for streptococcal antigen. He was diagnosed with acute rheumatic fever, and initiated on penicillin therapy, with a planned course of monthly penicillin G injections for 10 years. He was counseled to avoid strenuous sports for the duration of therapy.

**Discussion:** Acute rheumatic fever was once much more prevalent in the United States. The decreased incidence in recent decades can partly be attributed to improvements in public health and hygiene, but also is largely a factor of the unexplained proportional decrease of rheumatogenic strains of group A streptococcus. Several countries of the world, notably China and India have a high incidence of acute rheumatic fever, and a corresponding higher prevalence of rheumatic heart disease.

**Conclusion:** Though certainly a less common diagnosis in the United States, our patient was from the Midwest and had not traveled outside the country recently. This case serves as a reminder that clinicians must remain vigilant for uncommon diagnoses such as acute rheumatic fever, where initiation of proper therapy can prevent significant morbidity.

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### Vignesh Williams Palaniappan

**Sweet Peas: Ketoacidosis in the Setting of SGLT-2 Inhibition**

**Case:** A 52-year-old female with an 11-year history of Type II Diabetes Mellitus (DM) presented to a local emergency department with a 2-day history of nausea, vomiting, fatigue, and subjective fevers. She had returned from a work trip 2 days prior. The patient had not taken her prescribed insulin for a few days prior to presentation initially due to a hectic work schedule, and then due to her nausea. Other medications included aspirin, atorvastatin, metformin, and canagliflozin - an SGLT-2 inhibitor (SGLT2i).
On presentation, she was somnolent but oriented and notably tachypneic with RR of 40. Labs returned with a serum bicarbonate that was undetectable and a blood glucose of 230 mg/dl. Arterial pH was 6.88 with a pCO2 of 9. Her anion gap was 24. Salicylate and ethanol levels were negative and lactate was within normal limits. Beta-hydroxy butyrate returned at 9.8 mmol/L (normal <0.4 mmol/L). A diagnosis of euglycemic diabetic ketoacidosis (euDKA) likely associated with SGLT-2 inhibition was made and she was triaged to the medical ICU. Insulin drip was started at 0.1U/kg of actual body weight and she was maintained on a dextrose-10% drip to preserve a blood glucose in the 150-250 mg/dl range. After ~12 hours of treatment, her anion gap closed and her serum ketone levels normalized, with concomitant resolution of her acidosis and compensatory tachypnea. She was switched to subcutaneous insulin, transferred to a general ward, and was discharged home a few days later.

EuDKA was initially described in Type I Diabetic patients in the 1970s and defined as DKA occurring in the presence of a serum glucose of <300 mg/dl. In the interim ~40 years, further triggers have been identified with varying proposed pathophysiologies. SGLT2i’s are an increasingly prescribed class of medication for Type II DM as they do not cause hypoglycemia and are associated with reduction in cardiovascular risk. However, it has been posited that the persistent glucosuria induced by SGLT2i causes an increase in baseline ketogenesis that can tip into ketoacidosis during stressful episodes or with decrease in insulin dosing. A recent study reported 105 cases of euDKA where SGLT2i was thought to contribute to the presentation.

Conclusion: Overall, this case highlights the importance of considering ketoacidosis in patients with any form of diabetes. While uncommon, SGLT-2 inhibition can lead to euDKA. Prompt recognition of this life-threatening illness is challenging as normal or mildly elevated glucose levels often lead clinicians to alternative diagnoses. A thorough review of the patient’s risk factors as well as checking serum ketones in all critically ill patients with diabetes and increased anion-gap metabolic acidosis are essential in timely diagnosis and management of this critical illness.

Katrina Williamson  
Dr. Kimberly Johnson  
Dr. Garvan Kane

*Headache, Falls, and Weakness: Manifestations of Coccidioidomycosis Meningitis in an Immunocompetent Individual*

Introduction: Coccidioidomycosis is an important diagnosis to consider in those with a history of travel to the southwestern United States, Central, or South America. Primary infection frequently goes unrecognized with 60% of people being asymptomatic and the remaining presenting similarly to community-acquired pneumonia. Dissemination to the meninges should be a complication considered in all infected patients, even in those without an underlying immune compromising state.

Case Description: A 74-year-old man with a history of hypertension and CABG, presented to the emergency department for recurrent falls and progressive weakness. The patient had been diagnosed eight months prior with pulmonary coccidioidomycosis not requiring therapy. Six months following this, he presented with fevers, headache, lethargy, and confusion. CSF analysis was remarkable for a cell count elevated to 320 with a lymphocytic predominance and positive Coccidioides antibody and PCR, consistent with coccidioidal meningitis. Brain MRI/MRA showed enhancement of multiple cisterns and intracranial arterial vessel walls with punctate acute frontal and insular infarcts suggesting meningitis with associated vasculitis without evidence of aneurysmal disease. The patient was started on oral fluconazole with symptomatic improvement. The patient now presented two months later with reported falls at home preceded by staring and followed by 10-15 minutes of confusion. On examination, he was found to have subtle new left sided ataxia. Initial labs were largely unremarkable. EEG revealed diffuse cerebral dysfunction with maximal involvement of the left centroparieto-temporal region. Repeat lumbar puncture revealed a decreased cell count and negative Coccidioides PCR, consistent with a response to anti-fungal therapy. However, brain MRI demonstrated new acute lacunar infarct of the right internal capsule and subsequent MRA showed multi-
focal non-occlusive arterial narrowing, concerning for cerebral vasculitis. The patient was discharged on levetiracetam for seizure prophylaxis and a 4-week dexamethasone taper for vasculitis associated with his coccidioidomycosis meningitis.

Discussion: Coccidioidal meningitis occurs in an estimated less than 0.1% of those infected. Patients present with indolent, nonspecific symptoms—most commonly headache, making the diagnosis of coccidioidal meningitis challenging. However, without treatment, 95% of patients die within two years, making identification imperative. The diagnosis is made through CSF analysis—with the identification of CSF antibodies, coccidioidal antigen, or positive culture. Oral fluconazole is first-line therapy and should be continued lifelong to avoid relapse. New neurologic deficits identified in those on therapy should raise immediate concern for drug failure or complication of coccidioidal meningitis such as hydrocephalus, syrinx, abscess, or cerebral vasculitis. Repeat lumbar puncture and MRI/MRA help distinguish between these and guide appropriate therapy. Treatment failure warrants an immediate change in antifungal therapy. Evidence for treating concomitant vasculitis is limited, but steroids can be considered and may reduce subsequent cerebrovascular events.

Emily Witsberger
Dr. Bassim El-Sabawi
Dr. Ilya Shadrin
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| Case Description: A 30-year-old pregnant female at 16 weeks gestation (GSP2022) presented to the emergency department (ED) following a witnessed syncopal episode and subsequent four-hour history of chest discomfort and palpitations. Her heart rate in the ED was greater than 200 beats per minute (bpm). Basic labs were unremarkable, and a urine drug screen was negative. An electrocardiogram (ECG) obtained demonstrated narrow-complex tachycardia interpreted as sinus rhythm with supraventricular tachycardia. However, her tachycardia persisted despite vagal maneuvers, treatment with three boluses of intravenous (IV) adenosine, and two attempts for synchronized cardioversion. Her heart rate improved somewhat after receiving IV diltiazem, and she was transferred to an inpatient service. A repeat ECG then demonstrated atrial fibrillation (AF) with rapid ventricular response (RVR). IV fluids provided appropriate blood pressure support. She was given another diltiazem bolus and placed on a diltiazem drip with further heart rate improvement to 110-120 bpm. However, bedside telemetry readings still demonstrated AF with RVR. Given the teratogenicity of most antiarrhythmic agents, procainamide or synchronized shock were noted to be two of the best options for cardioversion. However, she ended up converting into normal sinus rhythm spontaneously about 12 hours after initial presentation. In retrospect, prior ECGs revealed a likely longstanding history of paroxysmal AF. A plan was developed for management of any potential future episodes of AF with RVR, and she was able to be discharged home. Prior to discharge, reassuring fetal heart tones were obtained.

Discussion: Multiple cardiovascular changes occur in women during pregnancy that predispose to arrhythmogenesis, including women without predisposing risk factors. While the tenets of managing AF in pregnancy are the same as those in non-pregnant women, faster intervention is necessary, and care must be taken to avoid potentially teratogenic medications. Secondary causes of AF should be evaluated including structural or valvular heart disease, rheumatic heart disease, alcohol use, electrolyte imbalance, stimulants, and hyperthyroidism.

Conclusion: Addressing rate control in pregnant patients with AF is often first done with either digoxin or sotalol. Other moderately safe rate-controlling medications include metoprolol, atenolol, and diltiazem. Rhythm control options include quinidine, procainamide, sotalol, flecainide, and propafenone. Of course, each drug carries its own host of side effects, which must be weighed on a case-by-case basis. Of note, synchronized cardioversion remains one method of rhythm control that has been performed safely during all trimesters of pregnancy. If rate has been controlled and the patient is hemodynamically stable, it is often possible to wait up to 24 hours before proceeding with rhythm.
control in case of spontaneous conversion to sinus rhythm. Finally, thromboembolic prophylaxis is recommended for all pregnant patients with AF except those with low thromboembolic risk, namely young individuals without clinical or echocardiographic evidence of cardiopulmonary disease.

Mysterious Linear Filling Defect of the Common Bile Duct – A Case Report of Suspected Biliary Liver Fluke

Case: A 39-year-old female immigrant from Myanmar with chronic hepatitis B presented to clinic for follow up and was found to have an incidental bile duct dilation on the screening right upper quadrant (RUQ) ultrasound for hepatitis B. The patient was born in Myanmar. She lived in Thai refugee camps for ten years prior to immigrating to the United States. She had completed the routine pre-departure physical exam and did not receive pre-departure treatment for intestinal parasites.

On presentation to clinic, she denied abdominal pain or changes in bowel habits. On physical examination, she appeared well, with no scleral icterus and no RUQ abdominal tenderness. Previous laboratory work included liver function tests which were within normal limits, stool ova and parasite exams, schistosoma IgG antibody and strongyloides IgG antibody which were negative. Hepatitis B serologic studies were: HbsAg positive, anti-Hbs negative, and Hepatitis B core Ab positive. Eosinophil count was 171 cell/mm3 (3%).

Abdominal ultrasound for cancer screening was done and found linear echogenicity without bile duct dilatation. Four years later, the ultrasound was repeated with unchanged linear echogenicity. There was no bile duct dilatation on the follow up ultrasound, but 9 months later, there was a mild common bile duct dilatation of 11 mm. The magnetic resonance cholangiopancreatography obtained 7 months later identified intrahepatic and extrahepatic bile duct dilatation up to level of ampulla of Vater. No obstructive lesion or stone was found. There were also small, calcified granulomas along the hepatic surface laterally.

Given the long history of asymptomatic bile duct dilatation without a clear etiology and the patient’s country of origin and travel history, liver fluke infection was the most likely diagnosis. The linear echogenicity likely represents calcification at the site of previous liver fluke infection. The patient was administered empiric oral praziquantel treatment. In this patient, no constitutional symptom and the condition was present for several years. The diagnosis of cholangiocarcinoma was unlikely.

Discussion: Although most patients with liver fluke infection are asymptomatic, the use of ultrasound has become an important method for establishing a diagnosis. Several ultrasound findings are suggestive of liver fluke infection, including hepatomegaly, periductal fibrosis, presence of gallstones, biliary sludge, intrahepatic duct stone, and poor contraction of gallbladder.

Conclusion: Liver fluke infection is related to dietary habit of eating raw fresh water fish. Several studies have found that liver fluke infection due to both Opisthorchis viverrini (prevalent in Southeast Asia) and Clonorchis sinensis (prevalent in East Asia) are strongly associated with cholangiocarcinoma. The liver fluke is hypothesized to explain the high prevalence of cholangiocarcinoma in Khon Kaen, Thailand. The Centers for Disease Control and Prevention recommends preemptive treatment with antiparasitic treatment for immigrants based on their country of origin.

A Case of PTLD

Introduction: Post transplant lymphoproliferative disorder (PTLD) is a recognized complication to solid organ transplant recipients. 10-15% of all adult solid organ transplants develop PTLD in 30-40months. The incidence of PTLD is about 1-2.3% in renal transplants but is reported with higher incidence in pediatrics due to EBV infection. Our case is unique as PTLD was diagnosed in a patient with a 24 years kidney transplant history in the setting of pulmonary histoplasmosis.

Case: A 74year old caucasian male presented with vomiting, fatigue, diarrhea
and cough to the E.D. His past medical history was significant for ESRD s/p living donor renal transplant in 1994, pulmonary histoplasmosis, HTN, type 2 Diabetes Mellitus, hyperlipidemia and osteoporosis. On Azathioprine, prednisone, lisinopril and itraconazole. Physical examination was benign except for fever of 39.3 C. CT-CAP showed an increase in size of several pulmonary solid masses with new innumerable hypoattenuating liver lesions. Primary work-up negative including bacterial and fungal blood cultures, AFB blood cultures, cryptococcal ag, BDG, galactomannan, histo serum antigen. Urine histoplasma antigen at 0.68. Itraconazole was changed to Amphotericin B and lisinopril was held for concern of interference with azathioprine metabolism.

7 months prior admission he had presented with constitutional symptoms, CT findings of a 1.8 cm Right lower lobe nodule with enlarged hilar & subcarinal Lymph nodes; positive BAL sputum culture, fungal blood culture, Histoplasma antibodies, urine histoplasma antigen and was diagnosed with pulmonary histoplasmosis. Azathioprine dose was reduced. Received 2 weeks of Amphotericin B then transitioned to itraconazole. EBV PCR 1,810 copies/ml. Repeat CT after 1 month showed improvement, urine histoplasma antigen was downtrending. Repeat EBV results were undetectable. Day 2 of admission, labs were noticeable for HgB 7.7, PLT 266, WBC 3.78, Ferritin 3,507. Prelim Lung biopsy suggestive of necrotizing granulomatous inflammation, BAL and bone marrow biopsy were non diagnostic. H-Score suggested HLH probability of <10%. EBV PCR 363,000 copies/mL. Finalized Lung biopsy was consistent with Polymorphic PTLD & positive to EBV-encoded RNA in situ hybridization. Liver biopsy noted for B-cell expansion. AZA was held and Rituximab was started. Repeat CT after 2 doses of Rituximab with small decrease in size of lung masses but increase in liver lesions & new Psoas muscle lesion. Due to febrile episodes R-CHOP chemotherapy was initiated. He was discharged home on itraconazole, bactrim, levofloxacin, vancomycin, valgancyclovir. EBV has been undetectable for 5 months.

Discussion: This case illustrates a unique case of polymorphic PTLD in a patient with pulmonary histoplasmosis and Kidney transplant. Given the role of EBV in the pathogenesis of PTLD, providers should have a high suspicion to rule out Infectious causes. Concerns were raised that the addition of lisinopril to his medication regimen may have reduced Azathioprine clearance causing over Immunosuppression and leading to EBV reactivation and PTLD. Also emphasizes reduction of Immunosuppression.

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Fever Without a Cause: An Unusual Case of Fevers and Hearing Loss

Introduction: Fever of unknown origin was originally defined as a fever of 38.3 C or greater on several occasions for at least three weeks without a clear source after one week of inpatient evaluation. While no longer requiring evaluation to take place inpatient, patients with unexplained fever can present significant diagnostic challenge. Leading etiologies include systemic rheumatic disease, infection, and malignancy.

Case Presentation: A 35 year-old female with a history of rheumatoid arthritis on prednisone and rituximab presents for evaluation of recurrent fevers. Her symptoms started 6 months prior when she began experiencing a decline in functioning along with a 38 pound weight loss. Other symptoms include dyspnea on exertion, cough, lower extremity edema, and diffuse joint swelling. She had recurrent fevers starting 4 months ago. She was evaluated at an outside facility and was ultimately diagnosed with viral meningitis. She also had two episodes of pancreatitis without a known cause. Around this time, she experienced progressive, complete sensorineural hearing loss. Laboratory studies included complete blood count showed lymphopenia (0.56 x 109 cells/L) without CD20 cells (0%), elevated C-reactive protein (23.5 mg/L), elevated hemoglobin A1c (7.1%), elevated cyclic citrullinated peptide antibody (186.1 units), negative antinuclear antibody (less than 1:80), hypogammaglobulinemia including immunoglobulin A (57 mg/dL), immunoglobulin M (27 mg/dL), and immunoglobulin G (650 mg/dL). Lumbar puncture was performed showing elevated total nucleated cells (35 cells/mcL),
elevated protein (83 mg/dL), normal glucose (40 mg/dL), and negative paraneoplastic autoantibody panel. Further infectious workup was also negative including human immunodeficiency virus screening, hepatitis B serology, hepatitis C antibody, Histoplasma antibody, Blastomyces antibody, Sporothrix antibody, (1,3)-beta-D-glucan assay, interferon-gamma release assay, JC virus DNA, and blood and cerebral spinal fluid cultures for bacteria, fungi, and mycobacterium. Positron emission tomography was performed, showing diffuse inflammation throughout nearly all soft tissues and organs. A punch and muscle biopsy was performed on her left lateral thigh. Punch biopsy pathology showed septal edema and lobular panniculitis with necrosis and chronic lymphohistiocytic inflammation. Muscle biopsy showed inflammatory muscle disease. Enterovirus PCR of the muscle biopsy was positive. The patient was started on intravenous immunoglobulin G for disseminated enterovirus, with symptomatic improvement.

Discussion: With advances in technology and diagnostic approach, greater numbers of patients with fever of unknown origin are able to be successfully diagnosed with their underlying disorder. A complete history and physical is the first and most essential step in evaluation. Next is a full laboratory and radiographic workup, particularly driven by features in the history. In patients who remain without a diagnosis, positron electron tomography can also be helpful in localizing pathology for more focused workup. For patient with disseminated enterovirus, there is no established standard therapy but intravenous immunoglobulin G may have benefit.

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Ambulatory Care with a Vodka Twist
Introduction: It is estimated that 2-9% of ambulatory patients have alcohol dependence. Alcohol withdrawal can occur in these patients 6-24 hours after the last drink. Here we present a case of an ambulatory gentleman who was found to be in acute alcohol withdrawal.

Case: A 29 year old man presented to primary care clinic to follow up from an emergency department visit for acute pain. He notes that the workup in the emergency department was negative for ominous pathology and his pain has improved slightly. Workup there was notable only for a mild elevation in liver enzymes and concern for alcohol abuse. In clinic he is afebrile, tachycardic, and hypertensive. Exam is notable for tongue fasciculations and bilateral fine tremor. He reports that he drinks 1.5 liters of vodka per week and his last drink was 36 hours prior to presentation. He is interested in quitting drinking and says that the liver damage discovered in the emergency department was a wakeup call.

This patient was found to be in alcohol withdrawal. He refused inpatient evaluation or chemical dependency referrals. He did not desire therapy with benzodiazepines as he had heard they were habit forming. He preferred to “stick it out on his own” and follow up in 1 week. He has since been lost to follow up. His case provides an opportunity to review the care of the ambulatory patient with alcohol abuse and withdrawal, risk stratification, and options for outpatient treatment.

Discussion: There are many potential outpatient treatment modalities for alcohol withdrawal, but they require knowledgeable providers and daily visits from patients which is a high barrier to providing evidence-based care. This patient did not receive the standard of care and his case offers the opportunity to review possible treatment regimens in the outpatient setting.

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Back pain? Don’t forget about Multiple Myeloma
Introduction: Multiple myeloma is the second most common hematologic malignancy in the United States. Complications from the disease are debilitating and include end-stage renal disease and skeletal fractures. Early diagnosis and treatment improve both prognosis and quality of life.

Case: A 65-year-old man presents with worsening back pain of two weeks
duration. He was hospitalized few months ago for an upper gastrointestinal bleed that presumably led to renal insult requiring initiation of hemodialysis. The rest of his medical and surgical history includes atrial fibrillation status post atrial appendage closure, gout, type 2 diabetes mellitus, and obstructive sleep apnea. His family history is significant for a sister who passed away from leukemia at age 38. He is a truck driver who never smoked and rarely drinks alcohol.
The back pain started gradually two weeks ago, and has steadily increased in intensity. It is described as stabbing, severe especially in the morning, and worsens with activity. No trauma, fall, or heavy weight lifting. He initially sought help from his primary care physician who diagnosed him with lumbosacral strain and sent him home with acetaminophen and cyclobenzaprine. Unfortunately he continued to deteriorate and required hospital admission. Physical exam on admission revealed tenderness on palpation of the thoracic spine with limited range of motion due to pain. Labs revealed multiple abnormalities including hemoglobin of 8.3 g/dL and sedimentation rate of 58 mm per hour. CT of the spine revealed multiple acute and subacute compression fractures and lytic lesions. Multiple myeloma was suspected. Bone marrow biopsy revealed 80% of marrow cellularity was composed of plasma cells. He was diagnosed with multiple myeloma and discharged on denosumab and a bortezomib based therapy with close oncology follow-up.
Discussion: Multiple myeloma is a hematologic malignancy caused by monoclonal proliferation of plasma cells with high morbidity stemming from multiorgan dysfunction. The median age of diagnosis is 66 years old. Bone pain, commonly of the back, is the initial complaint in more than half of the patients. When an elderly patient presents with low back pain, it is important for physicians to look for red flags such as pain without trauma that is refractory to over the counter analgesics, especially when accompanied by unexplained weight loss or neurological symptoms. Further work up would be justified and recommended in these cases and suspicion should remain high for malignancy. Initial screening includes a complete blood count with differential and peripheral blood smear, basic metabolic panel with serum calcium, and serum protein electrophoresis with immunofixation.
Conclusion: For most patients with suspected multiple myeloma, the best initial imaging modality is whole body low dose CT. This is crucial as early diagnosis and treatment significantly prolongs disease-free and symptom-free survival.