



**NEW JERSEY CHAPTER
AMERICAN COLLEGE OF PHYSICIANS
REGIONAL SCIENTIFIC MEETING**

**RESIDENT/FELLOWS AND
STUDENT
ABSTRACT COMPETITION**

MARCH 9, 2018

PARTICIPATING INSTITUTIONS

Thank you to all the programs who submitted abstracts for this year's abstract competition. Abstracts were received from the following programs:

- Atlanticare Regional Medical Center (Dominik Zampino, MD, FACP)
- Capital Health Regional Medical Center (Saba Hasan, MD, FACP)
- Cooper University Hospital (Brian Gable, MD, FACP)
- Englewood Hospital and Medical Center (Karlene Williams, MD)
- HackensackUMC Mountainside (Bijal, Mehta, MD)
- HackensackUMC Palisades (Sameh Elias, MD)
- Jersey Shore Medical Center (Arif Asif, MD)
- Monmouth Medical Center (Margaret Eng, MD, FACP)
- Morristown Medical Center (Donna Astiz, MD)
- Overlook Medical Center (Jeff Brensilver, MD, FACP)
- Raritan Bay Medical Center (Mayer R. Ezer, MD)
- Rutgers - New Brunswick (Ranita Sharma, MD, FACP)
- Rutgers - Newark (Neil Kothari, MD, FACP)
- Saint Barnabas Medical Center (Sunil Sapru, MD)
- Saint Francis Medical Center (Sara Wallach, MD, FACP)
- Saint Joseph's Medical Center (Muhammad A Khan, MD, FACP)
- Saint Peter's University Hospital (Nayan Kothari, MD, MACP)
- Trinitas Medical Center (William Farrer, MD, FACP)

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DISCLAIMER

It is assumed that all participants adhered to the rules as stated in the original abstract submission form. It is also assumed that the abstracts submitted were original works, represented by the true authors. The abstracts appear in no particular order. Judging was performed in an attempt to minimize bias. Judges were unaware of the authors unless they were directly involved with the associate. Although there were many excellent abstracts those selected to be presented as poster or oral presentation were chosen on the basis of content. This content was felt to be intriguing from a clinical education standpoint, thought provoking, or could stimulate debate regarding our current practice of medicine.

Thank You to Our Oral Abstract Presentation Judges

Beth Nalitt, MD, FACP – Governor Northern Region

Saba Hasan, MD, FACP – Governor Southern Region

Jon Shammash, MD, FACP – Governor Elect

Tamie Proscia-Lieto, MD, MBA, FACP-Chairperson

A Special Thank You to Our Abstract Poster Judges

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Daniel Silverstein, MD

Dominic Zampino, DO, FACP

ORAL PRESENTATIONS

Hypoglycemia - Who Is At Risk? Clinical Research

Anju Jose, MD; Sunil Asnani, MD, FACE; Mohammad Hossain, MD
Jersey Shore Medical Center (Mayer Ezer, MD)

We hereby present our data on the trends of inpatient hypoglycemia at Jersey Shore University Medical Center and describe factors associated with and likely causative of inpatient hypoglycemia.

Methods: IT generated hypoglycemia reports were used to identify subjects from January-June 2016. Charts were reviewed for labs, admitting service, severity of illness, steroid use, co-morbid illnesses, scheduled procedure or surgery and other variables affecting glycemetic control.

Results: 107 cases of hypoglycemia were confirmed. 76 (71%) were age 65 or older. 55 were females and 52 males. Only 4 (3%) of these episodes were seen while on insulin drip. Kidney disease (Chronic Kidney Disease (CKD) stage 3 or worse) was seen in 56 patients (52%). 55 patients (51%) had a 'nothing per mouth' (NPO) order or had poor oral intake. 32 subjects (30%) had traveled off unit for a procedure/surgery/test on or the day before the hypoglycemic event. 24 subjects (22%) were septic and 10 subjects (9%) had liver disease. Finally, 6 subjects (5%) were on steroid taper and 21 subjects (19%) had alteration in ability to self-report symptoms of hypoglycemia.

Conclusion: Elderly patients, patients with renal insufficiency, those who are NPO or have poor oral intake are at highest risk of developing inpatient hypoglycemia. Patients who have been off the unit for a procedure, those who are septic, those who have altered mental status and thus inability to report symptoms of hypoglycemia were found to be next higher risk. Patients who are on steroids, on insulin drip and those with liver disease should also be closely monitored for hypoglycemia based on our study results. Developing strategies to prevent or reduce hypoglycemic events should include identifying high risk patients, recognizing precipitating factors, use of appropriate scheduled insulin, and appropriate nutritional support in hospitalized patients.

Prevalence of Proteinuria in Hospitalized Patients: A Marker of Uncontrolled Hypertension

Clinical Research

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Christian Kauzinger, MD; Arif Asif, MD
Jersey Shore Medical Center (Mayer Ezer, MD)

Proteinuria is a marker for cardiovascular disease. Detection of such patients can identify those eligible for treatment to decrease the risk.

OBJECTIVE: Identify hospitalized patients with proteinuria, the prevalence of concurrent use of medications known to decrease the level of proteinuria, and how many were placed on anti-proteinuric medications.

METHODS: Retrospective review of 200 patients admitted to our institution 2010-2015. Abstracted data included demographics, medical history, labs, urinalysis (UA). “Appropriate Medications (BPMeds)” included antihypertensives known to mitigate proteinuria.

RESULTS: 201 charts reviewed, 121 had UA. Primary Endpoint: 41 patients (33%) had proteinuria on the first UA, 14 patients had a second UA done, 9(65%) of them subsequently had no proteinuria. 66% were on BPMeds as follows: 1 medication (n=21); >2 (n=6); 14 were on no BPMeds and 2 of that group had BPMeds initiated during the index admission. The mean SBP and DBP among those with Proteinuria \geq 3 (162+19/84+9,n=7) was higher than those with proteinuria $<$ 2 (127+4/71+13,n=33) and the total cohort (133+27/73+13,n=40), $p<0.001$. Proteinuria \geq 3 was found in 35% (n=20) of those diagnosed with chronic hypertension vs 5% without it (chi2 $p=0.02$). 53% of patients with chronic HTN had inadequate BP control (SBP $>$ 140/DBP $>$ 90).

CONCLUSIONS: Proteinuria is prevalent, more severe in those with chronic hypertension, and only 53% of inpatients with chronic hypertension had proper blood pressure control. Our findings suggest that detection of proteinuria on admission presents an opportunity to identify patients eligible for quality improvement projects regarding optimization of chronic hypertension.



**Effectiveness of the Real-Time Transfusion Decision
Support System in Preventing Inappropriate Transfusion Practices**
Quality Improvement/Patient Safety

Muhammad Sardar, MD; Ananta Subedi, MD; Muhammad Azharuddin, MD;
Prateek Ghatage, MD; Doantrang Du, MD; Arpad Szallasi, MD
Monmouth Medical Center (Margaret Eng, MD)

INTRODUCTION: Liberal transfusion strategies have not shown to improve outcomes. Inappropriate and excessive use of blood transfusion can lead to several complications including hemolytic reactions, fluid overload, infections, acute lung injury etc. The objective of this study is to assess the effectiveness of the real-time clinical transfusion decision support system adopted by Cerner at our hospital and to evaluate if blood transfusion practices at our hospital are in accordance with the latest guidelines.

METHODS: A retrospective analysis was performed for the blood transfusions at Monmouth Medical Center between November 2016 and January 2017. Cerner used at our hospital has a transfusion guidance support system that pops up an alert for Packed Red Blood Cell (PRBC) transfusions that are ordered for patients having hemoglobin above 7g/dl. We filtered those orders and calculated the percentage of orders that were canceled after seeing the alert. For the orders where the alert was ignored, we tried to assess if the PRBC transfusion was in accordance with the American Association of Blood Bank (AABB) 2016 guidelines. As per the guidelines, transfusions for patients having hemoglobin below 8g/dl with history of coronary artery disease and those scheduled for Cardio-Thoracic or Orthopedic Surgery were considered appropriate whereas for all other patients transfusion below 7g/dl were considered appropriate. Other transfusions that were considered appropriate irrespective of the hemoglobin level are 1) massive intra-Op blood loss, 2) symptomatic GI blood loss, 3) hyper transfusion strategy in thalassemia patients, 4) acute Sickle Cell crisis.

RESULTS: In a 3 month period, 100 PRBC transfusion orders were filtered out by the transfusion guidance support system based on cut off Hemoglobin level of 7g/dl. Out of 100 orders, only 12 orders were canceled and the rest were transfused. Out of the 88 transfusions, 41 (46.59%) were not indicated as per the AABB guidelines. The top 3 causes of inappropriate blood transfusion were: 1) Pre-operative/post-operative patients with anemia- 31.7%; 2) Asymptomatic drop of hemoglobin with unclear etiology – 26.82%; and 3) Asymptomatic GI blood loss- 14.63%. A single Unit of PRBC transfusion costs around \$1,600-\$2,400 (including cost of raw blood, handling and transfusion). So roughly \$82,000 worth of resources were utilized during the time period of this study for blood transfusions that were inappropriate.

CONCLUSIONS: A high percentage of PRBC transfusion ordered at Monmouth Medical Center did not meet the AABB guidelines. The real-time transfusion guidance system has not been very effective in reducing the number of inappropriate transfusions. We recommend that for the PRBC transfusion orders that are alerted as inappropriate, the ordering physician must provide justification to the blood bank before the PRBC unit is released. We also need to remind physician about the latest blood transfusion guidelines and its importance.

Lung Cancer Screening: Need for Better Compliance and Broader Guidelines Clinical Research

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Rabia Soomro, MD; Mohammad Arsalan, MD; Anand Kaji, MD
Saint Francis Medical Center (Sara Wallach, MD, MACP)

Background: Lung cancer is the leading cause of cancer-related death in the United States. Many patients report no symptoms whatsoever and the majority of them become symptomatic only with advanced local or metastatic disease. For clinicians, it becomes a diagnostic challenge at times to have an index of suspicion regarding lung cancer in patients, particularly smokers, who don't present with typical respiratory or systemic symptoms. The United States Preventive Services Task Force (USPSTF) recommends annual low-dose computed tomography (CT) of the chest in asymptomatic adults aged 55 to 80 years with a 30 pack-year smoking history for current smokers or those who quit smoking within the past 15 years. The purpose of this study was to compare the incidence of Non-Small Cell Lung Cancer (NSCLC) in patients who fitted the USPSTF guidelines to those who did not fit the screening criteria and to assess the prior compliance among those who fit the screening guidelines.

Methods: We retrospectively reviewed records of all patients with a biopsy proven primary lung cancer at our hospital from 2011 to 2017. All patients aged 18 to 80 years with available chest CT scans and histopathological reports were included. Patients were evaluated for baseline characteristics, smoking history, and prior screening if indicated.

Results: Of the 104 patients included, 55 (53%) fitted the USPTSF guidelines and 49 (47%) did not. None of this population underwent screening prior to diagnosis. Males constituted 73% of patients who fit the guidelines, and 51% of those who did not (p-value 0.48). Both groups matched for racial distribution (26% black, 67% white, and 7% other races in the fit group; and 23% black, 63% white, and 14% other races in the non-fit group; p-values: 0.49, 0.49, and 0.53 respectively). NSCLC occurred in 98% of patients who fit the screening guidelines and in 96% of those who did not (p-value 0.47).

Conclusion: All patients who met the screening guidelines were never screened before the diagnosis. This warrants the need for a strategy as an institution and community to get better compliance for screening. There was no statistical significance in the incidence of NSCLC between the study groups. This emphasizes the need for an effective smoking cessation programs in the community and the need for further studies to determine the usefulness of the current guidelines.

POSTER PRESENTATIONS
RESIDENTS

**Prevalence, Predictors, and Consequences of Outpatient Clinic Appointment
Noncompliance in an Internal Medicine Clinic**
Clinical Research

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Atlanticare Regional Medical Center (Dominik Zampino, DO)

Background: Clinic appointment noncompliance (also termed appointment no-show) is a common problem in health care systems. The prevalence of appointment noncompliance in the outpatient settings in the United States ranges from 15-45% per year. These no-shows disrupt patient care, affect clinical outcomes, increase health care cost, and lead to missed learning opportunities for internal medicine residents. Research suggests that appointment no-show rates are higher in clinics that serve low-income communities. Thus, this project aimed to estimate the prevalence, predictors, and consequences of appointment no-show and to develop a no-show prediction scoring system in an Internal Medicine clinic that serves an uninsured/underinsured population.

Methods: This is a retrospective cohort study that used administrative and medical records data of all scheduled appointments between July 1, 2015 and June 30, 2016. The primary outcome variable was an appointment no-show that was a binary variable that assessed whether a scheduled appointment was attended or not. There were two secondary outcome variables: a) emergency department (ED) visits and b) inpatient hospitalizations. These categorical variables measured the number of ED visits or inpatient hospitalizations that occurred among patients with scheduled appointments within the study period. The exposure variables were patient-related, provider-related, and practice-related variables. Logistic regression models were used to assess the association between appointment no-show and the exposure factors. Multinomial regression models were used to assess the relationship between appointment no-show and the secondary outcome variables controlling for the exposure variables. A prediction model was developed. Ethical approval was obtained from the Geisinger Institutional Review Board.

Results: Of the 7338 scheduled appointments contributed by 2099 patients within the study period, 1869 appointments were unattended – an appointment no-show prevalence rate of 25%. The prevalence of appointment no-show was also 25% among patients with two or more ED visits and 26% among patients with two or more inpatient hospitalizations. Multivariate regression analysis revealed that the statistically significant ($p < 0.05$) positive predictors of appointment no-show were: a) patient-level factors: younger age, Black and Asian race, uninsured, unemployed, tobacco use, illicit drug use, alcohol use, and same zip-code residence; and b) practice-level factor: appointment lead-time. However, provider level of training was negatively associated with appointment no-show ($p < 0.05$). Appointment no-show increased the risk of at least two ED visits and inpatient hospitalizations within the study period by 25% and 43% respectively ($p < 0.05$). An appointment no-show prediction score has been developed and is being used to assign risk of appointment no-show score to all scheduled appointments. An intervention to reduce the risk of appointment no-show is being implemented among patients with high propensity for no-show.

Conclusion: Reducing the prevalent appointment no-show in our Internal Medicine clinic will ensure continuity of care, reduce health care cost, and improve the graduate medical education.

Persistence of a Left Atrial Appendage Clot After 12 Months of Apixaban Therapy

Clinical Vignette

Muhammad Nabil, MD; Ejaz Khan, MD
Atlanticare Regional Medical Center (Dominik Zampino, DO)

Prevalence of a left atrial (LA) clot in patients with atrial fibrillation is well known and is a significant risk factor for thromboembolism. It is the predominant cause of cerebrovascular accidents in this population. Trans-esophageal echo (TEE) remains the gold standard for detecting LA thrombus. Effectiveness of novel oral anticoagulants (NOACs) is well established in prevention of LA clot formation and decreasing the incidence of a subsequent cerebrovascular event. However, their role is still under investigation in the treatment of an existing LA clot. There have been reports suggesting comparable results of NOACs to Coumadin in treatment of these cases. Even in such reports, Apixaban has been shown to be the most beneficial of all NOACs. We bring forward a case where apixaban failed to resolve a LA clot even after 12 months of treatment.

Our patient was a 60-year old male who presented to his cardiologist's office with complaint of shortness of breath. He had a past medical history of morbid obesity, hypertension and smoking. EKG showed a new onset atrial fibrillation. He was started on metoprolol succinate and apixaban. A transthoracic echocardiogram (TTE) was scheduled for later that week however his symptoms worsened and was hospitalized after a few days for congestive heart failure. TTE on the day of admission showed an ejection fraction of 20-25% with severe enlargement of left atrium. Cardiac catheterization showed normal coronaries. Patient then underwent a TEE which showed a left atrial appendage clot; hence he could not be cardioverted. On discharge, he was instructed to resume his apixaban and metoprolol. About a year later when he was seen at the office for a routine visit, he was recommended to have a repeat TEE with cardioversion for his atrial fibrillation. Patient reported compliance with his medications. But TEE showed persistence of the clot in the left atrial appendage. He was then switched from Apixaban to Coumadin which was continued for 6 months. He was reevaluated with another TEE which showed complete resolution of the clot.

Atrial fibrillation is commonly associated with LA thrombus and thromboembolism. Its detection rate among patients taking NOACs is comparable to patients on Coumadin however Apixaban has been shown to be the most effective of the NOACs. There have been case reports of resolution of LA clots with Apixaban which were resistant to Coumadin and Dabigatran. There have also been reports of NOAC failure in such patients but the duration of therapy rarely exceeded 4-6 weeks. This case highlights the need for high quality clinical trials to study the usefulness of NOACs in the management of LA thrombus.

**Rare Case of Duodenal Diverticula Causing Lemmel's Syndrome,
Cholangitis and Pancreatitis in an Elderly Woman**
Clinical Vignette

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Duodenal diverticula occur in up to 22% of the population with an increased incidence amongst the elderly. The majorities are asymptomatic; however on rare occasion they have been demonstrated to compress the hepatobiliary and pancreatic ducts. This compression can result in obstruction and strictures within the biliary tree resulting in hepatobiliary and pancreatic disease.

The case presented is a 90 year old female with a recent hospitalization for unexplained pancreatitis and surgical history of cholecystectomy ten years prior. She presented with altered mental status and septic, with a temperature of 102.6 and a leukocytosis of 19.8. The cause for her sepsis was unknown as chest X-ray and urinalysis obtained in the emergency department revealed no obvious infection. During her admission for acute pancreatitis one month prior, Magnetic Resonance Cholangiopancreatography (MRCP) performed demonstrated dilation in the hepatic and common bile ducts (CBD), with the CBD measuring up to 1.8 cm in diameter. Upon visualization of the biliary tree, there was no evidence of cholelithiasis or masses within the bile ducts. Additionally, a duodenal diverticulum adjacent to the major papilla measuring 4.2 cm in diameter by 3 cm in height was discovered. In the setting of acute pancreatitis and lack of evidence of obstruction on MRCP, the decision was made to defer Endoscopic Retrograde Cholangiopancreatography (ERCP) for outpatient. However, the patient did not return for follow-up and her symptoms of abdominal pain recurred. She became ill, quickly decompensated and was brought to the emergency department. Given her recent unexplained pancreatitis and MRCP findings, hepatic panel and bilirubin were ordered. Results were consistent with biliary obstruction with a direct hyperbilirubinemia and transaminitis. Treatment for cholangitis was initiated and ERCP was performed. ERCP revealed a single area of severe stenosis in the lower third of the CBD measuring 40mm in length. Again noted was marked dilation in the biliary and hepatic ducts and a large periampullary diverticula. A sphincterotomy was performed as well as ballooning and stent placement at the level of the CBD stricture. Cells of the stricture were sent for cytology which returned negative for malignancy. Endoscopic ultrasound was also performed to evaluate for pancreatic malignancy; no pancreatic masses or suspicious lesions were visualized. After stent placement and medical therapy for cholangitis, the patient improved and returned to her baseline mentation.

This case is unique as both pancreatic and hepatobiliary complications of periampullary diverticula occurred. Pancreatitis, Lemmel's syndrome and cholangitis were all sequela of the diverticula. Considering periampullary diverticula as the etiology of obstructive biliary disease in cases without evidence of cholelithiasis or pancreatic malignancy is pertinent in early diagnosis and appropriate treatment of this condition. Stent placement, and in certain situations surgery, are definitive treatment options for symptomatic periampullary diverticula.

The Tender Rash: A Rare Case of Cutaneous Collagenous Vasculopathy

Clinical Vignette

Anokhi Patel, MD; Vishal Patel, MD

Atlanticare Regional Medical Center (Dominik Zampino, DO)

Cutaneous collagenous vasculopathy (CCV) is an idiopathic condition in which capillaries become dilated and surrounded by thick perivascular deposition of hyaline-like materials affecting superficial dermal blood vessels. This condition was first described in 2000 and there have been 34 reported cases since 2017. Patients typically present with asymptomatic, symmetrical, blanchable, pinkish red macules, petechiae or telangiectasias. The rash starts on the lower extremities and extends to the upper extremities, abdomen and torso while sparing the face, mucosa and nail beds. CCV is clinically similar to generalized essential telangiectasia and requires a histopathological diagnosis to prove otherwise. The etiology remains unknown, however it is presumed to be caused by a genetic defect that alters collagen production in skin microvasculature.

We present the case of a 47 year old male with a history of hypertension, dyslipidemia, gout, and chronic pain who complained of a diffuse, telangiectatic rash for more than 10 years. The rash was present over his extremities, torso and back and extended up to his neck, sparing the face. The rash was described as blanching telangiectasias that were significantly tender to palpation. He underwent a full work up for connective tissue diseases including ANA, c-ANCA, p-ANCA, ds-DNA, SSA, SSB, anti-CCP, anti-centromere, anti-smith, and TTG IgA all of which were negative. An initial biopsy in 2012 was suggestive but negative for scleroderma. He was treated symptomatically with corticosteroids without improvement and was subsequently referred to dermatology for further workup. A repeat biopsy five years later revealed cutaneous collagenous vasculopathy with overlapping features of telangiectasia. This was likely attributed to a genetic defect causing alteration in collagen formation with possible autoimmune process component. PAS staining showed thickening of the walls of the superficial blood vessels. The patient had a negative direct immunofluorescence for IgG, IgA, IgM, C3 and fibrinogen. He was started on hydroxychloroquine and has had little improvement in his symptoms.

The exact pathogenesis of CCV continues to remain unknown. There are no known triggers or inciting factors related to the disease. Several cases have been linked to pre-existing autoimmune conditions including diabetes mellitus, Raynaud's disease, psoriasis, discoid lupus erythematosus, and hypothyroidism. Biopsies will show thickening of capillary walls characteristic of CCV. This illustrates the importance of obtaining a biopsy and even a repeat biopsy as cases may be underdiagnosed or mistaken for other conditions. It is crucial to differentiate CCV from similar vasculopathies to ensure the correct therapies are initiated. Most cases have not required treatment as the lesions are typically asymptomatic. While little is known about the efficacy of medical and non-pharmacological management, further therapies must be explored.

A Cranial Nerve Palsy Caused by Continuous Positive Airway Pressure

Clinical Vignette

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Capital Health Regional Medical Center (Saba Hasan, MD)

Background: Opportunistic infections are common in immunocompromised patients and those with certain co-morbidities as diabetes mellitus, chronic renal insufficiency, malignancies and cystic fibrosis. Here we present a previously healthy and immunocompetent adult who suffered from not one but two species of micro-organisms causing significant neurological deficits.

Case: A 72 year old male with history of uncontrolled hypertension and obstructive sleep apnea presented to the ER with headache, blurry vision and diplopia for 2 weeks. Positive findings on examination were elevated blood pressure and right lateral rectus palsy. CT scan of the head ruled out intracranial hemorrhage. ESR was 30 s and no tenderness on temporal artery was found. Cerebrospinal fluid analysis ruled out encephalitis or meningitis. MRI brain showed no infarcts, and no hypo or hyperdense areas, but demonstrated complete right sphenoid sinus opacification suggestive of mycetoma and bony erosion of lateral sphenoid sinus, along with mucosal thickening of ethmoidal sinus. Concerned about infectious vs neoplastic process involving the sixth cranial, he was started on antibiotics and endoscopic sinus surgery was performed where the contents of the sphenoid sinus were removed. The pathology showed presence of aspergillus and cultures reported achromobacter species. He was started on antifungals and antibiotics guided by the final sensitivity report. He completed the treatment with resolution of his diplopia within 3 months. Investigating the cause of this rare mycetoma, he was seen by his pulmonologist who found that his CPAP equipment was not serviced since many years and showed signs of poor maintenance. He was advised not to use his machine until repeating the polysomnography and change of equipment.

Discussion: CPAP is the standard of care for treatment of patients with obstructive sleep apnea, though little data is available on infections associated with use of CPAP machine. Few cases have been reported about CPAP equipment being colonized with organisms and causing sinusitis and pneumonia. This patient had a significant infectious neurological complication for which he underwent extensive work up. As internists, we should be aware of such possibilities and consider it as one of the differentials in such cases. Regular servicing of the CPAP equipment is important to avoid colonization by ubiquitous organisms that may cause complicated illness."

Out of Mind, Out of Sight; Keeping the Differential Diagnoses Open Clinical Vignette

Sofia Chaudhry, MD
Capital Health Regional Medical Center (Saba Hasan, MD)

Background: Systemic Sclerosis is an uncommon chronic progressive disease that involves multiple organ systems and has various clinical manifestations but which can be missed if it presents with less noticeable skin changes. Its pathophysiology involves endothelial damage as well as fibroblast activation and collagen deposition leading to tissue fibrosis in the skin and other organs. The pulmonary complications include interstitial lung disease, and the skin complications include Raynaud's phenomenon.

Case: A 33 year old female with prior ER visits for cough diagnosed as community acquired pneumonia and discharged on azithromycin presented to the ER with similar complaints. She had fever, chills, and increasing shortness of breath for the past several weeks. The patient had a CT chest done and it showed bilateral lung infiltrates.

As a result, she was admitted to treat for CAP with doxycycline; azithromycin was avoided because she mentioned that it had given her a rash on her face. On day 3, the patient's SOB had slightly improved but the cough was still present. On taking a closer look on the patient while performing a physical exam, it was noticed that there was a small degree of tightening of the skin. She was asked about her hands and offered information that they are very sensitive to the cold, getting pain with decrease in temperature. This is also when an observation was made that perhaps the rash on the patient's face was not simply an allergy to azithromycin as the patient had believed. This in turn led to basic rheumatologic work up. ANA levels returned at 1:320, SCL-70 was high at 7.6. She had a HRCT done and it showed "Peripheral reticular and ground glass opacities throughout the lungs." Putting together the patient's clinical findings as well as lab abnormalities, she had been diagnosed with systemic sclerosis. Upon having the diagnosis of systemic sclerosis, the patient was started on Mycophenolate Mofetil and was schedule to undergo the appropriate work up for pulmonary complications of the disease including PFTS and echocardiogram.

Discussion: Many times when a patient is admitted, it is seen that the admitting diagnosis can be carried on forward and less attention may be paid to a patient's subtle signs that can actually indicate another more likely diagnosis. This diagnostic momentum bias can therefore lead to delays in delivering appropriate patient care. In this case, it was the keen attention to the patient's subtle skin findings and the willingness to attribute her symptoms to an alternative diagnosis that led to the appropriate work up being ordered, the correct diagnosis being reached, and in turn to the delivery of care targeted at her disease.



Second
Place Tie

**Not All Granuloma's Are Created Equal: How Disseminated TB Can
Hide Under The Cloak of Sarcoidosis**
Clinical Vignette

Roozbeh Ghavami, MD; Aram Srapyan MD; Daniel Goldsmith MD; Kristina Zarkua MD;
Marc Whitman MD; Hermann Schumacher MD

Capital Health Regional Medical Center (Saba Hasan, MD)

Background: Granulomas are the hallmark structure of many granulomatous diseases, especially that of Mycobacterium tuberculosis (MTB). Historically, granulomas in MTB are believed to be a protective structure that walls off the infection. However, there are some new research that point to granulomas as a possible cause of disseminated MTB.

Case: We are reporting a case of disseminated Mycobacterium tuberculosis in a 72 year old Liberian American who presented through our Emergency Department with complaints of weight loss, anorexia, confusion and a non-healing scrotal surgical wound and fever. Patient had been seen prior and was diagnosed with sarcoidosis based upon inguinal lymph node biopsy that was negative for AFB and GMS stains. Several days prior to this admission patient was seen by urology for a scrotal mass and underwent orchiectomy. On exam, he appeared weak with reduced speech and as per wife, patient had been experiencing a decline in his cognitive ability with some somnolence. CT scan and MRI of the head demonstrated multiple enhancing lesions in the left temporal lobe and medial right frontal lobes of the brain. CT scan of the chest, abdomen and pelvic demonstrated multiple enlarged lymph nodes with reticular nodular appearance of the upper lobes consistent with the previous diagnosis of sarcoidosis. Patient's orchiectomy tissue was then specifically stained for AFB with positive results. Patient's groin lymph nodes were biopsied again for AFB which also returned positive along with positive culture confirmed by PCR and MTB rRNA detected in the sample. The CSF stain and culture were negative for AFB. Given the strong evidence on hand, the patient was diagnosed with disseminated TB and started on the RIPE regimen for anti MTB treatment.

Discussion: It is important to rule out any granuloma producing infectious process with due diligence. Although the pathology of the granulomas may be similar, the etiology is often drastically different, requiring different approach of treatment. In such cases that MTB could be in the differential diagnosis, a thorough workup including AFB stains and culture are needed to rule out MTB before starting steroid therapy.

Nutritional Acute Ascending Neuropathy Mimicking Guillain-Barre Syndromé

Clinical Vignette

Eseosa Edo-Osagie, MD; Emmanuel A Adomako, MD; Tanganyika Barnes, DO
Englewood Hospital and Medical Center (Jon Shammash, MD)

INTRODUCTION: Dry Beriberi remains an under-recognized cause of peripheral neuropathy despite reports in the literature, and may be misdiagnosed as Guillain-Barré Syndrome (GBS). We report a case of thiamine deficiency causing GBS-like neuropathy in the setting of remote alcohol abuse.

CASE: A 39-year old female with a history of alcohol abuse presented with an eight-day history of numbness involving her bilateral lower extremities and hands which had progressed proximally to involve her chest wall. She reported perioral and perianal numbness, lethargy and difficulty ambulating. Examination was significant for decreased sensation in a glove-stocking distribution, areflexia and ataxia. Initial laboratory data was significant for folate and magnesium deficiencies which were treated. MRI of the brain, cervical and thoracic spine were normal. A clinical diagnosis of GBS was made and she received IVIG and subsequently plasmapheresis with no symptomatic improvement. Serial negative inspiratory force and vital capacity measurements were normal. CSF analysis, viral, Lyme and rheumatologic serologies were unremarkable. EMG was significant for axonal sensory neuropathy. Thiamine levels returned low at 60nmol/L (normal 78-148). These findings were consistent with a diagnosis of dry beriberi and treatment with IV thiamine was started with mild improvement in her symptoms. She was eventually discharged on oral thiamine. At 4-month follow-up, symptoms had slowly improved but she remained dependent on a walker for mobility.

DISCUSSION: Thiamine deficiency causes clinical phenotypes of Beriberi and Wernicke-Korsakoff syndrome. Beriberi can be classified as “dry or wet”. Dry beriberi presents as weakness, paresthesia and neuropathic-type pain. The evolution of symptoms can occur very rapidly over the course of a few days (1). Limb involvement may be the only presentation in most cases of nutritional neuropathy compared with GBS where there is often involvement of the respiratory and bulbar muscles necessitating ventilation, which is extremely rare in beriberi (2). Recovery from nutritional neuropathy is a slow process. There is often considerable improvement in mild cases but in severe forms, recovery may not be complete (3). It is important that thiamine deficiency be part of the differential diagnosis in any patient presenting with an acute neuropathy which may mimic GBS especially in the setting of alcohol abuse whether remote or recent.

**Association of Updated AHA/ACC Heparin Dosing Guidelines for NSTEMI
Patients With Compliance and Bleeding**
Clinical Research

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Background: In 2015 AHA/ACC recommended lower heparin dosing for Non-ST elevation myocardial infarction (NSTEMI) patients. Maximum initial bolus decreased from 7500 units to 4000 units. Initial infusion rate decreased from 1500 units/hr to 1000 units/hr. We implemented those guidelines, and as part of the National Cardiovascular Data Registry (NCDR) quality assurance process, compliance and the rates of bleeding were recorded. **HYPOTHESIS:** Lower heparin dosing would be associated with fewer bleeding events. **OBJECTIVES:** analyze NCDR data at our institution to compare the following prior to and after 2015: 1) Rates of provider compliance with the new recommendations. 2) The number of patients who had NCDR bleeding events. 3) Patients who had Hemoglobin (HGB) Drop ≥ 3 g/dL entire admission.

Methods: NCDR data is collected by cardiology quality assurance advance practice nurses at our 546 bed tertiary care center. NCDR defines bleeding events as any of the following within 72 hours of starting heparin: 1)HGB drop ≥ 3 g/dL 2)Red blood cell transfusion with HGB < 8 g/dL 3)Procedural site intervention to address bleeding.

Results: After implementation of the new guidelines, the percentage of patients receiving heparin bolus >4000 units dropped from 62% to 22%, and the mean infusion rate dropped. The relative risk (RR) of receiving a heparin bolus >4000 units in 2014 vs. 2015 was 2.8 (95% CI [1.9,4.2]). NCDR bleeding rate: Was not significantly different in 2014 (2.5%) vs. 2015 (5%), (Wilcoxon Rank-sum $p=0.2$). Hemoglobin Drop ≥ 3 g/dL: The rate in 2015 was not significantly different from the 2014 rate. (RR 1.05. 95% CI [0.6,1.9]). Mean HGB Drop: No Difference 1.9 + 1.6 each year

Conclusions: Implementation of the new recommendations was associated with a 40% decline in excessive heparin dosing for NSTEMI patients at our institution, but the rate of NDCR defined bleeding did not change significantly. The stable bleeding rate could be explained by the presence of confounders that were not recorded as part of the nursing.

We Have the MEATS...or Hives: A Case of Tick-bite Induced Mammalian Meat Allergy

Clinical Vignette

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Case Summary: A 43 year old woman with no past medical history was camping in Pennsylvania, became “unwell” and developed diarrhea, vomiting, severe itching, generalized hives, and swelling of her extremities and face. The night of symptom onset, she had grilled steak for dinner. She endorsed removing multiple ticks after a hike in Monmouth County NJ 1 week prior and did not eat red meat until the night of the symptoms, which later improved with diphenhydramine. Physical exam at the office visit: normal vital signs, no systemic signs of illness including rashes, organomegaly, or edema. Immunological testing supported the clinical diagnosis of Alpha-gal allergy syndrome: Beef specific IgE levels = 1.39 kU/L (< 0.35), pork = 0.83 kU/L (<0.35), lamb = 0.64 (<0.35), as well as anti-galactose-alpha-1,3 galactose-IgE (Alpha-gal IgE) = 2.41 (<0.35). Following the diagnosis she avoided meat, but hives, diarrhea, and swelling recurred after consuming gelatin and dairy products but decreased in severity over 10 months.

Discussion: Alpha-gal allergy often manifests as delayed anaphylaxis, urticaria and or angioedema. It was first described in cancer patients treated with the monoclonal antibody cetuximab [PMID 18337601]. Recent data showed that tick bites, especially Lone Star tick (*Amblyomma americanum*) can induce Alpha-gal IgE by sensitizing the predisposed individual to the oligosaccharide which has been discovered in the gastrointestinal of the *Ixodis ricinus* tick [PMID 23414348]. Subsequent exposure to alpha-gal then results in delayed anaphylactic or urticarial reaction. One study showed that the prevalence of alpha-gal antibodies was 20% in tick prone regions compared to <1% in areas without. Within this study, prospective data on 3 patients showed a 20-fold increase in serum titers from baseline of anti-alpha-gal IgE antibodies after a tick bite with resulting delayed hypersensitivity in 2 out of 3 [PMID 22815061]. Additionally, a cross sectional study found that 97% of patients presenting to an allergy clinic with history of delayed anaphylaxis to red meat had Anti-alpha-gal IgE compared to only 11% of patients seen in that same practice for asthma [PMID 21453959]. The mainstay treatments for this condition are avoiding alpha-gal containing products and symptomatic relief. The long term prognosis of this condition is not well described; however it is thought that if a strict diet is followed and tick re-exposure is avoided, reactions may lessen over time [PMID 16261963]. This case demonstrates the need to recognize this emerging clinical entity.

**Acute Tubular Necrosis Secondary to Antacid-Induced Hypercalcemia:
Time to be More Aware!
Clinical Vignette**

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Throughout history, antacids were perceived to cause solely milk-alkali syndrome rather than causing a unique nephropathy. As the use and availability of calcium-based antacids become more prevalent in today's population, it is important to be aware of acute tubular necrosis caused by hypercalcemia due to excessive ingestion of antacid especially with any pre-existing kidney disease. Renal insufficiency secondary to hypercalcemia caused by impaired concentrating ability decreased glomerular filtration rate, calcium deposition, or nephrogenic diabetes insipidus. Acute tubular necrosis (ATN) is a rare manifestation of hypercalcemia.

Case: A 47 year old Caucasian female with past medical history of hypertension, diabetes mellitus, and COPD was admitted with complaints of generalized weakness, abdominal pain, diarrhea, and confusion. Patient also reported a history of taking excessive calcium-based antacids over the past several weeks for dyspepsia. Patient had been treated for similar complaints, hypercalcemia, and acute renal failure which required hemodialysis approximately eight years prior to this admission. Patient's vital signs were normal including negative orthostasis. Physical examination was unremarkable. Laboratory workup revealed BUN of 30 mg/dl, creatinine of 3.95 mg/dl, calcium of 16.3 mg/dl, and PTH of 6 pg/ml. Urine studies revealed random sodium of 71 mmol/dl, creatinine of 16.2 mg/dl, and osmolality of 168 mosm. Urine to serum creatinine ratio was less than 10 with a FENa of 13%. Urine analysis revealed muddy cast without proteinuria. Renal ultrasound was normal. Renal biopsy revealed acute tubular necrosis superimposed on underlying hypertensive arterionephrosclerosis. The patient was initially treated with intravenous normal saline. Later, in view of persistent hypercalcemia, calcitonin was added. After two days, serum calcium dropped to 9.2 mg/dl and the patient's clinical condition improved significantly.

Discussion: The calcium ion is an essential regulator of many body processes including muscle contraction, many secretory mechanisms and neuronal excitation. Most common causes of hypercalcemia are malignant tumors, primary hyperparathyroidism, immobilization, ingestion of vitamins A and D, thiazide diuretics, granulomatous diseases, milk-alkali syndrome, familial hypocalciuric hypercalcemia, and intrinsic bone diseases. The potential renal consequences of hypercalcemia are arteriolar vasoconstriction, decreased tubular sodium reabsorption, nephrogenic diabetes insipidus, nephrocalcinosis, tubulointerstitial fibrosis, and ATN. Etiology of ATN in hypercalcemia is not clearly understood but there are three well known alterations in tubular dynamics of ATN are obstruction, back-leak, and activation of tubular glomerular feedback. The degree of ATN is always directly proportional to the degree of hypercalcemia. Recurrent coexistence of ATN and hypercalcemia in this patient is suggestive that the association between these two phenomenon is a much more common entity than previously studied. The complications associated with ATN are often life threatening with in-hospital survival rate of approximately 50%. Any case presenting with hypercalcemia and acute renal failure should be investigated for ATN for early diagnosis and treatment.

**Clostridium Difficile Diarrhea Associated With Culture-Negative Peritonitis
in a Peritoneal Dialysis Patient**
Clinical Vignette

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Case Summary: An 85 year-old woman presented to the hospital with two days of crampy abdominal pain, vomiting, and watery diarrhea. She had started peritoneal dialysis three months prior for end stage renal disease. Her last dialysis session was one day prior to symptom onset. She was released from a rehabilitation center one month ago after a hospital admission for congestive heart failure. PMH: atrial fibrillation, chronic obstructive pulmonary disease, cervical cancer status post total abdominal hysterectomy. Exam: vitals were blood pressure of 179/74, pulse of 60, respiratory rate of 15, and temperature of 102.8. Abdomen: tender with voluntary guarding. Dialysis catheter was intact with no surrounding erythema, swelling, or purulent discharge. Peritoneal fluid aspiration via the catheter showed white cell count of more than 8000/uL (normal <1000) neutrophil count of 93% (normal <25), confirming the diagnosis of peritonitis. However, no organism grew on peritoneal culture. She received vancomycin and ceftazidime intraperitoneally via the catheter. Diarrhea workup revealed a positive stool PCR for *Clostridium difficile*. Metronidazole was added with subsequent rapid clinical response. *Clostridium difficile* peritonitis was suspected and the initial broad spectrum antibiotics were held, while the patient's symptoms continued to improve on metronidazole alone. Further fluid analysis taken from the peritoneal catheter on the third and fifth day after admission showed resolution of the leukocytosis. Upon surgical evaluation, it was decided to leave in the peritoneal catheter as the fluid culture was negative, fluid leukocytosis resolved, and the catheter did not appear to be the source. The patient's symptoms resolved and she was then discharged on a total of three weeks of metronidazole with continuation of peritoneal dialysis.

Discussion: Peritonitis was diagnosed based on classic clinical features of abdominal pain, vomiting, fever, cloudy effluent, along with peritoneal fluid leukocytosis with neutrophilic predominance. We considered *Clostridium difficile* as the causative organism for peritonitis despite negative culture as the patient's symptoms and fluid laboratory findings had normalized after starting metronidazole therapy. According to literature, about 20% of the *Clostridium difficile* peritonitis in peritoneal dialysis patients can have negative peritoneal fluid cultures. This case report highlights the importance of considering *Clostridium difficile* as the possible cause of culture negative peritonitis in peritoneal dialysis patients in the appropriate clinical setting.

**Two Primary Carcinomas for the Price of One: Synchronous Colorectal
Carcinoma in a Young Patient with No Risk Factors**
Clinical Vignette

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Case: A 48 year old male presented with acute, severe left lower quadrant abdominal pain accompanied by worsening constipation and a 20 pound weight loss. Family history: no colon cancer. CT scan: distended and stool-filled colon to the level of the mid to distal descending colon, where a 3 X4 cm soft tissue density was identified. He underwent an urgent left hemi-colectomy, and resection of the splenic flexure mass with end to side colo-colonic anastomosis in the sigmoid colon. Pathology: T3N0M0 poorly differentiated adenocarcinoma. There was no evidence of metastatic disease. Colonoscopy revealed 14 polyps throughout the entire colon ranging 10-25 mm, mostly tubular adenomas, with a few hyperplastic polyps. Surprisingly, an additional infiltrative ulcerated non-obstructing 6cm mass was found in the sigmoid colon, 32 cm from anal verge. Pathology: moderately differentiated colonic adenocarcinoma, staged as T3N1M0.

Germline mutation tests for familial adenomatous polyposis (FAP) and hereditary non-polyposis colorectal cancer (HPNCC) were negative (microsatellite instability analysis by PCR and APC gene testing). He underwent a second open sigmoid resection with recto-sigmoid anastomosis, followed by adjuvant chemotherapy with oxaliplatin. He will require yearly colonoscopy for 5-10 years.

Conclusions: Synchronous colorectal neoplasia (SN) is defined as two or more concurrent primary tumors in the same patient at the same time, and comprise 3-5% of cases of CRC. Intraoperative palpation may miss SN up to 69% of the time, so colonoscopy is necessary. Only 3% of SNs involve different types of malignancies, and 33-55% are villous adenomas. FAP is associated with APC gene mutation, development of hundreds to thousands of adenomas, younger age, and 100% progression to CRC. Other risk factors include ulcerative colitis, attenuated FAP, and serrated polyposis syndromes, although their association with cancer is lower. Continual surveillance for CRC is recommended in these cases.

Our patient was tested for these syndromes via germline testing, colonoscopy and review of family history, but ultimately was found to have numerous adenomatous polyps in the absence of a familial syndrome or genetic mutation. Adenomatous polyps are the classic neoplastic polyps, harboring a 5% or less chance of developing into a carcinoma.

Management of SN includes total abdominal colectomy with ileo-rectal anastomosis, favored in patients younger than 60 years, with non-metastatic disease and concurrent adenomatous polyps. Limited resection is preferred in older patients with increased comorbidity.

Impact of Patient Load on Documentation Quality Among Residents

Quality Improvement/Patient Safety

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Patient record form one of the most important part of clinical care as the primary source for patient information for primary team, consultants, nurses and other paramedic staff and help in providing a higher quality of care, as well as monitoring patient safety. Incomplete patient records are a source of gaps in patient care and vice versa. The importance of clinical documentation in medico-legal cases cannot be more emphasized. The current American College of Graduate Medical Education has ruled that a first-year internal medicine resident must not be responsible for ongoing care of more than 10 patients at a time. This is a randomly assigned number and the implications have not been studied. Here we assess the quality of patient health documentation by first-year residents of a community teaching hospitals and evaluate how patient numbers and workload affect documentation quality.

Interns in the hospital go on-call for admissions every fourth day. 80 progress notes, four each from 20 interns on different call status were assessed. On an average, first-year residents or interns during the study period took care of 9 ± 1 patients on a 'post-call' day, 7 ± 1 patients on a 'mid-call' day, 5 ± 1 on a 'pre-call' day and 4 ± 1 on a 'call' day. Responsible Electronic Documentation Checklist is a validated checklist scoring system to assess the quality of progress notes. We calculated the average scores of progress notes based on the checklist and compared it to their call status to evaluate how the patient load affected documentation quality. We also found that a resident on average access patient charts 221.9 times a day during a floor-rotation which gives an impact of the electronic workload.

The study found that notes were generally deficient in updated physical examination and summary statements while on days with maximum patient load there were minimal updates on assessment and plan, lacked a diagnostic plan for patient problems and physician interpretation of imaging studies. The average note scores dropped from 72.14% on a pre-call day to 57.06% on a post-call day. We found statistically significant difference in documentation quality between post-call day and any other call status and was most pronounced between the above two (p -value 0.00042; t =-3.626). Apart from the patient load of the residents, other factors like physical stress from the previous call-day might also be affecting the documentation quality. How the deficiency in documentation quality has affected patient safety and litigations have to be further studied. Whether this can be extrapolated to hospitalists and consultants will also, be of interest. With the advent of electronic health records, the electronic workload and the 'screen-time' for the physicians have increased and this might add to resident burn-out.

Occipital Condyle Syndrome as the Initial Presentation of Lung Cancer

Clinical Vignette

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Lung cancer is the most common cancer in men and leading cause of cancer mortality worldwide among women. Approximately 20% of Non-small cell lung cancers (NSCLC) have bone metastasis at presentation. The base of the skull is a less frequent site of metastasis and is most often silent. Occipital Condyle syndrome (OCS) is a rare clinical syndrome, consisting of unilateral occipital headache accompanied by ipsilateral hypoglossal palsy. To the best of our knowledge, this is the fourth reported case of OCS from primary lung cancer.

A 55-year-old Asian female with no significant past medical history presented to the emergency room with continuous left sided temporo-occipital headache of one-month duration and difficulty chewing food and deviation of tongue for 5 days. The headache was only partially relieved with over the counter medications. Physical examination revealed mild fasciculation of the tongue with deviation of tongue to right with no atrophy. A Computed Tomography (CT) imaging of the brain was done and was unremarkable. MRI brain was done which revealed 2.0 x 2.7 x 1.2 cm enhancing lesion destroying the right skull base in the region of the right hypoglossal canal. Subsequent screening with CT of the chest, abdomen and pelvis revealed 4.7 cm spiculated mass at the superior segment of the left lower lobe with metastatic lymphadenopathy to left hilar, mediastinal and right supraclavicular. A Right adrenal mass and sclerotic lesions in the T9 and L2 vertebral bodies without central canal compromise was found identified as possible metastasis. A CT guided lung biopsy confirmed the diagnosis of adenocarcinoma of lung. Patient was started on Bevacizumab, Pemetrexed, Carboplatin and also received radiation therapy. She was also given a tapering dose of prednisone and the headache resolved in a week. Metastasis or primary tumors involving an occipital condyle can directly invade the skull base involving the hypoglossal canal and result in lower cranial nerves neuropathies. Our patient experienced a unique pattern of a severe, continuous, unilateral pain in the occipital region, with symptoms typically improving by turning the head towards the painful side. Majority of cases remain silent. OCS is characterized by an isolated unilateral hypoglossal paresis and mastoid tenderness along with the headache. Diagnostic imaging of the skull base may appear normal on initial presentation. An awareness of the characteristics of OCS in healthy adults may lead to earlier diagnosis of the underlying etiology and prevent irreversible neurological damage."

Sarcoidosis, Everywhere but the Lung!

Clinical Vignette

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Sarcoidosis is a chronic inflammatory condition of unknown etiology most commonly involving lungs, but rarely affecting the musculoskeletal system. Axial sarcoidosis involving vertebral bodies and sacroiliac joints is rarer and might mimic cancer metastasis, myeloma and Langerhans cell histiocytosis requiring histopathological confirmation. Our case is an atypical presentation of a rare condition, extrapulmonary axial sarcoidosis affecting the skeletal system extensively without lung involvement.

A 53-year-old Caucasian male came to the outpatient office with lower back and bilateral thigh pain after lifting a heavy box at work a week ago. He denied any weakness, or bowel and bladder incontinence. Physical examination revealed no spinal tenderness or focal neurological deficits. Lumbosacral x-rays and nuclear whole-body bone scans were normal. An MRI lumbar spine and lower extremities demonstrated increased signal density of the L4-5 vertebral disc, T2 hyperintense lesions throughout the bony pelvis and bilateral femurs which was suspicious of myeloma or metastatic disease. However, blood counts, liver and renal function tests, PSA and serum protein electrophoresis were normal. PET-CT was done which revealed markedly hypermetabolic spleen and extensive osseous metastatic disease throughout cervical, thoracic and lumbar spine, pelvis, bilateral femur and right humerus as well as lesions in the ribcage. No FDG accumulation was noted in the lungs. Bone biopsy of the lesion showed multiple noncaseating granulomata and stromal fibrosis consistent with sarcoidosis. No micro-organisms were identified on gram staining and acid-fast stains. Patient was started on hydroxychloroquine and corticosteroids with resolution of symptoms and significant reduction of FDG avidness on repeat PET-CT in 2 months.

Sarcoidosis of bone occurs in 1-13% of sarcoid patients and might be an early manifestation of the disease. Presence of bone lesion is a sign of severe and chronic disorder and is seldom has not been reported without concurrent pulmonary involvement. Spinal disease is extremely rare with only case reports and series from our extensive literature search. The lesions are mostly silent and when lytic lesions are observed on radiographs or bone scintigraphy, they are often misdiagnosed as malignancy. Complications include fractures, cord compression and nerve impingements. Other conditions like lymphoma, myeloma and Langerhans cell histiocytosis also need to be ruled out. Magnetic resonance imaging is the most helpful diagnostic imaging in vertebral sarcoidosis while PET-CT will be helpful in assessing the extent of involvement. Histopathological confirmation of bone lesions is necessary for a final diagnosis. Glucocorticoids are the first line of treatment followed by antimalarial, methotrexate and in refractory cases, infliximab. Our patient responded to corticosteroid therapy and the changes between pre- and post-treatment imaging were remarkable. Knowledge about the condition is necessary to help follow proper diagnostic protocol and starting appropriate therapy preventing progression and complications.

Predictors of Post-Operative Myocardial Infarction After Carotid Endarterectomy

Clinical Research

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Introduction: Myocardial infarction (MI) is one of the complications that can occur after carotid endarterectomy (CEA). Our objective in this study was to perform a wide-based analysis of the American College of Surgeons (ACS) National Surgical Quality Improvement Program (NSQIP) database in an attempt to identify risk factors associated with occurrence of MI in the 30-day post-procedure period.

Methods: The ACS NSQIP database was queried among all participating institutions for CEAs performed between 2011-2015. Patient demographic data, co-morbid conditions, pre-procedure medication use, and operative variables were analyzed using multivariate logistic regression analysis to determine predictors of post-operative MI.

Results: 18,045 CEAs were available for review from the NSQIP database, of which 325 (1.95%) of patients developed a post-operative MI within 30 days. The incidence of MI increased by 0.2% (95% CI 1.000-1.0004; P=0.01) for each additional minute of operative time. Patients with an American Society of Anesthesiologists (ASA) classification of 4-5 were 56% more likely to experience a post-operative MI (CI 1.22-1.98; P=0.0003). Patients 80 years or older were 70% more likely to experience an MI (CI 1.34-2.14; P<0.0001). Approximately 2 times the risk of post-operative MI was found to occur in patients who were not functionally independent (CI 1.27-2.99; P=0.0022), and in patients with a history of congestive heart failure (CI 1.34-3.80; P=0.0021). Mortality was associated with an 8.1 times higher risk of post-operative MI (CI 5.18-12.71; P<0.0001). Pre-operative beta-blocker use was not associated with a decreased risk of post-operative MI (CI 1.42-2.26; P<0.0001).

Conclusion: The predictors of an MI occurring within a 30-day period after CEA include: longer operation time, ASA classification 4-5, age 80 years or older, absence of functional independency prior to procedure, history of congestive heart failure. MI occurred more often in patients who expired within the 30-day post-operative period. Beta-blocker use did not decrease the risk of post-operative MI.

An Unusual Complication of Chronic Lymphedema

Clinical Vignette

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Stewart-Treves syndrome (STS) is a rare condition that refers to the development of an angiosarcoma in the setting of longstanding lymphedema. Classically this is due to axillary lymph node dissection following a radical mastectomy. However, it is extremely uncommon for this syndrome to be reported in a patient with idiopathic chronic lymphedema of the lower extremities. This case highlights the importance of including Stewart-Treves syndrome in the differential diagnosis of chronic lymphedema in the setting of a non-healing ulcer as well as an understanding of the diagnostic workup of such a case.

A 52-year-old Caucasian female with past medical history of hypertension, diabetes mellitus type 2, peripheral vascular disease, and chronic lymphedema for over 20 years initially presented to the hospital for evaluation of a non-healing wound of her right lower extremity. Patient states that she has had this wound 6 months prior to admission and failed outpatient antibiotic treatment from her primary care physician. Upon physical exam, there was significant bilateral pitting edema. The right lower extremity had a 10 cm irregularly round ulcer with granulation tissue and drainage adjacent to an 8-cm round fungating black mass. Both lesions were surrounded by purpuric patches circumferentially around the lower extremity. An MRI of the right lower extremity was performed and showed signs suggestive of cellulitis. Therefore, patient was admitted for sepsis secondary to right lower extremity cellulitis and was treated with vancomycin and merrem. Despite antibiotic therapy, patient clinically did not improve.

Dermatology was consulted and was concerned about possible underlying neoplasm as there was a skin mass inferior to the ulcer. A wedge biopsy was performed which showed malignant cells with prominent nucleoli which formed rudimentary inter-anastomosing vascular canals with extensive hemorrhage and necrosis. Immunohistochemical study revealed diffuse expression of CD31 and CD34 confirming the diagnosis of angiosarcoma. Based upon the clinical presentation and immunoprofile, the patient was diagnosed with Stewart-Treves syndrome. Patient was then referred to a specialized surgical center where she underwent an above the knee amputation.

The development of angiosarcoma in chronic lymphedema was originally described in 1948 in women with breast cancer treated with radical mastectomy. However this case brings to the forefront that STS may present in the lower extremities in congenital, idiopathic, traumatic, infectious, or post-surgical lymphedema. As primary care doctors, it is imperative to recognize suspicious lesions in a patient with chronic non-healing ulcer and to consider STS in the differential diagnosis. Despite the poor prognosis of this entity, early recognition may help to improve patient quality of life.

Mysterious Bleeding

Clinical Vignette

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Case presentation: A 67-year-old female was admitted to the medical service in June 2017 for a syncopal episode. The patient had a history of colon cancer diagnosed in May 2016 during a colonoscopy. Soon after, she underwent left hemicolectomy with end-ileostomy without any bleeding complications. A month later, follow-up imaging showed a liver lesion and right lower lobe lung mass. She had been treated with 5-fluorouracil, folinic acid and oxaliplatin. In June 2017, a restaging scan showed no improvement in the lung mass. There was a concern for a second primary in the lung. The patient had just undergone a CT- guided elective right lung mass biopsy. She was being monitored in the recovery area. Medical service was called, one hour later, when she was complaining of dizziness and syncopized. She regained consciousness in a minute and had no further complaints. On exam, she was hypotensive and tachycardic. Post-operatively, chest x-ray showed a small right-sided hemothorax. Computed tomography of the chest revealed moderate to large hemothorax. In the next five hours, hemoglobin dropped from 11 g/dl to 8.3 g/dl. Activated partial thromboplastin time (APTT) was elevated at 61.1 seconds (reference range 23 to 37 seconds) with normal prothrombin time (PT). She was treated with fresh frozen plasma. However, there was no improvement noted in APTT. The improvement in acute bleeding was noted with recombinant factor VIIa infusion. One year earlier, her APTT had been normal. Coagulation studies showed decreased levels of factor VIII and positive factor VIII inhibitor. A diagnosis of acquired factor VIII inhibitor was made in the setting of metastatic colon cancer. She was then successfully treated with cycles of rituximab and steroids.

Discussion: Acquired factor VIII inhibitor disorder is characterized by the development of auto-antibodies against a clotting factor VIII. The condition is extremely rare with a potential to cause life-threatening bleeding. Therefore, it is important to be familiar with the diagnosis of this condition. Prompt treatment can be life-saving. Autoimmune diseases such as systemic lupus erythematosus and rheumatoid arthritis are most commonly associated with this condition. Factor VIII inhibitor can be rarely associated with solid tumours and hematologic malignancies. There has been no association found with specific type of a tumour. Our case highlights acquired factor VIII inhibitor in the setting of metastatic colon cancer which is a rare phenomenon.

**H. Pylori Eradication Combined with Iron Administration for
Effective Treatment of Iron Deficiency Anemia**
Clinical Vignette

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Introduction: Iron deficiency anemia (IDA) is a common cause of referral to gastroenterologists (4%-13% of referrals). In about 5%-10% of patients with IDA without gastrointestinal bleeding, the cause remains unknown despite extensive investigation. Studies have suggested that *Helicobacter pylori* (*H. pylori*) eradication therapy improves iron absorption. Iron therapy in combination with *H. pylori* eradication has been shown to be more effective than iron therapy alone for the treatment of IDA.

Case: Our patient is a 42-year-old male presented who presented to the emergency room with the complaints of generalized fatigue and tiredness. He was sent by his PMD after routine blood work revealed hemoglobin of 4 mg/dl. He denied any hematemesis, melena, or abdominal pain. His physical examination was unremarkable except for pallor. Initial labs were: Hemoglobin of 3.8 mg/dl, hematocrit of 14.3%, WBC of $7.4 \times 10^9/L$ and platelets of $504 \times 10^3/\text{micro liter}$. He was started on IV pantoprazole with serial checks of hemoglobin and hematocrit. After the anemia panel was send, he was transfused with packed red blood cells. Subsequently he underwent endoscopy and colonoscopy. His endoscopy showed mild gastritis and colonoscopy showed second degree hemorrhoids. His anemia work up showed iron deficiency anemia with an iron of 12 micrograms/L, ferritin of 2 micrograms/L, IBC of 451 micrograms/dL, and transferrin saturation of 3.0%. Pathology report showed extensive chronic active gastritis and *H. pylori*. The patient was discharged on appropriate treatment for *H. pylori* with iron supplements.

Discussion: *H. pylori* is associated with peptic ulcer disease and gastric cancer, which in turn can result in overt or occult bleeding resulting in iron deficiency anemia. However, chronic gastritis is the most common finding associated with *H. pylori* infection which is not associated with gastrointestinal bleeding. The proposed mechanism is that the iron uptake is impaired by *Helicobacter pylori* colonization in gastric mucosa which can lead to increase loss of iron and potentially to IDA. The British Society of Gastroenterology suggests eradication of *H. pylori* infection in IDA patients with normal upper endoscopy and colonoscopy (Grade C recommendation), and the Maastricht guidelines recommend eradicating *H. pylori* in patients with IDA (Grade A recommendation).

Conclusion: Gastric *H. pylori* infection may be a frequent cause of iron-refractory or iron-dependent anemia in adult patients, mainly in men and post-menopausal women. In these patients, *H. pylori* infection eradication.

Life Threatening Pericardial and Neurologic Sequelae in a Lupus Patient

Clinical Vignette

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Introduction: Reversible posterior leukoencephalopathy syndrome (RPLS) is a group of etiologies that included posterior reversible encephalopathy syndrome (PRES), reversible posterior cerebral edema syndrome, posterior leukoencephalopathy syndrome, hyperperfusion encephalopathy, and brain capillary syndrome. It is characterized by altered mental status, visual perception abnormalities and seizure. SLE is a rare etiology of PRES and affects less than 1% of SLE patients. The association is secondary to the effect of SLE on endothelial dysfunction and the effect of cytotoxic medications on the loss of auto-regulation of systemic hypertension. It is important to recognize this condition and important associations as early detection and intervention often lead to favorable outcomes. We present a case of a diagnostic and therapeutic challenge in a patient with Lupus-induced pericardial effusion complicated by intractable in-hospital seizures later found to have posterior reversible encephalopathy syndrome (PRES).

Case: A 21-year-old African American female underwent outpatient evaluation for dyspnea, arthralgias and alopecia. Laboratory evaluation returned ANA titer of 1:5210 speckled pattern, ESR of 130 and CRP of 4.94, low C4, normal C3/CH50, high IgG without M spike, anti-Smith+, rNP+, RF+, Ro+, Scl70+, DsDNA -, CCP -, SSA + and nephrotic range proteinuria. She was found to have a large circumferential pericardial effusion with borderline tamponade physiology on TEE. Successful pericardiocentesis revealed inflammatory pattern in the fluid studies. On the fifth hospital day, she developed a tonic-clonic seizure aborted with lorazepam. She was intubated and transferred to the ICU. MRI revealed bilateral parieto-occipital edema consistent with a radiographic diagnosis of PRES. After extubation she had another episode of tonic-clonic seizures and required re-intubation. During both seizures, her systolic blood pressure was relatively normotensive (107-149/62-104). Renal biopsy obtained prior to steroid initiation showed Class V membranous lupus nephritis. Induction therapy with mycophenolate was initiated and follow-up studies showed improvement in the cortical and subcortical regions on MRI and no evidence of pericardial effusion on TEE. Patient was discharged on hospital day 20.

Discussion: PRES is a rare complication of lupus and has been infrequently described in the literature. The prevalence in one retrospective case series has been estimated to be approximately 0.69% among SLE patients. The most common etiologies of PRES include malignant hypertension, eclampsia, chemotherapy agents and immunosuppressive medications. It is thought that the association is secondary to the effect of SLE on endothelial dysfunction and the effect of cytotoxic medications on the loss of autoregulation of systemic hypertension. To our knowledge, this is the first case report of SLE with associations of both PRES and circumferential pericardial effusion with borderline tamponade physiology, both of which are exceedingly rare in the SLE patient population.



Uveitis Caused By Neurosyphilis Clinical Vignette

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Several ocular symptoms are known to be associated with neurosyphilis. Our clinical observations suggested that uveitis is an under-recognized but common ocular manifestation of neurosyphilis that has important short-term and long-term prognostic significance. The low incidence with which it is encountered makes neurosyphilis a diagnostic challenge.

A 56 year old gentleman consulted his rheumatologist and ophthalmologist because of diffuse joint pain, lip swelling, and left eye blurry vision. Additional history revealed that at age 16, he had experienced urethral discharge and dysuria, and after diagnosis with syphilis, was given one dose of penicillin upon which symptoms resolved, but never followed up. A clinical diagnosis of bilateral uveitis and syphilis was made. Upon evaluation by the rheumatologist, an X-ray of all joints was performed, which were negative, and eye drops minimally alleviated symptoms. When seen by his ophthalmologist, the patient reported seeing floaters and was diagnosed with bilateral uveitis, as he was noted to have inflammation of the eyes, associated with ciliary flush. Subsequently, the rheumatologist performed bloodwork and he tested positive for syphilis (RPR 1:128 titer and *Treponema pallidum*). He then presented to the hospital for IV treatment and neurological evaluation. In addition to bilateral uveitis, the physical examination revealed mild impairment of hearing in the left ear during the whisper test. A lumbar puncture revealed CSF VDRL titer 1:1, with WBC 49 (19% segments, 77% lymphocytes), protein 93, glucose 65. Serology workup, as inpatient, revealed RPR 1:256 reactivity along with FTA-ABS reactive. Antibiotic therapy with Crystalline penicillin was initiated. After his first dose of penicillin, he experienced rigors, abdominal pain, nausea and vomiting. Because cultures were negative, symptoms were attributed to a Jarisch Herxheimer reaction, in which antibiotic treatment caused release of spirochetes, resulting in a systemic inflammatory response. Supportive treatment was initiated with IV fluids, Benadryl, and ibuprofen. Subsequently, symptoms subsided and patient was discharged from the hospital with PICC line in place for antibiotic therapy for two weeks. Upon outpatient followup, RPR titers decreased to 1:32, and later 1:2, along with resolution of headaches, visual changes, hearing loss, and arthralgias.

This case illustrates the potential for neurosyphilis to manifest as uveitis and the value of a complete history and physical exam. Although there are many causes of uveitis, syphilis remains an essential differential diagnosis to consider, despite its rare prevalence. Recognition of this manifestation is critical to institution of appropriate therapy and prevention of Inflammatory Ocular Hypertension Syndrome, where intraocular pressure acutely increases secondary to direct inflammation of the trabecula.

Platypnea Orthodeoxia Syndrome: A Diagnostic Challenge

Clinical Vignette

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Platypnea Orthodeoxia Syndrome (POS) is an uncommon and under recognized condition characterized by deoxygenating that occurs as a result from changing position from a recumbent to a standing position.

An 83 year old woman with a history of Alzheimer's Dementia, HLD, and a recent stroke presented to the ED for evaluation of dyspnea and lightheadedness on ambulation. At rehab she had dyspnea on ambulation during physical therapy with a SpO₂ of 80. She denied fevers, chills, cough, chest pain, orthopnea, PND, leg swelling, or ever smoking.

During her hospitalization, she was found to be more hypoxic in the standing position requiring oxygen therapy with high flow and BiPAP. A shunt was suspected. ABG in the supine position showed a pO₂ of 105 on FiO₂ of 60% and ABG in the upright position showed a pO₂ of 85 on 60% FiO₂. A TEE with bubble study showed a PFO vs. a fenestrated atrial septal aneurysm with significant right to left intra-cardiac shunting during the bubble study. The TEE also showed pulmonary hypertension with RVSP of 35 mmHg. V/Q and CTA were negative for PE, infiltrates, congestion, however it did show an ecstatic thoracic aorta and thoracic spine degenerative disease. There was no evidence of an extra-cardiac shunt. She underwent successful percutaneous closure of a PFO aneurysmal septum using a 30 mm multi-fenestrated cribriform septal occlude. Following the procedure, she was weaned of oxygen therapy and able to ambulate without any symptoms. Her A-a gradient improved and she was discharged home on room air. POS is characterized by hypoxemia that is exacerbated by postural changes from the supine to the upright position. This syndrome can be associated with a number of clinical conditions due to cardiac or extra-cardiac shunts. In POS, the hypoxemia is not solely due to increased right atrial pressure causing a right to left shunt. Other factors must be present to influence the intra-cardiac hemodynamics and cause the venous flow to cross the shunt toward the left atrium in the upright position. POS requires the presence of both an anatomical defect such as a cardiac or pulmonary shunt with a functional deformity. The functional deformity requires the redirection of IVC flow towards the atrial septum or a transient change in RV compliance. There are a number of proposed functional factors including aortic dilatation, aneurysms, pneumonectomy, diaphragm paralysis, kyphoscoliosis, thoracic vertebral fracture, lipomatous inter-atrial septum, right atrial mass, pulmonary fibrosis, and single lung transplantation. Our patient had evidence of an ecstatic thoracic aorta with degenerative changes throughout the thoracic spine and an atrial septal aneurysm that could contribute to the functional deformity causing hypoxemia with position changes to the upright position.

**Varicella-Zoster Virus Encephalitis with Focal Deficits but No Rash
in an Immunocompetent Adult**
Clinical Vignette

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Introduction: Encephalitis is an uncommon but serious complication of Varicella-Zoster Virus (VZV) infection. It is often associated with chickenpox rash in primary varicella, localized herpes zoster or disseminated zoster in VZV reactivation. Here we report a rare case of a young immunocompetent patient who presented with focal neurological findings, and was diagnosed with VZV encephalitis in the absence of herpetic rash.

Description: 36 year old Caucasian female with past medical history of migraine, inflammatory bowel disease not on any medications presented to the emergency department with severe generalized headache, right side weakness, and speech difficulty. Upon presentation she was afebrile, with BP 156/79, HR 110. She was awake, alert, agitated, and able to move all extremities but not fully following commands. She was able to articulate words but exhibited a fluent aphasia. She was not found to have any skin rash. Other physical and neurological exams were limited due to uncooperativeness. A stroke code was called and NIHSS performed at bedside was 14. CT and MRI of the brain did not reveal any hemorrhage or other acute pathology. Admission laboratory tests were unremarkable. She did not receive tPA being at low risk for stroke but underwent diagnostic lumbar puncture immediately. Cerebrospinal fluid analysis revealed white blood cell count of 187 with 90% lymphocytic predominance. PCR panel for meningitis and encephalitis revealed positivity for VZV which was further verified by viral DNA quantitative analysis. She completed acyclovir treatment for a total of 21 days, and achieved complete resolution of symptoms and return of baseline functional status.

Discussion: In a young, immunocompetent patient who presents with focal neurological findings and severe headache, differential diagnoses usually include migraine, stroke, subarachnoid hemorrhage, multiple sclerosis, or CNS infections. VZV encephalitis is usually not on the top of the differential diagnoses unless there is a dermatomal rash or pain. This unusual case not only demonstrates that focal VZV encephalitis can occur in immunocompetent patients in the absence of dermatological manifestations, but also proved that CSF analysis with viral and bacterial PCR panel is a vital diagnostic tool in prompt and on-target management of VZV encephalitis.

Tumefactive Multiple Sclerosis in a Patient With Hodgkin's Lymphoma Clinical Vignette

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Introduction: Hodgkins lymphoma (HL) is a malignant B cell neoplasm in a heterogenous reactive cellular infiltrate. Multiple sclerosis (MS) is an autoimmune disease involving polycellular inflammation (T cells, monocytes and B cells), gliosis, axonal and oligodendrocyte pathology. Although very few concurrent cases have been reported so far, these two clinically distinct conditions share common inherited, epidemiological and environmental features. Tumefactive MS is a rare diagnosis with only 1 to 2 per 1000 MS cases presenting initially as tumor-like demyelination.

Case Report: A 19-year-old lady was initially admitted for left axillary lymphadenopathy and new onset progressive left sided weakness. Lymph node cytology, lumbar puncture results and bone marrow biopsy showed histological features of classic HL Ann Arbor stage IIB. MRI brain without gadolinium revealed two well-circumscribed T2 FLAIR hyperintense lesions in the right frontal horn abutting the deep white matter and right thalamocapsular junction attributed to metastatic spread of HL. Steroid treatment was started and chemotherapy was planned. Two weeks later, she reported worsening left sided weakness and paresthesias. Neurological exam revealed 4/5 motor strength in left upper and lower extremity with proportionate dysmetria, reduced fine finger movements, brisk reflexes and left hemiparetic gait. No other neurological deficits were noted. Repeat MRI revealed a larger 1.7cm lesion at right thalamocapsular junction with conspicuous rim like diffusion restriction and surrounding edema leading to diagnosis of tumefactive MS. Patient was treated with a high dose steroid taper and her symptoms resolved with 4 weeks of treatment. Follow up MRI one month later revealed decreased lesion size.

Discussion: HL affects males and females equally and displays a bimodal age distribution, with peaks in young adults (15-34 years) and older adults (over 50 years). Onset is between 20 to 40 years and females are affected three times more than men. Both conditions are observed in similar age groups and mutually cluster in first degree relatives. Two large Danish and Swedish studies showed an increased risk of HL in first degree relatives of MS patients. A meta-analysis studying 404 thousand single nucleotide polymorphisms revealed genome wide overlap in the HLA region. Both have associations with EBV infection and UV light exposure. Tumefactive MS is a rare form, involving a large isolated lesion (usually over 2 cm) and presenting with atypical symptoms mimicking a space occupying lesion. Diagnostic features include typical periventricular location, targetoid appearance and peripheral restriction on diffusion weighted imaging. In our patient, the diagnosis was based on the clinical and radiological features that accompany tumefactive MS and the treatment response. Although the occurrence is rare, the observed associations and genetic overlap between MS and HL warrants a high level of suspicion for timely diagnosis and treatment.

Retrograde Amnesia Caused by Synthetic Cannabinoid (K2 Spice)

Clinical Vignette

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Case Presentation: A 30 year old man with no past medical history was transferred to our hospital for altered mental status after a reported heroin overdose. He was found unresponsive in the field by EMS with an agonal breathing pattern. EMS reports the patient to have overdosed on heroin so he was given naloxone and transferred to the nearest healthcare facility where he was agitated and combative. He was not speaking. He was given a total of 12 mg of lorazepam IV for agitation and 10 mg of haloperidol IM. He continued to be agitated and was transferred to our hospital. Upon arrival to our hospital, he was confused and had no recollection of any events that had happened that day. He denied any prior personal or family history of seizures, and denied using any recreational drugs.

On physical examination, he appeared to be in no acute distress. Vital signs were normal besides him being tachycardic. Cardiac exam revealed a regular heart beat with a visible apical impulse and visible epigastric pulsations that corresponded to the cardiac beat. Head examination revealed no signs of acute trauma and the right hand showed 3 scars from the formerly mentioned tendon repair. Neurologic examination revealed brisk stretch reflexes in both upper and lower extremities with non-sustained ankle clonus bilaterally.

The patient was alert and oriented to person but had some difficulty determining the place and the date. He exhibited a reduced attention span with moderate difficulty in serial subtractions and backward spelling. He had no recollection of what he had eaten for breakfast that day, the reason for which he was at the hospital or any events that occurred that day prior to the event of note. The blood cell counts were normal, as were results of the basic metabolic panel including a normal renal function tests. Creatinine kinase was elevated at 9500 units/liter. A CT scan of the head was negative. Urine drug test was negative.

Working diagnosis of drug overdose likely from artificial marijuana was made as urine toxicology was negative. Patient was adequately hydrated with normal saline and closely monitored. After 2 days patient started to remember set of events that happened and he admitted of using K2 (synthetic cannabinoid) before losing consciousness.

Discussion: This case illustrates one of the wide range of side effects that can happen with synthetic cannabinoids including retrograde amnesia and rhabdomyolysis as we report in our case. Urine toxicology of this drug is negative on routine screening and it requires more sophisticated and analytical approach for detection which is not available in our hospitals yet. Joint collaboration between agencies like health department, law enforcement and research laboratories is needed to tackle this outbreak.

Contrast Induced Neurotoxicity After Cardiac Catheterization Clinical Vignette

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Introduction: Contrast induced neurotoxicity (CIN) is a relatively rare complication with incidence reported to be 0.2-0.4%. Risk factors include advanced age, male gender and hypertension. It may present with non-specific symptoms like headache, nausea, vomiting, but in severe cases, may manifest as dysarthria, encephalopathy, hemiparesis, global amnesia, cortical blindness and internuclear ophthalmoplegia. We report a case of an 86 year-old male who was suspected to have developed CIN post PCI.

Case Presentation: 86 year old Asian male with past medical history of hypertension, hyperlipidemia, coronary artery disease s/p PCI and complete heart block s/p PPM presented with unstable angina for 3 days. He had 60 pack-year smoking history and quit smoking 10 years ago. He denied alcohol or illicit drug use. Patient's admission vitals and physical exam were unremarkable with no neurological deficit. Laboratory data revealed microcytic anemia with Hgb of 10.5 g/dl.

Patient was started on heparin infusion and underwent left heart catheterization with placement of DES to LAD. Total of 185 ml of non-ionic contrast media (iopamidol) was used during the procedure.

Patient was transferred to critical care unit with normal neurological function. However, about 10 hours later, he started complaining of diplopia and nausea. On neurological examination, he was found to have right eyelid ptosis and internuclear ophthalmoplegia involving both the medial recti. Pupils were miotic and reactive bilaterally. Rest of his neurological examination was normal.

CT scan of the head was negative for intracranial hemorrhage, infarct or mass. Due to his pacemaker, MRI was not a feasible option. CTA of the head and neck demonstrated stenosis in bilateral intracranial vertebral arteries. Patient's symptoms persisted with repeat CT scan planned to be done outpatient. Repeat CT scan, one week after onset of his symptoms was also negative for ischemic or embolic stroke validating the possibility of CIN.

Discussion: Pathophysiology behind CIN is complex and poorly understood. Involvement of the vertebrobasilar circulation has been reported in more than 50% cases of cardiac catheterization-associated neurologic events. The proposed mechanism of neurological insult is micro emboli precipitated by cardiac catheterization. We speculate that in our patient atherosclerotic burden involving the intracranial vertebral arteries might have caused micro emboli to the posterior circulation leading to neurological deficit. Furthermore, he had the risk factors of advanced age and hypertension and received a high dose of contrast causing disruption of the blood brain barrier and subsequent neurotoxicity.

Contrast induced neurotoxicity is a rare entity and should be considered as the probable cause of acute focal or generalized neurological symptoms that develop after procedures involving the use of iodinated contrast media. Although complete resolution can take up to a few months, the likelihood of persistent deficit is higher with ophthalmic involvement.

**Coronary Artery Ectasia Presenting as a non-ST Segment Elevation Myocardial
Infarction in a Young Adult**
Clinical Vignette

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Introduction: While acute coronary syndromes most commonly occur secondary to unstable atherosclerotic plaque, coronary aneurysms, also known as coronary artery ectasia (CAE), represent a less common etiology. Whereas coronary atherosclerosis accounts for about 50% of CAE, the remaining 50% are either congenital or secondary to a host of inflammatory and connective tissue disorders, with Kawasaki disease being a well-known association. Patients with CAE have worse outcomes than the general population regardless of the presence of associated atherosclerotic coronary artery disease.

Case Presentation: A 26-year-old man presents to the emergency department with atypical chest pain, a right bundle branch block on EKG and a positive troponin. His medical history, as well as his social history, is unremarkable. His family history is notable for hypertension, diabetes and peripheral arterial disease in his father and paternal grandfather. His BMI is 28. Vital signs are normal. CBC, CMP and a lipid panel are normal. HbA1C is 5.6%. Echocardiography reveals inferior wall akinesis and coronary angiography reveals moderately to severely dilated coronary aneurysms in the proximal segments of all three main coronary vessels with no flow-limiting lesions and no evidence of coronary atherosclerosis. The patient has no recollection of any febrile childhood illness that would be consistent with Kawasaki disease. The patient's aneurysms are probably congenital. He is discharged on dual antiplatelet therapy and a high-intensity statin, in addition to metformin for a new diagnosis of prediabetes.

Discussion: Coronary aneurysms are defined by a dilatation of a coronary artery segment to more than 1.5 times the diameter of the adjacent normal segment. Coronary atherosclerosis is the most common cause of CAE; accounting for about 50%. The next most common cause is congenital; accounting for 20-30%. Coronary vasculitis, connective tissue disorders, mycotic aneurysms and angioplasty have all been implicated as etiologies of CAE. There is a strong association between familial hypercholesterolemia and CAE with the incidence about 6-fold. Reported prevalence of CAE varies widely between angiographic studies from 0.3% to 12%.

In the absence of atherosclerotic coronary artery disease, CAE could cause chest pain via the coronary slow flow phenomenon or via aneurysm thrombosis secondary to stagnant non-laminar flow that is not related to plaque rupture.

Medical management of CAE in adults is largely anecdotal. Antiplatelet agents are used on the premise of an increased risk of aneurysm thrombosis and anticoagulation is suggested for larger caliber aneurysms. Percutaneous intervention is sometimes challenged by the large and non-uniform caliber and tortuosity of the lesions. Solitary bypass grafting is suggested as the intervention of choice for non-giant aneurysms by some authors. Other surgical options include aneurysm ligation, resection or marsupialization with interposition grafting.



Herbal Trouble! A Rare Case of an Ayurvedic Diabetes Remedy Causing Lead Poisoning Clinical Vignette

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Introduction: Complementary and alternative medicine use is as widespread in the United States today as it is across the globe. Its use is assumed to be safe and free from side effects. In addition to organic compounds Ayurveda uses lead, arsenic and mercury as active elements in its formulations. Cases of lead toxicity have been reported with the use of ethnic remedies when used for the treatment of haemorrhoids, psoriasis and tooth pain. We report a rare case of chronic lead poisoning from an herbal remedy used for diabetes management.

Case: A 60-year-old Indian male presented with complaints of multiple bouts of squeezing, non-radiating abdominal pain all over his belly for the past 5 years which had worsened in intensity and frequency over the last week. Peaking to an intensity of 10/10 in minutes the pain was often accompanied by nausea but not vomiting, fever, sweats, chills, rashes, flatulence, photosensitivity or urinary complaints. He recognized no aggravating factors or relation to food intake. Ibuprofen and acetaminophen had provided only marginal relief. Extensive testing over the past 5 years including computed tomography scans, ultrasounds and endoscopies had yielded no answers. His other complaints were significant for diffuse vague aches and pains, headaches, constipation, tingling in his feet, recent erectile dysfunction, insomnia and irritability. He was on treatment for diabetes, hypertension and anemia. His medications included: metformin, insulin (glargine & aspart), aspirin, simvastatin, losartan, iron and “Madhumehatakvati” – an herbal supplement for diabetes management from India which he had been consuming for the last 10 years. He lived in a house built in 1985, worked as a researcher at Colgate, did not drink or manufacture his own alcohol or abuse illicit drugs. His vitals were stable, conjunctivae were pale, and the junction of the teeth and the gums showed discolouration (Burton’s Lines). Abdomen was non-tender with no organomegaly. Both feet showed decreased sensation to monofilament testing.

Lab analysis showed microcytic anaemia (Hb 8.8 gm/dl; MCV 78.8 fL), basophilic stippling on peripheral smear and markedly elevated serum lead (142 mcg/dl) and zinc protoporphyrin levels (718 mcg/dl). Plain abdominal radiograph showed 5 pill-sized radiopaque foreign bodies. He was started on chelation with succimer and poison control was notified.

Discussion: In Ayurvedic medicine, lead is considered an aphrodisiac and its presence in diabetes preparations may be to counter the impotence associated with diabetic neuropathy. Analyses of some of these remedies have often found lead concentrations thousands of times the FDA’s upper limit of normal for water or candy. With the Ayurvedic market projected to grow at 16% over the next decade, this case questions the need to potentially revisit dietary supplements regulation in the United States.

**A Case Report of Drug Interactions Leading to Hypoglycemia: An Avoidable
Hospital Admission**
Clinical Vignette

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Objective: Drug interactions caused by Cytochrome P450 [CYP450] inhibitors and inducers are rarely recognized by general internists in clinical practice. So we would like to present our experience with an interesting clinical presentation of drug interaction.

Background: Severe hypoglycemia and elevated international normalized ratio [INR] from the interaction of clarithromycin and other drugs.

Case Report: A 77-year-old female with past medical history of well-controlled diabetes mellitus type II, hypertension, prior cardiac stenting and gallstone pancreatitis with pseudocyst presented with malaise, dizziness, sweating, and nausea. The patient reported decreased appetite for two months prior to starting *Helicobacter pylori* treatment with clarithromycin, amoxicillin, and omeprazole. She continued to take antidiabetic medications without dose adjustments, despite reduced appetite. Physical exam was unrevealing including no abdominal tenderness. On admission, her blood glucose was 30 mg/dl. Interestingly, her international normalized ratio was elevated to 7.1. During the hospital course, the hypoglycemia and INR improved. The patient was discharged on insulin regimen and coumadin. Recommended to follow-up with the primary care physician within one week of discharge.

Conclusion: Patient was started on triple *Helicobacter pylori* treatment without regards to the concomitant medical regimen including coumadin and sulfonylureas. She presented with dizziness and hypoglycemia and was incidentally found to have a suprathreshold international normalized ratio. This is due to increased levels of active metabolites of sulfonylureas and coumadin due to inhibition of CYP450 by clarithromycin.

Discussion: An estimated 56% of elderly population >65 years age are hospitalized for the peptic ulcer disease are positive for *Helicobacter pylori* infection. This age group has a high prevalence of diabetes and cardiovascular disease. The sulfonylureas and warfarin are the main drugs to be prescribed by the physicians. These drugs are metabolized in the liver by CYP450 to inactive metabolites. Clarithromycin inhibits liver CYP450, thereby increasing the levels of active sulfonylureas and warfarin. This potentiates the hypoglycemic effect and the risk of bleeding by increasing INR. Internists must be cautious before prescribing drugs which are inducers or inhibitors of CYP450. It is advised to adjust the dose of these drugs within 7 days of prescribing clarithromycin.

Laugh Till Your Feet Drop! Nitrous Oxide Begetting Vitamin B12 Deficiency Clinical Vignette

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Introduction: I felt a high, an extraordinary degree of pleasure, different from that produced by wine . . . and yet approaching nearer to it than any other sensation I am acquainted with.”

-Sir Humphrey Davy

The effects of nitrous oxide (N₂O) inhalation have been reported as far back as the 18th century, with its euphoria described as a brief but vivid intoxication - combining powerful euphoria with distortion of time, space and sensation. Today much as in Davy's time, students abuse this easily accessible agent for its euphoriant properties. We report a case of severe myelopathy culminating in bilateral foot drop secondary to its abuse.

Case: A 20-year-old woman started to feel numb on her soles 2 months prior to presentation. The numbness progressed in a sock like distribution up to her knees. Subsequently, she experienced difficulty walking, imbalance and began dragging her toes. On presentation, her debility had progressed to an inability to raise her feet or wiggle her toes. Prior to symptom onset, she had been inhaling 5-10 cartridges of “whippets” daily for 2½ months by filling them in balloons. B12 supplements given by a friend experiencing similar symptoms had halted the progression of her symptoms. On examination, she had a GCS of 15, bilaterally obtunded fine touch, tactile localisation, 2-point discrimination and vibration perception up till the L5 dermatome with foot drop. Lehermitte's sign was positive, Babinski's sign was negative and her gait was high steppage.

Investigations revealed hemoglobin: 14.2 g/dl, MCV: 88.8 fL, Vitamin B12 level: 874 pg/ml, homocysteine: 17.3 umol/L and normal methylmalonic acid level. T2/Short tau inversion recovery (STIR) signal abnormality was seen in the cervical dorsal column extending from C2-C6 with concomitant corticospinal tract involvement. A diagnosis of subacute combined degeneration of the spinal cord (SCDS) was made secondary to Vitamin B12 inactivation by N₂O. She received Vit. B12 injections and by 2 months achieved almost complete recovery.

Discussion: A potent oxidising agent, N₂O oxidises the body's reserves of Vitamin B12. This halts homocysteine methylation and isomerization of methylmalonyl CoA to succinyl CoA. DNA synthesis is impaired and accumulated substrate participates in fatty-acid synthesis that deposit in myelin thus impairing nerve conduction and precipitating SCDS. Our patient's normal Vitamin B12 and methylmalonic acid and low homocysteine levels is explained by toxic Vitamin B12 inactivation with partial resolution from self-administered B12 supplementation. Patients with N₂O intoxication may present with normal haematology parameters and low/normal Vitamin B12 levels. This should not misguide clinical suspicion as sometimes it is the Vitamin B12 activity which may be impaired and not the levels themselves. It is the elevated homocysteine and normal/elevated methylmalonic acid which helps clinch the diagnosis.

Paraneoplastic Pseudoachalasia Secondary to Ovarian Carcinoma: A Hard Pill to Swallow

Clinical Vignette

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Introduction: Pseudoachalasia is the dilatation of the esophagus which mimics achalasia, with narrowing of the distal esophagus but from reasons other than primary denervation. Paraneoplastic pseudoachalasia has been reported in cases of small-cell and adenocarcinoma of the lung, carcinoma of the gastric cardia, pleural mesothelioma and bronchial carcinoid but never from the ovary. Here we report the first such case.

Case: A 78-year-old Caucasian female with stage IIIc ovarian cancer diagnosed 1½ years ago presented with the complaint of persistent vomiting that began 8 months ago but which had acutely worsened over the week preceding presentation. The frequency of episodes had increased from 3-4 a day to 10-12 a day. She had also unintentionally lost 15 lbs. over the last 2 months. On evaluation, she appeared pale and cachectic. Mucous membranes were dry. Her abdomen was distended and revealed scars of a previous TAH-BSO. She was not tender to the touch and had normoactive bowel sounds. Lower extremities exhibited pitted edema up to the ankles. A plain radiograph of the chest revealed a widened mediastinum. CT scan showed a megaesophagus with an air fluid level but without obvious stricturing at the cardio-esophageal junction. This was compared with a CT scan done 1 year ago which showed a normal appearing collapsed esophagus. Esophagogastroduodenoscopy revealed an aperistaltic, uniformly dilated esophagus filled with 3 liters of fluid and undigested food. The scope was navigated through the lower esophageal junction without much resistance and biopsies were taken. Given the rapidity of onset of her symptoms, her advanced age, diffuse involvement of the esophagus, lack of esophageal denervation (characteristically seen in achalasia cardia and absent in pseudoachalasia) and no visible mural pathology, in the setting of active malignancy - she was diagnosed with paraneoplastic antibody mediated pseudoachalasia from the ovarian cancer. Paraneoplastic antibody panel tested positive for ANNA-1.

Discussion: On rare occasions, as seen in our patient, malignancy can give rise to paraneoplastic antibodies which interfere with the neuromuscular transmission at the myenteric plexus leading to pseudoachalasia. In such cases, no gross anatomic pathology can: paraneoplastic motility disturbance is ANNA-1 (Anti-Hu). Molecular mimicry and subsequent cross reactivity between onco-neural antigens and neurons in the myenteric plexus is responsible for esophageal dysfunction and achalactic transformation. Ovarian carcinomas are known to produce ANNA-1. However ovarian carcinomas have never been linked to Pseudoachalasia, making this case a first of its kind. Rapidly progressing achalasia like symptoms (mean duration of 10 months) in an older population, with evidence of weight loss and in the setting of active malignancy such as in our patient should always raise suspicion for Pseudoachalasia.

Pet or Host? A Case of *Pasturella Multocida* Spontaneous Bacterial Peritonitis and Bacteremia

Clinical Vignette

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Introduction: We present a rare case of bacteremia associated with spontaneous bacterial peritonitis (SBP) to review the importance of a detailed social history, including pet and animal exposures. We also offer the diagnostic and management review, a rare cause of SBP, and highlight the importance of timely initiation of antibiotics in suspected SBP.

Case: A 73 year old male with Hepatitis C and Alcoholic liver disease presented with two days of worsening dyspnea and confusion. Two days prior to presentation, he was feeling weak, tripped, and fell in the kitchen but did not lose consciousness or sustain injury. He was unable to stand by himself and denied preceding fever, chills, or abdominal pain. Vitals were stable on admission, and physical exam revealed a soft abdomen without any obvious fluid thrill. Laboratory exam revealed hyponatremia of 123 g/dL and transaminitis consistent with alcohol induced liver injury, (AST 211: ALT 89: ALP 121) lactic acid 4 mmol/L, white blood count 23K c/mm³. Blood cultures grew gram negative rods in less than 12 hours. Based on the history, a diagnosis of spontaneous bacterial peritonitis was suspected an ultrasound guided paracentesis was performed. The patient was given 1L fluid bolus and started on piperacillin-tazobactam and one dose of gentamycin. Peritoneal fluid was positive for *Pasturella multocida*. After furthering questioning, the patient mentioned he had recently been around his neighbor's puppy the past one week and it had licked a cut on his arm several times.

Discussion: *P. Multocida* a gram negative rod, and part of the normal oral flora isolated in 25% of dog bites and 60% of cat bites (II) It is rare cause of SBP in cirrhotic patients and patients receiving peritoneal dialysis. Disseminated infection with *p. multocida* has shown to have a mortality rate of 20-30% in immunocompromised hosts, there are less than 20 cases described in the literature. Early broad spectrum empiric antibiotics, with prompt narrowing to targeted treatment along with treatment of the underlying liver disease is important in management of this severe infection. This case highlights the severe effects an infection with *p. multocida* can have on an immunosuppressed patient and the importance of prompt paracentesis and blood cultures to obtain the causative agent of infection and targeted coverage.

IgG4 RD with Pancreatic Mass

Clinical Vignette

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Introduction: The IgG4-related disease is an increasingly recognized immune-mediated condition comprised of a collection of disorders. The commonly shared features include tumor-like swelling of variable organs, a lympho-plasmocytic infiltrate with IgG4-positive plasma cells and a “storiform” patterned fibrosis. Focal chronic pancreatitis and autoimmune pancreatitis are the two benign processes that are most commonly mistaken for pancreatic malignancy on CT or ultrasound. Here we described a case of IgG4 related pancreatic mass.

Case Description: A 48-year-old male with diabetes mellitus presented with yellowish discoloration of urine and skin for one month. This was associated with generalized itching and pale stool. Within one year prior to presentation, the patient lost almost 70 pounds of weight. No history of fevers, chills, abdominal pain, change in bowel frequency or blood transfusion. Vitals on admissions were within normal limits. Initial physical examination showed a thin male with icteric sclera, temporal wasting, thin extremities with yellow-colored skin and excoriation from scratching.

Initial investigations on admission showed T. Bilirubin 18.1 with ALP 255 and normal AST, ALT, Lipase and PT/INR. MRI abdomen showed intrahepatic and extrahepatic biliary duct dilation. Hepatitis B, Hepatitis A, Antimitochondrial antibody and Anti-smooth muscle antibody were negative. CA 19-9 was 41. EUS showed hypoechoic irregular 30 mm X 20 mm pancreatic head mass with celiac lymphadenopathy. The patient underwent ERCP and sphincterotomy and stent placement. Cytology showed benign glandular cells without malignant cells. Even after stenting patient’s bilirubin was increasing, therefore patient was scheduled for surgery for removal of the mass. Before the surgery IgG4 level was requested which was 539 mg/dl and biopsy was obtained which showed intrahepatic and intracanalicular without fatty changes or Iron deposition and it was positive for IgG plasma cells with IgG4. We canceled the surgery, the patient was started on oral glucocorticoids and discharged. On follow up, patient improved significantly and T. Bilirubin was 0.4

Discussion: This patient presented with biliary tract obstruction. The diagnosis and management represented a challenge. Although high initial suspicion for pancreatic cancer on further investigation, the raised IgG4 and response to therapy guided us towards the diagnosis of this rare entity. Because the epidemiology of IgG4 is not well understood always consider this entity on the investigation of a pancreatic mass, therefore EUS-guided biopsy is recommended if a diagnosis of chronic or autoimmune pancreatitis is suspected.

Extramedullary Hematopoiesis Presenting as Massive Hemothorax Clinical Vignette

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Introduction: Patients with Thalassemia Intermedia (TI) live in a constant anemic state. The physiological response to this chronic anemia is to produce red blood cells in other organs outside medulla of the bone-like spleen, lymph nodes, lungs or pleura. This case is an example of extramedullary hematopoiesis that presented as a massive hemothorax.

Case Description: A 43-year-old male presented to the emergency department complaining of sudden onset, bilateral, pleuritic chest pain that woke him up from sleep. It was characterized by a sharp pain that worsened with deep inspiration and was associated with shortness of breath. He had a past medical history of thalassemia intermedia. On arrival, the patient was hypotensive (Blood Pressure - 94/51 mm Hg). On physical examination he was found to have pallor, dullness to percussion on the right hemithorax, decrease air entry on the right side, massive splenomegaly, and hepatomegaly. Laboratory results showed a hemoglobin 6.6 mg/dl a mean corpuscular volume of 61.7, and a red blood cell number of 3.48, consistent with thalassemia. Chest X-ray revealed a right lower lobe opacity suggestive of pleural effusion. Right-sided Thoracentesis was done and 1250 ml of sanguineous fluid was removed, followed by right-sided chest tube placement. Pleural fluid cytology was negative for malignant cell, and culture and strain negative for microorganisms.

Despite multiple transfusions, patient's hemoglobin continued to drop. Chest computed tomography angiography showed multiple paravertebral soft tissue masses, the largest measuring 4.2 cm x 5.8 cm on right. Subsequently, thoracic aortography demonstrated abnormal neovascularity in the area of mass in posterior right chest supplied by 7th and 8th ICA and the feeding arteries were embolized. The patient underwent video-assisted thoracoscopy surgery for mass removal and biopsy confirming the histopathological diagnosis of extramedullary hematopoietic tissue.

Discussion: Extramedullary hematopoiesis refers to blood cells production outside of the bone marrow. It occurs in hematological disease, either due to bone marrow replacement (myeloproliferative disorder), or hemolytic anemia with ineffective erythropoiesis (thalassemia, hereditary spherocytosis).

Hemothorax due to EMH is a rare and atypical presentation but should be considered as a differential in the patient with a hematopoietic stimulus, not many case report has been documented in the past. The incidence of EMH in patients with TI may reach up to 20% as compared to poly transfused Thalassemia Major patients where the incidence remains <1%. Orient study reveals extramedullary hematopoiesis complicates 19% of non transfusion-dependent thalassemia intermedia.

Mainly involves spleen, liver and/or Lymph node. In rare cases, EMH can present as a mediastinal mass. It is particularly important due to its critical presentation and challenging management.

Neglected Tropical Disease in New Jersey

Clinical Vignette

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Introduction: Dengue Fever is caused by rapidly spreading mosquito-borne RNA virus. Patients presenting symptoms may range from asymptomatic fever to more complicated course like hemorrhagic fever and shock including death. The incubation period of Dengue infection ranges from 3 to 14 days and symptoms typically develop between 4 and 7 days after the bite of an infected mosquito *Aedes Aegypti* or *Aedes Albopictus*.

Case Description: 32 M comes in with the history of high-grade fever and chills for 5 days. He came back to the US from the Southern part of India 11 days prior to presentation and his symptoms started 4 days prior to presentation to hospital. He had severe pain in extremities and abdomen, also had a headache, generalized weakness, nausea, vomiting, diarrhea, and sore throat. On presentation, his physical exams were grossly normal and tourniquet test done was negative. His platelets count dropped to 19 from 152 and also had hemoconcentration. Malaria smear was negative. He also had mild transaminitis. Considering him to be a high risk for hemorrhagic conversion, he was transferred to ICU for close monitoring and managed symptomatically. He received one unit of platelets and intravenous fluids. Once his platelet count and hemodynamics improved, he was transferred to the regular floor. Blood cultures were negative. ID was consulted and CDC was also informed. His IgM and IgG dengue serology were positive.

Discussion: Dengue fever is a neglected tropical disease in the USA but there have been few instances of epidemics in some states of USA- Hawaii, Florida, and Texas. Although it is a self-limiting condition, the early and accurate diagnosis is very important to reduce mortality. Our patient was discharged home with improvement in platelet counts and hemodynamics. This case highlights the importance of recognizing Dengue fever early in New Jersey due to the high traveling immigrant population. Early recognition of a disease reduces its mortality. Physicians need to be alert about tropical diseases even in a temperate zone such as New Jersey. Practitioners in areas with dengue mosquito vector should be aware of the risk of imported dengue resulting in an epidemic.

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A Case of Spontaneous Right Upper Extremity Swelling in a Young Adult Male

Clinical Vignette

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Background: Isolated spontaneous thrombosis of the right subclavian vein is a rare clinical problem. Paget-Schroetter Syndrome or “effort” thrombosis is the name for this rare syndrome that presents with swelling and pain of the right upper extremity in an otherwise healthy athletic person.

Case Presentation: A 22-year old African-American male, with no past medical history, presented with 48-hour history of right upper arm swelling and pain, without any other symptoms. There was no history of anabolic steroids use, intravenous drug abuse or family history of thrombophilia. Physical examination showed edema of the proximal part of the right upper extremity, mildly cyanotic fingernails and tenderness over the biceps and lateral shoulder areas. The left arm was normal. The initial laboratory studies including complete blood count, electrolytes, and liver tests were normal. The thrombophilia panel was normal. Ultrasound venous duplex revealed an almost-occlusive thrombus in the right subclavian vein with a free-floating thrombus at the proximal axillary vein. Heparin infusion was started for 48 hours with minimal-to-none improvement, subsequently receiving catheter-directed thrombolysis with tissue plasminogen activator under venography. Catheter thrombolysis by interventional radiology failed to lyse the clot within 24 hours. Catheter exchange leading to clearance of the clot burden 48 hours later, was performed. He was discharged on rivaroxaban and scheduled for decompressive surgery.

Discussion: Primary subclavian vein thrombosis is a rare entity and its true incidence is estimated to be 1-2 per 100,000 per year. Most commonly found in males (2:1 ratio) and usually reported with a history of repetitive exercise, with propensity on the dominant side of the patient. All these features were present in our patient case, pointing by the diagnosis of Paget-Schroetter Syndrome. The diagnosis of Page-Schroetter Syndrome is established by the typical findings of edema of the arm, followed by simple ultrasound duplex of the upper extremity. Differential diagnosis should include possible occult malignancy, in this case, testicular cancer. Ultrasound venous duplex of the right upper extremity was found to be 78%-100% sensitive and 82%-100% specific, making it the initial diagnostic test of choice. The role of venography as a gold standard is seen where the ultrasound studies are indeterminate or when an invasive intervention is planned. Venography will exhibit an occlusion of the subclavian vein distal to the costoclavicular junction (Figure). Hypercoagulable states can be seen in up to 67% of subclavian venous thrombosis, therefore a hypercoagulable workup is indicated as it will further modify anticoagulation management.

Conclusion: Paget-Schroetter Syndrome is a rare, yet straightforward diagnosis, where a thrombophilia workup is indicated and providers should be vigilant for thrombolysis-catheter failure.

POSTER PRESENTATIONS
STUDENTS

Nickel Dental Implant and Recurrent Asthma

Clinical Vignette

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Introduction: Bronchial asthma is a common clinical presentation which may have several identifiable and non-identifiable trigger factors.

Case Presentation: A 15-year-old female with asthma presented with progressive worsening of coughing and vomiting during the night, occasional wheezing, and a scaly pruritic rash on the dorsum of her hands. Patient had suffered multiple exacerbations of asthma throughout her life. Her medications included albuterol multi-dose inhaler, inhaled corticosteroids, and montelukast, without much relief of her symptoms. Her mother recalled worsening of her symptoms weeks after a dental procedure; which required a nickel crown. Due to the pruritic rash on her hand and worsening of her asthma symptoms, she was tested for nickel allergy; which was positive. Her dental crown was removed. She started seeing significant improvement after 6 weeks of removal. A year later, the patient no longer required medical management for asthma. She experienced only one asthma attack since the removal of the cap, but remained asymptomatic subsequently.

Discussion: Bronchial asthma has many common triggers such as exercise, infections, and allergens (dust, pollen, animal dander). However, other triggers such as nickel are often more associated with contact dermatitis and rarely considered when it comes to asthma exacerbations. Nickel is found almost everywhere from jewelry, coins, and cellphones to braces, dental crowns, stents, and knee replacement implants. No routine testing for nickel allergy exists in the general or asthmatic population and the exact prevalence in the U.S. population is unknown to justify such testing. In one study a significant difference in worsening of nasal flow was observed in patients with nickel allergy before exposure to nickel and after the nickel provocation test. Avoidance of nickel exposure in this group resulted in reduction in nasal and bronchial symptoms, such as cough and dyspnea, as well as improvement in peak expiratory flow and FEV1. This showed that there may be a correlation between nickel allergies and obstructive airway disorders.

Conclusion: This case demonstrates the importance of paying attention to common preventable environmental factors, such as nickel exposure, when it comes to treating asthma in order to improve quality of life and reduce the need for medications.

Warfarin Related Nephropathy in a Patient with Underlying Chronic Kidney Disease Clinical Vignette

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Introduction: Approximately 4 million people in the United States are currently taking warfarin for anti-thromboembolic therapy. Warfarin is known to be a culprit for bleeding, but acute kidney injury due to warfarin itself has been more recently described as warfarin related nephropathy (WRN).

Case presentation: A 66 year-old male with history of laryngeal cancer status post radiation, COPD, hypertension and CKD-III presented with acute onset shortness of breath. His physical examination was remarkable for atrial fibrillation with heart rate of 180 beats per minute. The rest of the vital signs were within normal range. The rest of the examination was normal. His electrocardiogram showed atrial fibrillation with increased heart rate. His serum creatinine was 4.0 mg/dL, BUN 45 mg/dL, hemoglobin 11.5 g/dL. The rest of the complete metabolic panel, complete blood count, troponins, TSH were within normal limits. Patient received medical management for rate control for atrial fibrillation with rapid ventricular rate, followed by warfarin 5 mg daily therapy. After 5 days his INR increased to 3.2 and serum creatinine increased to 8.0 mg/dL. The etiology of his rapid decline in renal function remained unclear even after extensive diagnostic workup, including normal renal ultrasonography. A renal biopsy showed mild diffuse mesangial proliferative glomerulonephropathy with multifocal RBC casts and diffuse acute tubular injury. A diagnosis of WRN was made. Patient was started on hemodialysis and subsequently he remained stable and hemodialysis dependent.

Discussion: Since 2009, cases of unexplained acute kidney injury has revealed a rare and novel entity called WRN. WRN is defined as a rise in serum creatinine by 0.3 mg/dL with concomitant rise of INR greater than 3.0. Histologic findings include presence of glomerular hemorrhage and acute tubular obstruction from RBC casts. WRN occurs more frequently in patients who have underlying chronic kidney disease (CKD) but can also occur in non-CKD patients. Warfarin increases the risk of hemorrhage in the microvasculature of the kidneys causing progressively worsening renal disease, especially in patients with underlying CKD. Patients diagnosed with WRN have been noted to have increased mortality within two months of diagnosis, or they develop accelerated progression of CKD. A retrospective analyses of patients who experienced over-anticoagulation (INR>3) 37% had unexplained AKI, making WRN a reasonable possible etiology and may be more prevalent than previously thought. Given the risks of WRN, a cautious approach towards the dosing of warfarin in the setting of CKD may prove to improve mortality rate of CKD patients receiving warfarin therapy.

Conclusion: Anticoagulation with warfarin has been a standard therapy for stroke prevention and treatment of thromboembolic diseases. It is important for the medical community and our patients to understand the risks associated with its use prior to initiating therapy, especially WRN.

**A Case of Pembrolizumab Induced Diabetic Ketoacidosis
in a Patient with Tonsillar Squamous Cell Carcinoma**
Clinical Vignette

Spencer Ng; Dawnette Urcuyo, MD
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Case presentation: A 60-year-old male with a diagnosis of metastatic squamous cell carcinoma of the right tonsil (T1N2Mx) and papillary thyroid carcinoma (TxN1Mx) on current treatment with Pembrolizumab presented with a two-day history of increased urinary urgency and frequency. He was initially diagnosed with tonsillar squamous cell carcinoma in 2016 and was treated with Cisplatin and radiation therapy. His cancer persisted despite three months of treatment. Palliative immunotherapy with Pembrolizumab began in September 2017. The patient presented to the hospital with the symptoms of urinary incontinence and increased urgency after receiving his second cycle of Pembrolizumab. On admission to the hospital, his blood glucose was 536mg/dL, anion gap of 28 and a beta-hydroxybutyrate level of 6.18mmol/L. He was found to have an HbA1c of 7.9%. Prior to his treatment, his random blood glucose readings ranged from 85-104mg/dL. He had no personal or family history of diabetes. Islet Cell Antibodies and Insulin Auto-Antibodies (<0.4U/ml) were negative. However, he did have a mildly elevated anti-glutamic acid decarboxylase antibodies [anti-GAD 6IU/ml (reference <5IU/ml)]. His hospital course was uncomplicated and his urinary symptoms resolved after treatment of hyperglycemia. He was started on both long-acting insulin glargine and prandial short-acting insulin with improved glycemic control upon discharge. Patient did well after discharge but Pembrolizumab therapy was held due to uncontrolled diabetes.

Discussion: Pembrolizumab is an IgG4 monoclonal antibody that targets PD-1. It is approved for the treatment of metastatic melanoma, renal cell carcinoma and non-small cell lung cancer. It has been associated with the development of type 1 diabetes mellitus in seven patients (incidence of 0.1%). New diagnosis of diabetes occurred between one week and fifty-one weeks after receiving pembrolizumab. The association of immune related adverse events with the use of immune checkpoint inhibitors has been increasingly recognized. Besides type 1 diabetes mellitus, thyroid disorders, hypophysitis and adrenal insufficiency have occurred. Here, we describe a case of diabetes ketoacidosis in a patient who had recently started pembrolizumab therapy. In most cases, the patient had positive anti-GAD and ICA, suggesting an association of PD-1 inhibitors with autoimmune diabetes. Of note, diabetes does not seem to resolve spontaneously after termination of the treatment and requires long term insulin therapy.

Conclusion: The PD-1 inhibitors have been associated with multiple autoimmune adverse events. Although rare, pembrolizumab induced autoimmune diabetes can occur and has become more frequently recognized in recent years.



1st Place
Student

When Diarrhea Dominates, Check the Duodenum for Rare Possibilities

Clinical Vignette

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Introduction: Chronic refractory diarrhea can be challenging in terms of finding an accurate diagnosis, which at times, can be due to a rare but correctable etiology.

Case: A 43-year-old Caucasian male with hypercholesterolemia and history of injection heroin abuse on methadone maintenance presented with intermittent, crampy abdominal pain and 5-6 watery bowel movements daily for the previous 6 months. He was taking Atropine-Diphenoxylate to manage his symptoms with minimal relief. He denied fever, heartburn, weight loss, hematochezia, melena, anorexia, mucous or blood in stool, or recent international travel. His vital were within normal limits. Physical examination revealed a soft, non-tender, non-distended abdomen. His complete metabolic panel, complete blood count, thyroid stimulating hormone and erythrocyte sedimentation rate were unremarkable. Abdominal ultrasound revealed no abnormality. CT abdomen identified a filling defect within the proximal small intestine. Follow-up small bowel series localized the filling defect to the 4th portion of the duodenum. Stool evaluation revealed borderline elevation in fat content with a normal WBC count and negative culture and ova and parasite. Stool osmolality was less than 50 mOsm/kg, suggesting a secretory form of diarrhea. Twenty-four hour urine 5-HIAA was elevated at 32 mg and octreotide scan demonstrated increased uptake in duodenum. EGD and biopsy of distal duodenum demonstrated tumor cells which were markedly positive for CD56, synaptophysin, and chromogranin consistent with a carcinoid tumor. He underwent resection of the distal portion of the duodenum and proximal jejunum with removal of 2.6 cm tumor with pathology consistent with grade I neuroendocrine carcinoma with no angiolymphatic invasion. Since resection, the patient has remained asymptomatic.

Discussion: Carcinoid tumors are a relatively rare, well-differentiated neuroendocrine tumor arising from enterochromaffin cells (EC). The age-adjusted incidence for non-pancreatic primary carcinoid tumors is 4.7 per 100,000. Most carcinoid tumors originate in the gastrointestinal tract (73.7%), with the appendix being the most commonly involved organ; the remainder of tumors originate in the respiratory system (25.1%) and reproductive system (0.56%). Typically, carcinoid tumors are discovered incidentally as many patients are asymptomatic. However, carcinoid tumors can secrete bioactive substances including serotonin and kallikrein. 5-hydroxyindoleacetic acid (5-HIAA) is a degradation product of serotonin that is often elevated in the urine of patients with carcinoid tumors. Carcinoid syndrome is seen in 5% of patients with carcinoid tumors and occurs when bioactive substances enter systemic circulation; patients can present with skin flushing, diarrhea, bronchoconstriction, and secondary restrictive cardiomyopathy. This is an uncommon presentation with primary gastrointestinal carcinoid tumors due to hepatic degradation of vasoactive substances prior to their entry into systemic circulation.

Conclusion: Duodenal carcinoid tumors are a rare cause of chronic diarrhea. Confirmation of secretory diarrhea by stool osmolality assists in further work-up. Early diagnosis and treatment results into favorable outcome.



New Jersey Chapter