RESIDENT ABSTRACTS

PRESENTED:

FEBRUARY 7, 2019 THRU FEBRUARY 9, 2019
Abstract Title: Blastocystis in a patient with Acquired Immunodeficiency Syndrome (AIDS)

Abstract Information:

Introduction: Blastocystis species are anaerobic protozoa that are the most commonly detected parasites on stool ova and parasite smear in the United States. Unlike Giardia lamblia and Cryptosporidium parvum, both of which definitively cause diarrhea in the US, Blastocystis species are not always associated with severe diarrhea. Some experts suggest they are part of the normal gut flora. There is considerable controversy regarding the need to treat if they are encountered on a stool smear.

Case Description: A 40-year-old male with Acquired Immunodeficiency Syndrome (AIDS), colon cancer, and heart failure with reduced ejection fraction presented with nausea, vomiting, diarrhea, and right groin pain after a recent trip to Mexico. He had not been compliant with his medications, dolutegravir and emtricitabine/tenofovir alafenamide, having missed 2 weeks of treatment. He had not followed up with Infectious Disease (ID) for the last 4 months. At that time his CD4 count was 182 cells/μL with a viral load of 67 copies/mL. Upon admission, his lactate was elevated and his colostomy output was increased from usual. His CD4 count was 41 cells/μL with a viral load greater than 50,000 copies/mL. A computed tomography scan (CT) of the abdomen and pelvis demonstrated retroperitoneal lymphadenopathy and a testicular ultrasound demonstrated a small right hydrocele and a right epididymal head 6mm cyst. He was found to have Group C Streptococcus bacteremia as well as Blastocystis species in his stool. Lumbar puncture was negative for infection. He was seen by ID who started him on ceftriaxone for seven days then amoxicillin/clavulanate for seven days. He was started on metronidazole for five days for Blastocystis species isolated in stool culture. Due to his history of poor compliance, he received the full course of intravenous antibiotics as an in-patient.

Discussion: Smaller studies in which Blastocystis species were isolated in stool ova and parasite smear revealed a significant occurrence of copathogenicity, suggesting that the presence of Blastocystis species indicates co-infection with another parasite such as Giardia lamblia. In a 2009 study conducted amongst men with HIV/AIDS in Indonesia with CD4 counts <50, Blastocystis species was most commonly identified with Cryptosporidium parvum. The Infectious Diseases Society of America (IDSA) recommends that treatment be reserved for those patients with gastrointestinal signs and symptoms and many cysts in stool specimens. The drug of choice is metronidazole.
This case highlights that *Blastocystis* species, considered by many to be non-pathogenic and part of normal gut flora, can cause significant diarrhea if parasite burden is high. In these cases, especially when patients are immunocompromised, they should be treated with metronidazole. Otherwise, the IDSA recommends that asymptomatic individuals with few cysts on smear should not be treated.

References:

Leder K, Weller P. UpToDate article: *Blastocystis* species. Dec 05, 2017

Coyle C, Varughese J, Weiss LM, Tanowitz HB. *Blastocystis*: To Treat or Not to Treat... *Clin Inf Dis*, 2012 Jan; 54(1):105-110


Retrieved from https://www.cdc.gov/parasites/blastocystis/health_professionals/index.html
Abstract Title: Essentially Different Hypertension

Abstract Information:

Introduction:
Paragangliomas are an uncommon cause of secondary hypertension. We present a case of a 65-year-old female who presented to the hospital repeatedly for decompensated heart failure and poorly controlled hypertension prior to being found to have elevated plasma and urinary metanephrines and a left periaortic retroperitoneal mass.

Case description:
Patient is a 65-year-old female with past medical history significant for hypertension that presented to the emergency department on three separate occasions in a short time frame. On the first presentation, she reports severe headache and vague chest pain that woke her from sleep. Her initial blood pressure was 230/100mmHg and her cardiac enzymes were mildly elevated. She underwent a cardiac catheterization that revealed no coronary artery disease but she was diagnosed with takosubo cardiomyopathy. She had labile blood pressures and was discharged on a beta-blocker and ACE inhibitor. The patient returned a second time with complaint of shortness of breath, orthopnea, and hypertension. She was found to have pulmonary edema was treated with diuretics. She has systolic blood pressures that ranged from 82-193mmHg during this stay. She was discharged on a lower dose ACE inhibitor and loop diuretic but no beta-blocker. On her third presentation, she again complained of nocturnal headaches. During that admission, plasma and urine metanephrines were elevated and an abdominal/pelvis CT revealed a 4.2x3.6x4.2cm periaortic retroperitoneal mass. She was discharged on and alpha-blocker and beta-blocker. An outpatient I-123 MIBG scan showed intense radiotracer uptake over the tumor which confirmed the suspicion of functional paraganglioma. Her alpha blockade was increased and a repeat echo prior to surgery showed normalization of her left ventricular function. She underwent a robotic laparoscopic excision of the paraganglioma without complication.

Discussion:
Paragangliomas are uncommon tumors originating from chromaffin-negative embryonic neural crest cells. Paragangliomas are extra-adrenal and usually found in the head and neck
or in the abdomen [1]. This case illustrates the significant morbidity associated with these tumors prior to diagnosis and definitive management.

Catecholamine-induced cardiomyopathy is an unusual presentation occurring in approximately 10% of patients ultimately diagnosed with paragangliomas [2]. The pathogenesis of this cardiomyopathy is though to be related to both direct myotoxic effects of catecholamine metabolites as well as related to the vasoconstrictive effect of catecholamines [3]. The presentation is similar to that of takosubo cardiomyopathy and resolves with reduction of catecholamine levels. This patient had classic catecholamine-induced cardiomyopathy with worsening of symptoms after starting on an unopposed beta blockade and complete resolution of symptoms with alpha blockade and ultimate surgical resection of the paraganglioma. It is important for clinicians to recognize the full constellation of symptoms associated with a newly diagnosed cardiomyopathy to ensure proper treatment and reduced morbidity for patients.

References:
Abstract Title: Sustainable Improvement of Colorectal Cancer Screening Rates through a Resident Led Initiative

Abstract Information:

Introduction:
Colorectal cancer (CRC) is the third most common non-skin cancer in both incidence and mortality. However, screening patients remains a challenge, especially at Federally Qualified Community Health Centers (FQHC). In June 2017, the screening rate at the Denver Health Westside Adult Clinic was 54%, below a system wide goal of 65%. A team of internal medicine residents at the Westside Adult Clinic sought to improve the screening rate over the course of one year through increasing return rates of Fecal Immunochemical Test (FIT) cards.

Case Description:

Methods:
Through a root cause analysis, we identified that CRC screening was only ordered on 35% of eligible patients from June 2016-June 2017, and of those receiving FIT cards, only 62% had returned them.

We underwent three Plan-Do-Study-Act (PDSA) cycles. First, medical assistants (MA) pended a FIT test for patients due for CRC screening during the rooming process as a prompt for the provider to address CRC screening during the visit. Second, we developed a patient handout in both English and Spanish, for our patients to review the various options for CRC screening prior to seeing their provider.

In our third PDSA cycle, we identified patients who had received a FIT card but had not yet returned it within two weeks. We then sent a reminder letter (in both English and Spanish) encouraging them to turn in their FIT card.

Results:
In June of 2017 at the Westside Adult Internal Medicine Clinic 54% of nearly 3,000 patients eligible for CRC screening were considered to be up to date with screening.
implementing our three PDSA cycles our screening rates had increased to 63%. Of those receiving reminder letters, 20% returned their FITs.

Three months after concluding resident involvement in this project, the interventions of MA initiated FIT ordering and reminder letters for patients remain in effect through the support of clinic and clerical leadership. Additionally, Denver Health is now operationalizing “batch” letter functions within the EMR to make reminder letter generation a fully automated process.

**Discussion:**

Through this resident initiated quality improvement project, we substantially improved the rates of CRC screening. Additionally, through automated letter reminders, this project remains sustainable and has resulted in system wide changes in how patient outreach is approached. This project demonstrates that sustainable improvements in public health can be accomplished from a yearlong quality improvement project.
Abstract Title: A perfect storm: tacrolimus-induced DKA in a patient with anti-synthetase syndrome with interstitial lung disease

Abstract Information:

Introduction: Diabetic ketoacidosis is a rare but known complication in patients taking tacrolimus concurrently with steroids and is most commonly described in renal transplant patients. Here we present a case of a nontraditional patient for this life-threatening adverse reaction.

Case Description: The patient is a 59-year-old male diagnosed two years ago with anti-synthetase syndrome complicated by interstitial lung disease with repeated flares requiring high dose prednisone. The patient presented to the hospital with nausea, vomiting, and a glucose of over 700 and was found to be in new diabetic ketoacidosis. He had an A1c of 6.6 in November and had been on an extended prednisone taper as well as high-dose maintenance tacrolimus; recently, he had also been diagnosed with thrush and had completed a course of fluconazole.

He was admitted and started on a DKA protocol with rapid resolution of his anion-gap acidosis. His A1c upon recheck was 16, likely secondary to chronic high-dose prednisone for treatment of his interstitial lung disease flares. He was discharged from the hospital on a new basal-bolus insulin regimen. Both his tacrolimus and his prednisone were continued at the time of discharge in order to treat his anti-synthetase syndrome and his interstitial lung disease.

Discussion: Tacrolimus, an immunosuppressive medication used in transplant patients to prevent rejection, often causes elevated blood sugar. Tacrolimus-induced diabetic ketoacidosis is a rare complication that requires multiple factors for its pathophysiology. The few case studies of this condition often focus on renal transplant patients who are on chronic low-dose prednisone as well as tacrolimus who then have an initial DKA presentation. This patient represents a rare case where a non-transplant patient was diagnosed with tacrolimus-induced DKA. In this patient, who already had very high average blood sugars from chronic high doses of prednisone, the interaction of tacrolimus and fluconazole likely caused tacrolimus levels to become elevated, precipitating DKA. In patients taking tacrolimus, elevated blood sugar is a known and common side effect. When patients take multiple medications that affect blood sugar, it is important that any additional medicines be evaluated for interactions with chronic regimens of critical drugs as rare but life-threatening complications can arise with these interactions. This is especially important in patients who, like this one, do not have the chronic underlying conditions with which tacrolimus-induced DKA is associated. It is important for a
hospitalist to ascertain whether a patient’s uncommon but critical drugs have a narrow therapeutic index and incorporate that into the differential of an acute pathology.
Abstract Title: A Shocking Case of Neuropathy

Abstract Information:

Introduction:

Thiamine deficiency can present as two distinct clinical entities: beriberi and the Wernicke-Korsakoff syndrome. The typical risk factor for both diseases is chronic alcoholism, however, a post-mortem evaluation of 52 cases of Wernicke encephalopathy (WE) found 23% of the deceased to be alcohol non-abusers. This case illustrates a unique presentation of fulminant thiamine deficiency in severe protein-calorie malnutrition.

Case Description:

A 56-year-old woman presented to a community hospital with three weeks of progressive ascending weakness and numbness of the lower extremities. History was notable for necrotizing gallstone pancreatitis complicated by severe protein-calorie malnutrition requiring total parenteral nutrition (TPN). Lumbar puncture demonstrated cytoalbuminologic dissociation and polymerase chain reaction (PCR) studies were negative for West Nile Virus. With concern for Guillain-Barre Syndrome (GBS), she received intravenous immunoglobulin (IVIG) for five days with progression of her weakness and numbness. She then became encephalopathic and pancytopenic, requiring transfer to the University Hospital. On arrival, she was hypotensive with a lactate of 4 mmol/L requiring vasopressors and intubation for airway protection. High-dose thiamine was empirically administered on hospital day 1. Brain magnetic resonance imaging (MRI) on hospital day two demonstrated findings consistent with WE. Electromyography (EMG) revealed severe sensorimotor polyneuropathy without axonal loss nor fibrillation potentials. The serum thiamine level was 104 nmol/L (normal 70-180 nmol/L) after two days of supplementation. Ultimately, she was weaned from vasopressors on hospital day three, successfully extubated on day five, completed nineteen days of parenteral thiamine, and remains on oral supplementation more than 60 days following presentation.

Discussion:

Thiamine is a water-soluble vitamin that serves as a cofactor in glucose and ketone metabolism. Absorbed predominantly in the jejunum and ileum, thiamine has a half-life of only 10-20 days making continuous intake necessary to prevent deficiency. Patients with
chronic alcoholism, post-bariatric surgery, TPN-dependence, or severe protein-calorie malnutrition are at risk.

Dry beriberi is characterized by a distal symmetric peripheral neuropathy, typically with an axonal pattern on EMG, although it can be nonspecific in critically ill patients. Case reports describe dry beriberi mimicking acute inflammatory demyelinating polyneuropathy (AIDP) with ascending weakness and concurrent cytoalbuminologic dissociation in the cerebrospinal fluid. Thiamine deficiency in the form of wet beriberi can present with fulminant hemodynamic collapse, severe lactic acidosis, and circulatory overload, which is rapidly reversible with high-dose parenteral supplementation. No case reports have discussed dry beriberi and hemodynamic compromise, however the clinical syndromes can overlap (10). Imaging in WE most commonly reveals involvement of the thalamus and mammillary bodies. Empiric treatment of presumed thiamine deficiency is necessary, as serum levels take days to result, and timely treatment can rapidly correct hemodynamic instability and encephalopathy.

In critically ill patients with severe protein-calorie malnutrition or TPN-dependence who develop distributive shock with predominant peripheral neuropathy, prompt intravenous thiamine repletion should be considered.
Abstract Title: Factor of the Matter: A Case of Acquired Hemophilia

Introduction: Spontaneous hematomas can often be attributed to medications or known disorders that predispose the patient to bleeding. However, further evaluation can sometimes reveal disorders of coagulation. While hemophilia is a relatively common inherited condition, acquired factor deficiencies are seen less frequently. Even less frequent are acquired inhibitors of coagulation factors, but the most well-documented of these is acquired factor VIII inhibition. This condition can often initially present as a life-threatening bleed.

Case Description: A 67-year-old man presented with a large left thigh hematoma one month after a spontaneous upper extremity hematoma. He was found to have a significant isolated elevation of his partial thromboplastin time (PTT), which led to further evaluation for a disorder of coagulation. A coagulation factor assay demonstrated an undetectably low level of factor VIII and inhibitor assay resulted in a factor VIII inhibitor level of 7 Bethesda units, indicative of high inhibitor titer.

The patient initially received several units of FFP without benefit. Upon diagnosis of factor VIII inhibitor, the patient underwent therapeutic plasma exchange followed by IVIg infusions, neither of which provided noted benefit. The patient’s PTT remained significantly elevated to 85, and he continued to have increasing size of his retroperitoneal hematoma, which was causing nerve compression and left leg paralysis and paresthesia, in addition to intestinal ileus. He was started on recombinant factor VIIa, which helped to control the bleeding, while he was initiated on immunosuppressive therapy. He initially received IV steroids and this was followed by Rituximab and then, due to lack of clinical response, Cyclophosphamide was started. After about 2 weeks of therapy, he demonstrated improvement in his PTT levels and repeat assay revealed increased levels of factor VIII and decreased levels of factor VIII inhibitor to 3 Bethesda units.

The patient had no known family history of bleeding disorders and had no personal history of abnormal bleeding prior to his recent upper extremity hematoma. Workup was unrevealing for malignancy, autoimmune disease, and other disorders that have been known to be associated with acquired disorders of coagulation. He was ultimately discharged in improved condition.

Discussion: While acquired disorders of coagulation are uncommon, they must be considered when patients present with unexplained spontaneous bleeding. Small retrospective studies have shown poor outcomes for these patients and there is limited
data to guide physicians towards the most effective treatment. Therapy must focus first on control of the patient’s bleeding, which often requires recombinant factor VIIa. Immunosuppression with IV steroids and agents such as Rituximab and Cyclophosphamide is typically then needed to eliminate the offending antibodies and allow the patient’s natural factor VIII to function effectively.
Abstract:
Ertapenem is a carbapenem antibiotic used to treat infections caused by anaerobic and rare gram positive organisms. Neurotoxicity is a rare side effect of carbapenems and other beta lactam antibiotics, and it has has been described in the literature: most commonly presenting as seizures, encephalopathy, and hallucinations. In particular, ertapenem neurotoxicity typically presents as seizures and occurs most typically when given to patients with end stage renal disease (GFR <30). We present a case of a patient with moderate chronic kidney disease (GFR 30-59) who developed non-seizure related neurotoxicity after ertapenem treatment.

This patient is a 59 year old male who received intravenous ertapenem for treatment of a large empyema, cultures of which grew rare Actinomyces oris. The patient's empyema and associated clinical symptoms improved on this regimen; however, he developed increasing delirium beginning on hospital day seven (also day seven of ertapenem treatment). This delirium initially presented as “sundowning” but quickly progressed to worsening and constant agitation, insomnia, delirium, and visual hallucinations. No improvement in mental status was observed with cessation of other centrally acting medications or pharmacologic and non pharmacological delirium precautions and treatments. On hospital day 11, the patient was intubated due to inability to protect his own airway in the setting of his worsening mental status. After overnight observation in the intensive care unit, and discontinuation of the ertapenem, he was extubated and returned back to the medical floor with complete resolution of his neurological and psychiatric symptoms. He was discharged at his baseline neurological status the next day. Between escalation and resolution of the patient's mental status, the discontinuation of ertapenem was the only significant change in medical management other than pharmacologically-aided sleep/sedation. This dramatic resolution of the patient's delirium and temporal correlation with ertapenem administration make ertapenem-induced encephalopathy the most likely explanation for this patient’s clinical course.

References:


Abstract Title: The Price of Beauty: A Case of Refractory Hypercalcemia in Silicone Injection-Induced Granulomatous Disease

Introduction: Non-medical grade liquid silicone is a cosmetic filler commonly used for “underground” or unlicensed cosmetic procedures, likely due to its accessibility and lower cost than formal cosmetic surgery (1,2). Granulomatous disease is a known complication from silicone injections, and similarly, hypercalcemia is a known effect of granulomatous disease (3,4). Despite these known relationships, there are few reports in the literature of hypercalcemia secondary to a silicone-induced granulomatous disease.

Case: We present a case of a 48-year-old Hispanic female former sex-worker who presented with hypercalcemia we believe to be related to granulomatous disease caused by silicone injections performed in Mexico in 2002. She presented with symptoms of fatigue, anorexia, polyuria, and weakness and was found to have a calcium of 14. She had an appropriately low PTH, normal PTHrP, and calcitriol at the high end of normal. She had no evidence of sarcoidosis. She was treated with prednisone, pamidronate, minocycline, and fluid resuscitation with concomitant furosemide. After a week of treatment, her calcium levels lowered to 9.

Discussion: We believe this process took place in our patient given history of known granulomas and ulcerations throughout her lower extremities likely secondary to liquid silicone injections several years ago. Additionally, her workup was negative for other common causes of hypercalcemia including hyperparathyroidism, paraneoplastic syndrome, and multiple myeloma. Most interestingly, the patient’s serum calcitriol on the high end of normal, suggesting granulomatous disease as discussed above. Given this presentation, although not typical, it appears that the patient’s high levels of calcitriol from known granulomas are the cause of her hypercalcemia. The patient suffered greatly secondary to hypercalcemia with extensive calcium oxalate stones throughout her kidneys and nephrocalcinosis. She was extremely fatigued, anorexic, weak, and polyuric. Although these symptoms improved with normalization of her calcium levels, unfortunately she has had a difficult course with significant pain secondary to nephrolithiasis.

There have been several reports of silicone injections in the buttocks obtained from underground or unlicensed persons for cosmetic purposes (10,11,12,13). These tend to be
secondary to procedures performed in Latin American countries or in transgender or uninsured patients who are seeking a more affordable option. Because of the large soft tissue areas involved with granuloma formation, surgical correction is rarely an option although it has been curative when it is an option (14). In two cases, hypercalcemia was treated successfully with prednisone alone (11,13). In one other case, the liquid silicone spread from the buttocks down the legs and became a very extensive process. This patient required bisphosphonates in addition to prednisone to lower calcium levels (10). One case with refractory hypercalcemia, requiring minocycline. Described a few times in case reports, minocycline is thought to target the silicone granuloma itself and decrease focal inflammation, halting the cascade discussed above (12,15). Our patient had a very extensive disease process with granulomas, ulcerations, and scarring from her buttocks down to her ankles. After starting minocycline, we also saw improvement in calcium levels not seen with prednisone or bisphosphonates.
Abstract Title: Flash Pulmonary Edema: An Abnormal Presentation of Left Ventricular Non-Compaction Cardiomyopathy

Abstract Information:

Introduction:
Left Ventricular Non-compaction Cardiomyopathy (LVNC) is an uncommon cardiomyopathy caused by malformation of the myocardium during embryogenesis resulting in trabeculations in the left ventricle. The impact on patients ranges from asymptomatic to arrhythmias and even death. Management is similar to that of other cardiomyopathies including ACE-I/ARBs, betablockers, diuretics and Implantable Cardioverter Defibrillator Devices when indicated. We present a case of a 38 year old, otherwise healthy, Indian, male who presented with flash pulmonary edema found to have LVNC as the cause.

Case Description:
A 38-year-old Indian male with no significant medical history presented to a freestanding emergency room complaining of shortness of breath and productive cough with pink frothy sputum. On initial examination, he was found to have two-word dyspnea, elevated blood pressure, and tachycardia. A chest x-ray was significant for infiltrate representing pulmonary edema vs extensive pneumonia. He was placed on non-invasive positive pressure ventilation and given IV furosemide and ceftriaxone. Arrangements were made for his transfer and admission to our hospital. Prior to leaving the free standing ED he declined significantly and required intubation. On arrival to our ICU, pressor support and broad-spectrum intravenous antibiotics were initiated. Labs were significant for elevated lactic acid, leukocytosis (26.6), total creatine kinase of 268, troponin of 0.093 (peaked at 0.471), and a pro-BNP of 1090. An EKG revealed sinus tachycardia, right axis deviation, and non-specific S-T changes in the lateral leads. With these findings it was unclear if his presentation was infectious or cardiac until an echocardiogram the following morning showed mild concentric left ventricular hypertrophy, left ventricular ejection fraction of 28%, diffuse left ventricular hypokinesis and grade 3 diastolic dysfunction. A NC/C ratio of 2.1 was suggestive of non-compaction cardiomyopathy by Jenni criteria. The echocardiogram images showed increased trabeculations and intertrabecular recesses within the apex of the left ventricle and he was diagnosed with Left Ventricular Non-compaction Cardiomyopathy (LVNC).
**Discussion:**
LVNC is a rare phenotype of cardiomyopathy resulting from failure of endomyocardial compaction in utero, leading to prominent myocardial trabeculations. Clinical presentation can vary from asymptomatic to sudden cardiac death. Our case includes an otherwise healthy 38-year-old male presenting with flash pulmonary edema leading to respiratory failure requiring intubation, who recovered relatively quickly with diuresis and ultimately, Entresto. Our poster will discuss the acute and long-term management of this unusual presentation of an uncommon disease.
Lobular Panniculitis Due to Dermatomyositis

Introduction:
Dermatomyositis is an autoimmune connective tissue disease characterized by proximal muscle weakness, intense pruritus, and poikiloderma including the heliotrope, shawl and V-neck signs. Other findings can include ILD, dysphagia, GERD, arrhythmias and polyarthritis.

Case Description:
- A 34-year-old woman presented with six weeks of an itchy facial rash, innumerable exquisitely painful subcutaneous nodules, progressive lower extremity weakness and dyspnea on exertion.
- Vitals: 96.6 F, HR 88, 110/72,16, SpO2 99%
- Derm: scaly pink/red macular rash on the forehead and cheeks. Innumerable tender ulcerating 1-4cm nodules on the back, abdomen and thighs.
- Neuro: 3/5 strength in bilateral hip flexors and quadriceps. Unable to stand up from the bed due to leg weakness.
- CRP 14.6 mg/L (nml < 3) and aldolase 22.5 U/L (nml < 8.1)
- Positive for NXP-2 (nuclear matrix protein-2) autoantibody
- CT chest showed multifocal ground glass changes suggestive of interstitial lung disease, and CT abdomen and pelvis showed many calcified subcutaneous nodules
- Biopsy of skeletal muscle showed perimysial chronic inflammation and associated perifascicular atrophy
- **Biopsy of a subcutaneous nodule showed hyaline necrosis of the fat lobules, a lymphocytic infiltrate and calcification consistent with lobular panniculitis**
- She was diagnosed with dermatomyositis, started on a prednisone taper at 80mg per day and referred to Rheumatology for follow up. She is up to date on cancer screenings. Her intense pruritus and weakness have resolved, but her subcutaneous nodules have not.

Discussion:
Dermatomyositis has many cutaneous manifestations. Calcinosis cutis is common, especially in juvenile dermatomyositis. Panniculitis is an uncommon finding with less than thirty published cases. Treatment options include prednisone, methotrexate, azathioprine, cyclosporine A, and IVIG. In some cases the panniculitis improves slowly over months or resolves completely.
Abstract Title: Unnecessary Pre-Operative Cardiac Testing: This Patient MET his Goal

Abstract Information:

Introduction:
Proper application of preoperative risk assessment can prevent unnecessary testing and delays. DASI performs better than METs.

Case Description:
An 84-year-old man with coronary artery disease (CAD) and prostate cancer presented to the ED for hematuria. We did not identify any notable abnormalities on physical exam. Serum creatinine was 1.6 mg/dL (baseline 1 mg/dL), urinalysis showed hematuria, with normal PSA. Abdominal CT demonstrated unilateral hydronephrosis due to a pelvic mass. He was admitted with plan for ureteral stenting. However, on the day prior to the procedure, he experienced a single, brief episode of self-resolving non-exertional chest pain, with a normal EKG. The primary team calculated his Revised Cardiac Risk Index (RCRI) as 0 and determined he was able to perform > 4 metabolic equivalents (METs) asymptomatically. During preoperative evaluation, providers heard about the episode and requested formal cardiac evaluation. Based on his RCRI score, cardiology estimated a 0.4% risk for perioperative major cardiac events. He underwent treadmill stress testing; tolerating 12:50 minutes of exercise, achieving 8.2 METs, with evidence of ST depression during his recovery phase. Subsequent cardiac catheterization demonstrated a 90% right coronary artery lesion. Cardiology recommended medical management of his CAD: a beta-blocker was initiated, but was discontinued due to bradycardia. In the interval, he developed *C. difficile* colitis. He ultimately underwent ureteral stent placement 11 days after admission, which was delayed by five days for cardiac evaluation. He was discharged with follow-up.

Discussion:
This case – not an uncommon clinical scenario – highlights a misunderstanding of recommendations about preoperative cardiac risk assessment, resulting in extraneous medical testing. From a 2013 study of Medicare claims data it was estimated over 56,000 patients received unnecessary preoperative cardiac stress testing and the rate of unnecessary preoperative testing was increasing over time.¹ This unnecessary testing delayed therapeutic intervention, resulted in a medication adverse reaction, and exposed the patient to a hospital-acquired infection. The 2014 joint ACC/AHA guidelines
recommend against further cardiac testing when – as in this case – the preoperative risk of major cardiac events is less than 1% or the patient is known to have moderate functional capacity of greater than four METs, especially when findings would not impact treatment.² Non-indicated cardiac work-up did demonstrate cardiac disease; however, this did not impact his candidacy for the procedure. More recent data demonstrates the best assessment of non-cardiac surgery peri-operative 30-day mortality and MI is the Duke Activity Status Index (DASI), which performed better than METs (adjusted odds ratio 0.96, 95% CI 0.83-0.99, p=0.03).³ Like METs, DASI uses self-reported functional status as a proxy for cardiovascular health to identify individuals unlikely to benefit from further cardiac testing. Application of guidelines, as well as future consideration of using DASI, could avoid low-value cardiac workup.
Abstract Title: DOUBLE, DOUBLE VISION AND TROUBLE

Abstract Information:

Introduction: This case of a 79-year-old woman with migraines and chronic sinusitis who presented with left eye swelling, pain, and double vision highlights the importance of physical exam findings in differentiating between pre-septal and orbital cellulitis, two clinical entities with distinct management considerations and clinical courses.

Case presentation: A 79-year-old woman with migraines and chronic sinusitis presented with three days of headache centered over her left eye, double-vision, progressive swelling, and pain with eye movements. She had no recent upper respiratory illness, eye surgery, or immunosuppression. One week prior, her doctor felt symptoms to be most consistent with her known migraine headaches. Pain was refractory to sumatriptan and oxycodone. She was afebrile and left eye had marked proptosis, periorbital edema and erythema. Left eye movements were limited in supraduction, abduction, adduction, and infraduction. The right eye was normal. Both pupils were equal, round, and reactive to light. Orbital CT revealed bilateral sinusitis and a left intraorbital mass, contiguous with the left ethmoid sinus, compressing the superior rectus muscle. Findings were consistent with superior subperiosteal abscess. Antibiotics were started and she underwent orbiotomy and endoscopic sinus surgery, which immediately resolved the ophthalmoplegia, diplopia, and proptosis. Abscess cultures grew *Staphylococcus aureus* and *Staphylococcus lugdunensis*. Clindamycin was continued for 7 days and she was discharged with ophthalmology follow-up.

Discussion: This case presents a woman with chronic ethmoid rhinosinusitis who developed an initially misdiagnosed orbital cellulitis with periosteal abscess. Distinction between pre-septal and orbital cellulitis is essential to proper treatment. Orbital cellulitis is infection of the contents within the orbit, and often requires surgical as well as medical management. Pre-septal cellulitis, on the other hand, usually only requires oral antibiotics and is generally not vision or life-threatening. Both infection types can present with eye edema and erythema. Physical exam helps distinguish infection anterior to the orbital septum from that which originates posterior to the septum. Proptosis, diplopia, pain with eye movement or ophthalmoplegia all suggest infection posterior to the septum. Orbital
infections include more severe sequelae, namely orbital cellulitis, subperiosteal abscess, orbital abscess, and cavernous sinus thrombosis. Ethmoid sinusitis is the most common causative condition, occurring in approximately 98% of cases of orbital infection. This case serves as a reminder of the critical importance of a thorough ocular and neurological history and exam in all patients presenting with ocular-type symptoms.
Abstract Title: Up in the Air: A Story of Pneumorrhacis

Introduction

Pneumorrhacis (PR) or the presence of intraspinal air is a rare but interesting radiology finding that should prompt further investigation. It can be further classified into primary, secondary PR and descriptively into extra- and intradural PR. Most cases of pneumorrhacis are isolated to the cervical spine region, however a few cases describe PR throughout the spinal column. The differential diagnosis of intraspinal air should include degeneration, inflammation, malignancy and infection. Mechanisms include high pressure in the bronchoalveolar space leads to air leakage through the bronchovascular layers into the mediastinum. It is important to properly diagnose PR to distinguish it from other causes of intraspinal gas and create an appropriate and individualized treatment plan for the patient.

Case Report

An 18 yo male with history of ADHD and marijuana use presented with intractable nausea and vomiting. The patient had been admitted to a detoxification center the day prior and developed sudden onset of vomiting in which he was unable to keep any food or liquids down. Outpatient labs showed a Cr of 2.29. Vitals reviewed patient was afebrile, HR 106, RR 26, saturating 60% on RA. CT chest done showed small bilateral pneumothoraces, extensive pneumomediastinum with gas tracking throughout the neck and chest wall, pneumorrhacis in the spinal canal, as well as pneumoperitoneum, and pneumoretroperitoneum. Esophagram was negative for esophageal perforation. On physical exam, breath sounds were normal but subcutaneous air was present along the neck and chest. Labs were consistent with dehydration with H&H of 21/58.9. He was also noted to have an elevated WBC of 22.0 and Cr 1.7. Pulmonology was consulted and recommended aggressive anti-emetics and Unasyn for prophylaxis. The patient improved clinically and repeat imaging showed improvement in pneumorrhacis, pneumoperitoneum and pneumoretroperitoneum. He was discharged in stable condition.

Discussion

Various conditions can produce pneumorrhacis directly or indirectly including trauma, respiratory conditions producing high intrathoracic pressure, iatrogenic manipulations during surgery, malignancy or spontaneously. In our case, the patient’s PR was caused by high intrathoracic pressures caused by violent vomiting. The air dissected down the path of
least resistance from the upper respiratory tract into the paraspinal soft tissues of the neck and eventually into the spinal canal via the neural foramina. As with this case, PR is usually asymptomatic and self-limiting. Most cases can be managed conservatively as the air is eventually reabsorbed into the circulation slowly. Causes must be investigated and more insidious underlying pathology must be ruled out to prevent significant morbidity and mortality. Prompt recognition of underlying pathology is necessary to ensure adequate therapy.

However, in rare cases patients may develop neurological symptoms that may require surgical intervention. Although no guidelines currently exist for management in these cases given that it is so rare, spinal surgery should be consulted for evaluation.
Abstract Title: A Race Against Time: Descending Paralysis Associated with Injection of Black Tar Heroin

Abstract Information:

Introduction:
Botulism is a rare and potentially deadly neuroparalytic disease caused by release of neurotoxin from *Clostridium botulinum*. While most cases of botulism are food-borne, botulism can also be acquired from anaerobic wound infections. Wound botulism is rare with fewer than five cases reported yearly in the United States. Prompt recognition and administration of antitoxin to wound botulism patients is vital to help prevent morbidity and mortality associated with the disease.

Case Description:
A 26-year-old man presented to the Emergency Department (ED) with shortness of breath, difficult swallowing, and weakness. At presentation, the patient was nontoxic and had mild dysphonia, but was otherwise neurologically intact. Initial work up was remarkable for a white blood cell count of $12.2 \times 10^9$/$L$. Though he initially denied intravenous (IV) drug use, he was observed using IV black tar heroin while in the ED. The patient was kept for observation due to somnolence. Overnight, there was a progressive neurologic deterioration with bilateral ptosis, absent gag reflex, 1/5 strength in proximal musculature, 0/5 neck strength, severe dysphonia, and absent patellar and brachioradialis reflexes. Given his deteriorating respiratory status with negative inspiratory force (NIF) declining to -20mmHg, the patient was intubated and admitted to the ICU.

The patient’s symptoms were most concerning for wound botulism, particularly in the setting of active black tar heroin use. He was started on metronidazole for *Clostridium botulinum* coverage and the Center for Disease Control was contacted to facilitate the delivery of botulinum antitoxin from the Strategic National Stockpile in Los Angeles. Antitoxin was administered 28 hours after the patient’s initial presentation to the ED. He underwent drainage of an abscess in his right arm that subsequently grew *Clostridium botulinum*. On the fourth day of admission, his strength improved and NIF improved from -10mmHg to -30mmHg. The patient was extubated on hospital day five without complication and was discharged home two weeks later.

Discussion:
Although rare, it is important for providers to keep botulism on the differential diagnosis for patients presenting with bilateral cranial nerve palsies and descending, symmetric,
flaccid paralysis. This case illustrates the importance of early recognition of wound botulism as time to antitoxin administration and adequate source control have been correlated to length of ICU stay and ventilator-dependent days. Delay in care or misdiagnosis can result in a protracted disease course including prolonged neurologic deficits, nosocomial infections, and sequelae from extended mechanical ventilation.
**Abstract Title:** A Rare Type of Interstitial Lung Disease

**Abstract Information**

**Introduction**

Adult pulmonary Langerhans cell histiocytosis (PLCH) is an uncommon type of interstitial lung disease (ILD) that produces pulmonary nodules secondary to the release of inflammatory cytokines. PLCH has an unknown prevalence and incidence; with no occupational or geographical predisposition but nearly all patients diagnosed with PLCH have a history of smoking tobacco.

**Case Presentation**

A 35-year-old female with a pertinent past medical history of multiple arterial and venous blood clots, bilateral apical pulmonary cysts, and 20 pack year smoking history presented to the emergency department with a new onset left lower anterior, sharp chest pain associated with dyspnea. On physical exam, she had decreased breath sounds, left greater than right. A computed tomography (CT) confirmed a left sided pneumothorax and bilateral pulmonary cysts extending down into the middle and lower lobes. Pulmonology was consulted and advised to complete serial CXR to monitor the pneumothorax and scheduled a bronchoscopy for further evaluation of the pulmonary cysts. The bronchoscopy was non-diagnostic, and a video-assisted thorascopic surgery (VATS) was completed. The biopsy confirmed pulmonary Langerhans cell histiocytosis (PLCH). The patient was discharged home in stable condition but continues to smoke tobacco. Both pulmonology and her primary care physician follow her closely and continue to counsel on the importance of smoking cessation.

**Discussion:**

There are multiple modalities to diagnosis pulmonary Langerhans cell histiocytosis; high resolution computed tomography (HRCT) remains the diagnostic modality of choice of screening for PLCH and ILD. These cysts are characteristically bizarre in shape and permeate the entirety of the involved lung parenchyma; these findings along with a young adult smoker are virtually diagnostic of PLCH.
Patients with predominant nodular presentations have remission and recovery in around 70% of the cases reported and respond well to glucocorticoid treatment, with a 5-year survival rate of over 75%. The course of PLCH is variable and the disease may progress despite initial management. Treatment modalities for patients with more advanced presentations, such as cystic formation and decreased PFTs or other organ involvement, have limited choices.

Despite responses to certain treatment regimens, PLCH remains a progressive and ultimately fatal disease. Our patient, despite her young age, already has significant pulmonary function compromise and with continued smoking, it may rapidly progress to irreversible respiratory failure and death. A patient’s symptoms at time of presentation, should be investigated, especially if there is involvement of the lungs. Any symptoms indicating a decrease in effective exertional capacity should warrant further work up. If pulmonary function tests are inconclusive, and often they are with initial presentations, and clinical suspicion remains high after more conservative treatments are exhausted, imaging and consultation must be pursued early.
Abstract Information:

Introduction:

Polyarteritis nodosa (PAN) is characterized by segmental inflammation, with infiltration by eosinophils, and necrosis of medium-sized or small arteries. It is known to affect arteries in the kidneys, muscles, gastrointestinal tract, and heart. This results in many non-specific symptoms making diagnosis, or even consideration as a candidate for a differential, difficult. Moreover, it is even more difficult when this condition affects large vessels instead of medium sized vessels. Below is a case which encompasses this and includes a disease course which was not expected for PAN.

Case Description:

A 56 year old male with no significant past medical history presented to the hospital with generalized abdominal pain. CT angiography of the abdomen was performed and showed inflammatory stranding of the proximal aspect of the celiac trunk and the superior mesenteric artery which prompted Rheumatology referral and diagnosis of PAN by meeting 3 out of 10 American College of Rheumatology criteria. He was initiated on cyclophosphamide and methylprednisolone with improvement of his abdominal pain. He returned to the hospital two days later with a new complaint of shortness of breath. Echocardiogram revealed severe aortic regurgitation which was thought to be secondary to his vasculitis and was ultimately treated with aortic valve replacement. It is not common to see large vessel involvement in PAN. Since then, he continued to have an atypical course for PAN affecting multiple other organ systems including multiple lacunar infarcts, pericarditis on three separate occasions, 80% stenosis of the superior mesenteric artery, 85% stenosis of the left renal artery, 50% stenosis of the right renal artery, and renal multiple infarctions. Renal insufficiency is one of the most common manifestations of polyarteritis nodosa. However, this patient did not have any renal insufficiency. Surprisingly, his ESR and CRP were within normal limits initially and later in the course, they were only minimally elevated. Serologic studies showed only very mildly elevated complement C4 level but otherwise negative including ANCA and autoimmune panel. Patient is currently requiring chronic high dose corticosteroids to maintain remission.

Discussion:
This case illustrates that, although patient did not have typical presentation of polyarteritis nodosa, clinical suspicion should be high in order to diagnose it. Recognition of this disease is important and can be understandably difficult especially because of the overlap with the criteria for other vasculitides. We suggest that even with symptoms of large vessel disease, PAN remains in the differential to help identify this disease early in the disease course and requires multidisciplinary approach for monitoring and treatment.
Abstract Title: Rapid-onset Quadriparesis and Respiratory Failure in a Patient That Walked in to the Hospital

Abstract Information:

Introduction:
Extensive spinal epidural abscess (SEA) is a rare and emergent diagnosis that can rapidly lead to quadriparesis and death. Here we present a case of rapidly progressive paralysis and respiratory failure due to an extensive epidural abscess in a patient presenting with non-specific symptoms.

Case Description:
A 75-year-old man presented with 1-week of progressive generalized fatigue and weakness. He was confused, had slurred speech and reported difficulty with standing, dressing and bathing. He denied numbness, tingling, pain, focal weakness, fever, chills, shortness of breath, dysuria or diarrhea. Medical history included type II diabetes mellitus, chronic kidney disease, and eczema with frequent pruritis and skin wounds. Two weeks earlier he had been treated for leg cellulitis.

He was afebrile with normal vital signs. Cardiopulmonary and abdominal exams were normal. Strength, sensation and reflexes in all extremities were normal. White blood cell count was 10.8x10³/µL, sodium 126 mg/dL, blood urea nitrogen 83 mg/dL, creatinine 2.8 mg/dL and lactate 2.1 mg/dL. Urinalysis was bland. Chest x-ray and CT-head were normal.

He was started on intravenous fluids and cefazolin for cellulitis. Three hours later, he developed dense paresis in bilateral lower-extremities with 1/5 strength and hyporeflexia. Corticosteroids and broad-spectrum antibiotics were administered. While awaiting further neurologic imaging, weakness progressed to upper-extremities and core muscles, including respiratory muscle weakness requiring intubation.

Spinal MRI demonstrated an epidural abscess extending from C1 to L3 with multi-level spinal cord compression, vertebral osteomyelitis and discitis. Blood cultures and aspirated abscess grew MRSA. Despite neurosurgical interventions, he developed refractory septic-shock. Family opted to withdraw interventions and he expired.

Discussion:
Despite being uncommon (2 of 10,000 hospitalizations), the devastating implications of missed or delayed diagnosis of spinal epidural abscess necessitates all clinic and hospital-based internists be aware of this disease. Patients who are immunocompromised, have
diabetes, or use intravenous drugs are at greatest risk, although SEA most frequently occurs from spine instrumentation or bacteremia. Presenting symptoms can be subtle, including generalized malaise, fevers, back pain, and in advanced cases, neurologic symptoms. Leukocytosis and elevated ESR are suggestive, however, MRI is necessary for confirmatory diagnosis.

Prognosis is poor; 4-20% of cases result in irreversible paraplegia and 5% progress to uncontrolled sepsis and death. Extensive SEA, or a panspinal epidural abscess--as in this case--is rare, documented only in case-reports. SEA management includes broad-spectrum antibiotics and early surgical intervention. Antibiotic coverage should include Staph Aureus (60%) but also strep and gram-negative species. Prompt recognition of this disease and early intervention are key to its successful treatment.
Abstract Title: Gram Positive Coccobacilli on Peritoneal Fluid Culture, a ‘Slam Dunk’ Diagnosis?

Abstract Information:

Introduction:

Ninety-five percent of spontaneous bacterial peritonitis (SBP) is due to gram negative rods (e.g. Klebsiella, E. Coli) or S. Pneumoniae; however, incidence of SBP due to less common organisms, such as Listeria monocytogenes, has been increasing and carries a mortality of 30%.

Case Description:

A 56-year old male with past medical history of cirrhosis due to alcohol, Hepatitis B and non-alcoholic steatohepatitis, atrial fibrillation, diabetes mellitus type 2, umbilical hernia and lymphocytic colitis presented initially for chest pressure, massive ascites and hyponatremia of 121mmol/L. The patient’s chest pressure resolved with a therapeutic paracentesis of 3L and his sodium improved. However, on day 3 he developed malaise, right lower quadrant tenderness and diarrhea. The following day, he developed atrial fibrillation with rapid ventricular response and lab work showed creatinine of 1.5mg/dL (from 1.0mg/dL), sodium of 124mmol/L (from 129mmol/L) and elevated WBC of 10.7K with neutrophilia of 87%. Diagnostic paracentesis was significant for 7,441 nucleated cells with 86% neutrophils and the patient was started on Ceftriaxone for spontaneous bacterial peritonitis. Despite this, he continued to fever to 100.4F over the next 24 hours, developed worsening abdominal erythema and continued to decline clinically. On day 6, his peritoneal fluid culture grew gram positive coccobacilli and he was switched to ampicillin-sulbactam with addition of vancomycin for concern for possible abdominal wall cellulitis. The culture resulted Listeria monocytogenes on day 7, and with rapid clinical improvement, vancomycin was stopped on day 8 and patient was transitioned to ampicillin alone. Subsequent therapeutic paracentesis from 3 and 5 days into the course of ampicillin showed 1,557 and 118 neutrophils, respectively. The patient was discharged with a 28-day course of intravenous ampicillin.

Discussion:
Listeria monocytogenes is a gram-positive aerobic coccobacillus associated with meningitis and bacteremia in infants, elderly and the immunocompromised patients, gastroenteritis in immunocompetent individuals and less commonly peritonitis in peritoneal dialysis and cirrhotic patients. Proposed mechanisms for Listeria transmission include bacteremia with seeding of the peritoneal fluid and colonic bacterial overgrowth with translocation into the colon. Our patient's diarrhea may or may not have been due to Listeria, but his injured intestinal lining may have been more vulnerable to bacterial translocation. Proposed diagnostic clues to Listeria peritonitis include lymphocytosis, history of iron overload, and exposure to farm animals. While third generation cephalosporins are still recommended as empiric antibiotic coverage for SBP, individuals with Listeria risk factors or a lack of improvement in 48-72 should receive broader coverage. When Listeria is cultured, some reports suggest treatment for longer than 10-14 days as was done with our patient. Trimethoprim-sulfamethoxazole is recommended over fluoroquinolones for prophylaxis in cirrhotics with history of Listeria spontaneous bacterial peritonitis.
Abstract Title: A Rare Case of Non-Inflammatory Paraneoplastic Dermatomyopathy

Abstract Information:

Case Presentation:

51-year-old female with a remote history of Hodgkin’s Lymphoma and Stage IVB FIGO grade 3 undifferentiated endometrial carcinoma, s/p TAH BSO, was admitted with 2-week history of proximal muscle weakness, dysphagia and well-demarcated rash involving the face, neck, and upper chest. Initial concern was for dermatomyositis. Rash improved with high dose IV steroids however proximal weakness and dysphagia worsened. She developed respiratory muscle weakness with dysarthria and was transferred to ICU for higher level of care.

Laboratory studies showed elevated creatinine kinase, ESR and aldolase. CRP, Anti-Jo, myositis panel, HIV and hepatitis serologies were negative. Echocardiogram was obtained to evaluate for possible myocardial involvement and showed normal EF without myocardial wall dyskinesia or hypertrophy. Left deltoid muscle biopsy showed regional myofiber pathology without significant chronic inflammation, most consistent with Regional Ischemic Immune Myopathy.

Discussion:

Regional Ischemic Immune Myopathy (RIIM) is a rare type of necrotizing autoimmune myopathy (NAM). Similar to other paraneoplastic dermatomyopathies it is and characterized by proximal muscle weakness, dysphagia and rash however underlying histologic inflammatory findings generally seen with dermatomyositis are lacking. Commonly associated malignancies include gastrointestinal, lung, breast and prostate with rare case reports of endometrial cancer. Other cases of paraneoplastic NAM have demonstrated anti-HMGCR and anti-SRP antibodies. Treatment consists of steroids, IVIG, Rituximab and treatment of underlying malignancy.
Abstract Title: Post-cardiac arrest myoclonic status epilepticus requiring four anticonvulsants and one sedative

Abstract Information:

Introduction: Post-anoxic myoclonus status epilepticus, which was super-refractory and resistant to maximum doses of Keppra, Versed, Propofol and Depakote, required a loading dose of phenobarbital to finally break the seizure. This clinical presentation has variable etiologies ranging from severe ischemia in the brain, brainstem, and spinal cord. However, regardless of etiology, the condition is associated with an extremely poor predictive outcome. Few patients who experience myoclonus status epilepticus post cardiac arrest have good functional outcomes. With such a grim prognosis, it is reasonable to consider an aggressive approach where anticonvulsants are not only maximized but also stacked.

Case Description:

The patient is a 62-year-old male with a pertinent past medical history of asthma and ventral hernia who presented post cardiac arrest secondary to respiratory failure after choking on a hotdog. Per EMS, the patient was pulseless upon arrival, and CPR was initiated immediately. The airway was opened, and a large amount of food was cleared from the oropharynx. Return of spontaneous circulation was achieved after two rounds of epinephrine. The patient became hypoxic and was therefore intubated with endotracheal tube. Post cardiac hypothermia protocol was initiated; however, this was complicated by myotonic status epilepticus. The patient failed multiple anticonvulsants at various dosages; prior to a 24-hour EEG, he was on maximum doses of Keppra, Versed, Depakote, and Propofol. The EEG had burst suppression pattern that corresponded with the patient’s myoclonic whole body jerk, which lasted 12-18 seconds and reoccurred approximately every few minutes. EEG confirmed the diagnosis of myoclonus status epilepticus, and neurology advised to continue the above medications along with the addition of a loading dose of phenobarbital. This stopped the myoclonic jerking movement. The cessation of the status epilepticus was confirmed with a subsequent 24-hour EEG. Unfortunately, patient’s prognosis was poor and family decided to place the patient on comfort care. The patient succumbed to cardiogenic shock and passed away three days after admission.

Discussion:
This case illustrates the grim prognosis and difficulty of post-cardiac arrest myoclonus status epilepticus management. Furthermore, this patient required the use of three conventional anticonvulsants, one sedative and a third line therapy anticonvulsant in order to finally break the status epilepticus cycle. An EEG was completed after the initiation of the hypothermia protocol, which confirmed myoclonic status epilepticus. Overall, this case demonstrates the importance of aggressively treating status epilepticus and having the clinical knowledge to stack anticonvulsant medications and continuing to add medications until the cycle is broken. The prognosis for myoclonic status epilepticus remains poor despite this, however if there remains a goal of having a functional outcome for the patient, it is necessary to treat until the abortion of the myoclonic activity.
Abstract Title: No Stones, Bones, Moans, Groans, or Psychiatric Overtones

Abstract Information: Common presentation of Hypercalcemia

Introduction: Hypercalcemia presents commonly associated with malignancy

Case Description: MS is a 91 year old female with past medical history of hypertension, hyperlipidemia, mild chronic hypercalcemia, vitamin d deficiency, multiple falls, and failure to thrive who presented to the emergency department at the urging of her primary care physician due to hypercalcemia. Prior workup was done outpatient without any etiology for mild hypercalcemia. In the ED, lab studies showed moderate hypercalcemia (13.4), elevated Cr 1.3. Patient denied any symptoms of chest pain, palpitations, weakness, shortness of breath, or edema. She did report chronic diarrhea, unchanged for about a year, and slightly increased weakness for about 3 days. Physical exam was overall unremarkable, bilateral upper and lower extremity reflexes noted to be 3 /5, normal muscle tone, Chvostek’s sign negative, Trousseau sign not performed, and vitals were stable. A CT scan of the abdomen and pelvis showed new large splenic and pancreatic masses with questionable involvement of adrenal gland. A triple phase CT was performed after renal function improved. The patient’s EKG was similar to previous and showed no acute changes that could be related to hypercalcemia. She was admitted to the general floor for further monitoring and treatment with IV fluids, calcitonin, and pamidronate. Discussion was had with the family the next day about goals of care for the patient, as she had already mentioned that she did not want to have any ‘surgery or anything extreme’. Labs the next morning: Calcium 12.4, Angiotensin converting enzyme <15, Vit D 1,25-Dihydroxy 100, PTHrP <2, 24 hour urine, peripheral smear showed slight left shift. Later that day the patient with the help of her family decided to go home with hospice, she was discharged with a course of calcitonin 100IU BID for 4 days total to ensure her comfort care at home.

Discussion:
Differential diagnosis for hypercalcemia includes Vit-D excess, primary hyperparathyroidism, adrenal insufficiency, multiple myeloma, PTHrP producing tumor (ovarian, kidney, lung), osteolytic metastases sarcoid, TB. Pt did have history of mild hypercalcemia, and upon presentation she was asymptomatic. About 2 months prior to admission she had TSH 1.56, SPEP that showed no m-spike and mildly high alpha 2 globulin at 1.1, Vit D 25 hydroxy 56.1, 1, 25 dihydroxy 99.9, and PTH of 10. She had extensive testing done, it is felt that there was excessive testing and hospitalizations for her, malignancy was
always the most likely cause of her hypercalcemia. Much workup and cost could have been saved if Ct was performed earlier. Hypercalcemia is common in patients with cancer, it occurs with about 20-30% of cases. It is the most common cause of in-patient hypercalcemia. Typically malignancy is evident by the time it manifests with hypercalcemia, and is often a poor prognosis.
Secretory Diarrhea: A Very Important Presentation

Introduction:
Chronic diarrhea is a common presentation often encountered by internists and can present a diagnostic challenge due to its very broad differential, including infectious causes, inflammatory causes, osmotic causes, and secretory causes (eg. gastrinoma, hypothyroidism, serotonin syndrome, and VIPoma). Severe secretory diarrhea with associated life-threatening electrolyte abnormalities warrants pursuit of a definitive diagnosis, which can be life-saving.

Case Description:
An 80-year-old man was evaluated for five months of diarrhea. Exam was notable for severe abdominal distention and hyperactive bowel sounds. Clinical findings suggested secretory diarrhea, based on persistence despite fasting, stool volumes exceeding one liter per day requiring rectal tube placement, and low stool osmotic gap. Profound serum hypokalemia required 120 mEq daily potassium repletion to maintain low-normal range levels. Initial diagnostic evaluation was negative and included colonoscopy with biopsies for microscopic and collagenous colitis, levels of gastrin, 5HIAA, cortisol, calcitonin, TSH and extensive infectious work-up. Empiric treatment for SIBO with rifaximin and bulking agents was initiated with modest symptomatic improvement. The patient’s VIP level was ultimately found to be elevated at 96 pg/mL, and 63 pg/mL on repeat. CT of the abdomen and pelvis and triple phase CT of the pancreas were negative for malignancy, though subsequent Octreoscan localized a lesion to the region of the uncinate process of the pancreas, consistent with VIPoma. Comprehensive staging was deferred due to the patient’s poor candidacy for resection or systemic chemotherapy given age and comorbidities. The patient was started on octreotide, with production of formed stools and resolution of abdominal distension after 5 days of treatment. At time of discharge, the patient was producing 1 formed BM per day.

Discussion:
VIPoma is a rare cause of chronic diarrhea (1 in 10 million patients per year) and therefore can easily be overlooked as a culprit diagnosis. Also known as Verner-Morrison syndrome, VIPoma is a form of neuroendocrine tumor that can present with profound refractory hypokalemia and sometimes hypochloridria. Further confounding its diagnosis is its potential to present atypically. The case presented deviates from the classic presentation of VIPoma in several ways. VIP incidence has a bimodal age distribution with the majority of patients diagnosed in early childhood or between ages 30 and 50, in contrast to this elderly patient. While two CT scans were negative in this case, CT, particularly with
contrast, is highly sensitive for VIPoma. Tumor location within the uncinate process of pancreas also deviated from the most common location of VIPomas in the tail of the pancreas. Finally, VIP level was not consistently above the expected threshold of 75 pg/mL, highlighting the importance of confirmatory testing. Here we discuss a rare presentation of an exceptionally rare disease, underscoring the need for diagnostic perseverance in the face of an ambiguous, though critical diagnosis.