Obstruction as an initial presentation of Crohn’s disease

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Crohn’s disease (CD) affects individuals of all age groups, however, it is a diagnostic challenge in older adults. CD can lead to multiple complications, including bowel obstruction from fibrostenotic disease, but it is highly uncommon to have bowel obstruction as the initial finding.

A 77-year-old Caucasian man presented with persistent nausea and vomiting preceded by 3 months of decreased appetite, abdominal distension, intermittent fecal urgency, and loose stools with mucus. The patient had two screening colonoscopies in the past showing colitis, with one revealing granulomata, but was not diagnosed with Crohn’s disease. He denied any current abdominal pain, weight loss, or fever. Upon admission, he was hypertensive (168/79 mmHg) with HR 96. Physical exam revealed a severely distended tympanic abdomen without tenderness. CT of the abdomen and pelvis with IV contrast demonstrated segmental colitis of the descending colon leading to partial mechanical obstruction and marked gaseous distention of the proximal colon. Flexible sigmoidoscopy showed severe rectosigmoid colitis for which biopsies were suggestive of IBD, but without granulomata. CT angiogram of the abdomen and pelvis did not show any significant stenosis or vascular occlusion and biopsies did not represent ischemia. He was then diagnosed with Crohn’s disease and underwent a laparoscopic-assisted transverse loop colostomy. Despite surgical intervention, he continued to have significant distention and attempts to decompress further were unsuccessful. He was subsequently discharged home with comfort measures per family request.

This case highlights the severity of outcomes with nonspecific gastrointestinal symptoms due to colonic obstruction as the initial signs of CD. Given this atypical presentation, CD was almost overlooked as a potential diagnosis, but can have had more potentially serious outcomes, including death. This case serves as a reminder to practitioners that though the age of onset in CD has a bimodal distribution, the disease presentation is typically more subtle and insidious in elderly patients and is less likely to involve traditional symptoms, such as abdominal pain/cramps, diarrhea, and weight loss. A high index of suspicion must be had to employ proper diagnostic strategies for timely implementation of treatment.

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Abstract Form: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and stay within the borders!
A DEADLY MIMIC
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Mycotic aortic aneurysm (MAA) is an exceedingly rare and severe
disease representing less than 1% of all aortic aneurysms. Cases of
Klebsiella pneumoniae-induced MAA are even more uncommon with
less than 35 cases reported over the past 10 years. MAA causes an
irreversible vascular dilatation of the aortic wall instigated by microbial
invasion, usually due to Staphylococcus aureus or Salmonella. Given the
virulent nature of these microorganisms causing early aneurysm rupture,
clinicians must be vigilant in quick detection and treatment to avoid
devastating outcomes¹.

A 74-year-old woman with hypertension, diabetes mellitus type 2, and
hypothyroidism presented with a 3-day history of generalized weakness.
Initial vitals demonstrated tachycardia (105 bpm) and hypotension
(87/52 mmHg) that was responsive to intravenous fluids. Initially, she
was treated with one dose of ceftriaxone but given lack of improvement,
this was broadened to vancomycin and piperacillin/tazobactam until
blood cultures showed Klebsiella bacteremia in the setting of UTI,
therefore vancomycin was discontinued. On Day 3, she became fluid
unresponsive with BP (86/54 mmHg) requiring a dopamine drip. She
then developed chest pain due to NSTEMI, for which a heparin drip was
started. On Day 4, her chest pain resolved, and no further hypotension
was noted. The same evening, the patient reported recurrence of chest
discomfort despite improvement of troponin. On Day 5, a chest X-ray
showed vascular congestion, but upon closer review revealed a widened
mediastinum. CT angiography revealed an acute type B aortic dissection
with a contained rupture in the proximal descending thoracic aorta.
However, after a few hours, review of imaging by primary care team
with radiology revealed MAA based on evidence of periaeurysmal gas.
The patient was subsequently transferred to a tertiary care center for
thoracic endovascular aortic aneurysm repair, however over the next
several weeks, she continued to decline due to GI bleeding and
subsequent cardiac arrest, leading to death.

Management of MAA are clinically and surgically challenging,
particularly in aortic aneurysms involving the arch which have an
associated 50% mortality rate. Imaging findings of infected aneurysms
can be varied, ranging from a lobulated vascular mass to subtle
periaurysmal gas in the intima. A keen eye and understanding of this
devastating condition is imperative for prompt detection and timely
management.

CV 112

BREAKOUT ROOM 12

ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help
reproduction. Be sure to single-space and STAY WITHIN THE BORDERS!
Glioblastoma Multiform represents 19% of primary central nervous system tumors. Common presentations include headache, altered mental status, and seizures. Among medical patients admitted with hyponatremia, approximately 14% of cases can be linked to neoplasm. Most glioblastomas are present in the supra-tentorial region and rarely extend to involve deeper structures such as the rostrum of the corpus callosum (CC) and the hypothalamus.

Case Presentation:
A 72-year-old woman with a history of coronary artery disease, sarcoidosis, hypothyroidism, and hypertension presented with a two-month history of progressive confusion and headache. During the week prior to presentation, her confusion became more severe, and was associated with generalized weakness and urinary incontinence. There was no apparent seizure activity. Initial labs were notable for a serum sodium of 109mmol/L, which prompted admission to the ICU. Vital signs were unremarkable. Physical exam was notable for dry oral mucosa, poor skin turgor and delayed capillary refill. On neurologic exam, she was oriented only to person and was otherwise non-focal. Urine osmolality was high at 662mOsm/kg, serum osmolality at 235mOsm/kg and urine sodium was 70 mmol/L. Testing of thyroid function and adrenal axis was unremarkable. T2 weighted sequencing revealed an enhancing nodular mass involving the corpus callosum, extending to the left hypothalamus. Hypertonic saline resulted in improvement in her sodium. Once serum sodium reached 129mmol/L, her IV fluids were stopped, and she was placed on free water restriction. Biopsy of her intracranial mass revealed a high-grade glioma. Unfortunately, her mental status deteriorated rapidly and she was eventually transferred to inpatient hospice.

Conclusion:
High grade glioma's extending into the posterior corpus callosum with involvement of the hypothalamus is uncommon and their characteristics have not been well defined. Greater than 40% lie within the frontal lobes. Most cases of tumor related hyponatremia reported in head and neck cancers are due to ectopic production of ADH either through direct disturbance of sodium balance pathways or paraneoplastic syndrome. Due to the rarity of thalamic gliomas, mechanisms of this disease-causing hyponatremia is not well defined. It should be kept in mind that the severity of hyponatremia can be utilized as a potential predictor of disease progression in malignancies other than SCLC.
BLUE LIPS, LOW HEMOGLOBIN - SYMPTOMATIC METHEMOGLOBINEMIA FROM DAPSONE PROPHYLAXIS IN A PATIENT WITH APLASTIC ANEMIA. Dalal S, MD, Kashanian S, MD, Grier W, MD, Niyongere S, MD. The University of Maryland Medical Center, Baltimore, MD.

Methemoglobinemia is a rare functional anemia in which iron is oxidized from the ferrous to ferric state, and thus, increases hemoglobin's affinity for oxygen. Less oxygen is offloaded to the tissues, leading to perioral or peripheral cyanosis, headaches, fatigue, and dyspnea. Numerous medications can cause acquired methemoglobinemia, with dapsone being the most common.

A 57 year old female with aplastic anemia on dapsone prophylaxis presented with two episodes of syncope, subjective fevers, dyspnea, dry cough, chills, fatigue, nausea, vomiting, and dizziness. Physical examination was pertinent for a heart rate of 81 beats per minute and oxygen saturation of 88% on room air with improvement to 94% on 4 liters nasal cannula. Increased oxygen supplementation did not further improve oxygen saturation. Lungs were clear to auscultation. Laboratory values were pertinent for severe neutropenia of 0.35 Km/mL, hemoglobin 7.3 g/dL, and platelets 1 K/mcL. She was initially treated for neutropenic fever with meropenem. Chest imaging was unremarkable for a pulmonary embolism or consolidation. She had an episode of syncope on ambulation and was noted to have perioral cyanosis. Arterial blood gas on room air was visibly dark red and showed an oxygen saturation of 85%, PO2 of 95, carboxyhemoglobin of 6.9 g/dL, and methemoglobin level of 11.8%. Her glucose-6-phosphate-dehydrogenase (G6PD) level was normal at 10.2 U/gHb. One week after discontinuation of dapsone, her symptoms and hypoxia of methemoglobinemia resolved without methylene blue treatment.

This case illustrates symptomatic methemoglobinemia in a patient with aplastic anemia on dapsone prophylaxis. Although rare, methemoglobinemia should be suspected in clinical cyanosis with normal arterial oxygen levels, hypoxia not corrected with supplemental oxygen, and use of culprit medications. Patients with methemoglobin levels between 3-20% are usually asymptomatic but symptoms can be exacerbated by anemia, lung, or heart disease. Thus, in anemic patients, prescribing dapsone should be done cautiously.

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**ABSTRACT FORM:** Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and **STAY WITHIN THE BORDERS!**
A Case of Primary Idiopathic Chylopericardium. Angie M. Molina, MD. University of Maryland School of Medicine, Baltimore, MD.

Chylopericardium is a rare condition in which chylous fluid accumulates in the pericardial space. Most reports demonstrate development of chylous pericardial effusions from trauma of the thoracic duct after surgery. However, the exact pathophysiology is not well understood, with some reports demonstrating evidence of valvular damage of thoracic duct valves, communication of the thoracic duct into the pericardial space, or mediastinal obstruction causing increased pressure resulting in chylous fluid accumulation. Idiopathic chylopericardium is significantly less common and the pathophysiology is not understood.

A 48-year-old white male with a history of psoriasis (previously on adalimumab) and a large right-sided facial port wine stain presented as a transfer for a 2-week history of progressively worsening shortness of breath and productive cough. On his initial presentation he was found to have a large pericardial effusion with diffuse ground-glass consolidation of both lungs. He was treated for community acquired pneumonia and discharged home with follow-up echocardiogram but returned to the outside hospital days later for worsening shortness of breath, hypoxia to 80%, and hypotension. A repeat echocardiogram showed an enlarging pericardial effusion with tamponade physiology.

An urgent pericardiocentesis yielded approximately 600ml of chylous fluid (triglyceride level of 644 mg/dL). A drain was placed and removed within 24 hours with no significant accumulation after 2 weeks. The workup was negative for any autoimmune, malignant, or infectious causes (viral, fungal, or bacterial). He also had no prior recent history of trauma, malignancy, mediastinal mass, infection, tuberculosis, or thoracic surgery. Further imaging, which included two lymphangiograms, computed tomography, computed tomographic angiography, and magnetic resonance imaging of the chest did not show communication between the thoracic duct and pericardial space or anterior mediastinal mass. His diet was modified to a medium-chain triglyceride diet, and he was observed throughout his hospital stay, with no significant accumulation of the effusion. Cardiothoracic surgery deemed that the patient was not a candidate for a pericardial window. 

Idiopathic chylopericardium is rare, and a full workup is required to rule out more common causes. However, after a diagnosis of idiopathic chylopericardium is made, diet modification and serial echocardiograms are appropriate. Pericardial window or thoracic duct ligations are not necessary for management.
Angioedema Refractory to Steroids: A Cause for Concern

Introduction: Superior vena cava (SVC) syndrome occurs in only about 15,000 patients in the United States each year. Its rarity often excludes it from differential diagnoses. However, due to its association with malignancies, thrombotic events, and resultant complications, it must strongly be considered.

Case: A 78-year-old man with a history of hypertension and non-insulin dependent diabetes presented with subacute progressive facial and upper and lower extremity edema that originated periorally and spread in a descending manner. His symptoms were associated with dyspnea on exertion, hoarseness, and a new inability to tolerate oral intake. He completed two prednisone tapers in the outpatient setting without any resolution of his symptoms. He denied any rashes, wheezing, infectious symptoms, new chemical exposures, allergic history, or family history of angioedema. He does not take an angiotension converting enzyme inhibitor. His physical exam was notable for macroglossia, perioral edema, and edema of the upper and lower extremities. There was no flushing, erythema, or cyanosis. He could speak in full sentences without stridor. A bedside flexible fiberoptic laryngoscopy showed posterior pharyngeal wall water-balloon edema with a widely patent glottic airway concerning for angioedema. His complete blood count, basic metabolic panel, CI esterase inhibitor, trypsin, and complement levels were normal. The patient was started on a dexamethasone regimen. A CTA chest showed no pulmonary embolism but noted an irregular large lesion involving the entire anterior mediastinum with compression of the SVC and aortic arch. Steroids were discontinued out of concern for tumor lysis syndrome precipitation. Progressive tachypnea prompted a transthoracic echocardiogram that showed a large pericardial effusion with concern for cardiac tamponade. A pericardial drain was placed with resultant drainage of 600 cc of bloody fluid. He underwent an IR guided biopsy that indicated metastatic thymic carcinoma and SVC stent placement with near resolution of his symptoms. He was discharged with home hospice.

Discussion: This case illustrates the potential for SVC syndrome to be misdiagnosed as angioedema despite a lack of a sustained response to steroids. Although SVC syndrome is rare, recognition of its presence early in the disease course may prevent the above complications and may alter a patient’s goals of care.

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ABSTRACT FORM: Must be at least 10-point font. A sharp typeface will help reproduction. Be sure to single-space and stay within the borders!
An unusual presentation of Cat Scratch Disease

Background: Cat Scratch disease is a rare cause of fever of unknown origin (FUO). It is usually self-limiting in immunocompetent patients with minimal symptoms. We present a case of a patient with atypical features Cat Scratch disease.

Case: 31 year old man initially presented with fevers of 102-103F, chills and fatigue of two days duration. Initial suspicion was COVID-19 although he did not have any cough or shortness of breath. A SARS-COV-2 test was ordered which came back negative. He continued to have high fevers, mostly in the evenings. On further discussion with the patient, it was revealed that he had also been having B/L tender inguinal lymphadenopathy for a few days prior to the onset of the fevers.

A week after the onset of fevers, further labs were obtained which revealed a normal CBC, CMP, ESR, LDH and blood culture. Patient was negative for HIV. CRP was elevated at 71mg/L. At this point the patient was asked to come into the office for a thorough physical exam. The exam was normal except for B/L tender inguinal lymphadenopathy. No other lymphadenopathy in other regions were noted. A CT abdomen and pelvis showed 2 subcentimeter non enhancing lesions in the liver, 3 small splenic lesions which were hard to characterize, and B/L inguinal lymphadenopathy with the right side showing some necrosis or abscess formation. A right LN biopsy was done which showed mostly pus. No organisms were seen in the LN and the culture was negative. At this point the patient's fever had been continuing daily for 3 weeks. A detailed social and environmental history was obtained which revealed the patient had many cats which were in close contact with him all the time. A Bartonella IgG and IgM antibody with titer was ordered and both had high titers. The patient was started on Azithromycin and Rifampin for 2 weeks with which his symptoms improved.

Discussion: Cat Scratch disease is a rare cause of FUO. Our patient had many atypical features which makes it unique. He did not have a history of a cat scratch or bite which later developed into an erythematous papule which is reported in 70% of cases. Usually lymphadenopathy is regional and unilateral as opposed to our patient who had B/L inguinal lymphadenopathy. Cat Scratch disease is usually self-limited in immunocompetent patient, but our patient had FUO with hepatosplenic involvement.

Conclusion: Cat Scratch disease can have atypical presentations and should be considered in a patient with FUO.

CV 117

BREAKOUT ROOM 12
Bilateral Facial Palsy

Introduction: BFP occurs at a rate of 0.3 to 2% of all the facial palsies with annual incidence of 1 per 5 million[1]. Rare presentations of Lyme disease and GBS(Guillain-Barre Syndrome) have been previously reported in this clinical setting.

Case: 28 yo old woman presented with facial weakness of 3 days duration; started initially on the right side. She was diagnosed with Bell’s palsy and was prescribed prednisone. Two days later, she reports bilateral facial and lower extremity weakness with associated extremity tingling, numbness and bloody diarrhea. Past medical history included upper respiratory tract infection 3 weeks prior to admission. Physical exam was significant for decreased pinprick sensations in the toes and complete facial weakness bilaterally; lower extremity power of 4/5 with absent knee and ankle reflexes on both extremities. Lab work revealed wbc 14.3x10^9/L, absolute lymphocytes 5.42x10^9/L. Serum IgM for Borrelia burgdorferi was positive and was confirmed on western blot with negative IgG. Serum IgM for CMV, EBV capsid were positive along with heterophile antibodies. Serum CMV quantitative PCR was 1580 IU/mL CSF analysis revealed wbc <3, protein 68, negative Lyme serology and negative PCR for CMV, EBV. MRI brain showed mild bilateral facial neuritis. EMG was consistent with Acute inflammatory demyelinating polyneuropathy (AIDP). She was treated with ceftriaxone for 14 days and IVIG for 5 days.

Discussion: Few conditions have been reported to cause simultaneous BFP, most commonly Lyme disease, GBS, tuberculosis, HIV, sarcoidosis, trauma and autoimmune disorders[2]. Treatment with IVIG or plasma exchange has been shown to be beneficial with both approaches being equally efficacious. BFP can occur in up to 39% of the cases in Lyme disease[4]. Two-tier serological approach is considered as the standard diagnostic test for disseminated Lyme disease. Initial test utilizes Enzyme Immunoassay(EIA) or Immunofixation assay (IFA) and if positive or equivocal, will require confirmation with western blot. The use of ceftriaxone in early disseminated Lyme disease is recommended for certain neurological manifestations[5]. PCR for CMV, EBV was clinically insignificant in this case.

Conclusion: Bilateral facial palsy is a challenging entity due to a broad differential diagnosis. A thorough history and physical exam with extensive investigation is recommended to identify coexisting conditions. Careful monitoring of the patient vitals and respiratory capacity is necessary to avoid catastrophic outcomes.
Rare case of Primary Small-Cell Neuroendocrine Carcinoma of Prostate co-existing with High grade Acinar Adenocarcinoma

**Introduction:**
Primary neuroendocrine small cell carcinoma of the prostate is very rare and was first described in 1977. It accounts for less than 1% of total prostate cancers, with less than half mixed with conventional acinar adenocarcinoma. The prostate is the second most common site for primary small cell neuroendocrine carcinoma, after lung, accounting for 3% of total neuroendocrine tumors.

**Case presentation:**
A 83-year-old male presented with 2-day history of rectal pain with defecation and hematochezia. Patient had lost 40 lbs over the prior 6 months. Rectal examination revealed large painful prostatic nodules and an obstructed anal canal. PSA level was elevated to 148.3 ng/mL. CT abdomen/pelvis showed prostatic mass with circumferential infiltration/invasion and severe narrowing of the recto-sigmoid junction. The patient was started on bicalutamide 50mg daily, with plans for leuprolide hormonal therapy pending biopsy. Transrectal US guided prostate biopsy revealed mixed small cell neuroendocrine carcinoma–acinar adenocarcinoma (Gleason group 5+5 or GRADE Group 5).
Acinar adenocarcinoma expressed PSA, PSMA and NKX3.1 and high grade neuroendocrine carcinoma expressed CD56 and TTF-1 and negative PSA, PSMA, NKX3.1, chromogranin and synaptophysin. Tc-99m MDP whole body bone scan confirmed prostate as primary site and showed evidence of "SuperScan," with extensive diffuse osseous metastasis involving the axial and appendicular skeleton, sparing the skull. With evidence of primary prostatic neuroendocrine differentiation, patient was started on chemotherapy with carboplatin-etoposide and hormonal therapy. Follow up abdominal CT scan after 6 months did not show further progression of osseous metastases.

**Discussion:**
We present a very uncommon, biphasic carcinoma of the prostate with neuroendocrine differentiation from high grade adenocarcinoma, which can be aggressive on presentation. PSA levels can often be normal, and mixed tumors do not respond to hormonal therapy alone. There are no diagnostic tests for early detection of neuroendocrine differentiation, except confirmation with biopsy and ruling out primary source from lung. According to the SEER database, the presence of high-grade acinar adenocarcinoma in such biphasic tumors, as opposed to low grade, is a predictor of worse cancer specific mortality. Based on clinical studies, we suggest immediate attention and initiation of chemotherapy, given its poor prognosis with median survival of only 12-13 months.
Severe Cardiomyopathy from Chronic Chagas Disease

Introduction: Chagas disease, also known as American trypanosomiasis, is a parasitic infection caused by the protozoan Trypanosoma cruzi. Primarily transmitted by triatomine bugs, it is endemic to South and Central America. Chronic Chagas disease may involve the heart, leading to arrhythmias, thromboembolism, and dilated cardiomyopathy. Patients who were asymptomatic for decades after the acute infection may present with palpitation, syncope, or sudden death.

Case Description: A 47-year-old man with sudden onset biventricular heart failure was transferred for device therapy and heart transplant evaluation. Originally from El Salvador, he immigrated to the US 30 years ago and last visited his home 7 years ago. 5 weeks prior to transfer, he began to have exertional dyspnea associated with orthopnea, malaise, and generalized weakness, which worsened over the course of a week. He went to the emergency department of an outside hospital where he was found to have atrial fibrillation with rapid ventricular response, right bundle branch block, and cardiogenic shock. He experienced acute hypoxic respiratory failure and cardiac arrest. Echocardiogram showed dilated ventricles, global hypokinesis, and LVEF 10-15%. He was then transferred for veno-arterial extracorporeal membrane oxygenation and inotropic therapy. Coronary angiography showed normal arteries, and cardiac magnetic resonance showed heterogeneous late gadolinium enhancement without myocardial edema. Testing for ANA, HIV, adenovirus, coxsackie, Borrelia, and Toxoplasma serology were all negative. However, T. cruzi IgG was positive (IgM negative) and subsequently confirmed by CDC. Without a history of alcohol or illicit drug use, or family history of heart disease, and negative metabolic work-up (including normal troponin, thyroid function tests and low transferrin saturation) and positive T. cruzi serology, the patient was diagnosed with cardiomyopathy secondary to chronic Chagas disease. Given the high risk of arrhythmia with episodes of nonsustained ventricular tachycardia, an implantable cardioverter-defibrillator was placed. He continued to improve and was discharged home to complete heart transplant evaluation as outpatient.

Discussion: Antitrypanosomal therapy in chronic Chagas disease in adults remains controversial. The pharmacologic management of Chagas cardiomyopathy is mostly based on studies on other etiologies. Nevertheless, given the serious complications of the disease, awareness and appropriate index of suspicion are essential.