TEAM

8

CVs
2019 Mulholland Mohler Resident Meeting

<table>
<thead>
<tr>
<th>ALTERED MENTAL STATUS COMPLICATED BY NUTRITIONAL DEFICIENCY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jasdeep Sabharwal, MD, PhD; Callaghan Gillis; Steven Davies; Mark S. Gosnell, MD</td>
</tr>
<tr>
<td>Medstar Harbor Hospital, Baltimore, Maryland</td>
</tr>
</tbody>
</table>

**Introduction**

Nutritional deficiency can cause changes that complicate an altered mental status (AMS).

**Case Presentation**

A 42-year-old woman with a history of gastric bypass in 2008 presented with one day of AMS and muscle weakness. She was discharged seven days earlier from an outside hospital after a one-month hospital stay for abdominal pain, weight loss, fatigue, and inadequate oral intake. Later she became confused, developed a blank stare, had restless lower extremities and wanted to be left alone. On the day of admission, she was unable to converse and developed a rash on both lower extremities. She was started on lactulose for a possible diagnosis of hepatic encephalopathy. Head CT was normal, and workup for infection was negative. Multiple EEGs were negative though the patient had seizure-like activity. Mental status deteriorated, and the patient had to be intubated for protection of her airway. Further testing revealed decreased serum levels of copper and zinc, and IV supplementation was started. As a result, mental status and muscular strength gradually improved, and she was extubated.

**Discussion**

Gastric surgery is the most common cause of acquired copper deficiency. Other causes include excessive zinc ingestion, dietary copper deficiency, malabsorption syndromes, and chelation therapy in Wilson disease. The most common neurologic manifestation of acquired copper deficiency is that of a myeloneuropathy. Patients typically present with subacute onset of a gait disturbance. But a profound change in mentation may mask weakness.

**Conclusion**

In a patient with a history of gastric bypass presenting with anemia along with neurologic changes, nutritional deficiencies should be considered.

---

**Program Director’s Name:** Dr. Detterline

*(indicating review of abstract)*
2019 Mulholland Mohler Resident Meeting

AORTIC ANEURYSM IN A PATIENT WITH HLA-B52+ BEHÇET’S DISEASE & HLA-B27+ ANKYLOSING SPONDYLITIS
James Hughes, MD1, Caleb Anderson, MD2, Angelique Collamer, MD2
1National Capital Consortium Internal Medicine Residency
2Department of Rheumatology, Walter Reed National Medical Center

Introduction:
The coexistence of Behçet's disease (BD) and ankylosing spondylitis is an uncommon association but is gaining increasing recognition in the literature. This case of BD and AS is unique in its vascular involvement and its human leukocyte antigen (HLA) alleles. It is well known that BD is a vasculitis affecting any size vessel, though typically small, and aortitis can complicate AS. As for HLA alleles, HLA-B27 is most commonly associated with AS and HLA-B51 with BD, thus, the few reported cases of BD and AS are usually HLA-B51+ and/or HLA-B27+. HLA-B52 is associated with BD but is uncommon and typically only seen in Israeli, Mesifio Mexican, and Iranian Azeri patients. However, it is commonly known to be associated with another vasculitis, Takayasu arteritis (TA). Additionally, recent literature shows a significant association between HLA-B27+ AS and TA as well.

Case:
The patient is a 45-year-old man with a history of BD and AS positive for HLA-B52 and B27 diagnosed in January 2017. Symptoms first began in 2006 with chronic back pain along with recurrent painful oral ulcerations and acneiform lesions. Years later he was evaluated for BD, however, pathergy testing and ophthalmologic evaluation were negative. He was re-evaluated at WRNMMC in 2010 for AS. Sacroiliac (SI) joint MRI demonstrated joint edema, but he was lost to follow up. In 2013 he developed a spontaneous deep venous thrombosis of unknown etiology despite an extensive work up. In January 2017 he was hospitalized at Brigham & Women's Hospital for severe abdominal discomfort and was diagnosed with a 3.8cm thoracic aortic aneurysm and a hepatic vein thrombosis. Inflammatory markers were elevated but the remainder of lab testing was negative aside from HLA-B52 and HLA-B27. Xarelto was initiated, aortic endovascular stenting was performed, and he was started on daily corticosteroids. PET-scan performed later that month was negative for vasculitis. He was started on Humira in April 2017.

Discussion:
This is a unique case of concomitant BD and AS positive for HLA-B52 and HLA-B27 complicated by an aortic aneurysm, which has not been significantly reported in the literature previously. Additionally, recent literature shows a significant association between these alleles and TA. Could this suggest that in the setting of BD and AS these alleles could predispose at patient to large vessel vasculitis? This may be an association worth investigating.

First Author Information:
Name: James Hughes, MD PGY1
Institution: WRNMMC
Daytime Phone: (912) 660-6470

Co-Author(s) Associates:
CPT Caleb Anderson, MD & MAJ Angelique Collamer, MD

Program Director’s Name:
LTC Joshua Hartzell
(indicating review of abstract)
A BUS DRIVER WITH CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION (CTEPH)

Authors: Andi Shahu, MD1; Trisha Pasricha, MD2; Amit Goyal, MD1

Academic affiliations: 1 Johns Hopkins Hospital, Baltimore, MD

CTEPH occurs in ≤5% of survivors of acute pulmonary embolism (PE).1 Early diagnosis is key, requiring a high index of suspicion, multimodal approach and multidisciplinary team. A 54-year-old male bus driver with multiple prior deep vein thromboses (DVT) and PEs on anticoagulation with financial barriers to medication adherence presented with subacute progressive dyspnea and lower extremity edema. Physical exam was notable for hypoxia requiring 4L O2, elevated JVP and pitting lower extremity edema. Labs showed troponin <0.04 and pro-BNP of 1428. ECG revealed features of right heart strain (right axis deviation, R > S in V1, S1Q3T3 and precordial T wave inversions). Chest CT angiogram was negative for acute PE but showed bilateral peripheral pulmonary artery (PA) filling defects with distal branch attenuation and parenchymal mosaic attenuation concerning for chronic PE. Transthoracic echocardiography revealed a dilated, hypokinetic right ventricle (RV) and elevated RV systolic pressure (103 mmHg) indicative of severe pulmonary hypertension (PHTN). Following diuresis, right heart catheterization (RHC) showed normal right heart filling pressure (3 mmHg), elevated PA pressure (105/36, mean 53 mmHg), low pulmonary capillary wedge pressure (4 mmHg) and elevated pulmonary vascular resistance (10.7 Wood units) consistent with precapillary PHTN. VQ scan indicated a high probability of chronic PE and PA angiography demonstrated enlarged PAs with peripheral pruning, confirming the diagnosis of CTEPH causing group 4 PHTN. Extensive hypercoagulability testing was unrevealing; his occupation as a bus driver was felt to be his prevailing risk factor. He was discharged on furosemide, warfarin and 2L O2. This is a prototypical presentation of advanced CTEPH with severe group 4 PHTN. Early recognition is vital given the dire natural history of untreated disease and the advent of effective treatment.2 One must maintain a high index of suspicion and utilize multimodal diagnostics (TTE, CT, VQ scan, RHC, PA angiography) in consultation with a multidisciplinary team (pulmonologists, interventional radiologists, and thoracic surgeons). In addition to lifelong anticoagulation, the therapeutic armamentarium includes pulmonary thromboendarterectomy (definitive therapy), percutaneous balloon pulmonary angioplasty and pulmonary vasodilators. Our patient was referred to a CTEPH center for evaluation and management.

RHODOCOCCUS FASCIANS – A RARE CAUSE OF MENINGITIS IN A HUMAN HOST. Motwani K, MD. University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Rhodococcus species are obligate aerobes, gram-positive bacilli and partially acid-fast because of their mycotic acid-containing cell wall. They are isolated from a variety of sources including soil, ground water, plants and animals. The microbe is generally considered to have low pathogenicity, however has been known to cause disease in immunocompromised hosts.

A 76-year-old male with a history of hypertension and alcohol abuse was admitted for altered mental status which consisted of confusion, slurred speech, visual hallucinations and ambulatory dysfunction. He had recently arrived from the Dominican Republic where he was exposed to poor living conditions. Magnetic resonance imaging (MRI) of the brain displayed mild meningeal enhancement and small embolic events. Further work-up revealed a ruptured pseudoaneurysm of his ascending aorta, and he underwent repair with a graft placement. The patient remained hemodynamically stable, however, 7 days post-operation there was no improvement in his mental status.

Cerebrospinal fluid (CSF) studies revealed: glucose 28 mg/dL, protein 474 mg/dL, white blood cell count 260 mcl with 48% polymorphonuclear leukocytes, 47% lymphocytes, and 5% monocytes. Cytology, gram stain and cultures were all negative. He was started on ampicillin, ceftriaxone and doxycycline empirically. Repeat CSF analysis revealed an increase in leukocytosis, with persistently low glucose and elevated protein. MRI brain showed worsening interval meningesis versus ventriculitis. Given his lack of improvement, his treatment regimen was changed to rifampin, isoniazid, pyrazinamide, and ethambutol for suspicion for tuberculosis meningitis. Additionally, a trial of steroids for sarcoidosis was attempted, without improvement.

The patient underwent a leptomeningeal biopsy, which displayed evidence of necrotizing granulomas and 16S rRNA cultures were positive for Rhodococcus fascians. He was started on a three-drug regimen with vancomycin, meropenem, and azithromycin for at least an 8-week course with plans to transition to oral antibiotics. 

Rhodococcus fascians is a rare cause of meningitis and not easily identified with routine microbial testing. Treatment regimens are currently unknown due to the rarity of the disease. Recognizing lack of improvement in disease course in an immunocompromised patient should lead providers to consider alternate pathologies and seek further testing with 16S rRNA gene sequencing.
PRIMARY GASTRIC ASPERGILLOSI

Shailises Sinmkhada, MD; Rehan Farooqi, MD; Suresh Paudel, MD; Ritesh Shrestha, MD; Anita Naik, DO
MedStar Health Internal Medicine, Baltimore, MD

Invasive aspergillosis is most commonly seen in patients with immune disorders and primarily involves the lung. There are a few studies regarding primary and disseminated gastrointestinal aspergillosis in immunocompromised hosts. Only a few cases of primary gastric aspergillosis in non-immunocompromised hosts have been reported. Patients with primary gastric aspergillosis usually present with acute abdomen with perforation or impending perforation.

We present a 65-year-old woman with an extensive medical history including bladder cancer status post neoadjuvant chemotherapy, anterior pelvic exenteration with urostomy, recurrent pelvic mass and lung nodule in 2017 status post chemoradiotherapy with progression of disease requiring chemotherapy and immunotherapy. She presented with several episodes of loose, non-bloody and watery stool for three days. She also had crampy lower abdominal pain without radiation, nausea and vomiting, or fever. On exam, patient was afebrile and hemodynamically stable. She appeared cachectic with bitemporal wasting and lean extremities. She had diffuse tenderness without rebound tenderness with sluggish bowel sounds and a normal digital rectal examination. Relevant labs showed leukopenia of 0.2 with absolute neutrophil count of 0 and marked thrombocytopenia with platelets count of 32. Her creatinine was elevated to 2. CT of the abdomen and pelvis showed thickening of the descending colon and rectosigmoid region. Further workup revealed E. coli bacteremia and she was started on broad spectrum antimicrobials. Her hospital course was complicated with aspiration pneumonia which resulted in her being NPO pending further workup. She underwent endoscopy for feeding tube evaluation. During the procedure, she was noted to have an approximately 10-cm necrotic appearing ulcer in the greater curvature with a black base. Biopsies were taken. Histopathology revealed ulcerated gastric mucosa with fibrinopurulent debris with numerous fungal hyphae with septae and acute angle branching consistent with aspergillus infection. Due to ongoing concern for impending perforation, the decision for feeding tube placement was held. Patient was started on voriconazole and after extensive discussions with all the associated teams and palliative care team, the patient opted for home hospice.

Gastric secretions typically provide an inhospitable environment for aspergillus to grow. Hence, primary gastric aspergillosis is an extremely rare entity even the severely immunocompromised patient. There are just a few isolated clinic cases of gastric aspergillosis causing the complication of perforation or impending perforation with very high mortality.
2019 Mulholland Mohler Resident Meeting

WHEN SICKLE CELL DISEASE PRESENTS AS ALTERED MENTAL STATUS
Marzieh Keshtkarjahromi, MD; Louis Saade, MD; Seyed Hadi Mirhedyat Roudsari, MD; Medstar Health Internal Medicine, Baltimore, Maryland

In the United States, 6-10% of African-American newborns have sickle cell trait, and 0.2% have sickle cell anemia. The acute features of sickle cell disease are vaso-occlusive phenomena and hemolysis. Sickle cell crisis resulting in vaso-occlusions can lead to serious complications such as acute chest syndrome (ACS), stroke, renal, bone and myocardial infarction. ACS is a major cause of hospitalization and mortality. Diagnostic criteria of ACS include new pulmonary consolidation accompanied by fever and/or respiratory symptoms.

A 24-year-old African-American man with known history of sickle cell disease at birth presented with acute altered mental status. Past medical history was significant for an episode of acute crisis at age 6 requiring transfusion and one episode of cholelithiasis causing pancreatitis. Upon admission, he was lethargic and oriented to person and place. Vital signs were significant for temperature 39.3 C, heart rate 149 bpm, respiratory rate 42, blood pressure 96/44 mmHg. Physical exam revealed icteric sclera, laterally displaced PMI, 14 cm liver span in the midclavicular line and slight arachnodactyly. Labs were significant for elevation of WBC to 38K, LDH 262, Alk phos 311, bilirubin 22.4, AST 87 and reticulocyte 30%. Hgb was 8 g/dL which further decreased to 6.7 g/dL after aggressive fluid resuscitation. Imaging demonstrated an opacity with air-bronchograms in the left lower lung and a large calcified gallstone and atrophic spleen. As the clinical picture was compatible with ACS, the patient was begun on azithromycin and ceftriaxone and underwent exchange transfusion. Mental status improved to baseline shortly thereafter. Our patient remained symptom free after transfusion and was discharged from the hospital.

Vaso-occlusive phenomenon in sickle cell crisis in adults usually present as ACS or dacltytx. Neurologic events are rarely reported in patients over 20 years of age. In one study, neurologic findings occurred in 11% of patients, of which 56% had altered mental status (AMS). Vaso-occlusion in pulmonary microvasculature is the basic mechanism most often precipitated by infection and fat emboli. This patient presented with AMS which is not an usual presentation in ACS. Pigmented gallstone, elevated bilirubin and AST support hemolysis. Atrophic spleen and gallstones suggest repeated splenic infarction and chronic hemolysis. As pneumonia cannot be distinguished from ACS, empiric antibiotic therapy has been recommended. Transfusion of RBC is lifesaving. The goal of transfusion is to lower hemoglobin S to 30% to decrease the risk of vaso-occlusion and increase oxygenation. This case underscores the variation in clinical presentations of ACS, and reminds us to consider AMS as a potential presenting symptom for such a crisis.

AMERICAN COLLEGE OF PHYSICIANS
MARYLAND REGION
ANNUAL ASSOCIATES MEETING
MAY 9, 2019

Please check one. First author is:
(X) RESIDENT
Please check only one. Abstract is submitted to:
( ) Poster
( ) Oral
( ) Either
General Classification:
(X) Clinical Vignette
( ) Research Competition
( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

Indicate your participation in research process (4 sentences or less): Medical resident taking care of patient, writing abstract

First Author Information:
Name: Marzieh Keshtkarjahromi, M.D.
Institution: Medstar Health, Baltimore, Maryland
Daytime Phone: 443-742-1645
Co-Author(s) Associates:
Louis Saade, MD; Seyed Hadi Mirhedyat Roudsari, MD

Program Director's Name:
(indicating review of abstract)
2019 Mulholland Mohler Resident Meeting

A MISTAKEN CASE OF “ANGIOEDEMA”

Introduction: Dermatomyositis is a multisystem disease presenting with a variety of clinical features. Skin findings are very common which include facial erythema and heliotrope eruptions of the eyelids which can mimic angioedema. We present a case of a patient who presented with B/L eye lid edema and erythema who was initially diagnosed and treated for angioedema. He continued to worsen and eventually was diagnosed with dermatomyositis and treated accordingly.

Case: 74 yM with PMH of HTN, Type 2 DM and HLD who presents with periorbital swelling, tongue swelling and pruritic facial rash for one month. His ACEi was stopped by his PCP due to concern for ACEi induced angioedema and was started on a Medrol dose pack with minimal improvement. He later developed shortness of breath, orthopnea, increased fatigue, lower extremity swelling and intermittent dysphagia. He was admitted due to worsening of the above symptoms as well as a rash involving the neck, upper anterior and posterior chest, and both hands. On exam, He had a characteristic periorbital heliotrope rash with edema, erythematous rash on neck and anterior chest, back and shoulders, the face, knees and elbows. He also had Gottron’s papules, irregular and thickened cuticles, and cracked palmar fingertips. These were pathognomonic for Dermatomyositis. The diagnosis was confirmed by serology and muscle biopsy. Interestingly he had ground glass opacities on CT scan concerning for Interstitial Lung disease as part of the anti-synthetase syndrome which signifies a bad prognosis. He was also found to have new reduced ejection fraction heart failure with an EF of 40%. He was treated with high dose steroids which improved his rash as well as his heart failure.

Discussion: In a patient presenting with periorbital edema, angioedema is on top of the differentials especially if it affects the tongue and the oropharynx. Dermatomyositis can also present with eyelid swelling but typically does not involve the oropharynx. Only a few case reports have been published where the eyelid swelling and erythema being the dominant initial physical finding of dermatomyositis as seen in in our patient.

Conclusion: The eye lid findings of dermatomyositis can mimic angioedema. A detailed history and examination to look for other skin lesions and well as involvement of other organs could help in differentiating angioedema from other systemic diseases like dermatomyositis.
DIVERTICULITIS MASKING MESENTERIC VEIN THROMBOSIS
Sanjay Singh, MD; Sandeep Mishra, MD; Rehan Farooqi, MD
MedStar Health Internal Medicine, Baltimore, Maryland

Mesenteric venous thrombotic events are very rare and often present with non-specific abdominal presentations that can be easily missed without thorough history and imaging with a high index of suspicion. We present such a case to highlight need for close monitoring, early, and long-term management.

A 68-year-old woman presented with severe bilateral lower abdominal pain radiating to back, without hematochezia but with remote history of provoked deep vein thrombosis and recent diverticulosis. Clinical examination showed diffuse abdominal tenderness more localized in the left lower quadrant. She was initially diagnosed as diverticulosis and managed conservatively. However, after initial improvement, her symptoms worsened, and contrast CT scan showed presence of diverticulosis and inferior mesenteric vein (IMV) thrombosis. The patient required vascular surgical consult, heparin anticoagulation, continued antibiotics and extended hospital stay. She was finally discharged on anticoagulation for 6 months with DOACs.

Mesenteric ischemia remains a high mortality (20-50%) condition. Typical mesenteric thrombotic events are usually seen in the arterial system, often with supporting post-prandial abdominal symptoms. Mesenteric venous thrombosis constitutes about 10% of all mesenteric ischemia and typically affects the superior mesenteric vein (SMV), with IMV involvement being very rare. IMV thrombosis is neither common nor easily visualized in arterial-timed angiographic studies and is often missed, save for the tell-tale mesenteric stranding and bowel wall edema. Transmural ischemia leads to necrosis, with gastrointestinal bleeding, perforation, and peritonitis. Management is focused on urgent anticoagulation to achieve early restoration of circulation without loss of bowel integrity. Role of surgical intervention is now considered limited and warranted in cases with perforation or peritonitis. In absence of markers of infection, the use of antibiotics remains controversial, though there is data to suggest better outcomes with empiric antibiotics. Anticoagulation leads to recanalization in over 90% patients. Long term care of these patients is focused on comorbidity management with smoking cessation, blood pressure control, statin therapy and treatment of associated prothrombotic conditions.

Mesenteric venous ischemia outside of the portal venous system without circulatory decompensation is rare and requires early and urgent management, with or without antibiotics for improving outcomes.

Program Director's Name: Dr Stephanie Detterline
(indicating review of abstract)

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!
2019 Mulholland Mohler Resident Meeting

<table>
<thead>
<tr>
<th>COMPLETE RESOLUTION OF SKIN METASTASES IN MICROSATELLINE INSTABILITY-HIGH COLON CANCER WITH NIVOLUMAB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunita Timilsina, MD1; Prashanti Atluri, MD2</td>
</tr>
<tr>
<td>1MedStar Harbor Hospital, Baltimore MD; 2NYU Winthrop Hospital, New York, NY</td>
</tr>
</tbody>
</table>

Cutaneous metastases in colorectal cancer are uncommon occurring in 4.4% of patients. The median time to diagnosis for cutaneous metastasis is 32 months after the initial diagnosis of colon cancer. Patients with skin metastases have diffuse systemic disease. We present a patient with colon cancer who presents with isolated cutaneous metastases within six months of the initial diagnosis. Treatment with nivolumab leads to complete resolution of the lesions.

A 91-year-old woman with no significant comorbidities was diagnosed with colon cancer during evaluation for fatigue and constipation. Laparoscopic total colectomy with ileorectal anastomosis revealed two stage IIIIC synchronous primaries located at the cecum (adenocarcinoma, pT4a, N2a) and descending colon (adenocarcinoma, pT3N2a) with high microsatellite instability. We treated her with single-agent capecitabine. Two months into the treatment, the patient developed subcutaneous nodules at right deltoid (4×4 cm) and right back (2.5×2.5 cm) that were mobile, non-tender, and without any overlying skin changes. Fine needle aspiration cytology of the deltoid lesion was consistent with metastatic colon cancer. Imaging disclosed no other metastasis with complete resolution of the cutaneous lesions.

While skin metastasis is traditionally a poor prognostic marker, the prognosis of patients with oligo-metastatic skin lesions is not well defined. We hypothesize, early detection and treatment of skin metastases with systemic therapy can not only treat the skin lesion but may also address other micro-metastases and delay or prevent systemic disease. Nivolumab has been approved for advanced microsatellite instability-high (MSI-H) in colon cancer that has progressed following conventional chemotherapy. Patients with high microsatellite instability may respond dramatically to immune checkpoint inhibitors such as nivolumab.

---

First Author Information:

Name: Sunita Timilsina

Institution: MedStar Harbor Hospital

Daytime Phone: 6672281710

Co-Author(s) Associates:
Prashanti Atluri, NYU Winthrop Hospital

Program Director’s Name:
Stephanie Dettlerine
(indicating review of abstract)

**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!**
2019 Mulholland Mohler Resident Meeting

Moyamoya Presenting as Hypertension Secondary to Renal Artery Stenosis

Paul S. Jeong, DO, Walter Reed National Military Medical Center, Bethesda, MD.

Introduction: Moyamoya disease is a rare and progressive vaso-occlusive cerebrovascular disease characterized by bilateral stenosis or occlusion of the arteries around the Circle of Willis. Moyamoya usually presents with varying neurologic processes including stroke and seizure. The infrequency of diagnosis and non-specific presentation makes early diagnosis difficult. Moyamoya is described as a cerebrovascular disease and its extracranial manifestations remain to be elucidated.

Case: A 30-year-old Caucasian man presented with hypertension on hydrochlorothiazide. Secondary hypertension workup was performed due to his age, multiple paternal relatives with renal artery stenosis (RAS), and father with history of renal artery stenting and bypass in his 20s and several cerebrovascular accidents in his 40s from carotid artery stenosis. The patient’s CT angiogram of the abdomen demonstrated left RAS which was stented. MRA of the Circle of Willis showed no evidence of Moyamoya, but was followed by MRA four months later which was suggestive of Moyamoya with bilateral ICA stenosis and possible stenosis of the right A1 segment of the anterior cerebral artery. Patient underwent cerebral angiogram showing 60% stenosis of extracranial right ICA without evidence of Moyamoya changes. The plan was to continue monitoring.

Patient was lost to follow-up and was evaluated 2 years later with MRA showing worsened 99% stenosis of the right ICA, which was stented. Four months after stenting he had a tonic-clonic seizure during a combat fitness test and was hospitalized. Etiology of seizures was determined to be secondary to hypertension with blood pressures up to 250/180. CT angiography demonstrated occluded left renal artery stent and atrophy of left kidney for which there was no planned revascularization and medical management was pursued.

Discussion: This case illustrates a case of presumed Moyamoya disease which presented with secondary hypertension from RAS. The most commonly reported extracranial site involved is the renal artery. As most RAS is associated with atherosclerosis and fibromuscular dysplasia, it is difficult to predict that a patient’s RAS is due to Moyamoya disease. This case highlights the importance of including Moyamoya in the differential for young patients with hypertension secondary to RAS, even in the absence of neurological symptoms, and to be vigilant for such symptoms. It also emphasizes regular follow-up imaging in these patients who may be at higher risk for devastating cerebrovascular complications.

Program Director’s Name: Joshua D. Hartzell
(indicating review of abstract)

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!
TWO UNUSUAL CASES OF THROMBOTIC MICROANGIOPATHY COMPLICATED BY PURTSCHER-LIKE RETINOPATHY AND HEMOPHAGOCYTIC

Daniel H. Shu, MD, Avi Z. Rosenberg, MD, Lois J. Arend, MD, Samuel A. Merrill, MD, PhD

Thrombotic microangiopathy (TMA) is a hematologic syndrome characterized by widespread inflammation and endothelial damage. Its clinical findings of hemolytic anemia and thrombocytopenia require prompt recognition since certain causes of TMA, such as thrombotic thrombocytopenic purpura (TTP) and atypical hemolytic uremic syndrome (aHUS), can be fatal if left untreated. Purtscher-like retinopathy, an occlusive microangiopathy of the retina, is a rare presenting sign of TMA.

Two patients presented to our hospital with acute bilateral vision loss after an inflammatory prodrome including fevers and serositis. They were found to have cotton-wool spots, retinal hemorrhages, and Purtscher's fleckens on fundoscopic exam and were diagnosed with Purtscher-like retinopathy. The first patient had leukocytosis with marked eosinophilia, anemia, thrombocytopenia, and elevated creatinine at the time of admission, and the second patient developed these abnormalities after several days in the hospital. LDH and ferritin were also elevated and peripheral blood smear showed schistocytes. Both patients received plasmapheresis, steroids, and, after ADAMTS13 activity was found to be normal and stool Shiga toxin testing was negative, eculizumab for aHUS. Renal biopsy showed microangiopathy with fibrin thrombi. To our surprise, neither patient improved with these treatments. Given the presence of fever, cytopenias, and hyperferritinemia, as well as hypertriglyceridemia, elevated soluble IL-2 receptor levels, and hemophagocytosis on bone marrow biopsy, they were initiated on etoposide and dexamethasone for hemophagocytic lymphohistiocytosis (HLH), a rare, life-threatening condition caused by pathological immune activation. Both patients improved and eventually left the hospital, although neither recovered renal function or vision.

These two cases illustrate the importance of timely diagnosis of thrombotic microangiopathy and the need for a high index of suspicion for treatable forms of TMA, such as TTP and aHUS. They also provide an example of a rare presenting sign of thrombotic microangiopathy, Purtscher-like retinopathy, as well as an uncommon complication, HLH.

AMERICAN COLLEGE OF PHYSICIANS
MARYLAND REGION ANNUAL ASSOCIATES MEETING MAY 9, 2019

Please check one. First author is:
( X ) RESIDENT
Please check only one. Abstract is submitted to.
( ) Poster
( ) Oral
( ) Either

General Classification:
( X ) Clinical Vignette
( ) Research Competition
( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

Indicate your participation in research process (4 sentences or less):
I prepared the manuscript.

First Author Information:
Name: Daniel H. Shu, MD
Institution: Johns Hopkins School of Medicine, Baltimore, Maryland
Daytime Phone: 510-847-9484

Co-Author(s) Associates:
Avi Z. Rosenberg, MD, Lois J. Arend, MD, Samuel A. Merrill, MD, PhD

Program Director’s Name: Sanjay V. Desai, MD

(indicating review of abstract)

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!
Please check one. First author is:
( X ) RESIDENT
Please check only one. Abstract is submitted to.

(X) Poster
( ) Oral
( ) Either

General Classification:
(X) Clinical Vignette
( ) Research Competition
( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

Indicate your participation in research process (4 sentences or less): Resident caring for patient at initial presentation. I have followed patient’s treatment course and am reporting events as well as major learning points from case. I completed literature review of endocarditis and transient bacteremia after minimally invasive procedures for clinical vignette presentation.

First Author Information:

Name: Abhishek Mishra, MD

Institution: Medstar Health

Daytime Phone: 570-573-1129
Co-Author(s) Associates: Samuel English, DO; Michelle Zikusoka, MD; David Weisman, DO

Program Director’s Name: Dr. Stephanie Detterline
(indicating review of abstract)

STREPTOCOCCUS GALLOLYTICUS ENDOCARDITIS POST TUBULAR ADENOMA POLYPECTOMY IN A SPLENECTOMIZED PATIENT
Abhishek Mishra, MD; Samuel English, DO; Michelle Zikusoka, MD; David Weisman, DO
MedStar Health Internal Medicine, Baltimore, Maryland

Streptococcus galolyticus previously known as Bovis subspecies has a long, well-documented association with colorectal cancer. Transient bacteremia after inpatient or outpatient minimally invasive procedures is relatively common; however, this is predominately cleared by patient’s immune system without any manifestations of infection. Antibiotic prophylaxis with colonoscopy and polypectomy is generally not recommended in a normal population; however, American Society of Gastrointestinal Endoscopy states there is insufficient evidence to make a recommendation for immunocompromised individuals. Here, we report a case of streptococcus galolyticus infective endocarditis after low risk polypectomy in a splenectomized patient.

57-year-old man with history of multiple myeloma, asthma, hyperlipidemia, pancreatic cancer status post resection with splenectomy presents to the ED for workup of low-grade fever and fatigue for several weeks. In the ED his WBC count was 16,000 and he was sent home with a diagnosis of a viral illness and then called back after his blood cultures were positive within 24 hours gram-positive cocci in pairs in 2 of 2 samples. Blood culture speciation demonstrated Streptococcus galolyticus subspecies pasteurianus. New diastolic murmur was identified, and subsequent echocardiogram showed a large mobile echodensity on the aortic valve measuring 1.2cm with moderate aortic regurgitation. One month prior to presentation he underwent colonoscopy with removal of 1.5cm and 5mm pedunculated mid transverse colonic polyp with pathology showing tubular adenoma. The patient was treated with 4 weeks of ceftriaxone with clearance of bacteremia; however, repeat echo demonstrated persistent vegetation with worsening regurgitation and enlarging ventricular cavity. He underwent bioprosthetic aortic valve replacement and is currently recovering.

Splenectomy is an immunocompromising condition and places patients at greater risk for bacteremia by encapsulated organisms. Special consideration in prophylaxis and antibiotic administration at first signs of infection are essential in preventing complications.
2019 Mulholland Mohler Resident Meeting

THIRD TIME’S A CHARM: A CASE OF PERSISTENT HYPOXIA
Vaishnavi Raman, MD, David Weisman, DO
MedStar Health Internal Medicine, Baltimore, MD

Patent foramen ovale (PFO), a remnant of fetal circulation, is present in 20-25% of adults and often goes unrecognized due to patients remaining asymptomatic. When symptomatic, it is due to intracardiac right-to-left shunting of deoxygenated blood, which can cause hypoxia out of proportion to an underlying lung disease process.

A 49-year-old man with a history of end-stage renal disease (ESRD) on hemodialysis via tunneled right internal jugular catheter, heart failure with preserved ejection fraction, and anemia of chronic disease, presented to the hospital with 2-days of progressive exertional shortness of breath and pre-syncopal symptoms. He was afibrile, normotensive, tachypnic (respiratory rate 24) and hypoxic (84% on high flow nasal cannula FiO2 50%). Cardiopulmonary exam was notable for use of accessory muscles of respiration, diffuse bilateral fine crackles and no cardiac murmurs. His hemoglobin was 4.5 with no evidence of external loss or hemolysis, and chest imaging suggestive of pulmonary edema. Initial working diagnosis was volume overload in the setting of inadequate dialysis and symptomatic anemia of unclear cause. Despite urgent hemodialysis and blood transfusions, he required intubation due to increasing oxygen requirements and respiratory fatigue. A new diastolic murmur was appreciated and transthoracic echocardiogram (TTE) showed a severely incompetent aortic valve and small PFO with bidirectional flow. Blood cultures then grew Staphylococcus epidermidis in 2 of 2 bottles likely from the dialysis catheter. He began antibiotics for infective endocarditis (IE) and underwent emergent aortic valve replacement (AVR). Post surgery, he continued to be hypoxic and required prolonged mechanical ventilation via tracheostomy. At this point, PFO mediated intermittent right-to-left shunt was pursued as the culprit for hypoxia. A septal occlusion device was placed resulting in complete resolution of his intermittent hypoxia.

This case highlights the importance of maintaining a broad differential, especially when there is a sub-optimal response to treatment of initial working diagnosis. Native valve IE causing symptoms or signs of heart failure should prompt early referral to surgery for valve repair as this has been associated with reduction in mortality. Furthermore, PFO with right-to-left shunts cause hypoxia and correcting the shunt may improve clinical outcomes.

Program Director’s Name: Stephanie Detteline, MD

(indicating review of abstract)

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 RARE FORM OF INFLAMMATORY MYOSITIS

Evelyn Song, MD\(^1\), Chris Fan, MD\(^1\), Amit Goyal, MD\(^1\)
Johns Hopkins Hospital, Baltimore, MD

A 52 year-old male presented to the hospital with complaint of progressive dyspnea on exertion and weakness. Nine months prior he noticed progressive difficulty raising his arms. Seven months prior he developed bilateral hand joint pain and swelling, eventually diagnosed with rheumatoid arthritis (RA) with positive anti-nuclear antibody (ANA), rheumatoid factor (RF), and anti-cyclic citrullinated peptide (anti-CCP). He was treated with prednisone, methotrexate, and adalimumab. His arthritic pain improved, but over the subsequent months he developed progressive upper extremity weakness, myalgias, dyspnea on exertion, and orthopnea, prompting presentation to the Johns Hopkins Hospital.

Initial physical examination was notable for significant bilateral proximal muscle weakness involving the upper extremities associated with atrophy of the deltoids and shoulders, without fasciculations. Skin examination showed Gottron’s papules, mild erythema on face and chest, and buffalo hump. Initial labs were notable for normal creatine kinase and lactate dehydrogenase with mildly elevated aldolase. Autoimmune workup showed positive RF and anti-CCP with negative ANA. Dexamethasone suppression test and myositis antibody panel were negative. EMG/NCS suggested lower motor neuron (LMN) dysfunction in the cervical/lumbosacral distribution. Myositis protocol MRI of the arms showed symmetric active myositis of both upper extremities. Biopsy of his left deltoid revealed endomyosal inflammation and muscle necrosis as well as mild acute neurogenic atrophy. He was diagnosed with Brachial-cervico inflammatory myopathy (BCIM) and treated with high dose steroids, rituximab, and continued methotrexate on which regimen he reported modest improvement of his strength and dyspnea.

BCIM is a rare inflammatory myositis characterized by proximal weakness affecting primarily the arms and neck with prominent B-cell infiltrates seen on muscle biopsy, and is often associated with another autoimmune diagnosis, such as RA. This patient’s presentation and muscle biopsy findings are highly consistent with BCIM. Atypical features here include the lack of significant improvement with corticosteroids alone and the objective presence of concurrent LMN dysfunction raising the possibility of a myositis/motor neuron overlap syndrome. The rarity of BCIM made this a diagnostic challenge. However, following a stereotyped approach to common complaints, here the approach to weakness, is essential for making the diagnosis, even for the zebras.

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!
2019 Mulholland Mohler Resident Meeting

ADENOCARCINOMA IN A 39-YEAR-OLD
Shreenivas Sreenivasan, M.; Rehan Farooqi MD
MedStar Health Internal Medicine, Baltimore, MD

Colorectal cancer (CRC) is the 2nd leading cause of cancer-related deaths in the United States in males and females combined. Around 20% of patients with CRC have distant metastases at the time of diagnosis with involvement of the liver, lungs, peritoneum, or bone. At present, screening is not recommended for individuals under 50 years of age unless they have a positive family or other risk factors such as radiation or inflammatory bowel disease.

A 39-year-old male presented with 1-week history of intermittent vomiting and 1-day history of melena. He denied any changes in bowel pattern, abdominal pain, early satiety, weight loss, night sweats, or hematochezia. He had a 1-pack year history of smoking, endorsed social alcohol intake, but denied any illicit drug use. The patient was incarcerated from 2012-2017. He works as a trash collector & reports inhalation of unknown toxins at the site. He denied radiation exposure. Family history was negative for malignancy. Upon examination, he was afebrile, normotensive, and tachycardic (126). He had epigastric tenderness, bibasilar crackles, and diminished breath sounds bilaterally. Pertinent serology included hemoglobin of 7.9, PPD, AFB X 2, hepatitis panel, HIV and fecal occult blood test were negative. CEA level was elevated >10,000. Chest CT revealed multiple pulmonary nodules ranging from 1mm to 15mm with smooth borders. An abdominal CT was notable for hepatomegaly and multiple hypodense lesions concerning for metastases. An ultrasound guided liver biopsy demonstrated adenocarcinoma. Immunohistochemical staining biopsy sample was positive for CK20 and CDX2, making the colon a likely primary source. The immunohistochemistry testing for mismatch repair proteins revealed intact nuclear expression for MLH1, MSH2, MSH6 & PMS2. There was no loss of nuclear expression of MMR proteins. He was discharged with follow-up colonoscopy as an outpatient.

Colorectal carcinoma has been observed to occur with increasing frequency in adults <50 years of age. Most patients have non-specific symptoms such as subclinical fevers and weight loss. They present late with advanced disease and metastases. Therefore, a high index of suspicion is required, and colorectal cancer should not be excluded based on the patients age. It must also be noted that young patients may have intact nuclear expression on immunohistochemical staining but that does not exclude them from having aggressive CRC.
Suspected Myxedema Coma Refractory to Treatment Due to Interaction with Anti-Epileptic Drugs. Ning X, MD, Xiao E, MD, Lizama Hernandez S, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Myxedema coma is due to severe hypothyroidism causing decreased mental status and severe slowing of other organ systems. It is commonly treated with high doses of intravenous thyroid hormones, glucocorticoids, and supportive measures. Certain anti-epileptic drugs have an unrecognized drug interaction with levothyroxine which results in decreased serum concentration of levothyroxine and decreased therapeutic effect.

A 51 year old man with a history of thyroid cancer who underwent total thyroidectomy with radioiodine ablation two years prior to presentation was brought in after vehicular trauma where he was found pulseless. After achieving return of spontaneous circulation, he developed status epilepticus from anoxic brain injury for which he was started on a midazolam and ketamine drip. He was then loaded with fosphenytoin, lacosamide, and levetiracetam. During this time, he was found to have a TSH of 149.89 mIU/L (0.47-4.68 mIU/L) and a free T4 (FT4) of less than 0.1ng/dL (0.6-2.5 ng/dL).

Given his presentation and lab values, he was empirically treated with high dose IV levothyroxine, at an initial dose of 250mcg, followed by 100mcg daily and liothyronine 5mcg IV followed by 2.5mcg IV every 8 hours with minimal response in his free T4. Four days after starting treatment, his FT4 remained low, prompting an increase of levothyroxine. The remainder of his thyroid function studies at this time were as follows: TSH 50.18 mIU/L, total T3 (T3) 48 ng/dL (97-169ng/dL), total T4 (T4) 1.9 mcg/dL (5.1-13.8 mcg/dL). Over the next 11 days, his levothyroxine dose was increased incrementally to a maximum dose of 200mcg IV daily; however, his FT4 remained between 0.3-0.4ng/dL. At a dose of levothyroxine 200mcg IV daily, his labs were: TSH 51.77 mIU/L, T3 60 ng/dL, FT4 0.4 ng/dL, and T4 3.8ug/dL. During this period, the patient remained on phenobarbital, levetiracetam, fosphenytoin and lacosamide, often with supratherapeutic levels of free phenytoin. He did not gain meaningful neurological recovery, was transitioned to comfort care and ultimately expired.

This case demonstrates an example of drug-drug interactions. Phenytoin is known to displace thyroid hormones from their protein binding sites, increasing CYP450-mediated metabolism of the thyroid product, resulting in decreased serum concentrations or therapeutic effects of levothyroxine.

Program Director’s Name: Susan D. Wolfshal

(indicating review of abstract)

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!
2019 Mulholland Mohler Resident Meeting

| A CASE OF TRANSIENT GLOBAL AMNESIA |
| Sona Aloyan, MD |
| MedStar Harbor Hospital, Baltimore, MD |

**Introduction**
Transient global amnesia (TGA) is a clinical syndrome of reversible anterograde amnesia, a sudden loss of the ability to form new memories accompanied by repetitive questioning. TGA occurs in middle-aged and older individuals and lasts less than 24h.

**Case Report**
A 62-year-old man with a history of cluster headaches presenting with confusion and complete loss of memory for the last day. He had a long flight from China but could not recall any detail of the trip. He denied taking any medications or alcohol. He recognized his wife, could remember events before the trip, but repeatedly asks the date. A physical exam was otherwise remarkable for the inability to recall two out of three objects in three minutes. Toxicology screen was negative, vitamin B12 and TSH levels were normal, and RPR was nonreactive. MRI, carotid duplex and EEG were unremarkable. After several hours with no specific treatment, his confusion resolved and he returned to his baseline normal neurologic state.

**Discussion**
TGA is a clinical diagnosis, with no specific diagnostic tests to support it. It is a relatively rare syndrome, associated with a history of migraines. Patients are alert and oriented, and cognition is not impaired. No specific treatment is indicated because in most cases complete recovery is achieved in 24 hours with total amnesia for the event. Other conditions such as seizure, Korsakoff syndrome, TIA, or stroke should be considered. It is essential to obtain a detailed history, perform an accurate neurologic exam, and obtain blood work and imaging to rule out pathological causes for amnesia.

**Conclusion**
While the prognosis for TGA is generally benign, other diseases in the differential diagnosis carry the potential for long-term severe sequelae, making it essential for clinicians to be familiar with the diagnosis and evaluation.
**2019 Mulholland Mohler Resident Meeting**

**A Fortunate Anomaly**

**Introduction:** Total occlusion of the left main coronary artery (LMCA) and proximal left anterior descending artery (LAD) carry high morbidity and mortality and the presentation is usually catastrophic. We present a rare case of a patient presenting with total occlusion of the LMCA and proximal LAD who likely survived due to an anomalous left circumflex artery (LCX) originating from the right coronary cusp.

**Case:** A 60-year-old male with hypertension, dyslipidemia and a prior stroke presented complaining of constant, typical chest pain associated with dyspnea that started at rest, 4 hours before presentation. The chest pain improved upon arrival to the ED without any interventions and he remained hemodynamically stable. EKG revealed ST elevations in V2-V5 and ST depressions in the inferior leads. Troponin I was 2.54ng/mL. The patient was loaded with aspirin, heparin and ticagrelor and taken emergently to percutaneous coronary angioplasty (PCI). PCI revealed 100% LMCA occlusion extending into the LAD; lesions were stented. Coronary angiography also revealed an anomalous LCX artery originating from the right coronary cusp and right dominant circulation, which was likely the reason for patient’s survival and relatively benign presentation.

**Discussion:** Acute complete occlusion of the LMCA is rare and typically fatal unless intervened upon in a timely manner. Patients typically present with sudden death or profound cardiogenic shock, also known as left main shock syndrome. The treatments described in literature include placement of intra-aortic balloon pump, intracoronary thrombolysis, PTCA and emergency CABG however the optimal treatment modality is still undetermined. The prognosis of these patients is grave regardless of the treatment. A crucial factor affecting the prognosis is the presence of right dominant circulation and substantial collateral circulation to the left coronary artery with lower morbidity and mortality compared to patients without it. A review of literature did not identify cases of patients with total occlusion of the LMCA who presented with mild chest pain and who were hemodynamically stable due to the presence of an anomalous LCX originating from the right coronary cusp.

**Conclusions:** Complete occlusion of LMCA is a serious entity with grave prognosis. It is critical that physicians recognize possible LMCA involvement early and proceed with PCI in a timely manner to improve outcomes. Simultaneous implantation of IABP during PCI can help maintain the patient's hemodynamic status during the critical ischemic time.

---

**First Author Information:**

**Name:** Fahad Ahmed

**Institution:** St Agnes Hospital

**Daytime Phone:**

**Co-Author(s):** Sirajum Munira, Matthew Voss

**Program Director’s Name:** Sapna Kuehl, MD, FACP

(indicating review of abstract)
NEAR SYNCOPE AND COMPLETE HEART BLOCK AS A PRESENTATION OF INFECTED ST-ELEVATION MYOCARDIAL INFARCTION. Augenstein C, MD. The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Inferior myocardial infarction (MI) complicated by atrioventricular block (AVB) occurs in approximately 6 to 13% of patients with inferior MI and is associated with a poorer prognosis. The atrioventricular (AV) node is supplied from the posterior descending artery or posterior interventricular artery, a branch of the right coronary artery (RCA) in right-dominant individuals. Patients with AVB associated with acute inferior ST-elevation MI are expected to improve with percutaneous coronary intervention (PCI), thus permanent pacemaker is often not required.

A 54-year-old man with a history of liver and kidney transplant four months prior to this presentation, orthostatic hypotension and diabetes mellitus presented to the Emergency Department (ED) with a one-day history of fatigue and near syncope. As part of his pre-transplant evaluation he had undergone a nuclear stress test four months prior to this presentation, which was negative for ischemia. His fatigue had been attributed to his orthostatic hypotension, but his near syncope led to his presentation to the ED.

The initial electrocardiogram (EKG) showed complete heart block and inferior ST elevations. He was referred for emergent coronary angiography. He was found to have severe multi-level coronary artery disease. He was found to have acute total occlusion of the distal right coronary artery. He developed cardiogenic shock requiring vasopressors while in the catheterization laboratory. He underwent successful PCI of the mid to distal RCA with two overlapping drug eluting stents (DES). The complete heart block and cardiogenic shock resolved within minutes after restoring flow to the RCA. After PCI his fatigue resolved.

This case illustrates an inferior MI with RCA occlusion as an uncommon and reversible cause of complete heart block. When reviewing the EKG of a patient with near syncope it is important to look for signs of both ischemia and heart block since they may occur together. Revascularization may reverse the heart block and avert the need for permanent pacemaker.

First Author Information:
Name: Cheryl Augenstein, MD
Institution: University of Maryland Medical Center and Baltimore VA Medical Center
Daytime Phone: 410-328-7567

Co-Author(s) Associates:

Program Director's Name: Susan D. Wolfsthal

(indicating review of abstract)
TRACHEAL STENOSIS AFTER PROLONGED INTUBATION
Sahiba Khurana MD, Pavan Bhat MD, Phil Buescher, MD
MedStar Health Internal Medicine, Baltimore, Maryland

Tracheal stenosis is narrowing of the upper airway that can be caused by scar tissue or cartilage malformation. It is a feared complication of prolonged endotracheal (ET) intubation and tracheostomy but true incidence is not yet understood. Patients present with a constellation of non-specific respiratory symptoms and can be missed without appropriate diagnostic investigation.

A 74-year-old obese man presented to the hospital due to progressive dyspnea with inspiratory stridor. Two months prior following cardiac stress test, he underwent surgical aortic valve replacement and coronary artery bypass grafting (CABG). His postoperative course was complicated by myocardial infarction and respiratory failure requiring repeat CABG and 10 days of endotracheal intubation. He was eventually extubated and transferred to a rehabilitation facility. On re-presentation, he was thought to have pneumonia but respiratory decompensation persisted despite antimicrobial therapy and steroids. Initial CT scan of chest and neck reported no notable tracheal lesions. He was seen by ENT who saw intact vocal cords but did not probe below this level. His respiratory status worsened and he was transferred to ICU. He underwent emergent bedside bronchoscopy, which showed tracheal occlusion due to extensive granulation tissue. He then underwent tracheostomy and was discharged on tracheal collar.

ET intubation is a commonly performed, high-risk, lifesaving procedure for respiratory failure with almost half a million intubations performed in the US annually. Tracheal stenosis is an under-recognized complication. Incidence in literature is broad from 0.6 to 21% with 1-2% of these patients presenting with symptoms. Tracheal damage occurs when ET tube cuff pressure is greater than the mucosal capillary bed, resulting in ischemia of the underlying tissue. This can lead to ulceration followed by fibrosis leading to airway narrowing. Intubation for more than 10 days is a strong risk factor, and symptoms can develop up to 3 months post-extubation. Stenosis can be evident on imaging, but the mainstay of diagnosis is bronchoscopy. Given difficulty in differentiating the non-specific stridor and wheezing that accompany the dyspnea, patients are undiagnosed or misdiagnosed as asthma. In our patient, this diagnosis was not even in the initial differential. Delay in diagnosis can lead to inappropriate treatment and complications so clinicians must maintain a high level of suspicion for this condition.

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!
DIAGNOSTIC DILEMMA OF HYPERSENSITIVITY PNEUMONITIS IN A COMMUNITY SETTING

Man Kit Siu, MD; Kedaari Anant, MD; Raman Bharaj, MD
MedStar Health Internal Medicine, Baltimore, MD

Hypersensitivity pneumonitis (HP) is a spectrum of disease that stems from the lung responding to inhaled antigens. Only a small percentage of individuals exposed to an antigen will develop HP and for the majority of antigens this percentage is unknown. The presentation can mimic many other lung pathologies, often leading to misdiagnosis and delayed treatment. The importance of history taking and being familiar with the diagnostic criteria are important to identify cases as severe cases that can lead to death.

A 49-year-old Caucasian woman with a past medical history significant for rheumatoid arthritis presented with dyspnea and cough. She also recently visited her father in Alabama and was cleaning the old family home. Her review of systems was positive for myalgia, lightheadedness, headache and lethargy. Her vitals revealed hypotension and tachycardia. ESR and CRP were elevated to 105 and 91. ANA was positive with a titer of 1:80. Laboratory testing was negative for influenza, RSV, HIV, adenovirus, bordetella, coronavirus, chlamydia, human metapneumovirus, rhinovirus, parainfluenza, mycoplasma, streptococcus, legionella, staphylococcus, bacteremia, histoplasma, aspergillus, cryptococcus and beta D glucan. A CT scan revealed ground glass infiltrates. While the cause for her presentation was being worked up, she was presumptively diagnosed with pneumonia and started on empiric antibiotics and later antifungals with no improvement clinically and worsening of her infiltrates upon repeat CT. Given her history, clinical picture, and imaging findings, we diagnosed her with hypersensitivity pneumonitis and she was started on steroids with remarkable improvement in her symptoms and CT findings within three days.

The current diagnostic criteria for HP include an invasive procedure with either bronchoscopy, inhalation challenge testing, or compatible histopathology. These are not always possible in a community hospital and though it is ideal to perform the invasive procedures for accurate diagnosis, taking a good history and using good clinical judgement can help manage symptoms and prevent poor outcomes.

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!
2019 Mulholland Mohler Resident Meeting

HEADACHE AND OPHTHALMOPLEGIA

Tatiana Policarpo, MD; Orit Abrahim, MPH; Rupert Hung, MD; Amit Goyal, MD
The Johns Hopkins Hospital, Baltimore MD

Case: A 76-year-old Sudanese woman presented with two years of progressive periorbital edema, headache, and painful ophthalmoplegia. She was diagnosed with allergic conjunctivitis when she originally presented with bilateral conjunctival injection. However, this progressed to include unilateral right-sided headaches, diplopia worsened with bilateral gaze, and ultimately bilateral periorbital edema with proptosis. She sought care in Egypt where she was diagnosed with Tolosa-Hunt syndrome (a chronic inflammatory disorder) and cavernous sinus thrombosis based on imaging, for which she was started on chronic steroids with minimal improvement. She thus presented to the Johns Hopkins Hospital.

Exam was notable for conjunctival injection, prominent episcleral vessels, bilateral proptosis, and right-left cranial nerve VI palsy, with intact visual acuity. Extensive evaluation for autoimmune, infectious, and neoplastic etiologies were negative. Head CT/MRI primarily notable for bilateral superior ophthalmic vein thrombosis.

Subsequently, a cerebral angiogram revealed the culprit: bilateral carotid-cavernous sinus fistulae (CCF). A potentially curative transcatheter embolization attempt was aborted due to difficulty accessing the fistula as a result of SOV thrombosis and osteodural component of the fistulous connection. Surgery was felt to be too high risk. She thus underwent stereotactic radiosurgery targeting the osteodural track with improvement in her symptoms.

As clinicians, we frequently encounter headaches as a presenting symptom in both the inpatient and outpatient settings and must recognize warning signs that differentiate life-threatening from benign etiologies. In this case, the concomitant painful ophthalmoplegia, and the findings of prominent episcleral vasculature and cranial nerve palsy prompted concern for CCF -- an uncommon entity that may carry high morbidity and mortality with delayed diagnosis. In addition to highlighting a typical presentation of an uncommon disease, this case demonstrates the pitfalls of anchoring heuristics distracting our early diagnostic workup, which had focused on medical inflammatory etiologies rather than a structural one.

AMERICAN COLLEGE OF PHYSICIANS

MARYLAND REGION
ANNUAL ASSOCIATES MEETING
MAY 9, 2019

Please check one. First author is:
( X ) RESIDENT
Please check only one. Abstract is submitted to.
( ) Poster
( ) Oral
( ) Either

General Classification:
( X ) Clinical Vignette
( ) Research Competition
( ) Basic Science
( ) Evidence based medicine review
( ) Quality/Safety
( ) Clinical Research

Indicate your participation in research process (4 sentences or less):

First Author Information:
Name: Tatiana Policarpo, MD
Institution: The Johns Hopkins Hospital
Daytime Phone: 954-328-8470

Co-Author(s) Associates:
Orit Abrahim, MPH

Program Director’s Name:
Sanjay Desai, MD
(indicating review of abstract)
2019 Mulholland Mohler Resident Meeting

MANIFESTATION OF ANTI-PHOSPHOLIPID SYNDROME SECONDARY TO WALDENSTROM MACROGLOBULINEMIA.

Kim D, MD. University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Anti-phospholipid syndrome (APS) is a clinical syndrome of thrombosis due to anti-phospholipid antibodies (aPL) and can be primary or secondary in nature. The most common secondary cause of this disorder is Systemic Lupus Erythematosus (SLE). Other possible causes of APS include infection, malignancy and drugs. While rare, paraproteins can have lupus anticoagulant activity leading to APS.

The patient arrived as a transfer from an outside hospital with extensive necrotic lesions. Physical exam showed dusky hands and fingers along with extensive areas of purpura and multiple areas of eschar. There were partial necrotic lesions on the tip of the nose and bilateral pinnae of ears as well as splinter hemorrhages in the nailbeds. These findings were concerning for APS.

Labs showed aPL IgM titer of 148 micrograms with positive Lupus Anticoagulant activity with coagulation tests showing activated partial thromboplastin time (aPTT) of 77.4. SLE immunologic testing was otherwise unremarkable with negative double stranded deoxyribonucleic acid antibodies and smith antibodies. The patient was also found to be anemic to a hemoglobin of 6.8 grams per deciliter and work up showed a positive Coombs test and cold agglutinins titer raising suspicion for an associated malignancy. Serum protein electrophoresis (SPEP) revealed 0.27 g/L M protein and serum immuno fixation positive for IgM kappa. Urine protein electrophoresis (UPEP) was positive for free kappa light chain and urine immuno fixation positive for IgM bands. These were concerning for Waldenstrom Macroglobulinemia (WM).

For APS, the patient received plasma exchange in addition to rituximab and steroids. A bone marrow biopsy for WM was performed. After this, his course was complicated by multiple necrotizing soft tissue infections with Klebsiella bacteremia requiring a left lower extremity amputation. In addition, coagulopathies led to refractory bleeding. Patient ultimately elected for hospice and declined chemotherapy for WM. After discharge, the bone marrow biopsy confirmed lymphoplasmacytic lymphoma.

The phenomena of paraprotein by WM is likely the cause of the strong lupus anticoagulant activity leading to APS. IgM lambda paraprotein can display specificity for negatively charged phospholipids phosphatidyl serine and phosphatidyl inositol providing a potential mechanism for this phenomenon.
2019 Mulholland Mohler Resident Meeting

GASTRIC PNEUMATOSIS: A RARE COMPLICATION OF GASTRIC ULCERS
Saba Ahmed, MD
MedStar Harbor Hospital, Baltimore, MD

Background
Pneumatosis intestinalis (PI) refers to the presence of gas within the wall of the small or large intestine. Intramural gas less commonly affects the stomach and is referred to as gastric pneumatosis.

Case presentation
An 80-year-old man initially presented with hypotension and altered mental status. On exam, his abdomen was nondistended and non-tender, with normal bowel sounds and no masses or organomegaly. CT scan showed a distended stomach with pneumatosis. He had a partial gastrectomy, and he was found to have dense inflammation in the left upper quadrant between the stomach and the spleen. Large necrotic gastric ulcers in the anterior and posterior walls adjacent to the greater curvature. Upper G.I. series was done on postop day three and showed no sign of a leak. He tolerated a diet, had the return of bowel function, remained afebrile with normal vitals and labs, and was discharged back to his nursing home.

Discussion
There are many causes of gastric pneumatosis such as the invasion of the stomach wall by gas-forming organisms (gangrenous stomach), ischemic infarction of the stomach, penetrating injury (from instrumentation), and gastric emphysema (usually precluded by gastric distension and vomiting). Gastric ischemia is a rare cause (unlike pneumatosis intestinalis) due to the abundant blood supply of the stomach. In our patient, gastric pneumatosis was thought to be caused by perforation of a gastric ulcer. Since penetrating injury is an established cause of gastric pneumatosis, perforation of gastric ulcers can be thought of as a form of penetrating injury.

Conclusion
Gastric pneumatosis may be an incidental finding associated with a benign etiology, whereas in others, it portends a life-threatening intra-abdominal condition. The latter requires emergency surgery, as was the case for our patient.

Program Director’s Name: Dr. Detterline

(Indicating review of abstract)