BREAKOUT
ROOM
NUMBER TWO

CV 11 - 20
ACUTE RENAL FAILURE LEADING TO DIAGNOSIS OF HIV

Human Immunodeficiency Virus (HIV) - associated nephropathy is one of the leading causes of kidney failure. However, it is unusual for nephropathy to be the initial presentation of HIV. Here we discuss an otherwise healthy female presenting with acute renal failure due to HIV-associated nephropathy as the initial presentation of HIV.

A 55-year-old female presented to her primary care physician in November 2019 with worsening renal function in routine workup. The patient had a normal BUN and creatinine in October 2018. During an office visit in October 2019, BUN/Creatinine were elevated to 31/3.28. Nephrology follow-up showed proteinuria and microscopic hematuria on urinalysis. Kidney ultrasound showed increased echotexture of bilateral renal parenchyma. Repeat bloodwork showed BUN and creatinine of 34 and 4.24 respectively and patient was admitted to the hospital for evaluation. Abdominal CT without contrast showed splenomegaly, as well as prominent retroperitoneal and inguinal lymph nodes. CBC showed anemia and thrombocytopenia. The patient also admitted to 20 lbs weight loss in 2 months. A full work-up including serum protein electrophoresis (SPEP)/ urine protein electrophoresis (UPEP), Hepatitis B and C, HIV, ANA, and C3/C4 was done. SPEP showed monoclonal gammopathy and urine was negative for Bence-Jones protein. HIV testing came back positive with CD4 count of 98. A subsequent kidney biopsy showed diffuse global and segmental glomerulosclerosis with rare collapsing changes. Patient was started on anti-retroviral treatment (ART), as well as Pneumocystis jiroveci prophylaxis. She was started on Prednisone 60mg for 8 weeks, which was discontinued after only 4 weeks due to concern for worsening immunosuppression. Over the next several months, the patient’s kidney function remained stable with a creatinine of around 4.5. The patient is currently being evaluated for a possible kidney transplant and continuing with her ART.

Though nephropathy is a known complication of HIV infection, it is rare to see acute renal failure as the initial presenting sign of HIV infection. HIV-associated nephropathy is generally seen in patients with known HIV infection for many years. HIV infection is a serious infection which is life threatening without prompt and proper treatment. Furthermore, studies show significant increase in survival in HIV patients on dialysis when using ART compared to no HIV treatment. Therefore, early recognition of HIV, as well as HIV-associated nephropathy is very important for both survival benefit and quality of life.

Program Director’s Name: Stephanie Detterline, MD, FACP

(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:

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General Classification:

- [X] Clinical Vignette
- [ ] Research Competition
  - [ ] Basic Science
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  - [ ] Quality/Safety
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Indicate your participation in research process (4 sentences or less):

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First Author Information:

Name: Ufara Zuwasti, MD

Institution: MedStar Health Internal Medicine, Baltimore, MD

Daytime Phone: 503 464 6195

Co-Authors/Associates:

Adel Yazji, MD, MedStar Health Internal Medicine, Baltimore, MD
Ricardo Quarrie, MD, Heart and Vascular Institute, MedStar Union Memorial Hospital, Baltimore, MD
Elizabeth Allen, MD, Department of Pathology, MedStar Union Memorial Hospital, Baltimore, MD
Christopher Haas, MD PhD, Member, Department of Internal Medicine, MedStar Franklin Square Medical Center and MedStar Harbor Hospital, Baltimore, MD

Program Director’s Name: Stephanie Detterline, MD

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 RARE BUT DEADLY CAUSE OF ABDOMINAL PAIN
Muhammad Nadeem Yousaf MD, Prabij Dhungana MD, Albert S. Fleisher MD, Charmian D. Sittambalam MD FACP
Medstar Health Internal Medicine Residency Program, Baltimore, MD

A biloma is an encapsulated collection of bile outside the biliary tree with an incidence of 0.3%-2%. The rarity with which this occurs makes it an uncommon etiology on the differential of right upper quadrant (RUQ) abdominal pain. The most common etiologies are choledocholithiasis, abdominal trauma, and iatrogenic causes such as after cholecystectomy or hepatobiliary interventions.

A 91-year-old female presented with acute onset of RUQ pain for 1 day that was sharp, radiating to her back, and associated 2-3 episodes of non-biliary, non-bloody vomit. She denied fever, diarrhea, weight loss, abdominal trauma, or prior abdominal surgery. Clinical examination showed RUQ tenderness without rebound tenderness or Murphy’s sign. Laboratory workup showed lipase 5700 U/L, lactic acid 2.2 mmol/L, and creatinine 1.71 mg/dl. Computed tomography scan of abdomen and pelvis showed a distended gallbladder with wall thickening, but without evidence of pericholecystic or gallstones. Abdominal ultrasonography (US) showed trace intramural and pericholecystic fluid with no abnormality of the common bile duct (CBD). The hepatobiliary iminodiacetic acid (HIDA) scan findings were consistent with extrabiliary biliary leakage into the peritoneum. Magnetic resonance cholangiopancreatography (MRCP) revealed moderate pericholecystic and perihepatic fluid collection. A cholangiogram demonstrated a perihepatic biloma, which was drained under guidance of fluoroscopic imaging. On endoscopic retrograde cholangiopancreatography (ERCP), there was no clear evidence of contrast extravasation, however a blush of contrast at the junction of the cystic duct and the common hepatic duct was seen, which correlated with the location of biloma noted on cholangiogram. A 10 French plastic stent was placed in CBD across this point, after which the patient’s symptoms completely resolved, and he was discharged home.

The clinical presentation of a biloma is variable, ranging from abdominal fullness, pain, fever, and jaundice, to rarely peritonitis without fever. SBL is a rare etiology of biloma and may result in delayed diagnosis and interventions. HIDA scan is the most effective diagnostic imaging. A fluoroscopic-guided percutaneous drainage, ERCP biliary stenting results in the complete resolution of biloma and SBL. Emergent surgical exploration is required in hemodynamically unstable patients as septic shock, biliary peritonitis, biliopleural fistula, or bilhemia can increase risk of mortality.

CV 13

BREAKOUT ROOM 2

Program Director’s Name: Stephanie Deterline, 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!
SUICIDE LEFT VENTRICLE: OBSTRUCTIVE PHYSIOLOGY FOLLOWING TRANSCATHETER AORTIC VALVE REPLACEMENT. Fitch, J. MD, Davis, I. MD, The University of Maryland School of Medicine and VA Medical Center, Baltimore, MD.

Transcatheter aortic valve replacement (TAVR) is a percutaneous procedure increasingly used to treat symptomatic severe aortic stenosis. This vignette discusses a rare consequence of aortic valve replacement, known as suicide left ventricle, which presents a diagnostic challenge and requires close attention to the patient in the hours following TAVR. Patients with longstanding aortic stenosis may develop left ventricular hypertrophy (LVH) due to the increased afterload. The sudden decrease in afterload after TAVR can lead to dynamic intra-ventricular gradients, similar to those seen in hypertrophic obstructive cardiomyopathy. This can lead to acute hemodynamic collapse.

An 83-year-old female with a history of moderate aortic stenosis (aortic valve area of 1.1 cm² and mean gradient across the aortic valve 22 mmHg), LVH with baseline ejection fraction (EF) >70%, and hypertension, originally presented to an outside hospital emergency department with syncope. For a few months prior to admission, the patient had been experiencing dyspnea and had had several syncopal events. She was thought to have developed symptomatic severe aortic stenosis with outside hospital trans-esophageal echocardiogram demonstrating restricted left aortic leaflet. Patient was referred for aortic valve replacement evaluation and TAVR procedure was planned.

During the procedure, patient developed bradycardia secondary to complete heart block. She was given fluids, phenylephrine, and was transvenously paced. She returned to sinus rhythm about one hour later. Approximately 17 hours after her procedure, the patient’s mean arterial pressure (MAP) decreased to 50 mmHg. Bedside echocardiogram demonstrated hyperdynamic LV EF with a septal knuckle bulging into the LV outflow tract. Formal transthoracic echocardiogram (TTE) showed EF 85% with near-obliteration of the LV cavity. In order to increase preload, she was treated with 5L of intravenous fluids. Phenylephrine was started to increase vaso-constriction while minimizing any increase in contractility. Beta-blockade was strongly considered but not given due to the patient’s recent complete heart block. MAP improved to >65 mmHg over the next few hours and repeated TTE 24 hours later no longer demonstrated obstructive physiology.

Following TAVR, suicide LV can cause severe hemodynamic changes. Since treatment may differ from therapy for other etiologies of shock, early identification is paramount.
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( ) Research Competition
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Indicate your participation in research process (4 sentences or less):

First Author Information:

Name: Steven Liskov, MD

Institution: University of Maryland Medical Center and Baltimore VA Medical Center

Daytime Phone: 570-762-1162

Co-Author(s) Associates: Jeffrey Fitch, MD

Program Director’s Name: Susan Wolfsthal, MD

(indicating review of abstract)

DEEP VEIN THROMBOSIS PROVOKED BY COMPRESSION OF AN Iliac VEIN BY A UTERINE LEIOMYOMA. Liskov S, Fitch J. The University of Maryland School of Medicine and Veterans Affairs Medical Center, Baltimore, MD.

Deep vein thrombosis (DVT) can lead to pulmonary embolism, a significant cause of morbidity and mortality. The pathogenesis for DVT can be described by Virchow’s triad, which includes hypercoagulability, stasis, and endothelial injury. Risk factors for DVT include hereditary and acquired factors. Major acquired risk factors include recent surgery, malignancy, pregnancy, oral contraceptives, and immobilization. Uterine leiomyomas, or fibroids, occur in 20-30% of women above the age of 30. Given the uterus’s proximity to the pelvic veins, direct venous compression can lead to stasis and therefore thrombosis.

A 39-year-old nulliparous female with a history of menorrhagia presented to the Emergency Department for acute left-lower extremity (LLE) pain and edema. She denied history of DVT, active malignancy, autoimmune syndrome, tobacco use, oral contraceptive use, or family history of prothrombotic disease. The patient’s last menstrual period occurred 10 days before presentation and was associated with menorrhagia. Physical exam revealed asymmetric edema, erythema, and poster calf and thigh tenderness of the LLE. Initial evaluation included a venous duplex study of the LLE which revealed acute occlusive DVT extending from the superficial femoral vein to distal calf veins. Subsequent CT angiogram of the abdomen and pelvis with femoral arterial runoff was performed to identify any predisposing anatomical irregularities. The study captured bilateral segmental and subsegmental pulmonary artery emboli. It also revealed a large uterine leiomyoma compressing the left common iliac vein associated with acute thrombus of the left common and external iliac veins and left superficial femoral vein. Therapeutic anticoagulation was achieved with low molecular-weight heparin. The patient’s course was complicated by severe iron-deficiency anemia with initial hemoglobin 6.0 g/dL and ferritin 10 µmol/L for which transfusion was performed. The patient was discharged home in stable condition on apixaban and was scheduled for elective total abdominal hysterectomy.

Direct compression of the pelvic venous system by a uterine leiomyoma leading to DVT has been rarely described in the literature. In females who are diagnosed with DVTs without an obvious alternative etiology, advanced imaging may be considered to evaluate for unusual structural causes, including leiomyomata. Anticoagulation in these cases may be complicated by menorrhagia. Therapies including inferior vena cava filters and hysterectomy may be required to minimize the risk of venous thromboembolism.
443-257 INFECTIOUS COLITIS CAUSED BY CYTOMEGALOVIRUS, HERPES SIMPLEX VIRUS-1, AND HERPES SIMPLEX VIRUS-2 IN A PATIENT WITH ACQUIRED IMMUNE DEFICIENCY SYNDROME. Hale D, MD, Kathari Y, MD. The University of Maryland School of Medicine and Baltimore VA Medical Center, Baltimore, MD.

Diarrhea in a patient infected with Human Immunodeficiency Virus (HIV) has a significant impact on quality of life and, in patients with advanced HIV infection, can lead to increased mortality. Immunocompromised patients with symptoms suggestive of colitis, including abdominal pain and bloody stool, and supportive findings of inflammation on imaging, lead the examiner to consider diagnoses of cytomegalovirus (CMV), herpes simplex virus (HSV), and Clostridium difficile (C. difficile). These pathogens are typically singular causes of infectious diarrhea and co-infection is not well documented.

A 48-year-old man with HIV presented to the hospital with shortness of breath and a one-month history of diarrhea. The patient reported watery stools every 20 minutes associated with cramping abdominal pain. He was found to be hypoxic and hypotensive requiring admission to the intensive care unit with vasopressor and supplemental oxygen support.

Physical examination revealed bibasilar rales and hyperactive bowel sounds with diffuse tenderness to abdominal palpation without rebound or guarding. CD4 count was less than 20 cells/mm³. C. difficile testing was negative, fecal leukocytes were positive, and CMV viral load was 229,000 copies/mL. Computed tomography scan of the abdomen revealed multifocal areas of colonic wall thickening consistent with colitis. On the second day of admission, the patient had four bloody bowel movements with clotted blood. Subsequent colonoscopy revealed diffuse colitis with multiple ulcers, from which biopsies were taken. At that time, empiric treatment for CMV colitis was initiated with ganciclovir. His abdominal pain and diarrhea were treated symptomatically with dicyclomine. Pathology returned positive for CMV, HSV-1, and HSV-2. The patient was successfully transitioned to valganciclovir and completed 3 weeks of treatment with improvement in symptoms.

Although CMV and HSV are known to independently cause colitis in patients infected with HIV, we present a patient found to have co-infection with three viral etiologies for his diarrhea, likely attributing to his severe presentation. Further, this case exemplifies the importance of antiretroviral therapy in preventing opportunistic infections in a patient infected with HIV.

First Author Information:

Name: David E. Hale, MD

Institution: University of Maryland Medical Center and Baltimore VA Medical Center

Daytime Phone: (443) 257-3115

Co-Author(s) Associates:

Yamini K. Kathari, MD
Rhabdomyolysis Secondary to Rhinovirus

Rhabdomyolysis is a condition characterized by rapid skeletal muscle cell breakdown, resulting in muscle breakdown products that can cause a wide range of complications, from serum electrolyte derangements to life-threatening end-organ damage.

A 26-year old woman presented to the emergency department because of two days of pain and weakness in her shoulder girdle, thighs, and hips as well as nasal congestion and discharge. On presentation, her vital signs were within normal limits. Neurologic examination was significant for bilateral brachioradialis, patellar and achilles hyporeflexia and 4/5 strength in her leg and hip flexors, and 4/5 strength in her shoulder abductors. The remainder of the examination was within normal limits. A CBC showed mild anemia and mildly elevated WBC count, while the UA on presentation was significant for 3+ blood on dipstick but no RBC on microscopic examination. ESR and CRP were elevated at 108 mm/hr and 22.7 mg/dL respectively. CPK was elevated to over 380,000 mg/dL.

The patient used no medications and denied any recent injury, drug use, excessive exercise, or seizure. Rheumatologic evaluation, Hepatitis C antibody, amylase, and lipase were all unremarkable. Hepatic function panel was significant for AST and ALT of 1138 and 182 respectively. Influenza, strep swab and Epstein-Barr serology were negative. A respiratory viral panel was positive for rhinovirus but negative for other respiratory viruses.

The patient initially received 3L of lactated Ringers solution and started on continuous IV fluids at 250 mL/hr. Her creatinine peaked at 1.0 on the day of admission and quickly downtrended to < 0.5 mg/dL. Serum potassium did not exceed 4.3. The patient remained on continuous IV fluids at a high rate for 12 days, at which point her CK decreased to below 1000 mg/dL and she was discharged to home. By the time of discharge, her symptoms of weakness and pain had completely resolved.

This case illustrates a rare case of rhabdomyolysis in a healthy young patient with no injuries, medications, or drug use. Though several other cases of rhinovirus-induced rhabdomyolysis exists in the literature, most cases are mild in nature and occur in the pediatric population. Recognition of rhabdomyolysis in acute viral illness and rapid treatment with appropriate IV fluid therapy is important in preventing long-term sequelae.

CV 17

BREAKOUT ROOM 2

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!
ALL THAT FEVERS IS NOT INFECTION: ACUTE POLYARTICULAR GOUT MANIFESTING AS FEVER WITH ALTERED MENTAL STATUS
Merna Hussien, MD; Shannon Walker, MD

Introduction:
Flares of polyarticular gout occur more frequently in those with poorly controlled disease. These flares induce an intense systemic inflammatory response that can mimic sepsis. Gout-induced encephalopathy is, however, a rarely described entity. Here, we report a case of persistent fevers, encephalopathy, and supraventricular tachycardia (SVT) precipitated by a polyarticular gout flare, posing a diagnostic challenge.

Case Summary:
A 64-year-old African American man with known history of gout, alcohol use disorder, and suspected cirrhosis, presented to the ED with palpitations after abrupt cessation of alcohol consumption 5 days prior. He was admitted for alcohol withdrawal and started on benzodiazepine symptom-triggered therapy. He was initially cognitively intact, but became progressively delirious, even after 48 hours of cessation of benzodiazepine therapy. He began to have intermittent febrile, hypoxic, and SVT episodes. Physical exam was significant for tachypnea, GCS of 10, and bilateral knee swelling; noted to be chronic by family. He was empirically treated for presumed aspiration pneumonia with ceftriaxone, and for hepatic encephalopathy with lactulose, all without symptomatic improvement. He underwent an extensive infectious work-up, including multiple cultures of blood, urine, and sputum, an echocardiogram, and CT imaging of chest, abdomen, head and spine, which was unrevealing. LP was unsuccessful due to habitus and anatomy. Due to the presence of progressive knee swelling and warmth on subsequent physical exams, an arthrocentesis was performed and revealed monosodium urate crystals. Steroid treatment was started for gout flare with dramatic improvements in mental status and pain within the following 48 hours. His fevers and SVT episodes subsided. He was then transitioned to oral colchicine, and eventually started on allopurinol after resolution of his acute flare.

Discussion:
We propose that our patient's gout flare, which precipitated while hospitalized, was the underlying driver of his presentation of fever, and altered mental status. He was particularly susceptible to delirium from the systemic inflammatory response of gout, due to chronic alcohol use and suspected alcoholic cirrhosis. Sequential physical examinations and revisiting of differential diagnoses were crucial to identifying the culprit precipitant, and providing appropriate treatment for our patient.
WARBURG EFFECT IN A PATIENT WITH POORLY DIFFERENTIATED NEUROENDOCRINE TUMOR

John J. Lofrese, MD – ACP Member, WRNMMC, Bethesda, MD
Sarah Shulte – Temple University, Philadelphia, PA
J. Paul Happel, MD – ACP Member, WRNMMC, Bethesda, MD

Introduction:
Neuroendocrine tumors (NETs) most commonly originate from the GI tract, pancreas, or lung; however, around 10-14% of NETs are of unknown primary site. These malignancies can present with a variety of symptoms owing to the production of a diverse array of compounds. We describe a patient with a poorly differentiated NET of unknown primary with significant liver metastases complicated by a lethal lactic acidosis due to the Warburg effect.

Case Presentation:
A 64-year-old female, active smoker with a history of coronary artery disease, poorly characterized chronic hyponatremia, and two years of cognitive impairment of unknown etiology, presented to the ED with 1-week of diffuse abdominal pain, nonproductive cough and non-bilious, non-bloody vomiting. Initial lab work-up revealed leukocytosis, acute worsening of hyponatremia, anion gap acidosis, and cholestatic hepatitis. CXR revealed a new left upper lobe infiltrate and hilar fullness. Abdominal CT demonstrated new hepatomegaly with innumerable small hypoattenuating lesions. The patient was admitted to the ICU, started on empiric antibiotics, and given 4-liters of fluids with return to baseline sodium levels. A chest CT revealed a large left hilar lung mass concerning for primary lung malignancy. Liver biopsy revealed small blue cells strongly positive for synaptophysin and CD-56 suggestive of poorly differentiated NET; however, stains for TTF-1, classically associated with pulmonary origin, were negative. Urinary 5-HIAA and serum chromogranin A were not independently obtained. On hospital day six, the patient became hypoxic and was found to be acidic with a lactate of 9.8 mmol/L. After a brief period of PEA arrest with worsening lactic acidosis despite ongoing fluid resuscitation, the patient passed. Autopsy revealed a NET in the left lung with extensive hilar lymphadenopathy. Given the poorly differentiated tissue noted on histopathology, origin of the net could not be confirmed, but clinical suspicion remained for primary small cell lung cancer.

Discussion:
Poorly differentiated small cell carcinomas are rapidly growing and diffusely metastatic. The Warburg Effect was likely accelerated in this case due to metastatic burden in the liver, where roughly 60% of lactate is metabolized. Of interest in this case is whether a less aggressive NET had been present for some time, complicated by chronic paraneoplastic SIADH and encephalomyelitis or limbic encephalitis.

First Author Information:
Name: John Lofrese
Institution: Walter Reed National Military Medical Center
Email Address: johnlofrese@gmail.com
Cell Phone: (516) 457-0965

Program Director’s Name: Dr. Joshua Hartzell

(indicating review of abstract)
A CASE OF WERNICKE’S ENCEPHALOPATHY FOLLOWING ROUX-EN-Y GASTRIC BYPASS

Lucy Ma¹, MD, Dinh Dung², BS, Kevin Chien², BSE, Isaiah Horton¹, MD, Erika Walker¹, MD ¹National Capital Consortium, Bethesda, MD ²Uniformed Services University of the Health Sciences, Bethesda, MD

Introduction: Nutritional deficiency is a common complication of bariatric surgery. Despite vitamin supplementation protocols, rare and potentially fatal vitamin deficient states, such as Wernicke’s encephalopathy (WE), can occur. WE is an acute neuropsychiatric syndrome caused by thiamine (vitamin B1) deficiency with a classic triad of nystagmus and ophthalmoplegia, mental status changes, and ataxia. It more commonly occurs in patients who abuse alcohol but can be seen in patients who have had bariatric surgery. Initial symptoms of thiamine deficiency prior to progression to WE are non-specific thereby lending to the difficulty in diagnosis. We report a case of WE status post Roux-en-Y gastric bypass (RYGB).

Case: A 27-year-old woman with past medical history of obesity underwent RYGB in July 2019. She had no immediate post-operative complications, was tolerating a regular diet and taking over-the-counter multivitamins as directed. By postoperative month three, the patient reported increasing nausea and vomiting and was diagnosed with dumping syndrome. She was then instructed to avoid complex starches; however, her symptoms continued to worsen despite strict dietary adherence. In addition to the nausea and vomiting, she developed severe anorexia, profound weakness, extreme vertigo and blurry vision with hypokalemia and ketosis requiring hospital admission. Neurological exam revealed ataxia and horizontal jerk nystagmus on lateral gaze bilaterally that did not fatigue, suggestive of a central vestibular lesion. An MRI of the brain was normal. Thiamine levels were low at 50.8 ug/L (normal range: 66.5 - 200 ug/L). Given the constellation of symptoms in the context of a recent bariatric operation, the patient was diagnosed with WE and started on IV thiamine with symptomatic improvement within 12 hours. She continued to receive IV thiamine 500 mg every eight hours for 3 days with rapid resolution of nystagmus, ataxia, nausea, vomiting and fatigue. She was discharged on a fortified vitamin regimen based on the American Society for Metabolic & Bariatric Surgery guidelines.

Discussion: This case illustrates the potential for severe, irreversible consequences of vitamin B1 deficiency in RYGB patients. It underscores the importance of sufficient postoperative vitamin supplementation as well as recognition of the complex presentation and subtle physical exam findings in the proper and expedited diagnosis of WE.