INTRODUCTION:

An 87-year-old male with a history of Crohn’s disease (CD) and bowel resection presented with 2 weeks of diffuse abdominal pain. On examination he was hypertensive and somnolent with slurred speech. His abdomen was soft but tender, with scars from abdominal surgeries. Blood work revealed leukocytosis and anion-gap metabolic acidosis (AGMA) with LA. Testing for ketosis and toxic ingestion was negative. Gastrointestinal pathogen panel was negative. Computed tomography (CT) was negative for bowel thickening or enhancement, and CT angiography was negative for mesenteric thrombosis. Endoscopy did not reveal active CD. Lactic acid remained elevated despite intravenous fluids. The patient was noted consuming juice and several packets of sugar with tea, raising concern for D-LA. After restricting carbohydrates, lactic acid and AGMA rapidly normalized. A blood sample sent out from time of admission showed elevated D-lactic acid.

DISCUSSION:

This patient’s neurologic symptoms, AGMA and LA, simple carbohydrate intake and prior bowel resection raised concern for D-LA, a rare condition observed in patients with short bowel syndrome (SBS). Decreased intestinal absorption in SBS results in delivery of carbohydrates to the colon, where fermentation by colonic flora form L- and D-Lactic acid and other organic acids. Systemic absorption can cause AGMA, LA and neurologic symptoms, such as encephalopathy, dysarthria and ataxia. Diagnosis involves clinical suspicion, AGMA that resolves with carbohydrate restriction, and neurologic symptoms. D-lactate dehydrogenase assay confirms diagnosis. Treatment involves correction of acidosis and oral carbohydrate restriction. Enteric antibiotic use has been proposed to reduce acid-forming bacteria. Physicians should suspect D-LA in patients with CD post-bowel resection and otherwise unexplained AGMA, as this could prevent harm through inappropriate treatment including intravenous antibiotics and corticosteroids, radiation exposure, and invasive endoscopic evaluation.

Figure 1. Unenhanced cross section CT scan of the abdomen showing an ileocolonic fistula with aneurysmal dilatation of the small bowel.

Figure 2. Endoscopic picture showing the fistula at 20cm from the anal verge(left) and a small bowel fistulating mass(right).

Figure 3. H&E (left) and BCLC (right) stained histology slides showing Malignant high grade DLBCL cells infiltrating the small bowel.

S2783 Ileocolonic Fistula Due to Diffuse Large B-Cell Lymphoma: Unusual Presentation of a Rare Disease

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CASE DESCRIPTION/METHODS:

A 55-year-old male presented with 3 months of watery non-bloody diarrhea and 3 weeks of abdominal pain associated with nausea. He denied any vomiting, anorexia, weight loss, fever or chills. Labs revealed a CRP of 15.3 mg/dl and leukocytosis of 14.3 k/ul.

Follow up PET/CT scan showed no residual disease and was declared to be in complete remission.

DISCUSSION:

Auto-brewery syndrome can be difficult to diagnose given the episodic symptoms. Typical symptoms include dizziness, fatigue, somnolence and slurred speech. Diagnostic tests include alcohol and ammonia levels, fungal stool cultures as well as a carbohydrate challenge test, in which patients are observed eating a carbohydrate-heavy meal and monitored for symptoms. Antifungal therapy is a mainstay of treatment. Probiotic use has also proven effective, helping to prevent yeast overgrowth in the gut microbiome. Adherence to a low-carbohydrate diet also can alleviate symptoms by decreasing the ability of yeast to ferment ethanol. While gut-fermentation syndrome is a rare clinical disease, it can be an important differential in a patient with longstanding fatigue without a clear etiology.

S2784 Presidential Poster Award

Drunk on Life: Auto Brewery Syndrome

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INTRODUCTION:

Auto-brewery syndrome, otherwise known as gut-fermentation syndrome, is a rare condition where yeast overgrowth in the small intestine leads to ethanol fermentation in the gut, leading to alcohol intoxication in the absence of alcohol consumption.

CASE DESCRIPTION/METHODS:

A 67-year-old male with past medical history of gastro-esophageal reflux disease (GERD), irritable bowel disease diarrhea type (IBS-D) and hyperlipidemia presented to the office with a chief complaint of fatigue. For 4 to 5 months the patient’s wife noticed the patient would be sleeping more often than normal and was behaving “like he was drunk.” Both the patient and his wife stated that the patient had not been drinking alcohol on days he experienced symptoms. He also felt increased fatigue and required long naps, typically waking up with slurred speech. He was asymptomatic in the office and initial bloodwork included thyroid studies, lactic acid, prolactin, ammonia and ethanol levels were all normal. A hepatic function panel was ordered which revealed slightly elevated transaminases which were consistent with levels prior to the onset of symptoms. Patient also underwent MRI of the brain which was negative. Patient was told to have repeat blood work drawn when he was symptomatic. Repeat blood work revealed an elevated ammonia and alcohol level. On a repeat visit 2 weeks later he was started on probiotics as well as a low-carbohydrate diet and referred to his gastroenterologist for further workup. He was advised to obtain a breathalyzer and log alcohol levels after meals. Patient saw his gastroenterologist and declined any diagnostic workup. At his 4 month follow up patient stated he no longer had any symptoms.

DISCUSSION:

Auto-brewery syndrome can be difficult to diagnose given the episodic symptoms. Typical symptoms include dizziness, fatigue, somnolence and slurred speech. Diagnostic tests include alcohol and ammonia levels, fungal stool cultures as well as a carbohydrate challenge test, in which patients are observed eating a carbohydrate-heavy meal and monitored for symptoms. Antifungal therapy is a mainstay of treatment. Probiotic use has also proven effective, helping to prevent yeast overgrowth in the gut microbiome. Adherence to a low-carbohydrate diet also can alleviate symptoms by decreasing the ability of yeast to ferment ethanol. While gut-fermentation syndrome is a rare clinical disease, it can be an important differential in a patient with longstanding fatigue without a clear etiology.

S2785 Metastatic Mixed Adenoneuroendocrine Carcinoma Masquerading as Ileocecalis and Small Bowel Obstruction

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INTRODUCTION:

Mixed adenoneuroendocrine carcinomas (MANEC) are rare, accounting for 3-9% of colorectal cancers. They are comprised of at least 30% of both exocrine and neuroendocrine components. As disease is uncommon and clinical presentation is variable, masquerade by common differentials often leads to a delay in diagnosis and management.
CASE DESCRIPTION/METHODS: A healthy 62-year-old female presented with 2 months of nausea, vomiting, abdominal distension, and non-bloody diarrhea progressing to constipation. Initial computed tomography (CT) demonstrated ileocecitis, appendicitis, and partial small bowel obstruction (SBO). Vital signs were stable and labs unremarkable. Fecal calprotectin was 2292. Stool and blood cultures were negative. After discharge from an uneventful 2 days of conservative management for SBO, readmission occurred 4 days later for worsening symptoms. Repeat CT showed ileocecitis with high-grade SBO with transition point in the terminal ileum (TI). Conservative approach was attempted and request made for gastroenterology consult. Risk factors for adhesions, infectious, foodborne, and inflammatory diseases were denied. Abdomen was distended and tender, but vital signs and labs remained unremarkable. Enterography showed diffuse wall thickening and enhancement of the TI. Endoscopies were not pursued given SBO. As Crohn’s disease (CD) was the lead differential, intravenous steroids and pre-biologic workup were initiated; however, clinical and radiologic assessment on day 5 of IV steroids showed persistent SBO. Exploratory surgery was advised, revealing a 6.5 cm ileocecal mass with peritoneal implants. Right hemicolectomy with both nodal and implant biopsies were performed. Pathology revealed high grade MANEC, with extension into the visceral peritoneum and extensive lymphovascular invasion. Immunohistochemistry stain was positive for synaptophysin, with KI-67 proliferative index of 50-60%. Microsatellite instability was absent. Lymph nodes and peritoneal implants were consistent with metastases. Palliative chemotherapy was recommended.

DISCUSSION: Although immunohistochemical staining has allowed for greater detection of neuroendocrine (chromogranin, synaptophysin, and CD56) tumors, the diagnosis of MANECs remains rare, and grimly, as in our case, often occurs in advanced stage. Prognosis remains uncertain, but generally thought to be poor once local or distant metastasis is evident. Management also remains unclear, however if sought, treatment is guided at the more aggressive tumor component.