

normal except for diffuse abdominal fullness and tenderness to palpation without guarding. Liver and spleen were not palpable. Laboratory evaluation showed peripheral eosinophilia, which increased from 1,320 cells/ μ L (0-610) on admission to 12,770 cells/ μ L on day 7. She underwent upper and lower gastrointestinal endoscopies, which noted diffuse severe inflammation, characterized by erosions, erythema and friability within the stomach. Biopsies noted greater than 400 eosinophils/high-power field (HPF) and 80-120 mast cells/HPF in the gastric tissue, duodenum, terminal ileum, and colon. The mast cells were arranged in dense infiltrates and >25% were atypical or spindle-shaped. They expressed CD2 and CD25 in addition to CD117. Laboratory evaluation also revealed Trypsase and total serum immunoglobulin IgE elevated at 37 ng/mL (<11) and 11,021 kU/L ($\leq$ 114), respectively. Strongyloides serology and anti-neutrophil cytoplasmic antibodies were negative. Allergy and immunology was consulted and diagnosis of indolent systemic mastocytosis was made given significant mast cell infiltration of the GI tract and persistently elevated serum trypsin. Medication-induced eosinophilia was considered, but exenatide cessation did not resolve her eosinophilia or symptoms. She was discharged on Loratadine, Famotidine, Montelukast, Pantoprazole, and Cromolyn with subsequent improvement in her gastrointestinal symptoms. Systemic Mastocytosis with Predominately Gastrointestinal Manifestations

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Auto-Brewery Syndrome in a Diabetic Man

ACG Case Reports Journal Award

Presidential Poster Award

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Introduction: Auto-Brewery Syndrome (ABS), also known as Gut Fermentation Syndrome, is a rare medical condition in which ethanol is endogenously fermented via fungal overgrowth in the gut. The causative organism for our patient is *Saccharomyces cerevisiae*. Both Ethanol and Metformin can lead to lactic acidosis. Conventional methods to treating ABS are not always successful, especially if exacerbated by comorbidities, therefore other empiric methods have been utilized. **Case:** A 46-year-old male was eating dinner when he developed symptoms of progressively worsening vomiting, diarrhea, and slurring of speech. In the ED, he was given a diagnosis of acute pancreatitis secondary to alcohol intoxication, even though the patient had been abstinent of alcohol for 20-years. He has a PMH of diabetes and is compliant with his Metformin. The patient had undergone surgical and dental procedures in simultaneous weeks, and was prescribed antibiotics for both. Fungal stool cultures grew *Saccharomyces cerevisiae*. He was diagnosed with Auto-Brewery Syndrome (ABS), and was given a 21-day regimen of Fluconazole and probiotics, consistent with conventional approaches to treating ABS. The patient returned 2 weeks later appearing toxic, with elevated lactate and ethyl alcohol levels. He was admitted to the ICU for continuous monitoring, and the Infectious Disease team decided on an empiric approach of IV Micofungin. This treatment option had no supporting data due to the rarity of his condition alongside with his ongoing diabetes. The patient's lactic acidosis resolved in the following days, as well as his ethyl alcohol levels stabilized, and the Micofungin successfully decreased fungal overgrowth. **Discussion:** In patients with diabetes, one of the main-stem goals is avoidance of lactic acidosis (Non-ketotic diabetic acidosis). Lactic acidosis can be fatal and originates through inhibition of the Pyruvate Dehydrogenase Complex (PDH), a primary means of generating energy from glucose. Ethanol is an inhibitor of PDH. Biguanides such as Metformin have been shown to inhibit PDH as well. Conventional methods failed at treating this patient, and an empiric method by the Infectious Disease team proved successful. We cannot rule out that his ABS could have been exacerbated by his diabetic medication. We know fermentation and Metformin utilize similar pathways, and this alone could lead to break through studies in treatment options of ABS in patients with known comorbidities.

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Rumination, Eosinophilic Esophagitis and a Parasitic Infestation: A Diagnostic Conundrum

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Rumination syndrome is an underappreciated condition. It is a behavioral condition characterized by postprandial regurgitation which can often be mistaken for gastroesophageal reflux disease or vomiting. A 11 year male with a past medical history of pecan and walnut allergy presented with a 4 week history of progressive symptoms of nausea, vomiting and non bloody diarrhea. The onset of symptoms occurred following a trip to India. His symptoms sequentially progressed from loose stools, abdominal pain, vomiting to bloating and belching followed by regurgitation and solid & liquid dysphagia. He noted a varied diet which included consumption of sushi with raw sea food. His physical exam was normal. Labs showed a normal WBC count with eosinophilia of 15% and normal stool studies. Abdominal imaging revealed a markedly distended stomach which persisted on multiple days. He underwent an EGD which revealed edema, exudates and rings seen throughout the esophagus with an EREF score of 3. A flexible sigmoidoscopy with biopsies was normal. Biopsies showed pronounced eosinophilia in the esophagus (distal, mid and proximal at 60, 50 and 45 eos/hpf respectively) and 80 eos/hpf in the antrum. Despite twice daily therapy with a proton pump inhibitor and Budesonide slurry he progressed to severe oral intolerance with weight loss requiring inpatient admission. An upper GI series was normal. He was started on oral steroids and under a post pyloric Dobhoff tube placement for enteral feeds after continued emesis with gastric feeds. Work up was negative for Toxocara. Ig E was normal. He underwent repeat EGD given persistent symptoms which now showed resolved eosinophilia. An empiric trial of Albenadazole was given for possible parasitic infection. Given symptoms of painless regurgitation within 15 minutes of ingestion, he underwent high resolution esophageal motility testing which revealed supragastric belch associated rumination syndrome. His symptoms resolved with diaphragmatic breathing. This case highlights the importance of considering a parasitic infection in patients with a history of travel, acute onset of gastrointestinal symptoms and eosinophilia. Diagnosis was further delayed with occurrence of secondary rumination.

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Co-Infection of Blastocystis and Endolimax Nana and Their Association with Functional Disorders and Bowel Inflammation

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Blastocystis species is a parasite found in the colon and cecum with clinical significance of gastrointestinal infections presenting with diarrhea and non-specific findings of nausea and vomiting. *Endolimax nana* resides in the colon but can also be found in the appendix. There is limited evidence regarding pathogenicity for these protozoan. Transmission is proposed to be fecal-oral. We report a case of a 32-year-old Hispanic male presenting to clinic complaining of intermittent, diffuse abdominal pain, diarrhea for 10 months. The abdominal pain was sharp, aggravated with food, not relieved by any factors. Diarrhea was non-bloody occurring 3-4 times a day. He denied any fevers, chills, unintentional weight loss, nausea, vomiting, constipation, melena, hematochezia, recent travel, or antibiotic use. He worked as a waiter and dietary habit was mainly takeout food. He had no past medical or surgical history and family history was non-contributory. He denied any smoking, drug use but endorsed occasional alcohol use. Physical exam revealed a well-nourished man with dry mucous membranes. Abdominal exam showed mild, diffuse abdominal tenderness with no guarding or rebound tenderness. Laboratory tests displayed normal complete blood count, comprehensive metabolic panel, thyroid stimulating hormone. Erythrocyte sedimentation rate, C-reactive protein, HIV antibody, tissue transglutaminase IgA antibody were found to be negative. Fecal osmolar gap was calculated to be 10 (<50) consistent with secretory diarrhea. Stool studies for ova and parasite were positive for *Blastocystis* species and *Endolimax nana* cysts seen on wet prep and trichrome smear. Patient was started on Metronidazole 500mg 3 times a day for 10 days. Re-evaluation of symptoms at a follow up visit showed significant improvement. Laboratory tests with colonoscopy showed no significant abnormality. Due to incomplete resolution of symptoms patient was diagnosed with irritable bowel syndrome with diarrhea predominance. Studies indicate co-infection of the parasites and evidence of involvement in immune system stimulation and association with inflammatory bowel disease (IBD) and irritable bowel syndrome (IBS). Our case demonstrates the necessity for further investigation to fully recognize pathogenic and non-pathogenic components of these species. Also to raise awareness in diagnosing chronic diarrhea related to protozoan and perhaps incorporate stool studies for the mentioned species in patients with IBD and IBS.

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Premature Ticagrelor Discontinuation Secondary to Severe Diarrhea: A Case Report

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Dyspnea and easy bruising are the most commonly reported Ticagrelor side effects while gastrointestinal (GI) bleeding is one of the most worrisome complications. Here we present, to the best of our knowledge, the first reported case of severe diarrhea attributed to Ticagrelor that necessitated premature discontinuation of the medication. A 67 year old male presented to the clinic for follow up for celiac disease and microscopic colitis that were diagnosed 5 years prior.

The patient was doing well until about 10 months prior to this presentation when he started complaining of 6-7 large daily loose bowel movements. His symptoms started shortly following an admission for acute coronary syndrome requiring percutaneous coronary artery intervention (PCI). The patient denied dark stools, bright red blood per rectum or abdominal pain. He denied any upper GI symptoms. He adheres to a strict gluten-free diet. Last upper and lower endoscopies were 5 years prior to presentation both of which were normal examinations. Biopsies were consistent with celiac disease and microscopic (lymphocytic) colitis respectively.

The patient was started on aspirin and ticagrelor after his coronary artery stent placement 10 months prior. Otherwise he was on lisinopril and atorvastatin.

Physical examination was unremarkable. Laboratory work up including a complete blood count and comprehensive metabolic panel were unremarkable. Further testing revealed a negative Clostridium difficile polymerase chain reaction (PCR), stool studies including Giardia, and IgA tissue transglutaminase antibodies. The patient continued to have diarrhea despite trials of Budesonide, cholestyramine, atropine/diphenoxylate and bismuth. This was followed by a course of Rifaximin for possible small intestinal bacterial overgrowth which did not yield any changes in symptoms.

Given the correlation between the initiation of the suspected offending agent and the onset of symptoms, ticagrelor was switched to clopidogrel. The patient's diarrhea completely resolved within a week with no complaints on follow up after 1 month. Our review of literature did not find any reported cases of diarrhea associated with ticagrelor use with a very limited number of studies, however, those studies do not comment on the severity of symptoms or on any reported discontinuation of the drug. A thorough medical history can guide clinicians towards possible underlying etiologies and appropriate work up for chronic diarrhea.

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Recurrent Pancreatitis Due to Celiac Disease

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Celiac Disease (CD) is a small bowel disorder characterized by mucosal inflammation, villous atrophy, and crypt hyperplasia that occurs upon exposure to dietary gluten. Symptoms improve upon withdrawal of gluten from the diet. CD has other implications such as its association with pancreatico-biliary disease. Postulated mechanisms include reduced gallbladder emptying due to impaired cholecystokinin release