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A Rare Case of Nsaid Induced Gastric Body Diaphragmatic-Like Stricture

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ABSTRACT

Protein-losing gastroenteropathy is a rare syndrome of protein loss from the gastrointestinal system. It manifests with hypoproteinemic edema, which may be due to either lymphatic leakage due to increased interstitial pressure or leakage of protein-rich fluids due to intestinal disorders. Our case describes a 65-year-old female with life-threatening protein-losing enteropathy (PLE) requiring multiple transfers to intensive care unit for vasopressor support. In this rare instance, her extensive initial workup did not reveal any etiology for PLE, but she was later found to have underlying Crohn's colitis. Protein-losing enteropathy is an underdiagnosed complication of inflammatory bowel disease and must be considered while treating patients with colitis.

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Introduction

Protein-losing gastroenteropathy is disproportionately excessive loss of serum proteins into the gastrointestinal tract. Intestinal leakage of serum proteins can occur by intestinal loss of lymphatic fluid, increased mucosal permeability, or inflammatory exudation. Serum proteins like albumin, IgA, IgG, and IgM that have a slow catabolic rate are reduced more significantly in comparison to proteins with a rapid turnover rate [1]. Clinical manifestations of protein-losing enteropathy depend primarily on the underlying disease. Patients typically present with peripheral edema, diarrhea, dyspnea due to pleural/pericardial effusion, and abdominal distention due to ascites.

Case Presentation

A 65-year-old female with a past medical history of C. difficile infections, and ulcerative colitis (UC) in remission on vedolizumab and oral mesalamine presented to the hospital complaining of protracted diarrhea, generalized lethargy, and diffuse total body edema. Work up in the emergency room revealed significant hypoalbuminemia (albumin level of 2), moderate hyponatremia and right popliteal deep vein thrombosis (DVT). Physical examination was remarkable for anasarca and hypotension. The patient was initially started on apixaban for DVT. However, anticoagulation was stopped after multiple gastrointestinal bleeding episodes, requiring transfusion. The patient underwent extensive workup with endoscopy, colonoscopy and MRI enterography which only revealed featureless colon (Figure 1a – coronal section, 1b – axial section) and chronic inactive colitis, and no source of bleeding was found.

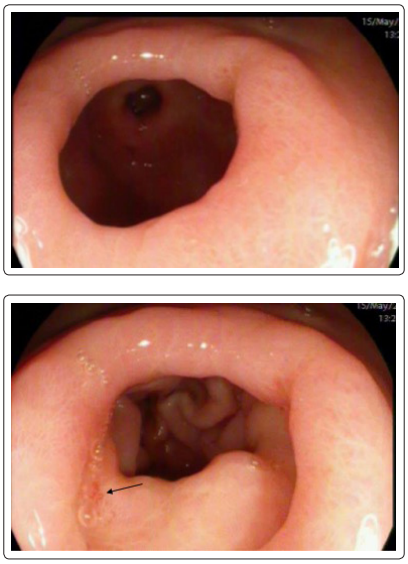

Figure 1a, 1b: Featureless colon (arrow mark in coronal section) without wall thickening or evidence of inflammation, consistent with known diagnosis of ulcerative colitis. There is no MR evidence of active disease

The patient continued to have a high output of persistent diarrhea. Infectious workup for C. difficile, extended fecal pathogen panel by PCR, and cytomegalovirus were negative. The patient also had a normal fecal osmotic gap of 108, and a normal anti-tissue transglutaminase IgA Ab and gliadin IgG Ab. However, the patient had a significantly elevated fecal calprotectin level of 1800 from an unknown etiology. The patient's albumin and pre-albumin levels

dropped drastically to 1.3 and 8, respectively. Multiple urinalyses showed none or mild proteinuria, and the liver appeared normal on abdominal ultrasound and CT scan of the abdomen. Further workup for hypoalbuminemia revealed a significantly elevated stool alpha-1-antitrypsin level of 125, leading to the diagnosis of protein-losing gastroenteropathy. Her diarrhea did not improve despite trials of multiple medications, including prednisone, mesalamine, psyllium, bismuth subsalicylate, rifaximin, and loperamide. The patient required multiple transfers to the intensive care unit for hypovolemic shock requiring vasopressors. Considering unclear etiology for protein-losing gastroenteropathy and ongoing high output diarrhea, the patient underwent a repeat MRI enterography, which now revealed distal ileum wall thickening (Figure 2a – coronal section, 2b – axial section) with asymmetric mucosal enhancement and adjacent mesenteric vascular engorgement concerning for ileitis. Her diagnosis of UC was changed to Crohn's colitis.

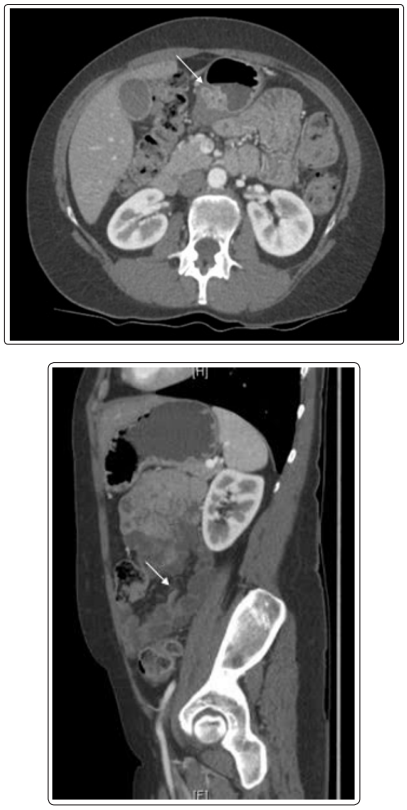

Figure 2a, 2b: There is distal ileum wall thickening (arrow mark in coronal and axial section) with asymmetric mucosal enhancement and adjacent mesentery vascular engorgement concerning for underlying Crohn's disease. The terminal ileum is unremarkable and not involved by disease

The patient was started on high dose intravenous methylprednisolone, vedolizumab, daily albumin infusions, and high protein diet which resulted in improvement of diarrhea, hypoalbuminemia, and hypotension. Vitamin B12 and copper deficiencies were replaced adequately as well. The patient had a protracted and complicated hospital stay of more than three months due to IBD induced protein-losing gastroenteropathy, resulting in substantial albumin loss, anasarca, and hypovolemic shock. Once diarrhea improved, the patient was switched to oral prednisone with a plan to taper slowly. On discharge to a skilled nursing facility, mesalamine was discontinued, and the patient was maintained on loperamide and bumetanide. The patient has continued to follow up with her gastroenterologist and receives vedolizumab every eight weeks. As patients with albumin loss are at high risk of thrombosis, apixaban was resumed at a low dose without any reoccurrence of bleeding episodes.

Discussion

We describe a rare case of life-threatening protein-losing gastroenteropathy secondary to Crohn's colitis leading to critically low albumin levels, anasarca, intravascular volume depletion, end-organ damage, vitamin and mineral deficiencies, and hypovolemic shock. Mild protein-losing gastroenteropathy is a relatively common condition that occurs in multiple gastrointestinal and non-gastrointestinal diseases, yet it is underdiagnosed as it is mostly overlooked.

PLE is caused by leakage of intestinal proteins due to either intestinal loss of lymphatic fluid, increased mucosal permeability, or inflammatory exudation [2]. Intestinal loss of lymphatic fluid can be caused by impaired lymph drainage due to primary intestinal lymphatic disorders or secondary causes [3]. Increased mucosal permeability may be the result of several non-erosive gastrointestinal diseases like celiac disease, rheumatic diseases, and amyloidosis [4]. Inflammatory exudation from mucosal erosions and ulcerations can occur in erosive gastrointestinal disorders like inflammatory bowel disease (IBD), gastrointestinal malignancies, and colitis due to Clostridioides difficile infection (CDI) [5].

Protein-losing gastroenteropathy is diagnosed by increased stool clearance of alpha-1 antitrypsin [6]. Alpha-1 antitrypsin is synthesized in the liver and is excreted in the stool. An elevated alpha-1 antitrypsin clearance is diagnostic of PLE. Patients with hypoalbuminemia and anasarca who do not have any evidence of protein malnutrition, underlying liver disease, or proteinuria should be evaluated for protein-losing gastroenteropathy. Protein-losing gastroenteropathy has been reported to occur in more than 60 different conditions. It is important to investigate the underlying etiology responsible for gastrointestinal protein loss. Our patient had excessive enteral protein loss secondary to IBD, which surprisingly was not evident on either colonoscopy or initial imaging. Patients with IBD, especially Crohn's colitis, experience enteral protein loss due to focal surface erosions and ulcerations. Sometimes in early mild disease, imaging might be non-revelatory; however, capsule endoscopy may reveal serous fluid leakage from intestinal erosions [7].

Management of protein-losing gastroenteropathy consists of treatment of the underlying disease and low-fat, high protein, medium-chain triglycerides diet [8]. Patients should also be tested for vitamin and mineral deficiencies and replenished if deficient.

Technique for MRI Enterography W/WO IV Contrast

MRI, multiplanar, T1 and T2 weighted images of the abdomen and pelvis were performed following neutral oral contrast, administered orally per MR enterography protocol. T1-weighted, fat-saturated images were obtained before and after intravenous contrast administration.

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