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CASE REPORT | COLON

Eosinophilic Enterocolitis: Gastric Outlet Obstruction

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ABSTRACT

Eosinophilic enterocolitis is a rare condition representing the least frequent manifestation of eosinophilic gastrointestinal disorders. We report a 49-year-old man who presented with abdominal pain, diarrhea, and intractable vomiting for 2 weeks. Abdominal computed tomography demonstrated gastric wall thickening with mural wall thickening of the small intestine and colon. Endoscopy revealed gastric outlet obstruction because of luminal stenosis from duodenal edema. Peripheral eosinophilia, endoscopic mucosal biopsies, and exclusion of differential diagnoses confirmed eosinophilic enterocolitis. The patient was successfully treated with a 4-week prednisone taper.

INTRODUCTION

Eosinophilic gastrointestinal disorders (EGIDs) are a spectrum of diseases that include eosinophilic esophagitis, eosinophilic gastritis, eosinophilic gastroenteritis, eosinophilic enteritis, and eosinophilic colitis. Eosinophilic enterocolitis (EEC) represents the least frequent subset of this condition. There are no data on the prevalence of EEC because of rarity, but the incidence of EGID is estimated to be 18 per 100,000 persons.[1] The etiology and pathogenesis are not well understood but are likely from a multifaceted interaction between immunological, environmental, and genetic factors.[2] Approximately 75% of patients have a history of allergy or atopy.[3] The Klein classification subdivides the disease based on the extent of eosinophilic infiltration within the bowel wall: mucosa, muscularis, and subserosa. The clinical manifestations are predominantly based on the affected layers. Diagnosis is defined by 3 criteria: (i) the presence of gastrointestinal symptoms, (ii) biopsies showing eosinophilic infiltration of one or more areas of the gastrointestinal tract, and (iii) exclusion of secondary causes of eosinophilic infiltration such as parasites, medications, inflammatory bowel disease, malignancy, autoimmune disease, and hypereosinophilic syndrome.[4] Treatment typically includes systemic corticosteroid therapy, which suppresses cytokine gene transcription and local inflammation.[5] Case studies have shown sodium cromoglycate and leukotriene inhibitors to be effective with possible role for targeted immunotherapeutic treatments in the future.[6] The duration of treatment remains controversial. However, a long-term follow-up is required because recurrent symptoms commonly develop.[7]

CASE REPORT

A 49-year-old Hispanic man with no medical history of atopy or defined food sensitivities presented with abdominal pain, diarrhea, and intractable vomiting for 2 weeks. He was admitted 8 months earlier with similar symptoms and an eosinophil level of 2,500 eosinophils/uL. Upper endoscopic workup then suggested diffuse erythematous gastropathy and duodenal erythema, edema, and friability. Gastric and duodenal biopsies showed moderate chronic inflammation with positive *Helicobacter pylori* immunohistochemical stain and mild chronic inflammation, respectively. However, there was preserved villous architecture with no evidence of increased intraepithelial lymphocytes or eosinophils. The patient was treated with clarithromycin, metronidazole, and pantoprazole without improvement.

On current admission, the patient was hemodynamically stable. The examination was significant for diffuse abdominal tenderness. Laboratory findings included leukocytosis (white blood cells 17.7 $\times$ 1,000/mm^3), peripheral eosinophilia (8,400 eosinophils/uL), and elevated hepatic chemistries (aspartate aminotransferase 43 unit/L, alanine aminotransferase 77 unit/L, alkaline phosphatase 307 unit/L, and total bilirubin 3.5 mg/dL). Liver ultrasound showed gallbladder wall thickening with nondilated bile ducts. Abdominal and pelvic computed tomography with contrast demonstrated moderate gastric wall thickening with dilatation and marked mural thickening of the duodenum, proximal jejunum, and descending colon (Figures 1 and 2). Repeat upper endoscopy revealed

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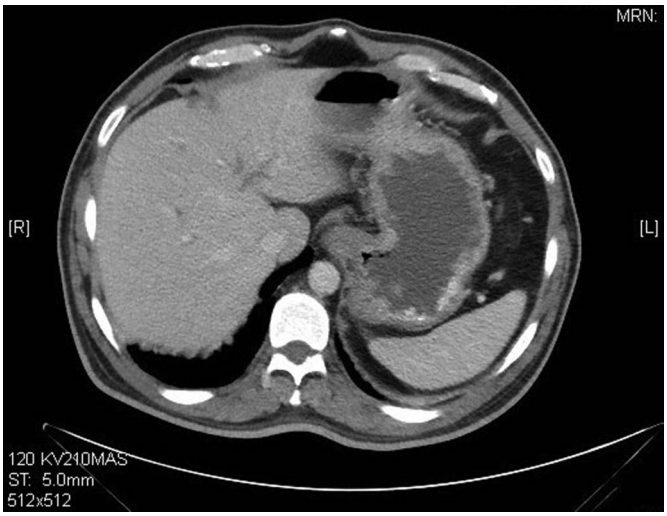

Figure 1. Axial abdominal computed tomography with contrast shows moderately distended stomach with moderate thickening of the wall of the stomach concerning gastric outlet obstruction.

severe duodenal edema and erythema with significant luminal stenosis resulting in a gastric outlet obstruction (Figure 3). Colonoscopy showed pancolonic edema with areas of hyperemia and edematous hemorrhagic folds without ulceration. Biopsies of duodenal mucosa showed normal villous architecture and extensive inflammation with increased eosinophils (>50 per high power field [HPF]) and colonic mucosa with areas of markedly increased eosinophils in the lamina propria, intraepithelial spaces, and muscularis mucosa (Figure 4). In addition, the endoscopic biopsies showed a resolution of *H. pylori*. Parasitic, fungal, and bacterial pathogens were ruled out. The patient was not on new medications and had no new dietary exposure since previous endoscopy. The autoimmune panel was negative for vasculitis biomarkers. Based on clinical history, laboratory, radiological, and endoscopic findings, EEC was diagnosed. Initiation of intravenous methylprednisolone 40 mg twice daily led to an improvement of symptoms within 24 hours. Given the lack of biliary ductal dilation, biliary stenting was not performed. The cholestatic liver chemistries were believed to be likely from the eosinophilic inflammatory process. The patient was discharged home with a 4-week prednisone taper and followed up 2 weeks later with complete resolution of symptoms, normalization of liver chemistries, and reduced peripheral eosinophilia (600 eosinophils/uL).

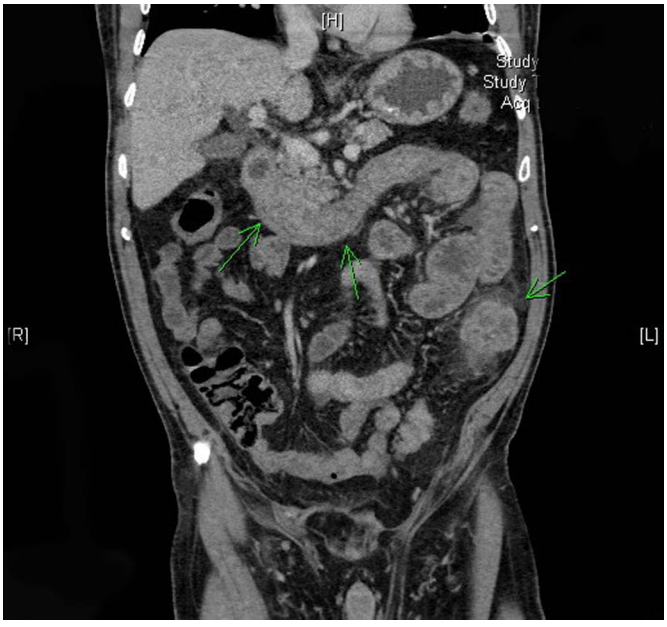

Figure 2. Coronal abdominal computed tomography with contrast shows moderate thickening of the small bowel and marked thickening of the duodenum and proximal jejunum.

DISCUSSION

EGIDs, a rare inflammatory condition, are established by functional gastrointestinal symptoms, peripheral eosinophilia, and segmental eosinophilic infiltration of the gastrointestinal tract, all of which were present in our patient on admission.[8] Although manifestations occur based on the affected gastrointestinal layer, abdominal pain is the most prevalent presenting symptom.[9] Involvement of the mucosal layer can cause diarrhea and vomiting, and involvement of the muscular layer can cause wall thickening leading to obstruction, as seen in our patient. From the initial presentation, computed tomography imaging has shown to be useful in revealing intestinal wall thickening, detecting the presence of ascites, evaluating for inflammatory and neoplastic lesions, and ruling out extraintestinal pathologies.[10,11] The preferred diagnostic modality is endoscopic biopsy. Endoscopic findings in EEC are variable and nonspecific including mucosal erythema, nodular appearance, edema, and loss of normal vascular pattern.[1] There is no diagnostic cutoff for the number of eosinophils per HPF on histologic examination. However, an absolute count of at least 20 eosinophils/HPF has been noted in most reports.[12] It is imperative to exclude other possible causes of eosinophilic infiltration, which was ruled out in our patient.

Regarding treatment, there have been no randomized controlled trials in the literature to describe the utility of different therapeutic options. The mainstay treatment remains prednisone by inducing eosinophil apoptosis and inhibiting chemotaxis.[13] Successful transition from oral, conventional glucocorticoids to budesonide (nonenterically coated) has been reported in patients involving the gastric antrum and small intestine. However, this is usually used for recurrent disease.[14] There have been some studies that show that the patients are placed on an elemental formula, which empirically eliminates potential food allergens, including soy, wheat, egg, milk, peanut/tree nuts, and fish/shellfish. However, the main limitation with this method is patient compliance.[15] Other treatment approaches, described in small case series, include leukotriene inhibitors, mast cell stabilizers, and biologic agents.[6] Further research and studies are needed to

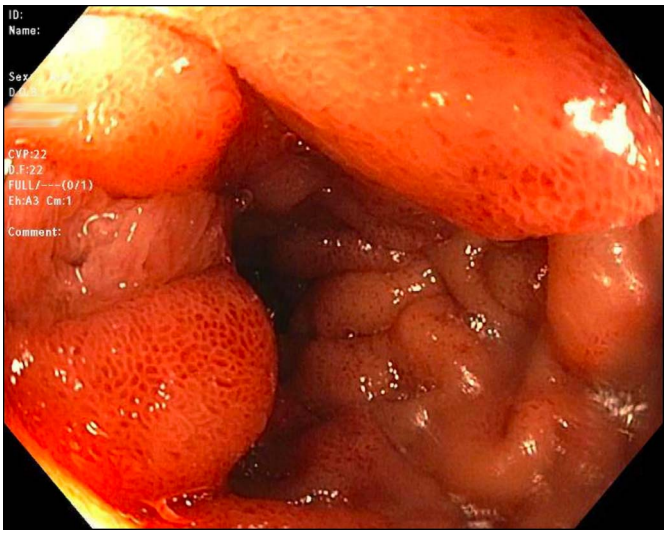

Figure 3. Endoscopic image of the duodenal edema and erythema with luminal stenosis.

determine the long-term effectiveness of the different treatment options. The natural course of the disease is also largely unknown, and many patients have relapse and recurrent episodes; therefore, close follow-up with these patients is crucial.[7]

Our clinical case report highlights a rare entity with a unique presentation. There have been a couple of cases of eosinophilic gastroenteritis causing gastric outlet obstruction because of primary eosinophilic infiltration in the antrum and/or associated gastritis with polypoid lesions; however, this is the first case of extensive intestinal edema from EEC resulting in gastric outlet obstruction. Although the elevated liver chemistries are presumed to be eosinophilic hepatitis, this was not proven via biopsy. Diagnosis of EEC should be considered in patients presenting with gastrointestinal symptoms, including gastric outlet obstruction and cholestatic liver pattern, with peripheral eosinophilia.

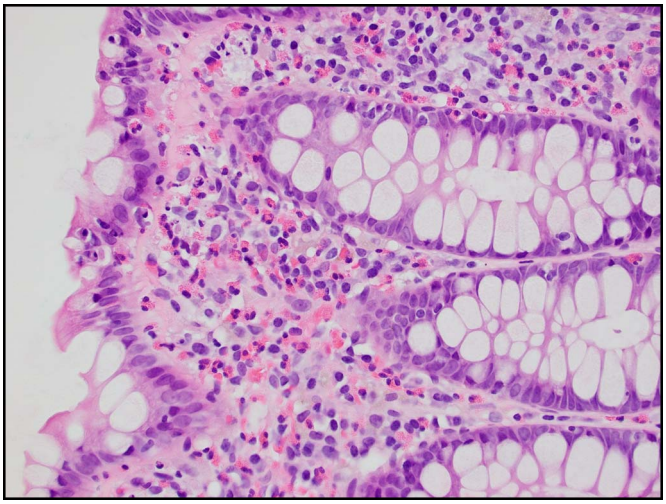

Figure 4. Biopsy of the left colon showing areas of markedly increased eosinophilic infiltration (400× magnification).

DISCLOSURES

Author contributions: Both authors contributed equally to this manuscript. I. Elkhatib is the article guarantor.

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