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CASE REPORT | STOMACH

Eosinophilic Gastroenteritis Causing Pyloric Stenosis: A Rare Manifestation

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ABSTRACT

Eosinophilic gastritis is a gastrointestinal disorder characterized by eosinophilic infiltration in the gastric wall. We present a rare case of critical pyloric stenosis secondary to eosinophilic gastritis in a 16-year-old adolescent girl who presented with nausea, vomiting, early satiety, and abdominal pain. Abdominal computed tomography and subsequent esophagogastroduodenoscopy confirmed the anatomical diagnosis, but histological confirmation of the eosinophilic etiology was challenging. After an unsuccessful trial of high-dose systemic corticosteroids, a laparoscopic gastrojejunostomy was performed and long-term immunosuppression with mycophenolate mofetil was commenced.

KEYWORDS: eosinophilic esophagitis; eosinophilic gastritis; eosinophilic gastroenteritis; pyloric stenosis; gastric outlet obstruction

INTRODUCTION

Eosinophilic gastritis (EoG) is an inflammatory, immune-related condition of the gastric mucosa driven by hypereosinophilia. It is a product of IgE-mediated and cell-mediated mechanisms using the T-helper-2 cell response.[1] EoG is strongly associated with a history of atopy. An enhanced understanding and recognition of the disease has led to an increased incidence, with female and White individuals more commonly affected.[2] While abdominal pain, nausea, vomiting, and diarrhea are typical features of mucosal involvement, deeper eosinophilic penetration may result in more complex presentations.[3] Involvement of the muscular layer may present as a gastric outlet obstruction while deeper penetration to the serosal layer may manifest with ascites or even perforation.[1] Mucosal biopsies are not always diagnostic, and surgically assisted full-thickness biopsies may be necessary.[1] Multiple biopsies from various gastric locations increase the diagnostic yield.[3]

This report illustrates a case of EoG presenting with critical pyloric stenosis due to severe antral fibrosis.

CASE REPORT

A previously well, 16-year-old White adolescent girl presented to the emergency department on multiple occasions with 5 weeks of nausea, vomiting, early satiety, and epigastric pain. She had no medical history, medications, smoking or alcohol use, no personal or family history of atopy or food sensitivities, and minimal travel history. Her full blood count, urea and electrolytes, liver function, and C-reactive protein were unremarkable. Given multiple presentations to the emergency department, her general practitioner ordered an outpatient ultrasound, which showed dilation of the stomach. A subsequent abdominal computed tomographic scan revealed a distended, fluid-filled stomach with thickened wall and tapering at the antrum and pylorus (Figure 1). An esophagogastroduodenoscopy (EGD) on day 1 of her admission revealed a fluid-filled stomach, severe erosive gastritis most pronounced in the antrum, and pyloric stenosis traversable only with a 5.4 mm gastroscope (Figure 2). The esophagus and duodenum were normal macroscopically. Eighteen biopsies from the proximal esophagus to the third part of the duodenum including the stenosis revealed only mild chronic gastritis, without *Helicobacter pylori*, lymphoid proliferation, dysplasia, or malignancy. Flow cytometry of the gastric antrum and body biopsies revealed normal T and B-cell markers. High-dose proton pump inhibition was commenced. A nasogastric tube was not tolerated physically or emotionally, so total parenteral nutrition was administered. Pediatric and surgical opinions were attained.

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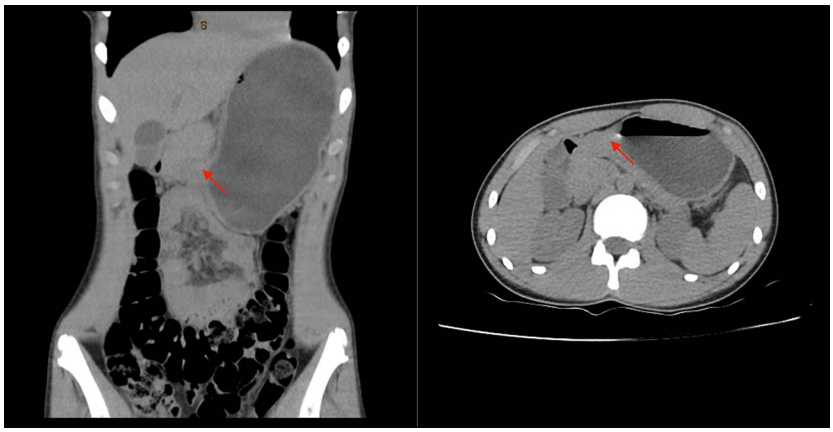

Figure 1. Axial CT showing fluid-filled stomach with the thickened wall and tapering at the antrum and pylorus (red arrow) with the corresponding coronal view. CT, computed tomography.

Her gastrin level, immunoglobulins (including IgG subclasses), anti-neutrophil cytoplasm antibodies/anti-saccaromyces cerevisiae antibodies, human immunodeficiency virus, and interferon gamma release assays were normal. An EGD on day 10 revealed persistent, erosive gastritis with ulceration and progression of the pyloric stenosis that precluded passage with the 5.4 mm gastroscope (Figure 2). Deeper tunneled mucosal biopsies (repeatedly from one biopsy site) obtained from the gastric antrum, body, and incisura this time showed active inflammation with focal intraepithelial neutrophils, lymphocytes, and more than 50 eosinophils per high-power field (HPF) in at least 5 HPFs across several specimens (Figures 3 and 4). Again, there was no Helicobacter pylori, granulomas, vasculitis, dysplasia, or malignancy. A colonoscopy of the terminal ileum was normal macroscopically and microscopically.

She received a daily dose of 100 mg IV hydrocortisone for 5 days before a daily dose of 1,000 mg IV methylprednisolone for 3 further days. An EGD on day 24 revealed a significant improvement in the inflammatory component, but persistent fibrotic antral changes not traversable with the 5.4 mm gastroscope (Figure 2). The patient declined sequential pyloric dilatation and favored surgical diversion. A laparoscopic gastrojejunostomy was performed on day 32, confirming a stenosis that encompassed both the pylorus and antrum. Diet was

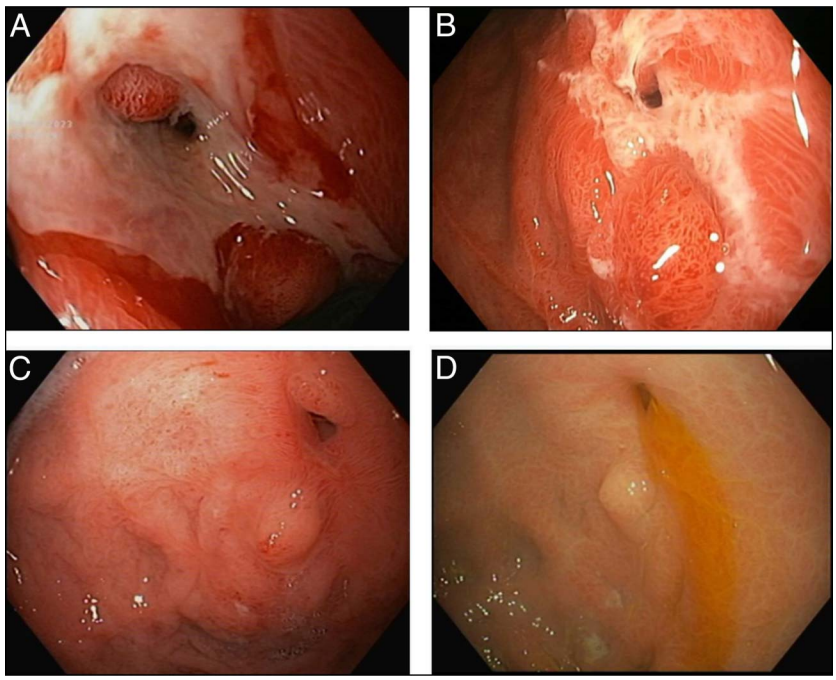

Figure 2. (A) Severe erosive gastritis most pronounced in the antrum and a pyloric stenosis traversable only with a 5.4 mm diameter gastroscope. (B) Severe erosive gastritis with ulceration and progression of the pyloric stenosis that now precluded passage with the 5.4 mm gastroscope. (C) Third EGD on day 24 revealed a significant improvement in the inflammatory component, but persistent fibrotic antral changes. (D) Repeat EGD 4 weeks later revealed a patent gastrojejunostomy with a noninflamed but fibrous stenosis. EGD, esophagogastroduodenoscopy.

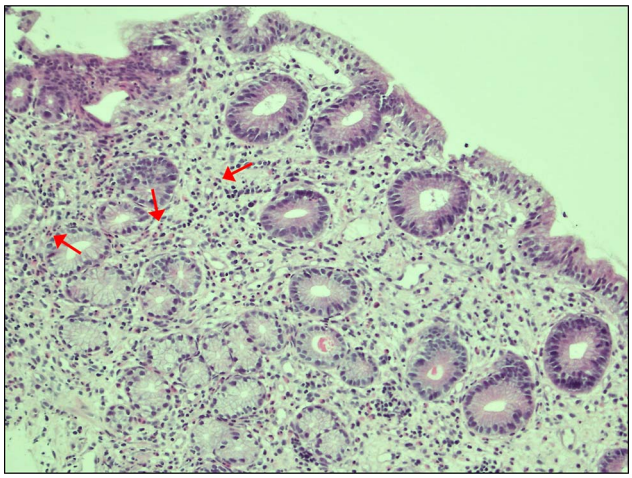

Figure 3. Histological image showing prominent eosinophil infiltration into the lamina propria of gastric mucosa. Eosinophil count is up to 50 per HPF, in greater than 5 HPFs. Focal intraepithelial eosinophilic infiltrates are seen. Eosinophils can be identified by orange granules and bilobed nuclei (red arrows). HPF, high-power field.

upgraded and total parenteral nutrition weaned. After a 4-week steroid-free postoperative period, 15 mg of oral prednisolone was commenced. A repeat EGD 4 weeks later revealed a patent gastrojejunostomy and noninflamed fibrous stenosis in the antrum (Figure 2). Biopsies obtained from the proximal esophagus to the small bowel by using the anastomosis did not reveal any eosinophilic infiltrates. Maintenance mycophenolate mofetil was introduced with oversight from a pediatric immunologist, and prednisolone was tapered. A follow-up gastroscopy 6 months later revealed antral scarring, traversable with a 5.4 mm gastroscope. There are no plans for future dilatations or surgeries.

DISCUSSION

Obtaining a histological diagnosis of EoG can be challenging. The number of eosinophils per HPF necessary for diagnosis varies depending on the biopsy location and is not formally defined.[3,4] It has been suggested that at least 30 eosinophils per HPF on a biopsy sample from the stomach with the exclusion of other causes of hypereosinophilia and presence of characteristic symptoms constitutes a diagnosis of EoG.[4] The HPF count of eosinophils coupled with the absence of response to proton pump inhibition in our patient renders a diagnosis of erosive gastritis unlikely.

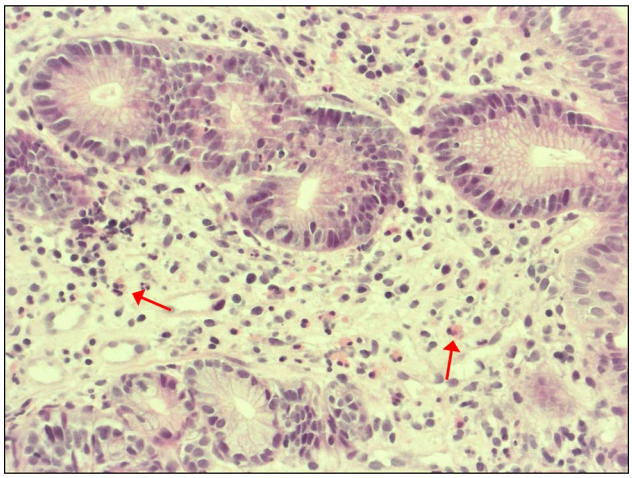

Figure 4. Histological image showing prominent eosinophil infiltration (red arrows) into the lining epithelium. Eosinophil count is up to 50 per HPF, in greater than 5 HPFs. HPF, high-power field.

Pyloric stenosis in EoG leading to gastric outlet obstruction is limited to several case studies within the literature.[5–7] Corticosteroids were used as first-line treatment in all cases and are considered the mainstay for induction of remission.[5–7] In established mucosal fibrosis, however, corticosteroid treatment may be inadequate. While elimination diets are a conservative alternative, evidence for the same is lacking. Adjunctive pharmacological treatments to corticosteroids include leukotriene receptor antagonists or mast-cell stabilizers while several monoclonal antibodies targeting interleukin-5 and interleukin-4/13 look promising for future use. In severe cases with complications such as gastric outlet obstruction or perforation, surgery may be necessary.[1] Endoscopic dilation is possible, but recurrence is common.[8] Given the sparse literature of pyloric stenosis management in EoG, treatment options for adult idiopathic hypertrophic pyloric stenosis have been used as surrogate. They include partial gastrectomy, gastroenterostomy, pyloromyotomy, and pyloroplasty.[9,10] In one pediatric case report, a distal gastrectomy with gastroduodenal anastomosis relieved the gastric outlet obstruction.[11] Three adult case reports used various types of gastrectomies.[12–14] In our case, a laparoscopic gastrojejunostomy was performed successfully and with potential for reversal subsequent to further immunosuppression. While there are case reports of EoG recurrence after treatment with systemic corticosteroids alone, the rate of recurrence at anastomotic sites after the aforementioned surgeries is unknown.[1,11–14]

Multidisciplinary care is essential for patients with EoG-associated pyloric stenosis given its variable and potentially complicated disease course.[1] Awareness of this rare manifestation and its diagnostic challenges will aid in a timely diagnosis, potentially preventing irreversible fibrosis and stenosis. Early surgical referral and intervention in the event of failed corticosteroid therapy in established fibrosis is crucial in preserving gastrointestinal function.

DISCLOSURES

Author contributions: FY Pan wrote the article, obtained consent, and is the article guarantor. M. Smale and M. Rennie revised the article for intellectual content. R. Langan is a pathologist who chose the histology images and provided descriptions. C. Rogge is the senior author who supervised this study.

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