MAR Gastroenterology (2025) 4:01

Case Report

Eosinophilic Gastroenteritis Mimicking Pyloric Stenosis in Infants: Medical Management over Hasty Surgery

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Received: 14 February 2025

Published: 19 February 2025

DOI: https://doi.org/10.5281/zenodo.15140207

Background

Eosinophilic gastrointestinal disorders (EGID) are rare disorders characterized by eosinophilic infiltration of gastrointestinal tract; eosinophilic gastroenteritis (EG) is a specific category of EGID referring to eosinophilic infiltration of the stomach and/or small intestine (1). EG affects both children and adults, and has a highly variable presentation (2). A US population-based study estimated the prevalence of EG/EoD at about 15 per 100,000 people, but recent research suggests it is likely underdiagnosed (4). While there is currently no standard for the diagnosis of EGID, presence of eosinophilic infiltrate on biopsy with greater than 10 eosinophils per high power field (hpf), associated Gastrointestinal (GI) symptoms, and exclusion of other causes of eosinophilia are generally accepted to diagnose EGID in children (1, 2). EG can be commonly misdiagnosed as infantile hypertrophic pyloric stenosis (IHPS) when infants present with projectile vomiting, and duodenal structuring (3). EG can be classified into three forms based on the predominance of eosinophilic infiltration: mucosal, muscular, and subserosal (3). The muscular form, which causes thickening of the bowel wall, can lead to obstructive GI symptoms and is often confused with IHPS given similarity of clinical presentation (1, 3). This can be detrimental given that treatment of EGID is medical while that of pyloric stenosis is surgical. Index of suspicion and standardized diagnosis of EGID is necessary to avoid unnecessary surgeries. The following two cases which we present exemplify this further and demonstrate the importance of appropriate diagnosis and pursuing medical management while avoiding hasty surgical intervention.

Case Reports:

Case 1

A 16-month-old male with no significant past medical history presented to the ED with acute onset coffee ground emesis, dehydration, and noted to have metabolic acidosis. Ultrasound of the abdomen showed a severely distended stomach with an elongated and thickened pyloric wall. NG Tube decompression yielded 400cc of breastmilk.

The upper GI series (Image 1) showed failure of contrast dye to pass through the gastric outlet and duodenum, suggestive of gastric outlet obstruction. An esophagogastroduodenoscopy (EGD) was performed (Image 2) which showed significant stenosis around the pylorus with duodenal thickening. The scope was unable to traverse the pylorus into the duodenum. He was given bowel rest, started on TPN and also IV methylprednisolone given the degree of intestinal inflammation.

Repeat EGD (Image 3) ten days later showed mild improvement in the duodenal thickening with a neonatal scope now able to traverse the pylorus, duodenal biopsies were now obtained. Duodenal bulb biopsies revealed prominent eosinophilia is the lamina propria (46/hpf) as also in the gastric antrum (62/hpf). A diagnosis of EGID was made, and he was started on a steroid taper, in addition to a leukotriene inhibitor (Montelukast), and was recommended to follow a six-food elimination diet. Repeat UGI series revealed resolution of the obstruction clinically correlated with resolution of symptoms. Over the course of the following months, food groups were reintroduced with the exception of dairy and wheat. Repeat endoscopy 3 months later showed resolution of eosinophilic gastroenteritis. He was tapered off Montelukast and, at one year follow-up, continued to be symptom free on a dairy free diet.

Case 2

A 2-year-old male with no significant past medical history presented to the ED with intermittent fevers, persistent abdominal pain, anemia unresponsive to iron supplementation, and elevated inflammatory markers. A computed tomography (CT) scan revealed duodenal thickening and a possible phlegmon. Given concerns for Crohn's disease, endoscopy was pursued which revealed the presence of a duodenal stricture with ulceration and friability. Given the narrow lumen which did not allow a standard endoscope to pass, a neonatal endoscope was used for intubation of the pylorus and duodenum and biopsies were obtained. Pathology findings indicated acute erosive duodenitis. He received treatment with methylprednisolone, nexium. 1 month later, he had an improvement in symptoms with appetite and abdominal pain improvement. He was continued on a proton pump inhibitor for 3 months, however, one week after being weaned off the PPI, he started to complain of abdominal pain again, needing PPI to be restarted. Six months after his initial presentation, UGI series was repeated which showed persistent duodenal narrowing. Another EGD was done which revealed esophagitis with an EREF score of 2, duodenitis with superficial erosions in the first part of the duodenum, and a duodenal stricture that could only be traversed with a neonatal scope. Esophagus revealed up to 55 eosinophils/HPF, and marked spongiosis and basal cell hyperplasia. Stomach antrum showed intestinal-type mucosa with 60 eosinophils/HFP. Duodenal biopsies showed preserved villous architecture, borderline intraepithelial lymphocytosis (up to 30 lymphocytes/100 enterocytes) and 75 eosinophils/HPF. He was given a short course of steroids, leukotriene inhibitor was considered but not started due to parental concerns. He was also put on a strict milk and wheat elimination diet along with a PPI. His symptoms improved over the following two months, and a repeated UGI series after steroid therapy demonstrated resolution of the duodenal narrowing. His endoscopy after 1 year of original presentation

showed resolution of duodenal stricture and reduction of gastrointestinal eosinophils.

Discussion

In both the cases, the initial presentation was that of duodenal strictures and only repeated biopsies showed evidence of EGID. These cases should serve as a reminder that multiple biopsies may be necessary to diagnose EGID due to the patchy nature of the eosinophilia. (4). For these cases, systemic steroids, proton pump inhibitors, leukotriene inhibitors (case 1 only), and food elimination diets were effective in treating the inflammation caused by EG.

Prednisone is the primary treatment for inducing remission in EG, and cases with no initial improvement with prednisone administration should be reevaluated for diagnosis of EGE (7). Numerous studies in the literature have demonstrated that leukotriene inhibitors (Montelukast), when used in combination with steroids or alone, induces significant clinical improvement in patients with EG (7). Montekulast is especially effective for treating dyspepsia in children aged 6-16 years with duodenal eosinophilia (5). Dietary elimination was also an effective treatment for these cases (4). Overall, these cases provide evidence that EG can be managed medically even in cases of complete obstruction of the gastric outlet to reverse the mechanical obstruction without resorting to surgery. While there are very few cases of EGID in published literature, and even fewer involving gastric outlet obstruction, there are some reports which support our findings where medical management was implemented and found to be therapeutic (8,6). Surgical interventions should be pursued when the case is refractory to oral steroids.

The concluding recommendation remains that EGID should be considered and ruled out before resorting to surgery, as even severe cases of gastric outlet obstruction can often be managed medically.

Acknowledgment:

Informed patient consent was obtained for the publication of the case details.

Disclosures:

The authors have no financial or conflicts of interest to disclose.

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