March of the Eosinophils: A Case of Eosinophilic Gastroenteritis, Immune Thrombocytopenia, and Iron Deficiency Anemia

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Introduction

- Eosinophilic gastrointestinal disorders (EGIDs) refer to eosinophilic infiltration of various sections of the gastrointestinal tract in the absence of secondary causes [1].
- Eosinophilic esophagitis has gained traction in recent years as a common cause of esophageal dysphagia in young adults, however eosinophilic gastritis (EG) and eosinophilic gastroenteritis (EGE) remain rarer clinical entities.
- The standardized prevalence of EG and EGE in the US was calculated to be around 6.3 per 100,000 and 8.4 per 100,000, respectively [2].
- Clinical manifestations of EGIDs vary depending on the section of the GI tract involved, as well as the layer of tissue that has been infiltrated by eosinophils [3]. A diagnosis of EGE requires endoscopic evaluation and histopathological examination of gastric and intestinal tissue [3].

Case Description

- A 16-year old male with a past medical history significant for autism, chronic ITP , GERD, melanoma, allergic rhinitis, and eosinophilic esophagitis presented for evaluation of chronic abdominal pain.
- He underwent an esophagogastroduodenoscopy (EGD) in April 2018 that revealed white exudates at the gastroesophageal junction, scattered erosions in the gastric body and fundus, severe antral gastritis, and scattered erosions noted throughout the duodenal bulb.
- Biopsies were taken and revealed distal eosinophilic esophagitis with greater than 50 eosinophils/HPF, eosinophilic gastritis, and chronic inflammation of the duodenal bulb with Brunner gland hyperplasia and villous blunting (Figure 1).
- Absolute eosinophil counts consistently remained higher than 2,000/uL

Case Description

- Given findings of persistent inflammation despite a six-food elimination diet, the patient was started on oral Budesonide, a proton-pump inhibitor, and an H2 receptor blocker.
- Surveillance EGD performed six months later revealed resolution of esophagitis and duodenal inflammation.
- Previous findings of duodenal metaplasia and villous blunting had resolved, as did the polypoid lesions noted in the gastric mucosa. Roughly 28 eosinophils/HPF were present in the gastric cardia near the gastroesophageal junction (GEJ), and numerous eosinophils remained in the other gastric tissues sampled.
- The patient's symptoms gradually improved, and oral budesonide was ultimately tapered and discontinued.
- On a follow-up visit three years later for surveillance EGD, the patient was found to have microcytic anemia, low folate, and iron levels, believed to be secondary to malabsorption caused by EGE. A diagnostic colonoscopy revealed no acute pathology in the colon or terminal ileum.
- EGD demonstrated persistent, diffuse eosinophilic gastritis involving the cardia, antrum, fundus, and body with minimal involvement of the distal esophagus (3 eosinophils/HPF) and no duodenal involvement.
- He was started on supplemental iron and folate, leading to resolution of the anemia; the patient currently remains on famotidine and pantoprazole with a plan for surveillance EGD every two to three years.

Figures

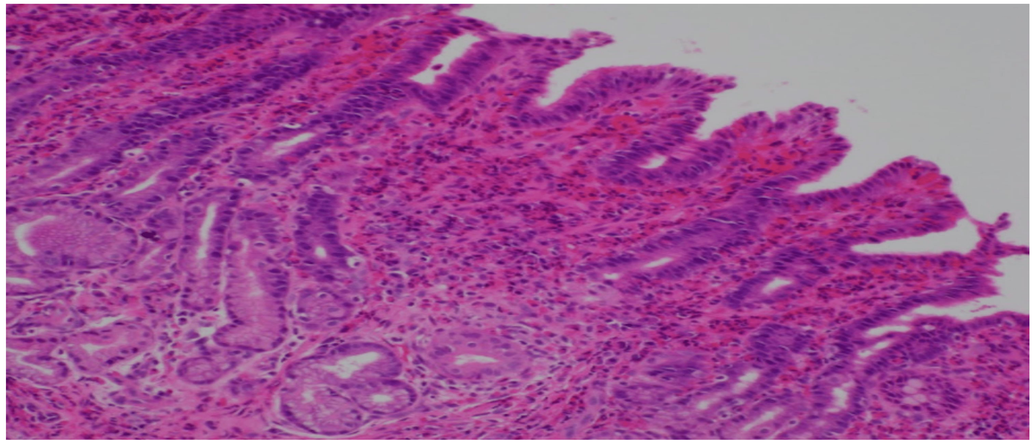

Figure 1: Cross-section of gastric tissue stained with hematoxylin and eosin demonstrating prominent eosinophils (greater than 100 per high-power field) within the lamina propria and gastric epithelium with associated epithelial injury.

Discussion

- Eosinophilic gastroenteritis is a rare clinical entity characterized by eosinophilic infiltration of the stomach and small intestine.
- Analysis performed by Jaffe et al and Caldwell et al using flow cytometry suggest that IL-13-mediated TH2 cell activation plays a role in EG/EGE [4,5].
- Food allergy has also been suspected to play a role, as food allergy free diets have demonstrated an improvement in disease activity [6]. The mainstay pharmacotherapy for EGE presently is glucocorticoids [1,7]
- EGE traditionally presents with nonspecific symptoms such as nausea, vomiting, abdominal pain, diarrhea, and weight loss [7]. Malabsorption can also be seen if there is involvement of the small intestine and can lead to various nutritional deficiencies [3,8]
- In their case report of an elderly woman with ITP and EG, Pan et al. recognized aberrant T-cell activation leading to B-cell stimulation and antibody production was the etiology underlying ITP [8].
- However, Ogawara et al. and Noriyuki et al. were able to identify that ITP is associated with a high TH1/TH2 ratio [9,10]. Given that much of the available literature surrounding EGID suggests a TH2-mediated response, it presently remains unclear if there a causal association exists between the two.

References and Disclosures

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