CASE REPORTS

Eosinophilic gastritis-a rare diagnosis

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ABSTRACT

Introduction: Eosinophilic Gastritis (EG), one of the less common amongst Eosinophilic Gastrointestinal Disorders (EGID), with a prevalence of 6.3 cases/100,000, is an uncommon cause of unspecific abdominal symptoms, including epigastric pain.
Case: A 23-year-old female, was admitted with recurrent episodes of nausea, vomiting, abdominal pain and weight loss. She was discharged one week previously after being treated for similar symptoms. The patient had failed an outpatient treatment with a Proton Pump Inhibitor (PPI). During her re-admission, Esophagogastroduodenoscopy (EGD) was performed, and results were significant for gastritis without ulcers or esophagitis. While awaiting biopsy results, the patient experienced a minimal improvement in her symptoms with intravenous fluids and PPI. Extensive workup remained negative except of eosinophilia in peripheral blood. Gastric mucosal biopsy revealed eosinophilic infiltrates in Lamina Propria, confirming the diagnosis of Eosinophilic Gastritis. *Helicobacter pylori* on immunohistochemistry was negative. The patient was counseled regarding the Six Food Elimination Diet for the management of this condition. Her symptoms began to improve under dietary restrictions. Repeated EGD with biopsy at 12 weeks showed resolved eosinophilic infiltrates.
Discussion: EG remains a rare diagnosis, and it should be included as a differential diagnosis in every patient with refractory symptoms of nausea, vomiting, abdominal pain, and failure to thrive. Diagnosis of EGID is established after the exclusion of other causes of eosinophilia in the gastrointestinal tract.

Key Words: Eosinophilic gastritis, Six food elimination Ddiet, Eosinophilic infiltrates in gastric mucosa

1. INTRODUCTION

Eosinophilic Gastrointestinal Disorders (EGID) are chronic inflammatory disorders associated with eosinophilic infiltration in the absence of an identifiable secondary cause. These disorders are well described in the pediatric population but are less common in adults.[1] Patients with EGID present with varying symptoms based on the type of organ affected and the extend of eosinophilic infiltration. Eosinophilic Gastritis is one of the less common types of EGID, with a reported prevalence of 6.3/100,000 patients. Other EGIDs include eosinophilic esophagitis, eosinophilic gastroenteritis, eosinophilic enteritis, and eosinophilic colitis.[2, 3]

Eosinophilic gastritis is more common in the female sex. The prevalence of EG increases with age as compared to Eosinophilic gastroenteritis (EGE), which is more common in children.[4] EG patients commonly present with nausea, vomiting, epigastric pain and discomfort, but may induce hematemesis, or melena. These symptoms are not specific; therefore, diagnosis of EG requires the presence of symptoms, histologic findings based on biopsies from upper endoscopy, and absence of possible secondary causes. Gastric biopsies with the eosinophilic count of > 30 HPF, support the diagnosis of EG in the appropriate clinical setting. The low prevalence of EG and its tendency to cause non-specific and

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common symptoms may lead to misdiagnosis and inadequate management of these patients.

2. Case presentation

A 23-year-old female initially presented to the Emergency Department with an 8-weeks history of nausea, vomiting, epigastric pain, weight loss, and inability to tolerate oral intake. The patient had a past medical history of Insulin-dependent type 2 diabetes, obesity, and depression. Lab tests on admission were unremarkable. Computed tomography scan (CT-scan) of the abdomen and pelvis, and right upper quadrant ultrasound were only significant for mild hepatic steatosis. Helicobacter pylori stool antigen and Celiac testing were negative. The patient was started on intravenous fluids, a Proton Pump Inhibitor (PPI), and antiemetics, which led to a temporary improvement of her symptoms. She was discharged home on an oral course of PPI for four weeks. The patient's symptoms recurred shortly after discharge leading to her readmission one week later.

Further workup included a specific gastroenterology consultation. Esophagogastroduodenoscopy (EGD) was performed on admission, and findings revealed gastritis without ulcers or esophagitis (see Figures 1, 2). Biopsies were obtained from gastric mucosa, but not from the esophagus as mucosa was macroscopically normal-appearing. Pending gastric biopsy results, the patient was continued on PPI and maintenance fluids, with minimal improvement in her symptoms. CT-scan of the brain, nuclear cholescintigraphy, and gastric emptying study were part of the extensive workup, performed in order to rule out other possible causes of persistent symptoms, the results of which were unremarkable.

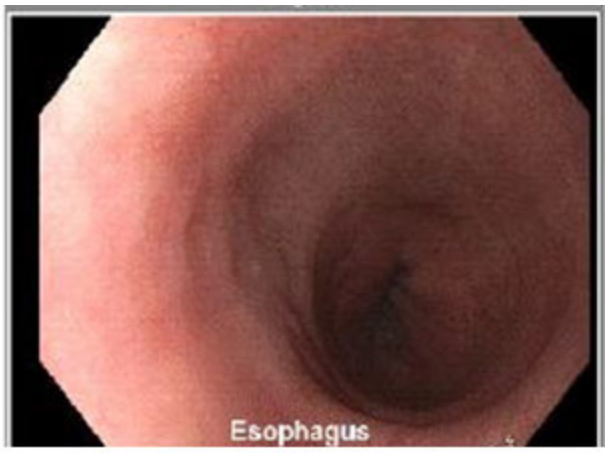

Figure 1. Image of patient's Esophagus from Upper endoscopy performed during hospitalization. Visually normal appearing mucosa without evidence of inflammation or injury.

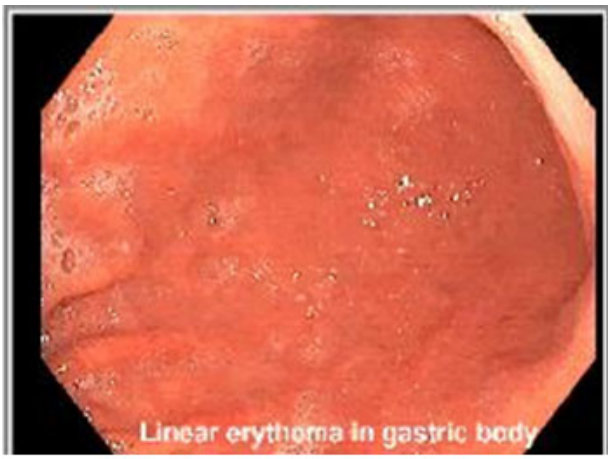

Figure 2. Image of Gastric body from Upper endoscopy performed during hospitalization. Gastric mucosa appears inflamed with linear erythema.

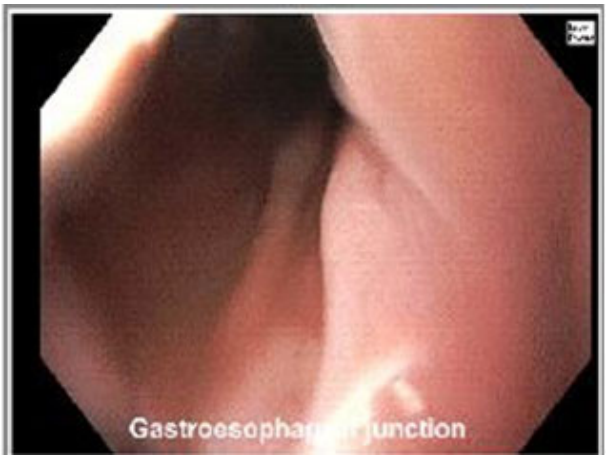

Figure 3. Image of Gastroesophageal Junction from Endoscopy performed in 12 weeks after discharge. Normal appearing mucosa without evidence of ulceration or inflammation

Peripheral blood eosinophilia was noticed on day 4 after admission. Previous complete blood count (CBC) with blood cells differentiation were unremarkable. The patient had no known history of atopy or allergies. The gastric biopsy revealed an extensive eosinophilic infiltration within the lamina propria, numbering greater than 50 HPF in most areas. The biopsy was negative for Helicobacter pylori immune-histochemical stain and dysplasia, findings that were consistent with the diagnosis of Eosinophilic Gastritis. In the absence of diarrhea or rectal bleeding, concurrent eosinophilic colitis was not clinically suspected; therefore, a colonoscopy was not recommended by our gastroenterologist.

As the patient was symptomatic, the Six Food Elimination Diet (SFED) was initiated as first-line therapy for her condition and was continued on oral PPI therapy. She was

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evaluated by a dietician to help give recommendations and promote adherence to the specific diet. The patient was discharged home with the recommendation of a follow-up visit in our Gastroenterology Clinic.

The patient's symptoms completely resolved in two months under the elimination diet. She was continued on SFED and repeat EGD with biopsy was performed at 12 weeks, demonstrating resolution of gastritis (see Figures 3, 4) and eosinophilic infiltration.

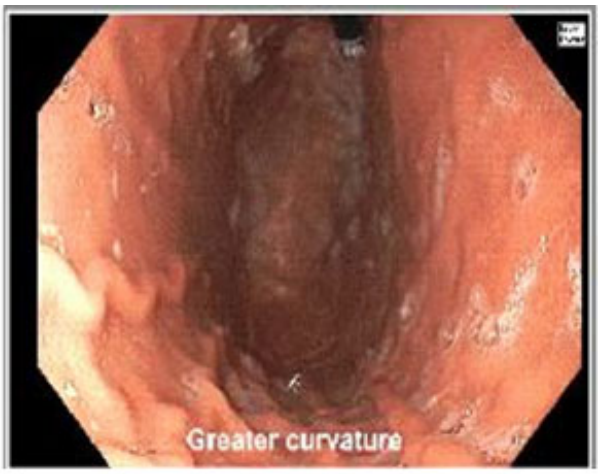

Figure 4. Image of Gastric body from Endoscopy performed in 12 weeks after discharge. Resolved linear erythema as compared to Figure 2.

3. DISCUSSION

The aspects of this case that merits discussion includes: diagnosing eosinophilic gastritis, management of a patient with EG, and Uniqueness of this case.

3.1 Diagnosing eosinophilic gastritis

The non-specific symptoms of EG, which can be present in many other conditions, makes it a challenging diagnosis. Varying symptoms are seen based on the depth of the eosinophilic infiltration of the gastric wall. Nausea, vomiting, and epigastric discomfort is seen with eosinophilic infiltration of the mucosal layer, while the involvement of muscularis layer can mimic gastric outlet obstruction.[5]

Esophagogastroduodenoscopy is warranted in all patients with refractory and non-specific abdominal symptoms (these symptoms are discussed 'Introduction'). As EG is histologically diagnosed, it is imperative to perform biopsy of gastric mucosa on evidence of inflammation macroscopically. Findings that can be appreciated during gastroscopy in a patient with EG include; normal mucosa, mild erythema and erosive ulcerations, presence of mucosal necrosis can be seen in extreme cases. Need for obtaining biopsies on grossly normal gastric mucosa in a symptomatic patient is debatable, and would require greater degree of clinical suspicion for EG when alternate diagnosis could not be derived. Eosinophil count of > 30 HPF on gastric biopsies supports the diagnosis of EG in appropriate clinical setting.

It is curial to rule out underlying potential etiologies, before ascertaining a diagnosis of EG. *Helicobacter pylori* infection, gastrointestinal helminth infection, use of non-steroidal anti-inflammatory drugs (NSAIDs), known food allergies, hyper-eosinophilic syndrome, and connective tissue diseases are some of the known etiologies associated with eosinophilic infiltration of the gastric mucosa. EGID are found to be associated with atopic conditions, peripheral blood eosinophilia, or protein-losing enteropathy. Our patient had no history of asthma, or allergies and presented with the acute onset of her gastrointestinal symptoms. Peripheral blood eosinophilia was seen later in the admission but is not part of the diagnostic criteria nor consistently present in patients who suffer from this condition. Greater than 50% of EGID cases occur independent of peripheral blood eosinophilia.[6] A range of 5% to 35% is reported in the literature for the prevalence of peripheral blood eosinophilia in EGID.[7]

3.2 Management of patients with symptomatic eosinophilic gastritis

There are no defined treatment guidelines for the management of the patients with EG. The elimination diet is one of the well-studied approaches to the management of patients with eosinophilic esophagitis (EOE),[8] but not yet established for EG. A prospective clinical trial studied Six Food Elimination Diet (SFED) in EGE, preliminary results were noteworthy for improvement in clinical symptoms, and resolution of peripheral and histologic eosinophilia at six weeks.[9] SFED excludes soy, wheat, egg, nuts, milk, and fish from dietary intake. We observed similar improvements in our patient (compared to preliminary results of the study), with adherence to 12 weeks of SFED. Although there is no definitive evidence to prove that food hypersensitivity causes EGID, clinical improvement after adherence to an elimination diet supports the role of food allergy or hypersensitivity.

Patients treated with oral steroids have shown clinical improvements in the literature in EGID, but this has only been studied in small series of patients.[10] As corticosteroids are known to have several potential sides effect and risks, their use is limited. Oral budesonide, is well established treatment for inflammatory bowel diseases and eosinophilic esophagitis; however, nausea, dyspepsia, and abdominal discomfort are commonly reported adverse reactions associated with its use. Lack of clinical data for the use of budesonide in EG and predominance of gastrointestinal adverse effects, limited

its use in this case. The use of prednisone was not an ideal treatment choice in this patient with diabetes due to the risk of hyperglycemia; therefore, SFED was initiated as a first line treatment option. The steroids were reserved as a second line treatment for refractory symptoms despite adherence to dietary restrictions.

3.3 The uniqueness of this case

The majority of EG cases are reported in the pediatric population. In adults, reported EG cases were related to a known etiology, had associated enteritis/colitis, or presented with complications of undiagnosed/untreated EG.

Only two cases of uncomplicated EG without underlying cause were reported. In the first case, 34-year-old male, presented with acute abdominal pain and peripheral eosinophilia. The patient had spontaneous resolution of his symptoms without any treatment.[11] In the second case, 37-year-old male was evaluated for chronic abdominal symptoms, unresponsive to conventional treatment. After confirmation of diagnosis of EG, patient was started on oral prednisone without trial of SFED. Treatment with steroids resulted in mild improvement, but symptoms relapsed on dose reduction. Patient was eventually started on Interferon alpha and continued on prednisone.[12] No further details of clinical response to Interferon alpha were discussed.

In addition to being a rare diagnosis, our case highlights the patient's response to the SFED, and the potential role of atopy in the pathophysiology of the condition.

4. Conclusion

Eosinophilic Gastritis remains a rare diagnosis. Clinical presentation with non-specific symptoms makes it a diagnostic challenge for clinicians, requiring a high degree of suspicion in patients with refractory symptoms of nausea, vomiting, abdominal pain, and failure to thrive in patients in whom an alternative diagnosis cannot be determined. Diagnosis is established on gastric biopsy, after exclusion of other causes of eosinophilia in the gastrointestinal tract.

Clinical trials on larger patient populations are needed to help formulate guidelines for the optimal management of patients with EG.

Conflicts of Interest Disclosure

The authors have declared no conflicts of interest.

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