Eosinophilic Gastroenteritis in a Young Filipino Adult: A Case Report

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Abstract

Introduction. A 23-year-old young female patient presenting with persistent diarrhea and eosinophilia, who had clinical improvement after administration of steroids

Case. A 23-year-old Filipino female, without comorbidities presented with abdominal pain, diarrhea and vomiting. Initial work-up showed peripheral eosinophilia. Computed tomography (CT) of the abdomen revealed massive ascites. An esophagogastroduodenoscopy (EGD) was done which revealed acute duodenitis. Gastric and duodenal biopsy revealed tissue eosinophilia. Total Immunoglobulin E (IgE) was elevated while work-up for intestinal parasitic infection was negative. On bone marrow biopsy, there was no eosinophilic infiltration. Oral prednisone was then started at 60mg/day and was subsequently tapered over 30 days resulting in resolution of symptoms.

Conclusion. This report illustrates a rare case of eosinophilic gastroenteritis (EGE). EGE may mimic a wide spectrum of gastrointestinal disorders, hence, prompt recognition of EGE and awareness of its clinical symptoms, diagnosis and treatment is important.

Keywords: Eosinophilic gastroenteritis, case report, eosinophilia, diarrhea

Introduction

EGE is a rare disease characterized by eosinophil-rich inflammation of the stomach and small intestine. The prevalence is estimated to be 5.1 in every 100,000 population. Diagnosis of EGE requires three criteria, which are the presence of gastrointestinal (GI) symptoms[1]; histologic evidence of eosinophilic infiltration in one or more areas of the GI tract[2] and exclusion of other causes of tissue eosinophilia[3]. This is a case report of a rare disease with seemingly common gastrointestinal symptoms that aims to discuss the diagnostic criteria and treatment of patients with EGE[1].

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Case Presentation

A 23-year-old Filipino female presented with a 6-day history of diarrhea, vomiting and epigastric pain. There was no associated fever, dysuria, and hematuria. She was initially seen at the emergency room and managed as a case of Acute Gastroenteritis and was sent home with oral rehydration salt, racecadotril, and domperidone. However, there was no noted improvement of symptoms. Hence, another consult at the emergency room was done after 4 days and the patient was subsequently admitted. She had no known comorbidities, no history of heredofamilial diseases, allergies to foods or medications, previous parasitic infection and use of steroids.

Pertinent findings on physical examination include presence of flabby abdomen with direct tenderness on the epigastric area, no palpable masses or organomegaly, a positive fluid wave and positive shifting dullness signifying ascites.

Her complete blood count (CBC) showed peripheral eosinophilia of 16%. Her Total IgE was normal at 28.01. Fecalysis, stool culture, and formalin ether concentration technique were unremarkable. CT of the abdomen exhibited massive ascites as shown in *Figure 1*

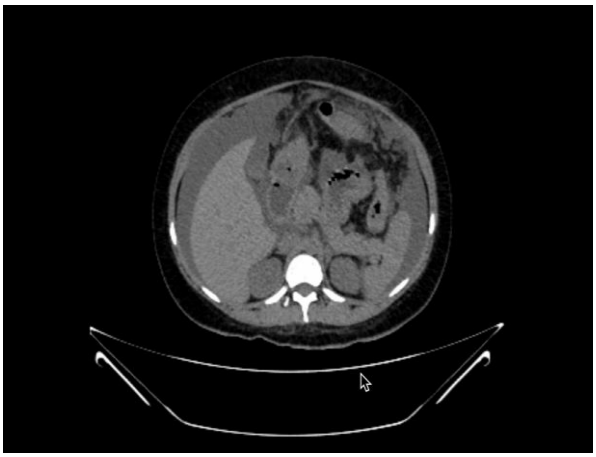

Figure 1. Whole Abdominal CT Scan of the patient showing massive ascites in the perihepatic, pericholecystic, perisplenic, meseteric, bilateral

EGD demonstrated acute duodenitis and no ulcerations or masses as shown Figure 2**.** Multiple biopsies were taken from the gastric mucosa and duodenal mucosa which showed chronic, mild active gastritis with tissue eosinophilia and villous blunting with tissue eosinophilia, respectively. Bone marrow biopsy was done revealing normocellular bone marrow for age and no eosinophil infiltration. Hence, hypereosinophilic syndrome was ruled out.

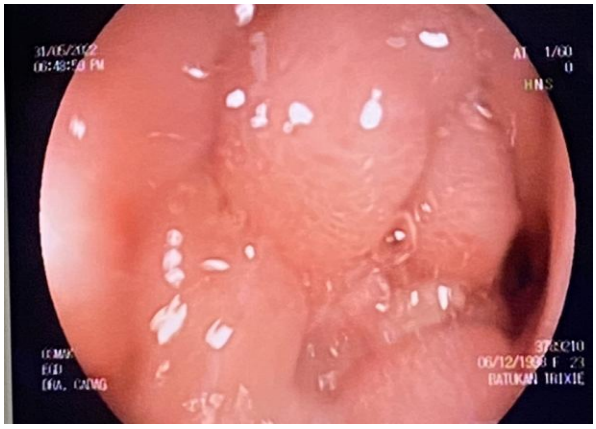

Figure 2. Endoscopic photos of the stomach with normal gastric folds and normal mucosa from cardia to pyloric ring.

Based on the clinical manifestations, tissue eosinophilia observed in gastric and duodenal biopsies, and a normocellular bone marrow aspiration, the patient was diagnosed with eosinophilic gastroenteritis (EGE). She was treated with prednisone at a dosage of 60 mg/day (1mg/kg/day) for 30 days, resulting in resolution of both her symptoms and peripheral eosinophilia.

Eosinophilic ascites is an uncommon presentation of EGE, caused by eosinophilic inflammation and subsequent edema of the small bowel serosal layer. Although paracentesis and ascitic fluid analysis were not performed to confirm the etiology of the patient's ascites, her condition improved markedly after steroid initiation.

The patient was discharged with instructions to follow a targeted elimination diet. On follow-up, she remained symptom-free, with a repeat CBC showing only 2% eosinophils. The prednisone dose was gradually tapered until discontinued, and she was monitored for any recurrence.

Discussion

Eosinophilic gastrointestinal disorders (EGID) is characterized by eosinophilic infiltration in the GI tract in the absence of known causes for eosinophilia[1,2]. EGID has six main types namely: eosinophilic esophagitis, eosinophilic gastroenteritis, eosinophilic gastritis, eosinophilic enteritis, eosinophilic duodenitis, and eosinophilic colitis. It is known that eosinophilic esophagitis usually presents with dysphagia while both eosinophilic gastritis and gastroenteritis is characterized by nausea, vomiting and abdominal pain. Abdominal pain, diarrhea and rectal bleeding are common symptoms in a patient with eosinophilic colitis[3].

In cases of EGID, it is important to rule out common causes of eosinophilia such as drug hypersensitivity, connective vascular disease, malignancy and infection[4]. General diagnostic evaluation includes complete blood count, Total IgE, erythrocyte sedimentation rate, skin-prick testing, infection work-up as well as lower and upper gastrointestinal endoscopy. In the presence of hypereosinophilia, bone marrow analysis, serum tryptase, serum vitamin B12, echocardiogram, and genetic analysis for FIP1L1-PDGFRA fusion gene[2].

EGE is a rare disease characterized by eosinophil-rich inflammation of the stomach and small intestine. According to the National Organization for Rare Diseases, it affects both males and females, but is slightly more common among men. In adults, peak prevalence is among those 20-50 years of age. The prevalence is estimated at 5.1 in every 100,000 population[5].

In our case, we diagnosed the patient with eosinophilic gastroenteritis which requires the fulfillment of three criteria, namely: presence of GI symptoms[1]; histologic evidence of eosinophilic infiltration in one or more areas of the GI tract[2]; and exclusion of other causes of tissue eosinophilia[6]. The patient had signs of gastrointestinal involvement such as abdominal pain, diarrhea and ascites. Secondly, the endoscopic tissue biopsy revealed marked eosinophilic infiltration in the gastric mucosa.

Eosinophils are highly specialized granulocyte effector cells that produce and store diverse biologically active

molecules which regulates local immune and inflammatory responses as they play vital role in gastrointestinal homeostasis by interacting with constituents of the intestinal mucosa such as the nerves, endothelium, and immune cells which mediate barrier integrity maintenance as well as tissue damage through the release of granule mediators[7,8]. EGE is idiopathic and the pathophysiology of EGE is still not well-established but an allergic component has been documented in most patients. When there is contact with an allergen, such as in food allergy, a cascade of inflammatory response occurs. This ultimately leads to recruitment of eosinophils which subsequently become cytotoxic by producing factors such as major protein, an eosinophil-derived neurotoxin eosinophilic cationic protein and eosinophilic peroxidase[9].

EGE has three different disease types based on the layer of gastrointestinal wall affected by eosinophilic infiltration. Patients with mucosal involvement commonly present with iron-deficiency anemia, enteropathy, protein loss or malabsorption. Involvement of the muscular layer is characterized by diffuse thickening of the abdominal wall which can cause obstructive symptoms as well as intussusception and perforation. Serosal involvement can present as peritonitis and ascites[10].

Targeted elimination diet is one of the strategies for the treatment of EGE. Since the most common pathophysiology of EGE is secondary to food allergy, elimination diet provides clinical improvement in most patients. It involves removal from the diet of the six most common allergenic foods such as dairy, eggs, wheat, soy, peanuts, fish/shellfish[1,2,11]. In a study[12] conducted by Yoshiyuki et al., which included three patients with EGE and treated with targeted elimination diet without systemic steroids, all patients had improved clinical symptoms and imaging or histological findings. In patients with EGE who had targeted elimination diet for 4-8 weeks, there was noted histological improvement in more than 70% of the population[13].

Steroid therapy is one of the mainstays for induction of remission. Prednisone, in particular, induces neutrophil apoptosis and inhibits chemotaxis, inhibiting eosinophilic infiltration in the GI tract. For systemic steroid therapy, a course of 2-6 weeks with relatively low doses is administered with an excellent response rate at 90%. Other therapies include azathioprine, leukotriene inhibitors, antihistamines and biologics agents such as omalizumab, infliximab and mepolizumab[1,2,14]. Moreover, a case report from China[15] reported a case wherein a patient with eosinophilic gastroenteritis had fast therapeutic response after FMT. However, further studies should be done in order to establish its safety and efficacy[15].

The prognosis of eosinophilic gastroenteritis (EG) in adults varies significantly based on disease severity, organ involvement, and the individual's response to treatment[16]. The case we reported involved a localized gastrointestinal tract involvement, and the patient responded well to corticosteroids. As a result, her prognosis is favorable, and with proper treatment and management, she is expected to have a normal or near-normal life expectancy. Key factors in improving outcomes for adults with eosinophilic gastroenteritis include close monitoring, adherence to dietary adjustments, and personalized treatment strategies.

One limitation of this case report is the absence of ascitic fluid analysis to assess eosinophil count, which would have provided more objective evidence to support the diagnosis of eosinophilic gastroenteritis (EGE) as the cause of the patient's ascites. Due to the unavailability of this test, the diagnosis relied primarily on the clinical presentation and the patient's response to treatment. This approach introduces a degree of uncertainty in the final conclusion.

Conclusion

EGE should be one of the differentials in a patient presenting with diarrhea and eosinophilia unresponsive to conventional treatment. Awareness of rare diseases such as EGE is important to avoid misdiagnosis and improve outcomes for patients. In patients who do not have severe manifestations, targeted elimination therapy is the initial management. Meanwhile, in other patients such as in this case with severe manifestations, steroid is the mainstay for induction of remission.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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