Young Male With Chronic Small Bowel Diarrhea : A Case Report

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Abstract

Eosinophilic Gastroenteritis (EoG) is a rare benign entity of unknown etiology characterized by focal or diffuse eosinophilic infiltration in the small intestine. Klein et al. categorized EoG into three different patterns relying on the concept of the predominant location, extent, and depth of intestinal eosinophilic infiltration. Diagnosis can only be obtained by high clinical suspicion and histopathological confirmation after the excluding of common causes of GI eosinophilia. Treatment is primarily based on systemic corticosteroids, which improve symptoms and endoscopic lesions dramatically. This report highlights an interesting case of chronic diarrhea and mucosal EoG that was successfully treated with steroids.

Keywords: Eosinophilic gastroenteritis • GI eosinophilia • Rare diseases

Introduction

Eosinophilic Gastroenteritis (EoG) is related to a group of Eosinophilic Gastrointestinal Diseases (EGID). These are rare benign diseases that are characterized by varied clinical symptoms with gastrointestinal (GI) eosinophilia on biopsy specimens [1]. Due to its rarity, most knowledge related to these disorders was derived from case series and histopathology observations only. EOG is ruled out from other causes of GI eosinophilia by their selective GI involvement, and dominant eosinophilic cell types with no other inflammatory cell infiltrate on biopsy. Of late, due to an increase in the number of diagnosed cases, the cellular and molecular basis are further identified for better understanding and treatment of this clinical entity. In this case report, we present an unusual case of chronic diarrhea with pain abdomen, which led to the diagnosis of mucosal subtype of EoG.

Case Presentation

A 32 years old male who is a army soldier presented to our outpatient department with complaints of pain abdomen and chronic watery diarrhea for 8 months. He described abdominal pain as epigastric in location and his diarrhea was of large volume and watery type occurring with more than 4-6 episodes per day. He also lost 5 kgs over 3 months duration. He was visited multiple medical centers and was recieving multiple medications for the same.There is no history of fever, abdominal distention, malabsorption, rash. He has no history of atopy, food, and pollen, or drug allergy. His physical examination was unremarkable. His laboratory values showed ESR of 36 mm/hr; hematocrit of 45%, AEC of 1240 cells/μL; total IgE levels of 2200 UI/ml, His liver and renal parameters were in the normal range. The stool routine for parasites and atypical organisms was normal. There are no remarkable features in abdominal ultrasound. Endoscopy with gastric and duodenal biopsy was taken showed no evidence of H pylori in the gastric antral biopsy. The duodenal biopsy showed EoG (>40 eosinophils/100 enterocytes). His symptoms improved after receiving proton pump inhibitors, Ivermectin, and enteric-coated budesonide.

Results and Discussion

With a prevalence of 8.4 in 100000 [2]. Eosinophilic Gastroenteritis (EoG) is an uncommon inflammatory disorder with no established cause [3]. Kaijser et al. initially described this condition in 1937 as a disease caused by patchy or diffuse eosinophilic infiltration of the gut wall, resulting in a variety of Gastrointestinal (GI) symptoms [4]. Klein et al. later classified this benign entity into three types of EoG depending on anatomical location in the small bowel: mucosal subtype, muscle subtype, and serosal subtype [5]. Each subtype of EoG has a different clinical presentation. The physician needs to attain sufficient knowledge of EoG to establish a positive diagnosis. The exact physiopathological mechanism is not fully understood in EoG. Eosinophils are uniquely distributed inhomogeneously throughout the GI tract. It is present in high abundance in ileocaecal and appendicular regions with absolutely none of them seen in the esophagus [6]. The frequent connection of EoG with other atopic and allergy illnesses suggests that the diseases may have an immunologic origin. Existing theories hypothesize that the disturbances both in IgE-mediated and cell-mediated mechanisms cause accumulation, activation, and degranulation of eosinophils culminating in tissue damage. Chemokines like Eotaxin (CCL11) have also been found to have a function in eosinophil homing and recruitment [7]. Additionally, non-atopic/non-allergic and familial

Table 1. Clinical features in EoG.

Site of involvement	Frequency (%)	Symptoms
Mucosal (most common)	45-80	Abdominal pain, Diarrhea, Malabsorption, GI hemorrhage, Protein-losing enteropathy.
Muscular is propria	12-30	Obstructive symptoms due to pyloric stenosis, gastric outlet obstruction and intussusception
Serosa (rarest presentation)	12.5-39	Exudative ascites

Table 2. Diseases compatible with the clinical picture of EoG.

Intestinal	Systemic
	1. Primary hyper eosinophilic syndrome 2. Vasculitis a. Churg-Strauss b. Polyarteritis nodosa
1. Parasites (helminths) 2. *Helicobacter pylori* infection 3. Food allergies 4. Pseudomembranous colitis 5. Chrons disease 6. Celiac disease 7. GI tuberculosis 8. Lymphangiectasis 9. Portal enteropathy	3. Connective tissue diseases a. Systemic lupus erythematosus b. Scleroderma c. Dermatomyositis 4. Neoplasms (lymphoma, cancers) 5. Graft versus host disease 6. Drugs a. Nonsteroidal anti-inflammatory drugs b. Interferon α c. Enalapril d. Carbamazepine e. Trimethoprim/ Sulfamethoxazole f. Clopidogrel

factors were also found to be important in tissue eosinophil recruitment [8].

Clinical presentation is usually elusive and heterogeneous. EoG can occur in any age group. however, the highest incidence is noted in the third to fifth decade of life with male gender preponderance. Depending on the extent and depth of eosinophilic infiltration the clinical symptom can fluctuate (Table 1) [9]. There are no definite diagnostic criteria followed. However, Features that strongly support the diagnosis of eosinophilic gastroenteritis include the presence of GI symptoms, the histological confirmation of GI eosinophilia (>20 eosinophils per high-power field) with or without peripheral eosinophilia and the absence of other causes for secondary eosinophilia [10]. Endoscopic and Radio-graphic changes are usually nonspecific and can be absent in >50% of patients [11]. Disease mimickers to EOG must be ruled out (Table 2). Most patients of EoG will respond to full course corticosteroid therapy. Proton pump inhibitors, Antihistamines, Mast cell stabilizers may provide additional benefits. Disease identification and treatment in the early course of the disease will provide superior results but some cases can also have a chronic disease course with unpredictable relapses [9,10]. Immunosuppressive drugs such as Azathioprine, 6-mercaptopurine, Methotrexate, Tumor necrosis factor α blockers, and vedolizumab have been tried in steroid-refractory or steroid-dependent disease courses, however, the treatment outcomes are variable. Our case was an interesting case of an army soldier who developed pain abdomen and chronic watery small bowel diarrhea who was diagnosed having EoG and successfully treated with corticosteroids [12].

Conclusion

Eosinophilic gastroenteritis (EoG) is an inflammatory disorder characterized by abdominal symptoms and GI eosinophilia (>20 eosinophils/HPF). The clinical symptom might vary depending on the degree and depth of eosinophilic infiltration. The majority of patients respond to steroids, while refractory cases necessitate immunomodulator therapy. Ours is an interesting case of chronic diarrhea was diagnosed with EoG after multiple referrals and successfully treated with corticosteroids.

Points to Remember

- Eosinophilic Gastroenteritis is a rare benign treatable gastrointestinal disease.
- Etiology and pathogenesis are not well understood. However, hypersensitivity responses to dietary allergens were postulated to play a role in eosinophils requirement.
- Disease presentation varies based on location and degree of intestinal involvement.
- Many therapeutic options have been suggested, including dietary considerations, leukotrienes inhibitors, mast cells stabilizers, and corticosteroid.
- Immunomodulator therapy and surgery are typically reserved for steroid-refractory patients.

Disclosure Statements

There are no financial disclosures. The authors declare no conflicts of interest.n.

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