

Eosinophilic gastroenteritis: A case report of a rare disease mimicking acute abdomen

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ABSTRACT

Eosinophilic gastroenteritis (EGE) is a rare disease that can present as acute abdomen and can be challenging for a clinician to differentiate amidst the numerous causes of abdominal pain. Due to the non-specificity of symptoms and scarcity of diagnostic guidelines, a high index of suspicion is required to diagnose EGE and thereby defer unnecessary surgical interventions. Herein, we present the case of a 17-year-old female who had presented to the emergency department (ED) with severe abdominal pain and peripheral eosinophilia mimicking an acute abdomen. The relevance of the case resides in highlighting the fact that such rare diseases are often missed in ED and a broader diagnostic spectrum should be considered while evaluating cases of undifferentiated acute abdomen.

Key words: Acute abdomen, Eosinophilic gastroenteritis, Peripheral eosinophilia, Steroids


Eosinophilic gastroenteritis (EGE) is a rare disease affecting the gastrointestinal (GI) system with a reported incidence of 1–30/100,000 population [1,2]. It was first described by Kajiser in 1937, and since then, about 300 cases have been described in the literature [3]. EGE manifests with varying GI symptoms which can mimic an acute abdomen and is characterized by eosinophilic infiltration of the GI tract. The most common anatomic sites involved are the stomach and proximal bowel; although, it may affect any part of the GI tract from the esophagus to the rectum [4]. EGE is clinically diagnosed by the triad of abnormalities of GI function, eosinophilic infiltration of GI tract, and exclusion of other diseases causing peripheral eosinophilia. EGE can masquerade among the numerous causes of the acute abdomen and can pose a diagnostic challenge in the emergency department (ED). Recent studies have shown that the prevalence of EGE is increasing, and therefore, it is imperative that clinicians are acquainted to this emerging medical condition [5,6].

CASE REPORT

A 17-year-old female with no prior health issues was referred to our ED with a provisional diagnosis of acute appendicitis from

a primary care center. She complained of diffuse abdominal pain with increased intensity over the right iliac fossa and three bouts of non-bilious vomiting. For the past 5 days, she had mild abdominal pain with a waxing and waning course, associated with dyspeptic symptoms, and bloody diarrhea. She also complained of a mild, persistent cough for the past month. She had no complaints of fever or dysuria. She had no history of allergies and her menstrual cycles were normal. On admission, her vitals in the ED were stable and she was afebrile. Focused examination revealed mild tenderness in the right iliac fossa. She was given analgesics and blood samples were drawn for laboratory investigations which showed a total count of 20,300 cells/mm³ with 11% neutrophils, 20% lymphocytes, and 65% eosinophils with an absolute eosinophil count of 13,195 cells/mm³. Other blood and urine tests were within normal limits. Peripheral smear revealed severe eosinophilia. Stool examination was negative for evidence of parasitemia.

To rule out a surgical cause for the abdominal pain, an ultrasound scan was done which revealed mild free fluid in the pouch of Douglas and right iliac fossa with a sonologically normal appendix. X-ray chest revealed bilateral reticulonodular shadows. A contrast-enhanced computed tomography (CECT) of the abdomen was done which showed pre and para-aortic lymph nodes with minimal free fluid. A CECT chest revealed bilateral centrilobular nodules with septa thickening suggestive of

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subacute hypersensitivity pneumonitis (Fig. 1). No surgical cause for her abdominal symptoms could be elicited despite a thorough evaluation. Her anti-nuclear antibody profile was negative for autoimmune diseases and perinuclear anti-neutrophil cytoplasmic antibodies were negative for Churg-Strauss syndrome. An endoscopy and duodenal biopsy were done in view of possible EGE.

Histopathological studies showed significant infiltration of lamina propria by eosinophils which clinched the diagnosis of EGE (Fig. 2). She was started on deflazacort 6 mg twice daily orally for 2 weeks along with Pantoprazole 40 mg, anti-histamines, and probiotics. She had a dramatic resolution of her symptoms within 2 days of initiating treatment and the patient was discharged in a stable state.

DISCUSSION

EGE belongs to a spectrum of GI disorders which also includes eosinophilic esophagitis, eosinophilic gastritis, eosinophilic enteritis, and eosinophilic colitis [7]. EGE may affect any age group, although the peak incidence is between the third and fifth decade of life with a slight male predominance [8,9]. Based on the anatomic location of eosinophilic infiltration in the intestinal wall, Klein *et al.* in 1970 classified the disease as mucosal, muscularis,



Figure 1: Contrast-enhanced computed tomography image of the chest showing prominent centrilobular nodules with interlobular septal thickening suggestive of subacute hypersensitivity pneumonitis

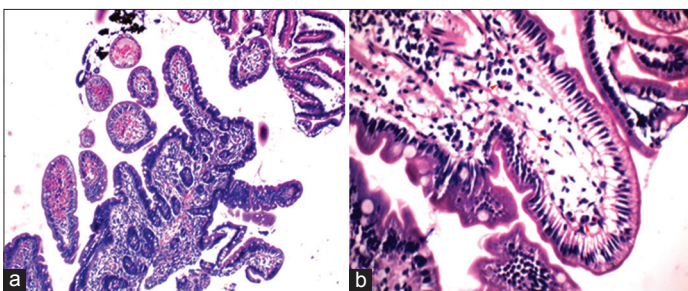


Figure 2: (a) Photomicrography of the duodenal biopsy specimen showing hypercellularity of the lamina propria with normal serosal and muscular layers; (b) magnified image of the duodenal biopsy specimen showing eosinophils infiltrating the lamina propria (indicated by red arrows) with normal glands

or serosal type [10]. The exact etiopathogenesis for EGE remains unknown, but eosinophilic inflammation of the GI tract from an allergic response is often thought to be an underlying cause.

The clinical manifestations of EGE depend on the site, as well as the depth of eosinophilic infiltration of the gut wall involved. Involvement of the mucosal layer often presents with abdominal pain, nausea, vomiting, bloody diarrhea, fecal occult blood loss, anemia, or protein losing enteropathy. Involvement of the muscular layer may present with signs and symptoms of gastric outlet and intestinal obstruction while the involvement of serosal layer presents with ascites [11]. Rarely, the serosal type of EGE may present with eosinophilic ascites without peripheral eosinophilia.

Due to the rarity of the disease and the absence of guideline recommendations, evaluation and management of EGE is challenging. Diagnosis of EGE requires a high index of suspicion when no other cause can wholly explain the clinical features. The history should be focused on eliciting allergies to food, medicines, or environmental agents, including a family history of atopy. Laboratory investigations often reveal peripheral eosinophilia but it is not necessary for the diagnosis of EGE. In patients with ascites, paracentesis may reveal ascitic fluid eosinophilia. Stool examination should be considered to rule out evidence of parasitemia. CT in EGE may show nodular, irregular folds, and thickening of the stomach and intestinal walls. Gross endoscopic findings in mucosal type EGE may include mucosal hyperemia, ulcerations, and focal erosions [12]. The conclusive evidence is by the histopathological demonstration of eosinophils in the layers of GI walls.

Restricted diet and steroids are the mainstays of treatment of EGE. Other adjunct treatment modalities include mast cell inhibitors, leukotriene receptor antagonists, antihistamines, and proton-pump inhibitors [11]. Several novel anti-eosinophil therapies have been reported effective in the recent literature which includes neutralizing monoclonal antibodies against Interleukin-5, anti-tumor necrosis factor alpha, anti-immunoglobulin (IG) E monoclonal antibodies, selective T_H2 cytokine inhibitors, intravenous IG, and interferon- α [13-17].

In our case, the patient was extensively evaluated for surgical causes of an acute abdomen as well as for secondary eosinophilia which were both negative. The presence of highly elevated blood eosinophil levels prompted us to contemplate an unusual cause for her symptoms. The histopathological findings along with the response to steroids helped us to conclude it as a case of EGE along with subacute hypersensitivity pneumonitis, which explains the persistent cough. The clinical relevance of the case lies in the fact that EGE is a treatable condition and may mimic an acute abdomen which can be missed easily in ED if a high clinical suspicion is lacking. There are several reports which have shown that EGE can masquerade among the numerous causes of the acute abdomen such as acute appendicitis, intussusception, pancreatitis, and cholecystitis [18-21]. A study by Abassa *et al.* in 2017 reports that the incidence of EGE is often underestimated due to several factors such as non-specificity of EGE symptoms, insufficient data in the literature, low indices of clinical suspicion, and lack of

histopathological evidence [2]. Another study in 2019 by Sunkara *et al.* also reports that EGE is commonly underdiagnosed or underreported and that its prevalence is expected to increase [22]. In the above context, we believe that this case report will be an eye opener for all emergency physicians to consider this unusual, but emerging disease along with other differentials in undifferentiated cases of acute abdomen.

CONCLUSION

EGE is a rare disease affecting the GI system with symptoms ranging from abdominal pain and dyspepsia to obstruction and ascites. The diagnosis is confirmed by the presence of eosinophil infiltration of the walls of the GI tract. EGE shows an excellent response to steroids and adjunctive therapy. Due to its potential for underdiagnosis, the physician should exercise a high index of suspicion when the symptoms cannot be explained by other common causes.

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