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Case Report

A case report of eosinophilic enterocolitis

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ABSTRACT

A 13-year-old boy was admitted with complaints of abdominal pain and loose stools. Abdomen was distended. Laboratory tests on admission revealed neutrophilic leukocytosis and a peripheral smear done showed severe eosinophilia (33%) and leukocytosis. CT abdomen revealed ascites. Ascitic fluid tapping was done and 1.5 L of straw colored ascitic fluid drained. Ascitic fluid study was suggestive of high protein low SAAG ascites. The possibility of eosinophilic enterocolitis was considered. For confirmation endoscopy and sigmoidoscopy were done and a segmental biopsy was taken. Biopsy was suggestive of significant mucosal eosinophilia of duodenum, stomach, rectum, and eosinophilic abscess in the muscularis layer in the duodenum, stomach, and descending colon.

Keywords: Eosinophilic enterocolitis, Ascites, Eosinophilia, Biopsy

INTRODUCTION

Eosinophilic gastroenteritis is a rare disorder of unknown etiology that affects both adults and children. Although cases have been reported worldwide, the exact incidence of eosinophilic gastroenteritis is unclear.[1] It can present with a broad spectrum of symptoms; it may affect essentially any part of the gastrointestinal system and can involve any or all layers of the gastrointestinal wall. This disorder could be pathologically classified into three major subtypes, affecting predominantly the mucosal layer, muscle layer, or subserosal layer. Patients with serosal type frequently have ascites, which may lead to the wrong diagnosis of Eosinophilic Gastroenteritis due to unusual clinical manifestation.[2] Therefore, it is an easily omitted situation that needs more consciousness of the internists and gastroenterologists for diagnosis.

CASE REPORT

A 13-year-old boy was admitted with complaints of abdominal pain and loose stools. He had diffused abdominal pain for 3 weeks which was intermittent dull aching non radiating without any aggravating or relieving factors. He was treated in various local hospitals for the same and had symptomatic relief. He also gave a history of loose stool 2 days before admission to the hospital which was large volume watery without mucus and blood. No h/o malena/bleeding per rectum. Later he developed multiple episodes of vomiting, non-projectile and non-bilious. There was no history of fever, abdominal distension, jaundice, weight loss, dysphagia, or heartburn. He had a history of bronchial asthma.

Systemic examination

Gastrointestinal system

Abdomen was distended with tenderness over the epigastric and right lumbar region. There was no organomegaly. No other significant findings were noted. Laboratory tests on admission revealed neutrophilic leukocytosis and a repeat peripheral smear done after 3 days showed severe eosinophilia (33%) and leukocytosis. D-dimer was elevated (>10000 and serum LDH-335). Serum electrolytes, TSH, TropI, serum lactate, serum profile, coagulation studies liver function, and kidney function tests were normal. Stool examination was

negative for ova, parasites, and other pathogens. USG abdomen and CT abdomen done showed gross ascites and circumferential edematous wall thickening involving duodenum, jejunum and ileal loops, stomach pylorus, caecum transverse colon, and hepatic flexure along with mesenteric lymphadenitis. Ascitic fluid tapping was done and 1.5 L of straw colored ascitic fluid drained. Ascitic fluid study was suggestive of high protein low SAAG ascites. A possibility of eosinophilic enterocolitis was considered. For confirmation endoscopy and sigmoidoscopy was done and a segmental biopsy was taken. The report came as significant mucosal eosinophilia duodenum, stomach, rectum, and eosinophilic abscess in the muscularis layer in the duodenum, stomach, and descending colon.

Diagnosis was eosinophilic enterocolitis.

Treatment

The patient was started on IV Methylprednisolone 40 mg od, for 3 days, later he was switched over to oral steroids. Tab. Wyslone 30 mg once daily and continued for 2 weeks.

Follow up

The patient showed improvement. His abdominal distension reduced, appetite improved and diarrhea subsided, oral steroids were gradually tapered and stopped after one-month patient improved.

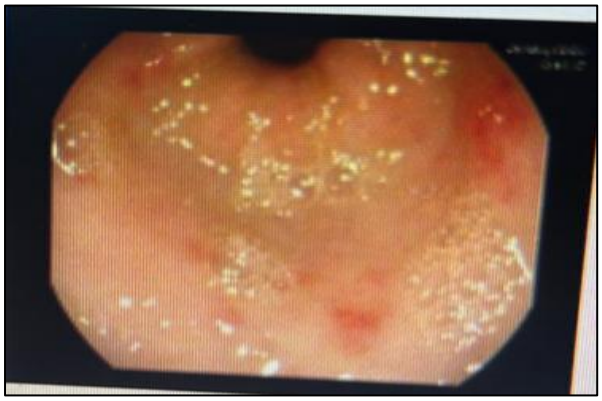

Figure 1: Antral erosions.

Figure 2: Ascitic fluid.

DISCUSSION

Eosinophilic gastrointestinal diseases (EGID) are chronic immune-mediated disorders characterized by a pathologic increase in eosinophil-predominant tissue inflammation and clinically by gastrointestinal symptoms.[3] In allergic patients with eosinophilic gastrointestinal disease, there will be differentiation of IL-5 and Th-2 cells leading to gut eosinophilia. Once eosinophils are recruited to the gastrointestinal tract they can persist through eosinophil-active cytokines such as IL-3, IL-5, and GCSF. Eosinophils can cause local inflammation by the release of eosinophils major basic proteins are most commonly involved areas body of stomach distal antrum and proximal small bowel.[4] The clinical features of eosinophilic gastrointestinal disease vary and are related to the location and extent of the organ involved as well as the layer of the bowel with eosinophilic infiltration.[5] The Mucosal disease presents as abdominal pain, vomiting, early satiety, and diarrhea. Muscular layer disease presents with features of intestinal obstruction and serosal disease presents with isolated ascites or ascites in combination with mucosal or muscular gastritis, enteritis, or colitis.[6,7]

Al Fadda et al study reported that Novel approaches to EGIDs focus on biologics, humanized monoclonal antibodies developed against targeted inflammatory mediators. Omalizumab, a recombinant, DNA-derived, humanized IgG1k monoclonal antibody, inhibits the binding of immunoglobulin E to the high-affinity IgE receptor, Fc epsilon R1 (FcεRI), and thus prevents an inadvertent anaphylactic reaction by limiting the degree of mediator release. Omalizumab when given every 2 weeks for up to 8 weeks reduces absolute eosinophil count after 3-4 months of therapy and provides symptomatic improvement in EGIDs. Mepolizumab, a humanized monoclonal antibody directed against IL-5, a key cytokine involved in the maturation, proliferation, and survival of eosinophils, shows promise; this antagonist at 750 mg IV every 2 weeks for 16 weeks significantly reduces tissue eosinophils in EE. Further clinical studies are needed to fully validate such experimental approaches.[8]

Kinoshita et al The diagnostic value of endoscopic examination findings varies among the different types of eosinophilic gastrointestinal diseases. For the diagnosis of eosinophilic esophagitis. These findings have been shown to be useful and provide important clues for the diagnosis of eosinophilic esophagitis. Longitudinal furrows, frequently found on the lower esophageal mucosal surface between longitudinal esophageal folds, are a characteristic finding noted in 90% of cases. Furthermore, white plaque and localized esophageal constriction, termed rings, are also frequently observed. These three findings of longitudinal furrows, white plaque, and the rings are considered important endoscopic findings for the detection of the eosinophilic esophagitis.[9]

CONCLUSION

Eosinophilic enterocolitis progresses from inflammation to a fibrostenotic state. Biopsies will give a confirmatory diagnosis. Earlier intervention improves quality of life and decreases morbidity. Hence it is desirable to consider eosinophilic enterocolitis in patients with allergies, isolated ascites, and diarrhea.

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