

## EOSINOPHILIC GASTROENTERITIS PRESENTING AS ACUTE SMALL INTESTINAL OBSTRUCTION: A CASE REPORT

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DOI: 10.5455/ijmsph.2014.250620142

Received Date: 13.05.2014

Accepted Date: 25.06.2014

### ABSTRACT

Eosinophilic gastroenteritis (EGE) is a rare disease of unknown etiology. It is characterized by eosinophilic infiltration of bowel wall to a variable extent and is associated with variable gastrointestinal (GI) symptoms. We elucidate here a case of eosinophilic gastroenteritis presenting as a small bowel obstruction in the post-operative period after groin surgery. A 66-year-old man was admitted in this hospital for bilateral direct inguinal hernia and bilateral hernioplasty was done. On the 7th post-operative day, he developed abdominal distension, abdominal pain, obstipation and vomiting. His abdomen was distended with diffuse tenderness and there was no guarding or rigidity. Abdominal radiograph revealed multiple air-fluid levels in small intestine. CT abdomen was taken which confirmed the distal small bowel obstruction and presence of ascites. An emergency laparotomy was performed and an area of induration in the ileum about 50 cm proximal to the ileo-cecal junction was noted. A segmental ileectomy was performed and primary end to end anastomosis was done. Histologically there was a dense transmural infiltration of eosinophils – so, eosinophilic enteritis was diagnosed. The patient recovered well. The present report has been made for the rarity of the lesion and its unusual presentation.

**Key Words:** Eosinophilic Enteritis; Small Bowel Obstruction; Eosinophilic Infiltration; Steroid Therapy

### Introduction

Eosinophilic gastroenteritis is an extremely rare disease which can affect any part of gastrointestinal tract. It is characterized by diffuse or patchy eosinophilic infiltration involving single or multiple layers of the gastrointestinal tract.<sup>[1]</sup> The pathogenesis and etiology of eosinophilic gastroenteritis remain unclear. Diagnostic criteria include the demonstration of eosinophilic infiltration in the bowel wall, lack of evidence of extra intestinal disease and exclusion of various disorders that mimic a similar condition.<sup>[1]</sup> Short courses of corticosteroids are the mainstay of treatment, although some patients with relapsing disease require long-term low-dose steroids.<sup>[2]</sup> Surgery in eosinophilic gastroenteritis should be reserved for complications like obstruction or perforation.

### Case Report

A 66-year-old man was admitted in our hospital for bilateral direct inguinal hernia and bilateral hernioplasty was done. On the 7<sup>th</sup> post-operative day, he developed abdominal distension, abdominal pain, obstipation and vomiting. His abdomen was distended with diffuse tenderness and there was no guarding rigidity. Laboratory investigation showed a white cell count of 9800/mm<sup>3</sup> with 3% eosinophils. Other laboratory studies were within normal limits. Abdominal

radiograph displayed multiple air-fluid levels in the small intestine (Figure 1). Computed tomography demonstrated dilatation of the small intestine and ascites (Figure 2) and there was no clear transition zone or mass. With the patient's abdominal distension and pain progressively worsening, emergency laparotomy was performed through a midline incision. Exploration revealed a small amount of yellowish ascites in the peritoneal cavity and focal stricture in the ileum about 50 cm proximal to the ileo-cecal valve (Figure 3). The remaining proximal small bowel was dilated. Further inspection of the peritoneal cavity did not find any other abnormality. The stricture was resected with proximal and distal margins of 10 cm each, and end to end anastomosis was performed. The mucosa of the specimen was edematous, but there was no inflammation (Figure 4). Histologically, there was a dense infiltration of eosinophils throughout the entire thickness of ileal wall (Figure 5). Postoperatively, the patient's stool was negative for ova and parasites and skin prick test was negative. The patient was recovered well and was free from gastrointestinal symptoms at the time when we reported his disease (Figure 6).

### Discussion

Eosinophilic gastroenteritis (EGE) is a rare and poorly understood condition presenting with variable symptoms.



Figure-1: Erect abdominal X-ray showing multiple air-fluid level in the small intestine

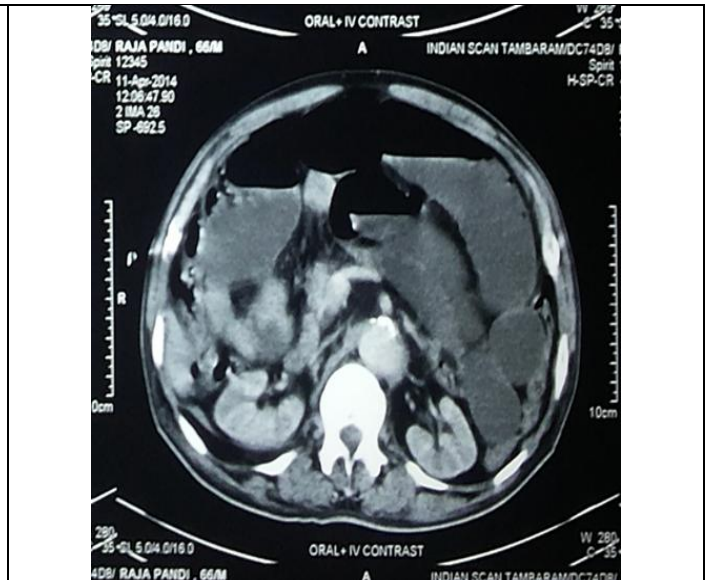


Figure-2: CT Abdomen showing dilated small bowel with ascites

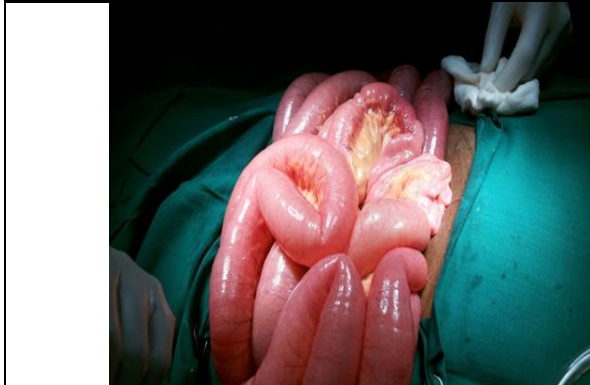


Figure-3: Intraoperative findings. Stricture lesion and dilated proximal small bowel



Figure-4: Resected specimen showing stricture lesion

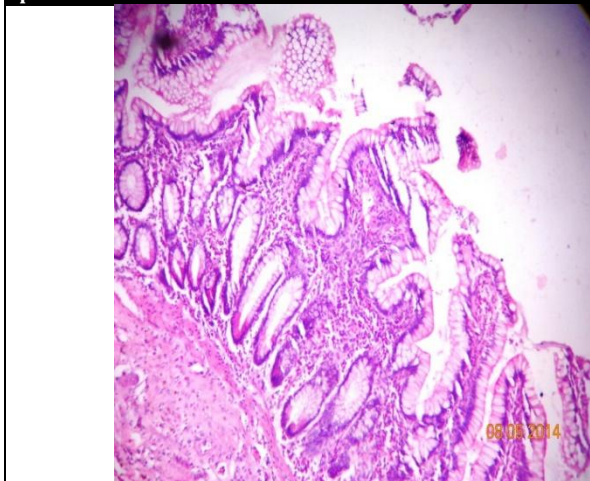


Figure-5: Microscopy of the resected ileum revealing eosinophilic infiltration



Figure-6: post-operative status

Eosinophilic gastrointestinal disorders may be primary or secondary in nature. Primary eosinophilic gastroenteritis is defined as a disorder that primarily affects gastrointestinal with eosinophilic inflammation in the absence of known causes for eosinophilia including drug reaction, parasitic infections and malignancy.<sup>[2]</sup> EGE

can occur secondarily in GI tract in inflammatory bowel disease, autoimmune disease, drugs, parasitic infections, hyper-eosinophilic syndrome and after solid organ transplantation.

Most of the cases reported in the literature are in whites

with some cases in Asians. Though it usually occurs in the third to fifth decade of life, it can affect any age group – from infancy through the seventh decade. A slight male predominance has been reported.<sup>[2-4]</sup>

Although the exact etiology is unknown, an allergic disorder is present in 50% of the patient. Recently, the role of cytokines IL-5, IL-4 and gamma-interferon has been described.<sup>[2]</sup> More than 80% of patients are associated with an elevated peripheral eosinophil count.<sup>[5]</sup> Although EGE can occur in any part of gastrointestinal (GI) tract, stomach and small intestine are the most common sites of involvement. Rarely colon, oesophagus, appendix, pancreatic cases are also reported in literature.<sup>[6]</sup>

This disorder was originally described in 1937 by Kaijser.<sup>[1,4,6]</sup> In 1970, Klein et al defined three patterns of disease manifestation related to the depth of the disease process involving of GI tract.<sup>[2,6]</sup>

- (i) The mucosal form (25-100%) of EGE is the most common one, characterized by vomiting, abdominal pain, diarrhoea, blood loss in stool, iron-deficiency anemia, malabsorption and protein-losing enteropathy.
- (ii) The muscular form (13-70%) is characterized by infiltration of eosinophils predominantly in muscle layer, leading to thickening of the bowel wall, which results in GI obstructive symptoms.
- (iii) The serosal form (12-40%) is least common and affects minority of patients, characterized by exudative ascites with elevated peripheral eosinophil count compared to other forms.<sup>[2,5]</sup>

Extra-intestinal manifestations such as eosinophilic cystitis, eosinophilic splenitis and hepatitis, obstructive jaundice have been described along with.<sup>[6]</sup> Hitherto, there are no existing standard criteria for the diagnosis of EGE. The most widely used criteria have been formulated by Talley et al.<sup>[6,7]</sup>

Talley et al have identified three main diagnostic criteria;

- (i) The presence of gastrointestinal symptoms
- (ii) Biopsies demonstrating eosinophilic infiltration of one or more areas of GI tract
- (iii) No evidence of parasitic or extra-intestinal disease

It is agreed that peripheral eosinophilia is uniformly associated with eosinophilic gastroenteritis. However, the definitive diagnosis of eosinophilic gastroenteritis requires histologic evidence of eosinophilic infiltration. The diagnosis of EGE is confirmed by biopsies that reveal

>20 eosinophils per high-power field on microscopic examination.<sup>[4-6]</sup>

In the present case, eosinophils infiltrated all three layers of the intestinal wall. Thickening of the muscle layers narrowed the intestinal lumen, causing intestinal obstruction, and serosal involvement produced ascites without peripheral eosinophilia.

Hyper-eosinophilia in the peripheral blood is usually absent in at least 20 % of the cases. Therefore, the absence of hyper-eosinophilia should not exclude consideration of the diagnosis of eosinophilic gastroenteritis in patients with unexplained GI symptoms.<sup>[4]</sup>

Radiologically, the hallmark of eosinophilic gastroenteritis on CT is nodular and irregular thickening of the folds in the distal stomach and proximal small bowel. However, similar thickening may also be seen in Menetrier's disease, lymphoma, scirrhous carcinoma, Crohn's disease, and granulomatous disease. It is, thus, not a specific sign of eosinophil gastroenteritis. Mesenteric inflammation as well as ascites are not uncommon, but are still nonspecific.<sup>[4,5]</sup>

Ultrasonography is a useful tool for detecting non-mucosal eosinophilic gastroenteritis in patients without peripheral hyper-eosinophilia. It may reveal generalized thickening of the intestinal wall as well as ascites. Sonography could also evaluate the response to treatment by measuring the thickness of the affected layer.<sup>[4]</sup>

The endoscopic appearances are non-specific, including erythematous, friable, nodular and occasional ulcerative changes. Eosinophilic infiltrates are usually patchy in distribution and may be present in otherwise normal, non-inflamed bowel wall. Therefore, multiple biopsies maybe required to avoid missing the diagnosis. Several different examinations, such as gastro-duodenoscopy and colonoscopy, and multiple deep biopsies may be necessary to establish the diagnosis. Even then, it may be difficult to evaluate accurately the degree and extent of disease in most patients, given the patchy distribution of the infiltrates.<sup>[4]</sup> A new technique using Tc-99m HMPAO labelled WBC SPECT may be useful in assessing the extent of disease and response to treatment.<sup>[4,5]</sup>

There have been no randomized prospective clinical trials regarding therapy. The treatment is empirical –

based on severity of symptoms. The role of modification of diet is controversial. [6] Oral glucocorticoids with anti-inflammatory properties are the primary therapy, especially for patient with mucosal/ serosal form and rarely for muscular disease. Most patients with eosinophilic gastroenteritis respond dramatically to oral prednisolone, recommended daily. Improvement occurs in 2 weeks regardless of the site of bowel involved. If symptoms recur, a low dose maintenance therapy with prednisolone is required.[4,6]

Successful treatment with other anti-inflammatory medications, such as leukotriene modifiers (montelukast) and mast cell stabilizers (cromolyn), anti-allergic drugs (budesonide) has been reported. Based on IL -5 involved in pathogenesis of EGE, therapeutics targeting IL-5 are used for trials in patients with EGE. The first reported patient with eosinophilic esophagitis treated with the humanized anti-IL-5 antibody, mepolizumab was an 18-year old male with long history of dysphagia, persistent vomiting, and severe stricture on endoscopy who failed to respond to steroids. In response to this treatment, there was diminished gross inflammation and stricture on endoscopy with improvement in symptoms like vomiting and difficulty in intake of solid food. Large randomized trials are needed to further clarify the efficacy and safety of this therapy and to clarify its role in the long -term management of patients with EGE.[2]

Surgical treatment is required only for complicated cases like intestinal obstruction, perforation or when

performing a full thickness intestinal biopsy to establish the diagnosis.[4,6]

The mortality related to EGE itself is rare. However, the morbidity due to profound weight loss, intestinal obstruction and peritonitis can occur. It does not predispose to GI malignancy. The long term prognosis for this condition is good.[6]

## Conclusion

EGE is a diagnostic dilemma and the clinical presentation and investigations are only contributory. So, surgeons must be aware of this condition as a rare cause of acute abdomen.

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**Cite this article as:** Rajkumar Bharani K, Anbarasu S, Essaki M, Padmavathy A. Eosinophilic gastroenteritis presenting as acute smallintestinal obstruction: A case report. Int J Med Sci Public Health 2014;3:1178-1181.

**Source of Support:** Nil

**Conflict of interest:** None declared