Predominantly muscular eosinophilic gastroenteritis with ascites: A Case Report and Review of the Literature

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Citation

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Abstract

Eosinophilic gastroenteritis is a rare pathologic entity characterised by eosinophilic infiltration of the wall of the gastrointestinal tract and manifests various dysfunctions of GI tract. Diagnosis requires a high index of suspicion and exclusion of other disorders that are associated with peripheral eosinophilia. We report a case of a patient, who presented with abdominal pain and ascites and on diagnostic work up turned out to be a case of eosinophilic gastroenteritis with predominantly muscular propria type of eosinophilic infiltration. Presentation with ascites is very rare with such type. Our patient was successfully treated with corticosteroid administration.

INTRODUCTION

Eosinophilic gastroenteritis, first reported by Kaijser [1] in 1937, is a rare gastrointestinal disorder that can present with various gastrointestinal manifestations, depending on the specific site of the affected gastrointestinal wall. Klein et al[2] have demonstrated that this disorder could be pathologically classified into three major types: predominant mucosal layer, predominant muscle layer, and predominant subserosal layer. However, its clinical features, aetiology, and treatment have not yet been definitely established. We report a case of eosinophilic gastroenteritis with features of the predominant muscle layer with ascites, abdominal distension, diarrhoea and high grade fever which is rare because ascites and muscular predominant eosinophilic gastroenteritis is not well reported.

CASE REPORT

A 45 year male Muslim patient presented with complains of abdominal distension, occasional vomiting, high grade intermittent fever and increased frequency of stool for one month. He did not have any significant history of other illness. He had mixed diet with normal appetite and no allergy to any food items. He had no positive findings on the general examination. In abdominal examination abdomen was distended, soft, no tenderness, no guarding and no rigidity, liver and spleen were not palpable, s/o intra abdominal free fluid were present. On per rectal examination fullness was appreciated. He was evaluated with CBC, in which total counts rose to 15291/cmm, absolute eosinophilic counts were 8410/cmm that is 55% of total WBC counts. His urine analysis, renal functions and liver functions were normal. His X ray chest showed right pleural effusion and abdomen standing showed ground glass appearance with few air fluid bowel loops. His ultrasound abdomen and thorax s/o moderate ascites and thickened bowel loops. His CT scan confirmed the USG findings and additionally showed diffuse thickened mucosal folds in non dilated almost entire small bowel loops. Diagnostic ascitic tapping was done which was reddish yellow turbid fluid; TC was 8060cells/cmm in which polymorphs 90% and lymphocytes 10%. Smear with gram stain and Z-N stain were negative. Ascitic fluid culture was negative. Ascitic fluid Adenosine De Aminase was negative. On cytological examination no malignant cells were seen. He had undergone upper GI scopy and sigmoidoscopy to rule out infiltrative disease and storage disease which were not conclusive on histopathology. Then it was decided to take full thickness biopsy from the bowel, peritoneum and from the liver through diagnostic laparoscopy. On laparoscopy there was moderate ascites with diffusely dilated small bowel. Biopsies were taken from liver, peritoneum and jejunum. On histopathological examination liver tissue found normal, peritoneal biopsy showed infiltration of the tissue by chronic inflammatory cells along with many eosinophils; jejunal biopsy consists chiefly of the muscularis propria which is infiltrated by many eosinophils. Thus his diagnosed was confirmed of eosinophilic gastroenteritis. He was prescribed prednisolone 20mg once a day for 6 weeks and then tapered off. His ascites was

relieved within 15 days, clinical improvement noted within one month and he was followed for one year after stoppage of the drug and he did not show any symptoms.

DISCUSSION

Eosinophilic gastroenteritis was first reported by Kaijser [1] in 1937. In 1970, Klein [2] classified the disease according to the predominance of eosinophilic infiltration in different layers of the intestinal wall. Talley et al [3] have identified four main diagnostic criteria: (1) the presence of gastrointestinal symptoms, (2) biopsies demonstrating eosinophilic infiltration of one or more areas of the gastrointestinal track, (3) absence of eosinophilic involvement of multiple organs outside the GI tract and (4) no evidence of parasitic or extra intestinal disease. Disease is rare, undiagnosed and surely underreported. [2,3] Patients typically present in third through fifth decades of the life, but the disease can affect any age group. An equal gender distribution or a slight male preponderance has been reported. [3] The cause is unknown and pathogenesis is poorly understood. Any segment of GI tract may be involved, including the oesophagus [5,6] or colon [7], but most commonly the stomach or small bowel is affected. Classification based on the layer of gut wall primarily affected. Most prevalent form is characterised by mucosal (and submucosal) disease. [2,3,4] Predominant mucosal layer disease present with pain, nausea, vomiting, diarrhoea, weight loss, iron deficiency anaemia, malabsorption, protein loosing enteropathy, while predominantly muscle layer disease present with the obstructive features. Rarest form of all is the serosal disease; all layers of the bowel are usually involved, and patients present with typical ascites. [2,3,8] Only rarely mucosal involvement is absent when serosal disease is present. [8] However, the definitive diagnosis of eosinophilic gastroenteritis requires histological evidence of eosinophilic infiltration. Blood investigation shows peripheral eosinophilia in about 80% cases; the absolute eosinophil count averages 2000 cells/µL in patients with mucosal disease and 1000cells/µL in patients with disease of muscle layer, although count may fluctuate markedly over time. [3] Other findings include iron deficiency anaemia, hypoalbuminemia. Radiological changes found in eosinophilic gastroenteritis are variable, nonspecific, and absent in at least 40% of patients. [9,10] The gastric folds are enlarged, with or without nodular filling defects. CT may demonstrate thickened intestinal wall and localised mesenteric lymphadenopathy; with serosal involvement, ascitic fluid usually is detected. When subserosal disease involves the small bowel, biopsy of the mucosal layer taken during gastroscopy or colonoscopy often fails to diagnose eosinophilic gastroenteritis. Laparotomy or laparoscopic full thickness biopsy of bowel is often required for confirmation in such case. On histopathology it is characterised by oedema and an inflammatory cell infiltrate that is almost entirely composed of eosinophils; the eosinophils may occur in clumps. [11] Treatment with a steroid is the mainstay in the management of eosinophilic gastroenteritis. Other modality includes diet modification, antihalminthic, mast cell stabilisers (sodium chromoglycate). Surgical intervention is required only when a definitive diagnosis cannot be made or when the complications like obstruction, perforation or bleeding occurs. Because the natural history of eosinophilic gastroenteritis has not been well documented, long-term follow-up is required. We have followed our patient for one year following the treatment.

Figure 1

Figure 1: Eosinophilic clumps in muscle layer

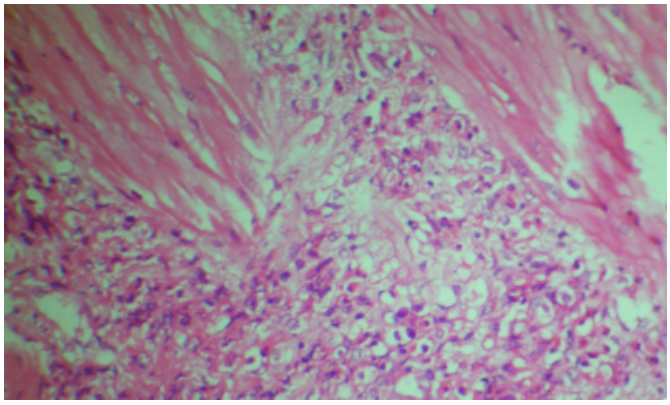


Figure 2

Figure 2: Predominantly muscular eosinophilia

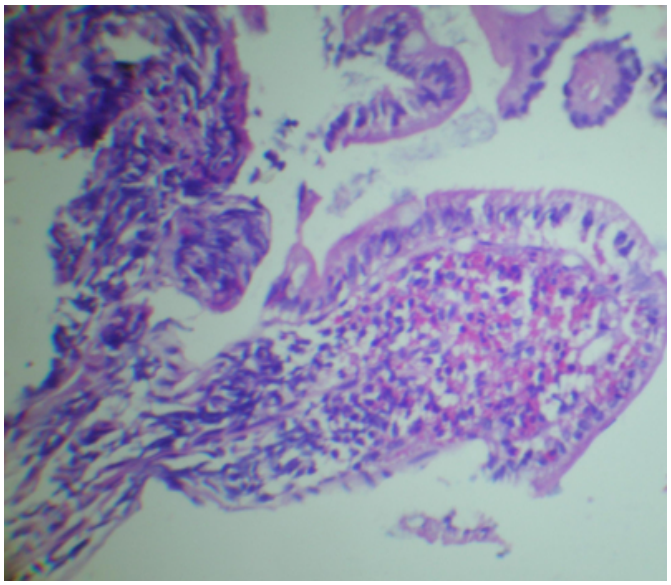
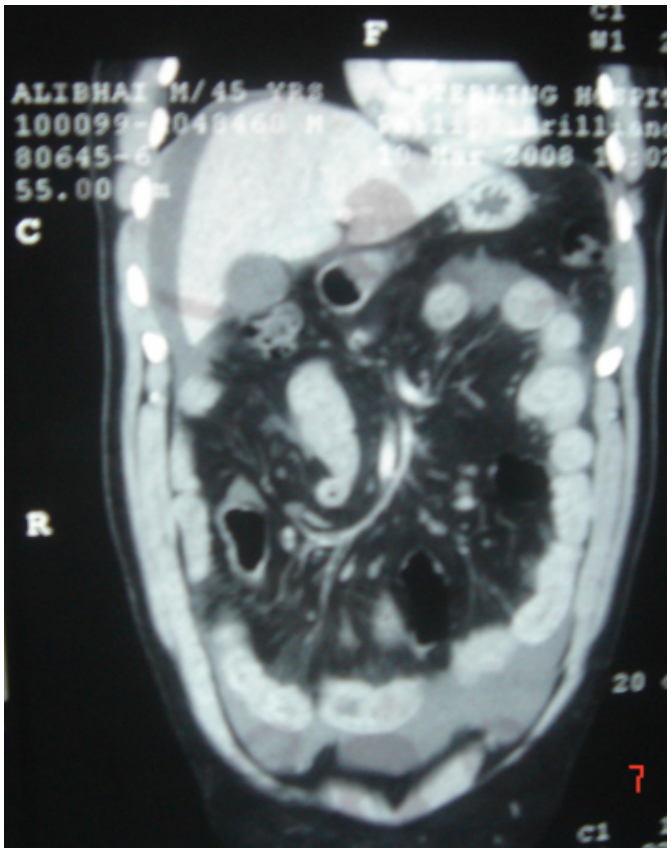


Figure 3

Figure 3: Ascites with generalised thickness of the small bowel



AUTHORS CONTRIBUTIONS

Sudhanshu I. Patwari, Mahendra S. Bhavsar managed the patient , Rahul P. Naik prepared the manuscript and figures; Vismit P. Joshipura and Nitin R. Patel revised the manuscript.

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