EOSINOPHILIC GASTROENTERITIS

(A Report of Two Cases)

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Introduction

Infiltration of the gastrointestinal tract with eosinophils in association with recognisable clinical disease is rare. Since first described in 1937 by Kaijser [1], this entity has been reported as isolated case report or small case series [2]. Eosinophilic gastro enteritis (EGE) should be suspected in presence of unexplained chronic or relapsing gastrointestinal symptoms. Even though the pathogenesis of this disease is obscure, the diagnosis should not be missed, as anecdotally steroid therapy may be effective [2]. We report two cases of eosinophilic enteritis diagnosed in relatively short period of one year.

Case Report

Case 1 : A 29-year-old male patient presented with 3 months history of pain epigastric region, anorexia and weight loss of 6 kg. He also complained of intermittent vomiting, constipation and had two episodes of melaena each lasting for 10-12 days, associated with giddiness and postural symptoms. There was no history of fever, diarrhoea or abdominal distention. The only positive clinical finding was a 2 cm hepatomegaly. All investigations including biochemical profile, total and differential leucocyte count, upper gastrointestinal endoscopy (UGIE), colonoscopy and ultra sound (US) abdomen revealed no abnormality. Barium meal follow through (BMFT) was suggestive of multiple small intestinal strictures. Laparotomy revealed oedema and thickening of entire small intestine with two skip areas of 15-20 cm of normal gut. In addition mesenteric lymph nodes were enlarged. There was no ascites. A full thickness biopsy of the ileum revealed oedematous lamina propria with broad villi. Blood vessels were congested. There was evidence of lymphoplasmacytic and eosinophilic infiltration upto the muscular layer with eosinophil predominance (Fig 1). The mesenteric lymph node also showed eosinophilic infil-

tration. Patient was treated with steroids for 3 months and has shown a good response in that he is asymptomatic and has gained 4 kg body weight.

Case 2 : A 32-year-old female patient presented with one year history of intermittent epigastric pain, vague dyspeptic symptoms, weight loss and small bowel diarrhoea. She had been treated for pulmonary tuberculosis 4 years back. Clinical, biochemical examination and investigations including UGIE, BMFT, US abdomen did not reveal any significant abnormality except for an 8 mm solitary gall stone. The blood eosinophilic count was normal. At laparotomy, small intestine was grossly normal, but there were few enlarged mesenteric lymph nodes. Full thickness jejunal biopsy showed oedematous lamina propria. There was lymphoplasmacytic and eosinophilic infiltration of the mucosa and the muscular layer, the predominant cell being eosinophils. The serosa was not infiltrated. Mesenteric lymph nodes also showed eosinophilic infiltration. She was treated with steroids for 3 months and has shown a good response.

Fig. 1 : Shows eosinophilic infiltrate extending from mucosa to muscular layer (X-100)

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Discussion

EGE is defined as presence of gastrointestinal symptoms associated with eosinophilic infiltration of the gut with no evidence of extra intestinal disease or parasitic infection [3]. Peripheral eosinophilia is not a universal finding and occurs in 20-90 per cent cases and is not included as diagnostic criteria [4]. In fact both our patients did not have peripheral eosinophilia. The Klein classification, where the patients are divided into those with predominantly mucosal, muscle layer or subserosal disease is widely used [5]. Talley et al [2] in a study of 40 patients of EGE found muscular disease in only 30 per cent of their patients. In their study the commonest form of disease was mucosal disease. They found that mucosal and muscle layer disease could be confused with functional bowel disease. Venkataraman et al [6] found abdominal pain, weight loss, diarrhoea and abdominal distention as the commonest presenting symptoms. In our study both the patients had muscle layer disease confirmed by mini laparotomy and full thickness intestinal biopsy. Abdominal pain and weight loss was present in both cases, while one had diarrhoea none had abdominal distention.

The pathogenesis of EGE is not understood; perhaps an excessive accumulation of eosinophils causes destruction of intestinal epithelium caused by the release of eosinophilic major basic protein, as has been found in the respiratory epithelium in asthma [7]. Steroids remain the mainstay of therapy for EGE with good symptomatic response be-

ing reported [2,4,5] and similar result was found in our cases. Relapses are common [6] but we have not had a relapse in our cases so far.

EGE patients present with vague gastrointestinal symptoms and are diagnosed and treated as cases of non ulcer dyspepsia or irritable bowel syndrome till they develop weight loss and other features of Malabsorption syndrome. These patients can be diagnosed early with high index of suspicion and prompt efforts at specific diagnosis. Reward of early diagnosis is almost complete response to steroid therapy.

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