

# A case of eosinophilic gastroenteritis

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**Eosinophilic gastroenteritis is a rare disorder characterised by eosinophilic infiltration of the bowel wall and various gastrointestinal manifestations. Diagnosis requires a high index of suspicion and exclusion of various disorders that are associated with peripheral eosinophilia. We report on a woman who had a short history of abdominal pain and ascites, and who responded dramatically to a course of low-dose steroid.**

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## Introduction

Eosinophilic gastroenteritis (EG) is a rare gastrointestinal disorder that can present with various gastrointestinal manifestations, depending on the specific site of affected gastrointestinal tract and specific layer of affected gastrointestinal wall. The majority of reported cases involve the stomach and proximal small bowel. The pathogenesis and aetiology of EG remain unclear. Diagnostic criteria include demonstration of eosinophilic infiltration of the bowel wall, lack of evidence of extra-intestinal disease, and exclusion of various disorders that could mimic a similar condition. One needs to consider this rare disease during the differential diagnosis of unexplained gastrointestinal symptoms, especially when they are associated with peripheral eosinophilia.

## Case report

In August 1994, a 34-year-old woman presented with mild upper abdominal pain and, 2 weeks later, with abdominal distension, frequency of bowel motion, and tenesmus. She did not give a past history of ulcer pain and had not had any abdominal operations; there had been no recent weight loss and no blood or mucus was present in the stool. The patient denied taking any drugs or herbal medicines. There was no history of drug allergy, asthma, or allergic rhinitis, except for occasional skin eczema. Abdominal examination

showed a soft, mildly tender abdomen, and presented as shifting dullness.

The patient was admitted to St Teresa's Hospital in August 1994. Gastroscopy showed only mild antral gastritis; no stomach or duodenal ulcers were present. Colonoscopy showed diverticula in the caecum and ascending colon, mild proctitis, and sigmoid colitis. Biopsy of the gastric antrum and rectum revealed non-specific inflammation. Computed tomography of the abdomen showed a moderate amount of ascites. The liver, spleen, and retroperitoneum were normal. Ultrasonography of the pelvis revealed the presence of small bilateral ovarian cysts. The haemoglobin level was 137 g/L (normal range, female, 115-155 g/L) and the white cell count was  $22.2 \times 10^9$  /L, with 40% neutrophils, 10% lymphocytes, 1% monocytes, and 47% eosinophils. The platelet count was  $403 \times 10^9$  /L and the erythrocyte sedimentation rate was 2 mm/hr. The tests for antinuclear factor, rheumatoid factor, and serum hepatitis B surface antigen gave negative results; however, antibody to hepatitis B surface antigen was present. The creatine phosphokinase level was 21 U/mL (normal range, 10-70 U/mL) and the lactate dehydrogenase level was 143 U/L (normal range, 50-150 U/L). The anti-amoebic titre was negative and stool microscopy for ova and cysts was negative.

Laparotomy was performed to establish the diagnosis and to rule out any ovarian malignancy. Operative findings included 1600 mL of turbid ascitic fluid; microscopy of the fluid showed abundant white cell counts which were predominantly eosinophils. Cytology, culture, and smear tests for acid-fast bacilli in the fluid gave negative results, however. There was marked inflammation at the gastric antrum and

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proximal duodenum, and biopsy of these inflamed areas showed inflammation with marked eosinophilic infiltration at the subserosal and muscular wall. Bilateral ovarian cysts were found and their biopsy showed them to be follicular cysts. In addition, slough and clots were found in the pouch of Douglas and their histological examination revealed an organising blood clot with the presence of abundant eosinophils and mesothelial cells.

After excluding the possibilities of malignancy, parasitic disease, and autoimmune disease, EG was diagnosed. This diagnosis was based on the presence of peripheral eosinophilia and eosinophilic ascites, and the eosinophilic infiltration of the serosa and muscle wall of the gastric antrum and proximal duodenum. Postoperatively, there was a persistence of peripheral eosinophilia and continuous drainage of ascites; thus, a small daily dose of steroid (prednisolone 10 mg) was given. There was a marked improvement: the eosinophil count normalised and ascitic fluid production immediately decreased. Steroid treatment was gradually reduced and eventually terminated after 6 weeks. During the follow-up period of 1 year, the patient was completely free of recurrence.

## Discussion

The important feature in this case of EG is the extremely high eosinophil count in the peripheral blood, ascitic fluid, and the subserosal and muscular wall of the gastric antrum and proximal duodenum. The diagnosis of EG was confirmed after the exclusion of other disorders that have such similar features as gut lymphoma, parasitic infection, carcinoma, inflammatory bowel disease, and allergy.<sup>1</sup>

Eosinophilic gastroenteritis is a rare disease and was first described in 1937.<sup>2</sup> In 1970, Klein classified the disease according to the predominance of eosinophilic infiltration in different layers of the intestinal wall—namely, the mucosal, muscle, and subserosal layers.<sup>3</sup> The involvement of different layers usually gives rise to different clinical manifestations. Mucosal disease generally presents with bleeding, protein-losing enteropathy, or malabsorption. Involvement of the muscle layer may cause bowel wall thickening and subsequent intestinal obstruction. The subserosal form usually presents with eosinophilic ascites, which was the manifestation in this patient.

Eosinophilic gastroenteritis can involve any part of the gastrointestinal tract from the oesophagus down to the rectum. The stomach and duodenum, however,

are the most common sites of involvement.<sup>4-7</sup> The pathogenesis and aetiology of the disease are not well understood. There is evidence to suggest that a hypersensitivity reaction may play a role.<sup>8</sup> The presence of peripheral eosinophilia, abundant eosinophils in the gastrointestinal tract, and a dramatic response to corticosteroid provide some support that the disease is mediated by a hypersensitivity-type reaction.<sup>8</sup> Moreover, a study at Mayo Clinic showed that 50% of patients with EG give a history of allergy such as asthma, allergic rhinitis, urticaria, drug allergy, and eczema.<sup>8</sup>

If EG is considered as a form of hypersensitivity reaction, the exact nature of the allergen requires investigation. Food, as a natural allergen, is considered to be associated with EG.<sup>9</sup> Nevertheless, clinical studies have shown no relationship between food allergy and EG.<sup>9</sup> A study of a patient with EG over a 2.5-year period showed that when the gut was challenged orally with different food allergens, the appropriate gastrointestinal symptoms of EG could be reproduced. During the challenges, however, there was no demonstration of any of the associated changes or eosinophilic infiltration in the jejunal biopsies.<sup>9</sup> This observation explains why although elimination diets often help to treat a food allergy, they have no role in the treatment of EG.

When there is a subserosal disease, as in this patient, biopsy of the mucosal layer (taken during gastroscopy) often fails to diagnose EG. Laparotomy or laparoscopy is often required to make a diagnosis in such cases. It must be emphasised that in the mucosal form of the disease, multiple biopsies must be taken during endoscopy, because mucosal involvement is often patchy in nature.

Treatment with a steroid is the mainstay in the management of EG. Dramatic clinical improvement is seen after treatment with a low dose of steroid. The duration of treatment is controversial, however. This patient was given prednisolone 10 mg daily for 6 weeks and responded favourably. Surgical intervention may sometimes be required for patients with obstruction complications or when performing a full thickness intestinal biopsy to establish the diagnosis. Using an elimination diet, as discussed, has no role in the therapy of EG. The long-term prognosis for this condition is good.

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