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Case report

Eosinophilic gastroenteritis presenting as tense ascites and ileal subobstruction

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SUMMARY

Eosinophilic gastroenteritis (EGE) is a rare condition of unknown aetiology characterized by peripheral eosinophilia, eosinophilic infiltration of the gastrointestinal tract, and abnormalities of gastrointestinal function. We recently treated a young man affected by eosinophilic infiltration extending from his stomach to his ileum and presenting as tense eosinophilic ascites and ileal subobstruction. We diagnosed him as a case of a diffuse infiltrative type of eosinophilic gastroenteritis with subserosal involvement, essential to obtain eosinophilic ascites. When given oral steroids, the patient's condition improved and he was discharged without symptoms. This report adds to the scarce data on sub-serosal involvement, the rarest form of presentation, and illustrates that EGE complicated by ascites can be effectively treated with steroids after other systemic disorders associated with peripheral eosinophilia have been ruled out.

Key words: Eosinophilic gastroenteritis, ascites, abdominal pain

INTRODUCTION

Eosinophilic gastroenteritis is a rare condition. Since its first description in 1937 by Kaijser[1] fewer than 300 cases have been reported[2]. Its cause is unknown. Although it was long believed to be related to food allergy, it is now clear that only about half the patients have a history of allergy or atopy[2,3].

The disease can affect any part of the gastrointestinal tract from the esophagus to the colon, although it affects the gastric antrum and the proximal small bowel most commonly[4,5]. Whether the reported prevalence of the disease in the gastric antrum reflects its biologic nature or merely the more frequent use of gastroscopic evaluation to make the diagnosis is unclear[2]. We report on a patient presenting with tense eosinophilic ascites and abdominal pain who responded promptly after steroid therapy was started.

CASE REPORT

The patient, a 22-year-old man was referred to our hospital because of diffuse and recurrent attacks of vague abdominal discomfort and slight abdominal distention for about 2 months. His past history included an appendectomy 18 months before admission and an episode of diarrhea one month earlier from which he had recovered after symptomatic treatment. The patient did not suffer from anorexia, weight loss, nausea or vomiting, and food allergy and atopy were ruled out by anamnesis.

When examined on admission, the physical condition of the patient was unremarkable, except for diffuse and soft abdominal tenderness and signs of ascites. Routine laboratory tests revealed a white blood cell count of 13.6×10^9/l (43% neutrophils, 17% lymphocytes, 5% monocytes and 35% eosinophils). Hemoglobin levels, erythrocyte sedimentation rate, electrolyte levels, coagulation test results, measures of liver and renal function, total protein count, urinary sediment and levels of vitamin B12 and folic acid were all in the normal range. Level

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of serum immunoglobulins and complement was normal and findings were negative for antinuclear antibodies (ANAs), smooth muscle antibodies (SMAs), rheumatoid factor, Mantoux reaction, serological markers for hepatitis B and C. Repeated stool samples were negative for bacteria and parasites. Serological search for Ecchinococcus, Filaria, Toxocara and Schistosoma remained negative. To rule out a malignant process, a bone marrow biopsy was performed, which showed infiltration by eosinophils.

Chest x-ray showed no pathological lung images. Upper gastrointestinal endoscopy revealed thickened gastric folds and bulboduodenitis. Endoscopic biopsies of stomach (Figure 1) and duodenum (Figure 2) showed a rich inflammatory infiltrate mainly consisting of eosinophils and lymphoplasmocytic cells. A colonoscopy was normal and mucosal biopsies revealed no abnormalities.

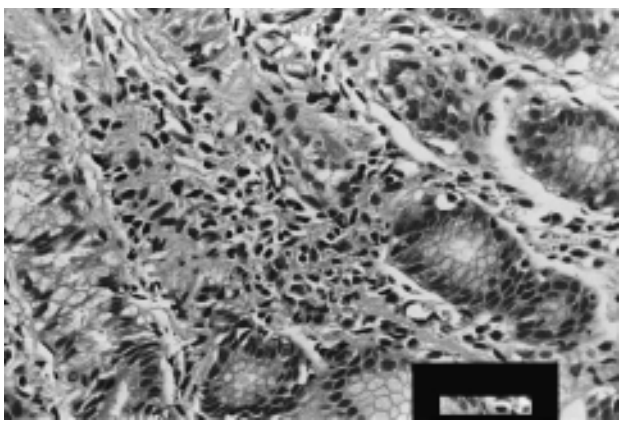

Figure 1. Photomicrograph from gastric antrum showing eosinophilic infiltration of the lamina propria (H/E 400x).

Abdominal ultrasonography and CAT scan revealed ascites. Paracentesis yielded 500ml of turbid fluid. Ascitic fluid contained 78g/l protein and had a leucocyte count of $5.2x10^9$/l (95% eosinophils) (Figure 3). Levels of glucose, amylase and lactate dehydrogenase in the patient's peritoneal fluid were normal, and the results of culture, cytological analysis and Gram staining of his peritoneal fluid were negative.

An upper gastrointestinal series with a small-bowel study showed normal folds in the proximal small bowel; segments in the distal small intestine had thickened folds; loops of small bowel were thickened, suggestive of mural thickening; a normal cecum was opacified.

A regimen of prednisone (20mg/day) was instituted. The dose of steroid was slowly tapered off over the following 2 months. Twelve months after his initial presentation, the patient is asymptomatic with a normal absolute eosinophilia.

DISCUSSION

EGE is characterized by peripheral eosinophilia and infiltration of the gastrointestinal tract by eosinophils, which may lead to a wide array of symptoms. There is a slight preponderance of the condition among men (m:f = 3:2) and the disease seems to have a bimodal age distribution with peaks in the third and sixth decade[6]. EGE typically involves the stomach and proximal small bowel but any area of the gastrointestinal tract may be involved[2,6]. Eighty percent of patients have symptoms for 1 to 25 years.

The pathogenesis of EGE is poorly understood. Eosinophils contain several proteins, such as major basic protein, eosinophil-derived neurotoxin, eosinophil cationic

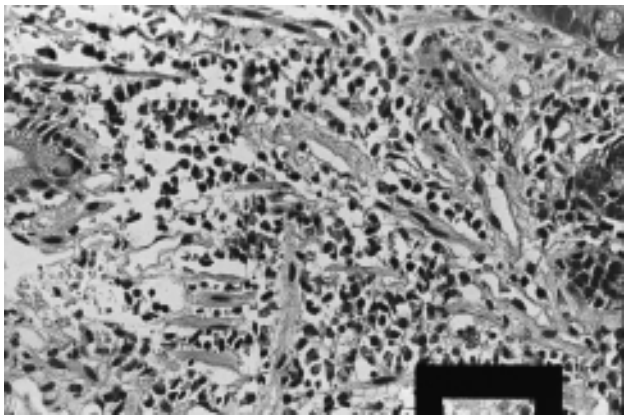

Figure 2. Photomicrograph from descending duodenum showing eosinophilic infiltration of the lamina propria (H/E 400x)

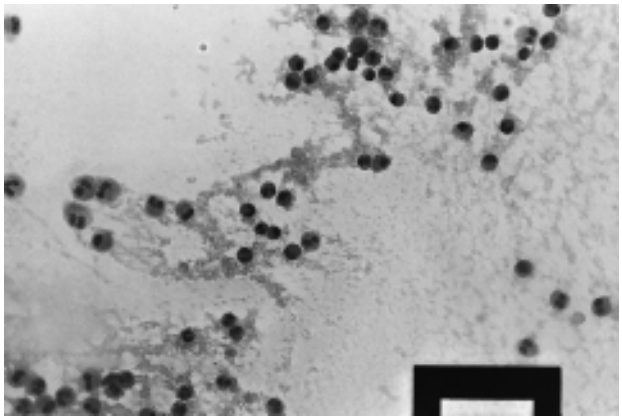

Figure 3. Photomicrograph from the ascitic fluid containing predominantly eosinophil leucocytes. (H/E 400x).

protein and eosinophil peroxidase[7]. Eosinophils may damage the gastrointestinal tract directly, through the release of toxic proteins[8], or indirectly, by triggering leukotriene synthesis[9] or by activating mast cells to release histamine. Although several mechanisms have been proposed to explain the degranulation of eosinophils in EGE, an alteration in intestinal epithelial integrity leading to antigen-induced eosinophil activation[7] appears to be the most acceptable hypothesis. However, only half of the patients with EGE have an atopic history, and in patients with predominantly subserosal involvement, food allergy is exceptional[2,10].

EGE is classified according to the layer of gastrointestinal tract involved. Mucosal involvement, the most common form of presentation, may result in abdominal pain, nausea, vomiting, diarrhea, anemia, and protein-loosing enteropathy while muscular layer disease generally presents as gastrointestinal obstruction. Serosal eosinophilic infiltration, the rarest form of presentation, may result in the development of eosinophilic ascites. EGE complicated by ascites can be effectively treated with steroids after other systemic disorders associated with peripheral eosinophilia have been ruled out.

Diagnosis of subserosal EGE is often difficult with non-diagnostic endoscopic biopsies and the need for surgical transmural tissue samples. This form of the disease comprises less than 10% of the cases reported in the world literature[11]. Although Kravis et al.[12] attempted to correlate pathological and clinical findings, it seems that only subserosal disease has distinctive clinical features[2]: a higher peripheral eosinophil count, eosinophilic ascites, abdominal bloating and a dramatic response to steroids. The ascitic fluid is usually a sterile exudate including an elevated percentage of eosinophils that can reach 95% of the cellular content. Our patient had very high absolute counts for peripheral eosinophils and paracentesis showed an exudate with 90% eosinophils. He was referred because of abdominal pain, the most frequent complaint in patients with subserosal disease[2,11].

The rarity of EGE and its nonspecific presenting features and radiologic findings make it a diagnostic challenge. It is often misdiagnosed initially as a malignant tumor or inflammatory bowel disease. The presence of peripheral eosinophilia may be helpful in establishing the diagnosis, but the peripheral eosinophil count is normal in about 25% of cases[2,11]. Even if peripheral eosinophilia is present, other disorders that may be accompanied by eosinophilia, such as parasitic infection, vasculitis, lymphoma, carcinoma or inflammatory bowel disease, must be ruled out[4].

EGE has an excellent prognosis when treated properly. Treatment of the subserosal form is usually based on a steroid regimen, starting with prednisone (20-40mg/day) for 2 weeks and followed by progressive tapering until discontinuation. A dramatic response has been documented in more than 90% of the reported cases[3,11,13]. The response should be monitored by assessment of clinical symptoms, the obtaining of peripheral eosinophil counts and possibly by use of non-invasive imaging techniques such as ultrasonography[14]. Surgical intervention is discouraged because of high recurrence rates. Our patient improved remarkably after administration of oral steroids and remains in clinical remission one year after the discontinuation of this therapy.

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