

Eosinophilic Gastroenteritis Presenting with Duodenal Obstruction and Ascites

Dear Editor,

Eosinophilic gastroenteritis (EG) is a rare inflammatory disorder of the gastrointestinal tract of unknown aetiology. It is characterised by eosinophilic infiltration of the bowel wall, peripheral eosinophilia and various gastrointestinal manifestations. We report CT appearances in a rare case of EG with duodenal obstruction, peripheral eosinophilia and eosinophilic ascites, which responded promptly to steroid therapy.

Case Report

A 31-year-old man was admitted with 3 weeks history of progressive epigastric discomfort, vomiting and diarrhoea, which was aggravated by meals. He had no history of fever, weight loss, abdominal surgery or any known food allergy. He denied taking any drugs or herbal medicines. On clinical examination, he appeared dehydrated and vitals were normal. His abdomen was soft and slightly distended.

Laboratory investigations showed a high white cell count of $24.1 \times 10^9/L$, with raised eosinophils count of $12.51 \times 10^9/L$. The haemoglobin level and platelet counts were normal. The liver function tests, erythrocyte sedimentation rate, C-reactive protein and autoimmune antibody screen were normal. The serum immunoglobulin-E (IgE) was mildly raised at 103 IU/ml (normal reference range, 0 to 87 IU/ml). Stool culture for pathogens and analyses for ova, cysts and parasites were negative. Serum anti-amoebic antibody and serology for strongyloides were negative.

Endoscopy revealed large mucosal folds in the gastric antrum and proximal duodenum causing obstruction (Fig. 1) and the endoscope was not able to pass through. Endoscopic ultrasound (EUS) showed thickened gastric antral wall involving all layers. Multiple biopsies were obtained from the gastric antrum and duodenum. During the admission, the abdominal pain and vomiting worsened. An abdominal radiograph showed dilated small bowel. Computerised tomography (CT) revealed dilated and oedematous duodenum and proximal jejunal loops (Fig. 2). The distal ileal loops were also dilated and no definite transition point of obstruction was seen. The colon was normal. So were the liver, spleen and pancreas. There was moderate amount of ascites with no evidence of inflammation. There was no lymphadenopathy.

The mucosal biopsies showed gastritis, duodenitis,

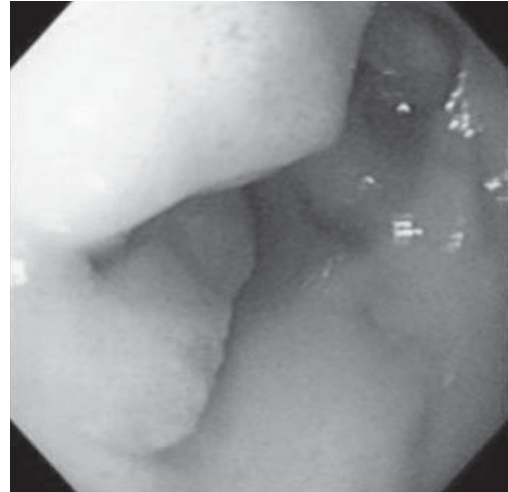


Fig. 1. Endoscopic view of the junction of the first and second parts of duodenum showing large mucosal folds.

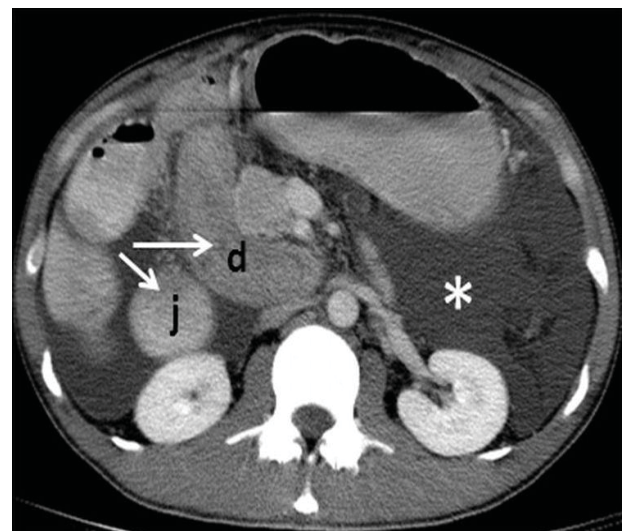


Fig. 2. Axial image of CT abdomen, with intravenous and oral contrast medium administration, showing dilated and oedematous duodenum and proximal jejunum (arrows). Incidental note of gut malrotation with the jejunal loop (j) located to the right of duodenum (d) is demonstrated. There is moderate amount of ascites (*).

duodenal intramucosal eosinophilic infiltration and eosinophils in the crypts of the epithelium. The peritoneal fluid from diagnostic tap showed sterile exudative ascites with predominant eosinophils. There was no evidence of malignant cells.

The combination of endoscopic, CT and ascitic fluid analysis led to the diagnosis of EG, after exclusion of other causes of peripheral eosinophilia. He was treated with oral prednisolone (30 mg daily). His symptoms improved, and the serum white cell and eosinophil counts returned to normal levels after 9 days. The steroid dose was gradually tapered at follow-up as outpatient and successfully stopped a year later. He has remained symptom-free for more than 3 years post-treatment.

Discussion

Eosinophilic gastroenteritis is a rare, benign inflammatory disorder of the gastrointestinal tract, characterised by eosinophilic infiltration of the layers of the bowel wall, in the absence of known causes of eosinophilia, including drug reaction, parasitic infections and malignancy.¹⁻³

EG affects all ages of both genders and diagnosed most frequently in the third decade of life.^{2,3} EG mainly involves the stomach and duodenum.^{1,2} The pathogenesis and aetiology remain unclear. A personal or family history of allergic disorder is reported in 70% of patients with this disorder.⁴

EG should be considered in the differential diagnosis of unexplained gastrointestinal symptoms, especially in the presence of peripheral eosinophilia. The 3 main diagnostic criteria are: (i) the presence of gastrointestinal symptoms, (ii) biopsies showing eosinophilic infiltration of one or more areas of the gastrointestinal tract (>20 eosinophils per high power field) or typical radiological findings with peripheral eosinophilia, and (iii) no evidence of parasitic or extra-intestinal disease.² Histopathology is the gold standard for diagnosis.^{1,3}

The disease is classified histopathologically into 3 major types: (i) predominant mucosal (60%)—manifested as abdominal pain, vomiting, diarrhoea, weight loss and malabsorption, (ii) predominant muscle layer (30%)—causing bowel wall thickening and intestinal obstruction, and (iii) predominant serosal (10%)—manifested as eosinophilic ascites and intense peripheral eosinophilia.^{2,3,5}

Our patient had features indicating serosal disease, involving all bowel layers as evidenced by the diffuse bowel wall thickening at CT and endoscopy. The large duodenal folds causing obstruction seen on endoscopy suggest muscle layer involvement, mimicking malignancy. This was supported by the EUS finding of thickening of all layers. Eosinophilic ascites indicates serosal involvement in

this patient. Other causes of eosinophilic ascites including abdominal lymphoma and parasitic infection were excluded.

EG is usually patchy in distribution, which explains the absence of eosinophilic infiltration in the gastric antrum specimen in our patient.^{2,3} Invasive laparotomy or laparoscopic full thickness biopsy may need to be performed to diagnose subserosal disease.¹ This was avoided in our case, as there was sufficient endoscopic, radiological and histological information available to arrive at the diagnosis and guide treatment.

Peripheral eosinophilia is seen in up to 80% of cases of EG.^{2,3} Raised serum IgE, as seen in our patient, was reported to be more prevalent in children with EG.⁵

Endoscopy, CT and ultrasonographic studies may provide indirect signs of EG. Thickened mucosal folds represent part of spectrum of endoscopic findings in EG, which vary from normal mucosa to frank ulceration.^{1,3} Barium contrast studies, CT and ultrasonography may show non-specific generalised bowel wall thickening, bowel obstruction, ascites and lymphadenopathy.^{1,3}

Ninety percent of patients with EG respond to steroid therapy, and more dramatic response is usually seen in the serosal subtype.^{2,3} The duration of steroid therapy reported in the literature is variable. Other treatment options include dietary modification, leukotriene receptor antagonists, mast cell stabilisers, and antibodies against interleukin-5 and IgE.^{1,3,5} Surgical intervention may be required for cases where a definitive diagnosis cannot be made or when perforation or significant obstruction occurs.^{1,3}

The natural history of EG is unclear, emphasising the need for long-term follow-up studies for patients with EG. Our patient remained free from the disease for more than 3 years after stopping treatment.

Conclusion

In summary, we present a case of unusual manifestations of EG secondary to serosal involvement demonstrated with CT. The clinical, radiological, endoscopic and pathological findings indicate serosal disease with diffuse wall involvement. Physicians should be aware of this entity as it can mimic several other gastrointestinal disorders.

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