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## CASE REPORT

# Eosinophilic Gastroenteritis: an Unusual Cause of Acute Abdomen

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## ABSTRACT

***Eosinophilic gastrointestinal disorders are relatively rare disorders characterised by eosinophilic infiltrate into any layer of the gastrointestinal tract, for example, mucosa, muscularis mucosae, and serosa, usually in association with peripheral eosinophilia. This report describes the computed tomography findings of a patient with oesophago-gastroenteritis presenting with acute abdominal pain and ascites. The computed tomography findings of a layered pattern of bowel wall thickening and mesenteric hyperaemia, which may mimic inflammatory bowel disease, are presented.***

***Key Words:*** *Abdomen, acute; Eosinophilia; Gastroenteritis; Tomography, X-ray computed*

## 中文摘要

### 嗜酸細胞性胃腸炎：急腹症的非常見病因

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嗜酸性粒細胞引發的胃腸功能紊亂屬於相對少見病症，特徵是嗜酸性粒細胞浸潤胃腸道的任何分層，如粘膜層、粘膜肌層和漿膜層；通常與外周血嗜酸性粒細胞增多有關。本文報告一名臨床症狀為急性腹痛和腹腔積液的食道–胃腸道炎症患者，其電腦斷層掃描結果為與腸道炎症表現類似的腸壁層狀增厚和腸系膜充血。

## INTRODUCTION

Primary eosinophilic gastrointestinal disorders (EGIDs) are defined as disorders that selectively affect the gastrointestinal (GI) tract, with eosinophil-rich inflammation in the absence of any known cause of eosinophilia-like parasitic infection, malignancy, or drug reaction. These disorders may affect any part of the GI tract, and include eosinophilic oesophagitis, eosinophilic gastritis, eosinophilic gastroenteritis, eosinophilic enteritis, and eosinophilic colitis.[1] There may be eosinophilic infiltration in any layer of the GI tract, and the symptoms may vary according to the layer and the part of GI tract involved. A history of atopy is found in up to 75% of patients.[2] The signs and symptoms may mimic other pathologies ranging from chronic recurring abdominal pain to acute abdomen.[3] This report describes a patient with eosinophilic oesophago-gastroenteritis who presented with acute abdominal pain and ascites.

## CASE REPORT

A 42-year-old man presented to the emergency

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department in January 2000 with severe abdominal pain for 2 days. He had no history of fever, night sweats, arthralgia, oral ulcers, or skin rashes or nodules. He had a history of a similar episode 1 year previously, which was treated by his general practitioner, and asthma for the previous 6 years.

On examination he had abdominal pain with tachycardia and tachypnoea, but his vital signs were otherwise normal. There was slight tenderness in the umbilical region, but no guarding or rigidity. His initial blood test results showed haemoglobin level of 110 g/L (reference range, 140-175 g/L) and white blood cell (WBC) count

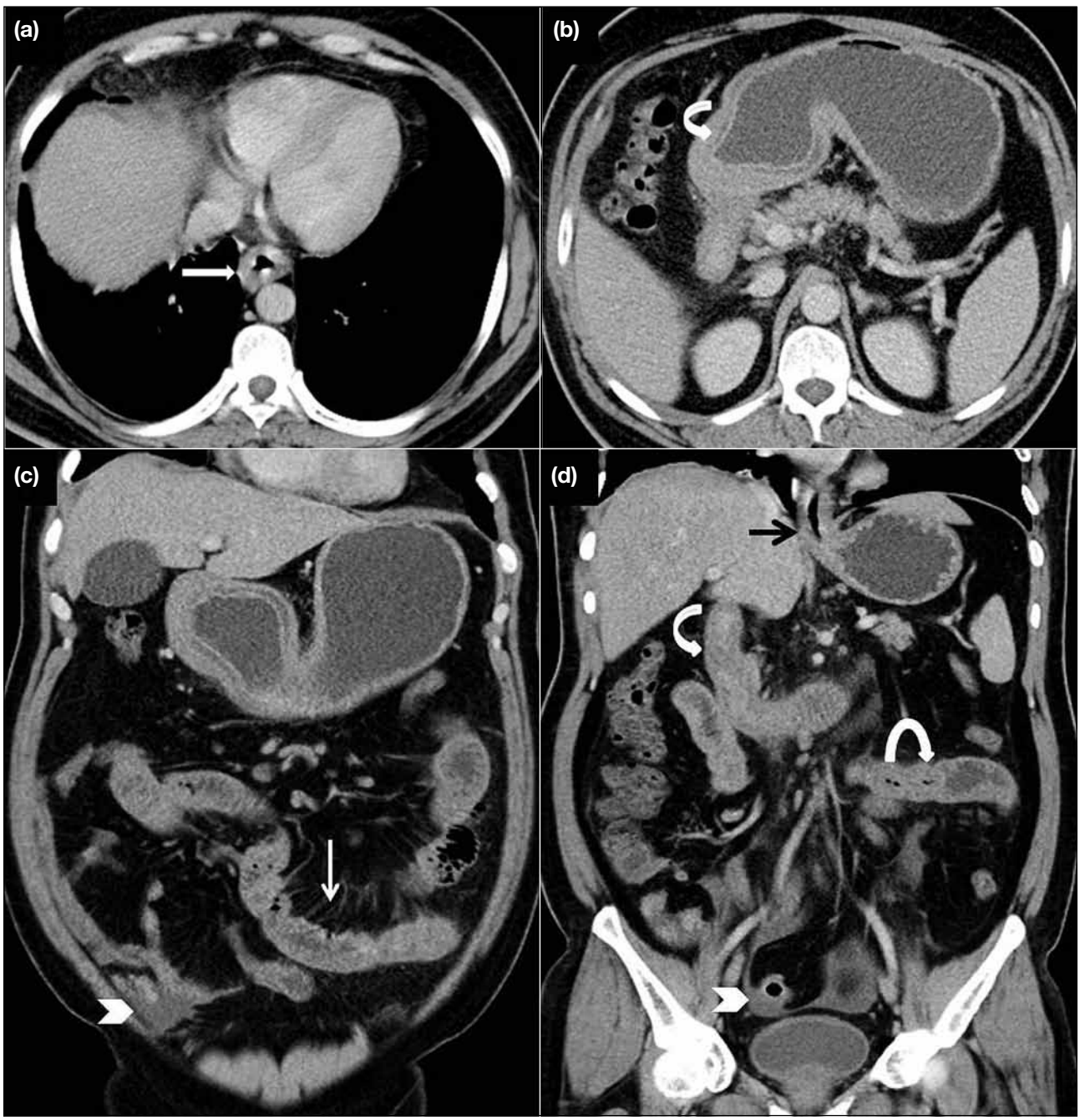


**Figure.** (a and b) Contrast-enhanced axial computed tomography images showing wall thickening of the lower dorsal oesophagus (straight arrow), with layered wall thickening of the antrum and a hypodense halo within (curved arrow). (c and d) Contrast-enhanced coronal computed tomography images showing markedly increased mesenteric vascularity (straight white arrow) with wall thickening of the duodenum, proximal jejunal loops (curved arrows), lower oesophagus (straight black arrow), and gastric antrum. A small amount of free fluid can be seen in the lower abdomen and pelvis (chevrons).

of 12.3 x $10^9$ /L (reference range, 4.5-11.0 x $10^9$ /L).

Emergency abdominal computed tomography (CT) revealed evidence of mural thickening in the stomach, duodenum, and proximal jejunum, as well as the lower dorsal oesophagus visualised on the abdominal CT scan. The wall showed a layered pattern of thickening with a prominent hypodense halo ranging from 5 mm to 15 mm in size. Mesenteric hyperaemia producing the 'comb sign' and mild ascites were also noted (Figure). No free air in the abdomen was seen. The pattern of involvement over a long segment with layering, mesenteric hyperaemia, and ascites suggested an inflammatory cause.

The patient underwent endoscopy, which revealed normal mucosa. Superficial endoscopic biopsies done on two separate occasions were reported as normal. Ascitic fluid polymerase chain reaction was negative for tuberculosis. Ascitic fluid cytology revealed WBC counts with 60% eosinophils. Peripheral blood smear also showed eosinophilia with 36% eosinophils on differential leukocyte count.

No evidence of connective tissue disorder / parasite infestation or extra-intestinal involvement such as cardiovascular, skin, or central nervous system manifestations or malignancy was noted.

The patient was treated with prednisolone (Wysolone; Wyeth, Mumbai, India) orally 40 mg/day for 2 weeks which was tapered over 6 weeks. This led to gradual remission of symptoms and ascites.

## DISCUSSION

Eosinophilic gastroenteritis is a rare disorder; the exact aetiology of which is not known. Eosinophilic gastroenteritis is characterised by eosinophilic infiltration of the wall of the GI tract along with GI symptoms and is often associated with peripheral eosinophilia.[1,4] In primary EGIDs there is selective involvement of the GI tract with no known causes for eosinophilia being evident.[5] In about 75% of patients, a history of allergy or atopy may be present.[2] This patient had a history of asthma, which was controlled by salbutamol inhaler, and no other cause of eosinophilia was evident.

Eosinophilic gastroenteritis is usually classified according to the layer of GI tract involved, for example, mucosal, muscular, and sub-serosal or serosal, as suggested by Klein et al.[6] The clinical presentation depends on the layer of GI tract involved. The most common pattern is mucosal involvement, and this may present clinically with abdominal pain, nausea, vomiting, diarrhoea, anaemia, and protein-losing enteropathy. GI obstruction is commonly seen in patients with muscular layer involvement. The least common variety is serosal eosinophilic infiltration, which may result in development of eosinophilic ascites and severe abdominal pain, and is more commonly associated with peripheral eosinophilia.[7] This pattern of presentation was seen in this patient who most likely had the serosal form of the disease. He presented with acute pain in the abdomen, and had eosinophilic ascites and peripheral eosinophilia, but normal superficial endoscopic biopsies. Other authors have reported that EGID may present as acute abdomen.[3] The presence of peripheral eosinophilia and eosinophilic ascites are useful pointers to EGID being a possible cause of acute abdomen. Although the diagnosis is usually established by demonstrating eosinophilic infiltration in the wall of the GI tract on endoscopic biopsies, these may be negative at times due to the patchy nature of eosinophilic infiltration; therefore, multiple biopsies may be required. Radiological features and the presence of eosinophilic ascites have also been used to diagnose the disease.[8]

The CT findings, which are usually non-specific, include wall thickening, bowel fold thickening with a layered pattern, intraluminal granuloma mimicking polyp, extraluminal mesenteric lymphadenopathy, inflammation, and ascites.[9-11] Ascites, a layered pattern of bowel wall thickening, and mesenteric hyperaemia were noted in this patient.

As the symptoms of EGID are varied and non-specific, the diagnosis is often delayed. The clinical differential diagnoses are extensive and include conditions ranging from acute intestinal obstruction to peptic ulcer disease, inflammatory bowel disease, and irritable bowel syndrome in patients with recurrent symptoms. Although there are no specific clinical features, EGID should be suspected in patients with recurrent non-specific GI symptoms of unexplained aetiology.[3,5,12] This patient had a history of a similar episode 1 year previously, for which he was treated by his general practitioner, but no investigations were done.

EGIDs are rare allergic disorders and the exact mechanisms involved are still unclear. The various

treatment options include exclusion of identified food allergens if possible, steroids, sodium cromoglycate, and montelukast (a selective leukotriene receptor antagonist). Since identification of food allergens may be difficult and dietary restrictions may not be possible if several allergens are identified, steroids remain the main therapy.[5,8,11] Steroids may be given as a short course or low-dose steroids may be needed over a long period for patients with repeatedly relapsing disease. Some authors have successfully used montelukast for long-term remission.[11] The present patient was given steroid therapy with gradual remission of ascites and symptoms.

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