



Unusual presentations of eosinophilic gastroenteritis: Two case reports

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ABSTRACT

Eosinophilic gastroenteritis is a rare disease that is characterized by eosinophil infiltration in one or multiple segments of the gastrointestinal tract. The etiology of this condition remains unknown. Eosinophilic gastroenteritis has heterogeneous clinical manifestations that depend upon the location and depth of infiltration in the gastrointestinal tract, and eosinophilia may or may not be present. This article reports two cases of eosinophilic gastroenteritis. The first is that of a 49-year-old woman with abdominal pain, ascites, eosinophilia, and a history of asthma. The second case is that of a 69-year-old male with a history of loss of appetite, belching, postprandial fullness, heartburn, and a 5-kilogram weight loss over a period of 9 months; ultimately, the patient was diagnosed with a gastric outlet obstruction due to pyloric stenosis. The rare character of eosinophilic gastroenteritis and its varied clinical presentations often lead to delayed diagnoses and complications. Case reports may help to disseminate knowledge about the disease, thereby increasing the likelihood of early diagnosis and intervention to prevent complications.

Keywords: Gastroenteritis, eosinophils, ascites, pyloric stenosis

INTRODUCTION

Eosinophilic gastroenteritis (EG) is classified in a group of diseases known as eosinophilic gastrointestinal disorders (EGIDs). The primary EGIDs are defined as diseases that selectively affect the gastrointestinal tract with the presence of inflammatory infiltrate that is rich in eosinophils in the absence of other causes of eosinophilia, such as drug reactions, parasitic infections, or malignancies. This group of diseases, the incidence of which has increased over the last few decades, includes eosinophilic esophagitis, eosinophilic gastritis, eosinophilic enteritis, eosinophilic colitis, and EG (1). All of these conditions exhibit significant infiltration of eosinophils in the gastrointestinal tract and a very strong association with allergies (2).

Eosinophilic gastroenteritis is defined by the infiltration of eosinophils occurring in multiple segments of the

gastrointestinal tract from the esophagus to the rectum and at different depths in the wall layer (3). According to Klein's diagnostic classification from 1970, the infiltration may be predominantly at the mucosal layer, the muscle layer, or the subserosal layer (4).

If the eosinophilic infiltrate occurs predominantly at the mucosal layer, the clinical manifestation usually involves nonspecific abdominal findings, such as abdominal pain, nausea, vomiting, diarrhea, fecal occult blood, anemia, and weight loss (3,5,6). According to the literature, this is the most prevalent subtype of EG; however, some have suggested the possibility of bias due to the relative ease of access to the mucosal layer when doing an endoscopic biopsy in order to confirm the presence of eosinophilia (6,7).

Eosinophilic gastroenteritis involving the muscle layer represents 13%-70% of all subtypes of EG and typically

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involves symptoms of gastric outlet obstruction and/or intestinal obstruction, because the infiltration of eosinophils within this layer causes an increase in the thickness of the wall of the gastrointestinal tract (8-10). The obstruction most frequently develops in the jejunum, but reports of obstruction in the colon and cecum have been published (11,12).

The subserosal form of EG is most frequently associated with increased abdominal volume (e.g., due to ascites), eosinophilia, and a great response to corticosteroids (5,13). Subserosal involvement is considered the rarest of the three forms of presentation (i.e., occurs in 12%-40% of all cases of EG), and patients typically present with ascitic fluid containing large numbers of eosinophils (5,8).

We describe two unusual case presentations of eosinophilic gastroenteritis. Patients have provided written, informed consent to participate in the study. This study was approved by our institutional review board under number 270.882, on May, 2013.

CASE REPORTS

Case 1

A 49-year-old woman was admitted to the hospital with abdominal pain in the periumbilical region that improved with the administration of simple analgesics and antispasmodics. Throughout the 7 months prior to admission, she exhibited a gradual increase in abdominal volume with no associated altered bowel habits, fever, or weight loss. She had a history of asthma since childhood but did not use medication to control it. Moreover, she complained of chronic dyspnea associated with moderate effort. She had a 5-pack-year history of smoking but stopped smoking 15 years ago.

At the physical examination, she presented with hypertension (i.e., 140x80 mm Hg) and tachycardia (i.e., 105 bpm) and was afebrile. Upon admission, a cardiac examination was performed, the results of which were normal. With percussion, hyperresonant lung sounds were detected. Moreover, pulmonary auscultation detected distant breath sounds with intermittent wheezing. Upon examination, the abdomen was distended with normal bowel sounds; however, mesogastric pain was present on palpation, and the presence of a Skoda semicircle below the umbilical scar, dullness, and the absence of visceromegaly were noted. No edema was present in the lower limbs. The results of the ordered laboratory tests were as follows: hemoglobin=11.4 g/dL, leukocytes=7560/mm³ (segmented=4348/mm³, lymphocytes=1897/mm³, monocytes=567/mm³, eosinophils=718/mm³, and basophils=30/mm³), platelet count=347,000/mm³, uptime prothrombin activity=78.1%, and serum albumin=3.3 g/dL. Moreover, a urinalysis was completed, and the results were normal. An ultrasound of the abdomen showed moderate ascites. Computed tomography (CT) of the chest, abdomen, and pelvis was performed, and the results in-

dicated pleural effusion and a large amount of free fluid in the peritoneal cavity. Paracentesis was performed to remove the ascitic fluid, which was subsequently analyzed. The exhibited total protein was 5.6 g/L, and the albumin level was measured at 3.2 g/L with a serum-ascites albumin gradient (SAAG) of 0.1 g/dL. Moreover, the ascitic fluid contained 600 cells/mm³ (i.e., 75% mononuclear cells, 8% eosinophils, and 7% neutrophils). The glucose level, lactate dehydrogenase (LDH), and adenosine deaminase (ADA) were measured at 97 mg/dL, 130 U/L, and 31U/L, respectively. An upper endoscopy was performed with normal results. A duodenal biopsy showed that there were 20 to 25 intraepithelial lymphocytes per 100 enterocytes, and staining with periodic acid-Schiff was negative for *Tropheryma whipplei*. A colonoscopy was performed and showed diverticular disease of the colon, but a small bowel transit time test was normal. All rheumatologic tests (i.e., anticardiolipin, anti-DNA, anti-La, anti-mitochondrial, anti-Ro, anti-RNP, anti-Sm antibodies, antinuclear antibodies, and rheumatoid factor) were negative. Tests for neoplastic markers (i.e., carcinoembryonic antigen, CA 19-9, and CA 125) were negative. A parasitological exam was conducted, and the results were normal in all three samples. The patient underwent a thoracoscopy and laparoscopic surgery to biopsy the pleura and peritoneum, which showed chronic pachypleuritis with severe hyalinization and increased numbers of eosinophils in the peritoneum (>20/high-power field) (Figure 1).

Prednisone therapy was administered at a dose of 20 mg per day. As a result, pain remission occurred in 8 days, and progressive improvement of ascites was observed. Moreover, the patient presented with a regular ultrasound and a blood test showing no eosinophilia in 30 days. The patient has now been asymptomatic for more than 1 year.

Case 2

A 69-year-old-man presented at the gastroenterology clinic with a history of foul smelling belches, heartburn, loss of appetite, and postprandial fullness over the past 9 months. He also complained of vomiting and constipation and had lost 5 kilograms in weight during this period. The patient reported no comorbidities and did not use any medication. The patient had a 78-pack-year smoking history and a 34-year history of drinking 60 g of alcohol per day with a prior hospitalization for alcoholism.

Upon physical examination, the patient was hypertensive (i.e., 180x90 mm Hg), eupneic, and afebrile. A cardiac examination was performed, the results of which were normal. An abdominal examination revealed a rounded abdomen with bulging flanks, normal bowel sounds, no pain, and no palpable masses or visceromegaly.

The results of the laboratory tests showed mild normochromic, normocytic anemia with a normal iron profile and hypoalbuminemia. Other lab results were as follows: CBC

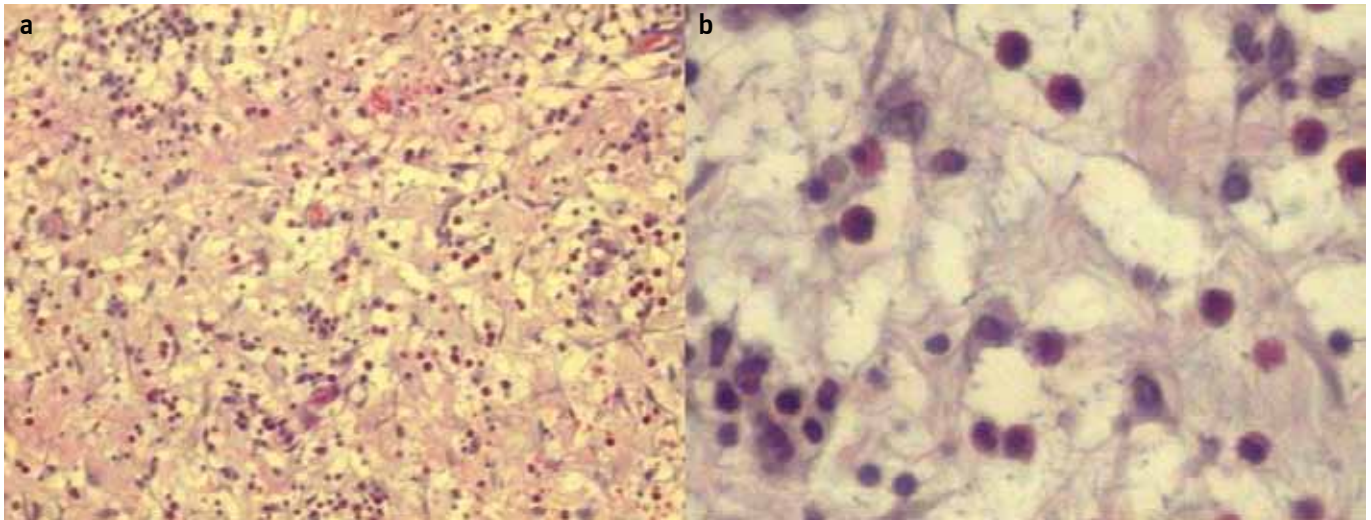


Figure 1. a, b. Microscopic image showing increased numbers of eosinophils in peritoneal adipose tissue. Hematoxylin-eosin stain (HE) 100X (a). HE 400X (b).

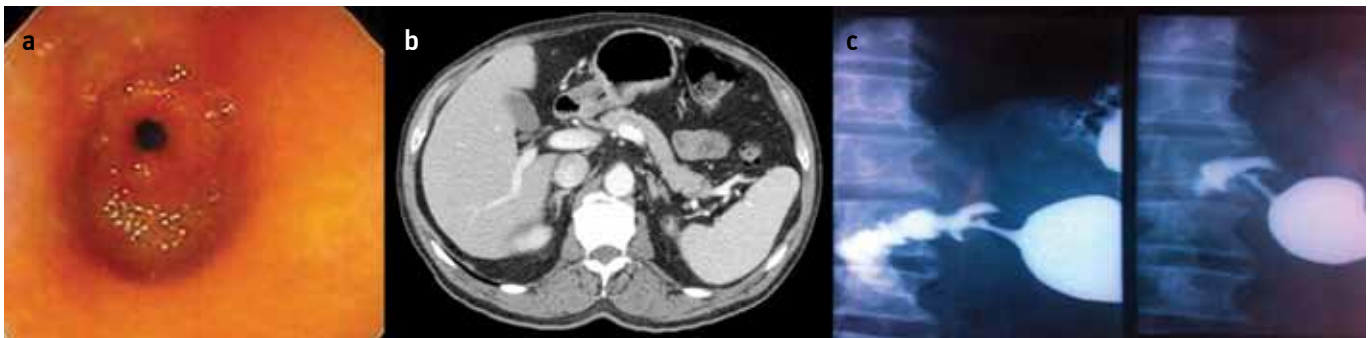


Figure 2. a-c. Images evidencing pyloric stenosis during clinical investigation. Endoscopic image showing hyperemia and concentric stenosis of the pylorus, which prevented the progression of an endoscopic device into the duodenum (a). CT evidences stenosis of pyloric lumen (b). Esophageal gastroduodenal serioscopy demonstrating significant reduction of the pyloric-antral segment (c).

showing hemoglobin=12 g/dL, leukocytes=6720/mm³ (segmented=4079 mm³, lymphocytes=1680/mm³, monocytes=759/mm³, eosinophils=181/mm³, and basophils=20/mm³), platelet count=190,000/mm³, transferrin=181 mg/dL, ferritin=300 ng/mL, serum iron=115 mg/dL, creatinine=1.1 mg/dL, uptime prothrombin activity=75%, and serum albumin=3.2 g/dL. An upper digestive endoscopy was performed and showed erosive esophagitis grade A (i.e., according to the Los Angeles classification), erosive gastritis in the antrum, pyloric stenosis (Figure 2a), and gastric stasis. The anatomic pathology division examined samples from a biopsy and found chronic gastritis with discrete activity, increased numbers of eosinophils, the presence of erosion foci, and areas of complete intestinal metaplasia and atrophy. However, *Helicobacter pylori* was not present. The results of a CT of the abdomen and pelvis showed thickening of the pyloric region associated with a narrowing of the lumen and two small adjacent lymph nodes, the largest of which measured 0.6 cm (i.e., the features were nonspecific); furthermore, no retroperitoneal or pelvic lymphadenopathy was indicated, and the presence of diverticular disease of the colon was noted (Figure 2b). The patient underwent an esophageal, gastric, duodenal serioscopy, which demonstrated a significant reduction of the pyloric-antral segment (Figure 2c).

After admission, the patient's condition progressively worsened to the point of requiring enteral feeding. Despite the administration of potent antiemetic medication, the patient's vomiting persisted, and a subtotal gastrectomy with reconstruction of the gastrointestinal tract using the Roux-en Y technique was indicated. The anatomic pathology division examined a surgical specimen and found the presence of coarse grain at the antral mucosa and thickening of the wall of the stomach with pyloric stenosis (Figure 3a). The results of histological tests of the mucosal layer and muscularis propria demonstrated increased numbers of eosinophils (> 25/high-power field) (Figure 3b, c).

The patient recovered well postoperatively and was discharged in 3 days; moreover, he remained asymptomatic for over a year. No corticosteroids were administered in this case.

DISCUSSION

Due to the diversity in the clinical presentation of this disorder, EG is a differential diagnosis from many other similarly presenting diseases. The first and second cases involve differentiation from ascites syndrome and gastric outlet obstruction, respectively.

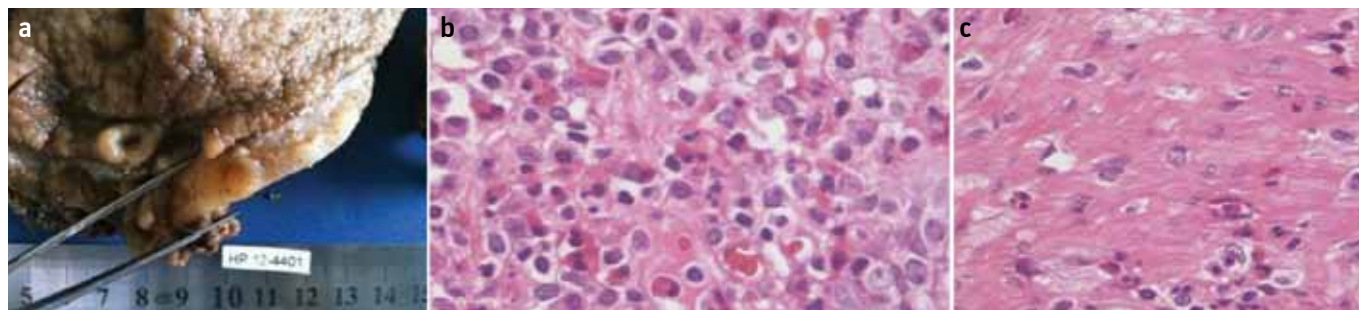


Figure 3. a-c. Surgical specimen of gastrectomy. Detail of picture demonstrates coarse grain at the antral mucosa, a stomach wall thickness of 1.5 cm, and light pyloric stenosis measuring 0.5 cm **(a)**. Microscopic image showing the mucosal layer with increased number of eosinophils in hematoxylin-eosin stain (HE), 400x **(b)**. Microscopic image showing the muscular propria with increased number of eosinophils (HE 400X) **(c)**.

Ascites is defined as the pathologic accumulation of free fluid in the peritoneal cavity. When an individual presents with ascites, liver cirrhosis is the diagnosis in 85% of cases (14). Other causes of ascites include congestive heart failure, liver metastasis, peritoneal carcinomatosis, pancreatic ascites, and peritoneal tuberculosis (15). Case history, physical examination, and paracentesis are essential for the differential diagnosis of EG. The SAAG replaces the old classification of ascites using the exudate and transudate and is performed by subtracting the ascites fluid albumin from serum albumin levels. A SAAG value ≥ 1.1 g/dL equates to a diagnosis of ascites secondary to portal hypertension with 97% accuracy (15). However, the paracentesis of the patient in the first case revealed a SAAG value of < 1.1 g/dL. Among individuals with a SAAG value < 1.1 g/dL, differential diagnoses usually involve diseases of the peritoneum, such as peritoneal carcinomatosis and peritoneal tuberculosis. Those diseases were excluded in the first case, because the abdominal CT showed no thickening and/or peritoneal implants, and the ADA level in the ascitic fluid was low. The possibilities of pancreatic ascites, intestinal obstruction, intestinal infarction, and biliary ascites were also excluded in the first case, because the clinical findings were not suggestive of such; moreover, the amylase and bilirubin levels in the ascitic fluid were normal, and the CT revealed no alterations in the pancreas, bile duct, or bowel. Nephrotic syndrome was not considered, because the patient in the first case had no anasarca, hypoalbuminemia was discreet, and the urinalysis showed no proteinuria (16). Moreover, all of the laboratory evidence that might indicate inflammatory serositis was normal. The patient's case was reviewed, and we noted a history of asthma and peripheral blood eosinophilia, in addition to the finding of 8% eosinophils in the ascitic fluid. Intestinal parasites, Crohn's disease, and Whipple's disease were rejected as the potential cause of eosinophilia. A laparoscopic peritoneal biopsy was essential for the diagnosis of EG, which showed increased numbers of eosinophils.

Although it is a rare cause of ascites, eosinophilic ascites due to EG has been reported frequently in the literature. In the past 5 years, a number of cases of ascites as a clinical manifestation of EG in adults have been published; most have been in Asia and Europe, but four were reported in the United States and two in South America (i.e., none in Brazil) (17-43). Note that we were

unable to analyze some of the articles because of language difficulties and access (18,25,26,31,33,36,40). In total, we reviewed 23 cases of eosinophilic ascites. In general, the most frequently described clinical presentation is abdominal pain associated with nausea and vomiting. Moreover, the presence of pleural effusion in subjects with eosinophilic ascites, as described in this report, is not rare and usually abates with the usual treatment (i.e., administration of a corticosteroid) (30,32,38,39,43).

Jaimes-Hernandez et al. (43) reported a case of a patient with ascites, hemolytic anemia, and positive autoantibodies with simultaneous diagnoses of eosinophilic ascites and systemic lupus erythematosus (SLE); the patient responded to methylprednisolone pulse therapy but 3 months later presented with an intestinal obstruction.

Aslanidis et al. (41) reported the case of a young patient with a history of SLE, scleroderma, and 6 years of immunosuppressive treatment, which resulted in a case of eosinophilic ascites with abdominal pain and thickening of the small bowel. The association between EG and SLE is rare i.e., only four cases reported in the literature (41,43-45)-and only these two were reported to have eosinophilic ascites (41,43).

Cha et al. (39) suggested an association between eosinophilic ascites and eosinophilic dermatitis i.e., an eosinophilic infiltrate was found to have a macroscopically normal skin biopsy-although further studies are needed to confirm this hypothesis.

Patients with subserosal eosinophilic gastroenteritis usually present with marked eosinophilia in the ascitic fluid (45%-90%) (17,29,30,34). Even though the patient in case 1 did not exhibit such a high eosinophil count in the ascitic fluid, as expected, histologic evaluation demonstrated 20 eosinophils/high-power field, which is considered the standard complementary method for the diagnosis of eosinophilic gastroenteritis in this clinical context. EG is a rare cause of ascites among individuals with SAAG < 1.1 g/dL; however, this condition is emerging as a more common differential diagnosis than previously thought and should be considered promptly, especially in individuals with a history of atopy and peripheral eosinophilia.

Case 2 describes gastric outlet syndrome in an elderly individual with weight loss and constipation. The differential diagnoses include benign disorders and diseases with a poor prognosis. The absence of ulcers revealed in the endoscopy ruled out the possibility of peptic stricture or gastric tuberculosis (46-51). The pyloric stenosis was benign with a regular centralized gastric outlet and no associated infiltrative lesions. However, due to the age of the patient, his history of alcoholism and smoking, and the presence of large areas of metaplasia found in the pyloric biopsy, the diagnosis of malignancy due to a distal gastric cancer could not be ruled out, because this type of tumor accounts for about 35% of gastric outlet obstruction cases (52). Moreover, pancreatic adenocarcinoma with extension and extrinsic compression of the stomach and duodenum is the cause of 15% to 25% of cases of gastric outlet obstruction (53); however, the CT results did not support this diagnosis. We also investigated different causes to account for the presence of the eosinophilic infiltrate. A variety of drugs, including azathioprine (54), gemfibrozil (55), carbamazepine (56), clofazimine (57), and enalapril (58), can cause infiltration of eosinophils in the gastrointestinal tract. The patient reported no history of the use of any of these medications, and *H. pylori* was not present, which is also another cause of eosinophilic infiltration (6). This patient showed no eosinophilia during hospitalization, which is similar to 20% to 50% of cases of EG (1). Intestinal parasitic infections are usually accompanied by the presence of eosinophilia; however, disseminated strongyloidiasis can exist without eosinophilia and evolve to intestinal obstruction (59). However, in case 2, three samples for parasitological stool examination were negative, and all biopsies showed no evidence of parasites.

In cases of EG presenting with obstructive complications, surgery may be indicated. Two cases were described in patients who were both about 40 years of age and had acute abdominal pain; one had an acute abdominal inflammatory complication, and the other had an obstructive complication. Both patients had a history of recurrent abdominal pain over the previous recent months, which worsened significantly at admission; one of them presented with a perforation of the wall of the small intestine. They both underwent surgery and received outpatient treatment with administration of 40 mg of prednisone for 3 months; both had good outcomes (60).

In another case, a patient had gastric dilatation with significant stenosis of the second and third duodenal portion, which was seen on endoscopy, and histological results demonstrating eosinophilic infiltration. The patient underwent dilation of the stenotic area with a pneumatic balloon in combination with a pharmacological treatment course (i.e., esomeprazol, domperidone, loratadine, and prednisone) (61). This was the first reported case in which the treatment involved an endoscopic intervention for a gastrointestinal tract obstruction due to EG, thereby circumventing the need for surgical resection. Sheikh et al. (62) reported cases of obstruction due to EG that were treated successfully through the use of corticosteroids and

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cases in which the use of corticosteroids (i.e., some associated with sodium cromoglycate and others not) resulted in therapeutic failure with recurrence and the need for surgical resection of the affected areas.

After an extensive literature review, we found four case reports of pyloric stenosis due to EG causing gastric outlet obstruction in adults; all underwent therapy with corticoids and had a good clinical responses (32,63-65).

Eosinophilic gastroenteritis is an uncommon disease with signs and symptoms similar to many other diseases. The presentation of pyloric stenosis with this disease is very rare, which is why it is often not considered in the differential diagnosis. Histology is essential for the diagnosis of RG and should be considered. Making the correct diagnosis early may result in effective treatments that avoid surgery.

Ethics Committee Approval: Ethics committee approval was received for this study from the institutional review board.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

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