Eosinophilic gastroenteritis is a rare gastrointestinal (GI) disorder characterized by nonspecific GI symptoms, peripheral eosinophilia, and eosinophilic infiltration of the intestinal wall. The disorder is classified into mucosal, muscular, and sub-serosal types, depending on the clinical picture and the depth of eosinophilic infiltration within the GI wall. Sub-serosal disease, which is complicated by ascites, usually results in the most severe clinical form of eosinophilic gastroenteritis and requires early corticosteroid therapy. In such cases, a favorable outcome can be achieved after a short course of corticosteroids. We present the case of a 28-year-old female with diffuse abdominal pain and distention for 2 weeks. Her physical examination was significant for moderate ascites. Initial work-up demonstrated severe peripheral blood eosinophilia, normal liver function tests, and elevated serum immunoglobulin E (IgE). Upper endoscopy, colonoscopy showed a thickening of the stomach and colon, and biopsies showed marked eosinophilic infiltration of the mucosa. Ascitic fluid analysis showed significant eosinophilia. Subsequent treatment with oral prednisone resulted in the normalization of laboratory and radiologic abnormalities 45 days after the start of the treatment. Despite its rarity, eosinophilic gastroenteritis needs to be recognized by the clinician because the disease is treatable, and timely diagnosis and initiation of treatment could be of major importance.

**Key words:** Eosinophilic ascites, eosinophilic gastroenteritis, gastrointestinal disorder

**INTRODUCTION**

Eosinophilic gastroenteritis (EGE) represents a member of a family of diseases that includes eosinophilic esophagitis, gastritis, enteritis, and colitis, collectively referred to as eosinophilic gastrointestinal (GI) disorders. EGE is a rare disease characterized by focal or diffuse eosinophilic infiltration of the GI tract, especially the stomach and duodenum. It has vague, nonspecific symptoms, including nausea, vomiting, abdominal pain, diarrhea, weight loss, ascites, and malabsorption.[1] There is no single diagnostic test or procedure that would point directly to the diagnosis, and there are no strict or uniform diagnostic criteria.[2] Despite its rarity, eosinophilic gastroenteritis needs to be recognized by the clinician because this treatable disease can masquerade as irritable bowel syndrome.

**CASE REPORT**

A 28-year-old Saudi female with no significant past medical history presented with abdominal pain and distention of 2 weeks’ duration. She denied any recent fever, chills, night sweats, weight loss, change in bowel habits, sick contacts, and consumption of raw food. In addition, she denied any chest pain, shortness of breath, joint swelling and skin rash. There was no history of recent travel. The patient was not taking any prescribed or over the counter medications or herbal compounds, and denied a history of allergies to food or medication.

Her physical examination showed that the abdomen was moderately distended, with active bowel sounds, diffuse tenderness without rebound, and moderate...
ascites. There was no hepatomegaly or abdominal masses. A complete blood count and comprehensive metabolic panel were significant for an elevated white blood count of 18,840/µL with 61% eosinophils. Liver function tests were within normal limits. Serum IgE level was elevated at 240 IU/mL (normal <180). Stool tests on three different occasions were negative for parasites. Abdominal and pelvic ultrasonography (US) showed moderate ascites with prominent bowel loops of thickened walls [Figure 1]. Abdominal and pelvic computer tomography (CT) showed moderate ascites with a thickening of the gastric antrum and proximal small bowel with multiple small mesenteric lymph nodes and mesenteric stranding [Figure 2]. The patient then underwent esophagogastroduodenoscopy, which demonstrated a diffuse thickening of the stomach and duodenum, and biopsies showed heavy infiltration of eosinophils 20 cell/HPF. Colonoscopy and ilioscopy showed diffuse thickening of mucosa and biopsies also showed eosinophilic infiltration of mucosa with more than 40 cell/HPF. Ultrasound guided abdominal paracentesis showed white blood cell count of 1638/mL, 97% of which were eosinophils, lactate dehydrogenase 481 mg/dL, albumin 2.7 g/dL (simultaneous serum albumin 2.2 g/dL). The constellation of clinical presentation and histopathological findings were suggestive of eosinophilic gastroenteritis.

The patient was started on oral prednisone (40 mg/day). Two weeks later, with noticeable symptomatic improvement, the prednisone was tapered off over a 2-week period. After the completion of the steroids, the patient's abdominal pain and ascites completely resolved and a peripheral blood count revealed an absolute eosinophil count of 300/µL (nL <450). Furthermore, IgE level dropped to 105 IU/mL and CT imaging of the abdomen and pelvis showed a complete resolution of the ascites and small bowel thickening. Six months have elapsed since treatment and the patient remains asymptomatic on no medications.

**DISCUSSION**

Eosinophilic gastroenteritis (EGE) is a rare condition characterized by recurrent eosinophilic infiltration of portions of the GI tract presenting with nonspecific GI symptoms in association with peripheral eosinophilia.[2,3] Eosinophilic tissue infiltration can affect any of the three layers of the digestive apparatus with symptoms varying according to the affected layer. The most common grouping of EGE based on the involved layer of the GI tract is known as Klein's classification, which describes three subtypes of EGE (mucosal, muscular, and subserosal), with some degree of overlap.[3,4] Data are insufficient with regard to the true prevalence of EGE and each of its subtypes. However, the mucosal form is the most common followed by muscular and lastly subserosal.[5]

The prevalence of the subserosal form of EGE varies in different studies. A clinicopathological study of 40 patients with EGE showed a predominance of mucosal disease in 57% patients with muscular and subserosal disease accounting for 30% and 13% of cases, respectively.[5] Patients with subserosal EGE have ascites as the source of their symptoms.[5,6] Furthermore, this subgroup is clinically distinct, with abdominal bloating, higher eosinophil counts, and dramatic response to steroid therapy.[4] Eosinophilia is a distinguishing feature of ascites in patients with subserosal EGE.[7] Further characterization of 42 patients with this subtype of EGE by Durieu et al. revealed a 75%
predominance of females who are 40 years and older. Moreover, the study showed that 69% of these patients had blood eosinophilia and 11% had pleural effusion. Eosinophilic infiltrations of the digestive tract were identified by tissue biopsy in 63% of the cases.\[8\]

Available data on the natural history and therapy of EGE remains scarce. Untreated patients can remit spontaneously or progress to develop severe malabsorption. In most cases, the disease is essentially benign and pharmacologic therapy is not always indicated.\[2\] Many patients have been reported to recover spontaneously in a matter of days. The outcome of eosinophilic ascites was favorable in 90% and relapses occurred in 26% of 42 cases studied by Durieu et al.\[9\] More symptomatic patients require therapy with prednisone (20–40 mg/day). A 2-week course produces a dramatic clinical improvement regardless of the histological subtype of EGE. Rapid tapering off over another 2 weeks is sufficient to keep the majority of patients in remission. Most patients have no recurrence; some relapse months to years after the prednisone has been tapered off, yet respond to a short course of repeat treatment. Only a minority of patients require long-term treatment with a low-dose of prednisone (5–10 mg/day). Other patients develop periodic flares after months or years. In addition to prednisone, small studies have described some success in using medications such as oral cromolyn, ketotifen, montelukast, and humanized anti-interleukin-5 antibody.\[5,9\]

In the study by Chen et al., 13 out of 15 patients with EGE required treatment with prednisolone (10–40 mg/day) resulting in complete resolution of symptoms within 2 weeks. However, more than one-third of the treated patients relapsed in 12 months, and 13% required long-term treatment with prednisolone (5–10 mg/day).\[10\] In cases that fail to respond to corticosteroids, treatment with azathioprine or 6-mercaptopurine should be considered.

CONCLUSION

This case report reviews some of the characteristic clinical, laboratory, and histopathological findings of a rare, readily treatable, and easily missed disease. Owing to the relatively nonspecific symptoms, this diagnosis should be considered in patients with ascites of unclear etiology, nonspecific bowel thickening by imaging studies and otherwise, negative workup for parasitic infection and malignancy. Although peripheral blood or ascitic fluid eosinophilia is suggestive, its absence does not exclude the possibility of this diagnosis. Furthermore, prompt therapy with low-dose prednisone may reduce the duration and severity of symptoms. Thus, an awareness of this condition and a timely diagnosis and initiation of treatment could be of major importance.

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Conflicts of interest
There are no conflicts of interest.

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