Eosinophilic Ascites: Singular Presentation of Eosinophilic Gastrointestinal Disorder

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Primary eosinophilic gastrointestinal disorders (EGID) are characterized by abnormal accumulation of eosinophils in the gastrointestinal tract in the absence of secondary causes of eosinophilia and mainly include eosinophilic esophagitis (EoE) and eosinophilic gastroenteritis (EGE) [1]. In EoE, the eosinophilic infiltrate is limited to the esophagus, but in EGE, any gastrointestinal segment might be involved, with the stomach and duodenum being the most commonly affected sites [2]. Eosinophilic ascites (EA) is an unusual presentation of EGE, occurring when there is serosal involvement of the affected section of the bowel [3,4].

A 36-year-old white man with a history of obesity, hypertension, and allergic rhinitis and asthma caused by sensitization to house dust mites came to the emergency department with a 4-week history of epigastric pain, abdominal distention, nausea, and vomiting. Omeprazole 40 mg/d was ineffective. He denied difficulty eating, heartburn, choking, dysphagia, regurgitation, or food impaction. He presented distended abdomen with active bowel sounds and ascites. Hepatomegaly, splenomegaly, and abdominal mass were ruled out. Urgent abdominal ultrasonography confirmed these clinical signs but ruled out portal hypertension. A complete blood count revealed an increase in the white cell count (15 340 m/L with 32% eosinophils [4940 m/L]), elevated erythrocyte sedimentation rate (16 mm/h), and normal results in the liver and renal function tests. Tumor markers, tryptase, antineutrophil cytoplasmic antibodies, celiac serology, and digestive parasites were normal during admission. Serology for human immunodeficiency virus, hepatitis B and C viruses, and *Echinococcus granulosus* was negative. The test for detection of *Helicobacter pylori* was also negative. Serum IgE was elevated (1832 kU/L). Specific IgE values for *Dermatophagoides pteronyssinus*, wheat, and grass pollen (*Phleum pratense*) were, respectively 1.07 kU/L, 6.03 kU/L,

and <0.10 kU/L. Results were negative for egg white/yolk, milk, legumes, fish, seafood, nuts, flours, and *Anisakis simplex*. The skin prick test was only positive to house dust mites (*D pteronyssinus*, 7×7 mm; and *Dermatophagoides farinae*, 4×4 mm). Prick-by-prick tests with egg white/yolk, milk, legumes, fish, seafood, nuts, and flours were only positive to wheat flour (5×5 mm). A thoracic-abdominal-pelvic computed tomography scan showed a moderate amount of pelvic free fluid, which was identified as moderate ascites without lymphadenopathies. A small right pleural effusion was also detected. Ultrasound-guided abdominal paracentesis revealed hematic fluid (total protein, 5.50 g/dL; lactate dehydrogenase, 158 mg/dL; adenosine deaminase, 0.20 U/L; white blood cells, 9100/mL [45% eosinophils]). No cytological signs of malignancy were detected. Laboratory testing of the ascites fluid for bacterial culture and tuberculosis was negative. After 5 weeks of treatment with proton pump inhibitors, upper gastrointestinal endoscopy up to the second part of duodenum did not reveal macroscopic anomalies. Histopathology showed heavy infiltration of eosinophils corresponding to esophageal epithelium (35 cells/HPF); stomach and duodenum biopsies (deep lamina propria) demonstrated nonspecific acute lymphocytic inflammation with eosinophils (40 cells/HPF). With a high suspicion of EA, we started the patient on oral prednisone (60 mg/day) and a diet excluding wheat. Symptoms resolved rapidly after a few days, the blood eosinophil count (1800 m/L) decreased, and the abdominal fluid gradually disappeared (confirmed by ultrasonography). Oral corticosteroids were maintained for 2 weeks and then tapered. The patient was recommended to continue with the wheat-free diet. After 4 months of follow-up under the same diet, the patient was clinically asymptomatic without medication, and his abdominal scan was normal.

We report the case of a young adult atopic patient in whom clinical presentation and histopathological findings confirmed EGE and EA, as previously described [5]. In 1970, Klein et al [6] divided the most common EGE classification into 3 different patterns, namely, the mucosal, muscular, and serosal patterns. The serosal pattern is the rarest of the three and is regarded as the special feature of this pattern. It is considered clinically distinct in that it is characterized by abdominal bloating, high eosinophil count, favorable response to corticosteroids [6], and pleural effusion [7]. All of these characteristics were present in the case we report. Although serosal involvement could not be confirmed by the pathologist, we do not reject the diagnosis. The classification of Klein et al is considered by many authors to be inaccurate, because the layers involved are still not easy to find in daily clinical practice. Only mucosal and submucosal biopsies were taken in almost all cases, and in EGE, clinical and pathologic features often overlap [4]. Fifty percent of patients with EGE have a history of allergy [8]. Nevertheless, IgE antibodies are rarely directed against identified food allergens. A limitation of our study is the simultaneous indication of 2 treatment modalities, which prevents us from knowing which was more effective in the resolution of symptoms. However, given the moderate degree of sensitization and its key role in dietary treatments in other eosinophilic gastrointestinal diseases (eg, eosinophilic esophagitis) [9], we considered wheat to be the potential initial trigger of EGE and EA and therefore recommended avoidance.

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Conflicts of Interest

The authors declare that they have no conflicts of interest.

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