

Eosinophilic ascites, as a rare presentation of eosinophilic gastroenteritis

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Abstract

Background: Eosinophilic ascites is the most unusual presentation of eosinophilic gastroenteritis (EGE), caused by edema and eosinophilic inflammation of the small bowel wall's serosal layer.

Case Report: We report the case of a 37-year-old woman, who presented with diffuse abdominal pain, nausea, abdominal distension, moderate ascites and diarrhea of two weeks duration. The rest of physical and clinical examination was unremarkable, and her past medical history was uneventful. Magnetic Resonance Imaging showed the presence of ascites and diffuse thickening of small bowel wall, but did not detect a primary malignancy in the abdominal cavity; and no signs of portal hypertension or liver damage. Laboratory test results revealed essential peripheral blood eosinophilia, elevated serum IgE and marked increase of eosinophils in the abdominal fluid. Treatment with corticosteroids normalized laboratory tests results, and the ascites resolved immediately.

Conclusions: EGE is a rare entity and it should be kept in mind in patients of unexplained ascites. The absence of primary malignancy on imaging, coupled with marked increase of fluid eosinophilia and immediate response to treatment with steroids, confirm indirectly the diagnosis of EGE. Hippokratia 2014; 18 (3): 275-277.

Key words: Eosinophilic gastroenteritis, ascites, corticosteroids, magnetic resonance imaging

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Introduction

Eosinophilic gastroenteritis (EGE) is a rare condition of unknown etiology characterized by vomiting, diarrhea, protein-losing enteropathy and eosinophilic infiltration of the gut wall. The disease is uncommon, but the incidence is difficult to be estimated because some patients may be misdiagnosed. There is no single diagnostic test or procedure that would point directly to the diagnosis and there are no strict or uniform diagnostic criteria¹. In the present case the definitive diagnosis is made, based on imaging, laboratory results, clinical findings and good response following treatment with steroids.

Case Report

A previously healthy 37-years-old woman presented to the emergency department of the University Hospital Center "Mother Teresa", with generalized abdominal pain, nausea, sporadic non-projectile vomiting, abdominal distention and occasional diarrhea, during the preceding two weeks. She did not report any recent fevers, chills, change in bowel habits, respiratory symptoms, joint swelling or skin rash. She reported no history of alcohol consumption, illicit drugs and was not taking neither medications, nor supplements or herbal compounds. There was no history of atopy, allergy, transfusion, recent travel, liver or heart disease. The patient was afebrile and hemodynamically stable. The physical examination was unremarkable.

Complete blood count revealed an increase in white cell count (WBC) of 13,000 m/L with segment nuclear neutrophils 39%, lymphocytes 20%, eosinophils 38% and monocytes 3%. C-reactive protein, serum liver tests and electrolytes were normal. Serum IgE level was elevated at 838.4 IU/mL (normal <150). Skin prick test results for foods allergens were negative. Parasitic infestations were excluded by negative stool test and serology for Strongyloides, Trichinella, Toxocara and Schistosoma. Gynecologic pathologies were excluded. Diagnostic paracentesis revealed a clear fluid with protein level 4.1 g/dL, albumin 3.4 g/dL, LDH 266 mg/dL and WBC count of 8,800/mL with remarkable eosinophilia of 94%, without cytological signs of malignancy (Figures 1, 2). Laboratory testing of the ascitic fluid for bacterial culture and tuberculosis were negative. Magnetic resonance imaging (MRI) showed the presence of ascites and diffuse thickening of small bowel wall, but no signs suggesting malignancy. MRI also showed edema of the small bowel wall and normal appearance of the liver and portal circulation (Figures 3,4,5,6). Upper gastrointestinal endoscopy showed hyperemia of the esophagus and antral mucosa. Histological examination of the duodenal mucosa showed no eosinophilic infiltrate. The patients refused to undergo laparoscopy. She was treated with prednisone (40 mg/d) with rapid resolution of her symptoms, normalization of the eosinophil count, and slow decrease of the IgE level and gradual disappearance of the abdominal fluid. The

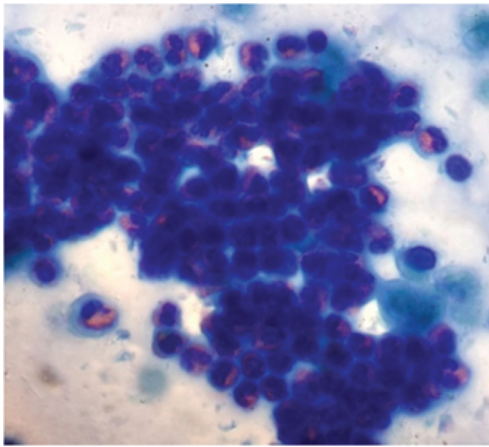


Figure 1: Cytology specimen showing eosinophils in 94% (Giemsa, x 40).

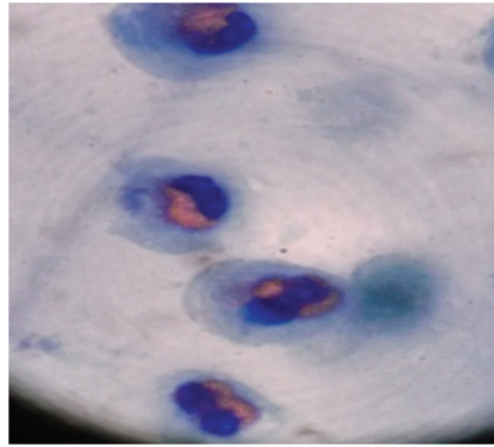


Figure 2: Cytology specimen showing eosinophils in 94% (Giemsa, x 40).

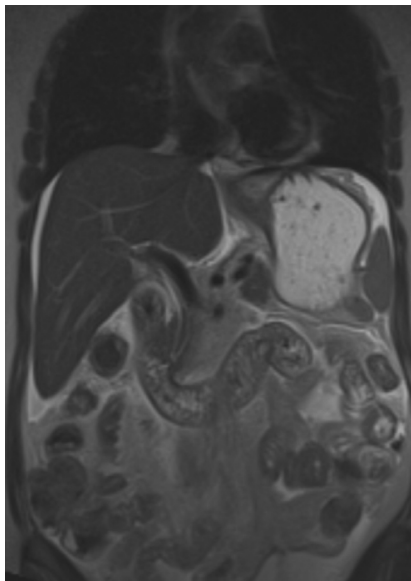


Figure 3: Magnetic resonance imaging, T2-weighted image (coronal view) showing moderate ascites and normal liver structure.

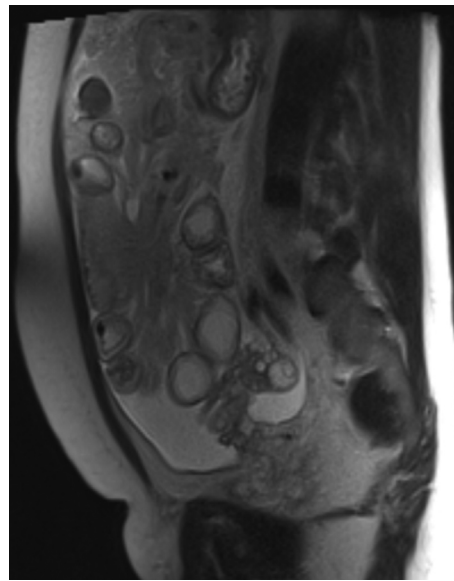


Figure 4: Magnetic resonance imaging, T2-weighted image (sagittal view) showing moderate ascites and normal ovary.

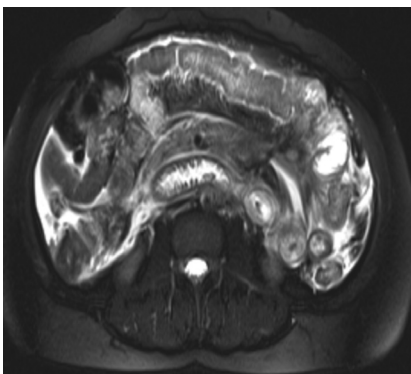


Figure 5: Magnetic resonance imaging, T2-weighted image with fat saturation (axial view) showing small bowel thickening and submucosal edema.

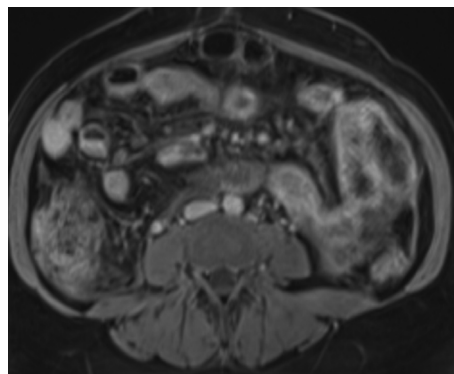


Figure 6: Magnetic resonance imaging, 3D gradient echo images after administration of intravenous contrast medium (axial view) showing small bowel thickening with mild enhancement.

patient's treatment with prednisone was maintained (15 mg every other day).

Discussion

EGE is a rare diagnosed condition that is characterized by recurrent prominent eosinophilic infiltration of the small intestine, generally localized to one level of the intestinal wall, presented with nonspecific gastrointestinal symptoms, in association with peripheral eosinophilia². Data regarding its prevalence and the demographic distribution of the disease is scarce. However, in the last decade, a progressive increase in incidence has been noticed in the both the pediatric and the adult population³. This condition may affect individuals of any age group⁴, typically presents in the third through fifth decades and is more common in the female population⁵.

Pathophysiology of the disease is not clear. Eventually, there is a strong association with atopy; around 80% of the patients reporting a personal history of asthma, eczema, allergic rhinitis or allergy, while half of the patients with gastrointestinal allergy show tissue eosinophilia^{6,7}. Despite difficulties in defining the exact immunological role of the eosinophils in this disease, there is evidence that the eosinophil remain a major effector cell in many types of allergic and non-allergic inflammations⁸. Credible investigations providing an insight into the pathogenesis of EGE support a critical role for allergens, eosinophils, T helper -2 type cytokines and eotaxin in mediating eosinophilic inflammation⁹. Depending on the depth of infiltration of the small bowel wall by eosinophils, there are two forms of EGE: the mucosal and sub-mucosal type, and the sub-serosal. The sub-serosal type is very rare, and eosinophil-rich inflammation affects all the layers of the bowel wall, typically presents with eosinophilic ascites, as in the case presented in this article. This sub-serosal type is also characterized by marked peripheral eosinophilia and has a dramatic response to corticosteroids. The differential diagnosis of Eosinophilic ascites (EA) often leads to confusion and in inability to exclude its multitude of causes in many patients¹⁰. EA should be kept in mind as a cause of unexplained ascites associated with gastrointestinal symptoms. The differential diagnosis include parasitic infestations (*Strongyloides Stercoralis*, *Toxocara Canis*), spontaneous bacterial peritonitis, abdominal tuberculosis, rupture of hydatid cyst, peritoneal dialysis, chronic pancreatitis, vasculitis (Churg-Strauss syndrome), hypereosinophilic syndrome, malignancy (ovarian cancer, Hodgkin lymphoma, peritoneal carcinomatosis) and Crohn's disease. The diagnostic approach comprises both histological and laboratory methods to assess eosinophils and eosinophil activation, as well as tools to assess the allergic disease, while excluding other gastrointestinal diseases, such as food intolerance, infec-

tions, inflammatory bowel disease, parasitic infestations, connective tissue disease, systemic mastocytosis, eosinophilic granulomas and tumors.

Treatment includes elimination or elemental diets and drug therapy using classical anti-allergic agents. Steroids are practically the mainstay of therapy. Most patients with sub-serosal type of EGE respond quickly to steroids. Usually, low-dose maintenance prednisone is needed to keep symptoms under control. Steroid-sparing therapy, such as cell inhibitors, antihistamines, leukotriene receptor antagonists, anti-interleukin or immunosuppressant should be considered¹¹.

In summary, EGE is a rare entity and it should be kept in mind in patients of unexplained ascites. Absence of malignancy, presence of ascitic fluid eosinophilia and a dramatic response to treatment with steroids confirm indirectly the diagnosis of EGE and EA, as was observed in this patient.

Conflict of Interest

Authors declare no conflict of interest.

References

1. Zhou HB, Chen JM, Du Q. Eosinophilic gastroenteritis with ascites and hepatic dysfunction. *World J Gastroenterol.* 2007; 13: 1303-1305.
2. Agostina A, Parenzi A. [Eosinophilic gastroenteritis. A case with predominant involvement of mucosal and muscular layers]. *Minerva Med.* 1994; 85:12: 655-658.
3. Hepburn IS, Sridhar S, Schade RR. Eosinophilic ascites, an unusual presentation of eosinophilic gastroenteritis: A case report and review. *World J Gastrointest Pathophysiol.* 2010; 1: 166-170.
4. Talley NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer and subserosal tissue. *Gut.* 1990; 31: 54-58.
5. Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). *J Allergy Clin Immunol.* 2004; 113: 11-28.
6. Bouhmidi A, Lorente Poyatos R, Romero Cara P, Ibáñez Martín JJ, Casado Caballero F, Quintero Fuentes D, et al. [Eosinophilic enteritis as a rare cause of ascites]. *Gastroenterol Hepatol.* 2003; 26: 480-481.
7. Bischoff SC, Ulmer FA. Eosinophils and allergic diseases of the gastrointestinal tract. *Best Pract Res Clin Gastroenterol.* 2008; 22: 455-479.
8. Moqbel R, Odemuyiwa SO, Lacy P, Ademko DJ. The human Eosinophils. *Wintrobe's Clinical Hematology.* 11th Edition, Lippincott Williams & Wilkins, Philadelphia, USA, 2009, 214.
9. Khan S. Eosinophilic gastroenteritis. *Best Pract Res Clin Gastroenterol.* 2005; 19: 177-198.
10. Gleich GJ, Leiferman KM. The hypereosinophilic syndrome: still more heterogeneity. *Curr Opin Immunol.* 2005; 17: 679-684.
11. Urek MC, Kujundžić M, Banić M, Urek R, Veic TS, Kardum D. Leukotriene receptor antagonists as potential steroid sparing agents in a patient with serosal eosinophilic gastroenteritis. *Gut.* 2006; 55:1363-1364.