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Case Report

Eosinophilic ascites, as a rare manifestation of eosinophilic gastroenteritis: A case report

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Abstract

Eosinophilic ascites (EA) can present as an unusual finding of eosinophilic gastroenteritis. We presented this case to remind eosinophilic acid in cases with unexplained etiology.

A 29-years old man presented to an emergency department with abdominal swelling, progressively worsening nausea, and fatigue over one month. The patient had no history of allergic disease. There was moderate ascites in the physical examination. Percent eosinophil was 60% in peripheral blood smear while IgE level was increased in the serum. There was ascites on abdominal computed tomography (CT) scan. Serum ascites-albumin gradient (SAAG) was non-portal. Eosinophilic infiltration was detected biopsy samples obtained by upper GI tract endoscopy and in bone marrow aspiration and biopsy. The abdominal pain, ascites and all laboratory tests were completely recovered after 12 weeks of prednisolone therapy.

Eosinophilic gastroenteritis should be considered in case of markedly increased eosinophilia in ascites fluid.

Keywords: Eosinophilic Gastroenteritis; Ascites; Abdominal Swelling.

INTRODUCTION

Eosinophilic gastroenteritis (EG) is a disorder characterized by eosinophilic infiltration of different parts or layers of GI tract. The disease is classified as mucosal, muscular or serosal according to histological layer involved. The most common type is mucosal infiltration. In addition, transmural involvement can also occur. The EG diagnosis is made based on criteria such as presence of GI symptoms, eosinophilic infiltration at one or more areas at GI tract proven by biopsy, lacking of eosinophilic infiltration in sites other than GI tract and lacking of parasitic infestation. The EG is typical seen between third and fifth decades of life but it can be seen at any age. The eosinophilic ascites (EA) can manifest as an unusual finding of EG which causes edema and eosinophilic inflammation at gastrointestinal wall and is characterized by vomiting, abdominal pain and diarrhea. In this case, the definitive diagnosis was made based on imaging studies, laboratory tests, clinical findings and dramatic response to steroid therapy. We presented this case to remind eosinophilic ascites in cases with unexplained etiology.

CASE REPORT

A 29-years old man presented to an emergency department with abdominal swelling, progressively worsening nausea and fatigue over one month. There was no history of transfusion, journey, eruption, respiratory symptom, liver disease or cardiac disease. However, he pointed out that food allergy against tomato sauce and mayonnaise. The patient had no history of alcohol consumption, illicit drug use or supplement use. Moreover, there was no familial history of liver disease, coagulation disorder or asthma.

In physical examination, there was no fever and hemodynamics were stable. No pathological finding was observed at skin and mucosal surfaces. Thyroid, cardiovascular system and thorax examinations were normal. In abdominal examination, there was grade 2 ascites but no rebound tenderness or abdominal guarding.

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In laboratory evaluations, the following findings were detected: WBC, 13.2x103/mm3; Hb, 12 g/dL; hematocrit, 37; platelet count 278x103/mm3; and erythrocyte sedimentation rate, 2 mm/hour.

Following findings were observed in peripheral blood smear: percent eosinophil count, 60%; percent neutrophil count, 30%, percent lymphocyte count, 9%; and percent monocyte count, 1%. The patient had normal hepatic function tests, thyroid function tests, serum electrolytes and coagulation tests.

The HIV was found negative in ELISA. The stool examination was negative for amoeba, parasites or other pathogens. Serum IgE level was found to be increased (349 IU/mL) and PDGFRA was negative. Serum tryptase and vitamin B12 levels were 5.42 µg/L (0-114) and 184 pg/mL (150-400), respectively. Skin prick test couldn't be performed since patient was on steroid therapy.

The EF was 60% while PAP was 25 mmHg on echocardiography. On abdominal CT scan, there was diffuse, free fluid at perihepatic area, perisplenic area and between intestinal loops (Figure 1A) and wall thickening (reaching up to 1 cm) in intestinal loops at middle and left quadrants (Figure 1B).

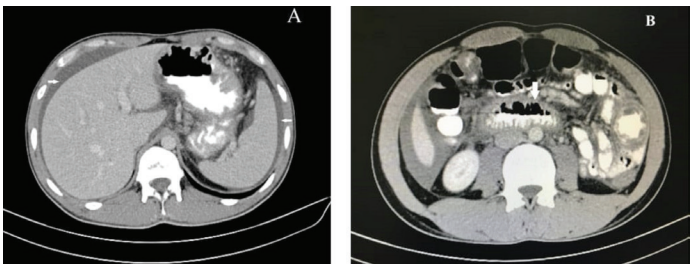

Figure 1 (A). Abdominal CT with oral intravenous contrast demonstrated ascites in perisplenic and perihepatic area (white arrows). **(B)**. Image study shows multiple segment of large bowel wall thickening (white arrow)

In paracentesis, SAAG (0.4) was non-portal with eosinophilia (70%). There was diffuse eosinophilia in pleural fluid (Figure 2A). In the histopathological examination of biopsy samples, there was edema at lamina propria of terminal ileum and mild, chronic inflammatory cell infiltration accompanied by eosinophils (15-20 eosinophils per field) occasionally involving epithelium (Figure 2B, 2C and 2D).

Eosinophilic infiltration was observed in bone marrow aspiration and biopsy (Figure 2E). On upper and lower GI tract endoscopy, there was diffuse mucosal edema, congestion and decrease in vascularity (Figure 2F). Prednisolone (40 mg/day) was prescribed to the patient, which was titrated over 8 weeks.

An additional 4 weeks of low dose prednisolone therapy (5 mg/day) was given. The abdominal pain and ascites were completely recovered after 12 weeks of prednisolone therapy. All laboratory tests, particularly percent eosinophil count and IgE level, were normalized (Table 1).

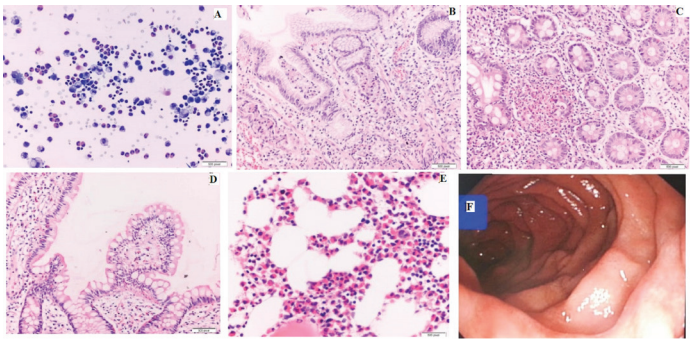

Figure 2 (A). Eosinophils with bilobed nucleus in pleural effusion cytology, MGG x 20, **(B,C,D).** Multiple eosinophils, some infiltrating crypts, and forming crypt abscess in the lamina propria of the gastric mucosa, jejunum, and ileum H&E, x20. **(E)**. Bone marrow biopsy revealed marked increase in eosinophils at all levels of maturation, H&E, x 40. **(F).** Endoscopic examination showed extensive congestion and edema in duodenum

Table 1. Laboratory results of the patient before and after steroid therapy

Parametres	Before therapy	3 months after therapy
White blood cell (normal: 4000–10000/mm^3)	13200	7800
Eosinophils (%) (normal: 0–400/mm^3)	7920(60)	234(3)
Eosinophils (%) (normal: 0–400/mm^3)	349	78

DISCUSSION

Eosinophilic gastroenteritis is a rarely seen disorder with unknown etiology. Although data regarding its prevalence and demographic distribution are limited, an increase has been observed in its incidence in recent years. The etiology and pathogenesis of the disorder hasn't been fully understood. It is thought that EG results from complex interactions among environmental factors, genetic factors and immune system.

The diagnosis is challenging since the EG manifests with several clinical variations and can be made by high level of suspicion. It should have to be considered in the differential diagnosis in patients with gastrointestinal symptoms and peripheral eosinophilia since this combination is observed in 30-80% of patients with EG. Again, there is history of atopia in 80% of these patients. In the literature, asthma, eosinophilic dermatitis, food intolerance and drug allergy have been reported in EG patients presented with ascites. In our patient, there was peripheral eosinophilia and history of intolerance to some foods; however, it was failed to prove by positive skin test as he was on steroid therapy. Positive skin prick test suggests a delayed hypersensitivity reaction against the food.

The mechanism triggering delayed hypersensitivity reaction is unknown but it is thought that food allergens induce lymphocyte transformation in lymphoid tissue at GI tract; as a result, a reaction

occurs in vulnerable individuals via cytokines and IgE released from lymphocytes and plasma cells, respectively. Eosinophilia is discriminating feature of the pathology. The eosinophilia was shown at both peripheral tissues and blood in our case.

The EG is classified as mucosal, muscular and serosal based on GI layer involved (Klein classification). There may be either isolated or synchronous involvement in these layers. Clinical findings depend on the layer involved. Serosal type can present with ascites. Our patient presented with abdominal pain, swelling and nausea. The ascites was defined by both physical examination and sonography; in addition, sampling was performed and SAAG and protein was studied in ascites fluid. Markedly high eosinophil count was detected in the fluid which displayed exudative features. It was reported that EG patients who had ascites with above-mentioned characteristics dramatically responded to steroid therapy, which was also proven by our patient.

In EG, there are some ambiguous macroscopic changes and the definitive diagnosis is made by histopathological examination. The EG causes no specific lesion but may display a wide spectrum of pathological changes from near-normal mucosal appearance to non-specific findings including thickened plica, mucosal ring, stricture, erythema, edema, erosion and ulcer. Histopathological examination may be reported as negative due to patchy involvement. Thus, multiple sampling is essential to prevent misdiagnosis. In our case, endoscopy revealed diffuse edema, congestion and decreased vascularity with eosinophilic infiltration in biopsy samples. The finding of thickening at intestinal wall and ascites suggested serosal involvement in our case.

Systemic causes should be excluded in cases with eosinophilia. Collagen tissue diseases, systemic lupus erythematosus, scleroderma, vasculitis, malignancy and systemic parasitic infestations can present as EG with eosinophilic ascites. In our patients, there was no abnormal clinical or laboratory finding suggestive for vasculitis or collagen tissue disorder. Bone marrow biopsy revealed marked increase in eosinophils at all levels of maturation. No parasite was detected in stool examination. Parasitic infestation was excluded before steroid therapy.

Although spontaneous recovery has been reported in a few cases, medical treatment is required in majority of cases. Although elimination of food allergens is mainstay of therapy, anti-allergic agents are also used in the treatment.

Exclusion of potential food allergen by skin prick test or food challenge tests isn't helpful in most cases. In our case, serum IgE measurement was preferred despite low diagnostic value, since it is a non-invasive method. Anti-parasitic treatment is recommended in all patients with eosinophilic ascites even it is failed to show parasites in stool test. Our case received mebendazole (100 mg, twice daily) over 3 days. Corticosteroid therapy is major treatment modality and patients typically respond to steroid therapy within 2 weeks. In particular, early initiation of steroid therapy is important in terms of both response to lower doses and prevention of ascites, gastric outlet obstruction and conditions requiring surgery. In some patients, it should be kept in mind that relapse may occur after discontinuation of steroid therapy. Thus, azathioprine, montelukast, ketotifen and cromoglycate are used to avoid adverse effects of long-term steroid use. In recent years, monoclonal antibodies against IgE and IL-5 have been used successfully in the treatment in recent years.

Biochemical and endoscopic parameters were used to assess response to steroid therapy. Biochemical parameters including eosinophil count and serum IgE levels were assessed at baseline and during follow-up. Significant recovery was observed in both parameters.

CONCLUSION

Although eosinophilic gastroenteritis is a rate entity, it should be kept in mind in case of unexplained ascites. Thus, eosinophil count in CBC test should be considered in all patients with ascites. It should be considered in ascites patients with eosinophilia in blood including history of atopia.

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