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Case Report

Relapsed Eosinophilic Ascites: A Rare Diagnostic and Therapeutic Challenge in Young Male

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Abstract

Eosinophilic ascites is a rare presentation of eosinophilic gastroenteritis and is often associated with the serosal form. The clinical manifestations are non-specific with history of allergy. The diagnosis is based on hypereosinophilia and eosinophil-rich ascitic fluid in absence of parasitic and extraintestinal diseases. Oral steroid is the treatment of choice giving marvelous response with few chances of recurrence. We present a 16 year-old male having moderate ascites with low serum ascites albumin gradient with high peripheral eosinophilia and gut wall thickening by ultrasonography. The patient was treated by prednisolone 20 mg twice daily for two weeks, then gradual tapering over one month. The patient responded well to treatment with follow up ultrasonography of the abdomen and pelvis showing no ascites after two weeks. After 6 month ascites recurred and repeated investigation diagnosed eosinophilic ascites but with a lower eosinophilia. The patient re-treated by prednisolone 20 mg twice daily with elimination of milk and milk products from the diet.

Introduction:

Eosinophilic gastroenteritis is a rare condition characterized by eosinophilic infiltration of the gastrointestinal tract with subsequent gastrointestinal symptoms [1]. Based on the different layers of the intestinal wall involved, eosinophilic gastroenteritis is divided into 3 groups (Klein classification): mucosal, muscular, and serosal type [2]. The subserosal form of EGE is most commonly associated with eosinophilic ascites [3]. Herein we present a case of relapsing ascites in a young male with a history of seasonal allergy, which was eventually diagnosed and successfully treated as a subserosal variant of eosinophilic gastroenteritis.

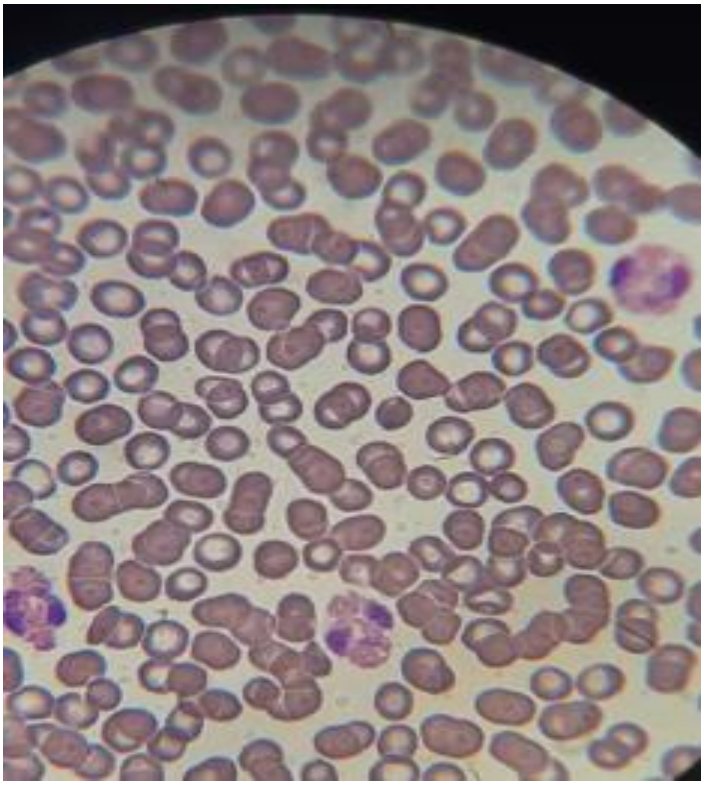
Case presentation:

A 16-year-old male presented to the hospital having generalized abdominal pain and swelling for three weeks with no relation to meals. Nausea and vomiting were reported during the previous week. There was no fever, weight loss, night sweats, chest pain, nor joint swelling. Also there was no history of drugs or herbal compounds. Past history was remarkable for seasonal allergies and skin atopy. On examination there was no pallor nor icterus and no lower limb edema. Abdominal examination revealed increased intestinal sounds accompanied by slight abdominal swelling, abdominal tenderness without rebound and grade 2 ascites was noted. Abdominal ultrasound showed moderate ascites with thickened gastric antrum and small bowel walls. Laboratory investigation revealed normal white blood cell count (10.800/µL) with marked absolute eosinophilia (5,400/µL).

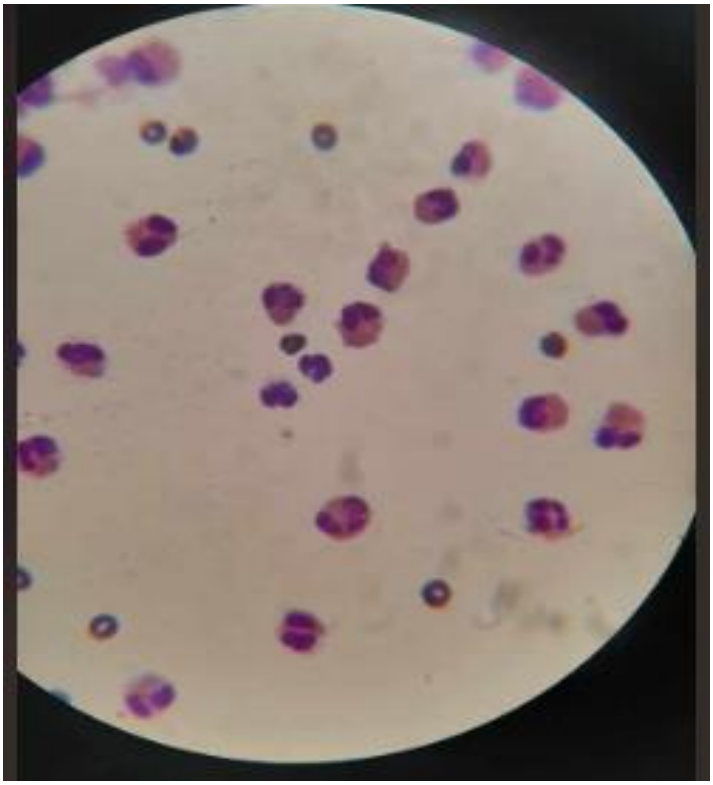
The thyroid function tests, erythrocyte sedimentation rate, C-reactive protein, and urine analysis findings were all normal. Additionally, anti-nuclear antibody, anti-ds DNA, anti-mitochondria antibody tests and urine culture results were all negative, and immunoglobulin E (IgE) level was 139 IU/mL (150 to 300 IU/mL). Stool parasites tests and toxocara, echinococcus and fecal calprotectin tests were also negative. Viral markers for hepatitis A, hepatitis B and hepatitis C were negative. Abdominal paracentesis and cytological examination of ascetic fluid revealed white blood cells 5000/mm3 and eosinophils 4500/mm3 (Figures 1a and 1b). Ascitic fluid Adenosine deaminase for TB was negative and ascitic fluid pH was 7.47; lactate dehydrogenase 250 mg/dL; and albumin= 3.18 g/dL (serum albumin= 4.0 g/dL). Skin prick test was negative.

As a consequence, a diagnosis of eosinophilic gastroenteritis with eosinophilic ascites was made. The patient was treated with prednisolone 20 mg twice daily for two weeks, then gradual tapering over one month. The patient responded well to treatment with follow up ultrasonography of the abdomen and pelvis showing no ascites after two weeks and serum WBCs became 6.8 with eosinophil 10%. The patient was followed every three month.
Sex months later, ascites recurred with abdominal pain. Repeated investigation diagnosed eosinophilic ascites but the number of eosinophilia in ascitic fluid were 2000 (Figure 2).

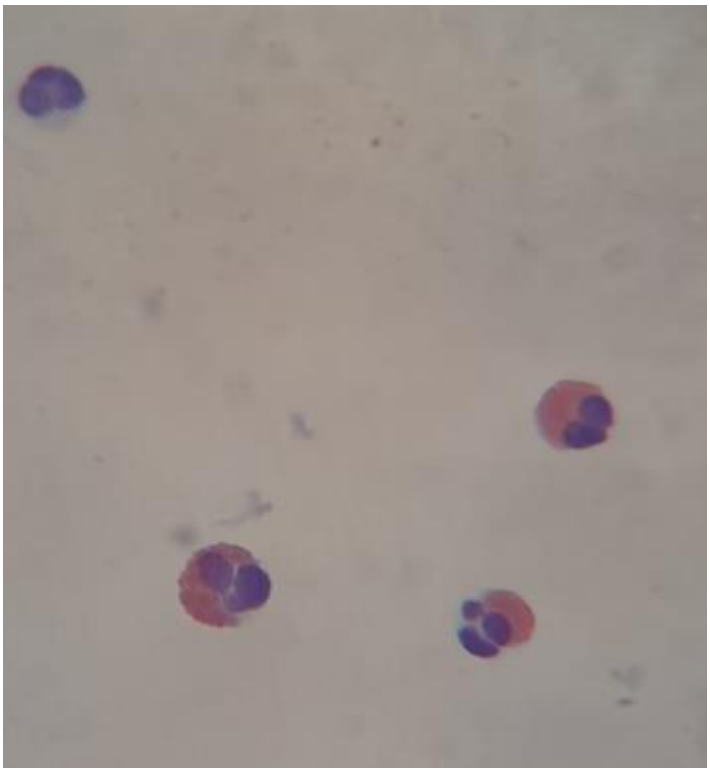
The patient re-treated by prednisolone 20 mg twice daily for two weeks, then gradual tapering over one month with elimination of milk and milk products from the diet. The ascites was completely resolved after two weeks of prednisolone. By the third month of treatment, he was asymptomatic while continuing the elimination diet.



Figures 1a



Figures 1b



(Figures 2).

Discussion:

Eosinophilic gastrointestinal (GIT) disorder is a rare heterogeneous diseases characterized by eosinophilic infiltration of GIT mucosa causing inflammation, without obvious cause of eosinophilia (e.g., drug, parasite, and cancer) [4]. It is classified into three pathologic types depending on the dominant gastrointestinal layer of eosinophilic infiltration [2]. The subserosal type characterized by primarily subserosal disease and eosinophilic ascites is the rarest presentation of EGE [5,6].

The approach to eosinophilic ascites warrants a thorough workup for diverse etiologies like parasitic infection, hyper eosinophilic syndrome, chronic pancreatitis, abdominal tuberculosis, ovarian cancer, and eosinophilic gastroenteritis [7,8]. An atopic predisposition is noted in patients with EGE with a history of allergy reported in 50% patients with EGE [9].

The mainstay of diagnosis of subserosal EGE is confirmation of eosinophil rich ascitic fluid on diagnostic paracentesis and peripheral hypereosinophilia [10].

Treatment of eosinophilic ascites consists of oral steroids (prednisolone 20-40 mg/day) for two weeks, followed by tapering over two weeks with excellent response [5].

Our patient had the serosal form of EGE with eosinophilic ascites because of peripheral blood eosinophilia, an eosinophilic infiltrate of the ascitic fluid, exclusion of alternative diagnoses and excellent response to steroid treatment even after relapse.

The clinical course may be characterized by periods of remission and relapses usually when the steroid therapy is discontinued in up to 50% patients [11]. Steroid-sparing therapy with antihistamines, mast cell inhibitors, leukotriene receptor antagonists, anti-interleukin drugs including ketotifen is useful in the treatment of relapses to avoid the sideeffects of steroids [12, 13].

Conclusions:

Eosinophilic gastroenteritis is a rare disease and warrants a strong clinical suspicion. Relapse was reported in 33% of EGE cases, hence in our case, we needed to ascertain before the administration of oral corticosteroids that ascites recurrence was not of tubercular etiology. The presence of eosinophil-rich ascitic fluid and prompt response to steroids in the absence of other etiologies confirmed the diagnosis of EGE.

Conflict of interests: None
Ethics Approval and Consent to Participate: Not applicable
Consent for Publication: Not applicable

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