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Case Report

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An unusual cause of ascites: eosinophilic gastroenteritis

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ABSTRACT

Eosinophilic gastroenteritis is a rare disease, characterized by eosinophilic infiltrates in the intestinal layers. Its etiology is not well known. Biopsy is mandatory for accurate diagnosis. Clinical presentationis variable and can be seen in numerous diseases. There are no patognomonic findings. Serosal type involvement is the rarestand usually is associatedwith ascites. In this case, we report a 21-year-old femalepatient presented with abdominal pain, diarrhea, vomiting and ascites.Diagnosis of eosinophilic gastroenteritis was made after eliminatingbroad-spectrummimicking causes of tissue eosinophilia.The patient recovered completely after treatment with steroids.

Keywords: Corticosteroids, eosinophilic gastroenteritis, ascites

INTRODUCTION

Eosinophilic gastroenteritis (EGE) despite its infrequent occurrence is one of the most significant primary eosinophilic gastrointestinal disorders. Most common symptom is abdominal pain. All levels of the gastrointestinal tract (GI) from the esophagus to the rectum may be affected.[1] There is a little data about the epidemiology of EGE. The prevalence of EGE in the United States is estimated to be 5.1 per 100,000 based on prior survey data.[2] EGE is well known to be more common among the pediatric population. Although it can affect all ages, the majority of cases in adults ocur from third to the fifth decades of life.[3] Three criteria are required for the diagnosis of EGE: GI symptoms, histological evidence of eosinophilic infiltration of the GI tract and exclusion of secondary tissue eosinophilia causes.[4] We report a case of EGE, that presented with abdominal pain, vomiting, diarhea and ascites.

CASE

A 21-year-old woman was admitted to the emergency room with complaints of abdominal swelling, diarrhea (stool frequency 5-6 times/day) started twenty days ago, and recently appeared epigastric pain and vomiting. İnprevious history it was learned that she was followed up by dermatology with the diagnosis of atopic dermatitis in childhood and adolescence periods. She has not recently traveled to a different place orchange her routinefood consumption. In addition she has no history of abdominal surgery or food sensitivity. On physical examination, her body temprature, blood pressure were normal, pulse rate was 95/ min and respiratory rate 21/min. Chest auscultation demonstrated bilaterally normal breath sounds. Her abdomen was distended, without rijidity and rebound, bowel sounds were evaluated as hyperactive. There was tenderness with dullnessin lower quadrants.

The laboratory tests were monitored as follows: hemoglobin 12,4 g/dL, leukocytes 15,1 10^3/uL (eosinophils 7.9 10^3/uL), platelet count of 359 10^3/uL. Electrolytes level were normal. The serum protein level was 7,5 g/dL; albumin 3,8 g/dL; blood urea nitrogen 14 mg/dL; creatinin 0,5 mg/dL; ALT 140 U/L; AST 288 U/L; LDH 576 U/L; C- reactive protein 0,79 mg/dL; total Ig E 203 IU/mL (upper limit 100 IU/mL), eriytrocyte sedimentation rate (ESR)16mm/hr. Coagulation parameters were normal. Her stool tests were negative for occult blood and enteric pathogens (bacteria, parasites).

A simple chest and abdominal radiograph was normal. A significant amount of ascites was observed in the abdominal ultrasound. Computed tomography (CT) showed free fluid in the perihepatic, perisplenic and pelvic regions (**Figure 1**).

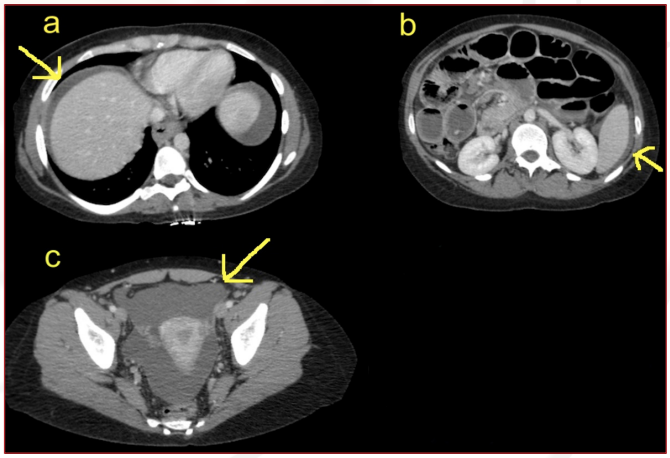

Figure 1. Perihepatic (a), perisplenic (b), and pelvic (c) peritoneal free fluid

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Paracentesis was performed from the ascitic fluid for biochemical, microbiological and cytological analysis. Acid fluid tests were monitored as follows: total protein 5.6 g/L, albumin level 3 g/dL, and caculated serum ascites albumin gradient 0.8 g/dL (<1.1, non-cirrhotic). In addition, ascitic fluid contained 24,000 cells/mm³ cells, 80% of which was eosinophil predominant leukocytes. The glucose level, lactate dehydrogenase (LDH), and adenosine deaminase (ADA) were measured as 74 mg/dL(normal), 241 U/L (normal) and 15 U/L (normal), respectively. The cultures for bacteria, parasites and Tuberculosis were negative. Malignant cells were not observed.

The gynecological examination performed to exclude malignancy was normal. Hematology consultation was made to rull-out hematological causes. Peripheral blood smear was consistent with complete blood count and no atypical cells were found. BCR-ABL, PDGFR Alpha , Flip 1-like 1 gene mutations which are seen in hematological diseases like hypereosinophilic syndrome or chronic myeloproliferative neoplasms were negative. Rheumatologic tests to exclude Celiac, collagen and vasculardiseases that may show similar clinical features were also negative (Anti- tissue transglutaminase, Antinuclear antibodies, ANCA).

Endoscopy and colonoscopy was performed. Gastroscopy showed slightly hyperemic and edematous mucosa in the antrum and duodenum, pylor was slightly deformed. Random biopsies were taken from esophagus and stomach. The histopatology showed focal chronic esophagitis with prominent eosinophilic infiltration (70 eosinophils per HPF, **Figure 2**), chronic gastritis and superficial erosions at the postbulber area.

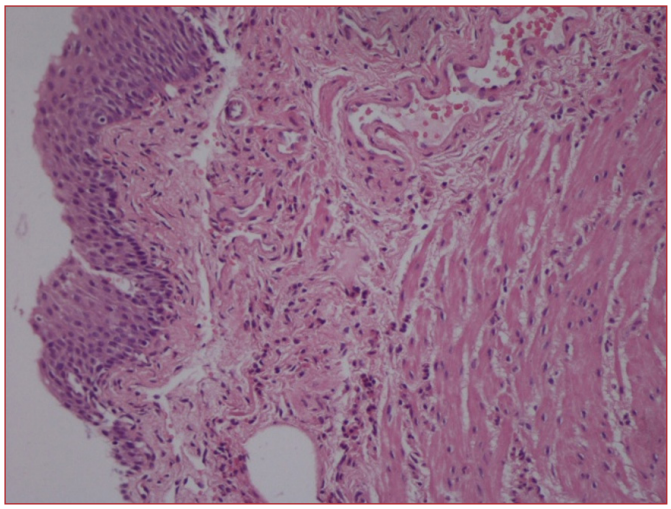

Figure 2. Microscopic image showing70 eosinophils/high power field (HPF) inesophagus(HE, 200x).

Helicobacter Pylori was negative. Endoscopic view in colonoscopy was normal, but randomly taken mucosal biopsies revealed synchronously eosinophilic infiltration of the terminal ileum (≥100 eosinophils/HPF) and ascending colon (≥20 eosinophils/HPF) (**Figure 3**). Accompanied by all these findings and no extraintestinal manifestation, diagnosis of Eosinophilic Gastrpoenteritis was made. 40 mg prednisolone per day was started. As a result vomiting, diarrhea and pain regressed at the third day of therapy. Control laboratory findings and abdominal ultrasound imaging return to normal. The steroid dose was gradually tapered and discontunied in eight weeks. She remained symptom free on a 12-month follow up.

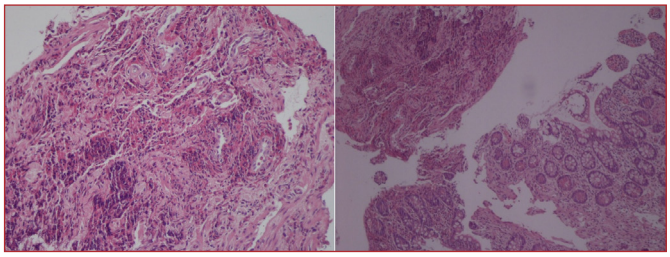

Figure 1. Microscopic image showing increased numbers of eosinophils in ileum. [More than 100 eosinophils / high power field (HPF) Hematoxylin-eosin stain (HE), 200x]

DISCUSSION

İmmunoglobulin E dependent and delayed TH2 cell mediated allergic mechanisms have been showed to be involved in the physiopathology of EGE. Interleukin 5 has also been demonstrated to play asignificant role in the expansion of eosinophils and their accumulation in GI. Chemokines like eotaxin 1, α4β7 integrin also contribute to aggregation of eosinophils in the intestinal wall. Other mediators particularly Interleukin 3,4,13, Leukotrienes and Tumor Necrosis Factor Alpha help to increase eosinophilic trafficking and prolong their activity together with lymphocytes. About %45 to %65 of patients has synchronously allergic diseases like asthma, allergic rinitis, eczema, drug and food intolerance.[5] In previous history of our patient, there was atopic dermatitis in childhood and adolescence periods, consistent with the literature.

As is mentioned above diagnosis is established with combination of clinical , pathological findings and exclusion of secondary tissue eosinophilia causes.[4]

The clinical features of EGE are related not just to the affected segment as well as the affected layer of the gastrointestinal wall. According to Klein's diagnostic classification from 1970, the infiltration may be predominantly at the mucosal, muscular or subserosal layer6[4]

Mucosal eosinophilic gastroenteritis is the most common type and the main affected areas are stomach and duodenum. Patients present with symptoms such as abdominal pain, weight loss, nausea, vomiting,and findings of iron deficiency, malabsorption, protein-losing enteropathy. Common endoscopic appearances are mucosal hyperemia, aphthae and ulcerations. In muscular type, wall thickening occurs and the patient presents with nausea, vomiting, abdominal distension as a result of impaired motility, stricture formation, and rigidity. Serosal type involvement is the rarest form and is seen with signs of eosinophilic ascites, intense peripheral eosinophilia and peritonitis.[7]

There is no established cut-off eosinophilic density on pathology. Along with the pathological findings (altered behavior and distribution of eosinophils, epitelial changes) the proposed numbers of eosinophils based on reported literature are: ≥15 per high-power field (HPF) in the esophagus, ≥30 per HPF in 5 HPF in the stomach, ≥30 per HPF in the duodenum, >56 per HPF in the ileum, >100 per HPF in right colon, >84 per HPF in transverse and descending colon, >64 per HPF in the rectosigmoid colon.[8]

Our case applied with the complaints of nausea, vomiting, diarrhea, abdominal distension and detected eosinophil predominant ascitic fluid. Peripheral eosinophilia occured at laboratory tests. Endoscopic biopsies were taken for differantial diagnosis. Histopathology revealed prominent eosinophilic infiltration and pathological changes in the

structure of mucosal layer. Number of eosinophils were highly above from proposed cut-offs.With all these data, EGE was confirmed after exclusion of parasitic diseases, drug or food allergy, reumatological diseases, hematological malignancies, inflammatory bowel, and celiac diseases. The presence of nause, vomiting, peripheral eosinophilia, eosinophil infiltration of mucosa and eosinophilic ascites indicate mucosal and serosal type of EGE, synhronously.

In the treatment of patients, the relationship of diet with symptoms should be questioned. In some cases there are patients treated with diet but empirical food elimination requires further study for long-term outcomes and efficacy in adults.[9,10]

In a study by Guıllaume Pıneton de Chambrun et al.[11] avoidance of culprit allergens was insufficient to resolve symptoms.The most commonly used treatment was oral corticosteroid therapy in 74% of patients. Corticosteroids were administered orally at a dose of 40-60 mg/day for a short period of time (1 week to 4 months), and subsequently reduced rapidly. Corticosteroid therapy appeared to be effective in 95% of patients. Based on case reports, other treatments include: Leukotriene inhibitors (montelukast), mast cell stabilizers (oral cromolyn), Interleukin-5 inhibitors, Ketotifen, immunosuppressive drugs, biologic agents like Vedolizumab, Mepolizumab (anti-IL 5 antibodiy) and Omalizumab (anti-IgE monoclonal antibody).[12,13]

In our patient symptomes and pathological findings regresed totally after eight weeks course of corticosteroids.

CONCLUSION

Eosinophilic gastrointestinal disorders are among the rare diseases. Its etiology has not been fully elucidated. Symptoms and signs are similar to many other diseases. Diagnosis requires high suspicion and exclusion of various disorders associated with peripheral and tissue eosinophilia. The disease course is variable. Corticosteroids have been used successfully to treat eosinophilic gastrointestinal disorders.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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