Pediatric Oncall Journal
DOI: https://doi.org/10.7199/ped.oncall.2022.23

PEDIATRIC ONCALL
CHILD HEALTH CARE

CASE REPORTS

# EOSINOPHILIC ASCITES

Sareena Kunnugothi.

Department of Pediatrics, Government Hospital, Minicoy, Lakshadweep, India.

**ABSTRACT**
Eosinophilic ascites (EA) is a rare disorder of unknown etiology that has been reported in both adult and pediatric patients. It is probably the most unusual presentation of the syndrome eosinophilic gastroenteritis (EGE). We report a 5-year-old male child who presented with bilateral painless scrotal distension for 5 months with ascites, eosinophilia and eosinophilic ascitic fluid. He received various anti-parasitic agents but had no response but finally responded to steroids.

**ARTICLE HISTORY**
Received 15 June 2021
Accepted 8 August 2021

**KEYWORDS**
eosinophilia, ascites

## Introduction

Eosinophilic ascites (EA) is a part of the rare and heterogeneous condition - eosinophilic gastroenteritis (EGE). EGE characterised by patchy or diffuse eosinophilic infiltration of gastrointestinal tissue.[1] It is an uncommon disorder characterised by tissue and peripheral blood eosinophilia in the absence of a known cause for the latter.[2] Pathogenesis of EGE is not entirely clear. It can present with a wide spectrum of symptoms. Presentations may vary depending on location as well as depth and extent of bowel wall involvement and can be classified into various types based on depth of involvement.[1] The diagnosis of EGE is established on high clinical suspicion in conjunction with suggestive histopathologic findings. No test specific for EGE is available and prior to establishing such a diagnosis, a few gastrointestinal and systemic diseases should be excluded. We present 5 years old who presented with idiopathic eosinophilic ascites and responded to steroids.

## Case Report

A 5-year-old male child, hailing from Nepal, weighing 16 kg, presented with bilateral painless scrotal distension for 5 months. He used to eat mud and he had a pet dog. There was no fever or diarrhea. He had taken anti-tuberculous therapy (ATT) for 7 months and had completed the course a year ago. Details of ATT are not available. On examination, he had bilateral hydrocele, hepatosplenomegaly with ascites. Abdominal ultrasonography (USG) showed ascites and bilateral hydrocele. X-ray chest showed right pleural effusion with right hilar haziness. Mantoux test was negative. His serial hemogram are depicted in table 1. Serum IgE was high (12430 IU/L); and HIV ELISA was non-reactive. Diagnostic paracentesis showed glucose less than 10 mg/dl, ADA less than 10.7 IU/L,

**Table 1.** Serial hemogram

| Date | April 2014 | May 2014 | June 2014 (1st week) | June 2014 (3rd week) | June 2014 (4th week) | July 2014 |
|---|---|---|---|---|---|---|
| Hemoglobin (gm/dl) | 9.6 | | 9.2 | 0.9 | 9.7 | 10.5 |
| White cell count (cells/ cumm) | 22400 | 13100 | 9300 | 9800 | 13600 | 9400 |
| Polymorphs (%) | 22 | 19 | 46 | 26 | 35 | 48 |
| Lymphocytes (%) | 17 | 21 | 43 | 59 | 38 | 46 |
| Eosinophils (%) | 61 | 60 | 8 | 15 | 26 | 6 |
| Absolute eosinophil count (AEC) cells/cumm | 13664 | 7860 | 744 | 1470 | 2522 | 564 |
| Platelet (cells/cumm) | 252000 | 232000 | 238000 | 315000 | 222000 | 271000 |
| ESR (mm at end of 1 hour) | 115 | | | | | |
| Treatment | Albendazole+ diethyl carbamazepine | Ivermectin + Albendazole | | Metronidazole | Steroids | |

**Address for Correspondance:** Dr Sareena Kunnugothi, Rammanjage House, Kudehi Village, Minicoy island, U.T. of Lakshadweep – 682 559.
**Email:** sareenameen@gmail.com

and 8962 cells/cumm with 85% of eosinophils. He was treated with 5 days of albendazole, 3 weeks of diethyl carbamazepine, and 2 days of ivermectin. Stool showed cysts of giardiasis for which he received metronidazole. However, he continued to have peripheral eosinophilia. (Table 1) After a bone marrow examination which had eosinophilia with normal other cells, and a normal echocardiography, he was started on steroids (prednisolone 2 mg/kg/day). Hemogram repeated after one week of starting steroids showed marked response; subsequently his scrotal swelling disappeared. Steroids were given for 1 month followed by gradual tapering. He continued to remain asymptomatic with no peripheral eosinophilia. He subsequently left for Nepal.

**Discussion**

Eosinophilic ascites is probably the most unusual and rare presentation of EGE. EGE is characterised by recurrent eosinophilic infiltration of portions of the gastrointestinal tract in the absence of known causes for eosinophilia.[3] The exact cause or incidence of EGE is not known in India or world and only 280 cases have been reported in the medical literature till date. Kim et al from Korea reported 31 new cases of EGE in his study over 33 years.[4] In India, 7 cases of EGE were reported by Venkataram et al over a period of 10 years.[5] EGE can present in any age, peaking between third to fifth decades with slight male predominance.[6]

The etiology and pathogenesis of EGE is not entirely clear. It seems to come about because of complex interplay of environment, genetics, and the immune system; an association between EGE, collagenoses, allergy and hypereosinophilic syndrome (HES) has been reported.[3]

EGE is classified according to the predominance of eosinophilic infiltration in the different layers of the intestinal wall (Klein classification): mucosal, muscularis and serosal forms.[2] Clinical manifestations depend on the affected layers and range from barely perceptible symptoms to intestinal obstruction or ascites. The most common mucosal form of EGE manifests with abdominal pain, nausea, vomiting, diarrhea, sometimes with hematochezia, and protein losing enteropathy, which may lead to weight loss and malnutrition. Muscularis involvement results in gut wall thickening and may lead to obstruction. The serosal form is the most unusual and leads to eosinophilic ascites.[7,8,9]

In a study of 15 patients with EGE, Chen et al. reported abdominal pain and diarrhea as the most common presenting symptoms. One third of the patients had history of allergy and more than 80% were found to have peripheral eosinophilia. Histological examination revealed that 47% of the patients had mucosal type of EGE, 13% muscular, and 40% subserosal.[10] The prevalence of subserosal form of EGE varies among different studies. A clinicopathological study of 40 patients with EGE showed predominant mucosal disease in 59% patients, muscular 30% and subserosal disease 13%.[2] Our patient did not have any symptoms related to intestinal involvement. He only presented with ascites suggestive of serosal form.

There is no single test or procedure that would point directly to the diagnosis of EGE, and there are no strict or uniform diagnostic criteria for it. When it is suspected based on clinical presentation or the results of tissue biopsy, other causes of hypereosinophilia, such as drug reaction, malignancy, parasites, infection, or systemic disease should first be excluded. Diagnostic evaluation of the patients with suspected EGE should include complete blood cell count and differential, erythrocyte sedimentation rate, C reactive protein, amylase, stool studies for ova and parasites, upper and lower gastrointestinal endoscopy with biopsies, bone marrow biopsy, allergen studies (skin testing and RASTs), and IgE and IL-5 levels. In the presence of ascites, paracentesis should be performed, and the ascitic fluid should be sent for cytology, cell count and differential, gram stain, culture including culture for tuberculosis.[2,3]

Parasitic infection with Strongyloidiasis and Toxocara may present with symptoms of gastroenteritis and eosinophilic ascites, and they need to be excluded prior to making the diagnosis of EGE.[11,12] Serum IgE elevation may point to occult parasitic infestation or an atopic variant of EGE.[13] Our patient was treated for various parasitic infestations with albendazole, ivermectin and DEC but did not have any response suggestive that parasites were not the cause of his eosinophilic ascites. He did have high serum IgE suggestive of atopic variant of EGE.

The diagnosis of eosinophilic ascites might be difficult to make since it is more common in the serosal form of EGE in which systemic eosinophilia might be absent and there may be no eosinophilic infiltration of the gastrointestinal mucosa. Although laparoscopic serosal biopsies may be required for a definitive diagnosis, ascitic fluid eosinophilia and a dramatic response to treatment with steroids indirectly confirm the diagnosis of EGE and eosinophilic ascites.[13] Our patient had no gastrointestinal symptoms and he presented only with ascites. He had a dramatic response to steroids.

Treatment modality depends on the severity of the clinical manifestations. However, corticosteroids are the mainstay of therapy with a 90% response rate in some studies.

Available data on the natural history and therapy of EGE remains scarce. Untreated patients can remit spontaneously or progress to develop severe malabsorption.[13] Corticosteroids are the mainstay of therapy with a 90% response rate in some studies.[13] Treatment modality depends on the severity of the clinical manifestation. Mild diseases are treated symptomatically, and small number of patients enter remission with dietary restriction alone. In patients with presentation or failure of dietary modifications or requiring surgical interventions, steroids are the cornerstone of treatment.[13] Duration of steroid treatment depends upon the response to treatment. If patient requires long term steroids to maintain remission, alternate day steroids for other modalities like anti histamine and mast cell stabilising agent, selective leukotriene receptor antagonist can be used.

**Compliance with Ethical Standards**

Funding None

Conflict of Interest None

**References:**


1. Kaijser R. Zur Kenntnis der allergischen Affektionen des Verdauugskanals vom Standpunkt des Chirurgen aus. Arch Klin Chir. 1937; 188: 36-64.
2. Tally NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. Gut. 1990; 31: 54-58.
3. Nguyen MT. Eosinophilic Gastroenteritis. Available from: http://emedicine.medscape.com/article/174100-overview#showall. Accessed on 14 Dec 2020.
4. Kim NI, Jo YJ, Song MH. Clinical features of eosinophilic gastroenteritis [in Korean]. Korean J Gastroenterol. 2004; 44: 217-223.
5. Venkataraman S, Ramakrishna BS, Mathan M. Eosinophilic gastroenteritis - an Indian experience. Indian J Gastroenterol. 1998; 17: 148-149.
6. Vanderhoof JA, Young RJ. Allergic disorders of the gastrointestinal tract. Curr Opin Clin Nutr Metab Care. 2001; 4: 553-556.
7. Durieu I, Nove-Josserand R, Cathebras P, Durand DV, Rousset H, Levrat R. [Eosinophilic ascites. 2 new case reports]. Rev Med Interne. 1992; 13: 446-448.
8. Kuri K, Lee M. Eosinophilic gastroenteritis manifesting with ascites. South Med J. 1994; 87: 956-957.
9. To Y, Ogawa C, Otomo M, Arai Y, Sano Y, Tashiro Y, Furuta K, et al. [A case of eosinophilic gastroenteritis complicated with ileus and ascites collection]. Arerugi. 1999; 48: 50-55.
10. Chen MJ, Chu CH, Lin SC, Shih SC, Wang TE. Eosinophilic gastroenteritis: clinical experience with 15 patients. World J Gastroenterol. 2003; 9: 2813-2816.
11. Chira O, Badea R, Dumitrascu D, Serban A, Branda H, al Hajjar N, Chiorean E, et al. Eosinophilic ascites in a patient with toxocara canis infection. A case report. Rom J Gastroenterol. 2005; 14: 397-400.
12. Lawate P, Singh SP. Eosinophilic ascites due to Strongyloides stercoralis. Trop Gastroenterol. 2005; 26: 91-92.
13. Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). J Allergy Clin Immunol. 2004; 113: 11-28.