

Parasitic Infection Presenting as Eosinophilic Ascites

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Citation this Article: Poonam Wade, Pramila Borse, Jane David, Radha Ghildiyal, “Parasitic Infection Presenting as Eosinophilic Ascites”, IJMSIR- February - 2021, Vol – 6, Issue - 1, P. No. 239 – 241.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Introduction

Eosinophilic ascites (EA) is a very rare clinical condition of unknown etiology.¹ It affects adults as well as children. It can be associated with abdominal lymphomas, peritoneal dialysis, eosinophilic gastroenteritis, hypereosinophilic syndrome and parasitic infestations.² Helminthic infestation and schistosomiasis are the important parasitic causes of eosinophilia.³

Eosinophilic gastroenteritis (EGE) is a curable disease of gastrointestinal tract, with an approximate incidence of 1/ 100000.⁴ Eosinophilic ascites is generally associated with serosal form of EGE. EGE is characterised by focal or diffuse eosinophilic infiltration of one or more layers of gastrointestinal tract. Ascitic fluid shows eosinophilic predominance with or without peripheral eosinophiia. It should be

considered in case of unexplained ascites with gastrointestinal symptoms specially when associated with peripheral eosinophilia. Prognosis is relatively good. In every case of eosinophilic ascites; it is essential to rule out parasitic infection prior to treatment with steroids. The antihelminthic therapy is efficient in most cases, however some of them may require steroid therapy.

Case report

A 5 year old girl presented with sudden onset of abdominal distension followed by pedal oedema since 4 days. Abdominal distension was associated with dull aching pain. She had history of repeated episodes of passing worms in stool. She had no history of icterus, fever, cough, weight loss, breathlessness or any past history of tuberculosis.

On examination patient was conscious, afebrile and hemodynamically stable. Systemic examination

revealed abdominal distension with ascites without organomegaly and signs of liver cell failure. Complete blood count revealed high white cell count (39100/mm³) with 62% eosinophils (absolute eosinophil count =24242). USG abdomen was suggestive of moderate ascites with normal echotexture of liver. Diagnostic ascitic tap was done which revealed a clear fluid with protein 2.4g/dL and total cell count of 280 /mL with very high eosinophilia of 95%, neutrophils 2% and lymphocytes 3%. Liver function tests were within normal range. Stool for parasitic infection was negative. Serum IgE level was 4161 IU/mL (normal < 180 IU/mL). Koch's work up was negative.

In view of history of repeated episodes of worms in stools and patient also passed round worm in stool during hospital stay, treatment with albendazole 400 mg one dose followed by diethylcarbamazine 6mg per kg per day for 21 days given. During hospital stay patients oedema & ascites decreased and it totally subsided on further follow up. After 3 weeks of diethylcarbamazine therapy patients eosinophil count decreased to 15% with absolute eosinophil count of 2100 which became normal subsequently.

Discussion

Usually Parasitic infection present with nonspecific symptoms. Eosinophilic ascites is a rare manifestation of parasitic infection.² Eosinophilic ascites can be unusual presentation of eosinophilic gastroenteritis. Eosinophilic infiltration generally involves more than one layer of gastrointestinal tract. Depending on the depth of infiltration by eosinophils, there are three types of eosinophilic gastroenteritis: mucosal type, muscularis type and serosal, constituting 70%, 20% and 10% of the diagnosis respectively.⁴ A history of allergy is generally present in patient with EGE.⁵ The

differential diagnosis of eosinophilic ascites include parasitic infestations, 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.⁶

Eosinophilic ascites is characterised by eosinophilic predominance in ascitic fluid with or without peripheral eosinophilia. Peripheral eosinophilia is present in approximately 75% of patients with eosinophilic ascites.⁷ Gastrointestinal system is the most common organ involved followed by respiratory system in a patient with eosinophilia. Visceral larva migrans may be caused by several nematodes of animals such as *Toxocaracanis*, *T.spiralis*, *Ascarissuum* and *Anisakis*. Human toxocariasis is caused by *T.canis*. Sometimes the manifestations of human toxicariasis resemble eosinophilic ascites.

Parasitic infections are generally self-limited and not life threatening. Parasitic infections are most common possible cause of eosinophilia. Treatment is reserved for only symptomatic patients. The antihelminthic therapy is sufficient in most cases but some cases may need therapy with steroids in eosinophilic ascites. Our patient responded well to treatment with albendazole and diethylcarbamazine. Ascites resolved and eosinophilic count decreased further. In a rare case report from Turkey a young patient of parasitic infestation had presented with eosinophilic ascites.² The patient was treated with albendazole for three months which successfully brought the eosinophilic cell count to normal.

In a systemic review of causes of EA by L Pinte among 171 cases of EA, 16 cases were associated with

parasitic infections.⁸ Six types of parasites identified were Toxocariasis (6 cases), Strongyloides stercoralis (5 cases), Giardia (1 case), Trichuris trichiura (1 case) [98], Ascaris (1 case), Enterobius vermicularis (1 case). In one patient parasitic etiology was assumed based on clinical response to albendazole treatment. Some studies on animal subjects showed that parasitic infection caused focal eosinophil infiltrate in the gastrointestinal layers, mimicking eosinophilic gastroenteritis along with marked eosinophilia.⁸

In every case of eosinophilic ascites, it is essential to rule out parasitic infection prior to treatment with steroids because direct treatment with steroids prior to treatment with anthelmintic may worsen the condition.

In our patient, the clinical features, laboratory findings and treatment results favours the diagnosis of parasitic infection (round worm) presenting as eosinophilic ascites. As patient had responded to the treatment endoscopic biopsy of gastrointestinal mucosa was not performed for histological confirmation of diagnosis of EGE.

We conclude that EA should be considered in a patient with ascites in the absence of liver disease, kidney disease, and with gastrointestinal symptoms, especially in the presence of worm infestations along with peripheral eosinophilia.

Acknowledgment: The authors thank Dr Ramesh Bharmal, Dean- T.N. Medical College & BYL Nair Hospital for granting permission to publish this manuscript.

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