Case Report

An Enigmatic Case of Eosinophilic Ascites

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ABSTRACT

Eosinophilic Ascites is a rare clinical entity of unknown etiology. It has been even rarely reported in a case of cirrhosis of liver and portal hypertension. We report a case of 48 years male, chronic alcoholic presented with progressive abdominal distension since one month. He was found to have cirrhosis of liver and portal hypertension along with eosinophilic ascites and peripheral eosinophilia. Patient was asymptomatic for eosinophilia excepting for eosinophilic ascites though presence of ascites can be attributed to portal hypertension. Absence of any single diagnostic test and uniformity in diagnostic criterion, makes diagnosis difficult. Our patient was treated empirically with Ivermectin for suspected occult parasitic infection. His eosinophilia improved with this treatment and absolute eosinophil count on subsequent follow up was normal.

Introduction :

Eosinophilic ascites (EA) is a rare entity and its prevalence remains unknown. It usually occurs as a part of syndrome of eosinophilic gastroenteritis (EGE). The exact etiology of EGE and EA remains idiopathic in majority of the cases; even though atopy (asthma, food allergy), parasitic infections, malignancy, etc are other causes. Some authors have reported it in a postpartum case[1]. It has a wide spectrum of presentation e.g. nausea, vomiting, diarrhoea, protein losing enteropathy, breathlessness, fever with chills ,joint swelling rash, etc[2]. It is reported in adults as well as in paediatric patients[1]. It can affect any age group population even though more common in third to fifth decade of life and more common in females[3,4]. It is even more uncommon in a patient of cirrhosis of liver to have eosinophilic ascites. We report a case of cirrhosis of liver and portal hypertension with eosinophila. Patient responded well to the Ivermectin and Diuretics.

Case Report :

A 48 years, alcoholic male, presented with progressive abdominal distension since one month. There was no history of fever, jaundice, breathlessness, rash, weight loss. There was no past history of allergy neither patient was on any Drugs. Clinical examination revealed presence of bilateral Pedal edema and ascites . There was no icterus or other features of chronic liver disease.

Blood investigtions showed normal Prothrombin time and Liver functions excepting for hypoalbuminemia (2.5 gm/dl). Viral markers were negative. Complete hemogram showed Hb of 10.2 gm%, platelet counts -2.87 lakh/mm^3, WBC-7900/mm^3 with markedly elevated eosinophils (29%). Ascitic fluid analysis was suggestive of high SAAG ascites and total cells 0f 870/mm^3 of which 92% were eosinophils. USG abdomen suggested liver parenchymal disease (cirrhosis), ascites with no evidence of abdominal malignancies. Upper GI endoscopy scopy demonstrated esophageal varices. Multiple biopsies taken from esophagus, stomach and jejunum did not show features of eosinophilic gastroenteritis .

Absolute eosinophil count (AEC) was 1530/mm^3 (reference range -40 to 440) . Stool examination and serology was negative for parasitic infection. Bone marrow aspiration and biopsy demonstrated eosinophilic hyperplasia.

He was diagnosed as a case of Alcoholic liver disease (ALD) with portal hypertension and eosinophilic ascites. He was managed with Furosemide plus aldactone. Ivermectin was given

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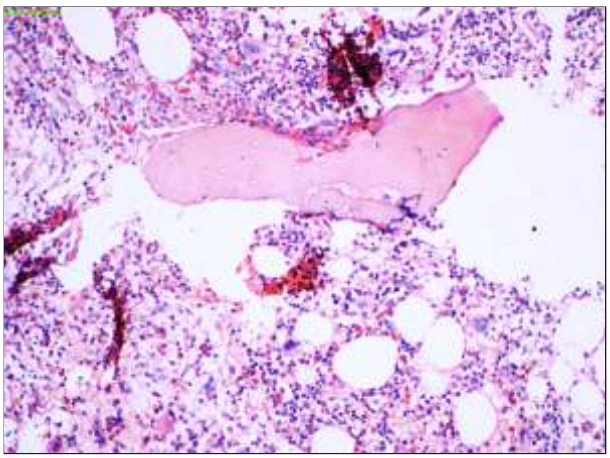
considering occult parasitic infection as a cause for peripheral eosinophilia and eosinophilic ascites.

Patient responded well to treatment, ascites decreased, peripheral eosinophilia reduced from 29% to 8% and ascitic fluid cells reduced to 180/mm^3 (67% eosinophils). He was doing well on with normal absolute eosinophilic counts.



Discussion :

Even though, the exact cause in majority cases remains idiopathic, there is a strong association of EGE and EA with atopy (80%) and almost 50% patients will have tissue eosinophilia[5,6].

The precise pathophysiological mechanisms that result in EA are poorly understood. Depending on the depth of infiltration of gastrointestinal layers, EGE is predominantly divided in two categories viz. mucosal and sumucosal; and subserosal. Subserosal layer; when affected, is characterized by peripheral eosinophilia and has dramatic response to corticosteroids[2].

Eosinophils have primarily innate immunity against parasites in the GI tract which ensures regulatory influence on the functions of other lymphocytes and probably tumor surveillance[7-10].

Strongyloidosis and Toxicarasis are two commonit parasitic infections known to be associated with EA and mandate exclusion before starting corticosteroids. Particularly, strongyloidosis warrants caution with corticosteroids use as it can cause dissemination of infection and death at times.[11,12]. However in our patient Corticosteroids were withheld as he had decompensated liver disease and as there was absence of substantial evidence suggesting parasitic infection .

Diagnosis of EA is difficult as it is common with involvement of serosal layer of GI tract and mucosal biopsies may be negative as in our case. Systemic eosinophilia may also be absent many a times. Presence of peripheral eosinophilia and elevated ascitic fluid eosinophils with along with increased AEC and eosinophilic hyperplastic marrow were suggestive of of EA in our case. Laparoscopic serosal biopsy is required for the definitive diagnosis[13]. Immunohistochemical processing of the biopsied tissue (endoscopic or laparoscopic) is known to deliver more accurate results[14,15].

To complicate the diagnosis, our case also had cirrhosis of liver with portal hypertension. Whether this case had mixed ascites (due to portal hypertension and eosinophilic ascites) or ascites was secondary to portal hypertension alone with occult parasitic infection (eosinophilia improved with antiparasitic treatment) will remain an enigma.

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