Case Report

Eosinophilic Ascites

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ABSTRACT

A 66 years old male patient, a known case of chronic bronchial asthma, presented with complaints of abdominal distention and discomfort of one week duration. There was no history of fever, nausea, vomiting, jaundice or past history of liver disease. Ascitic fluid examination revealed 1700 WBCS with 85% eosinophils. Peripheral blood had WBC count of 37,900/mm^3 with 70% eosinophils. Examination of stool did not reveal any ova or cysts. Diagnosis of eosinophilic ascites was made and patient was investigated.

Key Words : Eosinophilic Ascites

Introduction -

Eosinophilic inflammation of gastrointestinal tract (GIT) occurs in primary eosinophilic gastrointestinal disease (EGID) or secondary to other diseases. It can present as Eosinophilic esophagitis (EE), Eosinophilic gastro enteritis (EGE) or Eosinophilic proctitis (EP) and Food protein induced entero colitis (FPIEC). Last 2 conditions are seen in paediatric age group only while EE and EGE can affect all age groups.

Eosinophilic ascites occurs as a manifestation of EGE where serosal layer of the intestinal wall is mainly involved, giving rise to exudative type of ascites with high eosinophilic cell count. Treatment of choice for this condition is steroids. Association with bronchial asthma & other atopic conditions is often seen.

Case Report -

A 66 years old male patient presented with symptoms of abdominal distention and discomfort since 1 week. There was no history of nausea, vomiting, diarrhea, constipation, fever or jaundice. There was no swelling on feet or face.

Patient was a known case of bronchial asthma since 18-20 years and hypertension since 6 years. He was on tablet nifedipine for hypertension and tablet acebrophylline, and montelukast plus levocetrizine for asthma.

Patient's previous records showed that in November 2010, his peripheral blood eosinophilic count was 24% with total WBC count of 9500/mm^3. He was treated with Diethyl Carbamazine for 21 days, after which his eosinophil count dropped to 2%. His spirometry and allergy screening testing was done in May 2011 which did not reveal positive reaction to any of the allergens tested.

On examination, patient was afebrile, His BP was 140/90. He had no peripheral signs of liver cell failure. Chest examination showed few scattered rhonchi. Per abdomen examination did not show hepatosplenomegaly but evidence of ascites was present. 500ml of ascitic fluid was tapped under aseptic precautions.

Investigations showed Hb to be 11.4 gms%, normal routine urine and stool examination, ESR 11mm at the end of 1 hour, WBC count 37,900/mm^3 with eosinophils 70%, polymorphs 14%, Lymphocytes 16%, and adequate platelets. Immunological tests for diagnosis of parasitic infestation were not available and so not done. LFTs were normal. HBsAg was negative. KFTs and lipid profile were normal. ECG was normal and X-ray chest showed increased bronchovascular markings. Ascitic fluid examination showed light yellow colour, no cobweb, proteins 4 gm/dl, sugar 87mg/dl, cells 1700 WBCS/mm^3 with eosipophils 85%, polymorphs 10% and lymphocytes 5%. Malignant cells were not

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observed. Ascitic fluid was negative for AFB and ADA levels were normal *(Fig.1)*.

With these findings, diagnosis of eosinophilic ascites was made. Patient underwent upper and lower GI endoscopy and mucosal biopsies. Upper GI endoscopy revealed class B oesophagitis with normal stomach and narrowing of first part of duodenum. Biopsy taken from gastric antrum showed only chronic gastritis and no evidence of malignancy. Colonoscopy revealed oedematous mucosa with nonspecific colitis with normal terminal ileum and biopsy of colonic mucosa revealed chronic inflammation with presence of lymphocytes and few eosinophils. MDCT scan of abdomen revealed mild ascites, possibility of annular pancreas and severe focal duodenal narrowing with dilatation of proximal duodenum and stomach, web like mucosal thickening of gastric antrum and gastroduodenal junction mucosa and circumferantial symmetrical wall thickening of cardio-esophageal junction. Short length of descending colon also showed symmetrical wall thickening. Tumour markers CEA and CA 19-9 were negative. Eosinophilic ascites indicated serosal layer involvement. Laparoscopic biopsy was not possible as the patient was not willing. Ascites subsided remarkably after he received only 2 tablets of frusemide + aldactone combination.

Discussion -

Eosinophilic inflammation of GIT occurs as EGID or secondary to other systemic diseases. EP and FPIEC are seen only in paediatric age group while EE and EGE can affect all age groups. All these conditions exhibit prominent eosinophilic tissue infiltration & strong association with allergies[1].

Eosinophils are the granulocytes from myeloid series and they spend 8 days in bone marrow before migrating into peripheral blood (2-6% of peripheral granulocyte pool). Their circulating half life is 8-12 hours, then they move into tissues, GIT, thymus, mammary glands and haemopoetic organs. In GIT they survive for 1 week and then undergo apoptosis[2]. In GIT, highest concentration of eosinophils is seen in caecum and appendix. Esophageal epithelium is normally devoid of eosinophils.

Various experimental studies in animals and humans have shown the relation of respiratory (inhaled) allergens with eosinophilic esophagitis and gastro-enteritis. The link between the two is (TH) 2 helper cells[3]. Almost 80% cases of eosinophilic ascites cases are often accompanied by atopic conditions such as bronchial asthma[4] which this patient had.

EE, first reported in 1978 in children, has increasing prevalence in adults also which is parallel to increasing incidence of allergies. In one study, symptoms of chronic respiratory disease existed in 62% cases. History of allergy or atopy was present in 50-80% cases. Symptoms of EE are similar to GERD but respond only partially to anti reflux management, some patients may present with obstructive symptoms (dysphagia) with or without stricture. This patient had circumferential wall thickening at cardio-esophageal junction.

EGE affects both children and adults. Histopathologically there is intense eosinophilic infiltrate in one or multiple segments from esophagus to rectum and also may occupy various sites through the depth of GIT wall (involving either mucosal or muscular or serosal layer), causing unpredictability of presenting symptoms, ranging from pain, dysmotility, obstruction, bleeding or ascites.

Mucosal EG presents with abdominal pain, nausea, vomiting, dyspepsia, diarrhea, anemia, etc. Muscular EG presents with signs and symptoms of gastric outlet or intestinal obstruction[4] and in one series, 3 out of 7 patients had obstructive symptoms. This patient had thickening and narrowing of cardio esophageal junction, proximal part of duodenum and a short segment of colon, suggestive of muscular involvement. Serosal EG is seen in 10% cases of EGE, presenting as ascites. It is associated with significant bloating and high level of eosinophils in ascitic fluid with good response to steroids[5,6]. This patient is a typical case of serosal involvement and eosinophilic ascites.

Diagnosis of muscular and serosal EGE is many times difficult because of inaccessibility of much of the length of small bowel and difficulty in taking

biopsy from whole depth of the bowel wall because endoscope can take only mucosal biopsy. This patient's mucosal biopsies were not showing much eosinophilic infiltration but bowel wall thickening and ascites indicated muscular and serosal layer involvement.

Evaluation of EGE involves history taking regarding allergies, asthma, familial clustering. Laboratory investigations include peripheral blood smear for eosinophilia, stool exam to rule out parasitic infections, allergy testing and ascitic fluid examination. This patient had history of bronchial asthma but had no symptoms or signs suggestive of parasitic infestation such as abdominal pain, diarrhea, transient skin rash, fever, hepatosplenomegaly. Commonest differential diagnosis of EGE is parasitic infestation. Though eosinophilia is common with parasitic infestations, eosinophilic ascites is described very rarely. Only few cases are described with strongyloidosis and toxocaryosis. This patient had not received any anti helminthic treatment during this episode of ascites nor did he receive diethyl carbamazine. Treatment of EGE includes special diets (especially in children), steroids, mast cell inhibitors, anti histaminics and leukotrienes antagonists .This patient's ascites subsided without steroid therapy. Spontaneous resolution of eosinophilic ascites is described in few cases[4]. As the other possibilities for etiological diagnosis of eosinophilic ascites were ruled out EGID is a strong possibility in this case.

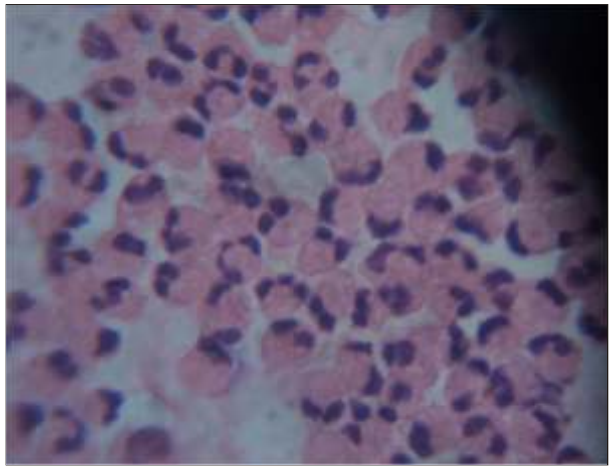

Fig. 1 : **Eosinophils in ascitic fluid**

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