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Eosinophilic Ascites as a Rare Manifestation of Eosinophilic Gastroenteritis

Aakash Shah [a], Nitesh Bassi [a], Pranjal Singh [a], Shishirendu Parihar [a], Ishan Mittal [a], Dipankar Das [a], V. K. Dixit [a] and D. P. Yadav [a*]

[a] Department of Gastroenterology, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India.

Authors' contributions

This work was carried out in collaboration among all authors. Authors AS and NB conceptualized the manuscript, collected data, wrote the first draft of the manuscript. Author PS helped in editing the manuscript draft and along with authors IM, SP and DD managed the literature search. Authors VKD and DPY contributed to supervision of manuscript. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Eosinophilic gastroenteritis (EGE) is a rare gastrointestinal (GI) disorder characterized by nonspecific gastrointestinal symptoms, peripheral eosinophilia, and eosinophilic infiltration of the intestinal wall. The disorder is classified into mucosal, muscular and sub-serosal types. Sub-serosal disease, which is complicated by ascites, is the most severe clinical form of eosinophilic gastroenteritis and requires early corticosteroid therapy. We report a similar case of a 47 year old male presenting with progressive abdominal distention, who on investigations was found to be having eosinophilic and low Serum Ascites Albumin Gradient (SAAG) ascites, patient was worked up further with upper and lower GI endoscopies and mucosal biopsies, Cross-sectional imaging, Bone marrow examination and finally diagnosed to be having eosinophilic ascites secondary to

**Corresponding author: Email: devesh.thedoc@gmail.com;*

Eosinophilic Gastrointestinal Disorders (EGIDs). He was started on steroids in a tapering dose with Six Food Elimination Diet (SFED) and improved remarkably. Patient is off steroids now and is planned to continue now on SFED.

Keywords: Eosinophilic ascites; eosinophilic gastroenteritis; gastrointestinal disorder.

1. INTRODUCTION

"Eosinophilic Gastroenteritis (EGE) represents a member of a family of diseases collectively referred to as eosinophilic gastrointestinal disorders (EGIDs) that includes eosinophilic esophagitis, gastritis, enteritis, and colitis. EGE is a rare disease characterized by focal or diffuse eosinophilic infiltration of the GI tract, especially the stomach and duodenum. It has vague, nonspecific symptoms, including nausea, vomiting, abdominal pain, diarrhea, weight loss, ascites, and malabsorption" [1]. "There is no single diagnostic test or procedure that would point directly to the diagnosis, and there are no strict or uniform diagnostic criteria" [2]. Despite its rarity, eosinophilic gastroenteritis needs to be recognized by the clinician because this treatable disease can masquerade as irritable bowel syndrome. Eosinophilic ascites [3] can be caused by parasitic infections, abdominal tuberculosis, eosinophilic pancreatitis, malignancies, crohn's disease, EGE etc. All other possible causes' needs to be ruled out before labeling the patient as having eosinophilic ascites due to EGE. Patient with sub-serosal variant of EGED present with abdominal pain, distention, eosinophilic ascites, lymphadenopathy, pleural Effusion etc. Mucosal biopsies can be negative in upto 10 % of the cases [4-6]. Bone marrow examination should be done to rule out clonal eosinophilia as cause of hyper-eosinophilic syndromes [7]. Radiographic picture has no characteristic appearance [8]. Treatment is by food elimination commonly consisting of Six Food elimination diet (SFED), oral glucocorticoids are given for patient who present with the obstructive symptoms and eosinophilic ascites.

2. CASE DESCRIPTION

A 47 year old man with a history of bronchial asthma presented with acute onset, gradually progressive abdominal distension for 1 month not associated with pedal edema, shortness of breath/cough or periorbital puffiness. He had generalized body weakness and constipation (passage of stool once in 3 days). Patient was a non addict, and had no similar complaints in past. On clinical examination, general examination was normal. On systemic examination he had hepatomegaly 6 cm below right coastal margin, horse shoe shaped dullness on percussion of abdomen. Respiratory system examination revealed monophonic rhonchi. Other systemic examination was normal. On investigation, CBC showed Hb – 14.1 gm %, leukocytosis of 16900 cells/mm3, differentials being N 22%/L25%/E 49%, with an Absolute eosinophilic count of 8281 / mm^3, Platelets were 2.68 L /mm^3 (Fig. 1). He was found to have low serum ascites albumin gradient (serum albumin 4.1 mg/dl, ascitic fluid albumin 3.21 mg/dl) with eosinophilic predominance (total cell 5673/mm3, neutrophils 02%, lymphocytes 03%, eosinophils 90%) with adenosine deaminase of 18 IU. Serum IgE was >2500 IU/ml (Biological reference <100 IU/ml). Renal and Liver function tests were within normal limit. Stool test for ova and parasites was negative, and serology for hepatitis-B and hepatitis-C were non-reactive. Ultrasonography of abdomen and pelvis showed mild ascites. 2D echocardiography revealed no abnormality. Upper GI Endoscopy revealed edematous mucosa of gastric antrum and body and Colonoscopy showed a normal study. Biopsies from gastric, duodenal ileal, and colonic specimen were suggestive of non specific gastritis, duodenitis, ileitis and colitis respectively. No specific mention about eosinophilic infiltration or predominance was made. Contrast-enhanced computed tomography (CECT) of the abdomen showed a diffuse circumferential wall thickening with post contrast enhancement in the multiple small bowel loops in the peritoneal cavity and associated intense peritoneal fat stranding (Figs. 2 & 3). Bone marrow examination (from Sternum) showed myeloid hyperplasia with remarkably increased eosinophilic precursors and mature eosinophils (44%) (Fig. 4). The patient was started on oral steroids, tablet Prednisolone 1 mg/kg to be tapered over 8 weeks, considering the diagnosis of eosinophilic ascites secondary to EGED, and was explained about SFED. The patient was followed up after 4 weeks, he had significant relief of symptoms, abdominal complaints and ascites resolved totally. Hemogram showed Hb of 14.8 g%, TLC 9100 /mm^3, DLC

N51%/L31%/E15%, Platelets of 3.5 L/mm^3. Ultrasound of abdomen revealed no ascites. The patient is now doing well and is to be tapered off from steroids and to be maintained on Six food elimination diet and will be followed up.

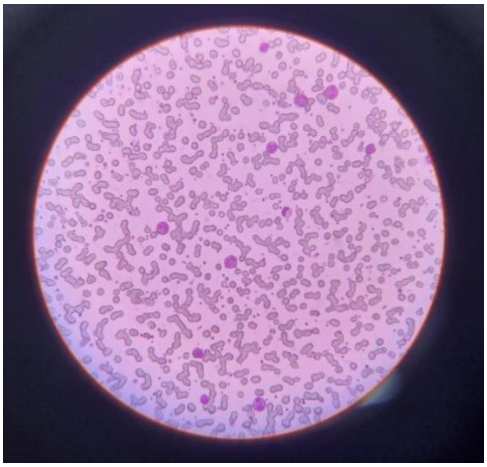

Fig. 1. Peripheral blood smear showing peripheral eosinophilia

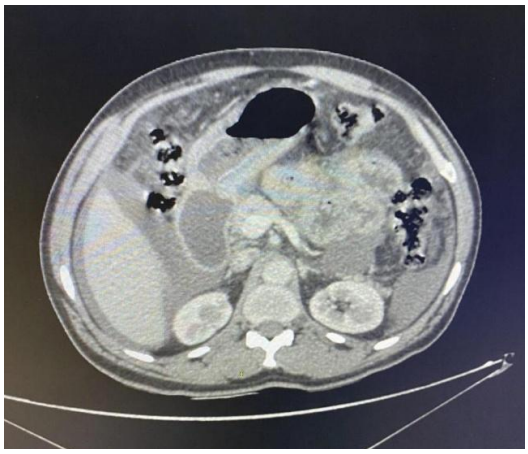

Fig. 2. CECT W/A showing presence of ascites and small bowel loop thickening

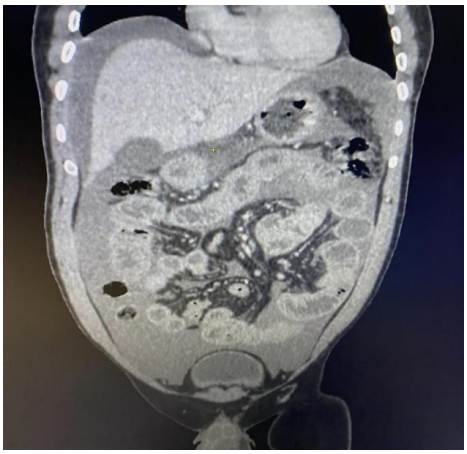

Fig. 3. CECT W/A saggital section showing ascites and small bowel lop thickening

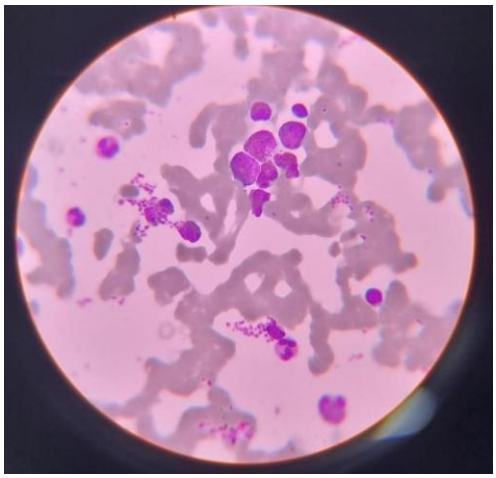

Fig. 4. Bone Marrow examination showing myeloid hyperplasia with eosinophilic predominance

3. DISCUSSION

Eosinophilic gastrointestinal disorder, which includes eosinophilic esophagitis, eosinophilic gastroenteritis, and eosinophilic colitis, is a rare group of heterogeneous diseases characterized by eosinophilic infiltration of gastrointestinal tract mucosa with subsequent inflammation and no apparent eosinophilic cause (e.g., drug reactions, parasitic infections, and malignancy) [9]. In 75% of the cases, it is significantly linked to atopic disease and an allergy-related family history [10]. Male adults in their third to fifth decades are more likely to have EGID [11]. "Eosinophils spend a brief time in peripheral circulation after maturing in the bone marrow and going through selective expansion. From there, they are trafficked to specific tissues like the gastrointestinal tract where they interact with endothelium to produce different types of inflammation with the aid of IL5, chemokines (eotaxin), platelet-activating factor, and cysteinyl leukotriene C4" [9].

"EGID can be of three subtypes: mucosal variety (most common 70%), presenting with diarrhea, melena, and iron deficiency anemia, protein losing enteropathy; muscular variety diarrhea, melena, and iron deficiency anemia, protein losing enteropathy; muscular variety (20%) manifests as intestinal obstruction; and the least common, sub-serosal variety (10%), manifesting as abdominal pain, distention, eosinophilic ascites, Lymphadenopathy etc" [9]. The diagnostic challenge about sub-serosal EGID is a rarity, nonspecific clinical presentation, non-diagnostic endoscopy since the biopsy sample is mostly taken from the mucosa.

The presence of gastrointestinal symptoms, the absence of parasitic or extra intestinal manifestations, a biopsy of the gastrointestinal tract showing eosinophilic infiltration, or radiological findings indicative of the disease like presence of ascites, peripheral eosinophilia are required for diagnosis. The differential diagnosis of Eosinophilic ascites (EA) often leads to confusion and in inability to exclude its multitude of causes in many patients. Eosinophilic Ascites should be kept in mind as a cause of unexplained ascites associated with gastrointestinal symptoms. The differential diagnosis include parasitic infestations (Strongyloides Stercoralis, Toxocara Canis), 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.

Treatment consists of elimination or elemental diet, oral steroids prednisolone 1mg/kg/day [12]. "Alternative second-line medications include mast cell stabilisers (sodium cromoglycate, ketotifen), leukotriene receptor antagonist (montelukast), an anti-IgE monoclonal antibody (omalizumab), anti-IL 5 monoclonal antibody (mepolizumab)" [12]. In 80% of instances, full symptom relief takes place within a week, and within two weeks, the eosinophil count returns to normal. 26% of the cases might relapse [13] and if they do short course of glucocorticoids work well. High AEC at diagnosis was found to be an independent predictor of relapses, as was extensive intestinal involvement [14].

Immunosuppressants such as azathioprine, cyclosporine and other steroid sparing therapies can be used in steroid- resistant patients [15]. In summary, EGE is an uncommon condition that patients with unexplained ascites should be aware of. Indirect confirmation of the diagnosis of EGED and Eosinophilic Ascites, as seen in this patient, is provided by the absence of malignancy, the presence of ascitic fluid eosinophilia, and a significant response to steroid therapy.

4. CONCLUSION

Because of the relatively nonspecific symptoms, this diagnosis should be investigated in patients with ascites of unknown etiology, nonspecific bowel thickening by imaging techniques, and a negative workup for parasite infection and malignancy. Absence of distinctive upper or lower gastrointestinal endoscopic biopsy findings does not rule out the presence of the disease in cases of eosinophilic ascites when there is abdominal discomfort, ascites, and peripheral eosinophilia. Excellent outcomes are achieved with early diagnosis, prompt diagnosis, and oral steroid treatment.

CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this case report and accompanying images.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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