Eosinophilic Ascites, A Rare Presentation of Eosinophilic Gastroenteritis

P Gupta¹, R Singla¹, S Kumar², N Singh¹, P Nagpal¹, P Kar²

Abstract

EGED is a rare entity; it should be kept in mind in patient of unexplained abdominal symptoms. Any age group and any part of GI tract can be involved. Eosinophilia may be absent in the peripheral smear and biopsy may be negative in up to 10% of the cases. Here we are reporting a 40 years old male who presented to us with eosinophilic ascites, a rare presentation of serosal variant of eosinophilic gastroenteritis (EGED).

Introduction

Eosinophilic gastroenteritis (EGED) is a rare disease. Kaijser first reported a patient with eosinophilic gastroenteritis in 1937.¹ Pathophysiology of the underlying disease is not clear, but most of the patients gave a history of atopy or food allergies. Patient with serosal variant of EGED present with eosinophilic ascites. 10% of mucosal biopsies are not helpful to reach to a diagnosis.²,³ Radiographic picture has no characteristic appearance.¹ Treatment is elimination of the foods implicated by skin testing, oral glucocorticosteroids is given for patient who present with the obstructive symptoms and eosinophilic ascites.

Case Report

40 years old male, present with abdominal pain since 15 days, epigastric in location, mainly post prandial, associated with occasional vomiting, which was non projectile, non bilious in nature, no c/o any alteration in bowel habits, no c/o anorexia, weight loss, fever, no past history of similar complaints, or any past history of HT, DM, TB, asthma, food allergies No H/o any prolonged drug intake. Patient was non alcoholic, non smoker non vegetarian. On examination patient was a febrile, PR – 90/min B.P. – 130/90 mmHg, There was no Pallor, Icterus, Cyanosis, Lymphadenopathy, Pedal edema or JVP was not raised. Abdomen was soft; there was no lump or organomegaly. Bowel sounds were present. Cardiovascular systems, Respiratory system, CNS, were within normal limits. On blood investigations, Hb -14gm%, TLC -15600/cumm, DLC –P6300 E2, PLT count -2.5 lakh/cumm, Urea-27mg%,Creatinine -0.9mg%, Na -142meq/L, K -4meq/L, S. amylase – 46u/l. On doing radiological investigation, USG abdomen reveals free fluid, CXR, ECG, were within normal limits. Ascetic fluid analysis reveals TLC- 7200, DLC- P2 L8 E90, sugar- 95, protein- 4.7, ADA 24, gram stain and AFB were negative, and there was no evidence of malignant cells. Further investigation were done which include stool R/M(3), peripheral smear for microfilaria, ANA, ANCA were negative, urine R/M –Normal, LFT-S.bil-0.7,AST/ALT-34/36, S.Pt/Alb-6.2/4, Lipid profile-T.Chol-234, TG-140. AEC was 7400/cumm, Bone marrow aspiration- Normocellular,

Myelopoeisis normal, with increase in Eosinophilic precursors, upto 35% of myeloid precursors, No evidence of parasitic disorders. On doing Upper GI Endoscopy Esophagus, Stomach was showing hyperemia of antral mucosa, Duodenum (1st part) showed evidence of edema along with multiple whitish nodular lesions (Figure 1), enteroscopy showed normal appearing jejunal mucosa, BAMFT showed thickening of gastric, duodenal wall, CECT abdomen revealed thickening of lower part of esophagus, stomach, duodenum, proximal jejunal wall and presence of ascites (Figures 2, 3, 4). Duodenum biopsy showed normal villous pattern with mild inflammation, no eosinophils were present. Patient was started on oral steroid. After two week patient was symptomatically better. His repeat Hgm after 2weeks showed Hb-14gm%, TLC-7800/cumm DLC-P64 L32 M1 E3, also his AEC decreased to 250/cumm. Repeat CECT abdomen revealed similar thickening of esophagus, stomach, duodenal, proximal jejunal wall but ascites had disappeared (Figure 5).

Discussion

Kaijser first reported a patient with eosinophilic gastroenteritis in 1937.¹ EGED most commonly involve stomach and small intestines, but it can involve any part of the G.I. tract.² It mainly affects children and adults but it can affect any age group. Here we are discussing about a middle aged male who presented to us with chief complaints of abdominal pain along with vomiting. On examination patient was found to have free fluid in the peritoneal cavity. Telly has formulated four Criteria for the diagnosis of EGE. These are the presence of abnormal GI symptoms, most often abdominal pain, eosinophilic infiltration in one or more areas of the GI tract, defined as 20 or more eosinophils per high-power field, the absence of an identified cause of eosinophilia, the exclusion of eosinophilic involvement in organs other than the GI tract.² EGE has three variant, Mucosal disease most common variant presented with abdominal pain, vomiting, diarrhea, protein-losing enteropathy, Muscle layer disease presents with bowel wall thickening, and gastric and small bowel obstruction, Subserosal disease, rarest form, associated with eosinophilic ascites.³ So this patient presented to us with feature of serosal and muscular variant of EGED. Peripheral blood eosinophilia is present in 20-80% of the patients. Absolute eosinophilic count is nearly 2000 eosinophils (Eos)/µL in patients with mucosal variant, 1000 eos/µL in patient with muscular layer involvement, and 8000 eos/µL with serosal layer involvement. CBC of the patient in our case showed an AEC OF 7,400. Before we make a diagnosis of EGED other secondary

¹Senior Resident, ²Director, Professor of Medicine, ³Post Graduate Student, ²nd Year, Department of Medicine, ³Post Graduate Student, ²nd Year, Department of Radio Diagnosis, Maulana Azad Medical College, New Delhi

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causes for eosinophilia must be ruled out which include stool examination for ova and parasitic cyst, skin prick tests for allergic reaction to inhalant allergens and food to identify sensitization to specific allergens, connective tissue profile to rule out connective tissue disease and bone marrow examination to rule out any hematological malignancy. In our cases work up for secondary causes was negative. Radiographic picture has no pathognomic appearance in EGED and may be absent in upto 40%. Gastric folds may be enlarged, with or without nodular filling defects, strictures, ulceration, or polypoid lesions sometime found. In EGE with muscle layer, localized involvement of the antrum and pylorus, causing narrowing of the distal antrum and gastric retention may be present. The small intestine may show dilation, with increase in the thickness of the folds. Ultrasound and CT scans show thickened intestinal walls and localized lymphadenopathy. Ascetic fluid is usually present in patients of serosal variants. Abdominal paracentesis shows eosinophilic ascites in such patient. Exploratory laparotomy may be indicated, especially in patients with serosal eosinophilic gastroenteritis. In our patient on USG, BAMFT, CECT ABD, UGI ENDOSCOPY there was thickening of the proximal part of small bowel along with thickening of the antrum of stomach was found, along with presence of ascites. The ascites was exudative in nature and was full of eosinophils. In EGED Endoscopy shows grossly prominent mucosal folds, hyperemia, and ulceration. Few patient shows presence of nodularity. Histopathology shows increased numbers of eosinophils in the lamina propria, muscularis and serosal layers. In EGED because of patch involvement of GI tract by eosinophils it is advised to take at least 6 biopsy from abnormal and normal area to reach to a diagnosis and it has been observed that in upto 10% of the cases biopsy may not be helpful to reach to a diagnosis because of mucosal sparing. Telly has described case series of 40 patient of EGED in which 4 patient were biopsy negative like in our case. Patient was started on steroid and his symptoms and ascites has resolved and AEC decreased significantly. From India only one such case was reported previously.
Conclusion
EGED is a rare entity, it should be kept in mind in patient of unexplained abdominal tract symptoms, eosinophilia may be absent in the peripheral smear and biopsy may be negative in upto 10% of the cases.

References