BMH MEDICAL JOURNAL

BMH Med. J. 2025;12(1):8-13. **Case Report**

An Unusual Case Of Ascites

Mansoor Ahammed, Abhishek Konnur, Reshmi KR, Ayshath Shamna, Biju I K, Gouri S, Sylesh Aikot, Ramalingam T, Mithun VV, Sangeet Krishnan, Anju Chacko

Departments of Medical Gastroenterology, Surgical Gastroenterology and Pathology, Baby Memorial Hospital, Kozhikode, Kerala, India

Address for Correspondence: Dr. Mansoor Ahammed, Department of Medical Gastroenterology, Baby Memorial Hospital, Kozhikode, Kerala, India. E-mail:dr.mansoor.ahammed@gmail.com

Abstract

Eosinophilic gastroenteritis (EGE) with eosinophilic ascites is a rare disease that can present with abdominal pain and can be challenging for a clinician to differentiate amidst the numerous causes of abdominal pain with ascites. Due to the non-specificity of symptoms and scarcity of diagnostic guidelines, a high index of suspicion is required to diagnose EGE and thereby defer unnecessary surgical interventions. Herein, we present the case of a middle aged female who had presented to the Gastroenterology department with severe abdominal pain and her basic blood test showed hypereosinophilia mimicking an acute abdomen. The relevance of the case resides in highlighting the fact that such rare diseases are often missed and a broader diagnostic spectrum should be considered while evaluating cases of undifferentiated acute abdomen. The clue for the diagnosis may be hidden in the basic lab reports.

Keywords: Acute abdomen, Eosinophilic gastroenteritis, Peripheral eosinophilia, Steroids

Introduction

Eosinophilic gastroenteritis (EGE) is a rare disease affecting the gastrointestinal (GI) system with a reported incidence of 1-30/100,000 population [1,2]. It was first described by Kajiser in 1937, and since then, about 300 cases have been described in the literature [3]. EGE manifests with varying GI symptoms which can mimic an acute abdomen and is characterized by eosinophilic infiltration of the GI tract. The most common anatomic sites involved are the stomach and proximal bowel; although, it may affect any part of the GI tract from the esophagus to the rectum [4]. EGE is clinically diagnosed by the triad of abnormalities of GI function, eosinophilic infiltration of GI tract, and exclusion of other diseases causing peripheral eosinophilia. EGE can masquerade among the numerous causes of the acute abdomen and can pose a diagnostic challenge. Recent studies have shown that the prevalence of EGE is increasing, and therefore, it is imperative that clinicians are acquainted with this emerging medical condition [5,6].

Case Report

A middle aged female, with no comorbidities presented with complaints of abdominal pain since 1

month, which was colicky type, intermittent and localized to periumbilical area and lower abdomen. It was associated with 3-4 episodes of loose stools per day, with increased gastrocolic reflux and occasional vomiting. She also complained of decreased appetite and weight loss of 2kg in 1 month.

She had similar symptoms 6 months back, for which she was evaluated; CECT abdomen reported left paraumbilical hernia with intermittent strangulation and underwent umbilical hernia surgery. Her symptoms were relieved for a duration of 3 months. On follow up, she complained of similar complaints, hence further evaluations were done - peripheral smear reported microcytic hypochromic anaemia with mild eosinophilia. OGD showed pan gastritis with Rapid urease test (RUT) positive. She was treated with H pylori eradication treatment and her symptoms improved.

On examination, she had rebound tenderness with shifting dullness. On investigation, she was found to have anaemia (Hb-9.1, MCV-73.4), eosinophilia (48%, WBC-8.9 x 10^3) and normal liver, renal function tests. USG abdomen showed no liver or renal disease, moderate to gross ascites, mildly distended GB with minimal sludge, diffuse subcutaneous edema noted in anterior abdominal wall in umbilical and periumbilical region. To rule out a surgical cause for the abdominal pain, CECT abdomen (**Figure 1**) showed hepatomegaly (16.3cm), circumferential long segment wall thickening involving gastric antrum, pylorus and small bowel loops - likely infective/inflammatory pathology, mild to moderate ascites with right mild pleural effusion. Ascitic fluid analysis showed low SAAG high protein (S.albumin-3.8, ascitic fluid albumin-2.9, ascitic fluid protein-5) with high eosinophilic predominance (WBC-4480, eosinophils-90%). Ascitic fluid cytology (**Figure 2**) showed eosinophilic rich effusion and ADA was negative. Upper OGD (**Figure 3**) showed pangastritis with duodenitis (RUT negative), Endoscopic biopsy showed normal duodenal mucosa with inflamed gastric mucosa with mucosal and submucosal infiltration of eosinophils. Colonoscopy (**Figure 4**) showed normal mucosal study and biopsy showed edematous and inflamed colonic mucosa with normal ileal mucosa. HPR of colonic mucosa showed eosinophil count 15/HPF. Considering the very high eosinophil count in ascites with thickened gastric wall and mucosal infiltration with eosinophil diagnosis of eosinophilic enteritis with ascites was considered.

She was managed with short course of steroids for 4-6 weeks and tapered off. She is asymptomatic and ascites has resolved. Antihelminthic coverage was also given. Repeat hemogram differential counts showed resolution of eosinophilia and USG screening showed no ascites. Plan is to keep her under close follow up and do bone marrow biopsy and work up to rule out connective tissue disorders.

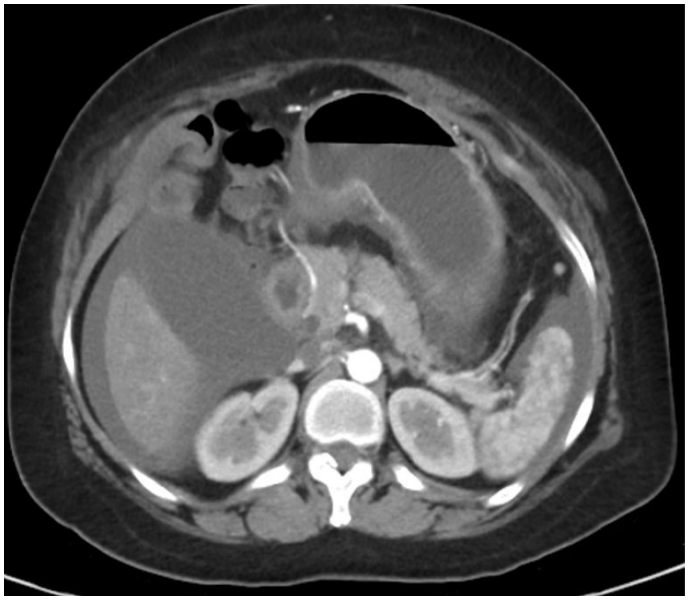

Figure 1: CECT abdomen: Thickened Gastric mucosa

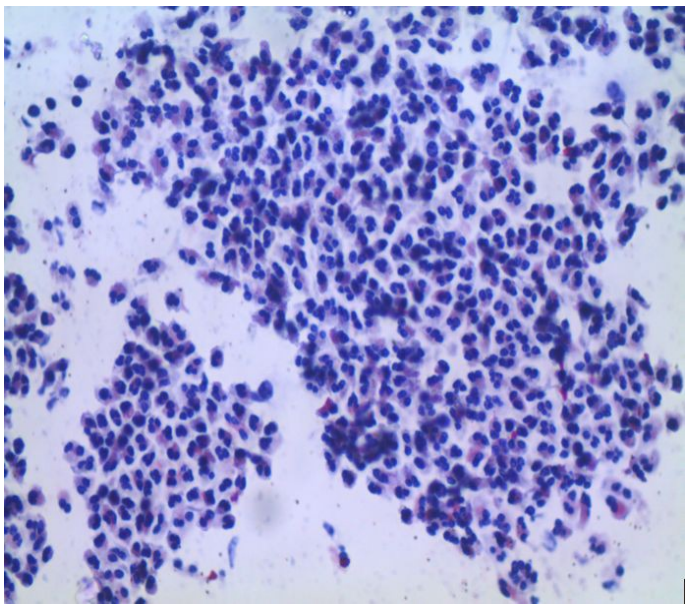

Figure 2: Ascitic fluid cytology showing eosinophils

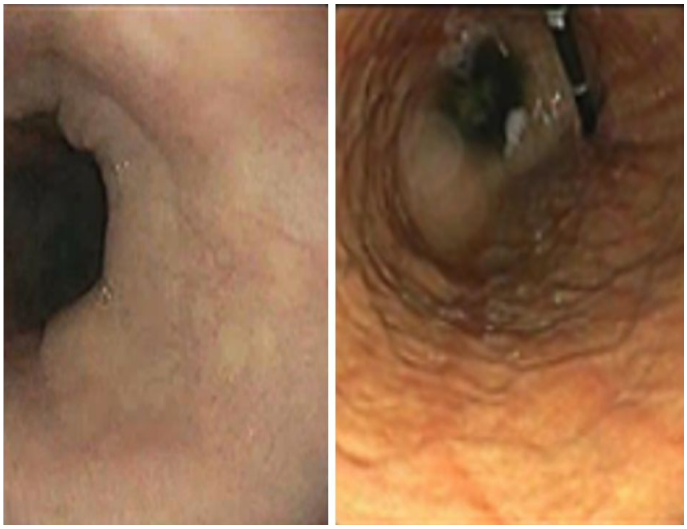

Figure 3: OGD: Erythema in antrum, fundus and body

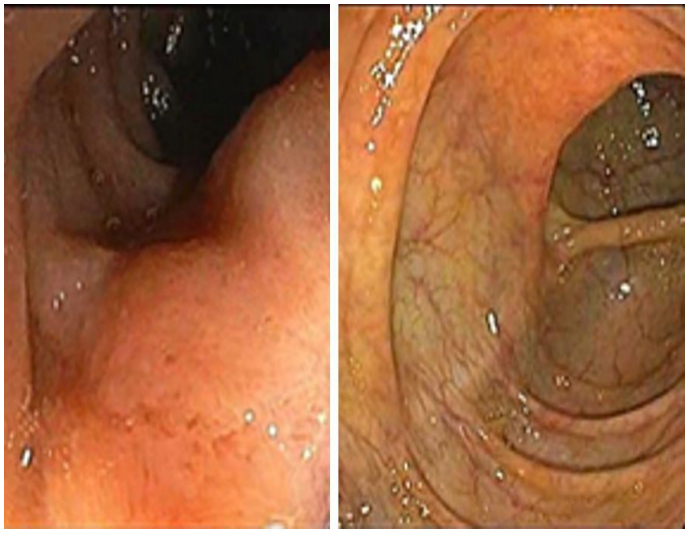

Figure 4: Colonoscopy - Normal study

Discussion

EGE belongs to a spectrum of GI disorders which also includes eosinophilic esophagitis, eosinophilic gastritis, eosinophilic enteritis, and eosinophilic colitis [7]. EGE may affect any age group, although the peak incidence is between the third and fifth decade of life with a slight male predominance [8,9]. Based on the anatomic location of eosinophilic infiltration in the intestinal wall, Klein et al. in 1970 classified the disease as mucosal, muscularis, or serosal type [10]. The exact etiopathogenesis for EGE remains unknown, but eosinophilic inflammation of the GI tract from an allergic response is often thought to be an underlying cause. The clinical manifestations of EGE depend on the site, as well as the depth of eosinophilic infiltration of the gut wall involved. Involvement of the mucosal layer often presents with abdominal pain, nausea, vomiting, bloody diarrhea, fecal occult blood loss, anemia, or protein losing enteropathy. Involvement of the muscular layer may present with signs and symptoms of gastric outlet and intestinal obstruction while the involvement of serosal layer presents with ascites [11]. Rarely, the serosal type of EGE may present with eosinophilic ascites without peripheral eosinophilia. Due to the rarity of the disease and the absence of guideline recommendations, evaluation and management of EGE is challenging. Diagnosis of EGE requires a high index of suspicion when no other cause can wholly explain the clinical features. The history should be focused on eliciting allergies to food, medicines, or environmental agents, including a family history of atopy. Laboratory investigations often reveal peripheral eosinophilia but it is not necessary for the diagnosis of EGE. In patients with ascites, paracentesis may reveal ascitic fluid eosinophilia. Stool examination should be considered to rule out evidence of parasitemia. CT in EGE may show nodular, irregular folds, and thickening of the stomach and intestinal walls. Gross endoscopic findings in mucosal type EGE may include mucosal hyperemia, ulcerations, and focal erosions [12]. The conclusive evidence is by the histopathological demonstration of eosinophils in the layers of GI walls. Restricted diet and steroids are the mainstays of treatment of EGE. Other adjunct treatment modalities include mast cell inhibitors, leukotriene receptor antagonists, antihistamines, and proton-pump inhibitors [11]. Several novel anti-eosinophil therapies have been reported effective in the recent literature which includes neutralizing monoclonal antibodies against Interleukin-5, anti-tumor necrosis factor alpha, anti-immunoglobulin (IG) E monoclonal antibodies, selective TH 2 cytokine inhibitors, intravenous IG, and interferon-α [13-17]. Eosinophilic ascites is a unique presentation of serosal eosinophilic gastroenteritis. Entire bowel wall is usually involved in serosal eosinophilic gastroenteritis. The clinical relevance of the case lies in the fact that EGE is a treatable condition and may mimic an acute abdomen which can be missed easily if a high clinical suspicion is lacking.

In our case, she had unexplained abdominal symptoms with hypereosinophilia, suspicion of eosinophilic gastroenteritis should be considered. She had gastrointestinal symptoms like altered bowel for at least 6 months with features of subacute intestinal obstruction suggestive of mucosal involvement and abdominal free fluid suggestive of serosal involvement. Her ascitic study showed low SAAG, high protein, high eosinophilic predominant and cytology also confirmed it. So if clinical suspicion is high, discussion with pathologist is essential.

There are several reports which have shown that EGE can masquerade among the numerous causes of the acute abdomen such acute appendicitis, intussusception, pancreatitis, and cholecystitis [18-21]. A study by Abassa et al. in 2017 reports that the incidence of EGE is often underestimated due to several factors such as non-specificity of EGE symptoms, insufficient data in the literature, low indices of clinical suspicion, and lack of histopathological evidence [2]. Another study in 2019 by Sunkara et al. also reports that EGE is commonly underdiagnosed or underreported and that its prevalence is expected to increase [22]. In the above context, we believe that this case report will be an eye opener for all emergency physicians to consider this unusual, but emerging disease along with other differentials of acute abdomen.

Conclusion

EGE is a rare disease affecting the GI system with symptoms ranging from abdominal pain and

dyspepsia to obstruction and ascites. The diagnosis is confirmed by the presence of eosinophil infiltration of the walls of the GI tract. EGE shows an excellent response to steroids and adjunctive therapy. Due to its potential for underdiagnosis, the physician should exercise a high index of suspicion when the symptoms cannot be explained by other common causes.

References

1.Cianferoni A, Spergel JM. Eosinophilic esophagitis and gastroenteritis. Curr Allergy Asthma Rep 2015;15:58.

2.Abassa KK, Lin XY, Xuan JY, Zhou HX, Guo YW. Diagnosis of eosinophilic gastroenteritis is easily missed. World J Gastroenterol 2017;23:3556-64.

3.Kaijser R. Zur kenntnis der allergischen affektionen des verdauugskanals vom standpunkt des chirurgen aus. Arch Klin Chir 1937;188:36-64.

4.Ingle SB, Hinge CR. Eosinophilic gastroenteritis: An unusual type of gastroenteritis. World J Gastroenterol 2013;19:5061-6.

5.Du L, Shen J, Kim JJ, Yu Y, Ma L, Dai N. Increased duodenal eosinophil degranulation in patients with functional dyspepsia: A prospective study. Sci Rep 2016;6:34305.

6.Hui CK, Hui NK. A prospective study on the prevalence, extent of disease and outcome of eosinophilic gastroenteritis in patients presenting with lower abdominal symptoms. Gut Liver 2018;12:288-96.

7.Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). J Allergy Clin Immunol 2004;113:11-28.

8.Chang JY, Choung RS, Lee RM, Locke GR, Schleck CD, Zinsmeister AR, et al. Shift in the clinical spectrum of eosinophilic gastroenteritis toward the mucosal disease type. Clin Gastroenterol Hepatol 2010;8:669-75.

9. Talley NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: A clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. Gut 1990;31:54-8.

10. Klein N, Hargrove R, Sleisenger M, Jeffries G. Eosinophilic gastroenteritis. Medicine (Baltimore) 1970;49:299-319.

11. Khan S. Eosinophilic gastroenteritis. Best Pract Res Clin Gastroenterol 2005;19:177-98.

12. Katsumi N, Yamaguchi Y, Yamato T, Abe T, Ishida H, Takahashi S, et al. Multiple ulcerative lesions of the stomach: A rare case of eosinophilic gastroenteritis. Gastrointest Endosc 2002;56:762-4.

13. Fenoglio LM, Benedetti V, Rossi C, Anania A, Wulhfard K, Trapani M, et al. Eosinophilic gastroenteritis with ascites: A case report and review of the literature. Dig Dis Sci 2003;48:1013-20.

14. Turner D, Wolters VM, Russell RK, Shakhnovich V, Muise AM, Ledder O, et al. Anti-TNF, infliximab, and adalimumab can be effective in eosinophilic bowel disease. J Pediatr Gastroenterol Nutr 2013;56:492-7.

15. Foroughi S, Foster B, Kim N, Bernardino LB, Scott LM, Hamilton RG, et al. Anti-IgE treatment of eosinophil-associated gastrointestinal disorders. J Allergy Clin Immunol 2007;120:594-601.

16. Shirai T, Hashimoto D, Suzuki K, Osawa S, Aonahata M, Chida K, et al. Successful treatment of eosinophilic gastroenteritis with suplatast tosilate. J Allergy Clin Immunol 2001;107:924-5.

17. Lu E, Ballas ZK. Immuno-modulation in the treatment of eosinophilic gastroenteritis. J Allergy Clin Immunol 2003;111:S262.

18. Tran D, Salloum L, Tshibaka C, Moser R. Eosinophilic gastroenteritis mimicking acute appendicitis. Am Surg 2000;66:990-2.

19. Huang FC, Ko SF, Huang SC, Lee SY. Eosinophilic gastroenteritis with perforation mimicking intussusception. J Pediatr Gastroenterol Nutr 2001;33:613-5.

20. Maeshima A, Murakami H, Sadakata H. Eosinophilic gastroenteritis presenting with acute pancreatitis. J Med 1997;28:265-72.

21. Jimenez-Saenz M, Villar-Rodriguez JL, Torres Y, Carmona I. Biliary tract disease: A rare manifestation of eosinophilic gastroenteritis. Dig Dis Sci 2003;48:624-7.

22. Sunkara T, Rawla P, Yarlagadda KS, Gaduputi V. Eosinophilic gastroenteritis: Diagnosis and clinical perspectives. Clin Exp Gastroenterol 2019;12:239-53