Case Report

Eosinophilic ascites: Uncommon Presentation of Eosinophilic Gastroenteritis in children

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Summary

Eosinophilic gastroenteritis (EGE) is a rare condition characterized by eosinophilic infiltration of the gastrointestinal tract. It is classified into mucosal, muscular, and sub-serosal types, depending on the clinical picture and the depth of eosinophilic infiltration within the gastrointestinal wall. Sub-serosal type, the most uncommon variant, which is complicated by ascites and peripheral hypereosinophilia, usually results in the most severe clinical form of eosinophilic gastroenteritis and requires early therapy. The clinical features are non-specific with history of atopic predisposition and allergy. Endoscopic biopsy is frequently non-diagnostic due to an uninvolved gastrointestinal mucosa rendering its diagnosis a challenge. The backbone of diagnosis is peripheral hypereosinophilia and eosinophil-rich ascitic fluid on diagnostic paracentesis. Oral steroid therapy is usually the first line of treatment with dramatic response. Due to a propensity for relapse, steroid-sparing therapy should be considered for relapses of EGE. We report a case of subserosal EGE with diagnostic clinical features and treatment response and review the current strategy in the management of eosinophilic ascites.

Keywords: *Eosinophilic gastroenteritis, hypereosinophilia, steroid therapy*

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Introduction

Eosinophilic gastroenteritis (EGE) is a rare condition, it is defined as a disorder primarily affecting the gastrointestinal tract with eosinophil-rich inflammation, in the absence of known causes of eosinophilia (e.g. drug reactions, parasitic infections or malignancy)[1]. To date, 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.

Case Report

A 12-year-old female presented to the clinic with complaints of abdominal distension and an episode of self-limiting diarrhea three weeks ago. Past medical history was remarkable for recurrent allergic bronchitis. On examination there was no evidence of pallor, icterus or peripheral edema and abdominal examination revealed marked distention with a doughy consistency. Abdominal ultrasonography demonstrated marked ascites with no signs of portal hypertension, liver, cardiac or renal disease, tab. (1). Laboratory examination revealed peripheral eosinophilic leukocytosis with 27% eosinophils (total leukocyte count 26500 cells/ mm^3) and no immature myeloid precursors. After one week during investigation repeat CBC, tab. (2) revealed peripheral eosinophilic leukocytosis with 64% eosinophils (total leukocyte count 32000 cells/mm^3) The C-reactive protein and erythrocyte sedime-ntation rate were within normal limits. Viral markers for hepatitis A, hepatitis B and hepatitis C viruses were negative, liver function revealed serum bilirubin 0.9 mg/dl, normal enzyme, serum albumin 4.5 gm/dl. Kidney function test within

normal. Serum amylase, autoimmune antibodies within normal and glucose level was normal. Diagnostic paracentesis revealed yellow aspect, total leucocyte count 60 cell/ cmm, Ziehl Nielsen stain was negative for acid fast bacilli, lactate dehydrogenase 142 U/L, total protein 4.6 gm/dl, negative for cytological malignant cells. Skin prick test results for food allergens and stool examination for bacteria, ova and parasites was negative. Upper endoscopy and colonoscopy not done as invasive maneuver and her parents refused. Echocardiographic findings were normal.

Table (1) Follow up ultrasonography during treatment

Date	Abd US
26/7/2017	Marked abdominal ascites and left pleural effusion
2/8/2017	Mild abdominal ascites
9/8/2017	No ascites
16/8/2017	No ascites
23/8/2017	No ascites
30/8/2017	No ascites

Table (2) Follow up of CBC during treatment

Date	TLC s/cm^3	Eosinophilic %	Neutrophil %	Lymphocyte %	Absolute neutophilic count	Hg%	Platelets
26/7/ 2017	32	64%	13%	21%	4.2	12.7	318000
2/8/2017	13.4	3.1%	35%	53%	4.7	12.2	292000
9/8/2017	22.9	0.70	65%	33%	13.7%	13.8	464000
16/8/2017	21	0.3	68%	26%	13.2	11.7	397000
23/8/2017	16.5	0.5	64%	28%	11.3	12.0	312000
30/8/2017	12.9	o.5	62%	30.%	8.O	12.5	224000

Discussion

Eosinophilic gastroenteritis (EGE) is a rare condition characterized by eosinophilic infiltration of the gastrointestinal tract. The clinical features of EG vary depending on the location, extent, and depth of infiltration of the gastrointestinal wall. Eosinophilic ascites is an unusual form of presentation of EGE[3]. It is characteristic of the predominantly subserosal pattern of the disease (the least common, followed by the intramural and the mucosal types) and is rare in young males[4]. Though, even in the predominantly subserosal type, there is frequently significant mucosal and submucosal eosinophilic infiltration[5], which permits for the diagnosis to be obtained through endoscopic biopsies in most cases. Talley et al[5], have defined three diagnostic criteria for EGE: **(1)** presence of gastrointestinal symptoms; **(2)** biopsies of the gastrointestinal tract showing eosinophilic infiltration or characteristic radiologic findings with peripheral eosinophilia or eosinophil-rich ascites with; and **(3)** no evidence of parasitic or extra intestinal disease. Characteristic findings of EGE on abdominal computed tomo-graphy include thickening of bowel wall or fold, layering of the bowel wall, luminal narrowing without obstruction, intra- or extraluminal

granuloma, mesenteric lymphadenopathy with peripheral rim-like enhancement or necrosis and ascites[6]. The etiology of EGE is unclear however; an atopic predisposition is noted in patients with EGE with a history of allergy reported in 50% patients with EGE[5]. A genetic predisposition is suspected as 16% of patients with EGE have a family member with a similar condition[7]. Depending on previous studies, the predominantly serosal pattern of EG seems to have a good prognosis, with excellent response to steroids and presenting mainly with a single flare and no chronic course[8]. In the study by Chen et al., 13 out of 15 patients with EGE required treatment with prednisolone (10-40 mg/day) resulting in complete resolution of symptoms within 2 weeks. Yet, more than one-third of the treated patients relapsed within 12 months, and 13% required long-term treatment with prednisolone (5-10 mg/ day)[9]. In cases that fail to respond to corticosteroids, treatment with azathioprine or 6-merca-ptopurine should be considered.

Comments

Case characteristics

A 12-year-old female presented with abdominal distension and diarrhea and was diagnosed with subserosal eosinophilic gastroenteritis (EGE) Eosinophilic ascites (EA). She responded to oral steroid therapy with complete resolution of the ascites and normalization of peripheral hypereosinophilia.

Clinical diagnosis

Subserosal EGE, Eosinophilic ascites (EA)

Differential diagnosis

Ovarian cancer, abdominal tuberculosis, vasculitis, parasitic infection, congestive heart failure.

Laboratory diagnosis

Peripheral hypereosinophilia and exclusions of other causes by diagnostic paracentesis.

Imaging diagnosis

Abdominal ultrasonography.

Treatment

Oral steroid therapy.

Experiences and lessons

A high index of suspicion of subserosal EGE in patients with abdominal pain, ascites and peripheral hypereosinophilia. Oral steroids adequate for initial therapy and montelukast, a leucotriene receptor antagonist may be considered for relapse of EGE.

Conclusion

Eosinophilic gastroenteritis diagnosis should be considered in patients with ascites of uncertain etiology, nonspecific bowel thickening by imaging studies and negative workup for parasitic infection and malignancy. Even though peripheral blood or ascitic fluid eosinophilia is suggestive, its absence does not exclude the possibility of this diagnosis. Short courses of corticosteroids are the backbone of treatment however; some patients with relapsing disease require long-term low-dose steroids.

References

1- Rothenberg M. Eosinophilic gastrointestinal disorders (EGEID). **J. Allergy Clin Immunol.** 2004; 113 (1): 11-28.

2- Naylor A. Eosinophilic gastroenteritis. **Scot Med J.** 1990; 35: 163-165

3- Zhang M, Li Y. Eosinophilic gastroenteritis: A state-of-the-art review**. J. Gastroenterol Hepatol.** 2016, Epub ahead of print.

4- Durieu I, Nove-Josserand R, Cathebras P, Durand D, Rousset H, Levrat R. Eosinophilic ascites. 2 new case reports (in French). **Rev Med Interne.** 1992; 13: 446-448.

5- Talley N, Shorter R, Phillips S, et al. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissues. **Gut.** 1990; 31: 54-58.

6- Zheng X, Cheng J, Pan K, Yang K, Wang H, Wu E. Eosinophilic enteritis: CT features. **Abdom Imaging.** 2008; 33:191-195.

7- Elliott J, McCormack O, Tchrakian N, Conlon N, Ryan C, Lim K, Ullah N, Mahmud N, Ravi N, McKiernan S, et al. Eosinophilic ascites with marked peripheral

eosinophilia: a diagnostic challenge. **Eur J Gastroenterol Hepatol.** 2014; 26: 478-484.

8- Pineton de Chambrun G, Gonzalez F, Canva J, et al: Natural history of eosinophilic gastroenteritis. **Clin Gastroenterol Hepatol**. 2011; 9: 950-956.

9- Chen M, Chu C, Lin S, Shih S, Wang T. Eosinophilic gastroenteritis: Clinical experience with 15 patients. **World J Gastroenterol**. 2003; 9: 2 813-2 816.