

Eosinophilic Ascites Due to Eosinophilic Gastroenteritis: A Case Report and Review

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1. Abstract

Eosinophilic gastroenteritis (EGE) is a rare disease characterized by the presence of gastrointestinal symptoms with an abnormal eosinophilic infiltrate of the intestinal wall and exclusion of other causes of secondary eosinophilic infiltration. EGE has various gastrointestinal symptoms depending on the depth of eosinophilic infiltration of the bowel wall: mucosal, muscular, or subserosal. In particular, EGE with ascites is uncommon among them, and mainly appeared in subserosal type. We have experienced a case of diagnosing and effectively treating EGE accompanied with ascites in a 40-year-old woman.

A 40-year-old woman visited the emergency room presenting with abdominal pain and diarrhea with a duration of approximately three weeks. Laboratory test results showed leukocytosis of 11,900/mm³ with eosinophilia (5,378 /mm³, 45.2%), and high serum level of IgE (1,484 IU/mL). On abdominopelvic CT, long segment edematous wall thickening of stomach to colon and a large amount of ascites in pelvic cavity were demonstrated. Paracentesis was performed, and eosinophilia was observed in ascites. On endoscopy, there was diffuse mucosal edema in the entire stomach, duodenum, and colon. Random biopsies were performed, and significant eosinophil infiltration was identified. Parasitic infection was excluded through stool examination, and there were no meaningful results in blood tests including food allergen tests and ANA, ANCA, C3 and C4.

The patient had a history of eosinophilic gastroenteritis, and she was thought to have recurrence of eosinophilic gastroenteritis with ascites, therefore steroid administration was begun (prednisolone 40 mg/d). Within one week, symptoms and eosinophil count improved, and improvement of intestinal edema and ascites were also observed in follow-up CT.

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2. Introduction

Eosinophilic Gastroenteritis (EGE) is a rare disorder of unknown etiology with an abnormal eosinophilic infiltrate of the intestine wall [1]. It can present with various gastrointestinal symptoms depending on the depth of eosinophilic infiltration of the intestinal wall, and is classified into 3 subtypes as follows: mucosal, muscular, and subserosal [2, 3]. Its main symptoms are abdominal pain, nausea, vomiting, diarrhea, weight loss, etc, [4]. EGE with ascites appears in the subserosal type, which is known to be the rarest form, representing approximately 10% of the total eosinophilic gastroenteritis [5]. Confirmation of eosinophilic infiltration in gastrointestinal wall through biopsy is essential of diagnosis. Peripheral eosinophilia appeared in most cases, but it is not an essential factor for diagnosis, and other diseases that cause secondary eosinophilic increases should be excluded [1].

We have experienced a case of diagnosing and effectively treating eosinophilic gastroenteritis with ascites in a 40-year-old female patient who complaining of abdominal pain

3. Case Report

A 40-year-old woman visited the emergency room presenting with abdominal pain and diarrhea that lasted about three weeks. She also complained of nausea and intermittent vomiting, accompanied by mild abdominal distention. Blood pressure was 112/71 mmHg, heart rate was 103 beats per min, respiratory rate was 20 breaths per min, and oxygen saturation was 97% in room air. On physical examination, there was diffuse tenderness of the whole abdomen, but rigidity was not observed. She was taking methima-₁

zole for hyperthyroidism and had a history of hospitalization for eosinophilic gastroenteritis 6 years prior. She did not have allergic conditions such as asthma, rhinitis, or food intolerance.

Initial laboratory test results showed leukocytosis of 11,900/mm³ with eosinophilia (5,378/mm³, 45.2%), high serum level of IgE (1,484 IU/mL), eosinophil count (9,340/mm³, normal range 50~500/mm³) and eosinophilic cationic protein (ECP >200 ng/mL). Other blood tests showed no significant abnormalities. Abdominopelvic CT showed long segment edematous wall thickening of stomach to colon. Additionally, there was a large amount of fluid collection in subhepatic space and pelvic cavity with mesenteric infiltration (Figure 1). Diagnostic paracentesis was performed and approximately 1L was drained. Ascitic fluid was translucent, and WBC was 5600/mm³ (eosinophil cell 91%, mononuclear cell 7%, and lymphocyte 1%). No malignant cells were detected and culture of the ascetic fluid was negative.

Upper endoscopy revealed diffuse mucosal edema in the entire stomach and duodenum. Random biopsies were performed at each segment of stomach and duodenum. Colonoscopy showed diffuse mild mucosal edema in the entire colon, and random biopsies were performed (Figure 2). Biopsy results showed significant eosinophil infiltration was observed in the entire intestine, especially co-

lon (more than 30/high power field) (Figure 3). Parasitic infection was excluded through stool test and specific antibody tests, and there were no specific findings in the food allergen skin prick test. In addition, blood test including antinuclear antibody, ANCA, C3 and C4, and peripheral blood smear were performed, and results were not significant.

The patient was diagnosed with recurrent eosinophilic gastroenteritis with ascites, therefore oral corticosteroid administration was begun (prednisolone 40 mg/day). Blood testing after 3 days showed eosinophil cell count decrease 2.7% (initial 45.2%), and after 5 days, patient's nausea and abdominal pain symptoms were also improved. The eosinophil count was also decreased to 150/mm³ (50~500 mm³), within normal range.

Abdominopelvic CT was performed on the 14th day of hospitalization, and diffuse edematous wall thickening from stomach to colon was significantly decreased. The substantial amount of ascites seen in subhepatic space and pelvic cavity were no longer visible (Figure 4). After a week of steroid administration, the dose was reduced 30 mg/day, and it was planned to be tapered every 7 days for the 2-month follow-up period. A week after discharge, the eosinophil count was in the normal range in outpatient care, and the patient complained of no symptoms.

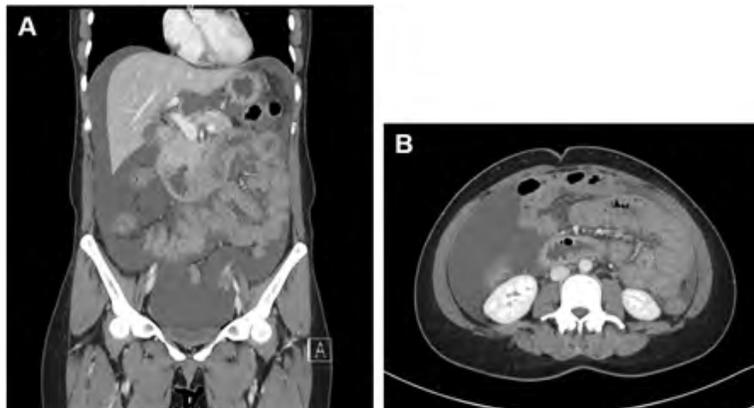


Figure 1: Abdominopelvic CT (A, transverse view; B, coronal view) Long segmental edematous wall thickening of stomach~colon. Large amount of fluid collection in subhepatic & pelvic cavity with mesenteric infiltration.

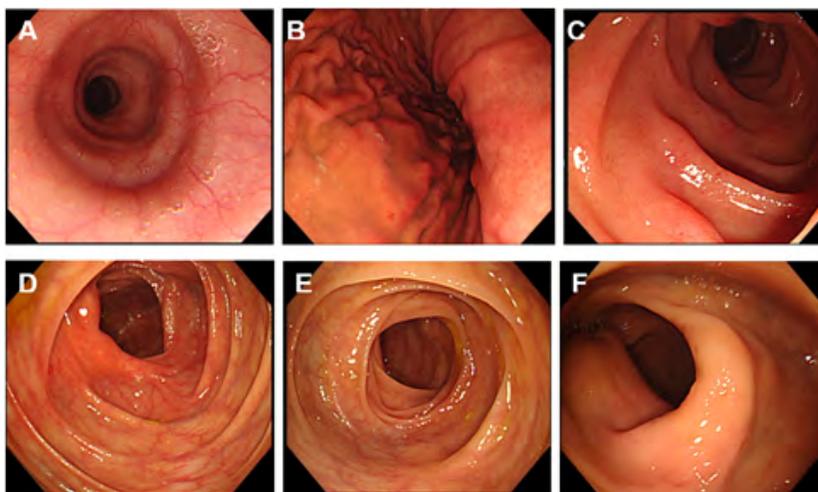


Figure 2: Endoscopic findings (A, esophagus; B, stomach body; C, duodenum; D, A-colon; E, T-colon; F, Rectum). Diffuse mucosal edema in the entire esophagus, stomach, duodenum and colon

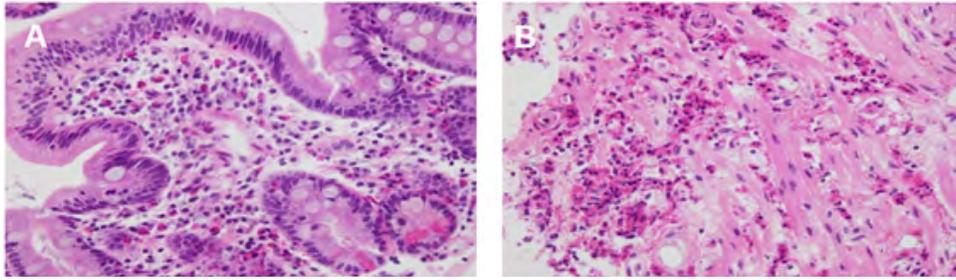


Figure 3: Histopathologic findings (A, duodenum; B, colon). Eosinophil infiltration was observed in the entire intestine, especially colon (more than 30/high power field)



Figure 4: Abdominopelvic CT (A, transverse view; B, coronal view). Decreased in the extent of edematous wall thickening of stomach~colon. Disappearance of large amount of fluid collection in subhepatic space & pelvic cavity

4. Discussion

EGE is a rare disease characterized by the presence of gastrointestinal symptoms with an abnormal eosinophilic infiltrate of the intestinal wall and exclusion of other causes of secondary eosinophilic infiltration [3]. In particular, EGE with ascites is uncommon, so we are reporting this case in comparison with previously reported 20 similar cases (Table 1). 6-20 EGE's prevalence is from 8 to 24 per 100,000 and most patients are diagnosed between 30-50 years of age [6]. The patient in this case was also a 40-year-old woman, and 13 of the 20 cases compared were in this age range.

The etiology remains obscure, but there are many reports that cytokines such as IL-4 and IL-5 which activate eosinophil are related [4]. In addition, the fact that EGE patients often accompanied allergic diseases such as asthma, rhinitis, or food intolerance also provides clues to the etiology [1]. Of the 20 cases we compared, 7 patients had allergic diseases. Although patient in our case did not have a history of allergic disease, the possibility of allergy to methimazole is being evaluated.

Based on the dominant layer of the intestinal wall infiltrated by eosinophil, EGE is divided into three types: mucosal, muscular, and subserosal (Klein classification) [3, 7]. The most common form (70%) is the mucosal infiltration type, with symptoms related to dysfunction of the mucosa with malabsorption and protein-losing enteropathy (diarrhea, steatorrhea, weight loss, hypoalbuminemia). The muscle layer infiltration type accounts for about 20% <http://www.acmcasereport.com/>

of the total EGE. This type is associated with localized or diffused bowel wall thickening, with features of pyloric narrowing and obstructive symptoms (colicky pain, nausea, or vomiting). The third type is subserosal infiltration type, characterized by ascites rich in eosinophils. This type is reported to be about 10% of the total eosinophilic gastroenteritis, and it is known to occur mainly in women of childbearing age, such as the patient in our case [3, 8].

Verifying eosinophilic infiltration in gastrointestinal wall by biopsy is essential for diagnosing EGE, and peripheral eosinophilia is not necessarily observed. But, peripheral eosinophilia is observed in most EGE patients, and it was found in all 20 cases compared, including our case. In this regard, one of the things to be careful about in the diagnosis of EGE is the need to differentiate other diseases that cause secondary peripheral eosinophilia, such as parasitic infection (*Strongyloides stercoralis*, *Toxocara canis*), vasculitis (Churg-Strauss syndrome), inflammatory bowel disease, malignancy (eosinophilic leukemia, lymphoma), and hypereosinophilic syndrome [3]. In this case, we excluded these diseases through tests such as stool test, specific antibody test, food allergen test, blood test including anti nuclear antibody, ANCA, C3, C4 and peripheral blood smear.

Although the increase in eosinophils is an important factor in EGE, eosinophils are also present in the normal mucosa of the gastrointestinal tract. There is no clear standard for how many eosinophils should be infiltrated to be considered abnormal yet, many

researchers have suggested that at least 20 eosinophils/HPF infiltration is necessary [5]. In our case, more than 30 cells/HPF were observed, and in all 20 cases compared, more than 20 eosinophils/HPF infiltration was observed.

EGE produces much less marked macroscopic changes, and so histopathological diagnosis is essential. As in the 20 cases we compared, endoscopic exam demonstrates nonspecific findings such as thickened mucosal folds, erythema, edema, erosion, and ulcer. Multiple samplings should be performed in endoscopic biopsies, because lesions can be distributed locally [1].

Major treatment of EGE is corticosteroid, which is generally effective in most patients. In most of the 20 cases, including our case, it has also improved through steroid therapy. The recommend dosage is daily 20–40 mg prednisolone for 7–14 days and then tapered [7]. Approximately 80% of patients improve their symptoms within one week and eosinophil count are normalized within 2 weeks [3]. The prognosis is good in most patients, but an estimated 50% of patient experience recurrence after discontinuation of steroid therapy, sometimes requiring long-term administration. A patient

in our case, also had a history of EGE before, and this event was second EGE. One caution before steroid treatment is that parasitic infections should be completely excluded, because steroid treatment in the presence of occult parasitic infection may result in catastrophic disseminated disease. Second-line therapies include antihistamines (ketotifen), mast cell stabilizers (sodium cromoglycate), leukotriene antagonists (montelukast), and immunomodulators (azathioprine). As well, it has been reported in recent years that monoclonal antibody (omalizumab) developed against IgE and IL-5 is beginning to be used successfully in treatment [7-9].

Ascites in EGE is rare. There are no clear diagnostic criteria yet, and endoscopic findings are also non-specific, so it is a disease that can be missed in clinical practice. In particular, if eosinophil count increase and ascites are observed in a patient complaining of gastrointestinal symptoms, it is necessary to distinguish this disease, and the exclusion of other diseases that cause eosinophil increase is also important. Since most such patients respond well to steroid treatment, prognosis is favorable. Therefore, we hope this case report will be an opportunity to advance the diagnosis and treatment of eosinophilic gastroenteritis.

Table 1: Case reports of eosinophilic gastroenteritis presenting with ascites

Reference	Age/ Sex	Symptoms	Peripheral eosinophilia	Involved organ	Endoscopic findings	Allergic disease	Treatment
Fenogolio LM, (2003) ⁸	29/F	Abdominal distension, Diarrhea, Vomiting	+	stomach, duodenum	Hyperemia	-	PD 25mg/d
Hepburn IS, (2010) ⁹	20/F	Abdominal distension/pain, vomiting, diarrhea	+	-	erythema	-	PD 40mg/d
Bleibel F, (2012) ¹⁰	55/M	Abdominal distension/pain	+	duodenum	hyperemia	-	PD 20mg/d
Liao WH, (2012) ¹¹	43/M	Abdominal pain	+	colon	erythema	-	Spontaneous resolution
De Matos Brasil AA, (2013) ¹²	49/M	Abdominal distension, bloating	+	Peritoneum, mesenteric lymph node	erythema	+(rhinitis, lactose intolerance)	PD 30mg/d, Restricted diet
Cuko L, (2014) ¹³	37/F	Abdominal distension/pain, diarrhea	+	Esophagus, stomach	hyperemia	-	PD 40mg/d
Baek MS, (2014) ¹⁴	68/F	Abdominal pain, nausea, diarrhea	+	duodenum	Hyperemia, edema, erythema	-	PD 30mg, Montelukast 10mg
Caglar E, (2015) ¹	35/M	Abdominal distension, Nausea	+	esophagus, duodenum	edema	+(asthma, rhinitis)	PD 40mg/d
Alsulaiman RM, (2015) ¹⁵	28/F	Abdominal distension/pain	+	Stomach, duodenum, ileum, colon	edema	-	PD 40mg/d
Khalil H, (2016) ¹⁶	25/F	Abdominal distension/pain, nausea, skin rash	+	Esophagus, stomach, duodenum, rectum	erythema	+(asthma, rhinitis, dermatitis)	PD 30mg/d, Restricted diet
Lourenco LC, (2017) ²	27/M	Abdominal distension/pain	+	stomach	none	+(asthma, rhinitis)	PD 40mg/d
Ferreira AA, (2017) ¹⁷	41/F	Abdominal distension, diarrhea, anorexia	+	-	none	-	PD 40mg/d
Santos C, (2018) ¹⁸	32/F	Abdominal pain, nausea, diarrhea	+	Ileum, colon	hyperemia	-	PD 40mg/d

Shi L, (2018)¹⁹	57/F	Abdominal pain/ distension	+	duodenum	Erosion, hyperemia	-	Ketotifen
Saban J, (2019)²⁰	30/F	Abdominal pain/ distension,	+	Esophagus	normal	-	PD, Restricted diet
Wan Feng, (2020)⁸	26/M	Abdominal distension/ pain, diarrhea	+	Stomach, colon	Erythema, edema	-	PD 40mg/d
El Ray A, (2020)³	30/F	Abdominal distension, vomiting, epigastric pain	+	Esophagus, stomach, duodenum	hyperemia	+(iron intolerance)	PD 40mg/d
Devi S, (2020)⁶	44/M	Abdominal pain/ distension	+	Esophagus, stomach	erythema	+(asthma)	PD 40mg/d
Tian XQ, (2021)⁵	34/M	Abdominal pain, diarrhea, nausea	+	Stomach, ileum, colon	Hyperemia, edema	-	PD 40mg/d
Sequeira M, (2022)⁴	24/F	Abdominal pain, diarrhea, vomiting	+	colon	Hyperemia	+(iron intolerance)	Suspend iron supplement
Present case	40/F	Abdominal pain/ distension, diarrhea	+	Esophagus, Stomach, colon	edema	-	PD 40mg/d

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