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Eosinophilic gastroenteritis with ascites at Children's Hospital No.2: a case report and review of the literature

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Abstract:

Eosinophilic gastroenteritis (EGE) is a digestive disorder in children and adults that is characterised by eosinophilic infiltration in the stomach and intestine. Depending on the dominant layer of infiltration, this disorder is classified into three types, namely, mucosal, muscularis, and subserosa. Subserosa clinical presentation usually begins with ascites and is sometimes associated with symptoms of intestinal obstruction. Ascites is characterised by marked eosinophilia in ascitic fluid. EGE with ascites simulating appendicitis and peritonitis is rare in paediatric patients. This publication aims to report a case series of EGE with clinical features, treatment responses, and a review of the current strategies in the management of eosinophilic ascites.

Keywords: **endoscopic biopsy, eosinophilia in children, eosinophilic ascites, gastrointestinal.**

Classification number: **3.2**

Introduction

EGE is a rare disorder presenting with a cluster of gastrointestinal symptoms associated with eosinophils histological infiltration. This disease was first described by Kaiser in 1937. The prevalence of EGE in the United States is estimated to be 22 to 28 per 100,000 persons. EGE usually occurs between 20 and 50 years of age, but recently it has been shown to manifest in children [1]. EGE can affect the entire digestive tract from the stomach to the colon. The clinical features of EGE are related to the affected gastrointestinal tract segment and the degree of eosinophil infiltration. Pathogenesis of the disorder is still unclear. It is hypothesised that there is a role for IgE-mediated immunity and T-lymphocyte (Th2)-mediated immunity.

The presentation of EGE is usually nonspecific gastrointestinal symptoms, and ascites is rarely seen [1, 2]. About 75% of cases have elevated eosinophil in blood. While no gold standard of diagnosis has yet been established, diagnoses are mainly based on the history and presence of eosinophils in tissue biopsy after excluding other conditions including peripheral eosinophilia [1]. Currently, there is no consensus of diagnosis nor are there specific treatment guidelines, for example, on the dosage, administration, and duration period of therapeutic steroids. Some drugs such as Cromolyn, Ketotifen, Montelukast, and Omalizumab can be used in some cases, but are not recommended.

From 2018 to 2020, we recorded 5 cases of ascites due to EGE admitted to the Department of Gastroenterology at Children's Hospital No.2. The process of establishing the diagnosis included a long period of monitoring clinical presences and laboratory findings. Although our clinical cases have similar GI symptoms, each case still has its own characteristics.

Case series

Case 1

A 14-year-old female patient came to us with a chief complaint of intermittent epigastric pain for 4 months. During this admission, the patient had epigastric pain continuously for 3 days. She had no febrile or other GI symptoms except for a little vomiting. Personal and family history had no documented tuberculosis or allergies. Physical examination revealed soft abdomen, mild distention, right iliac fossa pain, positive Blumberg sign, and unclear abdominal wall resistance. Laboratory results are detailed in Table 1 below. The accumulation of abdomen fluid in ultrasound and the elevation of eosinophils count in blood as well as the presence of inflammatory cells clusters, mainly eosinophils, in the intestinal wall were prominent in these findings (Fig. 1).

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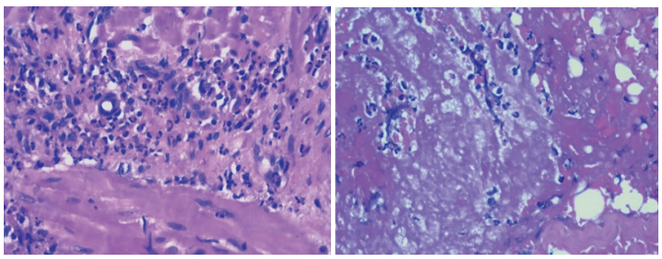

Fig. 1. Eosinophilic infiltration of the muscular layer of the intestinal wall and peritoneum biopsy sample of case 1 (Hematoxylin-Eosin staining×100).

Table 1. Clinical features and laboratory results of case reports 1, 2, and 3.

Case presentation	Case 1	Case 2	Case 3
Symptoms	Epigastric abdominal pain, vomiting, no diarrhoea, no weight loss	Periumbilical pain, vomiting, loose stools, weight loss	Periumbilical pain, increasing ascites, vomiting
Clinical examination	Mild abdominal distension, right iliac fossa pain, rebound tenderness (+/-), non- significant guarding sign.	Ascites, muscular resistance (-)	Ascites, tenderness around the umbilicus, no guarding sign, moist rales, bilateral alveolar hypoechoic, no oedema of limbs, face
Initial diagnosis	Acute appendicitis, enteritis	Unexplained peritoneal effusion, enteritis	Multi-membrane effusion of unknown causes-Pneumonia-follow-up eosinophilic enteritis or malignancy
Complete blood count	WBC: 16.7 K/Ul Neu: 10.6 K/ul Eo: 1.44 K/ul Hb: 11.8 g/d PLT: 448 K/Ul	WBC: 12 K/ul Neu: 6.9 K/ul Eos: 1.9 K/ul Hb: 18.4 g/dl PLT: 382 K/Ul	WBC: 12.9K/Ul Neu: 6.62 K/ul Eos: 1.16K/Ul Hb: 12.4 g/dl PLT: 428 K/ul
Immunoassay	ANA-8 (-) profiles (anti-dsDNA, anti-cardiolipin, antiphospholipid, anti-Sm, anti Jo-1, anti SS-A, anti SS-B)* Normal protein electrophoresis Normal C3, C4; Normal IgA, IgG, IgM		
Parasitic serology	Fasciola (+) Strongyloides stercoralis (+)	(-)	(-)
Peritoneocentesis	Exudate, neutrophil predominance, PCR of common agents (-), normal ADA	Exudate eosinophil predominance, normal ADA	Exudate, inflammatory cell diversity, no foreign cells recorded
Tuberculosis test	ADA peritoneal fluid* (-); VS* (-), Sputum BK* (-), Stool BK (-)		
Ultrasound	Abundant abdominal fluid, intestinal wall thickened ileocecum	Abundant abdominal fluid, thickened small intestinal wall	Large amount of abdominal fluid, no hepatosplenomegaly, not found abdominal lymph nodes
Abdominal CT scan	Abdominal free fluid, thickened small bowel wall, no abnormal dilation of intestinal loops	Enlarged liver, normality in liver tissue density Abdominal free fluid, thickened and oedematous wall of small intestine and ascending cecum, no enlarged abdominal lymph nodes.	Large amount of ascites. Thickened of the pericardium and momentum, no enlarged mesenteric and retroperitoneal lymph nodes. Pneumonia
Upper gastrointestinal endoscopy	Nodular gastritis	Congestive gastritis	Active chronic gastritis *H. pylori* test (+)
Lower gastrointestinal endoscopy	Did not perform	Inflammation of the ileum	Ulcerative ileocecal ulcer
Pathology	Clusters of inflammatory cells mainly eosinophils within intestinal wall; infiltration of fatty and fibres in connective tissue.	Active chronic proliferative ileitis, with clusters of inflammatory cells mainly eosinophils	Considerable infiltration eosinophils within all intestinal submucosal

*ANA: antinuclear antibody; anti-ds-DNA: anti-double stranded DNA; anti-Sm: anti-Smith; anti Jo-1: histidyl tRNA synthetase; anti SS-A: anti-Sjögren syndrome A; anti-SS-B: anti-Sjögren syndrome B); *C3, C4: complement 3 and 4; *BK: bacille de Koch; *VS: erythrocyte sedimentation rate; *ADA: adenosine deaminase, (+): positive, (-): negative.

Treatment progress: the patient was initially treated with intravenous antibiotics and parasite eradication (Ivermectin and Albendazole) along with exploratory laparoscopic surgery. The protocol recorded that abdomen was filled with yellow-brown fluid and the entire small intestine was inflamed, thickened, and congested. Similar condition with appendix. Stomach, colon, uterus, and appendages were normal. Appendix removal and abdominal fluid aspiration were performed. After the surgery, our patient remained with abdominal pain and increasing distention in addition to bilious vomiting. She had no fever and no hepatosplenomegaly or abnormal lymph nodes. Since the biopsy results proposed enteritis caused by increased eosinophils, we started to use intravenous corticosteroids therapy (2 mg/kg/day) and continued parenteral nutrition. After 5 days, distention state was greatly improved, eosinophils returned to normal range, and abdominal ultrasound showed a reduction of ascites. Treatment was to gradually reduce the dose of corticosteroids, switching to an oral route, and applying a diet towards avoiding allergies. There was a significant recovery and the patient was discharged from the hospital.

Case 2

A 16-year-old male patient was admitted to the hospital because of abdominal distention. He had abdominal pain mainly in the periumbilical area for 1 month and gradually distention along with nausea, vomiting, and loose stool 5-6 times/day, which was not accompanied by fever or weight loss. Physical examination recorded a soft belly, ascites, slight tenderness around navel, no resistance in abdominal wall and no enlarged liver and spleen. Personal history: enteritis, peritoneal infection, hospitalised for 21 days. Significant laboratory results: WBC 12 K/ul, eosinophil 1.9 K/ul, normal range for high sensitivity C-reactive protein as well as hepatic and renal function. Immunoassay: negative. Serological diagnosis of parasites: negative. Abdominal CT scan: hepatomegaly (#170 mm), normal density of liver tissue, large amount of intra-abdominal fluid. Thick oedematous wall of small intestine and ascending cecum, no enlarged abdominal lymph nodes. Peritoneal fluid: exudate, cellblock: eosinophil predominance 60%, ADA: 4.41 U/l. Tuberculosis test: negative. Endoscopy: congestive gastritis, ileitis. Pathology images of intestinal wall tissue biopsied: inflammatory proliferative chronic ileal mucosal activity, with clusters of inflammatory cells mainly eosinophils (see Fig. 2). Diagnosis: EGE, eosinophilic peritoneal effusion. The patient was treated with steroids therapy and an allergenic diet restriction.

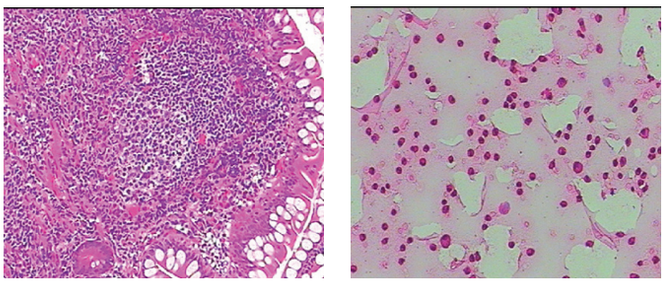

Fig. 2. Left: eosinophilic infiltration of the peritoneum in a biopsy sample of case 2 (Papanicolaou×100 method), right: histology of proliferative ileal mucosa, with clusters of inflammatory cells predominantly eosinophils of case 2 (Hematoxylin-Eosin staining×100).

Case 3

A 7-year-old female patient was hospitalised with multi-membrane effusion and malnutrition. She had gradually increased abdominal pain around the umbilicus for 1 month with ascites accumulation. Besides that, she had a phlegm cough without pyrexia. Clinical examination: significant ascites with soft abdomen, hepatosplenomegaly, slight tenderness around the umbilicus with no guarding, abdominal wall resistance, or signs of mucosal bleeding. She also had some moist rales in addition. Personal history announced the malignancy monitoring diagnosis due to multi-membrane effusion because of intra abdomen para vascular nodes findings. The patient was scheduled for exploratory surgery but, unexpectedly, the operation was not performed due to her absence with the reason being that ascites had improved according to parents' consideration. Abdominal CT scan at previous admission: massive abdominal effusion, mainly localised in front of stomach and liver; slightly inflammation of the intestinal wall, associated at the beginning of the ileum; oedematous condition of the mesentery and the great omentum; small lymph nodes (d#12 mm) adjacent to the abdominal aorta. At this admission period: large amount of ascites with high density, mostly localised in the epigastrium; thickened of the pericardium and momentum (d#15 mm). There are no enlarged mesenteric and retroperitoneal lymph nodes. Moreover, inflammation images related to the middle lobe of the lung. Endoscopy showed several scattered superficial ulcers effected along the first region of the right colon. The cecum had oedematous inflammation and numerous superficial ulcers too. Through the ileocecal valve for 10 cm, the ileal mucosa finding was similar to the under-gut disorder (see Fig. 3). Pathology had demonstrated the numerous presences of eosinophils in the intestinal submucosa, active chronic gastritis with positive *H. pylori* test. Other laboratory investigations are summarised in Table 1. For management, we also combined corticosteroids and parenteral feeding intervention, then continued by a hypoallergenic diet and oral medications. The patient responded well and left the hospital.

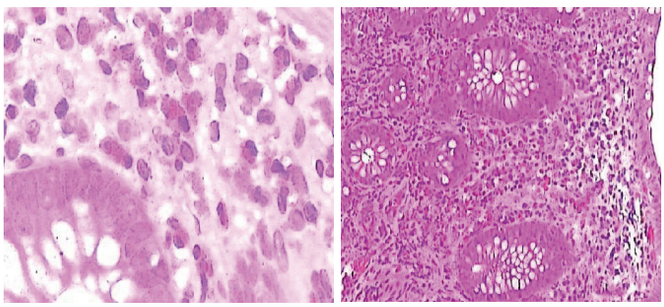

Fig. 3. Histology of infiltration of eosinophils in the intestinal submucosa (Hematoxylin eosin).

Case 4

A 14-year-old female patient was hospitalised due to a 10-day history of dull epigastric and periumbilical pain. She had other nonspecific GI symptoms like little vomiting after having meal, anorexia, and weight loss. We found a soft abdomen and no warning signs on clinical examination. Laboratory results showed the peripheral eosinophilia and a massive peritoneal effusion together with a diffusely thickened ileal wall in abdominal ultrasound demonstration. Therefore, we performed both upper and lower gastrointestinal endoscopy, exploratory laparoscopy along with tissue biopsied, and peritoneal fluid analysis to confirm the ailment. Upper gastrointestinal endoscopy showed nodular gastritis together with *H. pylori* infection, which was determined by CLO test and microbiological culture. Regarding the lower gastrointestinal endoscopy, we only found a polyp near the edge of the anus. On laparoscopy we recorded clear yellow abdominal fluid with medium volume accumulation, especially at the beginning region of the jejunum, which showed as thickened, inflamed, and speckled with white. Histopathological images revealed that the ileal tissue sample has considerable infiltration of eosinophils in the muscle layer and serosa (>200 Eos/HPF), and fewer in the mucosal layer (>50 Eos/HPF). Other clinical features and laboratory testing on admission are presented in Table 2. Intravenous corticosteroids combined with gastritis medicines were the main treatment during the hospitalised period.

Table 2. Clinical features and laboratory results of case reports 4 and 5.

Case presentation	Case 4	Case 5
Symptoms	Epigastric and dull periumbilical pain, anorexia, little vomiting after meal, constipation, weigh loss	Cystitis unresponsive to conventional treatment, Gradually increase abdominal pain, vomiting after meal
Clinical examination	Soft abdomen, no significant guarding signs	
Initial diagnosis	Following gastroenteritis	Cystitis Gastroenteritis
Complete blood count	Elevated eosinophils	
Immunoassay	Normal	IgE elevation
Parasitic serology	(-)	Toxocara (+)
Peritoneocentesis	Exudative, predominant neutrophils, PCR common agents (-)	
Tuberculosis test	ADA peritoneal fluid* (-); VS* (-), Sputum BK* (-), Stool BK (-)	Did not perform
Ultrasound	Massive ascites volume. Diffusely thickened intestinal wall (duodenum jejunum and the first region of ileum)	Ascites Thickened inflammation of stomach, antrum, pylorus, and duodenum Cystitis
Abdominal CT scan	Did not perform	Lymphoma (-)
Upper gastrointestinal endoscopy	Nodular gastritis *H. pylori* test (+)	Congestive oedema inflammation related the entire lining of the oesophagus, stomach, duodenum
Pathology	Eosinophil infiltration in the muscle layer and serosa, fewer in the mucosal layer of the ileum	Eosinophil infiltration in all tissue samples of oesophagus, stomach and duodenum

*BK: bacille de Koch; *VS: erythrocyte sedimentation rate; *ADA: adenosine deaminase.

Case 5

A 4-year-old male was admitted to hospital with a chief complaint of abdominal pain after 18 days of illness. Firstly, he had dribbling disorder and hypogastric burning pain condition when urinating. The patient was concluded cystitis syndrome at local hospital and was treated with 10 days of oral medicines (unknown type) but there still no relief of abdominal pain and urinary frequency.

After that, he had constant dull abdominal pain, vomiting 10 times per day after eating, and a slight headache in the forehead and temples. During the period of the illness, he had no fever, no blood vomiting, or other GI symptoms. Personal history recorded a mild dull periumbilical pain 2 times a week for 4 months prior to this admission. For the current presentations, the abdomen was soft, and the pain was continuous for 10 days. Some major laboratory findings included peripheral and central eosinophilia, positive Toxocarids antibody, IgE elevation and the abdominal ultrasound demonstrated an inflamed disorder mainly related to the stomach, antrum, the pylorus, specifically the thickness of duodenum wall with the diameter of 6 mm, and cystitis ailment in addition. Endoscopy: congestive oedematous inflammation related the entire lining of the oesophagus, stomach, duodenum. Pathology images: inflammatory cell infiltration with eosinophil predominance in all tissue samples of oesophagus, stomach and duodenum. We showed other clinical data and significant paraclinical testing in Table 2. Although antibiotic (Amoxicillin/Clavulanic acid) for ten days and parasite eradication (Albendazole) were used for initial treatment, the child still had abdominal pain and little vomiting about 3 times a day. When EGE was confirmed by the combination of physical examination and histology-proved biopsy, we used intravenous corticosteroids (Methyl prednisone) continually for 7 days with 2 mg/kg/day dosage for management. As a result, the patient had an obvious improvement of his abdominal pain and vomiting and was discharged after 23 days of hospitalisation.

Discussion

Clinical features and diagnosis

In the first case, the process of diagnosis was difficult because the patient had no specific or suggested symptoms nor an allergy history that would explain the ascites presence. Following the algorithm of peritoneal effusion, we performed clinical and paraclinical examinations to investigate appropriate causes such as peritoneal tuberculosis, liver disease (cirrhosis, portal hypertension), cardiovascular disease (heart failure, pericardial disease), kidney disease (nephrotic syndrome, chronic kidney disease), malignancy, parasite infection... [3] and none of these causes were involved in this patient.

According to the literature, EGE with the status of ascites includes 3 types: mucosal, muscular, and serosal types based on the depth of the lesion. The serosal type is the least common, ranging from 4.5 to 9% in Japan and 13% in the United States. Its mechanism can be the irritation of the peritoneum leading to ascites, along with peripheral elevation of eosinophil while peritonitis and perforation may occur in severe cases [2]. In comparison with other studies, we noted serosal ascites, exudative ascites, and predominant inflammatory cells, mainly eosinophils [4, 5].

Occasionally, EGE is associated with reflux esophagitis, dysphagia, and other vague symptoms such as abdominal pain, nausea, vomiting, diarrhoea, and weight loss, making it difficult to perform early diagnosis. In a case series in Australia, EGE was initially misdiagnosed as functional dyspepsia because the clinical presentation depends on the location, extent, and depth of the lesion in the gastrointestinal tract [6]. Furthermore, due to the characteristics of right iliac fossa pain and Blumberg sign

(+), we consulted with the surgeon about the appendicitis and peritonitis condition. As a result, our staff decided to perform emergency appendectomy and exploration.

A positive diagnostic serologic test for *Fasciola hepatica* and *Strongyloidiasis* may explain the eosinophil increases in the WBC, and fascioliasis may also cause multi-membrane effusions [7]. However, when treating these agents by anthelmintic, the clinical signs did not improve.

In the second case, peritoneal effusion was the initial presentation at hospital admission and many related diseases were investigated. However, based on the increase of eosinophil in the blood and the histological infiltration, we established the diagnosis earlier.

In the third case, the clinical features were similar to the second one with multi-membrane effusion. However, in the previous hospital admission, CT scan results had noticed para vascular nodes, so that made it impossible for us to rule out malignancy despite the recording of peripheral eosinophilic and peritoneocentesis. Therefore, the indication for exploratory surgery with lymph node biopsy that had been set at that time was completely appropriate. However, the follow-up process for that episode was interrupted because the family did not bring the child back to the hospital as scheduled (seeing that the ascites had improved). In this admission, with the suggestion of elevated eosinophils, multi-membrane effusion, and after excluding other causes, especially screening for cancer markers, along with peritoneal fluid test and pathology results, we confirmed the diagnosis with EGE. A considerable amount of literature suggests that 70% of cases have elevated eosinophils in blood especially in serosa type which has the higher risk of recurrence later [8].

Regarding the endoscopic features of the gastrointestinal tract, we recorded nodular gastritis in cases 1 and 4, congestive gastritis in case 2, and active chronic gastritis in case 3, which was accompanied with *H. pylori* infection. There was a distinctive characteristic in case 5 with congestive oedematous inflammation related the entire lining of the oesophagus, stomach, and duodenum. Meanwhile, when conducting lower gastrointestinal endoscopy on clinical cases 2 and 3, we recorded an ulcerative ileocecal ulcer and case 2 showed damage to both the upper and lower gastrointestinal tract.

Among our EGE with ascites patients, *H. pylori* presence was determined in two cases. Here, we explored the similar findings with a recent study in Japan that the rate of *H. pylori* infection was significantly lower in EGE, and Eosinophilic Esophagitis patients compared with controls, and this may explain the imbalance between Th1/Th2 in the aetiology of these disorders [8]. Countries with low hygienic environments and low socioeconomic conditions (overcrowding, low income, etc.) have higher infection rates and lower allergy-related diseases, such as asthma, eczema, allergic rhinitis, and others. The association between EGE and *H. pylori* infection has not been adequately considered in the literature. Additionally, prospective studies in children are needed to further investigate this association.

In terms of histopathological images, all cases of intestinal biopsies were infiltrated with many clusters of inflammatory cells, mainly eosinophils.

Treatment

Due to the diversity of clinical manifestations as well as the low morbidity rate, there have not been many studies on the disease progression, which makes it difficult to develop a treatment regimen for the disease. Many therapeutic options have been suggested including dietary considerations and medicinal remedies such as steroids, leukotrienes inhibitors, and mast cells stabilizers but no conclusive evidence has been published to describe the efficacies of these treatments. The therapeutic role of steroids in the treatment of EGE is not established. The recommendation dose for prednisone is 1-2 mg/kg/day for 7-10 days and then discontinuation after 4 months may be considered [9].

Dietary strategy means eliminating foods that are likely to cause allergies such as beef, cow's milk, eggs, soybeans, peanuts, etc. A series of paediatric EGE patients showed remission of symptoms in 40% of cases after dietary treatment, which consisted of an elemental diet in children under 6 months and hypoallergenic feeding in older children [10].

A similar case to ours was described by G. Ming, et al. (2015) [5] with an 11-year-old male patient admitted to the hospital because of abdominal pain for 7 days. The patient had a history of allergy to eggs. Clinical examination noted ascites and other organs were normal; eosinophil test increased, accounting for 31% of total white blood cells; ascites exudate with white blood cells increased, along with mainly eosinophils accounting for 91%. Endoscopy revealed oedema, congestion, and haemorrhagic spots of the antrum and duodenum and pathology revealed eosinophil infiltration into the mucosa. The patient was treated with corticosteroids and cetirizine, clinical and laboratory symptoms improved rapidly within 2 weeks.

In fact, our patient due to clinical conditions of vomiting, abdominal distension, and abdominal pain, the treatment was combined with intravenous feeding and corticosteroids at the dose of 2 mg/kg/day. After 1 week, the patient improved clinically and eosinophil counts in the blood returned to normal. As a result, the patient was discharged after two weeks starting EGE treatment.

Therefore, clinical physicians should have a high suspicion of EGE when children have nonspecific gastrointestinal symptoms such as nausea, vomiting, weight loss, bloating, abdominal discomfort, abdominal pain, diarrhoea, ascites, or oedema along with peripheral eosinophilia. Additionally, histological biopsy should be performed to obtain findings the infiltration of eosinophils into the submucosa, eosinophils within the epithelium of the crypts or villi or crypt hyperplasia, etc., which is helpful to confirm the diagnosis.

Recently, no therapeutic treatment available has been proven to have a specific efficacy on the disease. Instead, the treatment is based on the hypothesis of pathophysiology including the mechanism of allergy and inflammatory response, thereby diet remedy and anti-inflammatory medicines such as corticosteroids have been suggested [11]. Indications for surgical treatment are suggested when there are complications of perforation, intussusception, or intestinal obstruction [12].

Prognosis

C. Reed, et al. (2015) [13] conducted a follow-up for 26 months to evaluate the progression of the disease based on clinical symptoms, endoscopic images, and pathology. The results showed 60% clinical response, 51% improvement in endoscopic lesions and 69% histological recovery; in which there is 27% improvement in all 3 criteria above. The follow-up process is not effective due to the invasive means of monitoring [13, 14]. In fact, our patients continue to be monitored clinically and will have endoscopic examination when necessary.

Conclusions

EGE is a rare disease and collaboration between clinicians, endoscopists, and histopathologists can reduce missed diagnoses. Diagnosis is based on findings of dense eosinophilic infiltrates in the stomach and/or intestine combined with the clinical setting.

In terms of treatment and long-term follow-up, more clinical trials with larger sample sizes are sorely needed to compare the effectiveness of each remedy option as well as to establish the most optimal therapeutic method and appropriate duration of maintenance treatment.

Ethical approval

The need for institutional ethics approval for this case report was waived. All patients provided informed consent for their case details and images to be published.

COMPETING INTERESTS

The authors declare that there is no conflict of interest regarding the publication of this article.

REFERENCES

[1] D.Y.M. Leung, et al. (2016), "Allergic and eosinophilic gastrointestinal disease", *Pediatric Allergy*, Elsevier, 568pp.

[2] S. Tagore, et al. (2019), "EGE: diagnosis and clinical perspectives", *Clin. Exp. Gastroenterol.*, **12**, pp.239-253.

[3] A. Bavdekar, N. Thakur (2016), "Ascites in children", *Indian J. Pediatr.*, **83(11)**, pp.1334-1340.

[4] S.M. Belkhir, et al. (2018), "Eosinophilic ascites, a challenging diagnosis", *Int. Arch. Intern. Med.*, **2(1)**, DOI: 10.23937/iaim-2017/1710009.

[5] G. Ming, et al. (2015), "EGE with ascites in a child", *Indian Pediatr.*, **52(8)**, pp.707-708.

[6] M.M. Walker, et al. (2018), "EGE and other eosinophilic gut diseases distal to the oesophagus", *Lancet Gastroenterol. Hepatol.*, **3**, pp.271-280.

[7] S. Montembault, et al. (1997), "Hemorrhagic ascites disclosing massive *Fasciola hepatica* infectio", *Gastroenterol. Clin. Biol.*, **21(10)**, pp.785-788.

[8] S. Ishiharaa, Y. Kinoshitaa, A. Schoepfer (2016), "Eosinophilic esophagitis, EGE, and eosinophilic colitis: common mechanisms and differences between east and west", *Inflamm. Intest. Dis.*, **1(2)**, pp.63-69.

[9] F.M. Tien, et al. (2011), "Clinical features and treatment responses of children with EGE", *Pediatr. Neonatol.*, **52(5)**, pp.272-278.

[10] V.B. Busoni, et al. (2011), "Eosinophilic gastroenteropathy: a pediatric series", *Arch. Argent. Pediatr.*, **109(1)**, pp.68-73.

[11] A.J. Lucendo, A. Arias (2012), "EGE: an update", *Expert Rev. Gastroenterol. Hepatol.*, **6(5)**, pp.591-601.

[12] R.A. Abou, H.W. El (2016), "EGE: approach to diagnosis and management", *World J. Gastrointest. Pharmacol. Ther.*, **7(4)**, pp.513-523.

[13] C. Reed, J.T. Woosley, E.S. Dellon (2015), "Clinical characteristics, treatment outcomes, and resource utilization in children and adults with EGE", *Dig. Liver Dis.*, **47(3)**, pp.197-201.

[14] E. Cavalli, et al. (2019), "Eosinophilic esophagitis in children: doubts and future perspective", *J. Transl. Med.*, **17**, DOI: 10.1186/s12967-019-2014-0.