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EOSINOPHILIC GASTROENTERITIS: A RARE CAUSE OF PERSISTENT ABDOMINAL PAIN, DIARRHOEA AND ASCITES.

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ABSTRACT

Eosinophilic gastroenteritis is a rare disease, and it can pose diagnostic challenges in areas where parasitic infection is common. The condition is characterized by eosinophilic infiltration of the bowel wall with various clinical manifestations based on the bowel layers predominantly affected. It is typically treated with corticosteroid. In this case report, we describe a 33 years old lady with gastrointestinal symptoms of epigastric pain, vomiting and diarrhoea with mild ascites. She underwent extensive investigations including ultrasound guided paracentesis with ascitic fluid analysis which showed predominantly eosinophils cells. She was eventually diagnosed with eosinophilic gastroenteritis and was treated with tapering dose of prednisolone, and her symptoms gradually resolved.

Keywords: Abdominal pain, Diarrhoea, Eosinophilic gastroenteritis, Eosinophilic ascites, Gastrointestinal.

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ABSTRACT

Eosinophilic gastroenteritis is a rare disease, and it can pose diagnostic challenges in areas where parasitic infection is common. The condition is characterized by eosinophilic infiltration of the bowel wall with various clinical manifestations based on the bowel layers predominantly affected. It is typically treated with corticosteroid. In this case report, we describe a 33 years old lady with gastrointestinal symptoms of epigastric pain, vomiting and diarrhoea with mild ascites. She underwent extensive investigations including ultrasound guided paracentesis with ascitic fluid analysis which showed predominantly eosinophils cells. She was eventually diagnosed with eosinophilic gastroenteritis and was treated with tapering dose of prednisolone, and her symptoms gradually resolved.

Keywords: Abdominal pain, Diarrhoea, Eosinophilic gastroenteritis, Eosinophilic ascites, Gastrointestinal.

INTRODUCTION

Eosinophilic gastroenteritis (EG) is a spectrum of primary eosinophilic gastrointestinal disorders (EGIDs).[1] EG can involve the mucosa, submucosa or serosa. The clinical features of EG depends on gastrointestinal wall involvement. Mucosal involvement is typically characterized by non-specific abdominal pain and some degree of malabsorption; Submucosal involvement, central abdominal pain and obstructive symptoms; and serosal involvement, generalized central abdominal pain and ascites. However, symptoms can overlap based on infiltration severity and affected layers.[2,3]

Diagnosis is often challenging and a high clinical index suspicion of peripheral hypereosinophilia is often required, with histological confirmation via bowel biopsy is frequently necessary. Due to the rarity of EG, other gastrointestinal disorders must be ruled out. Once the diagnosis is confirmed, the condition can be treated with steroids.

Herein, we report a young female patient presenting with epigastric pain, diarrhoea and ascites, whose diagnosis of eosinophilic ascites was finally confirmed with thorough medical examinations and appropriate investigations.

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CASE REPORT

A 33-year-old woman with no significant medical history, consulted a general practitioner due to epigastric pain. She was diagnosed with dyspepsia, and symptomatic treatment with antacid was provided. However, she again visited her physician the following day due to the onset of new symptoms of diarrhoea and vomiting but no fever. She was diagnosed as acute gastroenteritis and treated symptomatically but with no antibiotics at this stage.

Her symptoms persisted and she presented to the Emergency Department. She was given analgesia which relieved the pain. She was discharged home and was referred to Gastroenterology Unit for gastroscopy. Upper gastrointestinal endoscopy was performed one week after that. The gastroscopy was reported as normal and biopsy was taken.

She again represented to the emergency department because of recurrent epigastric pain and diarrhoea. On abdominal examination, there was mild shifting dullness, and no stigmata of chronic liver disease. Other examinations were unremarkable. The full blood count (FBC) results were as follows: white blood cell (WBC) count, $15.7x10^3$/uL (4.2–12.6 $x10^3$/uL) and eosinophil count, $5.1x10^9$/L (0.0–$0.7x10^9$/L). The rest of other blood test results were normal. Abdominal and pelvic ultrasonography revealed moderate amount of ascites and normal liver, uterus and ovary. The patient was admitted to Gastroenterology unit for further evaluation.

She underwent ultrasound-guided paracentesis and the ascitic fluid sample was taken for analysis. The ascitic fluid's red blood cell count was 9,050/high power field (hpf), and the WBC count was 7,040/hpf. No organisms were identified on gram staining, and the acid-fast bacilli (AFB) staining result was also negative. The serum ascites albumin gradient was 7 g/L, and the protein was extremely high at 46 g/L. Due to concerns of secondary bacterial peritonitis, the patient underwent abdominal and pelvic computed tomography (CT) scan, which was reported as ascites with no evidence of bowel disease or perforation. She was started on IV ceftriaxone 1 g twice daily. At that time, antihelminth treatment was not considered due to the low risk of helminth infection.

Colonoscopy was conducted, and was reported as normal. The CA 125 level increased to 423 (normal range: 0.0–35.0) U/mL and other tumour markers were normal. The biopsies taken during gastroscopy and colonoscopy were reported as normal. In addition, her stool microscopy and culture findings were negative. A repeat FBC on the 5th day of admission revealed WBC eosinophil count of $6.3x10^9$/L. The ascitic fluid cytology result also confirmed moderate cellularity mostly comprising eosinophils (90%) with few neutrophils, histiocytes and mesothelial cells. However, no malignant cells were observed (Figure 1). With these findings, the final diagnosis of eosinophilic gastroenteritis with eosinophilic ascites was made. Oral prednisolone 30 mg once daily was started. The patient was discharged home and was followed up in the Gastroenterology clinic after treatment with oral prednisolone for 16 days. On follow-up review, her symptoms

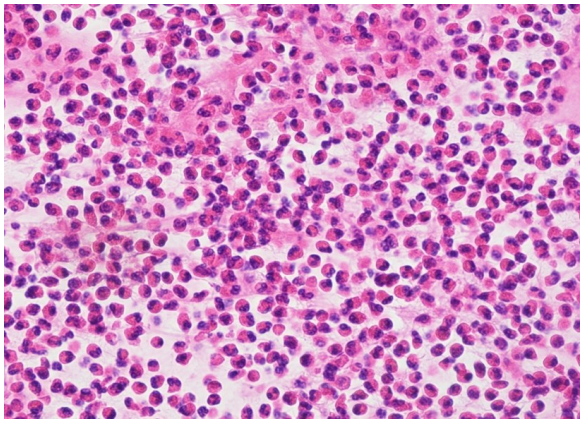

Figure 1: Abundant eosinophils comprising more than 90% of the cells. No malignant cells were observed. (Click on the image to enlarge).

have resolved completedly. Repeat eosinophil count was $0.7x10^9$/L. The prednisolone dose was tapered down and gradually discontinued.

DISCUSSION

EG is characterized by eosinophilic inflammation of the bowel wall with various gastrointestinal manifestations. Its prevalence in the United States was about 8.4 per 100,000 persons.[4] EG can affect any age group. However, it is commonly observed in children age less than 5 years old with a female preponderance.[4]

EG is a spectrum of primary EGIDs. Other than EG, primary EGIDs also comprise of eosinophilic oesophagitis, eosinophilic gastritis and eosinophilic colitis.[1,4] Primary EGID is a spectrum of inflammatory gastrointestinal disorders caused by infiltration of eosinophils into the gastrointestinal wall.

The cause of EG is still unknown and not well understood. EG was found to be associated with seasonal allergy, food sensitivity, eczema, asthma and atopy.[5,6] This result indicates that hypersensitivity reactions play a major role in the pathogenesis of the disease.

The clinical features of EG are based on the depths of eosinophilic infiltration of the gastrointestinal wall.[5] This disease can mimic various gastrointestinal diseases due to its wide spectrum of presentations. The mucosa is most commonly affected in 25%–100% of cases, and the clinical features include generalised central abdominal pain, diarrhoea, dyspepsia, anorexia, bloating, nausea, vomiting, gastrointestinal haemorrhage and weight loss due to malabsorption.[2,5,6] If muscularis of the gut wall is affected, it can present as pyloric outlet and small bowel obstruction. If the serosa is involved, it causes eosinophilic ascites (EA) and peritonitis.[5] In addition, EG involving the biliary system and pancreas can present as obstructive jaundice and acute pancreatitis, respectively.

EG can sometimes mimic acute abdominal conditions such as acute appendicitis. Hence, its diagnosis can be challenging.[6] The serum eosinophil count is increase in 20%–90% of cases.[5] The serum eosinophil count is indicative of symptom severity.[5] However, EG can also occurs independent of serum eosinophil count. Secondary causes of gut eosinophilia should be ruled out, for instance, gastrointestinal infections caused by different organisms (helminths and fungi), hypereosinophilic syndrome (HES), systemic disease (connective tissue disease, vasculitis, coeliac disease and inflammatory bowel disease), malignancies (gastric cancer and lymphoma) and other conditions attributed to the use of drugs (e.g. naproxen).[1,2]

There are no set standard criteria for diagnosing EG. The conclusive diagnosis of EG is obtained based on histological evidence of eosinophilic infiltration.[6] Endoscopic examination with multiple mucosal biopsies for histological confirmation remains the gold standard for diagnosis. However, due to the patchy distribution of the lesions, the affected area can be missed. Thus, biopsies at multiple sites are recommended to prevent obtaining false-negative results. Histological examination must reveal more than 20 eosinophils per hpf to confirm the diagnosis.[5,6] Other findings on histological examination include crypt hyperplasia and eosinophilic infiltration of the lamina propria.[2] In the presence of ascites, EA is characterised by the presence of up to 95% eosinophil counts in the ascitic fluid. [5,7]

Other medical examinations should be performed to rule out other causes of hypereosinophilia. Stool examination must be conducted to assess for parasitic infection. Abdominal ultrasonography usually reveals ascites in EA. CT scan shows thickening of the

bowel wall. However, this finding is not specific as it can be observed in other inflammatory bowel diseases.

Glucocorticoid is the mainstay treatment for EG. Prednisolone at a starting dose of 20–40 mg is commonly recommended and the dose is gradually tapered down. It is prudent to exclude parasitic infection prior to starting steroid therapy since initiation of steroid therapy in patients with this condition may lead to disseminated infection and death. Symptoms usually resolve within a few weeks after starting treatment. The optimal treatment duration is unknown.[5,6] Steroid-sparing medications such as sodium cromoglycate, ketotifen and montelukast have also been used for the management of EG.[5,6]

The clinical course of EG varies. Some patients with refractory or recurring symptom might require long-term treatment with prednisolone at maintenance dose.[5,6] Flare up of the disease can occur after months or years of remission, and this can be managed by providing prednisolone at tapered doses.

CONCLUSION

In summary, EG is a rare disease, and it can mimic other gastrointestinal diseases. Clinicians should have a high index of suspicion for EG specifically when a patient presents with gastrointestinal symptoms and high serum eosinophil count, after ruling out other causes that can increase peripheral eosinophil count. Endoscopy with multiple biopsies at multiple sites are recommended for confirmation of EG. EG is treatable with steroids.

DECLARATION OF CONFLICTING INTEREST

We declare that we have no conflict of interest with regards to publication of this case report.

INFORMED CONSENT

Verbal informed consent (verbal) has been obtained from the patient to publish the material.

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