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Case Report

A Case of Eosinophilic Gastroenteritis in a Young Female Peritoneal Dialysis

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Abstract

Eosinophilic gastroenteritis in peritoneal dialysis patients is a rare disease reported in the literature that is unrecognized. Diagnosis requires histological confirmation by gastrointestinal tissue biopsy. We report a case of a 23-year-old peritoneal dialysis female diagnosed with eosinophilic gastroenteritis who was treated with steroids and who has responded to treatment.

Keywords: Eosinophilic Gastroenteritis; Peritoneal Dialysis; Steroids

Introduction

Eosinophilic Gastroenteritis (EGE) is an inflammatory disorder characterized by eosinophilic infiltration of the stomach and duodenum, and in some cases, the esophagus and colon without any known cause of eosinophilia [1]. Although reported in patients of any age, it may typically present in the third through fifth decades and has a peak age of onset in the third decade with an incidence rate of 22 to 28 per 100,000 persons in the United States [2,3]. It has been suggested that it is underdiagnosed due to lack of recognition. However, to our knowledge, eosinophilic gastroenteritis in peritoneal dialysis patients is a rare disease that is not reported in the literature when we search PubMed using the key words.

Case History

We present a case of a 23-year-old female Peritoneal Dialysis (PD) who was admitted to hospital for symptom of refractory abdominal pain, diarrhea and anorexia. She, a HBV carrier, had no past allergic history and was taking regular renal replacement and medical treatment. She was a nonsmoker and did not drink alcohol. The mode of Continuous Ambulatory Peritoneal Dialysis(CAPD) is 1.5% Baxter dialysis solution containing sodium lactate 2L×4 circles per week. The catheter worked normally. Blood routine examination reveal that the proportion of eosinophil is up to 33.2% (peripheral eosinophil count of 3.48×10^9/L) . Urine analysis showed specific gravity 1.007, 3+ protein,1+ blood and red blood cell 46.3/μl. Routine stool test and stool culture was negative. Serologic evaluation for Antinuclear Antibody (ANA), Anti-Neutrophil Cytoplasmic Antibody (ANCA), complement, HIV, and hepatitis C virus was negative. This patient is hepatitis B surface antigen and hepatitis B core antibody positive. Laboratory evaluation revealed BUN of 7.10mmol/L, serum creatinine of 745.9μmol/L, serum potassium of 3.18mmol/L, calcium of 2.12 mmol/L, phosphorus of 1.45mmol/L, albumin of 34.5g/L, procalcitonin of 0.42ng/ml, C-reaction protein of less than 5.0mg/L. Hepatic function (liver enzymes, total bilirubin) is normal. Hematological malignancies were suspected but bone marrow puncture is negative. The ascites culture is negative. The CT Scan-Abdomen and abdomen ultrasound both revealed the existence of ascites and narrowing kidneys without obstruction, stones, or masses. Physical examination revealed that T was 36.5℃, BP was 133/83 mmHg, pulse was 98. Other physical examination features, apart from midsection tenderness, are unremarkable. There was no clinical evidence of congestive heart failure or peripheral vascular disease. After anti-infective therapy and symptomatic and supportive treatment, the symptom of abdominal pain, diarrhea and anorexia remained. However the suspension of PD made symptomatic relief. When we continue PD, the symptom occurred again. To

maintain renal replacement therapy, we changed the PD mode to Hemodialysis (HD. The initiate of HD made symptomatic relief. Based on these findings a gastrointestinal endoscopes was ordered. Results of gastrointestinal tissue biopsy showed active chronic mucosa inflammation and massive eosinophils infiltration (20-60/HP) (Figure 1), and a diagnosis of EGE was made.

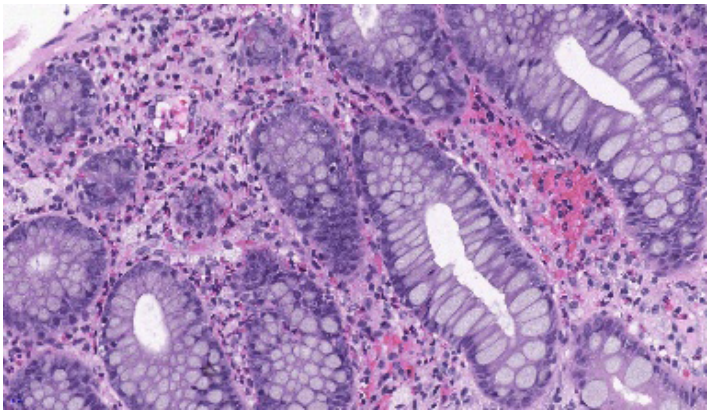

Figure 1: Results of gastrointestinal tissue biopsy (HE 40×10).

Peritoneal dialysate was collected soon. Fluid analysis show eosinophilia (Figure 2). Our patient was commenced on a course of intravenous steroids and following this her symptom rapidly controlled and routine analysis of blood show the reduction of eosinophil to 0.2% (peripheral eosinophil count of 0.01×10^9/L).

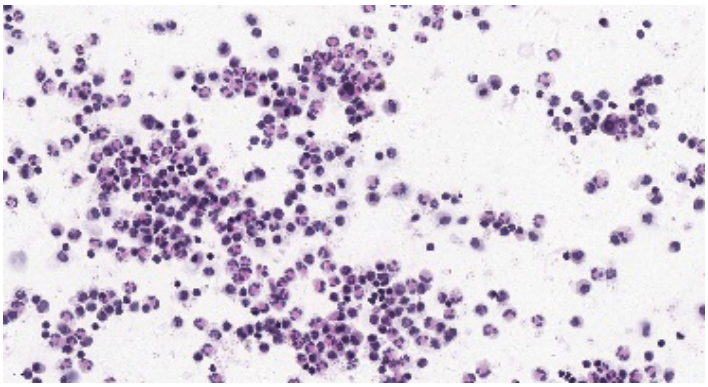

Figure 2: The fluid analysis of peritoneal dialysate (HE 40×10).

Discussion

This case report presents several teaching and learning points about the diagnosis and management of EGE. It has been reported that EGE is under recognized with physicians not familiar with this disorder. The pathogenesis of EGE is not well understood, but epidemiologic and clinical features suggest an allergic component [2,4,5]. Approximately one-half of patients have a history of allergic disease including asthma, defined food sensitivities, eczema, or rhinitis [4,5]. EGE should be suspected in a patient with abdominal pain, nausea, vomiting, early satiety, diarrhea, weight loss, or ascites that are associated with peripheral eosinophilia and/or a history of food allergy or intolerance. The definitive diagnosis of EGE is based on the presence of eosinophilic infiltration of the gastrointestinal tract on biopsy and/or eosinophilic ascitic fluid, lack of involvement of other organs, and absence of other causes of intestinal eosinophilia. Other diseases such as intestinal parasites, malignancy, inflammatory bowel disease, hypereosinophilic syndrome and rheumatoid vasculitis in which gastrointestinal symptoms are associated with peripheral eosinophilia can be distinguished from Eosinophilic Gastroenteritis (EGE) based on the clinical presentation, laboratory tests, and/or biopsies of the gastrointestinal tract.

The biopsy results of the eosinophilic infiltration of the muscle layer strongly support the diagnosis. Our patient is steroid-responsive. The guideline recommend that in patients who decline a dietary approach or whose symptoms do not improve after dietary therapy for six weeks, we suggest a trial of prednisone (20 to 40 mg/day) (Grade 2C) [6,7].Improvement usually occurs within two weeks regardless of the layer of bowel involved. Prednisone should then be tapered rapidly over the next two weeks. However, some patients require prolonged therapy (up to several months) for resolution of symptoms. Patients who relapse immediately after steroid cessation may need long-term, low-dose maintenance therapy with prednisone or budesonide. In our patient, peritoneal dialysate or/and accessory device may be the cause of her illness. Due to the patient's poor economic situation, serum immunoglobulin E (IgE) and Interleukin(IL)-3, IL-5, and granulocyte macrophage-colony stimulating factor couldn't be evaluated. Regular follow-up should be conducted to know the prognosis.

Conclusions

EGE is an underdiagnosed and rare disorder and should be considered in the differential for any patient presenting with gastrointestinal symptoms and peripheral eosinophilia. Gastrointestinal endoscopy and biopsy may increase detection of EGE. EGE is responsive to corticosteroids.

Acknowledgements

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