Eosinophilic colitis as an unusual cause of severe bloody diarrhea

Şiddetli kanlı ishalin bir sebebi olarak eozinofilik kolit

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Eosinophilic gastrointestinal diseases may affect the colon; however, isolated colonic involvement is very rare. Diagnosis of eosinophilic colitis requires clinical suspicion and it should be differentiated from other disorders causing eosinophil accumulation in the colon tissue. Herein we present a 46-year-old female admitted to the hospital with the complaints of bloody diarrhea (25-30 times a day), fever, abdominal pain and weight loss. Eosinophilic colitis was diagnosed and she was treated with steroid successfully.

Key words: Eosinophilic colitis, hypereosinophilia, bloody diarrhea

INTRODUCTION

Eosinophilic gastrointestinal disease is rare, and isolated colonic involvement appears to be very sporadic (1, 2). Since clinical and endoscopic features of eosinophilic colitis (EC) are not characteristic, it may be difficult to determine isolated colonic involvement. The differential diagnosis of EC requires clinical suspicion.

Herein we present a new case with EC. We also discuss the pathogenesis and therapeutic approaches in these patients in the light of the pertinent literature.

CASE REPORT

A 46-year-old female was admitted to the hospital with the complaints of bloody diarrhea (25-30 times a day), fever, abdominal pain and weight loss (12 kg) for a month. Past medical history revealed mitral valve replacement 12 years ago. She denied any exposure to toxins or other drugs other than warfarin and digoxin, and she had no asthma or allergic diseases.

Physical examination revealed all quadrant tenderness and rectal bleeding. Laboratory findings were as follows: hemoglobin 10.0 g/dl, WBC 12,100/mm³, eosinophil count 2,300/mm³ (0–700/mm³), and platelet count 488,000/mm³. Biochemical tests were within normal limits other than albumin 3.1 g/dl. Stool was positive for Charcot-Leyden crystals. Serum immunoglobulin E (IgE) concentration was normal. The relevant serological tests (antinuclear antibody and antibodies to extractable nuclear antigens) were negative.

Endoscopic examination of upper gastrointestinal system revealed antral gastritis. Mucosa and lumen of the duodenum were normal. An abdominal sonography revealed thickening of the wall of the rectum (7.8 mm). Colonoscopy showed mucosal ulcerations localized to the transverse colon, splenic flexura, and descending and sigmoid colon (Figure 1). Histology of colonic biopsy samples showed active inflammatory reaction with focal clusters of eosinophils in lamina propria, consistent with EC.
There was no cryptitis, cryptic abscess or atrophy. No ischemic or vasculitic features were identified (Figure 2).

She was treated with systemic steroids for two weeks and became well. At a follow-up visit one month after the discharge, colonoscopy and control biopsy were normal.

**DISCUSSION**

Eosinophilic infiltration of the gastrointestinal tract in relation with a recognizable clinical picture is an uncommon entity. Diagnosis of EC requires clinical suspicion and it should be differentiated from other disorders (inflammatory bowel disease, parasitic diseases, eosinophilic gastroenteritis, infections, drug reactions, vasculitis) causing eosinophil accumulation in the colon tissue (3).

The pathogenesis of EC is not clear and it is considered to be idiopathic. Role of eotaxin-1 and eosinophils in the pathogenesis of the disease provides a possible hypothesis for eosinophil-induced gastrointestinal dysfunction. Excessive accumulation of eosinophils results in release of eosinophil major protein causing destruction of the intestinal epithelium (4, 5). Recently, gastrointestinal hype-reosinophilic disorders have been classified into three categories: IgE-mediated, partially IgE-mediated and cell-mediated (6). In contrast to other gastrointestinal hypereosinophilic disorders, EC is usually a non-IgE–associated disease. Some studies point to a T lymphocyte–mediated process, but the exact immunologic mechanisms responsible for this condition have not been identified (7). Similarly, our case had normal serum IgE.

Similar to eosinophilic gastroenteritis, there are a variety of symptoms associated with EC, depending on the degree and location of tissue involvement. There are three specific patterns of eosinophilic infiltration of the gastrointestinal tract reflected in the presentation. Mucosal disease is usually associated with protein-losing enteropathy, subserosal disease with eosinophilic ascites, and transmural disease with ileus or perforation (8). Hypoalbuminemia may have been due to protein-losing enteropathy or active gastrointestinal bleeding in our case.

Three criteria were defined for EC diagnosis: the presence of gastrointestinal symptoms, biopsies showing eosinophilic infiltration of one or more areas of the gastrointestinal tract from the esophagus to colon, or characteristic radiologic findings with peripheral eosinophilia and no evidence of parasitic or extraintestinal disease (5). All of these criteria were present in our case. The peripheral eosinophilic count seems to be normal in some patients with EC, suggesting that it is not a reliable diagnostic criterion. Therefore, no single test is the gold standard for diagnosis, but peripheral blood eosinophilia or eosinophils in the stool are suggestive of EC (5, 9, 10).

Treatment of EC varies, primarily depending on the disease subtype. Drugs such as cromoglycate, montelukast, and histamine receptor antagonists are generally not successful. Anti-inflammatory drugs, including aminosalicylates and systemic or topical glucocorticoids, are commonly used and appear to be efficacious. There are several forms of topical glucocorticoids designed to deliver drugs to the distal colon and rectum, but EC typically also

**Figure 1.** Colonoscopic finding of the patient showing mucosal ulcerations.

**Figure 2.** Histology of colonic biopsy demonstrating active inflammation and focal eosinophilic clusters in lamina propria.
involves the proximal colon, as in our case. In severe cases, refractory or dependent on systemic glucocorticoid therapy, intravenous alimentation or immunosuppressive therapies such as azathioprine or 6-mercaptopurine are alternatives (11).

In conclusion, EC may present with severe bloody diarrhea. It should be kept in mind especially in the differential diagnosis of inflammatory bowel diseases.

REFERENCES