Turk J Gastroenterol 2008; 19 (1): 54-56

Eosinophilic colitis as an unusual cause of severe bloody diarrhea

Şiddetli kanlı ishalin bir sebebi olarak eozinofilik kolit

İbrahim ERTUĞRUL[1], Aysel ÜLKER[1], Nesrin TURHAN[2], Ülkü DAĞLI[1], Nurgül ŞAŞMAZ[1]

Departments of [1]Gastroenterology and [2]Pathology, Türkiye Yüksek İhtisas Hospital, Ankara

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

Eozinofilik gastrointestinal hastalıklar kolonu etkileyebilir. Bununla birlikte izole kolon tutulumu oldukça nadirdir. Eozinofilik kolit tanısı için klinik olarak şüphelenmek gerekir. Kolonda eozinofil birikimine neden olan diğer hastalıklardan ayırtedilmelidir. Burada günde 25-30 defa kanlı ishal, yüksek ateş, karın ağrısı ve kilo kaybı şikayetleri ile hastaneye başvuran 46 yaşındaki bayan hastada eozinofilik kolit tanısı konulmuş ve steroid ile başarılı bir şekilde tedavi edilmiş bir vaka sunulmuştur.

Anahtar kelimeler: Eozinofilik kolit, hipereozinofili, kanlı ishal

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/mm^3, eosinophil count 2,300/mm^3 (0–700/mm^3), and platelet count 488,000/mm^3. 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.

Address for correspondence: İbrahim ERTUĞRUL
Demirlibahçe Mahallesi Plevne Caddesi, 38/27
06080, Aktaş, Mamak, Ankara, Turkey
Tel: +90 312 320 22 32 • Fax: +90 312 312 41 20
E-mail: ibrahimer16@yahoo.com

Manuscript received: 01.03.2007 **Accepted:** 19.04.2007

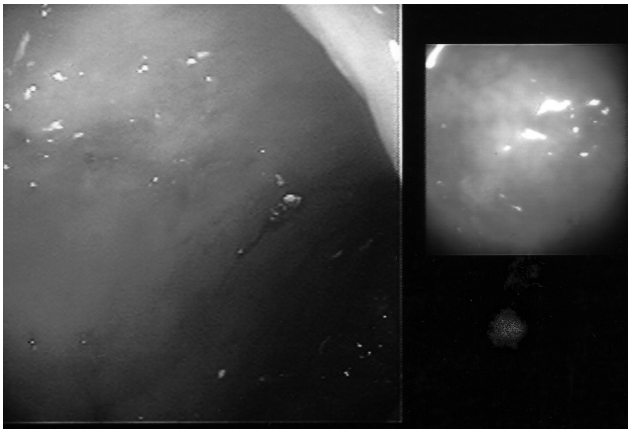

Figure 1. Colonoscopic finding of the patient showing mucosal ulcerations.

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).

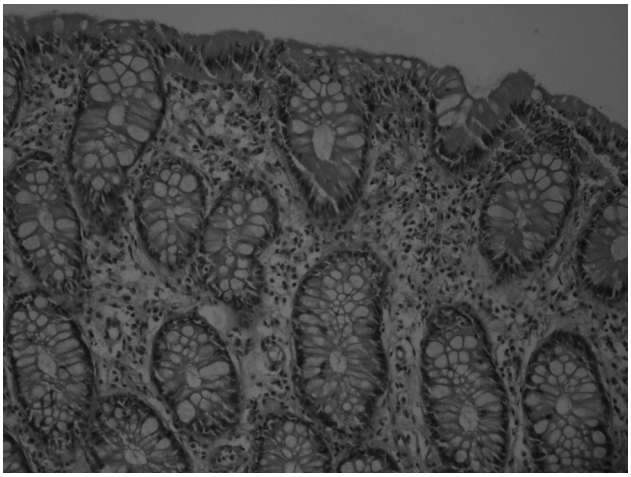

Figure 2. Histology of colonic biopsy demonstrating active inflammation and focal eosinophilic clusters in lamina propria.

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 eosinophilic major protein causing destruction of the intestinal epithelium (4, 5). Recently, gastrointestinal hypereosinophilic 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

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

1. Naylor AR, Pollet JE. Eosinophilic colitis. Dis Colon Rectum 1985; 28: 615-8.
2. Moore D, Lichtman S, Lentz J, et al. Eosinophilic gastroenteritis presenting in an adolescent with isolated colonic involvement. Gut 1986; 27: 1219-22.
3. Rothenberg ME. Eosinophilia. N Engl J Med 1998; 338: 1592-600.
4. Carpenter HA, Talley NJ. The importance of clinicopathological correlation in the diagnosis of inflammatory conditions of the colon: histopathological patterns with clinical implications. Am J Gastroenterol 2000; 95: 878-96.
5. Talley NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosa, muscle layer, and subserosal tissue. Gut 1990; 31: 54-8.
6. Sampson HA. Food allergy: Part 1. Immunopathogenesis and clinical disorders. J Allergy Clin Immunol 1999; 103: 717-28.
7. Van Sickle GJ, Powell GK, McDonald PJ, Goldblum RM. Milk- and soy protein-induced enterocolitis: evidence for lymphocyte sensitization to specific food proteins. Gastroenterology 1985; 88: 1915-21.
8. Losanoff JE, Kjossev KT, Katrov E. Eosinophilic enterocolitis and visceral neuropathy with chronic intestinal pseudoobstruction. J. Clin Gastroenterol. 1999; 28: 368-71.
9. Whitington PF, Whitington GL. Eosinophilic gastroenteropathy in childhood. J Pediatr Gastroenterol Nutr 1988; 7: 379-85.
10. Hoefer RA, Ziegler MM, Koop CE, Schnaufer L. Surgical manifestations of eosinophilic gastroenteritis in the pediatric patient. J Pediatr Surg 1977; 12: 955-62.
11. Rothenberg ME. Eosinophilic gastrointestinal disorders. Allergy Clin Immunol 2004; 113: 11-28.