

Severe exudative ascites as an initial presentation of Crohn's disease

Crohn hastalığında ilk başvuru yakınması olarak ciddi eksudatif asit

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Ascites in the course of Crohn's diseases is rarely seen and usually is a result of a concomitant malignancy, infection or thrombosis of portal or hepatic veins. In this article, a 55-year-old female patient suffering from severe ascites and finally diagnosed as Crohn's disease is presented. Further investigations revealed no malignancy, peritoneal infection or vein thrombosis. Treatment of Crohn's disease with steroids and 5-aminosalicylic acid also resulted in the disappearance of the ascites.

Key words: Ascites, Crohn's disease, etiology, malignancy, thrombosis

Crohn hastalığı seyrinde asit nadiren görülür ve genellikle eşlik eden bir maligniteye, enfeksiyona veya portal veya hepatik ven trombozuna bağlı gelişir. Bu yazıda, asit ile prezente olup Crohn hastalığı tanısı alan 55 yaşında kadın olgu sunulmaktadır. İleri incelemelerde herhangi bir malignite, peritoneal enfeksiyon veya ven trombozu saptanmamıştır. Crohn hastalığına yönelik uygulanan steroid ve 5-aminosalisilik asit ile olgunun asidi de kaybolmuştur.

Anahtar kelimeler: Asit, Crohn hastalığı, etyoloji, malignite, tromboz

INTRODUCTION

Crohn's disease is a chronic inflammatory granulomatous disease of the gastrointestinal tract. The well-known associated hypercoagulability may contribute to occurrence of portal or hepatic vein thrombosis which may result in ascites (1, 2). In addition, chronic inflammation is thought to contribute to the development of malignancy, particularly lymphoma, in patients with Crohn's disease. These patients with malignancy may also present with ascites (3, 4). Furthermore, infections may also be one of the etiological factors of ascites. However, ascites in the course of Crohn's disease without any other cause is rare, and still has unknown mechanism(s) (5, 6). We present a patient with Crohn's disease with exudative ascites as an initial presentation without any other cause.

CASE REPORT

A 55-year-old female patient was admitted to our department with a 10-day episode of bloody diarr-

hea occurring four to six times per day and associated with abdominal pain. Her history revealed no recent travel or use of antibiotics. Family history was negative for inflammatory bowel disease. At admission, her physical examination was unremarkable except for the presence of ascites. Her abdomen was distended and tenderness with a fluid wave was detected during percussion. Blood pressure was 110/60 mmHg, temperature 36.5°C, and pulse rate 82/min. The laboratory findings revealed anemia due to chronic disease (Table 1). Folate and vitamin B₁₂ levels were normal. Albumin level was in normal ranges. Erythrocyte sedimentation rate was 48 mm/hour and C-reactive protein was 2.9 mg/dl (normal: <0.5 mg/dl). Liver and renal function tests, electrolytes, and urinalysis were normal. Tests for antinuclear antibody and anti-double-stranded DNA were negative. Stool culture was negative three times. The purified protein derivatives test revealed a negative reaction 3 mm in diameter.

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Table 1. Laboratory data of peripheral blood and ascites on admission

Peripheral blood		Ascites	
White blood cell	7700/mm ³	White blood cell	100/mm ³
Hemoglobin	7.8 g/dl	Culture	negative
Hematocrit	26%	PCR for tuberculosis	negative
Platelet count	349x10 ³ /mm ³	Culture for tuberculosis	negative
Total protein (6.5-8.5 g/dl)	6g/dl	Total protein (g/dl)	4 g/dl
Albumin (3.5-5.0 g/dl)	3.3g/dl	Albumin (g/dl)	2.6 g/dl
Iron (50-140 µg/dl)	11µg/dl	SAAG	0.7
Transferrin saturation	35%	Cytology	benign
Ferritin (13-150 ng/ml)	148ng/ml	Glucose	76g/dl
LDH (230-460 U/l)	280U/L	LDH	170U/L

PCR: Polymerase chain reaction, SAAG: Serum-ascites albumin gradient, LDH: Lactate dehydrogenase

Abdominal ultrasonography confirmed the ascites and revealed normal echo and size of the liver, and no thrombosis of hepatic or portal vein was detected by Doppler ultrasonography. Serum-ascites albumin gradient (SAAG) was 0.7, which strongly excluded portal hypertension. Analysis of ascitic fluid showed no bacterial or tuberculous infection (Table 1). In addition, cytological examination of the ascitic fluid was repeatedly negative for malignancy. Meanwhile, two units of erythrocyte replacement were performed because of severe anemia.

Upper gastrointestinal system endoscopy was normal. Laparoscopy showed no pathological finding to identify the etiology of ascites. Colonoscopy revealed normal mucosa of the colon until the cecum. There was severe mucosal inflammation at the cecum including friability, hyperemia and extensive ulcers. Histopathological examination of the specimen obtained from the cecum corroborated the inflammation, and polymerase chain reaction (PCR) for tubercle bacilli was negative. The radiography of the small bowel with air contrast

barium enema showed thickening of the bowel wall, luminal narrowing, and mucosal ulceration at the level of the terminal ileum and cecum, which was consistent with Crohn's disease (Figure 1). She was diagnosed as Crohn's disease with these clinical, colonoscopical, histopathological and radiological features. Treatment with steroid (40 mg/day), and 5-aminosalicylic acid (2 g/day) was started. The patient was given no antibiotics, diuretics or albumin replacement. Her symptoms began to diminish in the 1st week and disappeared in the 3rd week of the treatment. In addition, near disappearance of the ascites was observed in the 3rd week of the treatment. The patient is still free of symptoms and there has been no recurrence of ascites after three months of follow-up.

DISCUSSION

Ascites in the course of Crohn's disease is an unexpected complication, and few cases are reported in the literature. Some cases with ascites have also had hepatic or portal vein thrombosis since hypercoagulability exists in patients with Crohn's disease. In a recently published study, 98 cases with inflammatory bowel disease and confirmed deep venous thrombosis or pulmonary embolism were checked for thrombophilic state. The authors reported that thrombophilia was present in 33% of the 40 patients tested (7). The prevalence of Leiden mutation, the most common inherited thrombophilic state, was reported as 14.28% in Crohn's disease in another study (8). Magro *et al.* found that the frequency of two or more thrombophilic abnormalities was related to disease activity in Crohn's disease (9). Thus, when a patient with Crohn's disease suffers from ascites, presence of portal or hepatic vein thrombosis has to be kept in mind. Tsujikawa *et al.* reported a patient with Crohn's disease and portal vein thrombosis which was successfully treated with anticoagulant

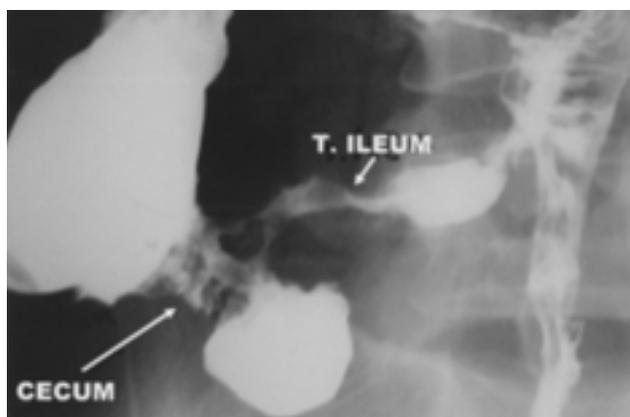


Figure 1. The radiography of the small bowel with air contrast barium enema. Note the luminal narrowing and mucosal ulceration at the level of the terminal ileum (small arrow) and cecum (big arrow)

therapy (2). In our patient, Doppler ultrasonography was performed and revealed no thrombosis of portal or hepatic veins. In addition, SAAG was smaller than 1.1, which excluded portal hypertension. Hypoalbuminemia may cause ascites in patients with Crohn's disease, but serum albumin level was in normal ranges in our patient. Since SAAG was smaller than 1.1, we especially focused on malignancy and infections. Another known etiology of ascites in the course of Crohn's disease is malignancy, especially lymphoma (3, 4). It is thought that chronic inflammation contributes to the development of lymphoma in patients with Crohn's disease. In our patient, repeated cytological examinations of the ascitic fluid revealed no malignancy, and microbiological investigations showed no bacterial or tuberculous infection. Furthermore, laparoscopy excluded both peritonitis carcinomatosa and peritoneal infection. No cause of the ascites except Crohn's disease itself was found. To our knowledge, only two cases with Crohn's diseases and severe exudative ascites ha-

ve been reported in the literature previously (5, 6). It can be speculated that the inflammatory nature of Crohn's disease may be the reason for the associated ascites via transmural inflammation and/or lymphatic stasis. If so, ascites would usually be seen in patients with Crohn's disease when it is more severe or exacerbated. However, to our observation, ascites does not appear in most of the patients at the exacerbation time of Crohn's disease. Thus, it is difficult to explain the occurrence mechanism(s) of ascites by only severity of the disease. On the other hand, treatment of Crohn's disease with anti-inflammatory drugs without any antibiotics, diuretics or albumin replacement resulted in the disappearance of ascites in our patient.

In conclusion, occurrence mechanism(s) of ascites in the course of Crohn's disease remains unknown. Treatment of Crohn's disease with anti-inflammatory drugs may result in dramatic disappearance of ascites; however, further investigations with large series are needed.

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