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Toxic Amebic Colitis Coexisting with Intestinal Tuberculosis

A patient with a fulminant amebic colitis coexisting with intestinal tuberculosis had a sudden onset of crampy abdominal pain, mucoid diarrhea, anorexia, fever and vomiting with signs of positive peritoneal irritation. Fulminant amebic colitis occurring together with intestinal tuberculosis is an uncommon event and may present an interesting patho-etiological relationship. The diagnosis was proven by histopathologic examination of resected specimen. Subtotal colectomy including segmental resection of ileum, about 80 cm in length, followed by exteriorization of both ends, was performed in an emergency basis. Despite all measures, the patient died on the sixth postoperative day. The exact relationship of fulminant amebic colitis and intestinal tuberculosis is speculative but the possibility of a cause and effect relationship exists. Fulminant amebic colitis may readily be confused with other types of inflammatory bowel disease, such as idiopathic ulcerative colitis, Crohn's disease, perforated diverticulitis and appendicitis with perforation. This report draws attention to the resurgence of tuberculosis and amebiasis in Korea, and the need for the high degree of caution required to detect it.

Key Words: *Dysentery, Amebic; Colitis; Tuberculosis, Intestinal*

Seung Cheol Park, Hae Myung Jeon, Jeong Soo Kim, Won Woo Kim, Ki Whan Kim, Seung Taek Oh, Eung Kook Kim, Suk Kyun Chang, Eun Jung Lee*

Departments of Surgery and Clinical Pathology*, St. Mary's Hospital College of Medicine, The Catholic University of Korea, Seoul, Korea

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Address for correspondence
Hae Myung Jeon, M.D.
Department of Surgery, St. Mary's Hospital, College of Medicine, The Catholic University of Korea, 62 Yoido-dong, Youngdeungpo-gu, Seoul 150-010, Korea
Tel: +82.2-3779-1175, Fax: +82.2-786-0802
E-mail: colon@cmc.cuk.ac.kr

INTRODUCTION

Fulminant amebic colitis with perforation is an extremely rare manifestation of *Entamoeba histolytica*, and intestinal tuberculosis comprises less than 1 percent of all proven or suspected cases of tuberculosis (1, 2), mainly affecting the ileum, jejunum, large bowel and appendix. It was concluded that amebiasis and intestinal tuberculosis are more common in the rural population and underdeveloped countries; a fact largely due to poor health awareness and consequently poor hospital attendance. In reviewing the literature, we were unable to find a previous report of fulminant amebic colitis with perforation combined with intestinal tuberculosis. We here present a case of fulminant necrotizing amebic colitis with intestinal tuberculosis, which was diagnosed during laparotomy performed for acute abdomen, and note that it may readily be confused with other types of inflammatory bowel disease, such as idiopathic ulcerative colitis, Crohn's disease, perforated diverticulitis and appendicitis with perforation.

CASE REPORT

A 49-year-old vagabond, homeless man with poor hygiene and in malnutrition state had a sudden onset of diffuse abdominal pain, mucoid diarrhea, anorexia, fever and vomiting. One week before admission, he suffered a spell of mucoid diarrhea with dull abdominal pain. Temperature was 38.9℃, blood pressure was 100/50 mmHg, and pulse was 120 beats/min. There was diffuse guarding and tenderness, especially at the right and left lower quadrants, and there was a rebound tenderness. No mass was palpable and bowel sounds were diminished. Hematocrit was 32.4 percent and white blood cell count was 12,900/μL with a marked predominance of polymorphonuclear leukocytes. Serum electrolytes levels were sodium 124 mEq/L, chlorides 98 mEq/L and potassium 4.1 mEq/L. The prothrombin time and activated partial thromboplastin time were markedly prolongated. Upon x-ray examination, gas accumulation and fluid levels in the free abdominal cavity were found. These findings suggested diverticular perforation.

An emergency operation was done and there were copious feculent turbid ascites fluid, estimated to be about 2,500 mL, was found in the peritoneal cavity, especially in the ileocecal region and pelvic cavity. A transverse perforation of irregular margin about 3 cm in diameter in the anterior wall of the cecum was found. Another small perforation was seen in the rectosigmoid junction. The colonic walls were often attenuated and

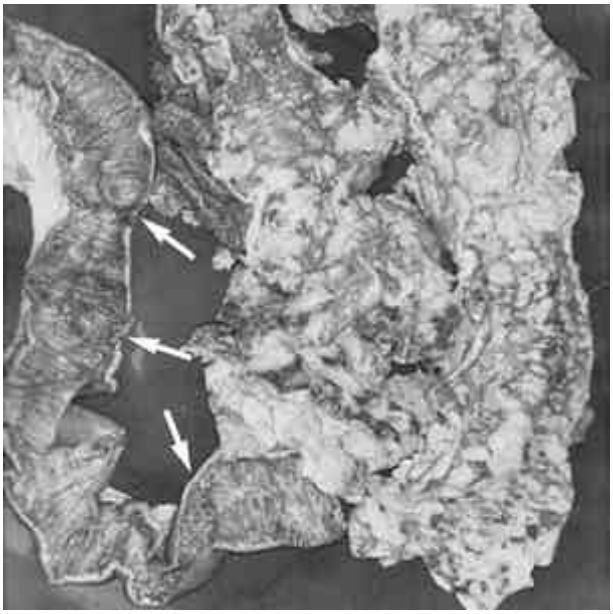

Fig. 1. The terminal ileum shows multiple circumferential ulcers (arrow). The mucosae of entire colon shows serpiginous and irregular ulcers covered by yellowish-green pseudomenbranes. Note the multiple perforations in the cecum and sigmoid colon.

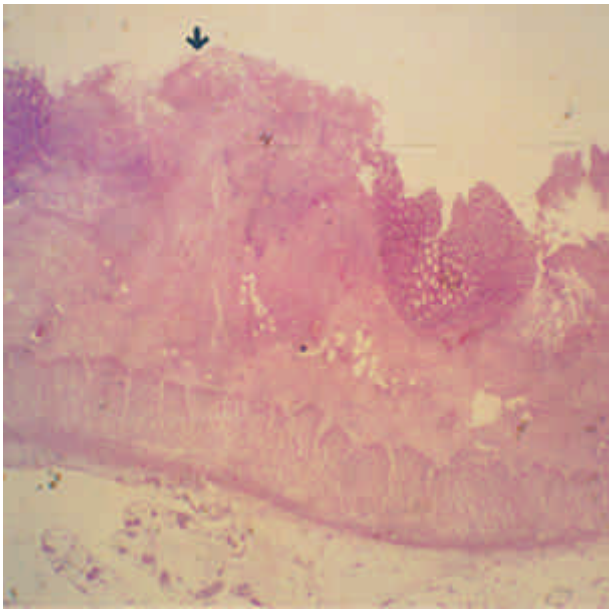

Fig. 2. Characteristic mucosal ulceration of amebic colitis with granulation tissue at the base (arrow) and overlying necrotic debris (H&E stain, ×20).

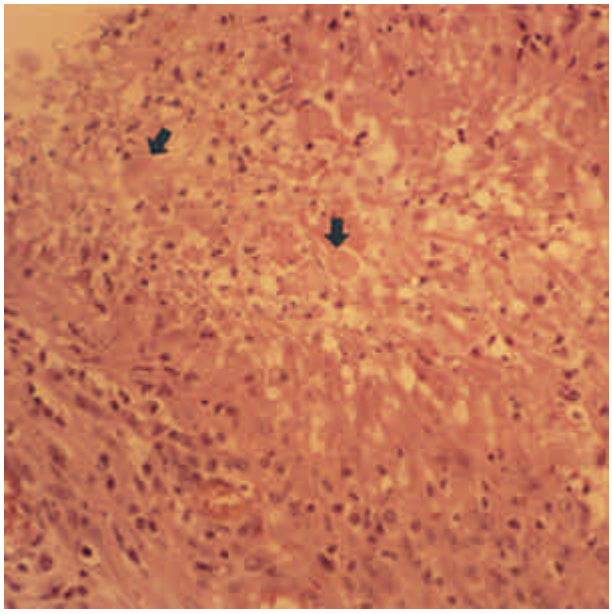

Fig. 3. Trophozoites of *Entameba histolytica* (arrows) were seen within the inflammatory exudates of the pericolic fat of the perforated site (H&E stain, ×200).

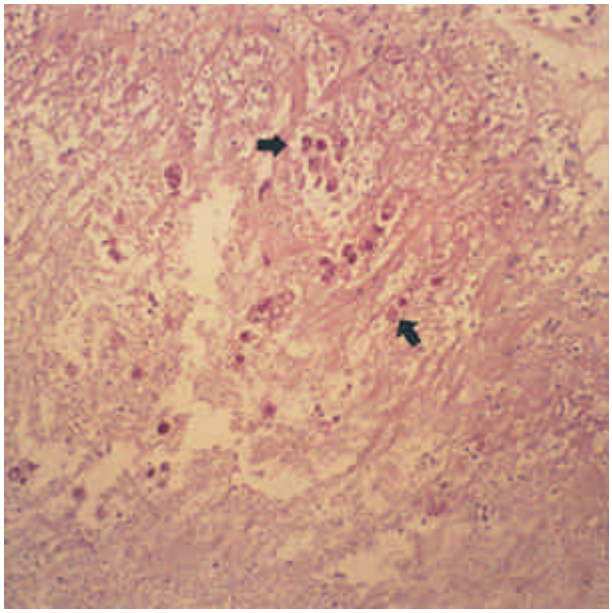

Fig. 4. Trophozoites of *Entameba histolytica* (arrows) are strong positivity for PAS stain (PAS stain, ×100).

extremely friable, having the consistency of 'wet blotting paper'. A subtotal colectomy with resection of ileum and ileostomy was carried out. Colonic mucosae showed serpiginous and irregular ulcer covered by yellow-green pseudomembranes (Fig. 1). The intervening mucosae were hyperemic. The ileum had multiple circumferential ulcers and some enlarged lymph nodes. The lymph nodes showed necrotic cheese-like material in the center. In spite of the above procedure, the patient was died on the sixth postoperative day due to sepsis. Histopathologic examination showed acute to subacute transmural necrotizing inflammation in the colon. The overlying pseudomembrane was composed of granular necrotic debris. Flask-shaped ulcers were expanded through out muscu-

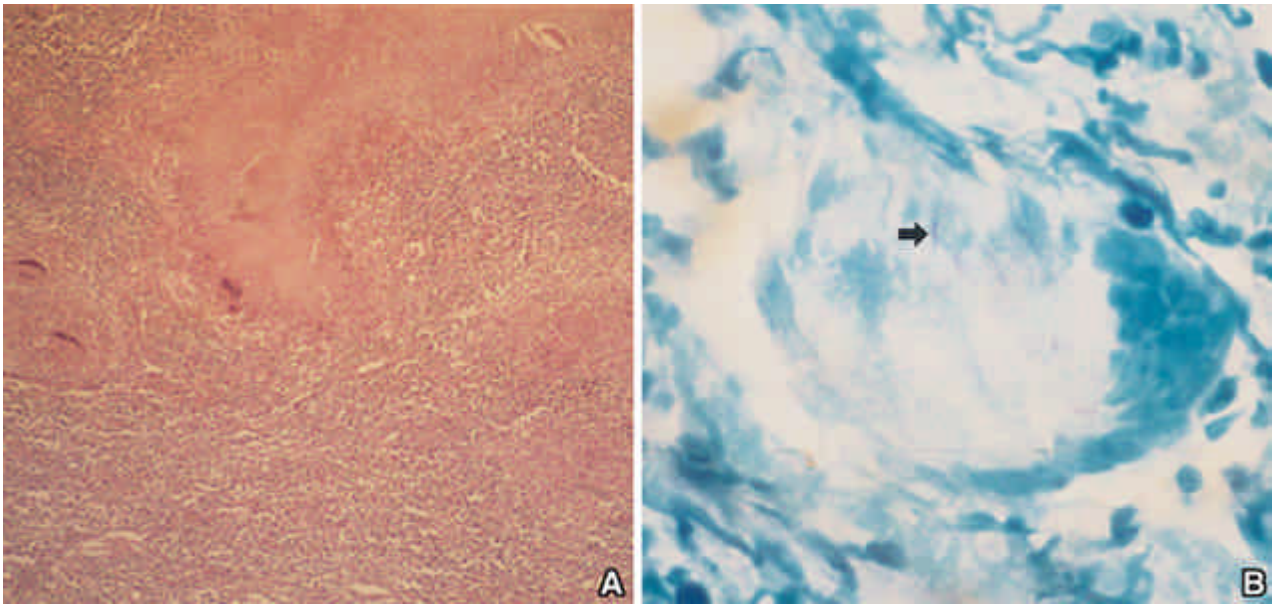

Fig. 5. **A:** It shows tuberculosis including caseating granulomas with multinucleated giant cells (H&E stain, ×40). **B:** Ziehl-Neelson stain showing several acid-fast bacilli (arrow) (×1,000).

laris propria, which was covered by purulent exudates (Fig. 2). Perforated lesions, trophozoites, were seen within the accompanying inflammatory exudates of the pericolic fat (Fig. 3). Intervening mucosa was often ischemic, and lamina propria showed marked lymphoplasmacytic infiltrates. It showed active inflammation with cryptitis and crypt abscess. However, no significant alteration of the crypt architecture was present. Periodic acid-Schiff stain for amebic trophozoites revealed strong positivity (Fig. 4). The terminal ileum and lymph nodes revealed chronic granulomatous inflammation with caseous necrosis and multinucleated giant cells (Fig. 5). The Ziehl-Neelsen stain for tuberculosis was positive.

DISCUSSION

Similar to other infectious diseases, amebiasis and tuberculosis in the worldwide distribution are not confined to endemic areas. We can attribute the recent spread of these diseases to at least four factors: the acquired immunodeficiency syndrome epidemic, immigration from high-risk areas, homosexuals and drug users and inadequate funding for these diseases and for other public health efforts (3, 4).

In the majority of amebiasis, the healing powers of the host balance the destructive effect of the parasite, and a state of equilibrium is achived (5). When the resistance of the host is lowered due to poor general condition or lower immunity, the disease will become acute and fulminant (6). Also in some people who have tuberculosis infection, the immune system cannot keep the tubercule bacilli under control, and the bacilli begin to multiply rapidly causing disease. Because tuberculosis is relatively common among Koreans, the probability of coexistence of tuberculosis and amebic colitis is remote. Only a very few isolated cases have been reported thus far (7, 8). It is unclear whether this is the result of underreporting or of patients with tuberculosis being protected from amebiasis by the frequent courses of antibiotics that are prescribed as part of the management of tuberculosis. The role of intestinal tuberculosis in the pathogenesis of fulminant amebic colitis is not known.

Amebic colitis is mild in most patients, but occasionally it is severe or even fulminant. Fulminant or necrotizing amebic colitis is an unusual form characterized by severe toxemia, rapid and extensive colonic necrosis, multiple perforations and is almost invariably fatal (9, 10). The rate of death from acute amebic colitis is estimated to be about 3%, and nearly all caused by the fulminating form of the disease. The incidence of perforation in amebiasis is less than 3%, but Chen et al. (11) have reported a higher rate of 6% in Taiwan. When the amoebae invade mucosal and submucosal tissues of the colon, they form typical flask-shaped ulcers. If the amebic infection is fulminating, a cavity containing necrotic material and abundant *E. histolytica* trophozoites develops beneath the ulcer. Such cavities, or submucosal abscesses may join together and destroy muscle layers, and further outward invasion of tissue results in perforation of the gut and lead to peritonitis (12). This invasion of tissues depends on the virulence of the ameboma and host resistance. The

role of these parasites in the pathogenesis of fulminant amebic colitis is not known. Various precipitating factors for perforation have been mentioned (13, 14); eq. malnutrition, chronic illness, parturition, external trauma, surgery and intestinal infections such as in our case.

Colon perforation due to amebiasis presents a bizarre and complicated clinical picture so that diagnosis remains a problem. Clinical pictures of this complication may exactly simulate acute abdominal conditions of other causes because of abdominal pain, muscle spasm and decreased intestinal activity. In our case, as in many others, surgery was undertaken because of a worsening clinical condition with an acute abdomen. The diagnosis of perforating amebic colitis was made postoperatively after pathologic analysis of the resected colon. Site of perforation in our case was seen at the cecum and sigmoid colon. The cecum, ascending colon and rectosigmoid areas are common sites of invasion and ulceration but the entire colon may be involved. In a majority of cases, perforations are multiple and may be seen clearly when the abdomen is opened. Cases with perforation are easily misdiagnosed as perforated appendicitis, diverticulitis or complicated inflammatory bowel diseases (15, 16).

The type of surgical procedure required in fulminant amebic colitis depends on the extent of colonic injury found at surgery and general status of the patient. Because of high risk of suture breakdown in tissues containing amoebae, resection and anastomosis should be avoided. Azar et al. (17) found gangrenous areas of colon both proximal and distal to anastomotic sites in a necropsy study. Several surgeons (18-20) recommend diverting and exteriorizing procedures if excision is carried out. They feel that there is no need for total colectomy, and hence only limited resection should be done as amebic colitis is a reversible condition and the colon can be used subsequently for restoring bowel continuity. In a few cases, the whole colon is so extensively necrotic that total colectomy seems the only satisfactory procedure (21). However the outcome in such case is likely to be fatal owing to toxemia and/or other combined intestinal infection as in our case.

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