Fulminant Amebic Colitis in an HIV-infected Homosexual Man

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Abstract

We present a case of fulminant amebic colitis in a human immunodeficiency virus (HIV)-infected homosexual man. The patient developed colonic perforation over a short time despite empirical therapy with metronidazole, and underwent right hemicolectomy. Amebic colitis was pathologically diagnosed by identifying invasive trophozoites of Entamoeba in a surgical specimen. Amebic colitis is one of the important differential diagnoses of acute abdomen in HIV-infected patients and/or homosexual men, especially in East Asia. Although fulminant amebic colitis is a rare manifestation of amebiasis, early diagnosis and treatment are thought to be important to improve the outcome of this highly fatal complication.

Key words: fulminant amebic colitis, colonic perforation, HIV infection, homosexual man


Introduction

Amebiasis is caused by the protozoan parasite Entamoeba histolytica. This disease is endemic to South Asia, Africa, and Central and South America (1), where poor sanitary conditions could be associated with the infection. In developed countries, most cases of amebiasis are reported in returning travelers and immigrants from endemic areas (2). Homosexual behavior is another important risk factor for amebiasis as well as human immunodeficiency virus (HIV) infection. Although amebiasis in homosexual men in the United States is predominantly caused by asymptomatic E. dispar infection rather than by E. histolytica infection (3), E. histolytica infection is more frequently seen in homosexual men in East Asian countries such as Taiwan, Korea, and Japan (4-8). It was reported that 13.4-20.4% of Japanese homosexual men test positive for amebic antibodies, according to enzyme-linked immunosorbent assay (ELISA); this is surprising considering that the hygiene status of the country is rather high (9).

Generally, more than 80% of E. histolytica infections are asymptomatic (10). However, in some cases of E. histolytica infection, the infected person develops amebic colitis, which is defined as amebic diarrhea with discharge of mucus or blood. Fulminant amebic colitis, which presents with a more aggressive clinical course including colonic perforation and necrotizing colitis, is a rare condition occurring in 0.5% of amebiasis cases, with a mortality rate of more than 50% (3, 11). We describe a case of amebic colitis in an HIV-infected homosexual man who developed colonic perforation, and review previous reports of fulminant amebic colitis.

Case Report

A 39-year-old man was admitted to our hospital because of severe diarrhea, abdominal pain, and acute fever. Two weeks before admission, the patient began to lose his appetite. Ten days later, watery diarrhea developed; the patient passed watery stools more than 10 times a day, which was accompanied by right abdominal pain, nausea, vomiting, and acute fever. The patient consulted a primary care physician because of the persistent symptoms and was then referred to this hospital and admitted. He had no remarkable medical history. He had had homosexual intercourse with many men. Until 2 years previously he had traveled to Singapore, Indonesia, and Thailand several times a year.

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The HIV viral load was 1.8×10^5 copies/mL, and the CD4 cell count was 72/mL. HBV-DNA load was 8.6 LGE/mL by ELISA, which indicates invasive disease of E. histolytica; however, repeated stool examinations did not show the presence of amoeba. Cultures of the peritoneal fluid revealed scant Enterococcus faecium and no acid-fast bacilli. Administration of metronidazole at 2,250 mg/day was resumed in the immediate postoperative period and continued for 10 days after the operation; vancomycin 1.5 g/day was added later. On the ninth postoperative day, initial colonoscopic examination was performed and showed ulcerated, aphthoid erosive mucosa, with friable and bleeding mucosa in the rectum.

Microscopic examination of the surgical specimen revealed multiple erosion sites or ulcers in the mucosal surface, and almost all of the epithelial tissue around the perforation sites was defective. Granulation, necrosis, and multiple abscesses were observed in the submucosa, and marked neutrophil and eosinophil infiltration was noted in the entire thickness of the bowel. Immunohistochemistry showed ghosts of Entamoeba trophozoites in the necrotic tissue (Fig. 2B), on the basis of which amebic colitis was diagnosed.

The patient showed slow improvement in bowel movement, with discharge of semisolid stools from stoma. Abdominal pain improved, and his temperature gradually returned to normal in the postoperative course. The patient’s general appearance improved, and he was discharged on the 26th postoperative day. After discharge, treatment for HIV was stopped; pulse rate, 118 beats per minute; respiratory rate, 18 breaths per minute; and blood pressure, 110/69 mmHg. The patient was uncomfortable and was under acute distress. There was moderate tenderness on the entire right side of the abdomen without rebound or guarding; the remainder of the physical examination revealed no abnormalities. The white cell count was 12,380/mm³, with 87% neutrophils. The total protein level was 7.4 g/dL; albumin level, 2.5 g/dL; creatinine level, 1.8 mg/dL; aspartate aminotransferase level, 68 U/L; alanine aminotransferase level, 90 U/L; and potassium level, 2.6 mEq/L. The levels of bilirubin, alkaline phosphatase, urea nitrogen, and sodium were normal. The patient tested positive for serum anti-amebic antibody by ELISA, which indicates invasive disease of E. histolytica; however, repeated stool examinations did not show the presence of amoeba. Cultures of the peritoneal fluid revealed scant Enterococcus faecium and no acid-fast bacilli. Administration of metronidazole at 2,250 mg/day was resumed in the immediate postoperative period and continued for 10 days after the operation; vancomycin 1.5 g/day was added later. On the ninth postoperative day, initial colonoscopic examination was performed and showed ulcerated, aphthoid erosive mucosa, with friable and bleeding mucosa in the rectum.

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Fulminant amebic colitis is a rare complication of amebiasis and is associated with high mortality rates. A retrospective analysis of 55 patients with fulminant amebic colitis diagnosed by autopsies and surgical operations in Mexico showed that it had a mortality rate of 89% (12). In Colombia, a study of 50 cases of fulminant amebic colitis, between 1971 and 1989, showed perforation in more than 70% of cases and a mortality rate of 60% (13). Ozdogan et al reported a case series of 8 patients with amebic colitis perforation in Turkey; their survival rate after colectomy was 50% (14). These studies were reported in endemic regions, and the cases were not complicated with HIV infection. In contrast, Mitarai et al reported 6 cases of amebiasis in HIV-infected Japanese men, 5 of whom were confirmed to be homosexual (7); 2 of these 5 patients died of colonic perforation soon after admission. In the United States of America, Saltzberg and Hall-Craggs reported a case of fulminant amebic colitis resulting in colonic perforation and death in a young homosexual man in the pre-HIV endemic era (15). In each report, delay in the diagnosis of amebiasis was associated with poor prognosis, because it is challenging to establish a prompt diagnosis in most situations. Moreover, high mortality after bowel resection in amebic colitis results from difficulties in placing the sutures due to the vulnerability of the intestinal wall (13).

In fulminant amebic colitis, the symptoms may be abrupt at the time of onset and progress rapidly. The present case was of an HIV-infected patient with acute abdomen, who was consequently diagnosed with fulminant amebic colitis after emergency surgery. Apparent abdominal symptoms developed only 4 days before admission, which was a somewhat early onset; patients with amebic colitis typically show a gradual onset of symptoms over several weeks, unlike patients with bacterial dysentery (3, 10). Although we started empirical therapy with metronidazole against supposed amebic colitis immediately after admission, colonic perforation developed 5 days after the initiation of treatment. Early diagnosis and treatment, including surgical intervention, are important for successful outcome of treatment for this condition.

It is unclear whether the severity of amebiasis is associated with cell-mediated immunity disorders in HIV-infected patients. In HIV-infected patients, asymptomatic intestinal colonization shows an increase, while invasive amebiasis is less common (16). A study of 203 HIV-infected patients in Mexico City showed a prevalence of *E. histolytica* and/or *E. dispar* infection of 34.1%, but none of them developed a clinical disease during the study period (17). A study on an animal model showed that depletion of CD4+ cells diminished parasite burden and inflammation, and decreased the severity of amebic colitis (18). However, the findings in the present case indicate that HIV-infected patients with low CD4+ cell counts could also develop even fulminant forms of amebiasis, the mechanism of which is unclear.

In conclusion, amebic colitis is one of the significant differential diagnoses of acute abdomen in HIV-infected patients and/or homosexual men, especially in East Asia. Although fulminant amebic colitis with perforation is a rare condition with a high mortality rate, early diagnosis and treatment might improve the outcome of the disease. Empirical therapy for amebiasis should be considered in such high-risk patients.

**The authors state that they have no Conflict of Interest (COI).**

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**Figure 2.** Macroscopical and histopathological findings of the specimen. The surgical specimen consisted of 5 cm of terminal ileum and 16 cm of ascending colon (panel A) showing 2 sites of perforation, sized 35×10 mm (arrow) and 35×30 mm (arrowhead). Fissures can be seen in the ascending colon, caused by the operative procedure. Immunohistochemistry showing ghosts of *Entamoeba* trophozoites in the necrotic tissue (Panel B, arrow).
References