

CLINICAL STUDY

Amebic perforation of small bowel: an unexpected localization of a fatal complication

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Abstract: The intestinal protozoan parasite *E. histolytica* is the causative organism responsible for human amebiasis and amebic dysentery. Although it is primarily an infection of the colon, it may also be spread by hematogenous path to other organs, especially the liver. In general, the clinical spectrum of colorectal amebiasis ranges from the state of asymptomatic carrier to severe fulminant necrotizing colitis with bleeding and perforation. Here we present an extremely rare case of necrotizing amebiasis of small bowel with a fatal outcome (Fig. 1, Ref. 4). Full Text (Free, PDF) www.bmj.sk.

Key words: amebiasis, necrosis, perforation, small intestine.

Amebiasis is a parasitic disease caused by a protozoan *Entamoeba histolytica*. Although it is primarily an infection of the colon, it may also be spread by hematogenous path to other organs, especially the liver. In general, the clinical spectrum of colorectal amebiasis ranges from the state of asymptomatic carrier to severe fulminant necrotizing colitis with bleeding and perforation. Invasive amebiasis is the second most common cause of mortality due to parasite infections worldwide (1).

Here we present an extremely rare case of necrotizing amebiasis of small bowel with a fatal outcome.

Case report

A 50-year old man was admitted to our emergency department with a chief complaint of abdominal pain and vomiting. He had a seven-day history of hospitalization in another medical center due to diarrhea. One day after being discharged from the latter medical center, he developed sharp abdominal pain and became worse. His past medical history was not significant and contained no record of abdominal surgery.

Clinical examination revealed a temperature of 39 °C, with general peritonism and abdominal distention. The white blood cell count was $1.8 \times 10^3/\mu\text{l}$ with 92.8 % neutrophils. Hemoglobin was 12.6 g/dl and the platelet count was 91000/ μl . Electrolytes were normal. Liver function markers were slightly high, namely lactate dehydrogenase and creatin phosphokinase. Prothrombin

time was 17.6 sec with an INR of 1.3. The abdominal plain roentgenogram revealed air-fluid levels in small bowel. Upon the detection of free fluid in the abdominal cavity by means of abdominal ultrasonographic examination, an emergency operation was planned with a preoperative diagnose of perforation.

During the operation, nearly 1500 cc of fluid of intestinal content was aspirated from the abdominal cavity and we observed an ulcer perforation of 0.5 cm at the anti-mesenteric side of small bowel located 200 cm from Treitz ligament. No other pathology was observed. After peritoneal lavage with 5000 cc of saline, a wedge resection and a Bogotá bag procedure were performed.

Seftriaxon treatment was prescribed due to the clinical suspicion of *Salmonella typhi* and daily peritoneal lavage was performed after the surgery. The patient was followed up at the intensive care unit during this period and fed only by parenteral nutrition. The pathological examination of the specimen revealed intestinal necrotizing amebiasis (Fig. 1A). Thus, metronidazole was added to the treatment.

On the 7th postoperative day, diffusion of necrosis was observed. Thus, a 100-cm segment at 20 cm distal of Treitz ligament and a 60-cm segment at 40cm distal of it (160 cm distal of Treitz ligament) were resected. End-to-end anastomoses were performed at both sides. A Bogotá bag procedure was performed after 5000 cc peritoneal lavage. The specimen had the signs of intestinal necrosis (Fig. 1B).

During the postoperative follow-up of the second operation, blood culture, which was taken during a high fever attack, revealed the presence of *Candida albicans*. Thus, intravenous fluconazole treatment was prescribed. On the 4th postoperative day, an anastomotic leakage was detected. Necrosis was observed at the distal anastomotic site and a 10-cm segment was resected at 40 cm distal of Treitz ligament with addition of a jejunostomy procedure.

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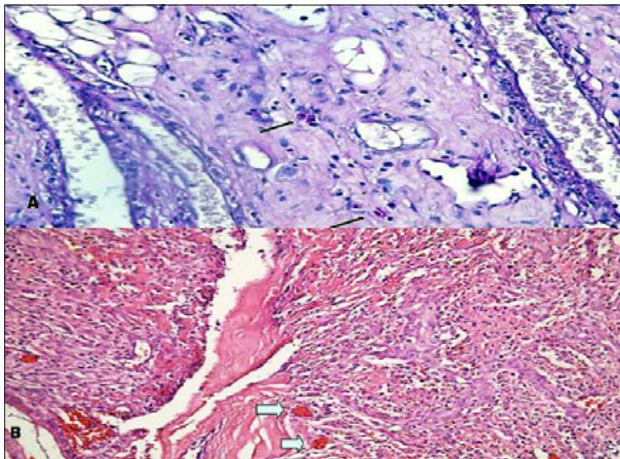


Fig. 1. A – Trofozoid of *Entamoeba histolytica* can be seen in the intestinal wall tissue. B – Mucosal necrotic areas can be seen at specimen of the second operation.

During the postoperative period of third operation; fungal pneumonia and toxic hepatitis were detected. His bilirubin levels and liver functions were progressively increased. Proper medications were prescribed. He was entubated due to low PO₂ saturation on the 3rd postoperative day. It was agreed that *Candida* sepsis and intrahepatic cholestasis due to sepsis were involved. On 7th postoperative day, conjunctival hemorrhages were detected in the right eye of the patient due to *Candida* infection, and ophthalmic polymyxin-B was prescribed. The patient died on the 32nd postoperative day after the third operation due to multiorgan failure.

Discussion

The intestinal protozoan parasite *E. histolytica* is the causative organism responsible for human amebiasis and amebic dysentery. Of epidemic proportions, it afflicts millions of people worldwide in developing countries. Transmission is mostly by ingestion of contaminated food and water; however, venereal transmission *via* the fecal-oral route can also occur. Trophozoites are responsible for invasive disease and may lead to colonic mucosal ulceration. The gastrointestinal tract and liver are the two main organ systems affected by the parasite. It is very rare

that patients with long-standing infection develop ulcerative exophytic inflammatory masses that are indistinguishable from carcinomas and can gain considerable size, reportedly up to 15 cm in diameter (2).

Santos et al suggested Multiplex-PCR as a tool for the confirmation of microscopic results when *E. histolytica* falls under suspicion (3). Unfortunately, both molecular and antigen testing are significantly more expensive than microscopy. Thus, our case was diagnosed by microscopy.

Invasive amebiasis is the second most common cause of mortality due to parasite infections worldwide, accounting for 40.000 to 100.000 deaths annually. High risk populations for developing invasive amebiasis include infants, pregnant women, and patients who are taking immunosuppressives (1). Our patient was a 50-year old man with no history of immunosuppressive or any other drug usage or additional disorder.

We performed Bogotá bag procedure to achieve a suitable abdominal toilet by daily lavage. On this account, we also decided to perform two end-to-end anastomoses at second operation. We think that the *Candida* sepsis was related to the antibacterial therapy. Also, the nosocomial candidemia that can be seen in intensive-care-unit patients might be another cause. Bouqnoux et al reported that the mean interval between ICU admission and candidemia was 19.0±2.9 days⁴. The ICU mortality in their candidemic patients was 61.8 %. We detected candidemia on the 10th day of our patient's hospitalization at intensive care unit and he died 31 days after our detection.

References

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