

Fulminant necrotising amoebic colitis: a report of two cases

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Hong Kong Med J 2022;28:172–4

<https://doi.org/10.12809/hkmj209107>

Case reports

Case 1

In January 2019, a 31-year-old man with a history of amphetamine abuse presented with a 10-day history of watery diarrhoea and abdominal pain. On admission, he had a fever of 38.1°C and tachycardia but normal blood pressure. Abdominal examination revealed tenderness and guarding over the lower abdomen. The patient was resuscitated and intravenous antibiotics prescribed since an infective cause was considered most likely. Abdominal plain radiograph showed grossly dilated small and especially large bowel (diameter up to 9 cm). White cell count was $22 \times 10^9/L$ (neutrophil differential count $19 \times 10^9/L$), liver and renal function were unremarkable. Contrast computed tomography (CT) scan of the abdomen and pelvis revealed extensive colitis, suggesting pseudomembranous colitis or diffuse colitis. Stool culture for *Clostridium difficile* was negative and stool microscopy revealed no ova or cysts. Human immunodeficiency virus (HIV), hepatitis B and C virus serologies were non-reactive. He was treated as pseudomembranous colitis by the medical gastrointestinal team and commenced on oral vancomycin. Later sigmoidoscopy revealed inflamed mucosa with multiple ulcers, especially at the sigmoid colon (Fig 1). Biopsies were taken. The patient's condition deteriorated and he developed septic shock. A new contrast CT scan showed pneumoperitoneum. Emergency laparotomy was

performed and revealed generalised faecal peritonitis with the whole colon necrosed and communicating with the peritoneal cavity. Debridement of necrotic tissue and multiple segmental resections of bowel with end ileostomy were performed in stages. The diagnosis of fulminant amoebic colitis (FAC) was made on histopathological evaluation of the biopsy and resection specimen (Fig 2). Antibiotics were switched to metronidazole accordingly. The patient's condition was later complicated by an intra-abdominal fluid collection and image guided drainage was performed. He was initially nursed in the intensive care unit and then transferred to a surgical ward for rehabilitation. He was discharged from hospital 4 months later.

Case 2

In February 2020, a 61-year-old man presented with a ≥ 1 -week history of diarrhoea, abdominal pain, high fever of 39°C and tachycardia with normal blood pressure. Abdominal examination showed diffuse tenderness, but without peritoneal signs. The patient's medical history was otherwise good, and initial investigations including blood tests and plain radiographs were all unremarkable. In view of the progressive abdominal distension, urgent contrast CT was arranged and showed a suspected perforated caecum. Emergency surgery found ischaemic colon extending from the caecum to mid sigmoid, with perforations over the proximal



FIG 1. Endoscopy photos showing severely inflamed mucosa in Case 1

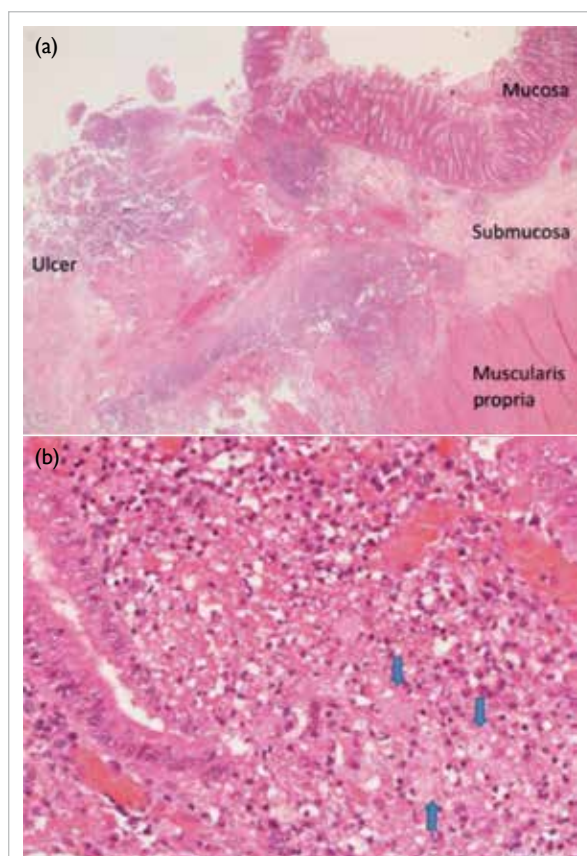


FIG 2. Histological examinations from Case 1. (a) Perforated ulcer. Haematoxylin and eosin (H&E) stain $\times 100$. (b) Amoebae labelled with arrows in the lamina propria. H&E stain $\times 100$

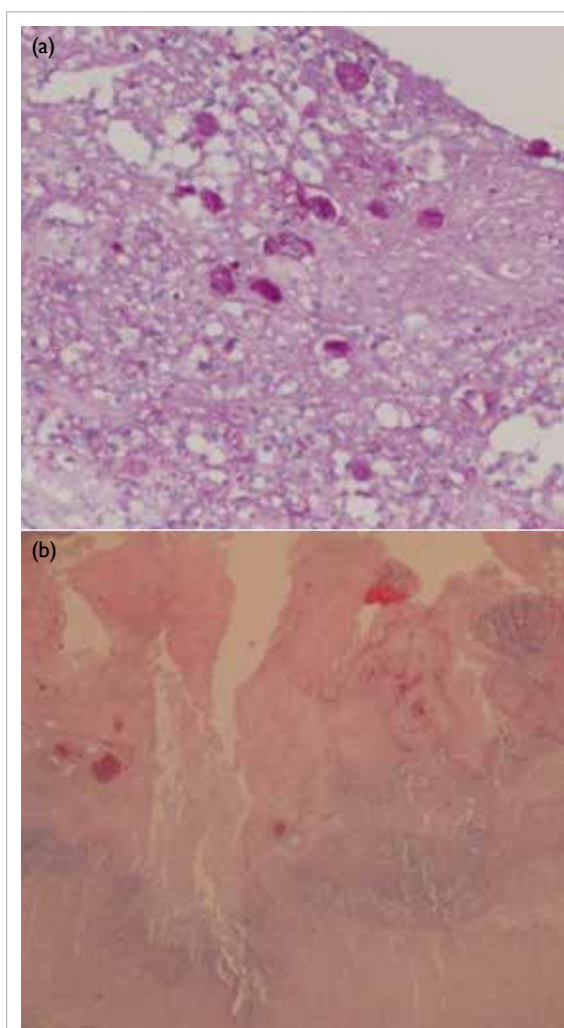


FIG 3. Histological examinations from Case 2. (a) Presence of *Entamoeba histolytica*, stained purple due to glycogen content, in necrotic ulcer debris. Periodic acid-Schiff $\times 200$. (b) Deep penetrating ulcer, with extensive necrosis, down to the muscularis propria. H&E stain $\times 10$

transverse colon and hepatic flexure. Subtotal colectomy with end ileostomy was performed. He was transferred to the intensive care unit after surgery and required vasopressor and continuous venovenous haemofiltration due to severe sepsis. He showed a good response and was weaned off vasopressor support on postoperative day 4. Pathology of the surgical specimen later confirmed amoebic colitis with extensive ulcer and perforation (Fig 3). There were no features of atherosclerosis, vasculitis, thromboembolism, or inflammatory bowel disease. Microscopic results were also available after surgery. Stool culture for *C difficile*, stool microscopy for amoeba, stool microscopy for ova or cysts, stool polymerase chain reaction for virus, and HIV serology test results were all negative, but *Entamoeba histolytica* serology test was positive. He was prescribed metronidazole for 14 days and then oral diloxanide furoate for 10 days as suggested by the microbiologist. The patient's recovery was later complicated by wound dehiscence and abdominal cocoon. Repeat surgery for debridement was performed and skin closure changed to an ABThera temporary abdominal closure system. The patient recovered gradually and was transferred to a rehabilitation unit 3 months after surgery.

Discussion

Entamoeba histolytica is a protozoan parasite and the cause of amoebiasis in humans.¹ Early diagnosis and treatment are essential to avoid progression to fulminant colitis. Amoebic dysentery is classified as a notifiable disease in Hong Kong. Prevalence of amoebiasis is higher in developing countries such as India, Mexico, and parts of Central and South America,² mainly related to poor socioeconomic and sanitary conditions. In developed countries, where faecal-oral transmission is unusual, amoebiasis is more often seen in immigrants from or individuals with a travel history to endemic areas. Other groups at risk include male homosexuals³ with or without HIV, infants, pregnant women, and those taking immunosuppressants, especially corticosteroids.¹ Neither of our two cases had a relevant travel history in the past year, and they presented several months apart, so they are unlikely to be imported cases or

to have had the same source of infection. Case 1 had some risk factors for amoebiasis: he was a male homosexual living in rental housing with shared rooms. A detailed history taking from patients who present with severe diarrhoea is crucial. Prompt initiation of anti-amoebic treatment should be considered in cases where there is a high index of suspicion.

Although *E histolytica* can be cultured in vitro, this is neither routinely performed nor is it a gold standard in the diagnosis of amoebic colitis because amoebic culture is insensitive (25%). The most commonly used laboratory test is stool microscopy to identify trophozoites or cysts, although this also has low sensitivity (<60%) and specificity (10%-50%). As exemplified by our two cases, both had negative stool microscopy. In some laboratories, stool antigen detection of *E histolytica* might be available. However, neither stool microscopy (in the absence of ingested erythrocytes in the trophozoites) nor some antigen detection kits can distinguish between pathogenic *E histolytica* and commensal *Entamoeba dispar*. Serum antibody is commonly positive in patients with invasive amoebiasis,⁴ especially in endemic areas. It may be difficult to differentiate past from active infection since antibodies may persist for some time. Recent commercially available multiplex polymerase chain reaction systems offer a rapid and sensitive way of detecting *E histolytica* DNA in stool; however, cost and availability limit their application in most patients.

Another popular non-invasive investigation is different modalities of imaging, especially contrast CT scan. Some CT features specific to amoebic colitis have been reported. These include extended submucosal ulcers with intramural dissection caused by flask-shaped ulcers typical of amoebiasis and omental “wrapping” indicating adhesions with neovascularisation due to ischaemic foci of transmural amoebic colitis.⁵ Other non-specific findings include pancolitis with areas of target signs, discontinuous bowel necrosis and coexistence of liver abscess. None of these features were evident on CT scans in our patients. In clinical practice, CT scan is not sensitive and has a small role in diagnosing FAC. It is mainly used to distinguish the severity of colitis, looking for complications such as bowel perforation or ischaemia.

Sigmoidoscopy and/or colonoscopy with biopsy can also be performed as a diagnostic tool. However, there is a high risk of perforation, especially of inflamed or even ischaemic bowel. We do not recommend endoscopic investigations in cases of severe colitis or in patients with high fever.

Preoperative diagnosis of FAC remains a challenge and detailed history taking plays an important role in identifying high-risk cases.

Mild cases of amoebic colitis can be treated medically with metronidazole to control systemic invasion and diloxanide furoate, a luminal agent, in addition to eliminating luminal cysts. If the condition

becomes transmural, conservative treatment is no longer appropriate. Early diagnosis and extensive surgical treatment are important to reduce morbidity and mortality.¹ In both our cases, with both complicated by bowel perforation, extensive bowel resection was immediately performed.

Intra-operatively, skipped lesions with multiple transmural perforations of bowel were noticed. However, some gross features make differentiation from other pathology difficult, especially Crohn’s disease. In our patients, necrotic tissue was more friable with little bleeding from the necrotic bowel wall. These features are quite unique compared with other causes of bowel ischaemia. This may be related to severe necrosis with consequent poor vascular supply to the bowel. In the worst case, the necrotic bowel wall communicates with the peritoneal cavity, making bowel resection more difficult as the dissection planes may be disrupted. Therefore, extensive debridement of necrotic tissue or even staged operations are required. Special gross features of FAC are seldom mentioned in other case reports. Early identification of these features enables early commencement of empirical anti-amoebic treatment and aids the recovery of patients.

Author contributions

Concept or design: LM Tam, KC Ng, CH Man.

Acquisition of data: LM Tam

Analysis or interpretation of data: LM Tam, FY Cheng, Y Gao.

Drafting of the manuscript: LM Tam.

Critical revision of the manuscript for important intellectual content: KC Ng, CH Man.

Conflicts of interest

The authors have no conflicts of interest to disclose.

Funding/support

This study received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics approval

The patients were treated in accordance with the Declaration of Helsinki. The patients provided verbal informed consent for the treatment/procedures and consent for publication.

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