

Case Report

Caught in the act... a case of fulminant amoebic colitis

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Introduction: Amoebic colitis is a common worldwide infection with a risk of serious complications, including invasive intestinal and extra-intestinal disease. Although newer tests are available, the mainstay of diagnosis remains stool microscopy. Early treatment with metronidazole followed by paromomycin generally results in a clinical cure.

Case presentation: We describe a case of fulminant colitis secondary to *Entamoeba histolytica* requiring bowel resection that went unrecognized until the operative histology was reviewed.

Conclusion: Clinicians worldwide need to be aware of this condition, particularly in patients originating from endemic areas, to ensure early diagnosis and appropriate treatment leading to optimal clinical outcomes.

Keywords: *Entamoeba histolytica*; fulminant amoebic colitis.

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Introduction

Entamoeba histolytica is a common, well-recognized pathogenic amoeba, associated most often with intestinal but also extra-intestinal infection. It is found worldwide but has a higher incidence in countries with inadequate infrastructure to limit faecal–oral transmission. Clinical disease can vary from asymptomatic colonization to invasive intestinal amoebiasis; dissemination most commonly occurs to the liver but can affect other body sites (Haque *et al.*, 2003). When diagnosed early, treatment of colonic disease is generally straightforward and the prognosis is good. Here, we report an encounter with a patient in whom diagnosis was delayed resulting in progression to fulminant colitis.

Case report

A 74-year-old previously healthy Indian man visiting family in Melbourne, Australia, presented to our emergency department with severe abdominal pain following 2 weeks of worsening dysentery. On arrival he was hypotensive, tachycardic and hypothermic with signs of peritonitis. Initial laboratory results were (reference range given in parentheses): haemoglobin 149 g l⁻¹ (119–160), white blood cell count 47 × 10⁹ l⁻¹ (4–11), creatinine 1418 μmol l⁻¹ (55–105), urea 58.7 mmol l⁻¹ (2.5–9.6), bilirubin 76 μmol l⁻¹ (0–20), albumin 14 g l⁻¹ (35–45), alkaline phosphatase 129 U l⁻¹ (30–120), alanine transaminase 41 U l⁻¹ (7–56), C-reactive protein 244 mg l⁻¹ (0–5) and

lactate 1.2 mmol l⁻¹ (0.5–2). Broad-spectrum antibiotics were commenced and an urgent laparotomy was performed on the suspicion of ischaemic bowel. A necrotic caecum and appendix with peritonitis were found intra-operatively; a right hemicolectomy and peritoneal lavage were performed.

Post-operatively the patient was admitted to intensive care and progressively improved. Renal and liver function returned to baseline over several days. Operative histology (Fig. 1) revealed changes consistent with extensive infection from *E. histolytica*. A 10-day course of oral metronidazole 600 mg every 8 h was administered, followed by paromomycin 500 mg every 8 h for 7 days. A liver ultrasound was performed to exclude extra-intestinal disease. Amoebic serology by indirect haemagglutination was strongly positive (titre 1:4096). Clinical improvement continued and he was discharged from hospital feeling well 2 weeks after his initial presentation. He subsequently returned to India.

Discussion

E. histolytica infection may be asymptomatic, cause dysentery or disseminate, most commonly to the liver. Humans are the primary known reservoir, and transmission occurs via the faecal–oral route (Stanley, 2003). There are several closely related *Entamoeba* spp. that are morphologically indistinguishable, with *Entamoeba dispar* traditionally thought to be non-pathogenic. Recent DNA evidence is

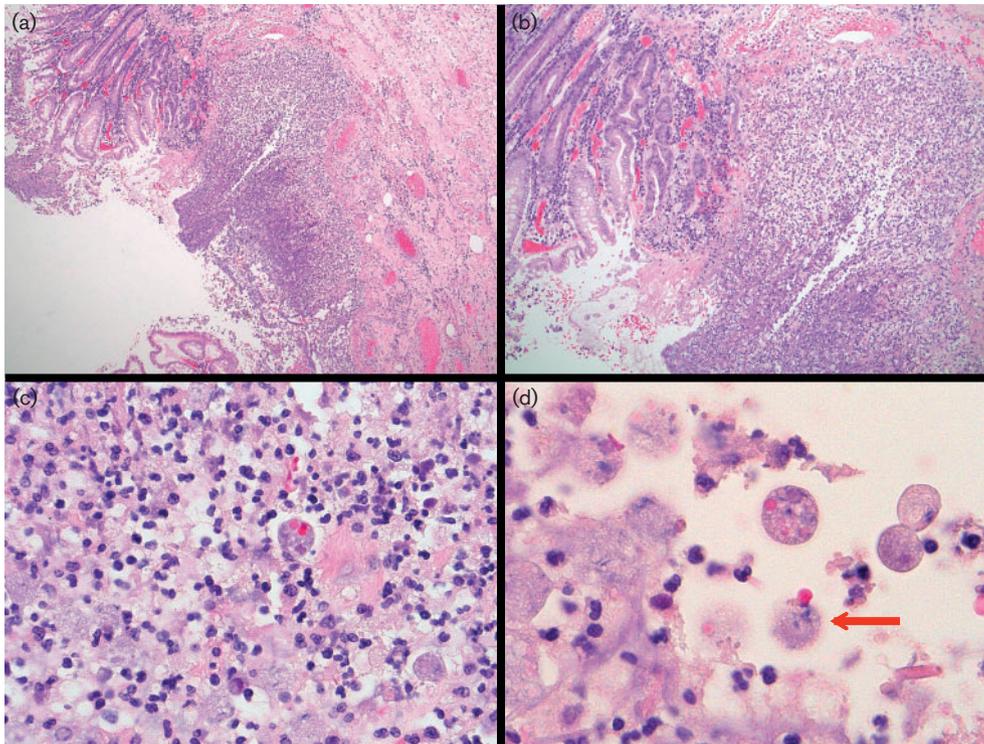


Fig. 1. (a, b) Haematoxylin and eosin stains of the caecum of the patient showing the classic flask-shaped colonic ulcer with adjacent normal colonic tissue. (c, d) Haematoxylin and eosin stained high-power images showing multiple trophozoites invading the colonic wall, including one that appears to be caught in the act of ingesting an erythrocyte (arrow in d). Magnification, $\times 40$ (a); $\times 100$ (b); $\times 400$ (c); $\times 600$ (d).

questioning this paradigm, however, with cases of liver abscess due to *E. dispar* described (Ximénez *et al.*, 2010).

Following ingestion of cysts, excystation occurs within the intestinal lumen and trophozoites develop. In the majority of cases, this results in self-limiting, asymptomatic infection. Colitis occurs when they penetrate the intestinal mucous layer, resulting in an inflammatory response (Haque *et al.*, 2003). Delayed diagnosis or failure to initiate prompt effective therapy can result in progression to severe fulminant colitis with a high morbidity and mortality (Gupta *et al.*, 2009). One recent case series (Ortiz-Castillo *et al.*, 2012) of 24 patients from an endemic area described a mortality rate of 50 %, with 20 patients requiring surgery within 24 h of presentation. An older study of 55 patients (Takahashi *et al.*, 1997) reported a co-existent liver abscess in 54 % of patients and had a higher mortality of 89 %.

Despite limitations, the mainstay of diagnosis worldwide remains stool microscopy. Antigen detection and PCR-based diagnostics are being increasingly utilized where available, and are more sensitive and specific. Serology is used mainly in extra-intestinal disease but does not reliably differentiate between current and past infection; more accurate enzyme immunoassay kits are commercially available and have largely replaced the indirect

haemagglutination test used by our laboratory (Tanyuksel & Petri, 2003; Santos *et al.*, 2013).

Treatment with metronidazole or tinidazole is generally curative but is active against the trophozoite stage only; paromomycin is required to prevent relapse by eliminating cyst carriage (Pritt & Clark, 2008). Broad-spectrum antibiotics are often indicated in fulminant disease where bacterial translocation across the damaged intestinal wall can occur, and therapeutic aspiration of a liver abscess is occasionally required as adjunctive therapy (Petri, 2003). Nitazoxanide is a possible treatment alternative; however, its role has not been well defined (Rossignol *et al.*, 2007).

In summary, we have described a patient with amoebic colitis who progressed to fulminant disease requiring urgent laparotomy and hemicolectomy. Our patient had seen a medical practitioner twice before presenting to our institution; no investigations had been performed and empiric amoxicillin had been prescribed. Even after his deterioration, the diagnosis of amoebic colitis had not been considered until operative histology was available, which was several days post-operatively. Despite this, fortunately he survived with minimal sequelae. Given increasing international migration and travel, clinicians worldwide need to maintain a high index of suspicion of this condition, particularly in patients

originating from endemic areas, to ensure early diagnosis and appropriate treatment to avoid complications.

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