

Invasive amoebiasis complicating inflammatory bowel disease

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Introduction

Amoebiasis, which is caused by the intestinal protozoan *Entamoeba histolytica*, is a ubiquitous parasitic infection affecting approximately 10% of the world's population and causing more deaths every year (100,000 deaths) than any other parasitic infection, with the exception of malaria and schistosomiasis [1–3]. Most individuals with an *E. histolytica* infection are asymptomatic, but some develop severe invasive disease, such as amoebic colitis. Other manifestations, such as pulmonary, cardiac or brain involvement, are rare. Intestinal amoebiasis can probably also present as a chronic, non-dysenteric syndrome of diarrhoea, weight loss, and abdominal pain that can last for years and mimic inflammatory bowel disease. Fulminant colitis with bowel necrosis leading to perforation and peritonitis occurs in only about 0.5% of cases, but it is associated with a mortality rate of more than 40%. Patients with invasive amoebiasis living in the United Kingdom and other developed countries generally acquire the infection in another country in which the pathogenic species is endemic. Areas that have high rates of amoebic infection include India, Africa, Mexico and parts of Central and South America. Infection with pathogenic *E. histolytica* is not a common cause of travelers' diarrhoea, and gastrointestinal infection is uncommon in travelers who have spent less than one month in endemic areas.

Despite the availability of sophisticated investigative procedures, differentiating invasive colonic amoebiasis from idiopathic inflammatory bowel disease (IBD) may be difficult. This case is presented to remind clinicians of the similarities in the clinical presentation and endoscopic features of these two conditions, and to highlight the difficulty in differentiating them.

Case report

We present the case of a 68-year-old woman who was diagnosed with ulcerative colitis (UC) in 1983. Since then she had frequent flare-ups and was repeatedly treated with courses of steroids. She was receiving 5-amino salicylic acid as a maintenance therapy. She presented to the clinic with a flare-up that coincided with her return from Thailand 22 days earlier. She complained of passing 7–8 loose motions per day and having generalised symptoms of malaise, low grade fever and tachycardia. With these symptoms and a C-reactive protein (CRP) of 55, she was hospitalised and placed on intravenous steroids. Stool samples were negative by both microscopy and culture.

Despite intravenous steroids, her diarrhoea worsened and became bloody. Sigmoidoscopy showed severe inflammation with cobblestone formation, bleeding, and friability of the mucosa from the rectum to beyond the sigmoid colon, which is not characteristic of UC. Therefore, multiple biopsies were taken from the rectum and sigmoid colon; the histology showed focal mucosal ulceration due to invasive amoebiasis, and *E. histolytica* parasites were evident (Fig. 2). Steroids were stopped and the patient was started on a course of metronidazole at 750 mg three times daily for 10 days, followed by oral diloxanide furoate (500 mg) three times a day for 10 days. She eventually recovered and was discharged on 5-amino salicylic acid. Since then she has remained in complete remission.

Discussion

Amoebic colitis is a disease with diverse clinical manifestations that frequently lead to confusing it with other types of colitis. When misdiagnosed as UC, undesirable outcomes might occur from the use of steroids, including colectomy or even death. Clinicians must keep amoebiasis in mind in the differential diagnosis of patients suspected of having UC or Crohn's disease [3]. A recent study in Turkey showed that amoeba infection in patients with IBD, especially those with UC, is more prevalent than in the normal population [4]. With more frequent air travel, amoebic colitis is an important "not to miss" diagnosis even in the developed world. However, it should be noted that amoebiasis can be contracted by people, especially children, who have never been abroad.

Stool microscopy is a relatively poor method for diagnosing intestinal amoebiasis because it is time-consuming, laborious, and requires specific expertise. Culture methods are often unrewarding, with a sensitivity of only about 50%. However, monoclonal antibodies that specifically recognise pathogenic *E. histolytica* strains but not nonpathogenic *E. dispar* in faeces and serum are commercially available. Antigen detection kits using enzyme-linked immunosorbent assay (ELISA), radioimmunoassay, or immunofluorescence have been developed. Serology is also useful for diagnosing amoebiasis. Infection with *E. histolytica* results in the development of antibodies, whereas *E. dispar* infection does not. Antibodies will usually be detectable within five to seven days of acute infection and may persist for years. Up to 25% of uninfected individuals in endemic areas have anti-

amoebic antibodies due to previous, often undiagnosed infection with *E. histolytica*.

Sigmoidoscopy and/or colonoscopy can be performed either to diagnose amoebiasis or to exclude other causes of the patients' symptoms. Microscopy may reveal cysts or trophozoites in scrapings or biopsies, which are best taken from the edge of ulcers. Colonic lesions in amoebic dysentery range from nonspecific mucosal thickening and inflammation to classic flask-shaped amoebic ulcers. However, colonoscopy is not recommended as a routine diagnostic approach because intestinal amoebic ulcerations increase the likelihood of perforation during instillation of air to expand the colon.

Asymptomatic carriers of *E. histolytica* should be treated with a luminal agent to minimise the spread of disease and the risk of developing invasive disease. Recommended drugs for treatment of symptomatic intestinal disease and for hepatic abscess are metronidazole and tinidazole. These drugs may not eliminate the intraluminal cysts, and so they should be immediately followed by treatment with iodoquinol, paromomycin, or diloxanide furoate.

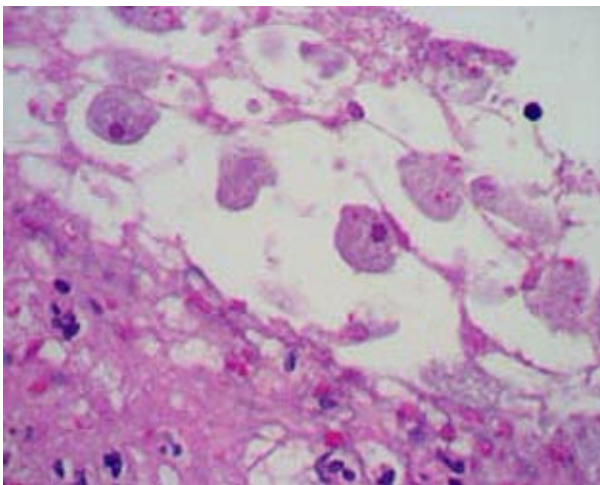


Figure 1: Numerous *E. histolytica* trophozoites at the base of ulcerated colonic tissue stained with hematoxylin-eosin.

Conclusion

E. histolytica continues to be an important, worldwide source of disease. It is the primary cause of amoebic dysentery in developing countries. Amoebic colitis needs to be kept in mind in the differential diagnosis of inflammatory bowel disease, particularly in high-risk individuals. Early diagnosis and treatment with metronidazole or tinidazole should be instituted once acute amoebic colitis is suspected. We believe that the remission of the case presented was due to treatment of the amoeba infection. We suggest that amoebiasis

should be sought and treated in every patient with ulcerative colitis.

References

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