

Not only coughs and sneezes

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Lancet 2008; 372: 1706

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In February, 2008, a 57-year-old man developed diarrhoea and abdominal pain, and saw his general practitioner. Stool samples contained no *Escherichia coli*, salmonella, shigella, or campylobacter; microscopy showed no ova, cysts, or parasites. Gastroenteritis was diagnosed. 5 weeks later, the patient came to our emergency department with a swollen abdomen, after 3 days of absolute constipation. He had no history of inflammatory bowel disease, and had not recently been abroad.

He looked dehydrated. He was clammy, sweating profusely, hypotensive, and tachycardic: we suspected sepsis. His abdomen was distended and diffusely tender, with guarding in the periumbilical region and right iliac fossa. Blood results revealed neutrophilia ($13 \cdot 2 \times 10^9$ cells per L), and a high concentration of C-reactive protein (245 mg/L). Abdominal radiography revealed several loops of dilated small bowel; chest radiography did not show free air under the diaphragm. CT showed dilatation of the small bowel, caecum, and ascending colon, and a localised perforation from the transverse colon into a fluid collection deep to the right rectus muscle. We did an emergency laparotomy, which confirmed our provisional diagnosis of faecal peritonitis, caused by perforations of the transverse colon. We did an extended

right hemicolectomy (figure), protecting the anastomosis with a loop ileostomy. Histopathological examination of excised bowel showed extensive necrosis of the wall, resulting in countless perforations. Within the necrotic tissue, the histopathologist saw many amoebic trophozoites. We gave the patient intravenous metronidazole, and oral diloxanide furoate to clear amoebic cysts. On further questioning, the patient revealed that he had recently had oral-anal sexual contact with a man born in Bangladesh. HIV serology was negative. The patient recovered well from the operation, and was discharged 10 days later. When the patient was last seen, colonoscopy showed a normal-looking bowel; reversal of the ileostomy is planned.

Amoebiasis is a parasitic infection, caused by *Entamoeba histolytica*. It kills up to 100 000 people a year.¹ Its prevalence is more than 20% in some areas of poor sanitation and poverty, and less than 5% in the USA.^{1,2} *Entamoeba* is transmitted by ingestion of cysts: usually in food or water contaminated by human faeces, but occasionally by oral or anal sex, or contaminated enema apparatus.¹ Hence, a sexual history can be crucial. Traditionally, microscopic examination of stool has been used for diagnosis; however, PCR and ELISA can be used to distinguish *E histolytica* from the harmless commensal *E dispar*.³ Many infected people are asymptomatic. A typical syndrome develops over several weeks, and consists of bloody diarrhoea and abdominal pain, sometimes with weight loss and fever.¹ People who are pregnant or immunocompromised are at high risk of developing fulminant colitis, which is also associated with diabetes and alcohol misuse.¹ Fulminant amoebic colitis causes intestinal perforation in more than 75% of patients, and kills more than 40% of patients.¹ Amoebic colitis is treated with metronidazole or tinidazole; diloxanide furoate is used for cyst clearance.¹ Colonic resection is mandatory in the presence of colonic necrosis. Bowel resection with primary anastomosis has a high mortality rate, caused by anastomotic breakdown,⁴ so surgeons are generally advised to form stomas from the bowel ends either side of the resection. In Europe and North America, where amoebiasis is rare, it is often misdiagnosed—for instance, as ulcerative colitis⁵ or appendicitis with perforation—although overseas travel is not a prerequisite for tropical infection.

References

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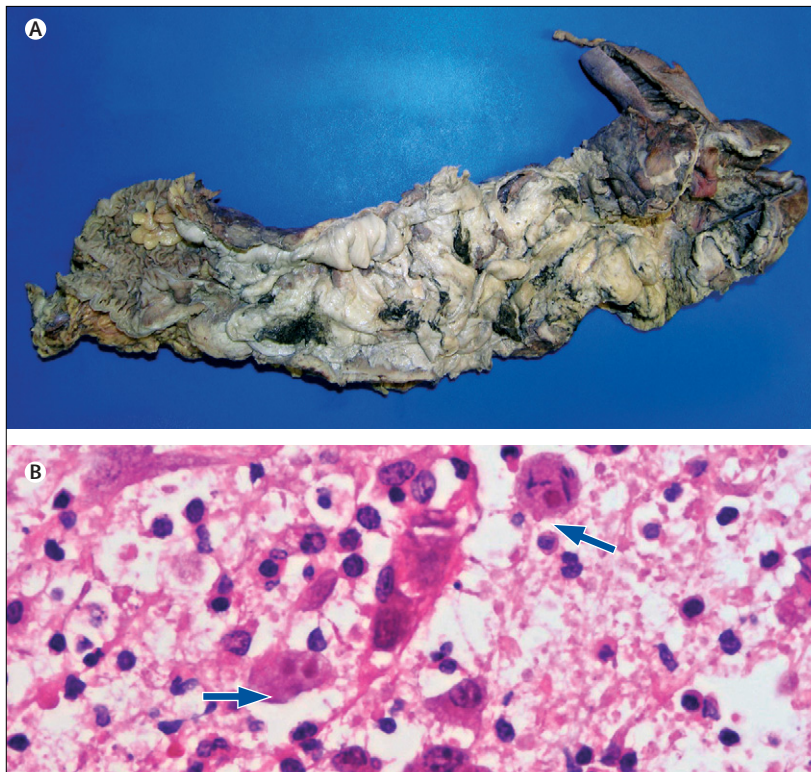


Figure: Amoebic colitis

(A) The resected colon. (B) Amoebae. The red inclusions are red blood cells.