Lesson of the Week

Indigenous amoebiasis: an important differential diagnosis of chronic inflammatory bowel disease

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Chronic inflammatory bowel disease often presents with chronic diarrhoea in childhood.^{1,2} Infection with microbial agents may, however, mimic this condition. Tuberculosis, yersiniosis, and amoebiasis must be considered in all cases. We describe a child with amoebiasis who had already been given treatment appropriate for chronic inflammatory bowel disease.

Case history

An 8 year old white boy presented to his general practitioner with chronic bloody diarrhoea; he was of normal intelligence and had never been out of England. He passed eight stools daily associated with blood and mucus. Two months later he was admitted to the paediatric unit of his local hospital with continuing bloody diarrhoea, weight loss, and anaemia. Sigmoidoscopy and rectal biopsy showed an inflamed colonic mucosa with ulceration; the presumptive diagnosis was chronic inflammatory bowel disease. Request for examination of a fresh stool for amoebas was refused on the ground that the child had never been abroad. He was transfused and began treatment with sulphasalazine and high dose oral steroids.

Despite an initial improvement he was still having bloody diarrhoea four weeks later and was therefore referred to the paediatric inflammatory bowel disease clinic at this hospital. On admission he was a frail, unwell child with a mildly tender abdomen. Erythrocyte sedimentation rate was 5 mm in first hour, serum albumin concentration 20 g/l, and C reactive protein concentration 5 mg/l. Blood was taken for serological screening for yersiniosis and amoebiasis; chest radiography and Mantoux test were performed to exclude tuberculosis. Colonoscopy showed a very inflamed mucosa with deep ulcers. Biopsy samples contained granuloma formation. The amoebic screen yielded a positive result with cellulose acetate precipitin and a fluorescent antibody titre of 1/640 to *Entamoeba histolytica*. Microscopy of a fresh stool then showed motile amoebas. A diagnosis of amoebic colitis was made. An unstained section of the original rectal biopsy sample was obtained and stained with periodic acid Schiff instead of haematoxylin and eosin; this showed amoebas in the tissue. Abscesses in the liver were excluded by ultrasound.

He was treated with intravenous metronidazole for 24 hours before being changed to oral medication. After five days a five day course of diloxanide furoate was added to eradicate any cysts. His stools were of normal consistency within three days of beginning treatment. He was discharged home. Three weeks later a repeat colonoscopy showed an improvement in

Amoebiasis may be contracted by children who have not been out of Britain; the clinical features and results of investigations may mimic those of chronic inflammatory bowel disease

his mucosa but some ulcers were still present. A further colonoscopy four months later showed completely normal mucosa endoscopically and histologically.

Comment

This case highlights three important features. Firstly, amoebiasis may closely resemble chronic inflammatory bowel disease in childhood. This has been reported in adults both in the United States³ and in Britain.⁴ This latter survey showed that in the 25 years after 1947 amoebiasis had a mortality of 1% and that 12 of the 30 patients who died were not diagnosed until necropsy. An editorial on the misdiagnosis of amoebiasis in Britain soon followed.⁵ The similarity to Crohn's disease in our patient was not confined to the clinical features alone but also included the findings of colonoscopy and colonic biopsy. Secondly, amoebiasis may be contracted by children who have never been overseas. A survey of children with amoebiasis presenting to the Queen Elizabeth Hospital for Children, London, and this hospital⁶ showed that two out of six had never been out of Britain. None of the relatives of our patient had recently been abroad, nor had there been any illness in the family. Thirdly, although the symptoms may disappear entirely within days of treatment, the mucosa may not return to normal until one to four months later.

Amoebiasis may be diagnosed either serologically or by examination of fresh stool. Serological investigations should include both a cellulose acetate precipitin test, which is highly specific, and a fluorescent antibody test, which is very sensitive. Microscopy of fresh stool is simpler but in practice may often be more difficult to organise; nevertheless, it should ideally be performed in any child presenting with chronic bloody diarrhoea.

References

- 1 O'Donoghue DP, Dawson AM. Crohn's disease in childhood. *Arch Dis Child* 1977;8:627-33.
- 2 Chong SKF, Bartram C, Campbell CA, Williams CB, Blackshaw AJ, Walker-Smith JA. Chronic inflammatory bowel disease in childhood. *Br Med J* 1982;284:101-3.
- 3 Krogstad DJ, Spencer HC, Healey GR, et al. Amebiasis: epidemiological studies in the United States. *Ann Intern Med* 1978;88:89-97.
- 4 Stamm WP. Amoebiasis in England and Wales. *Br Med J* 1975;iii:452-3.
- 5 Anonymous. Misdiagnosis of amoebiasis [Editorial]. *Br Med J* 1978;iii:379-80.
- 6 Sanderson IR, Williams CB, Walker-Smith JA. Indigenous amoebiasis in the United Kingdom. Plenary poster: annual general meeting of British Paediatric Association, 1984. York: BPA, 1984.

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