Amoebic hepatic abscess – potential causes of delay in diagnosis

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Summary
Delay in diagnosis can be serious in amoebic hepatic abscess, which is readily treatable. Two cases are presented to illustrate potential causes of delay. The first case had never been to an endemic area, apparently contracting the infection from his wife. The second case had negative serological tests.

Introduction
Stamm (1975) estimates that there may be about three deaths and about 200 new cases of amoebiasis annually in England and Wales. Amoebiasis is readily treated, and at least some of these deaths, and considerable morbidity, can be attributed to delay in diagnosis. The authors wish to report two cases of amoebic hepatic abscess which illustrate possible causes of delay in diagnosis.

It is frequently assumed that amoebiasis, being a tropical disease, can only be contracted in the tropics. It is thought that the first patient described below contracted the condition in the U.K. The second patient demonstrates that negative serological tests for amoebiasis should not deter the physician from a trail of metronidazole if hepatic amoebiasis seems a possible diagnosis. Both cases show the value of ultrasonic scanning of the liver in the differential diagnosis of solid from cystic hepatic masses.

Case 1
A 31-year-old man became ill in June 1973 with pleuritic pain in his right shoulder and under the right costal margin. Chest X-ray showed right lower lobe consolidation. The pain partially settled after 2 weeks, but his clinical condition did not improve. Over the next 4 months he lost 2 stone in weight, and suffered frequent night sweats. On admission in October his liver was enlarged 5 cm below the costal margin. Chest expansion was decreased on the right, with dullness to percussion below the right fourth intercostal space. There was a tender swelling laterally over the right lower chest. Temperature was 37.8°C on admission.

Investigations:
Hb 9.2 g/dl; white cell count (maximum) 15.7 x 10^9/l (88% neutrophils); ESR (Westergren) 115 mm/hr. Plasma: electrolytes normal; alkaline phosphatase 28.4 KAu. Serum: bilirubin <17.1 μmol/l; albumin 29 g/l; globulin 40 g/l; SGPT 3 units (normal <50).

Chest X-ray showed elevation of the right hemidiaphragm. Straight abdominal X-ray showed hepatomegaly. The diagnosis of hepatic abscess was confirmed by the ultrasonic scan (Fig. 1a) which demonstrated a 12-cm diameter trans-sonic mass occupying most of the right lobe of the liver. Indium 113 ferric chloride liver scan showed a large cold area posteriorly in the right lobe. Needle aspiration on 2.11.73 obtained 125 ml of typical ‘anchovy sauce’ pus. The diagnosis of amoebic abscess was confirmed by a fluorescent amoebic antibody test titre of 1:128. Treatment with metronidazole, 1-2 g/day for 10 days, two courses of emetine (60 mg i.m./day for 4 days each) and chloroquine 250 mg b.d. for 10 days brought about a remission of all symptoms, but repeated ultrasonic scans showed only partial reduction in the size of the hepatic abscess (Fig. 1b). Repeat aspiration of the abscess in January 1974 obtained a further 750 ml of pus, after which the abscess cavity progressively disappeared.
(Fig. 1c). At follow-up to July 1975 the patient remained asymptomatic, had regained his weight, and his liver size had returned to normal clinically. The patient had only once been outside Britain, and that was for a 2-week holiday in northern France in 1964. The source of his infection was therefore uncertain until his wife recollected suffering from severe 'colitis' between 1962 and 1964 while living in Nigeria. In 1964 she was advised at another hospital in the U.K. to have a total colectomy for 'ulcerative colitis', but discharged herself against medical advice. It was at this time that the patient and his wife met. The wife has had no symptoms since 1964, but her immunofluorescent amoebic antibody test in 1973 was 1:32, suggesting previous amoebic infection. The authors suspect that her illness in 1964 was due to amoebic colitis, and that her husband was infected directly at that time. Clinical examination of the patient's wife by sigmoidoscopy, and laboratory examination of the stools, show no evidence of amoebiasis at present (1977).

Case 2
A 37-year-old man presented in June 1975 with a 4-month history of increasing lethargy, anorexia, rigors, intermittent diarrhoea and a weight-loss of 15·9 kg. Examination showed a swinging pyrexia up to 39·6°C. The liver was enlarged six finger-breadths below the costal margin, and was firm, and not particularly tender. There was no adenopathy, splenomegaly or other abnormalities. The patient had served in the Armed Forces in Malta between 1957 and 1962 and had revisited Malta again in 1965 for a holiday. While in the Forces in Malta he had suffered from abdominal pain and bloody diarrhoea but no diagnosis was made. From that time he had noted a short episode of diarrhoea without blood twice yearly. In 1969 he had been jaundiced, which was thought to be due to infective hepatitis.

Investigations:
Hb 9·6 g/dl; HB,Ag, negative; white cell count (maximum) 14·0 × 10^3/l (86% neutrophils); ESR (Westergren) 105 mm/hr; film hypochromic with target cells. Plasma: electrolytes normal; alkaline phosphatase 36·5 KAU. Serum: bilirubin 8·55 µmol/l; albumin 23 g/l; globulin 40 g/l; α-fetoprotein, negative.

Liver scan (June 1975) multiple cold areas. Liver biopsy (July 1975) '. . . the liver contains an ill-defined area of young fibrous tissue in which there are collections of polymorphs, and there is what appears to be part of the wall of a subacute abscess. No amoebae were seen. There was no evidence of malignancy. The appearances suggested portal pyaemia with abscess formation'. Ultra-sound scan (Fig. 2a) showed a rounded trans-sonic area in the anterior part of the right lobe. The wall could not be completely traced but the findings were compatible with a cyst or abscess. Repeated stool examinations showed no amoebic cysts or other parasites. Sigmoidoscopy was normal. Rectal wall scrapings showed no evidence of vegetative amoebae.
A clinical diagnosis of multiple abscesses was made, and therapy with metronidazole 200 mg t.d.s. was commenced, and continued for 4 weeks. The patient’s fever settled to normal within 72 hr, and he made an uneventful recovery. When reviewed 6 weeks after the commencement of treatment he was asymptomatic, and had gained 6·35 kg in weight. His liver had regressed so that it was just palpable below the costal margin. A repeat liver scan still showed decreased uptake at the base of the right lobe, but otherwise was normal. Repeat ultrasound scan 3 weeks after commencement of treatment showed no abnormality (Fig. 2b). A fluorescent amoebic antibody test at the height of the patient’s illness was negative, with a titre of less than 1/32. A subsequent specimen one month after treatment showed an indirect fluorescent antibody test titre of 1:64 (no significant change). Examination of liver biopsy slides by an immuno-fluorescent stain for amoebae (Dr W. P. Stamm, Amoebiasis Diagnostic and Research Unit) showed one possible amoeba only, of doubtful significance.

Discussion
The majority of cases of amoebiasis seen in this country are in patients who have at some time lived in an area where amoebiasis is endemic. The short period of stay in the endemic area required to contract the infection, and the long period before symptoms develop may lead to difficulty in the diagnosis. A small proportion of cases are contracted indigenously, and case no. 1 is presented to emphasize this point. There are a small number of previously reported cases of amoebic hepatic abscess contracted indigenously (Paulley, 1961; Wright, 1966). There are in addition numerous reported cases of amoebic colitis contracted indigenously (Morton, Neal and Sage, 1951; Morton, Stamm and Seidelin, 1952; Conway and Watt, 1961; Wright, 1966). As in several previous cases it seems likely that patient no. 1 acquired his infection from a member of his family who had been infected abroad.

In case no. 2, the history of bloody diarrhoea while in Malta, followed by the development of hepatic abscess 15 years later was compatible with a diagnosis of amoebiasis, although the possibility of a pyogenic abscess could not be excluded. The initial titre of 1/32 in the immuno-fluorescent antibody test was not diagnostic of active amoebic infection, similar levels being found in patients with previous amoebic infections, and in patients with other, non-amoebic disorders (Jeanes, 1969). However, three out of sixty-one cases of extra-intestinal amoebiasis studied by this test had titres of 1/32 or less (Jeanes, 1969) and the authors would stress that such a low titre should not delay a trial of metronidazole therapy if amoebiasis seems probable clinically. The fact that many cryptogenic pyogenic abscesses are due to anaerobic organisms sensitive to metronidazole further supports its early use. It is also important to note that there are now several reports of the failure of this drug to cure hepatic amoebiasis, even using doses up to 2-1 g/day (Griffin, 1973). It was for this reason that when persistence of the abscess cavity was noted in case no. 1 the authors advised therapy with emetine and chloroquine in addition to the metronidazole.

In both cases, isotope scanning demonstrated a filling defect in the liver, and ultrasonic scanning was used to confirm that the defect was fluid-filled rather than solid. It has been suggested (Mathews et al., 1973) that amoebic hepatic abscess may be more difficult than pyogenic abscess to delineate by ultrasound examination because of the lack of a
clear echo-producing wall around an amoebic abscess. It was possible to find a clear wall in both the cases described above, although in case no. 2 the wall could not be traced completely around the abscess. These cases also confirm the value of serial scanning in the follow-up of treatment of these abscesses.

References

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