Lesson of the Week

Amoebic liver abscess in a Norfolk factory worker

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Amoebiasis is normally regarded as a tropical disease, and most cases in this country are imported. Indigenous cases are rare but must be considered in the differential diagnosis of dysentery[1] and liver abscess, as the following case illustrates.

Amoebiasis should be considered in patients with a liver abscess even though they have never been abroad

Case report

A 35-year-old Norfolk factory worker was admitted to hospital in November 1978 with a three-month history of right-sided pleuritic chest pain, weight loss of four stones (25·4 kg), and night sweats. There had been no change in bowel habit. He had never been abroad. On examination he was febrile but not jaundiced. His liver was palpable 8 cm below the costal margin. Posterolaterally over the lower right ribs there was superficial oedema and point tenderness. A clinical diagnosis of liver abscess was made.

Investigations showed Hb 10·2 g/dl; white blood cells $15{\cdot}2 \times 10^9$/l (15 200/mm^3); erythrocyte sedimentation rate 128 mm in 1st hour; urea and electrolyte concentrations normal; bilirubin 6 μmol/l (0·35 mg/100 ml); alkaline phosphatase 264 IU/l; aspartate transaminase 37 IU/l. An abdominal ultrasound examination and an isotope liver scan showed an abscess 16 cm in diameter in the posterolateral part of the liver. No cysts or amoebae were seen in the stools, but the fluorescent amoebic antibody test (FAT) was positive to a titre of 1/640 and the cellulose acetate precipitin (CAP) test was positive.

He was treated with metronidazole 800 mg daily for two weeks followed by diloxanide 500 mg tds for 10 days. He recovered and gradually put on weight, but follow-up ultrasound scans showed that the abscess cavity had increased in size so that by June 1979 the diameter was 19 cm. In September, 10 months after treatment, needle aspiration of the abscess was performed because of the risk of traumatic rupture. One and a half litres of clear fluid were drained. No amoebae or cysts were seen, and the fluid was sterile. He was treated with a further course of metronidazole. In March 1981 ($2\frac{1}{2}$ years after admission) he remained well, ultrasound examination showed a persistent abscess cavity with a diameter of 10 cm, the fluorescent amoebic antibody test was positive to a titre of 1/160 and the cellulose acetate precipitin test was weakly positive. Further questioning disclosed that his father (now deceased) had served with the Norfolk Regiment in Burma during the second world war and this was thought to be the source of the infection.

Comment

The diagnosis of amoebiasis must be considered in patients who have never been out of England who present with the clinical signs of a liver abscess. In an area where amoebiasis is not endemic the fluorescent amoebic antibody test is the most useful serological screening test. A titre of 1 in 256 or more, especially if associated with a positive cellulose acetate precipitin test, strongly suggests active amoebiasis.[2] In the convalescent patient the cellulose acetate precipitin test may remain positive in the absence of active disease.[3]

Serial liver scans show that most amoebic liver abscesses heal within four months of starting chemotherapy but occasionally healing may be delayed as long as one year.[4] The patient reported on is unusual because the abscess cavity has persisted for $2\frac{1}{2}$ years. Needle aspiration of an amoebic liver abscess may be unnecessary if the abscess heals rapidly after chemotherapy, but large abscesses and those considered at risk of rupture should be aspirated.[1] As in amoebic colitis treatment of amoebic liver abscess with corticosteroids may be disastrous.[5]

In the absence of the clinical signs of superficial oedema and point tenderness the clinical picture of an amoebic liver abscess with weight loss and a hard enlarged liver may mimic advanced malignancy.

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References

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Correction

ABC of 1 to 7: Infectious diseases

We regret that an acknowledgment was inadvertently omitted from this article by Dr H B Valman (17 October, p 1038). The illustrations were taken from Krugman S, Katz L. *Infectious diseases of children*. 7th ed. St Louis: The C V Mosby Co, 1981, by kind permission of the authors and publisher.