Tuberculous abscess of the liver

A case report

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Summary

Tuberculous abscess of the liver is rare. A case is reported and the southern African literature briefly reviewed. The clinical similarities to amoebic and pyogenic liver abscesses and primary or secondary neoplasms of the liver are emphasized. Laboratory tests and hepatic scintiscans may not assist in differentiating between these conditions.

Tuberculosis is common in southern Africa. Infection of the liver, however, is less frequent. Hepatic manifestations include fatty change, mononuclear infiltrates, granulomatous hepatitis, miliary tubercles, solitary or multiple tuberculomas, porta hepatis nodes1-3 and, rarely, tuberculous liver abscesses. There are few reports of a primarily hepatic presentation of TB in the southern African literature,4-9 and no case of abscess formation has been documented. Hersch5,6 reviewed 200 cases of hepatic TB seen between 1955 and 1961 and found no cases of abscesses, although in 2 cases the latter were simulated. We reviewed 98 consecutive cases of proven hepatic TB seen during 1974 through 1976 and 1978 through 1981, and found only 1 case of abscess. Indeed, only 90 cases had been reported worldwide up to 1975-1976.10-12 To our knowledge this is the first report of a tuberculous liver abscess in southern Africa.

Case report

An ill-looking 3-year-old Black child was admitted with swelling of the feet and face and a history of passing brownish stools mixed with blood. Kwashiorkor and TB had been diagnosed previously at a community clinic. He had marked pallor and mild pedal oedema. The lung fields were clear. Abdominal distension was observed but no free fluid could be demonstrated and there were no palpable masses. Rectal examination confirmed the presence of blood mixed with stool.

Investigations

The tine test was positive. The haemoglobin concentration was 4,1 g/dl (normal 9,6-15,5 g/dl) with normal red blood cell indices; the white cell count was 8 900/µl with a normal differential count, and the platelet count was 85 000/µl (normal 150-400 000/µl) with 4% reticulocytes. The blood sugar, urea, electrolyte, serum vitamin B12, and folate levels were normal. The erythrocyte sedimentation rate was 109 mm/1st h. The serum iron level was 16,7 µmol/l (normal 15-30 µmol/l), the transferrin level 0,8 g/l (normal 1,9 and 4,0 g/l) and the percentage saturation 79% (normal 15-50%). Microscopic examination and culture of stool and urine showed no pathogens or parasites. Blood cultures were negative. Agglutination and complement fixation tests for typhoid, Brucella, Rickettsia, Yersinia, amoebae and Echinococcus showed no rise in titres. The prothrombin index was 66%. The serum albumin level was 22 g/l (normal 35-50 g/l), and the globulin level 47 g/l (normal 25-30 g/l). The total bilirubin and transaminase levels were normal, but the serum alkaline phosphatase level was raised to 343 IU/l (normal 36-92 IU/l). Hepatitis B surface antigen was absent. Chest radiographs showed equivocal signs of TB. Barium studies of the gut revealed no lesions.

Progress

Over the ensuing 2 weeks the patient developed a spiking temperature, leucocytosis (16800 /µI) and a rapidly enlarging, tender liver which was palpable 6 cm below the costal margin. No hepatic bruit or friction rub was noted. Amoebic or pyogenic abscess was thought to be probable in view of the rapidly enlarging liver and the earlier haematochezia. Consequently a trial of both metronidazole and broad-spectrum antibiotics was commenced, with no improvement.

Hepatic scintiscanning and ultrasonography performed 2 weeks apart confirmed the presence of an enlarging mass in the region of the porta hepatis (Fig. 1). Isoniazid (INH) and ethionamide were commenced in the 5th hospital week, with a remarkable stabilization of the temperature. However, since the hepatomegaly persisted, the presence of a primary or secondary hepatic neoplasm was also considered. In the 9th hospital week laparotomy was undertaken.

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Fig. 1. Hepatic scan: filling defect seen on lateral view (arrows).
This revealed an abscess in the right lobe of the liver, from which thick pus was aspirated. The adjacent peritoneum was similarly involved. Adhesions were noted around the spleen, but the local lymph nodes, stomach and intestines were normal. Histopathological examination showed a focus of massive caseous necrosis with surrounding multinucleated giant cells, fibroblasts, acute and chronic inflammatory cells and acid-fast bacilli (Figs 2 and 3). Random biopsy samples taken from the right and left lobes were normal. No associated hepatic lesion was noted. The patient made an uneventful recovery on antituberculosis therapy, and the liver decreased in size over a period of several weeks. He was discharged from hospital but was lost to follow-up.

Fig. 2. Liver biopsy specimen (x 70). Note the caseous necrosis (bold arrows) bordered by granulomas, Langhans giant cells (double arrows) and chronic inflammatory cells.

Discussion

Liver involvement is common in cases of miliary TB. However, the primarily hepatic forms of TB are much less frequent, tuberculous liver abscess being one of the least common manifestations, with an incidence of 0.34% (1/298 cases of hepatic TB) at Baragwanath Hospital. These abscesses may represent caseous necrosis of large tuberculomas, although they could follow haematogenous spread. Patients characteristically present with rigors, fever, weight loss, sweating and sometimes abdominal pain. Unusual manifestations include diarrhoea and jaundice. The condition is most frequently confused with hepatoma and amoebic or pyogenic liver abscess, and indeed may be difficult to differentiate even after extensive investigation. This is understandable in the presence of marked wasting, chronic illness, pyrexia, a progressively enlarging tender liver, normal or slightly elevated serum transaminase levels, a markedly elevated alkaline phosphatase level, the presence of large filling defects on hepatic scintiscanning or ultrasonography, and sometimes unhelpful chest radiographs. Here, the term hepatic 'pseudotumour' seems appropriate.

The diagnosis becomes evident after the demonstration of acid-fast bacilli on staining or culture of the pus or cavity wall. However, the presence of TB elsewhere together with a typical laparotomy appearance, the presence of caseation, the failure to demonstrate other pathogens and the response to specific therapy are often deemed sufficient criteria. Rarely, the diagnosis is suspected when fistulas develop after performance of liver biopsy. The diagnostic dilemma is illustrated by the frequency with which laparotomy is resorted to.

In southern Africa amoebic liver abscess, hepatoma and TB are all common, and it is therefore important to identify an eminently treatable condition such as hepatic TB.

REFERENCES