Case report

Amoebic abscess in the cirrhotic liver

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SUMMARY Though amoebic liver abscess and liver cirrhosis occur very commonly in hospital practice in the tropics, they have not to the knowledge of the present authors hitherto been reported to occur simultaneously in the same patient. The patient described here, who had clear-cut clinical and histological features of chronic liver cirrhosis with portal hypertension and ascites, presented somewhat acutely with liver pain and an amoebic liver abscess that contained 'chocolate sauce' on needle aspiration. The amoebic abscess, although, no doubt, superimposed on chronic irreversible cirrhosis, rapidly regressed on metronidazole therapy. The infrequency with which liver abscess and liver cirrhosis coexist cannot be satisfactorily explained. It is probable, however, that extensive scarring in the liver may prevent entamoeba histolytica from thriving.

Amoebic liver abscess and liver cirrhosis tend to occur very commonly as separate clinical entities in tropical practice. In spite of the frequency with which these two disorders independently occur, it would appear that they only rarely coexist in the same patient. A deceptive clinical picture may be given by a primary liver carcinoma developing in liver with advanced cirrhosis; this strikingly common association may sometimes closely mimic coexisting amoebic abscess formation. However, an amoebic liver abscess superimposed on chronic cirrhosis is, in our own experience, quite unusual and to our knowledge has not been reported in the literature; hence, therefore, the justification for this case report.

Case history

The patient, OK (LUTH 002862), a 45-year-old factory worker was admitted to the Lagos University Teaching Hospital on 19 July 1978 having presented with a two-week history of right hypochondrial pain and swelling of an insidious onset accompanied by pyrexia with chills and sweats, weight loss, and general prostration. His past medical and social history revealed heavy alcohol consumption until recently and an attack of jaundice in 1960 which was self-limiting.

On specific questioning he denied drug abuse with potentially hepatotoxic compounds and also denied any history of recent dysenteric symptoms or gastro-intestinal haemorrhage. On examination he was ill-looking, pale and toxic and had digital clubbing. He had a pulse rate of 94/minute and the blood pressure was 130/80 mmHg. Examination revealed a 7 cm smooth tender hepatomegaly and conspicuously engorged superior epigastric venous collateralisation in the upper abdomen with demonstrable circulatory flow in the cephalic direction. Moderate ascites was readily elicitable but there were no other enlarged organs of intra-abdominal masses, and specifically no arterial bruit could be heard. Rectal examination was normal, as was the examination of the central nervous system and the cardiopulmonary system.

At this stage a definite diagnosis of liver cirrhosis with portal hypertension and ascites was made and the patient was further investigated to exclude either a primary liver-cell cancer developing in pre-existing cirrhosis—a well-recognised association¹—or an amoebic liver abscess superimposed on chronic cirrhosis.

INVESTIGATIONS

Haemogram on admission showed Hb 6.9 g/dl, haematocrit 20%, total white blood count 16,200/cu mm with 80% polymorphonuclear leucocytosis and ESR 112 mm/h (Westergren). Three consecutive faecal smears were negative on microscopy for amoeba trophozoites and cysts and for bacterial pathogens on culture.

Radiographs of the chest showed that the right dome of the diaphragm was markedly raised with clear lung fields (Fig. 1A), a radiological sign which has been reported to be highly diagnostic of amoebic

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Serum chemistry studies showed the deranged liver function characteristic of hepatic cirrhosis: total serum protein concentration 43 g/l, with albumin fraction severely depressed to 17 g/l (normal range 31–51.8 g/l); AST (SGOT) 103·9 IU/l (normal 4–12 IU/l); ALT (SGPT) 60·9 IU/l (normal 5–15 IU/l); alkaline phosphatase 241·5 IU/l (normal 21–65 IU/l). Hepatic cirrhosis was confirmed histologically by liver biopsy, which, on microscopic examination, showed patchy destruction of liver parenchyma by broad zones of fibrous tissue containing mononuclear infiltration and separated by nodules of regenerated liver cells. In addition, further diagnostic support was given by the appearances on liver scan which showed the patchy uptake pattern of cirrhosis, with an adjoining cold area in the right lobe which could signify either cancerous invasion or an amoebic abscess cavity (Fig. 2a).

The diagnosis of amoebic liver abscess was unequivocally proven by the results of a diagnostic needle aspiration of the abscess; this readily yielded 150 ml of typical ‘chocolate sauce’ which, using the indirect fluorescent antibody test for amoeba, showed a positive titre of 1:250 thus confirming recent past or present infection with the parasite.4

Thus, on the basis of the history and clinical examination, supported by the results of the ancillary laboratory investigations described above, this patient clearly had amoebic liver abscess superimposed on a coexisting chronic, and probably alcoholic, cirrhosis.

COURSE

Metronidazole therapy (800 mg for five days) instituted for amoebic liver abscess led to a striking improvement in the patient’s condition. Within 72 hours he had become apyrexial, liver pain and tenderness had waned, and hepatomegaly had regressed to 5 cm below the right costal margin. The liver, however, remained quite firm in its consistency with fine nodularity of its anterior surface. Auscultation revealed no sign of an arterial bruit. A follow-up radiograph of the chest within three weeks of admission and before discharge from the hospital showed right hemidiaphragmatic descent to a near normal level (Fig. 1B) and a return also to normal of the brisk leucocyte response. The abnormalities in serum chemistry remained essentially unchanged with the exception of serum alkaline phosphatase which showed a significant improvement (Fig. 2b).
Amoebic abscess in the cirrhotic liver

phosphatase activity which fell to 153.3 IU/l, thus suggesting that the abnormal liver function tests before treatment were due to underlying cirrhosis.

Discussion

The 'pus' derived from an amoebic liver abscess is known to consist of liver cells which have undergone necrosis and liquefaction as a result of the damaging action of amoebic invasion of the organ. It would seem reasonable to speculate that an appreciable loss of hepatocytes, as occurs in hepatic cirrhosis, may hinder amoebae from gaining a foothold within liver parenchyma in which the normal lobular architecture has become disorganised—liver cells being replaced by extensive connective tissue proliferation and scarring. It is not unlikely that the only susceptible target cell within the liver is the hepatocyte. This might explain the relative rarity of amoebic implantation in a liver with advanced cirrhosis, in spite of the vulnerability of such patients to intercurrent sepsis due presumably to a depression in their immunological status. Moreover, information gained from an MRC study on amoebiasis in Gambia, West Africa, which noted the rare occurrence of overt amoebiasis in that country in spite of high infection rates, suggested that cell-mediated immunity must play a large part in the development of the overt disease. It could therefore be argued that a predilection for hepatic amoebiasis might be a feature of chronic cirrhosis, but, on the contrary, the relationship between the two clinical entities has been a fortuitous one. It is accordingly suggested that extensive scarring in the liver probably constitutes a physical impediment for Entamoeba histolytica to thrive.

References