

NOTA PRÉVIA

LIVER ABSCESS AND SCHISTOSOMIASIS. A NEW ASSOCIATION

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The most common predisposing cause for pyogenic hepatic abscess is currently biliary tract disease. Infection in areas drained by the venous system (e.g., appendicitis, diverticulosis, Crohn's disease) may also result in pylephlebitis and pyogenic hepatic abscess. Other causes include penetrating or blunt abdominal trauma, septicemia, and infection arising in necrotic primary or secondary tumor deposits. Pyogenic liver abscesses may be single or multiple. Multiple small abscesses occur particularly in association with pylephlebitis, biliary tract obstruction with cholangitis, and septicemia. In many cases, no predisposing cause is apparent^{3,6}. It is reported here 2 patients with acute schistosomiasis and pyogenic liver abscesses and we present experimental evidence that schistosomiasis may be a predisposing cause for hepatic abscesses.

CASE REPORTS

Patient 1

A 10-year-old boy was admitted to hospital following a 10-day fever, weakness, loss of weight, abdominal pain, and diarrhoea. He appeared acutely ill. He had a furuncle on his left elbow. The liver was palpable 8 cm below the right costal margin and the spleen 3 cm below the left costal margin. Three hemocultures performed on different occasions were positive for *S. aureus*. Coproscopy disclosed the presence of viable eggs of *S. mansoni*. Routine laboratory studies revealed leukocytosis (Global: $18.3 \times 10^9/L$; neutrophils: $13.2 \times 10^9/L$, and eosinophils: $1.6 \times 10^9/L$), anemia (hematocrit: 0.25 l), and slightly elevated serum alkaline phosphatase. Ultrasonography showed multiple liver abscesses. After a three-day course of oxacilin and clindamicin surgical drainage of two abscesses was undertaken. Gram's stain

and culture of the material confirmed *S. aureus* as the sole microorganism in the abscess. Liver biopsy showed the presence of many necrotic-exsudative granulomata formed around *S. mansoni* eggs. The patient responded well to treatment with antibiotics showing convincing and rapid improvement, and was discharged from hospital 15 days after admission. Two days before dismissal he was treated for schistosomiasis with oxamniquine (20 mg/kg, body weight, single dose).

Patient 2

The patient was a 10-year-old boy who had a 4-day history of fever, vomiting, abdominal pain and hyporexia. On admission his body temperature was 39.2°C and he appeared acutely ill. A well defined, non-secreting area of folliculitis was observed on his left buttock. His abdomen was distended and tender on palpation. The liver was palpable 6 cm below the right costal margin. Hemocultures were negative. Coproscopy demonstrated viable eggs of *S. mansoni*. Liver function tests were abnormal (aspartate aminotransferase: 2.90 $\mu\text{kat/L}$; alanine aminotransferase: 2.32 $\mu\text{kat/L}$; bilirubin, total: 44.5 $\mu\text{mol/L}$; alkaline phosphatase: 3.2 $\mu\text{kat/L}$), hematocrit (0.30 l). The leukocyte count was $13.9 \times 10^9/L$ with $11.7 \times 10^9/L$ neutrophils. Ultrasound examination of the abdomen diagnosed multiple liver abscesses. His clinical condition deteriorated rapidly in the next 3 days, notwithstanding the use of oxacilin and amikacin. 4 days after admission he was operated on. Surgery revealed multiple liver abscesses in the left lobe of the liver. Liver biopsies showed *S. mansoni* eggs surrounded by hyperergic granulomata and culture of material from the abscesses isolated *S. aureus*. In the next 15 days the patient presented progressive improvement on oxacillin and amikacin. Specific treatment for *S. mansoni* infection was commenced after a convincing clinical response with antibiotics. The patient was discharged from hospital 32 days after admission and was asymptomatic on his last follow up, 3 months later.

Some common findings presented by our patients should be emphasized: 1. Both are children; 2. Both responded well to treatment with antibiotics, surgical drainage and oxamniquine⁴; 3. They reported recent contact with stream water in an *S. mansoni* endemic area and both had liver biopsies with hyperergic granulomata characteristic of acute schistosomiasis; 4. Both had had skin pustules beginning some

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time before the appearance of symptoms of liver abscess.

It is possible that schistosomiasis is a predisposing cause for liver abscesses, in view of: 1. Pylephlebitis has been described as an important aspect of liver pathology in schistosomiasis, and pylephlebitis is known as a predisposing factor for liver abscess; 2. It has been shown that necrotic areas in the liver may be infected by bacteria in patients with primary or secondary tumor deposits. In acute schistosomiasis a miliary distribution of eggs/granulomata (with central necrosis) occurs in the liver; 3. Transient immunodepression has been described in the acute phase of schistosomiasis, in animal models^{1 5} and in man².

The missing link would appear to be the absence of bacteremia. In our patients we suggest that the skin pustules (furuncles) represented the reservoir for *S. aureus* and gave rise to transient bacteremia.

To confirm that schistosomiasis is a predisposing factor for liver abscess, an experimental study was done. 60 days after being infected percutaneously with 70 *S. mansoni* cercariae (LE strain), 16 outbred albino mice (with appropriate controls) were injected intravenously with *S. aureus* (2×10^5 bacteria/ml), isolated from a patient with septicemia. 10 to 14 days later, 5 mice developed multiple pyogenic liver abscesses. *S. aureus* was recovered from the abscesses and from the blood of 2 mice.

Based on this preliminary report we should like

to call the attention of clinicians working in endemic areas of schistosomiasis to this association.

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