HEPATIC HYDATIDOSIS: DIAGNOSTIC AND THERAPEUTIC ASPECTS

R P Jalleh, R Nuruddin, M M S Krishnan

ABSTRACT

A rare case of hepatic hydatidosis in non-endemic Malaysia is reported. It is important that clinjcians treating the occasional patient be familiar with current diagnostic and therapeutic modalities.

Key words: Hepatic hydatidosis, Echinococcus, Malaysia.

SING MED J. 1989; No 30: 210 — 212

INTRODUCTION

Hydatid disease is a parasitic infection caused by several species of the cestode Echinococcus. Although endemic in many parts of the world, including Australia, the Middle East and the Meditteranean and Baltic areas, the disease is extremely rare in Malaysia. We report the first documented case seen at the University Hospital, Kuala Lumpur with emphasis on diagnostic modalities and management.

CASE REPORT

A 59 year old Indian female presented with fever and right hypochondrial pain for 3 days. She had no contact with animals and had visited the Trichi area in South India in 1957. Examination revealed an ill, febrile patient, with a temperature of 38.5°C. There was no jaundice. The enlarged liver, span of 17cm, was markedly tender. Investigations showed a normal haematocrit with a leucocytosis of 14100/μL. Liver enzymes were within normal limits.

Ultrasonography revealed a well encapsulated multiloculated anechoic lesion occupying most of the right hepatic lobe. The biliary tree and rest of the liver were normal. Computed tomography confirmed the large non-enhancing low-attenuation multiseptate lesion (Fig 1). A curvilinear calcification was noted at the periphery of this abnormality (Fig 2).

A clinical diagnosis of a multiloculated liver abscess was made, with differential diagnosis of infected hydatid cyst and necrotising hepatocellular carcinoma. Initial management consisted of rehydration, correction of coagulation profile and parenteral antibiotics. As this regime failed to elicit a response within 24 hours, emergency laparotomy was performed.

A large cystic lesion was noted in the right hepatic lobe. Clear fluid was obtained an aspiration. After the field was packed off with towels soaked in povidone iodine, an incision was made into the cyst. Hundreds of daughter cysts, each measuring 1-5 cm, were evacuated (Fig 3). Great care was taken to prevent spillage of cysts and fluid of the hydatid. After all daughter cysts were completely removed, the paracyte derived ectocyst was scraped out. Cetrimide (0.5%) was instilled into the residual host cavity and left in situ for 10 minutes. Primary closure without drainge was employed. Post-operatively, the patient was started on mebendazole 40 mg/kg/day for 1 month.

Two weeks after operation, the patient developed an abscess of the residual cyst cavity. This was drained via an indwelling pigtail polythene catheter inserted under fluoroscopic control. The patient responded dramatically. There was, however, persistent drainage of bile. Contrast examination confirmed a cyst-biliary fistula (Fig 4). This was treated conservatively with occasional flushing of the catheter. Subsequent examinations revealed the cavity to be decreasing in size and the catheter was removed 4 months after operation. She was well at 1 year follow-up examination.

DISCUSSION

Although human hydatid disease was known to Hippocrates and Galen and the parasitic nature of this disease was strongly suspected in the 17th century, it is only during the present century that significant advances were made in the accurate diagnosis and treatment of this condition (1). Of the two forms of human hydatid disease, it is the variety caused by Echinococcus granulosus that is much commoner, as seen in this case. Infection occurs when man becomes an accidental intermediate host for the larval form of the parasite. The commonest site of involvement is the liver, particularly the right lobe (2).

The diagnosis of hepatic hydatidosis has, in the past, been based on a history and clinical findings that are compatible with the disease, especially in endemic regions. However, as a result of ease of travel and migration, the disease is now encountered in locations in which it is non-endemic (3, 4). It is important, therefore, that clinicians treating the occasional patient be familiar with current diagnostic and therapeutic modalities.

Initial clinical suspicion is now confirmed by various radiological and serological tests. Computed tomography (CT) is the most effective imaging technique available (3, 5). It is non-invasive, highly sensitive, demonstrates the unique characteristics of hydatid cysts and may also discover additional small intrahepatic or unsuspected extrahepatic lesions. Its particular value is the accurate anatomic localisation of the cyst which is of special value to the operating surgeon. The findings of a hypodense lesion containing multiple daughter cysts and the presence of cyst wall calcification, as demonstrated in this patient, are characteristic features (5, 6). Ultrasonography has been found to be a useful alternative (1, 6).

Various sensitive and specific serological tests have now replaced the Casoni intradermal test for primary

Department of Surgery and Radiology
University Hospital
59100 Kuala Lumpur Malaysia

R P Jalleh, FRCS (Ed), FRCS (Glas), FRCSI
Lecturer
R Nuruddin, MBBS (Aust), M Sc (Nuclear Med), DMRD, FRCR
Lecturer
M M S Krishnan, MS, FRCS (Ed), FRCS (Glas), FICS
Associate Professor

Correspondence to: Dr Jalleh

LEGENDS

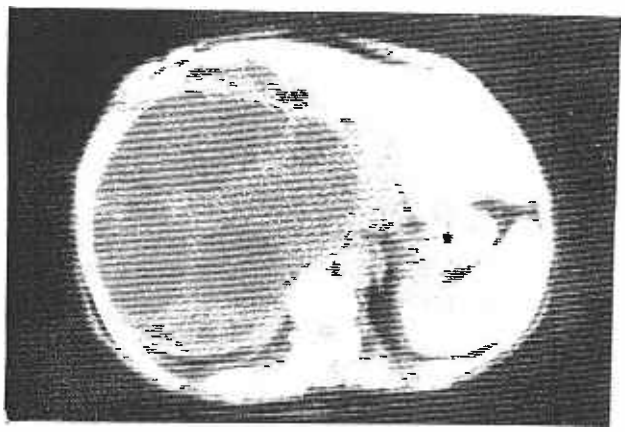

Fig.1: CT Scan showing multiseptate hypodense lesion in the right hepatic lobe.

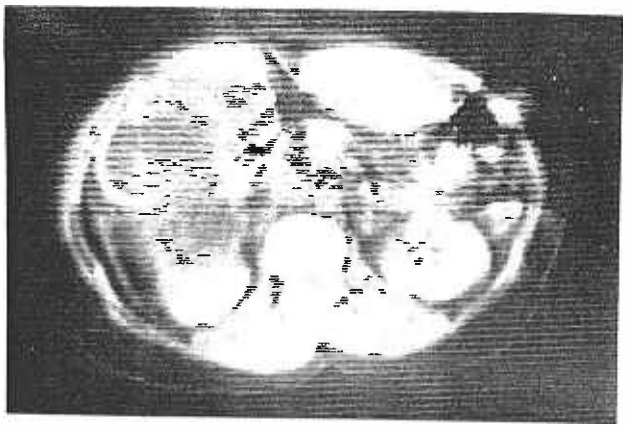

Fig. 2: CT Scan showing curvilinear rim of calcification.

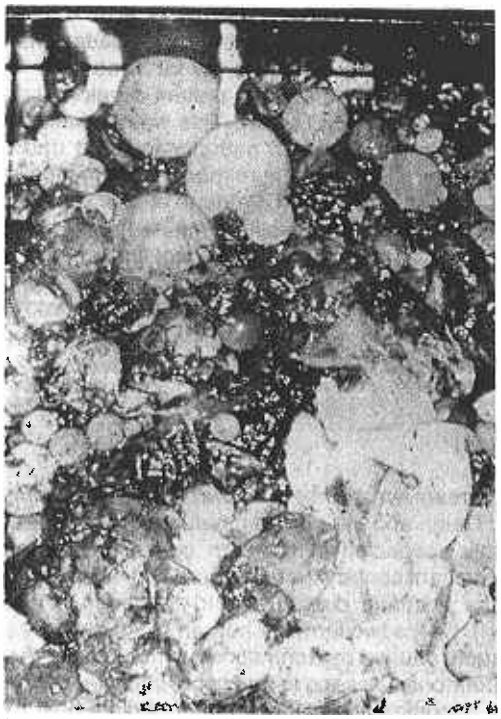

Fig. 3: Multiple daughter cysts.

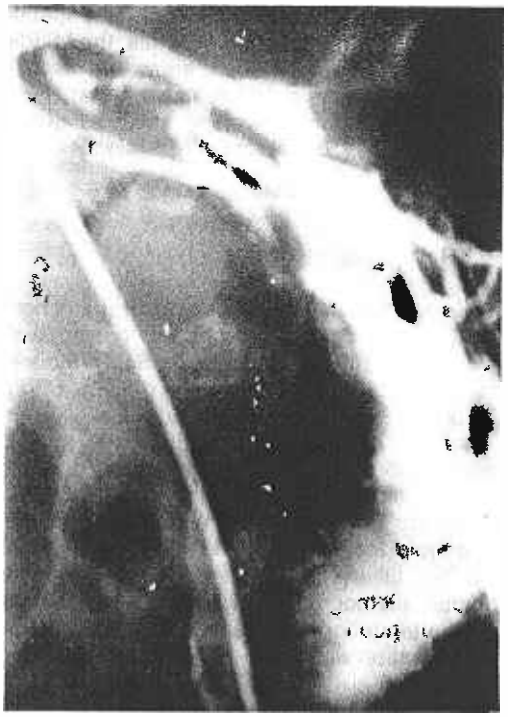

Fig. 4: Tube sinogram demonstrating intrahepatic biliary ducts indicating presence of cyst biliary communication.

diagnosis (1). Disadvantages of the latter include false positive and negative rates, persistence after successful treatment and the occasional problem of anaphylaxis (1, 7). The current investigation of choice is hydatid immunoelectrophoresis. This technique depends on the formation of a specific arc of precipitation (called 'Arc 5'), produced by the interaction of the serum of the hydatid patient, and hydatid antigen, as compared to a control. Other methods include the complement fixation, indirect haemagglutination and enzyme-linked immunosorbent assay tests. These were not available at our institution.

Most authors agree that the primary treatment of hydatid cyst of the liver is surgery (6). Although controversy exists regarding the choice of surgical procedure, the more conservative approach of cyst evacuation and management of residual cavity is currently favoured (2, 3). Features of the surgical technique described in this case represent standard operative procedure. Various scolicidal agents may be used to sterilise the residual cavity. These include hypertonic saline (15-20%), 0.5% cetrimide, chlorhexidine and 80% alcohol. The previously recommended formalin (2, 8) is absolutely contraindicated in view of the serious side effects of obliterative cholangitis and occasional fatal acidosis (6, 9).

The complication of cyst-biliary fistula, as seen in this case, has been described in detail (1). Although elosure of these communications under direct vision at operation has been recommended, the identification of such fistulae may prove difficult to the occasional surgeon. In the absence of biliary obstruction, the fistula in this patient closed spontaneously.

Medical management has recently attracted interest. Following Bekhti's report on the use of high dose mebendazole (10), anecdotal reports on the use of this agent (11, 12) and albendazole (6, 13) have featured in the literature. Their use has been occasionally associated with hepatotoxicity and reversible neutropenia. As medical treatment is not curative (6), its present role should be limited to use as an adjunct to surgery and in the treatment of patients unfit for surgical intervention.

ACKNOWLEDGEMENT

The authors wish to thank Puan Zureena Binti Haji Haron for her secretarial assistance.

REFERENCES

1. Kune GA. Hydatid disease. In: Schwartz SI, Ellis H. eds Maingot's Abdominal Operations, 8th ed. Norwalk: Appleton — Century — Crofts. 1985: 1605-24.
2. Dugalic D, Djukic V, Millicervic M, Sterovic D, Knezeric J, Pantic J. Operative procedures in the management of liver hydatidoses. World J Surg 1982; 6: 115-8.
3. Langer JC, Rose DB, Keystone JS, Taylor BR, Langer B. Diagnosis and management of hydatid disease of the liver — a 15 year North American experience. Ann Surg 1984; 199: 412-7.
4. Pitt HA, Korzelius J, Tompkins RK. Management of hepatic echinococcosis in Southern California. Am J Surg 1986; 152: 110-5.
5. Ismail MA, Al-Dabaqh A, Al-Janabi TA, et al. The use of computerised axial tomography (CAT) in the diagnosis of hydatid cysts. Clin Radiol 1980; 31: 287-90.
6. Langer B. Surgical treatment of hydatid disease of the liver. Br J Surg 1987; 74: 237-8.
7. Yarzabal LA, Schantz PM, Lopes-Lemes MH. Comparative sensitivity and specificity of the Casoni intradermal and the immunoelectrophoresis tests for the diagnosis of hydatid disease. Am J Trop Med Hyg 1975; 24: 843-8.
8. Heslop JH. Hydatid cysts of the liver. In : Dudley H, Rob C, Smith R Sir eds. Operative Surgery : Abdomen 3rd ed. London: Butterworths 1977; 320-8.
9. Aggarwal AR, Garg RL. Formalin toxicity in hydatid liver disease. Anaesthesia 1983; 38: 662-5.
10. Bekhti A. Schaaps JP, Capron M, et al. Treatment of hepatic hydatid disease with mebendazole : Primary results in four cases. Br Med J 1977; 2: 1047-51.
11. Davidson RA. Issues in clinical parasitology : the management of hydatid cyst. Am J Gastroenteral 1984; 79: 397-400.
12. Gil-Grande LA, Boixeda D, Garcia-Hoz F, et al. Treatment of liver hydatid disease with mebendazole : a prospective study of thirteen patients. Am J Gastroenteral 1983; 78: 584-8.
13. Saimot AG, Meulemans A, Cremieux AC, et al. Albendazole as a potential treatment for human hydatidosis. Lancet 1983; ii: 652-6.