Journal of Gastroenterology and Hepatology (1999) **14**, 931–934

PERCUTANEOUS DRAINAGE OF THE PANCREATIC CYST

CASE REPORT: Percutaneous drainage of the pancreatic head hydatid cyst with obstructive jaundice

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Abstract We report a rare case of a patient with a primary hydatid cyst in the head of the pancreas who presented with obstructive jaundice caused by extrinsic compression of the intrapancreatic portion of the bile duct. The patient was treated successfully by ultrasound-guided percutaneous drainage of the cyst using hypertonic (20%) saline as the scolicidal agent and albendazole chemoprophylaxis before and after the drainage. The cyst was not visible on ultrasonography at 6 months follow up. Clinical, sonographic and serological follow up to 35 months showed no evidence of cyst recurrence or dissemination. In endemic areas of hydatid disease, hydatid cyst should be a differential diagnosis in cystic lesions of the pancreas in patients presenting with obstructive jaundice.

Key words: cholestasis, cyst, echinococcosis, interventional procedures, liver, pancreas, parasites, percutaneous drainage.

INTRODUCTION

Common cystic lesions of the pancreas presenting with obstructive jaundice include pseudocysts, cystic neoplasms and true congenital and post-traumatic cysts.[1] Hydatid cysts are rarely found in the pancreas. Data taken from the literature give values ranging between 0.19 and 2%.[2–4] Obstructive jaundice caused by compression of the common bile duct from a hydatid cyst in the head of the pancreas is even rarer.[2,5–7] The embryos of hydatid cysts end up in the pancreas mainly by haematological and very rarely by retroperitoneal spread. Biliary or pancreatic canals have not been shown to be involved in such dispersion.[5] All the pancreatic hydatid cysts reported to date have been treated surgically with significant morbidity.[2,3,6–8] Promising results of percutaneous drainage (PD) in the treatment of hepatic hydatid cyst (HHC)[9–16] and the unique location of the cyst reported herein prompted us to treat the patient by this non-surgical technique to assist pancreatic preservation, decrease morbidity and shorten the hospital stay.

CASE REPORT

An 18-year-old male patient was admitted to the SK Institute of Medical Sciences hospital for evaluation of painless progressive cholestasis of 1.5 months duration. Examination revealed a pulse of 94/min, blood pressure 100 mmHg systolic and 80 mmHg diastolic, temperature 37.6°C, deep jaundice and scratch marks all over the body. His liver was palpable 6 cm below the right costal margin with a span of 16 cm in the midclavicular line and with smooth surface. The gallbladder was visibly enlarged in the right hypochondrium. Examination of the chest and cardiovascular system did not reveal any abnormality. Per rectum examination was normal and the faeces were clay coloured. Urine was dark coloured and contained bile pigments. Urobilinogen, however, was absent. His haemoglobin level was 10.2 g/dL, leucocyte count 9.0×10^9/L, with neutrophils 78%, lymphocytes 18%, eosinophils 3% and monocytes 1%. Serum bilirubin was 16.6 mg/dL, aspartate aminotransferase 70 IU/L, alanine aminotransferase 14 IU/L, alkaline phosphatase

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Accepted for publication 5 March 1999.

1820 IU/L, total proteins 7.0 g/dL, albumin 3.0 g/dL and serum amylase 230 U/L. Abdominal ultrasonography revealed an enlarged liver, dilated intrahepatic bile ducts, dilated common duct (1.9 cm) and distended gall-bladder (transverse diameter 4.2 cm). No echogenic structures were seen in the biliary tree. The lower end of the bile duct was displaced medially by a rounded anechoeic mass 4.7 × 4.7 cm in the head of the pancreas. The mass revealed posterior acoustic enhancement suggestive of a cyst (Fig. 1). An endoscopic retrograde cholangiopancreatogram revealed normal duodenoscopic examination but attempts at cannulation of the bile duct and pancreatic duct failed. A percutaneous transhepatic cholangiogram (PTC) revealed a dilated intrahepatic bile duct and common duct. The common duct at its mid length revealed sudden narrowing and obstruction. No contrast drained into the duodenum. Casoni's intradermal test was immediately positive and hydatid serology performed by an enzyme-linked immunosorbent assay revealed a positive titre of immunoglobulin (Ig) G and M antibodies to *Echinococcus granulosus* (normal 1:160). A diagnosis of hydatid cyst in the head of the pancreas causing obstructive jaundice was made. After getting an informed consent from the patient, it was planned to drain the cyst by PD. The patient was started on albendazole 10 mg/kg per day, orally. On 10th day of drug therapy, PD of the cyst was performed. Albendazole (400 mg) was administered on the morning of the procedure 4 h prior to cyst puncture. Under continuous monitoring and ultrasound guidance, PD was done in three steps. In step 1, the cyst was punctured through the biopsy port of the puncture probe (3.5 MHz) with the use of a 22-gauge, 22-cm long cholangiography needle (Cook Europe, Bjaeverskov, Denmark) and the cyst contents were rapidly aspirated. In step 2, the cyst cavity was nearly filled with sterile hypertonic (20%) saline, which was left in the cavity for 20 min. In step 3, the cyst was aspirated completely and the cavity was irrigated and left partially filled with sterile normal (0.9%) saline to minimize exudation or bleeding from the pericyst. During aspiration and irrigation, the separation of endocyst from the pericyst was observed as an echogeneic structure floating in the cyst cavity on real-time ultrasonography. The cystic fluid was subjected to cytological and microbiological examination. Cyst fluid at the initial aspiration was clear, watery, sterile on culture and contained hooklets and scolices of *E. granulosus* on microscopy. The scolices were motile and viable on dye test.[17] The fluid at repeat aspiration was opalescent, sterile on culture and contained scolices and hooklets. The scolices were non-motile and non-viable on dye test. Cyst communication with the biliary tract was excluded because the biochemical analysis of cystic fluid aspirated before and after injection of hypertonic saline did not show bilirubin. During and after the procedure the patient remained well and had no evidence of anaphylaxis, laryngeal oedema, bronchial spasm or skin rash. Albendzole therapy was continued for 8 weeks. The patient was discharged from the hospital on the 4th day after the procedure and was followed up monthly. The patient had dramatic clinical improvement following PD of the cyst. The itching disappeared and stools became yellow. The jaundice subsided and he was anicteric on the next hospital visit at 1 month. The hepatomegaly had regressed and the gall-bladder lump was no longer palpable at the 1-month follow up. Serial assessment revealed improvement in liver function tests and regression in the diameters of bile ducts and gall-bladder and a progressive fall in the titres of hydatid serology, both IgG and IgM (Fig. 2). Ultrasonography at 1 month follow up revealed appearance of high-level internal echoes in the cyst cavity. At 3 months the cyst had a heterogeneous echo pattern and at 4 months the cyst became uniformly echogeneic (pseudotumour echo pattern). The cyst was not visible on ultrasonographic examination at 6 months of follow up. A PTC was repeated at 3 months after PD and revealed normal bile duct diameters and free flow of contrast into the duodenum. Serial clinical, sonographic and serological examination have been performed up to 35 months after the PD. The patient is asymptomatic, has a normal biliary tree at ultrasonographic examination and negative hydatid serology. There is no evidence of recurrence or dissemination of the cyst in the peritoneum or other organs.

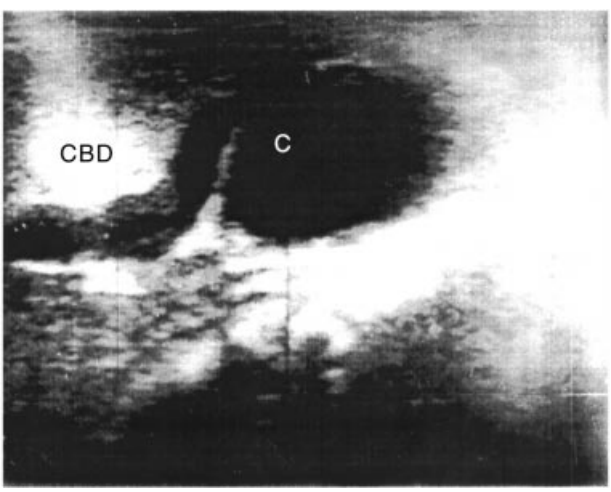

Figure 1 Right subcostal view of the pancreas. Composites of two real-time images showing a dilated (1.9 cm) common bile duct (CBD). The lower end of the duct was displaced and lifted by an anechoeic rounded structure (4.7 × 4.7 cm) with well-defined margins (C). The scan shows posterior acoustic enhancement suggestive of a cyst.

DISCUSSION

Hydatid disease has a worldwide distribution and is endemic in cattle-raising regions such as Mediterranean countries, Australia, New Zealand, the Middle and Far East, South America and South Asia. Immigration has led to the increased prevalence of the disease in Europe and North America.[18] Although practically any organ can be the site of a primary hydatid cyst, the pancreas is a portion of the gastrointestinal system where it is rarely found in humans. Data taken from the literature

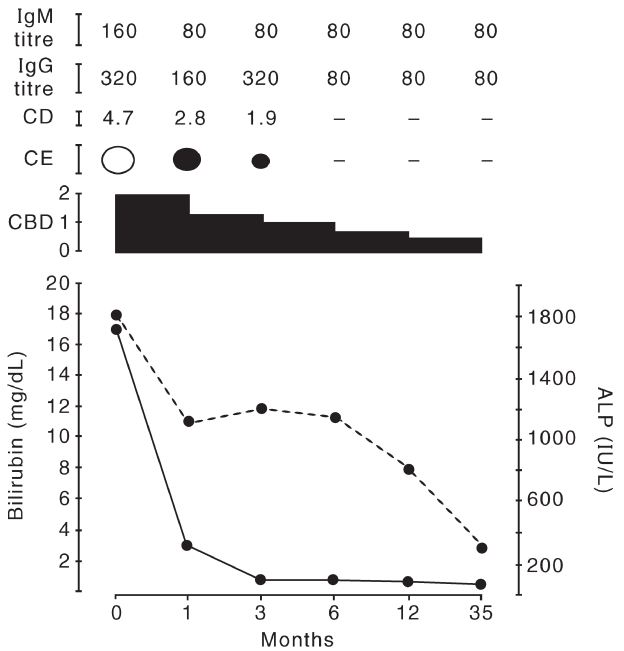

Figure 2 The biochemical, sonographical and serological course of the patient following percutaneous drainage. CBD, common bile duct; CD, cyst diameter; CE, cyst echo pattern. ALP, alkaline phosphatase (---). (—) Bilirubin levels.

quote the occurrence of pancreatic hydatid cysts at 0.19–2%.[2–4] The hexacanth embryo that becomes embedded in the pancreatic tissue forms an adventitia by the pressure it causes while growing and expanding. This adventitia is then encircled by an area of chronic inflammation of the pancreatic tissue. This mutual neighbouring of the surrounding inflamed pancreatic tissue, mainly the vasculature and the pancreatic secretory canals, the adventitia and the germinative membrane of the cyst, gives rise to concern as to how to deal with this disease by surgical means.

Biliary disease secondary to a hydatid cyst include: (i) rupture of the HHC into the biliary tree; (ii) extrinsic compression of bile ducts by a centrally placed HHC; (iii) sclerosing cholangitis, a well-established complication of surgery for HHC,· is due to the leakage of the scolicidal agents into the biliary tree;[19] or (iv) extrinsic compression of the intrapancreatic portion of the bile duct by a pancreatic head hydatid cyst, as in the case reported herein. The latter is a very rare clinical entity and, to date, only a few cases of hydatid cyst in the head of pancreas presenting with obstructive jaundice have been reported in the literature.[2,5–7]

The conventional treatment for hydatid cysts in all organs is surgical. Depending on the organs affected, the state of the development of the cyst or complications, a number of surgical techniques are available with pericystectomy being the technique of choice. The latter, however, may cause haemorrhage owing to close adherence of the outer cyst membrane to the parenchyma of the affected organ. In addition, the location of the cyst near major biliary or vascular structures may prevent complete pericystectomy and force surgeons to resort to partial pericystectomy or deroofing of the cyst. These techniques, however, expose the patient to the risk of recurrent disease. Furthermore, in some patients (elderly, surgical high risk or those experiencing relapses of disease), surgery is not advisable. Moreover, surgery has an attendant risk of mortality, significant morbidity and prolonged hospital stay.[20] Recently a number of reports have appeared in the literature in which hydatid cysts of the liver and kidney have been successfully treated by ultrasound-guided PD using hypertonic (20%) saline or absolute alcohol as scolicidal agents.[9–16] No immediate life-threatening complication, like anaphylaxis, has been encountered. In view of encouraging results of PD with albendazole chemoprophylaxis in the treatment of HHC, the unique location of the cyst in the pancreatic head with no biliary or pancreatic ductal communication and having mastered the procedure over the years, we attempted successfully to manage this cyst by this technique to assist pancreatic preservation, decrease morbidity and shorten the hospital stay. To our satisfaction, the cyst gradually decreased in size, gave a heterogeneous echo pattern at 3 months and disappeared at 6 months follow-up. The biliary compression and obstruction was relieved as evidenced by clinical, sonographical and cholangiographical findings on follow up. To date, the patient has completed 35 months follow up and the patient has no symptoms related to biliary or pancreatic disease. We took the risk of peritoneal fluid spillage by approaching the cyst through the direct transperitoneal route. Spillage of the hydatid fluid into the peritoneal cavity with slow formation of new cysts is more difficult to assess in view of the slow cyst growth. However, the use of the fine needles and catheters, advances in sonography and computed tomography techniques and sudden decompression of the fluid collections make the chances of spillage extremely low.[13] To cover the risk of the spillage and secondary peritoneal seeding, a combination of PD with medical chemoprophylaxis using praziquantel and/or albendazole before and after PD have been reported to be potentially advantageous and reasonably safe.[12–16] In a randomized, controlled trial of efficacy of albendazole in intra-abdominal hydatid disease,[21] 18 patients received no albendazole treatment (controls), 18 received albendazole (10 mg/kg daily) for 1 month (group A) and 19 received the drug for 3 months (group B). All patients underwent surgery on completion. Parasite studies of surgically removed cysts investigated protoscolex viability, whether cysts developed after intraperitoneal inoculation in mice and changes in the germinal layer of the cysts. Eight (50%) of the cysts in the control group, 13 (72%) in group A and 16 (94%) in group B were non-viable ($P=0.015$). Treatment was also significantly associated with total cyst membrane disintegration. Protoscolex and cyst viability were significantly ($P=0.039$ and 0.018, respectively) lower in treated patients than in controls. The message is important and clear that albendazole prophylaxis is justified to prevent or reduce the risk of development of secondary hydatid cysts after spillage. With such experience, the present patient was treated with albendazole, 400 mg daily, for 10 days prior to PD and the therapy was continued for 8 weeks thereafter. Albendazole

(400 mg) was administered 4 h prior to the cyst puncture so that maximal drug levels were attained at the time of possible spillage.

Management of cysts packed with daughter cysts (multivesicular cysts) could present problems. The scolicidal agent may not penetrate septations or the walls of the daughter cysts and they may remain potentially infective. However, in the Sheri-Kashmir Insitute of Medical Sciences, we puncture and treat large vesicles separately. If the vesicles are small and multiple, we puncture as many vesicles as possible during PD. Thereafter, hypertonic (20%) saline was very effective in rupturing the remaining vesicles. Long-term follow up has shown that there was no persistence of secondary vesicles in multivesicular cysts.[13,15] This experience is limited only to PD of HHC where the transhepatic route is used for aspiration.[13] At present we will not recommend PD of hydatid cysts with complex morphological appearance where the transperitoneal route is used for aspiration.

We conclude that: (i) hydatid cyst should be a differential diagnosis in cystic lesions of the pancreas presenting with obstructive jaundice; and (ii) PD and hypertonic saline injection for treatment of purely cystic hydatid disease of the pancreas may be an alternative to surgical treatment.

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