

Rupture of a Hydatid Cyst into the Bile Duct

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ABSTRACT

Cholestasis secondary to a cystobiliary communication is a rare complication associated with hepatic hydatidosis. The most established surgical procedure is the evacuation of the contents of the cyst (daughter cysts) without spills, sterilization of the cyst cavity with scolicide agents to prevent the dissemination of the hydatids to the peritoneal cavity, and cavity management (capitonnage) together with the closing of the communication.

Key words: Liver hydatid cyst, biliary duct obstruction, cystobiliary communication

INTRODUCTION

Hydatid cysts (HCs) are the result of an infestation by the larval stage of the mediterranean tapeworm *Echinococcus granulosus*, a parasite that most commonly affects the liver (humans are accidental intermediate hosts). It is often an incidental diagnosis in patients for whom an ultrasound study is performed for other reasons. However, HC may have several symptomatic manifestations as cystobiliary communications, after rupture of a cyst into the bile duct. Small communications usually not only remain asymptomatic but also may manifest by the presence of a bile leak, whereas major communications usually cause obstructive biliary obstruction, jaundice, and secondary cholangitis.^[1,2]

There is no standardized treatment; hence, it should be done according to the World Health Organization- Informal Working Group on Echinococcosis classification. A rare case of cholestasis secondary to cystobiliary communication in hepatic hydatidosis is presented.

CLINICAL CASE DESCRIPTION

A 53-year-old male was presented with a history of hepatitis C virus treated in 2005 with a sustained viral response and a 3 cm

left hepatic cyst diagnosed in 1994 without follow-up, consulted after suffering abdominal pain, nausea, choluria, acholia, and distress during the previous week. There was no history of fever, vomiting, or trauma. Physical examination revealed discrete mucocutaneous jaundice and a large cystic swelling occupying the upper abdomen, mainly right hypochondrium, measuring 12 cm × 7 cm. Laboratory investigations and liver function tests revealed a pattern of cholestasis (total serum bilirubin of 3 mg/dl [direct bilirubin 2.4]), aspartate transaminase of 83 mg/dl, alanine aminotransferase of 169 mg/dl, gamma-glutamyl transpeptidase of 288 mg/dl, raised alkaline phosphatase of 589 mg/dl (normal up to 117), and leukocytosis with striking eosinophilia (44%).

Abdominal ultrasonography showed a large cystic lesion in the left hepatic lobe with multiple cysts within suggestive of hepatic HC, with secondary compression at the confluence of the left and right biliary radicals. Computerized tomography (CT) of the abdomen showed a thin-walled cystic lesion measuring 8 cm × 7 cm × 12 cm with multiple daughter cysts within. The cyst was seen to involve a part of the left lobe. The common bile duct was normal [Figures 1 and 2]. Magnetic resonance cholangiopancreatography revealed a liver of normal size and morphology with a lobulated multicystic lesion affecting the left lobe, 8.6 × 6.8 × 12.6 cm (AP × T × L)

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Figure 1: Abdominal CT, sagittal section, showing a multicystic lesion in the left hepatic lobe



Figure 2: Abdominal CT, sagittal section, showing the lesion in contact with the gallbladder

showing a homogeneous and low signal in T1, high in T2, with gross mural calcifications in its upper margin, smooth wall and septa, no nodules and no significant enhancement of the cysts after intravenous contrast administration. The cystic lesion causes compression at the level of the confluence of left and right biliary radicals with normal-caliber common bile duct [Figures 3 and 4].

Hydatid serology was positive by enzyme-linked immunosorbent assay. The patient was started on albendazole therapy (400 mg bd), but after 4 weeks from the initial presentation, jaundice became more severe and surgery was scheduled.

The patient underwent open surgical exploration. Intraoperative, a large cystic lesion was seen to be arising from the liver anteriorly involving the II, III, and IV liver segments and compressing the vascular structures. A hydatid cyst-biliary fistula was identified by bile leak from the

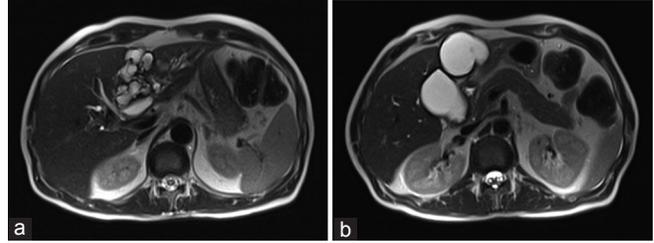


Figure 3: (a and b) MR cholangiopancreatography, sagittal sections - Multicystic lesion in the left hepatic lobe with gross wall calcifications and smooth septa. Absence of significant enhancement of the cysts after contrast administration

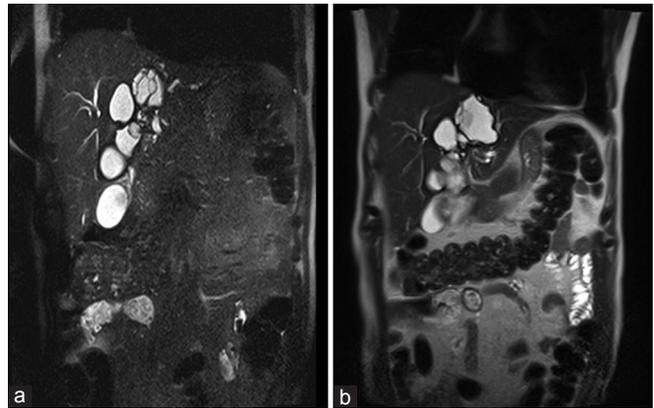


Figure 4: (a and b) MR cholangiopancreatography, coronal sections - The cystic lesion causes compression in the confluence of left and right biliary radicals with normal-caliber common bile duct

cyst wall. An intraoperative cholangiography confirmed the communication, together with the presence of hydatid vesicles in the distal common bile duct. The patient underwent cholecystectomy, deroofting of the cyst, and evacuation of cyst contents with closure of the hepatic tree communication using 3-0 polyglactin. Hypertonic saline along with povidone-iodine was used as the scolicalid agent. A sutured omentopexy and intracavitary drain placement were performed. The drain output changed to bilious 150–200 ml/day from the 3rd day and increased to 400–600 ml/day in the next 17 days. Spontaneously, the bile output decreased over the following 1-week, so intracavitary drain was then removed. The patient discharged and is presently asymptomatic in follow-up.

DISCUSSION AND COMMENTS

Communication between the HC and the biliary duct varies from an occult communication to frank intrabiliary rupture. The incidence of cystobiliary communication varies from 5% to 25% of patients.^[1]

A majority of minor communications remains asymptomatic and manifests postoperatively by the presence of a bile leak while major communications, as the current case presents

with obstructive jaundice or cholangitis. HC growth causes displacement, distortion, and stenosis of the biliary tree.

The development of a cystobiliary communication is favored by continuous compression in the hepatic duct, which causes it to become atrophic and susceptible to leakage.^[3] This biliary communication can be suspected preoperatively by anamnesis, analytical, or radiological examinations.^[4] Imaging techniques such as echography and CT of the abdomen are the prime examination, accompanied by magnetic resonance imaging in cases of jaundice. As in our case, large HC located medially and near the hepatic hilum are more inclined to suffer a cystobiliary fistula.^[1] The presence of a cystobiliary communication in a HC prevents the percutaneous approach or *Percutaneous drainage of echinococcal cysts* (puncture, aspiration, injection, and respiration technique) as a treatment option.

Surgery is consequently the best therapeutic option, which may be radical or conservative depending on the location and anatomical relationships of the cyst. Before the intervention, it is important to start treatment with benzimidazoles that will be maintained 3–6 months after surgery. During the

intervention, it is also essential to isolate the cyst as well as using scolicide agents to prevent the dissemination of the hydatids to the peritoneal cavity.

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