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Case Report: Hepatic Hydatid Cyst Revealed by an Exceptional Mode of Presentation

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Abstract **Case Report**

Hydatid cyst (HC) is a benign parasitic disease that predominantly affects the liver. It is relatively common in countries around the Mediterranean basin; however, it remains an exceptional cause of portal hypertension. We report a new case of a patient with a hepatic hydatid cyst revealed by a portal hypertension syndrome, highlighting its diagnostic and therapeutic particularities, along with a review of the literature.

Keywords: Digestive hemorrhage, portal hypertension, hepatic hydatid cyst.

INTRODUCTION

Hydatid cyst is a benign parasitic disease that predominantly affects the liver. It is relatively common in countries around the Mediterranean basin; however, it remains an exceptional cause of portal hypertension. We report a new case of a patient presenting with a hepatic hydatid cyst revealed by a clinical and ultrasonographic syndrome of portal hypertension, together with a review of the literature.

CASE PRESENTATION

This was a 40-year-old man from a rural area of Ksar El Kebir who presented with chronic right hypochondrial pain for 4 months, of moderate intensity, intermittent in course, not related to meals, associated with low-volume hematemesis and intermittent food vomiting, without bowel transit disorders or abdominal distension, evolving in a context of unquantified weight loss and absence of fever.

Clinical examination revealed dysarthria, cutaneomucosal pallor, and stable hemodynamic status, while abdominal examination showed clinical signs of portal hypertension (collateral venous circulation, splenomegaly) and a right upper quadrant abdominal mass extending to the right iliac fossa, with moderate tenderness in the right hypochondrium, without Murphy's sign or signs of hepatocellular failure; digital rectal examination showed no melena.

Laboratory investigations showed normochromic normocytic anemia at 11.7 g/dL, platelet count of 250,000/mm3, and white blood cell count of 6,560/mm3. Liver transaminases were normal, with no biological cholestasis.

Upper gastrointestinal endoscopy revealed stage 2 esophageal varices without red signs. *(figure 1)*

Abdominal ultrasound revealed a cystic lesion projected in the epigastric region, intersplenorenal collateral venous circulation, and hepatosplenomegaly. This was complemented by abdominal computed tomography, which demonstrated a large oval calcified cystic lesion in segment I measuring 71 × 90 mm, exerting a mass effect and compressing the hepatic pedicle, with dilatation of the splenomesenteric trunk and presence of periportal, perisplenic, and periesophageal varices with secondary portal hypertension, without dilatation of the intrahepatic bile ducts *(figures 2 and 3)*.

Figure 1: Endoscopic appearance on upper gastrointestinal endoscopy: stage 2 esophageal varices without red signs

Figure 2: Ultrasonographic appearance of a large hydatid cyst of segment I of the liver, with ultrasonographic signs of portal hypertension

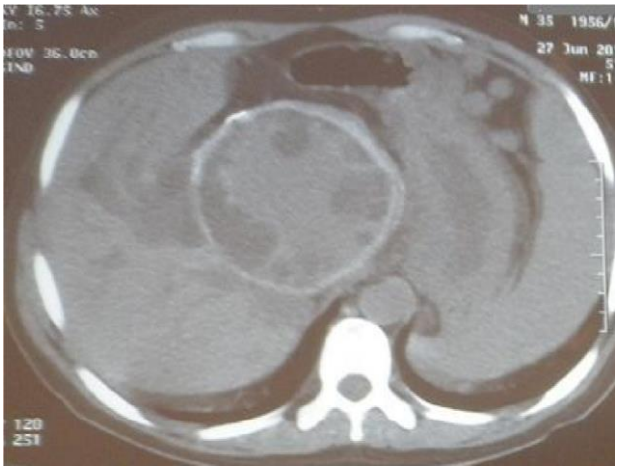

Figure 3: CT-scan appearance of a large hydatid cyst in segment I of the liver

Hydatid serology was positive. Hepatitis B and C viral serologies were negative. The patient was referred to visceral surgery for operative management. The patient was started on albendazole before surgery and was expected to resume it after surgery.

DISCUSSION

Hepatic hydatid cyst is a parasitic disease caused by the intrahepatic development of *Echinococcus granulosus*. It remains frequent and constitutes a public health problem in highly endemic countries such as those of the Mediterranean basin and North Africa. [1,2]

In rare cases, hepatic hydatid cyst is revealed by a vascular complication such as portal hypertension, as illustrated in our case. This complication is explained by compression of intrahepatic or perihepatic vascular structures (portal vein, its branches, inferior vena cava, hepatic veins) under the pressure exerted by the cyst during its expansion in the absence of treatment. To our knowledge, about ten cases revealed by portal hypertension have been described in the literature since the publication of the first case in the 1970s. [6,8] Compression is most often asymptomatic and well tolerated. The clinical presentation is variable and depends mainly on the size and location of the hepatic hydatid cyst [3]. It is exceptionally revealed by digestive hemorrhage due to rupture of an esophageal varix, as reported in the literature, and this complication is often associated with compression of the bile ducts. Hepatocellular insufficiency is generally mild, but it depends mainly on the duration of progression and whether bilioportal obstruction is relieved. Upper gastrointestinal endoscopy makes it possible to diagnose esophageal or gastric varices and to perform endoscopic hemostatic treatment in the event of bleeding while awaiting etiological treatment, which is based on surgical relief of the obstruction. A classification of portal hypertension of hydatid origin was proposed by Bourgeon *et al.,* in the 1980s; it includes 4 stages, but it is not used. [5,7]

Diagnosis is based on imaging. Ultrasound coupled with Doppler is the examination of choice; it establishes the diagnosis of hydatid cyst in most cases by specifying the location, cyst contents, number of cysts, and intrahepatic vascular and biliary relationships, and by establishing the Gharbi classification. Doppler ultrasound makes it possible to assess the relationship of the hydatid cyst with the vascular axes (portal vein, hepatic veins, inferior vena cava) and to demonstrate, depending on the level of compression, direct or indirect signs of portal hypertension, and sometimes portal cavernoma or Budd-Chiari syndrome. CT scan is not essential for diagnosis, but it is indicated in cases of diagnostic difficulty on ultrasound, especially in large central cysts or type IV cysts. Magnetic resonance imaging does not appear to provide any advantage. Hydatid serology is inconsistently positive [3,4].

Treatment is medical and surgical, and its objective is to remove the obstruction caused by the hydatid cyst by resection of the protruding dome or pericystectomy. Surgical management should be accompanied by albendazole starting four days before the procedure and continued for up to four weeks afterward to prevent hydatid dissemination and recurrence. The prognosis remains good, but the reversibility of portal hypertension after treatment of the hydatid cyst is still uncertain. [2,8]

CONCLUSION

Hydatid disease remains frequent and benign; however, vascular complications are rare and severe. Ultrasound combined with CT scan plays an important role in the diagnosis of these vascular complications by showing their point of impact and by establishing a precise functional vascular map. Portal hypertension due to a hepatic hydatid cyst appears to have a better prognosis according to the reported cases.

Conflicts of interest: The authors declare no conflicts of interest.

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