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Case Report

Hydatid cyst of the liver invading the inferior vena cava ☆

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ABSTRACT

Hydatid disease is a parasitic infestation due to the development of Echinococcus granulosus in the organism. This disease is particularly frequent in Morocco where echinococcosis is endemic. The liver is the most common organ to be affected by hydatidosis, and several complications have been described. Vascular complications secondary to hepatic echinococcosis such as fistulization or rupture of hydatid liver cysts to the inferior vena cava (IVC) are an extremely rare and life-threatening condition. This report aims to describe a case of invasion of the IVC by a hydatid cyst of the liver resulting in portal hypertension in a 60-year-old female patient. The diagnosis was established in the preoperative phase by a CT scan. IVC invasion remains an infrequent complication that should be routinely looked for in patients with hydatid disease of the liver, and few cases have been reported in the literature to date.

Introduction

Hydatid disease, also known as Echinococcosis, is a zoonotic disease caused by the echinococcus parasite, which belongs to the family of Taeniidaes [1]. Dogs, wolves, and coyotes are the primary hosts, whereas sheep, cattle, and deer are the intermediate hosts. Transmission to humans happens when they consume contaminated food or water or come into close contact with their animal hosts. Although the liver (60%-70%) and lungs (20%-30%) are the most frequently affected organs [2]. It may develop in almost any part of the body, Although the liver (60%-70%), and lungs (20-30%) are the most usually affected organs [2]; It arises in other abdominal organs in 10% of cases [3]. Hydatid cysts frequently compress the inferior vena cava, however, fistulization or rupture of hydatid liver cysts to the inferior vena cava (IVC) is a very unusual and potentially fatal disease [4,5]. This report aims to describe a case of invasion of the IVC by a hydatid cyst of the liver resulting in portal hypertension in a 60-year-old female patient. The diagnosis was established in the preoperative phase by a CT scan. IVC invasion remains an infrequent complication that should be routinely looked for in patients with hydatid disease of the liver, and few cases have been reported in the literature to date [5].

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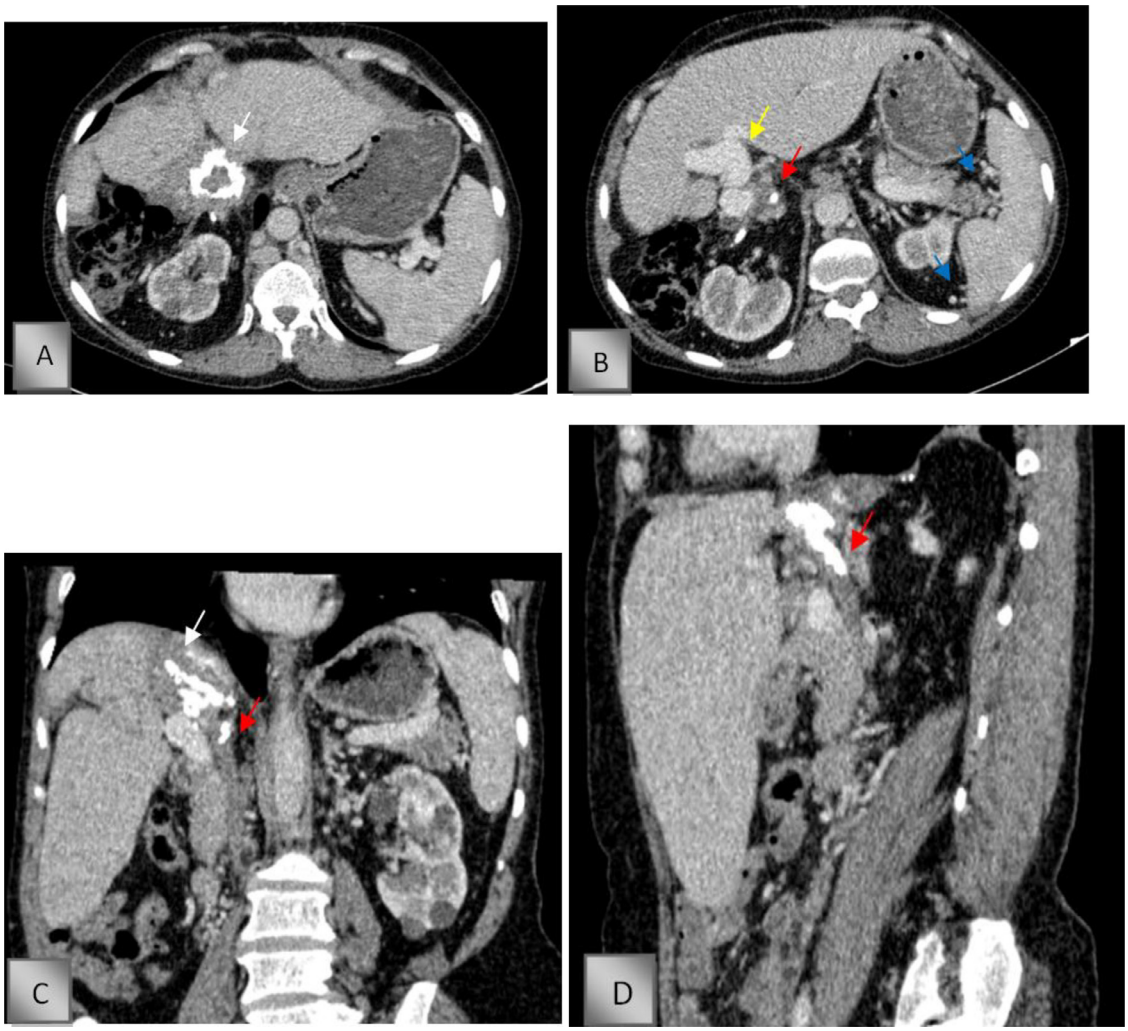

Fig. 1 – (A) Abdominal contrast-enhanced CT scan revealed a 4.6 cm mass in the left lobe of the liver with a thick calcified wall (A, C: White arrow). It lay immediately adjacent to the IVC, with individualization of calcified material within its lumen (B, C, and D: Red arrow). The retrohepatic and sus hepatic segment of the IVC was not totally filled with contrast agents. Dilatation of the portal trunk and right portal branch (B: Yellow arrow) with splenorenal shunts (A and B: Blue arrow), related to portal hypertension.

Case report

We report the case of a 60-year-old otherwise healthy female patient, hailing from a rural area, without significant history, who presented with 2 years' history of abdominal pain and a gradually increasing lump in the right hypochondrium. The vitals of the patient were stable. Examination of the head, neck, chest, and cardiovascular systems were normal. Abdominal examination revealed hepatomegaly, with a regular, insensitive surface. No splenomegaly was detected. There was no pedal edema or ascites. Her laboratory investigations revealed hemoglobin at 10.3 g/dL (normal value: 14-16 g/dL); and eosinophilia at 800 μL (normal value: 100-400 μL). Serum bilirubin 9 mg/L (normal value: $<$ 12 mg/L), serum aspartate aminotransaminase 56 IU/L (normal value: $<$ 38 UI/L), serum alanine aminotransaminase 80 IU/L (normal value$<$ 41 UI/L), alkaline phosphatase 475 IU/L (normal value: $<$ 270 UI/L), and prothrombin time at 75%. Upper gastrointestinal endoscopy was normal, and varices were not seen. Hydatid serology was positive.

The patient was referred to our department for a CT scan (Fig. 1), which revealed a 4.6 cm mass in the left lobe of the liver with a thick calcified wall, According to the Gharbi classification, the cyst was type V. It lay immediately adjacent to the IVC, with individualization of calcified material within its lumen. The retrohepatic and sus hepatic segment of the IVC was not completely opacified. This finding suggested a direct extension into the lumen of the vessel. Dilatation of the portal trunk and right portal branch with splenorenal shunts, as well as dilatation of the azygos venous system, related to portal hypertension. There was no hydatid cyst in the lung parenchyma.

Curative anticoagulation was initiated. Accounting for the risk of dissemination of daughter cysts and pulmonary embolization, surgery was considered, but the patient refused

it. The patient was put on medical treatment with antihelminthics antihelminthics (Zentel 400 mg daily, 3 weeks out of 4). The evolution was favorable, with the same appearance of the hydatid cyst of the liver, and the IVC persisting on the follow-up CT scan 1 year later.

Discussion

The hydatid cyst of the liver is a cosmopolitan disease that is endemic in developing countries and represents a serious public health problem in Morocco [6]. The most frequent complications, which occur in 40% of cases, are infectious, biliary, and thoracic ones [4]. Vascular complications are extremely rare. They include rupture into the large vessels (portal branch or IVC) and portal hypertension secondary to portal flow obstruction by an infra-, intra- or suprahepatic obstacle [7]. Spontaneous rupture of the hepatic hydatid cyst into the IVC is very rare, with only a few cases reported in the literature [3,4,6]. The richness of the vascular wall in elastic fibers explains its compliance potential, hence the rarity of opening openings in large vessels [8,9], unlike bile ducts, whose rigid wall easily predisposes to rupture [9].

The main symptomatology is pulmonary embolism, which is rarely described and most often discovered at autopsy [13–15]. Intraoperative and postmortem data indicate that embolism is due to purely mechanical obstruction by the cyst and daughter vesicles. There is no evidence of associated caillot or thrombosis [13,15]. In theory, hemorrhagic shock due to spontaneous rupture of the hydatid cyst of the liver into the IVC should be the main complication. described in the literature, and this can probably be explained by the fact that these patients die before arriving at the hospital.

Radiologically, a CT scan remains the imaging method of choice to assess the vascular links of the hepatic hydatid cyst especially with the IVC [5]. The rupture of the IVC concerns generally hydatid cysts located in the posterior segments of the liver (VII, VIII, and I), and in contact with the IVC. The latter may be compressed or laminated by the cyst, and sometimes it can be partially or totally thrombosed due to the presence of vesicles in its lumen [4]. Doppler ultrasound and MRI angiography confirm the presence of cystic lesions in the retrohepatic vena cava and pulmonary artery [6].

The ruptured hydatid cyst of the liver in the IVC is treated surgically. Two main complications are to be feared intraoperatively: massive intraoperative pulmonary embolism and hemorrhagic shock [6]. The rupture of a liver hydatid cyst in a vessel creates communication between cavities with different pressure regimes. The resulting gradient of cystovascular pressure is significant [9,10], and the vascular fissure is blocked by the hydatid attached to the adventitia. Preoperatively, after evacuation of the cystic contents, the prodigal membrane leaves the breach Intracystic bleeding and sudden drop in blood pressure are reported in the literature [9,11]. Other mechanisms have also been suggested to explain the appearance of the fissure. The mobilization of the liver in the presence of a fissured ICV leads to the migration of daughter vesicles into the systemic circulation, which may be responsible for massive and fatal pulmonary embolism [9,12]. Hence the need for early control of the vascular axes, with cautious mobilization of the liver to avoid the unfortunate consequences of such an incident. Medical treatment is indicated in cases of chronic embolism, extensive dissemination of daughter vesicles involving the entire pulmonary arterial trunk, or when surgery is contraindicated [13,15]. In our case, the CT scan enabled us to establish the diagnosis preoperatively, and given the significant risk of the procedure and the patient's rejection of surgery, the decision was made to abstain from surgery, and put on medical treatment, with regular monitoring for complications.

Conclusions

The rupture of the hydatid cyst of the liver into the VCI is a rare and serious complication. CT scan is the examination of choice for diagnosing this complication. Pulmonary hydatid embolism remains the most frequent complication. Treatment of ruptured hydatid cyst of the liver in the IVC is surgical and must be carried out under vascular control. The prognosis is reserved.

Author's contributions

All authors contributed to this work. All authors have read and approved the final version of the manuscript.

Patient consent

Written informed consent for publication was obtained from patient.

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