

Case Report

Pulmonary Embolism Due to Liver Hydatid Cyst in Children: Follow Up Over 15 Years

Hasni Bouraoui I^{1*}, Sanogo K¹, Beji O², Tarmiz A³ and Jemni H¹

¹Imaging Department, Sahloul Hospital, Tunisia

²Intensive Care Unit, Sahloul Hospital, Tunisia

³Department of Cardiac Surgery, Sahloul Hospital, Tunisia

*Corresponding author

Imaging Department, Sahloul Hospital, Sousse, Tunisia,
Tel: 0021-6986-439-07; Email: ibtissehasni@yahoo.fr

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- Hydatid
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Abstract

We report a case of hepatic hydatid cyst ruptured in the inferior vena cava (IVC) with pulmonary embolism in a child. We detail his disease story on a follow-up of 15 years (2002-2017).

ABBREVIATIONS

HPE: Hydatid Pulmonary Embolism; IVC: Inferior Vena Cava; CT: Computed Tomography; MRI: Magnetic Resonance Imaging

INTRODUCTION

Hydatid pulmonary embolism (HPE) is a rare clinical entity. It may complicate hepatic or cardiac hydatidosis and can evolve in different modes: superacute, subacute or chronic over a number of years. The progression of the disease may cause symptoms like dyspnea, hemoptysis, and chest pain, and anaphylactic shock may develop due to leakage of the hydatid cyst fluid [1,2]. We report here the case of a 19-year-old patient with hepatic hydatid cysts complicated with rupture in the inferior vena cava (IVC) with HPE when he was 11 years old. Eight years later, the pulmonary hydatid disease becomes chronic and involves the vital prognosis.

CASE PRESENTATION

The disease history began in 2002 when the patient was 4 years old; he was operated on a hydatid cyst of the left liver and supposedly cured. In April 2009, the child was asymptomatic but imaging control; Sonography discovered the recurrence of three hepatic hydatid cysts. Computed tomography (CT) scan showed a univesicular cyst and a multivesicular one in the left liver and the third one comes into contact with the IVC without fistula (Figure 1). The cardiac Doppler ultrasound did not find intra-cardiac cysts but an extrinsic compression of the right atrium and the IVC by the hepatic cyst. A few days before the scheduled surgery, he presented fever, chest pain, and dyspnea evolving for 5 days. The hemodynamic state was stable.

Emergency thoracic CT scan showed an endoluminal defect of the right pulmonary artery and multiples daughter vesicles in the IVC secondary to the spread of hydatid cyst of the liver (Figure 2). The cardiac Doppler ultrasound showed dilation of the

right heart cavities without intra-cardiac cysts. The patient was operated on an emergency basis. The operation was performed through a median sternotomy incision under extracorporeal circulation. There was no cyst in cardiac cavities. Many daughter vesicles were extracted from IVC Ostia and only one from the pulmonary right artery after arteriotomy. The fistula between the hydatid cyst and the IVC was repaired.

Others liver cysts were treated 3months later. The child was regularly monitored, after one year of treatment with albendazole, we reviewed the child, and he had a chronic cough and insufficient growth. The chest x-ray of control showed bilateral parenchymal nodules of variable size and thoracic CT demonstrated multiple bilateral parenchymal hydatid cysts and chronic pulmonary emboli (Figure 3). The hydatid disease of the lungs has become chronic with the growth of countless pulmonary hydatid cysts. Several episodes of bacterial infections were treated with antibiotics (Figure 4). Until January 2017, the hydatid cyst of the liver did not recidivate. Pulmonary surgery is no longer indicated due to advanced disease and the patient is currently followed by a cardiologist.



Figure 1 April 2009, Thoracic computed tomography showed in A: hepatic cysts, univesicular 7 cm in diameter (asterisk) and multivesicular cyst (white arrows), and in B: multivesicular hepatic cyst adjacent to the IVC (black arrow).



Figure 2 June 2009, Coronal (A), axial(B) thoracic CT scan showing complete occlusion of pulmonary right artery by a mass with fluid density (white arrow) and hepatic cyst expanding into the IVC (black arrow).

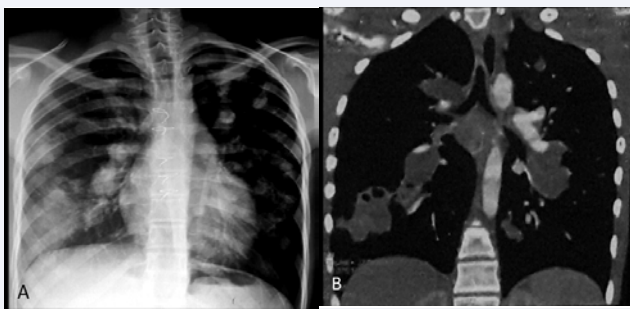


Figure 3 June 2015, Radiography of the chest(A) showing multiple nodules in the left and the right lung, coronal thoracic CT scan(B) showing multiple bilateral parenchymal hydatid cysts and chronic pulmonary emboli.

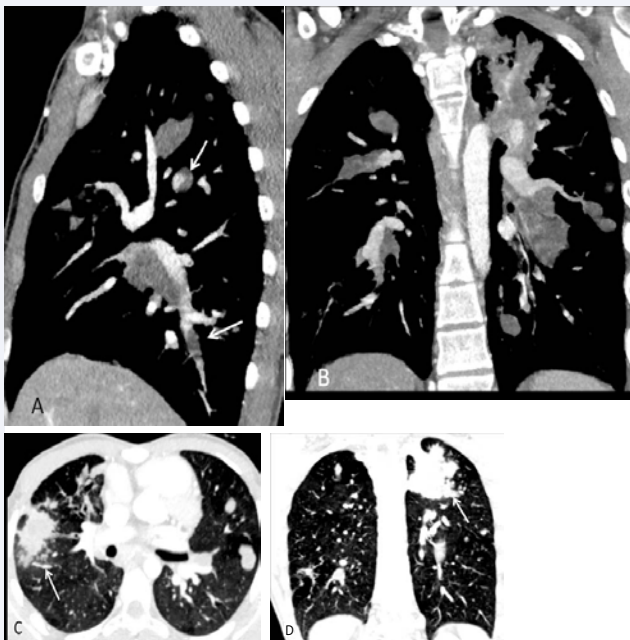


Figure 4 January 2017, Sagittal (A), coronal thoracic CT scan (B) showing persistence of multiple bilateral parenchymal hydatid cysts and chronic pulmonary proximal and distal emboli. Note in parenchymal window (C) and (D) bilateral pulmonary condensation secondary to cysts infection (arrows).

DISCUSSION

HPE is a rare entity and a serious complication of rupture of a hepatic hydatid cyst in the IVC or a consequence of the rupture of cardiac hydatid cysts [1]. This complication is lethal in 50% of cases [3,4]. Different clinical signs arise depending on the size and number of emboli. HPE may be superacute and lethal through anaphylactic shock or massive pulmonary embolism confirmed at autopsy. It may be subacute evolving within a few months towards cardiac failure and death. HPE can become chronic over a number of years with a progressive appearance of pulmonary hypertension and post-embolism hydatid pulmonary heart through repetitive migrations of microemboli. Early diagnosis with imaging studies and treatment are the main aspects of preventing complications [3,5].

Clinical manifestations of the HPE are not specific although hemoptisy is the most frequent sign [4]. In the absence of a medical history of a visceral hydatid cyst, clinicians should use all the means necessary to reach hydatid disease. The diagnostic investigation of patients with suspected HPE should involve a two-dimensional echocardiography CT scan and magnetic resonance imaging (MRI). If there is no previous history of hydatid disease its existence can be suspected by the presence of anti-echinococcal antibodies and eosinophilia in blood tests. Eosinophilia is uncommon except for cyst rupture [6]. The visualization of endoluminal cystic images does not take contrast in CT scan or sometimes hydatid membranes and the presence of hydatid cysts in contact with the IVC or hepatic veins is a more plausible argument in favor of the hydatid origin of pulmonary embolism as in our case. Ultrasonography shows relationships of the liver hydatid cyst with IVC but hardly ever shows an image of the communication [7]. MRI is the best means of asserting the cystic nature of the embolus, the cysts are in hyposignal T1 and hypersignal T2 except in the presence of cysts rich in proteins, in which case the signal will be hyper intense in T1 and T2 [3]. In our case CT scan is always sufficient for the follow-up of the disease.

Once the pulmonary vascular obstruction has been diagnosed, the surgical treatment of the hepatic cyst at the origin of the embolism must be carried out as the first step. However, mobilization of the liver may cause hydatid embolism and hemorrhagic complications when the hydatid cysts are in contact with the walls of the IVC. To prevent a fatal intraoperative pulmonary embolism a wide laparotomy to control the IVC and an extracorporeal bypass ready are recommended [8]. Surgery for a hydatid pulmonary vascular obstruction is quite similar to that for usual pulmonary embolism: embolectomy by arteriotomy for proximal pulmonary intra vascular hydatid cysts using cardiopulmonary bypass.

Chronic pulmonary arterial hypertension may worsen even after hydatid embolectomy as the vascular obstruction is also distal and associated with a granulomatous reaction and vascular fibrosis as our case [8]. Pulmonary transplantation may be a therapeutic option because immunosuppressive treatment does not adversely affect the course of hydatid disease [3,8].

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