

Pulmonary Hypertension Secondary to Cardiac Hydatid Cyst Embolism

Serdal Citil¹, Mehmet Sait Menzilcioglu², Tuna Sahin¹, Mahmut Duymus² and Ertuğrul Mavili³

¹Ministry of Health Kahramanmaras Necip Fazıl City Hospital, Division of Radiology, Kahramanmaras, Turkey

²Gazi University School of Medicine, Department of Radiology, Ankara, Turkey

³Erciyes University Hospital, Department of Radiology, Kayseri, Turkey

*Corresponding author: Sait Menzilcioglu, Department of Radiology, Gazi University, Bahçelievler, Ankara-06100, Turkey, Tel: +90-3124844928; Fax: +90-3124844929; E-mail: dr.m.sait@hotmail.com

Received date: May 06, 2015; Accepted date: June 24, 2015; Published date: June 26, 2015

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Abstract

Hydatid pulmonary embolism is an uncommon and rare complication secondary to the rupture of cardiac hydatid cyst. Cardiac hydatid cyst is a silent disease and may be diagnosed with the serious complications such as heart block, valvular obstruction, cerebral and pulmonary embolization. Pulmonary hypertension related with embolism is even rare. Radiological findings of pulmonary embolism secondary to the hydatid cyst rupture are important to prevent further potentially fatal complications. In this case report we present a case of cardiac hydatid cyst causing pulmonary hypertension secondary to pulmonary arterial embolism.

Introduction

Cardiac hydatidosis (CH) is a rare parasitic disease caused by larval forms (metacestodes) of the genus Echinococcus. It constitutes approximately 0.02-2% of all cases of human hydatidosis [1]. Hydatid pulmonary embolism is a complication of CH that generally occurs after an iatrogenic or spontaneous rupture of the right ventricular or right atrial hydatid cyst or from systemic circulation [2-5]. Before the introduction of cross-sectional imaging techniques the diagnosis was based on clinical and laboratory findings. Echocardiography is a noninvasive effective tool for the diagnosis of the CH. But it is not always suitable for the detection of pulmonary cysts. In this condition Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) fascilitates the definite diagnosis [1,6-8]. We present a patient presenting with pulmonary hypertension due to pulmonary embolism of the CH that was detected with CT.

Case Report

13-year-old girl admitted to the pediatric cardiology outpatient clinic complaining of dyspnea related with effort. Two years ago she was operated from right ventricular hydatid cyst.

On physical examination, the vital findings were as follows: blood pressure was 90/60 mmHg; cardiac rate was 64 beats/min, body temperature was 36°C and respiration was 24 breaths/min. On auscultation, there was 2/4 systolic ejection murmurs on mezocardiac area.

There was an increase in eosinophil count of 1100/mm³ (over 5%) in laboratory tests. Serologic tests of hydatid cyst were positive. Other parameters were normal. Spirometric examination revealed FVC: 85%, FEV1: 83%, FEV1/FVC: 100%, PEF: 85%.

Echocardiography was performed to demonstrate a residual cyst but no cardiac lesion was seen. Right ventricular dilatation and pulmonary hypertension were detected. Pulmonary artery pressure was 45-50 mmHg. CT examination was performed which revealed total occlusion of the right upper lobe pulmonary artery. It was filled with a hypointense cystic material, which enhanced peripherally after contrast injection (Figure 1).



Figure 1: Coronal reformatted image demonstrates two cystic dilatations in the distal part of the pulmonary vessels on the right (asterix).

Moreover multiple cystic lesions in the pulmonary arteries in different locations were seen. The lesions were unevenly distributed and they were predominantly in the periphery of the lung. The lesions were also seen in pulmonary veins (Figure 2). A cyst was also seen at the level of right upper lobe pulmonary vein draining into the left atrium. With these findings the patient was diagnosed as pulmonary embolism of CH. Since hydatid disease was disseminated surgery could not be performed and Albendazole treatment was started.



Figure 2: The lesions were also seen in pulmonary veins on Coronal reformatted image (asterix).

Discussion

Cardiac cysts constitute 0.02-2% of all hydatidosis cases [1]. Coronary circulation brings the larva to the heart. The mostly involved sites are left ventricle (60%), right ventricle (15%) and interventricular septum (9%) respectively [5]. Rupture of a right ventricular cyst may result in pulmonary embolism, which is a rare cause of PAH. The embolism can occur spontaneously or during surgical removal of the hydatid cyst [2-5]. Direct spread of a visceral cyst to the pulmonary arteries via inferior vena cava and the right cardiac chambers is another possibility. The embryos reaching the lung change into small cysts and begin to grow. When the cysts are in an intra-arterial location they finally occlude the pulmonary arteries. The growing rate is slow so pulmonary perfusion is not affected quickly. Therefore these lesions may initially be asymptomatic. Compression of vital structures or interruption of blood circulation may cause symptoms in these patients [2]. In our case, the first symptom was progressive dyspnea. Therefore a cardiac hydatid disease was suspected first. But on echocardiography PAH and enlargement of the right ventricle were seen.

Before the advent of CT and MRI clinical and laboratory tests were used to diagnose hydatid disease. Definite diagnosis using ECG and chest X-ray was not possible [6]. The Casoni skin test, Weinberg reaction and the peripheral blood eosinophil count tests were used in the diagnosis. But these tests also have low value due to the false negative results [6]. CT, MRI and echocardiography have the superior advantage of direct visualization of the emboli, pulmonary hypertension and parenchymal lesions, like in our case [1]. On CT images, non-complicated cysts are usually manifested as hypodense lesions, with possible calcification in the periphery. Only the wall of the cyst enhances with the administration of intravenous contrast agent [1-4]. Our findings were in concordance with the previously reported cases.

Hydatid cyst has a characteristic appearance on MRI. It is observed as an oval lesion that is hypointense on T1-weighted images (WI) and hyperintense on T2-WI. A peripheral hypointense ring on T2-WI represents the pericyst and is a typical finding. But it cannot always be detected [1-4]. In this case we ruled out the acute pulmonary thromboembolism and primary arterial tumors from differential diagnosis list. The acute thromboembolic disease was excluded clinically, because of the lack of predisposing conditions, and no history of deep vein thrombosis in the lower legs [3]. The clinic manifestation of primary arterial tumor is more aggressive than our case [9]. Thus, the clinical presentation may be misleading and confused with other more frequent causes of pulmonary embolization; however, a combination of the clinical and radiological features and the medical history can lead to the correct diagnosis [1]. Once diagnosis has been established CT or MRI is effective for follow-up to search for recurrences or the formation of pseudo aneurysms [2-3].

Although surgery combined with medical treatment may improve the prognosis, the treatment of this rare presentation should be individualized [2,4]. Surgical intervention can be complicated by rupture of the cyst. This rupture can cause the dissemination, anaphylactic shock, embolism, and pseudo aneurysm formation [4]. Albendazole is widely used and limited success has been reported. Rupture of a hydatid cyst of the lung during and after the cessation of albendazole treatment has been reported [10]. Therefore surgery has been advocated for intraarterial hydatid cysts. Our patient could not undergo surgery due to disseminated hydatid cyst, therefore only Albendazole treatment was given. In cases of diffuse and severe involvement of the pulmonary arteries like presented here, mortality is high [4].

Conclusion

Even it is rare, cardiac hydatidosis should be kept in mind in the assessment of pulmonary hypertension secondary to embolism. Cardiac evaluation should be included in the imaging procedure.

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