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CASE REPORT

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# Pulmonary hydatid embolism : A case report

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## ABSTRACT

Hydatidosis is a parasitic disease caused by the *Echinococcus granulosis* parasite. It is a cosmopolitan zoonosis prevalent in farming areas, particularly around the Mediterranean. Cardiovascular localization is rare, and pulmonary hydatid embolism is a rare and potentially serious complication. It results from the rupture of a hydatid cyst into the venous return circulation, often of hepatic origin, or from the direct rupture of the hydatid cyst into the right cardiac cavities. Imaging techniques, particularly thoracic CT angiography, are essential for diagnosis and to investigate other localizations of the hydatid cyst. The management of pulmonary hydatid embolism is delicate and remains poorly defined. We present a case of pulmonary hydatid embolism resulting from the rupture of a hepatic hydatid cyst into the inferior vena cava, leading to pulmonary lesions that are consistent with infarction.

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## Introduction

Hydatid disease, also known as echinococcosis, is a parasitic infection caused by the larval stage of Echinococcus granulosis. This disease is prevalent in various agricultural areas, where dogs serve as definitive hosts while humans become accidental intermediate hosts. The liver and lungs are the most commonly affected sites for hydatid cysts, although cardiovascular involvement is infrequently observed [1,2].

Pulmonary hydatid embolism is a rare yet severe complication, typically caused by the rupture of a hepatic or cardiac hydatid cyst into the venous system (including hepatic veins, inferior vena cava, or right heart chambers). Clinically, it may resemble thrombotic pulmonary embolism. Imaging is crucial for diagnosis and for demonstrating the cystic nature of the intravascular lesions.

We present a case of pulmonary hydatid embolism resulting from the rupture of a hepatic hydatid cyst into the inferior vena cava, leading to pulmonary lesions that are consistent with infarction. To the best of our knowledge, instances of pulmonary infarction in this specific situation have been described only infrequently.

## Clinical Observation

A 40-year-old female patient, who had been diagnosed with hepatic hydatid cysts a year prior, was admitted for planned surgical intervention. She did not have any significant past medical or surgical history and lacked known risk factors for venous thromboembolism, such as recent surgery, extended immobility, deep vein thrombosis, or cancer. During her hospitalization, the patient reported chest pain and dyspnea. No hemoptysis or syncope were reported. The patient had a normal blood pressure (117/83 mmHg) with a normal oxygen saturation ($SpO_2$: 96%).

On laboratory examination, the eosinophil counts were in the normal range. D-dimer was positive, with an increased value of 1258 µg/l. liver transaminases (ALAT = 114 UI/l and ASAT = 64) were elevated also. Serologic testing was positive for antibodies against cystic hydatidosis (IgG 27.4 S/ cut-off).

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The thoracic CT angiography showed multiple, well-defined cystic filling defects within the bilateral lobar and segmental branches upper and lower lobes of the pulmonary arteries, some exhibiting arterial enlargement, which is consistent with hydatid emboli (Figs. 1 and 2). In the adjacent lung tissue, ground-glass opacities and peripheral, pleural-based wedge-shaped consolidations were noted in the segmental vascular territories, suggestive of pulmonary infarction (Fig. 3).

Abdominal CT imaging indicated the presence of hepatic hydatid cysts closely adjacent to the inferior vena cava, with small cystic formations visible within the lumen of the inferior vena cava, indicating a rupture of the hepatic cyst into the venous system (Figs. 4 and 5). Complementary thoracic MRI (only a coronal T2 sequence was performed) confirmed the cystic nature of the intra-arterial lesions (Fig. 6).

Transthoracic echocardiography did not visualize any cardiac localization or signs of portal hypertension.

Due to the widespread dissemination of daughter cysts into multiple segmental branches bilaterally, surgical intervention was deemed high risk and technically unfeasible. Following a multidisciplinary meeting that included cardio-thoracic surgery, anesthesia, visceral surgery and radiology, the decision was made to forgo surgical embolectomy and instead pursue medical management alone. Albendazole was initiated at a dosage of 400 mg twice a day.

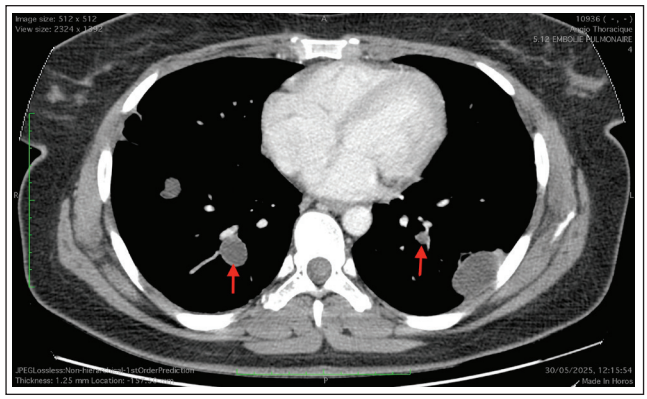


**Figure 1.** Coronal and axial CT scans showing cystic emboli in the pulmonary arteries (red arrow).

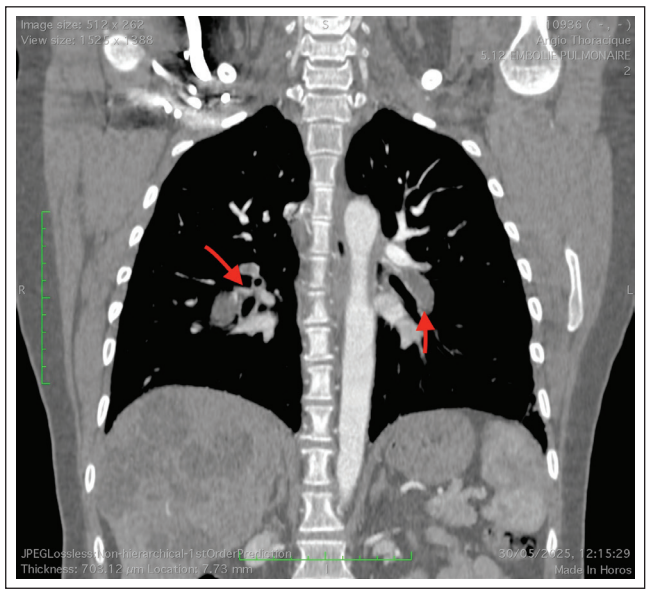


**Figure 2.** Coronal and axial CT scans showing cystic emboli in the pulmonary arteries (red arrow).

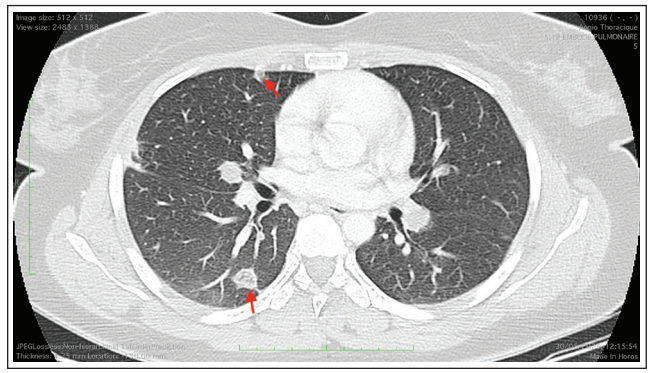


**Figure 3.** Axial CT scan illustrating a pulmonary infarction in the right lower lobe (red arrow).

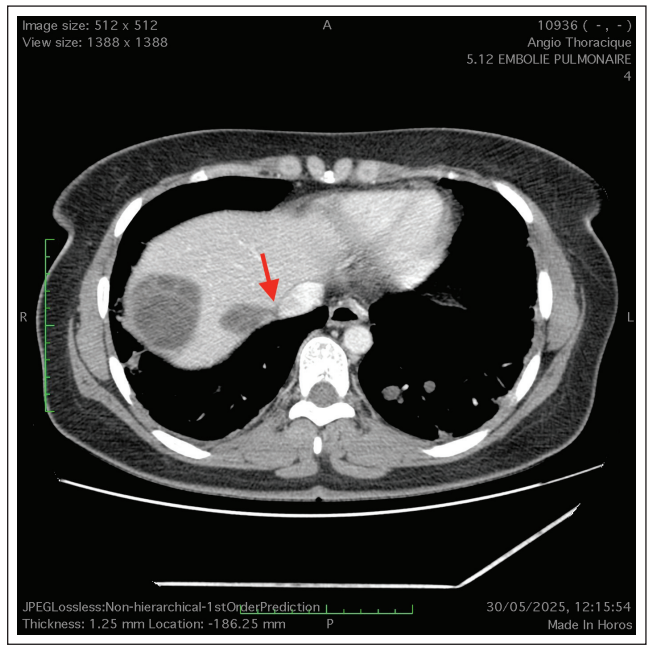



**Figure 4.** Axial CT scan showing a hydatid cyst in contact with the inferior vena cava (IVC); (red arrow).

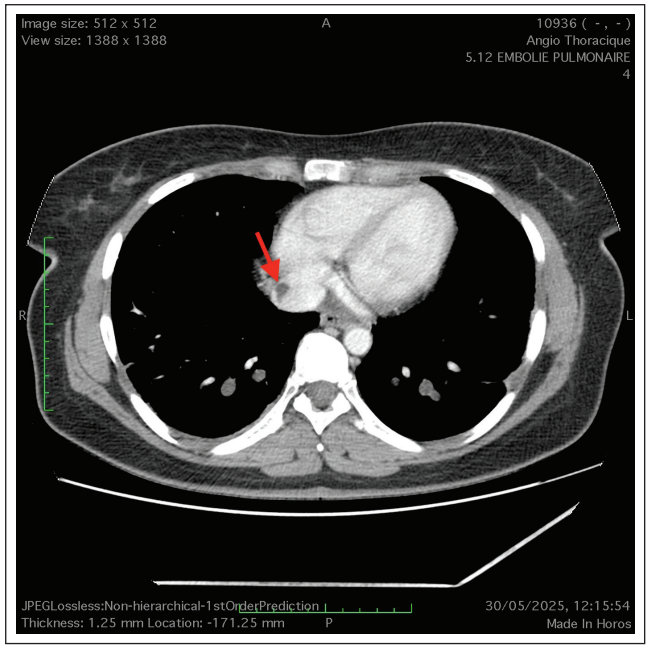



**Figure 5.** Axial CT scan showing a daughter cyst in the region of the inferior vena cava (IVC). (red arrow).

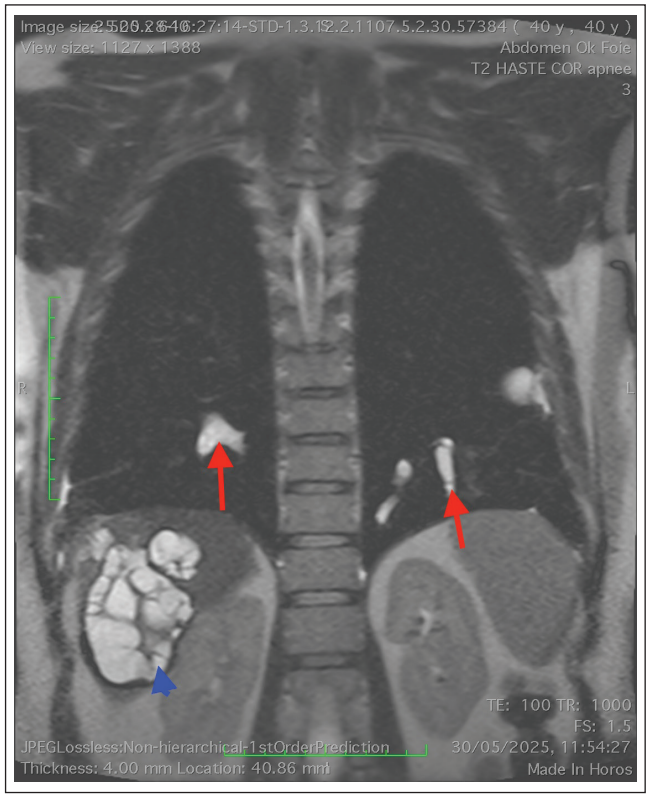



**Figure 6.** Coronal T2-weighted sequence showing hepatic hydatid cysts (red arrow head) and pulmonary arterial cystic emboli (red arrow).

The patient was contacted recently and is continuing her medical treatment. She has noted a slight improvement in her dyspnea. However, she did not attend her follow-up CT scan.

## Discussion

Echinococcosis, or hydatid disease, is a common parasitic infection and represents a significant public health issue in areas of high endemicity. Humans serve as accidental intermediate hosts, while dogs are the definitive hosts. Human contamination occurs through the accidental ingestion of food or water contaminated with *Echinococcus granulosis* embryophores, or through direct contact with an infected dog [1,2].

The liver and lungs are the most common sites for hydatid cysts (HCs). Cardiovascular localization is rare, and pulmonary hydatid embolism is a rare and potentially serious complication of this localization [3].

From a pathophysiological perspective, pulmonary hydatid embolism results from the rupture of a hydatid cyst into the venous return circulation, often of hepatic origin, or from the direct rupture of the HC in the right heart chambers into the cardiac cavities [4].

The clinical presentation of pulmonary hydatid embolism is nonspecific, with symptoms often resembling those of a thromboembolic pulmonary event. In the acute phase, symptoms may be minimal, consisting of a skin rash or even an anaphylactic shock [5].

In later stages, clinical signs of pulmonary embolism may appear due to obstruction of the pulmonary arterial branches. It can also be incidentally discovered during the assessment of lesions related to hydatid disease [6].

This variability in presentation underscores the importance of maintaining a high index of suspicion for hydatid disease, particularly in endemic regions, to ensure timely diagnosis and intervention.

Thoracic CT angiography and MRI are the two imaging modalities of choice for diagnosing pulmonary hydatid embolism. They not only facilitate the diagnosis but also allow for the localization of hydatid cysts, the assessment of their morphology, and the evaluation of their extension into the vascular branches.

These imaging techniques are crucial for understanding the extent of the disease, which aids in planning appropriate management strategies and potential interventions.

On CT scans, hydatid emboli appear as well-defined liquid-density masses with enhanced walls after contrast injection, similar to the case of our patient [7,8]. The imaging can also reveal other parenchymal involvement, notably ground-glass opacities or foci of pulmonary infarction, indicating

pulmonary distress due to chronic obstruction of the pulmonary arterial tree. To the best of our knowledge, instances of pulmonary infarction in this specific situation have been described only infrequently.

On MRI, the content of the HC exhibits a liquid signal, showing low signal on T1-weighted images and high signal on T2-weighted images [9]. Occasionally, a peripheral ring with low signal on T2 may be noted, corresponding to the pericyst, which is a dense fibrous reactive capsule.

Transthoracic echocardiography is useful for evaluating the impact of pulmonary hydatid embolism on cardiac function and, most importantly, for ruling out cardiac localization. This non-invasive imaging technique helps assess any potential cardiac involvement, ensuring comprehensive management of the patient's condition.

There is no defined treatment plan for hydatid pulmonary embolism. Many authors recommend surgery (embolectomy and addressing the cardiac or hepatic source) when possible, often along with perioperative albendazole therapy. In cases with extensive distal embolization, serious comorbidities, or extremely high surgical risks, treatment with albendazole alone might be chosen, though results are usually less favorable and long-term outcomes can be poor [10,11].

## Conclusion

Pulmonary hydatid embolism is an uncommon and serious complication of hydatid disease that can mimic thrombotic pulmonary. Keeping a high level of suspicion in endemic areas, along with careful clinical evaluation and layered imaging, is essential for forming an accurate diagnosis. Management should be customized for each patient, typically requiring a multidisciplinary discussion, and the outlook remains cautious, especially when surgical options are limited.

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