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An 88-year-old dyspneic woman: What Is Your Diagnosis?

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An 88-year-old woman presented to our clinic with a long-term history of cough, dyspnea, and fatigue. She had no history of smoking or biomass exposure. Her medical history revealed that she was treated with an antibiotic for three months eight years ago, but she did not recall the name of the drug or the indication. The results of routine blood testing and physical examination were unremarkable however, renal function test results were poor. The C-reactive protein level was 23 mg/L. Cardiac enzymes were at normal levels. Chest X-ray revealed a right hilar lobulated mass. Intravenous contrast could not be infused due to high creatinine levels. Non-contrast-enhanced multidetector computed tomography (MDCT) of the chest showed a well-delineated, nodular, hypodense mass with fluid attenuation in the right pulmonary artery (Figure 1) and multiple millimetric, hypodense lesions filling the upper lobe branches and surrounding the parenchyma (Figure 2, 3). The right ventricle volume and main pulmonary artery width were within normal limits. There was no flattening or paradoxical bowing of the interventricular septum. There were no visible lesions in the right atrium. A hypodense lesion with visible septa was shown adjacent to the inferior vena cava at images through the abdomen (Figure 4).

a. Pulmonary thromboemboli
b. Pulmonary artery sarcoma
c. Fat embolism
d. Hydatid cyst embolism
e. Lung cancer invading pulmonary artery

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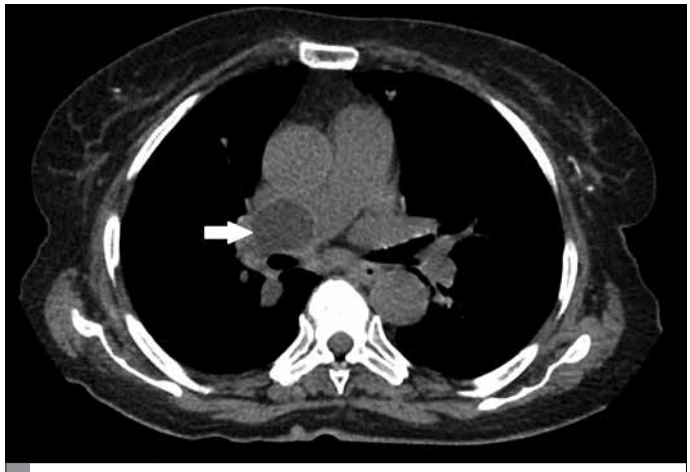

Figure 1. Axial non-contrast-enhanced CT image through the pulmonary artery bifurcation shows a round, well-defined cystic mass in the right pulmonary artery (arrow). The main pulmonary artery diameter is within normal limits.

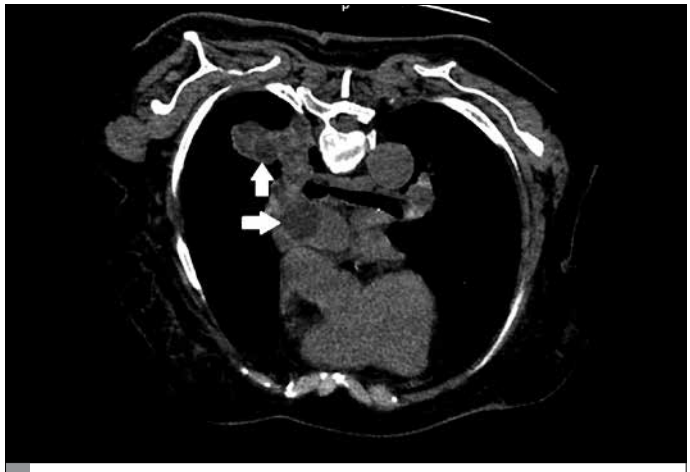

Figure 3. Continuity of the lesions, and the cystic mass is better demonstrated in the reformatted image (arrows).

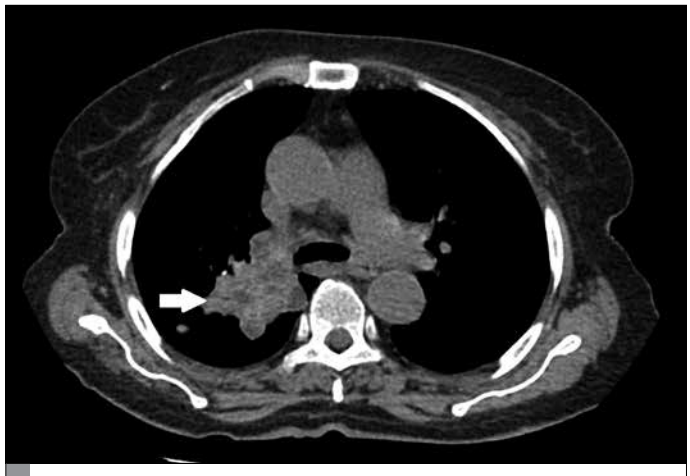

Figure 2. CT image from an upper level shows multiple cystic lesions in the upper lobe artery and adjacent parenchyma (arrow).

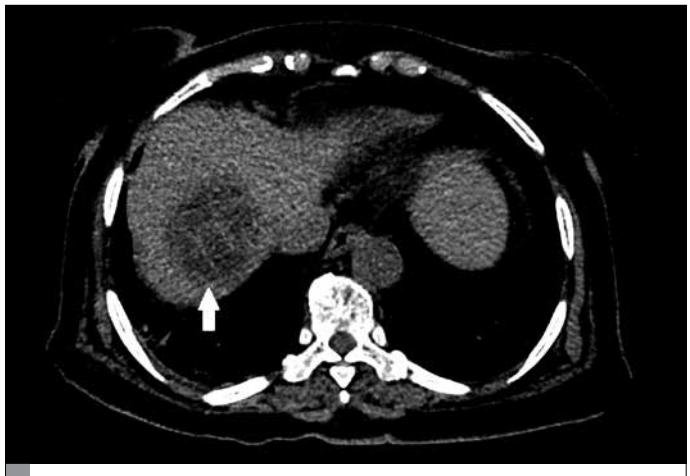

Figure 4. On the axial image through lung bases, a cystic mass with thin linear septa is seen at the hepatic dome (arrow)

Diagnosis:

Pulmonary embolism with pulmonary and hepatic hydatidosis

Hydatidosis is a parasitic disease caused by *Echinococcus granulosus and* remains a conspicuous health problem in sheep-breeding areas, mainly affecting the Mediterranean region, the Middle East, and South America (1). Dogs are definitive hosts. The adult form lives in the intestine of dogs, and eggs are spread via feces. Eggs are very resistant to environmental conditions and may stay infective for up to a year under suitable conditions. Humans may serve as intermediate hosts if contaminated food is consumed or they contact infected dogs. Once eggs are ingested by humans, oncospheres are released with the help of enzymes and bile acids. Oncospheres then penetrate the small intestine mucosa, enter the portal circulation, and reach the liver, where they develop into metacestodes, the so-called hydatid cyst (HC). Eventually, protoscoleces, which are fertile metacestodes, are produced (2).

Serologic and immunologic tests for hydatidosis have high sensitivity, but low specificity, due to cross reactions (2). Diagnosis mostly depends on radiologic and clinical findings. HCs can be single or multiple, and their size may vary. Symptoms vary according to their location, size, and number. They can involve any part of the body but most commonly affect the liver. The lungs are the second most common site, and HC has a predisposition for the involvement of the right posterior lung segments and lower lobes. Bilateral involvement is present in 20% of cases (3).

Pulmonary artery embolism is an extremely rare complication of hydatidosis and generally results from cardiac hydatidosis and less commonly from hepatic HC rupture into the inferior vena cava (4). Recurrent pulmonary embolism due to hepatic HC has also been reported in the literature (5). Patients present with symptoms of thromboembolic disease. Clinically, pulmonary embolism with HC may result in three conditions: fatal acute embolism, subacute pulmonary hypertension, and chronic pulmonary hypertension (6). A predilection for prolonged hypertension with acute embolic episodes has been reported (7).

On MDCT, HCs present as intra-arterial, well-demarcated, non-enhancing, round lesions with fluid attenuation. Peripheral calcifications and membranes can also be seen (8, 9). Contrast-enhanced images may demonstrate the lesion more clearly; however, in patients with supporting clinical findings and history, intra-arterial HC can also be diagnosed with non-contrast-enhanced MDCT.

In our case, after MDCT, a detailed history was obtained from the patient's family, and it was learned that she was treated with albendazole in the past due to hepatic hydatidosis. Indirect hemagglutination tests were also positive for hydatidosis. Her history and multiple intra-arterial, millimetric cystic lesions led us to a diagnosis of pulmonary artery HC embolism. The differential diagnosis should include venous thromboemboli and other causes of nonthrombotic emboli such as arterial wall tumors and fat emboli.

The treatment of pulmonary artery HC consists of the surgical removal of cysts by embolectomy with/without enucleation and subsequently, albendazole, particularly if the lesion is localized (6, 9, 10). However, rupture of the cyst may result in disease dissemination or anaphylactic shock (1, 4, 10). Our patient had a poor performance status, and surgery could not be performed. She also declined medical therapy because of the risk of anaphylaxis. Eventually, she was lost to follow-up.

Informed Consent: Written informed consent was obtained from patient who participated in this study.

Peer-review: Externally peer-reviewed.

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