

A Journey from Vertebra to Pulmonary Artery: The Silent Threat of Pulmonary Cement Embolism Following Vertebroplasty

Vertebradan Pulmoner Artere Yolculuk, Vertebroplasti
Sonrası Sement Embolisinin Sessiz Tehdidi

A 76-year-old female patient presented to the cardiology clinic with complaints of dyspnoea, palpitations and irregular blood pressure. New-onset dyspnoea limiting exertional capacity was present. She had a history of hypertension and underwent percutaneous vertebroplasty for vertebral compression fractures fifteen years ago. On initial examination, the electrocardiogram (ECG) showed sinus rhythm. An echocardiography revealed a normal ejection fraction, mild tricuspid regurgitation and systolic pulmonary artery pressure of 33 mmHg. Both the right atrium and right ventricle appeared normal and laboratory tests for Troponin I and BNP were within normal limits. The D-dimer level was measured at 0.67 mg/L.

Chest X-ray showed linear and nodular opacities in the right lung, in addition to material from the previous vertebroplasty procedure (Figure 1). A computed tomography (CT) angiography was subsequently performed, which identified

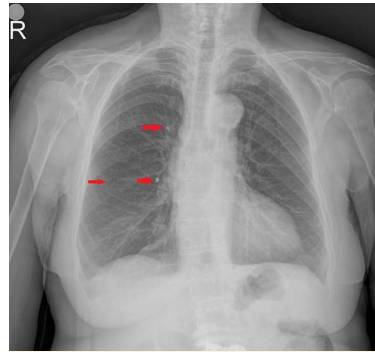


Figure 1. Chest X-ray, pulmonary cement pieces and vertebroplasty materials in L3 vertebral body.

CASE IMAGE OLGU GÖRÜNTÜSÜ

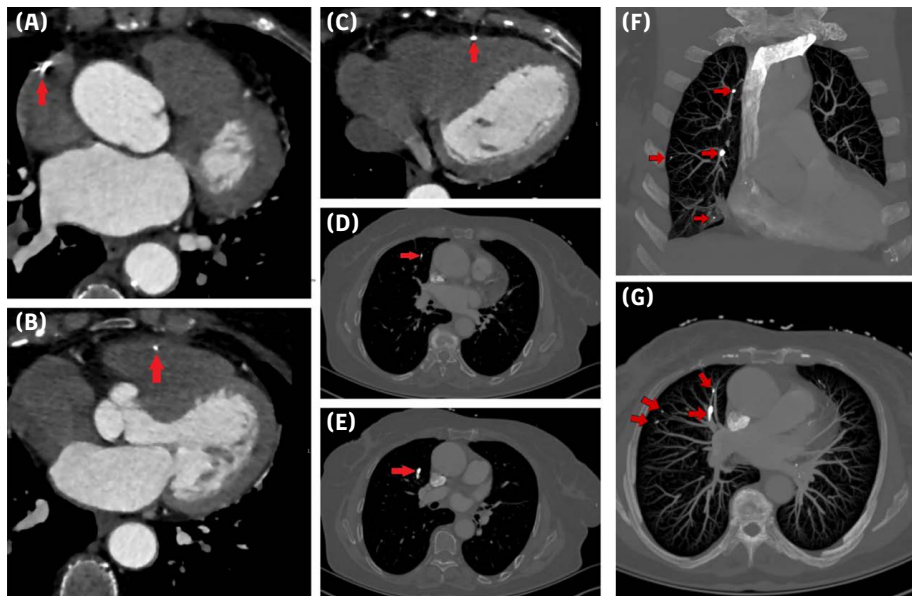


Figure 2. Cardiac and Pulmonary CT, arrows demonstrate multiple hyperdense foci compatible with cement embolism. (A) Right atrial cement pieces, (B, C) Right ventricular cement pieces, (D, E) Pulmonary cement embolism, (F, G) Axial and coronal bone window MIP images.

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multiple millimetric radio-dense materials in the right atrium, right ventricle and segmental pulmonary arteries, suggestive of pulmonary cement embolism (PCE) (Figure 2).

Following the identification of PCE, antihypertensive therapy and anticoagulant treatment were initiated, leading to significant improvement in the patient's symptoms. The diagnosis of PCE was made incidentally while investigating cardiac etiology in a patient with dyspnoea.

Given the increased operative risks associated with vertebral fractures in elderly patients, percutaneous vertebroplasty is commonly preferred. Cement particles can extravasate from the paravertebral space into the venous system, traveling through the vertebral venous plexus and then into the azygos venous system. From there, they drain into the superior vena cava, right heart and eventually the pulmonary arteries, facilitated by the lack of valves in the vertebral venous system.

Although rare, PCE can be life-threatening. Clinical manifestations can range from local compression to progressive embolization and may mimic conditions such as coronary artery disease, pulmonary embolism or heart failure. Symptoms may include chest pain, dyspnea, tachypnea or, in severe cases, hypoxia, hypotension, arrhythmias and cardiac arrest. The presence of cement particles in the right heart can lead to serious complications, such as cardiac rupture and tamponade.

PCE may present shortly after the surgery or may develop after a long period of time. It can be classified as central and peripheral according to the location, and as symptomatic and asymptomatic according to the clinic. When the literature is reviewed, it is seen that there is no consensus

in terms of treatment for PCE. In general, no additional treatment is recommended in asymptomatic patients with peripheral embolism and clinical follow-up is recommended. Anticoagulation is recommended in patients with symptomatic peripheral embolism and in the group with central embolism. Anticoagulation with coumadin is generally recommended for 3 to 6 months.

Therefore, PCE should be considered in the differential diagnosis of patients with a history of vertebroplasty and relevant symptoms. Chest X-ray findings of linear opacities in such cases should prompt further investigation with CT angiography, and echocardiography should be planned for a comprehensive cardiac evaluation.

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