Case Report

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Diagnosis of pulmonary cement embolism using only the bone window setting on computed tomography: a case report

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Abstract

Pulmonary cement embolism (PCE) is one of several complications of percutaneous vertebroplasty and kyphoplasty. Generally, PCE can be easily diagnosed based on typical chest radiograph findings such as single or multiple radiographically dense opacities with a tubular or branch shape in the lung field along with a recent history of percutaneous vertebroplasty or kyphoplasty. These findings can be alarming and may be encountered on routine chest radiographs, even in asymptomatic patients. One study showed that PCEs that were not visualized on chest radiograph were also not shown on chest computed tomography. However, we encountered a patient with dyspnea who had normal chest radiograph findings but was diagnosed with PCE through only the bone window setting on chest computed tomography. The present case will be beneficial to all physicians examining older patients with dyspnea.

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Bone window, dyspnea, pulmonary cement embolism, vertebroplasty, chest radiograph, computed tomography

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Introduction

Pulmonary cement embolism (PCE) is one of several complications of percutaneous vertebroplasty (PVP) and kyphoplasty.[1–3] Generally, PCE can be easily diagnosed based on typical chest radiograph findings; i.e., multiple radiographically dense opacities with a tubular and branching shape scattered sporadically or distributed diffusely throughout the lungs along with a recent history of PVP or kyphoplasty.[1,2,4] The presence of cement emboli in the branches of the pulmonary arteries can be confirmed by chest computed tomography (CT). However, although one study showed that CT could not show PCEs that were not visualized on chest radiographs,[1] we encountered a patient with dyspnea who had normal chest radiograph findings but was diagnosed with PCE through only the bone window setting on chest CT. The present case will be beneficial to all physicians examining older patients with dyspnea.

Case report

A 74-year-old man with a medical history of hypertension presented with dyspnea that had worsened during the past 3 weeks. He had been experiencing mild dyspnea on exertion for the past 6 months. On admission, he had a cough but denied other symptoms. His vital signs were as follows: respiratory rate, 20 bpm; temperature, 36.7°C; pulse, 77 bpm; and blood pressure, 130/70 mmHg. Arterial blood gas analysis revealed a partial oxygen pressure of 65 mmHg, partial carbon dioxide pressure of 39 mmHg, pH of 7.44, and oxygen saturation of 93% while resting in room air. Laboratory data included a D-dimer level of 0.44 μg/mL (reference range, 0.00–0.50 μg/mL) and an N-terminal B-type natriuretic peptide level of 15 pg/mL (reference range, 0–500 pg/mL). A chest radiograph indicated no active lesions in the lung (Figure 1). Contrast-enhanced chest CT was performed to exclude pulmonary thromboembolism or other lung disease and revealed no evidence of filling defects in vessels, thus suggesting pulmonary thromboembolism. Transthoracic echocardiography indicated a mean pulmonary arterial pressure of 47 mmHg (reference range, 10–22 mmHg). On further evaluating the chest CT through the bone setting, however, we identified several hyperdense linear lesions in both peripheral pulmonary arteries that could not be identified using the lung or soft tissue windows, and this raised suspicion for PCE (Figure 2). We confirmed that the patient had undergone PVP with a diagnosis of vertebral compression fracture at another hospital 14 months previously. During hospitalization, his symptoms resolved slightly after starting treatment with diuretics.

Ethics

This study was a case report involving only one patient and only involved observation of the clinical effects. Therefore, approval by an ethics committee was not required. We obtained verbal consent from the patient for publication of this case report and images.

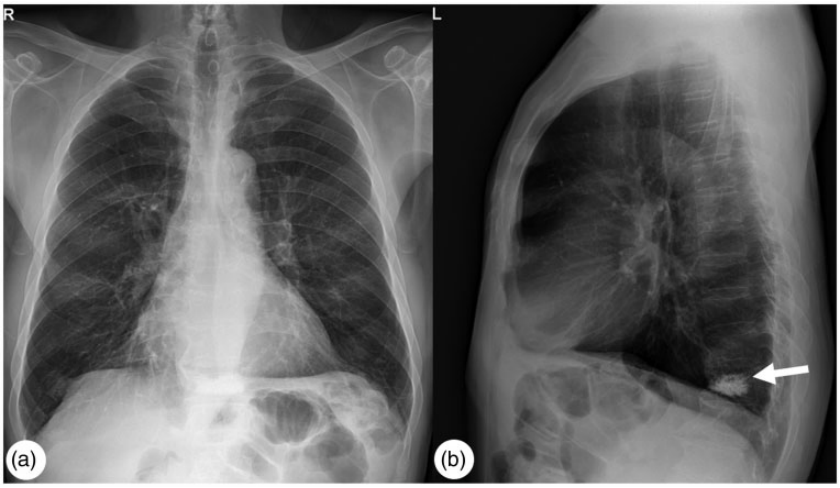

Figure 1. (a) Posteroanterior and (b) lateral chest radiographs on admission showed no active lung parenchymal lesions or bone cement deposition in the T11 vertebral body (arrow).

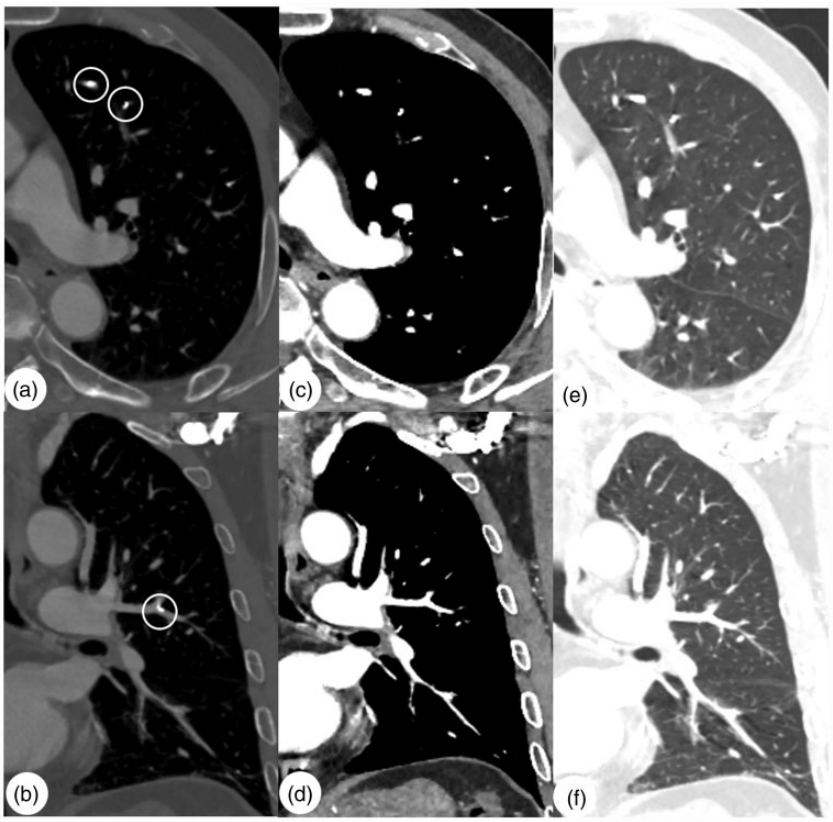

Figure 2. (a, b) Selected axial and coronal images of the bone window settings on chest computed tomography showed several small nodular or linear hyperdense cement emboli (circles) in the pulmonary artery branch in the left upper lobe. Images obtained using the (c, d) soft tissue window and (e, f) lung window settings on chest computed tomography showed no evidence of cement emboli.

Discussion

PVP is a minimally invasive procedure that was first introduced in 1987 and is now widely used for pain relief in patients with symptomatic vertebral compression fractures secondary to osteoporosis, multiple myeloma, metastatic tumors, or hemangioma.[5–7] During this procedure, polymethylmethacrylate (PMNA) cement, a rapidly setting bone cement, is transcutaneously injected into the vertebral body under imaging guidance.[1] Generally, complications of PVP are rare, and most are related to leakage of PMNA into the spinal canal or perivertebral venous system.[5] PCE is a recognized complication of distant leakage of cement by either penetration into the venous plexuses or retrograde migration through the arterial vessels into the aorta.[8] Cement leakage occurs frequently after PVP, with reported rates up to 81% depending on the imaging method.[9] Most cases of cement leakage are clinically asymptomatic, and this complication is usually incidentally detected on chest radiographs. Therefore, the incidence of PCE might be underestimated; it may actually be higher than the reported range of 2% to 26%.[2,5,7]

Most patients with PCE remain asymptomatic, with 0.4% to 0.9% of patients showing symptoms. Common clinical signs and symptoms include dyspnea, cyanosis, palpitation, chest pain or tightness, and acute respiratory distress syndrome in rare cases.[10,11] These symptoms rarely occur immediately after PVP; most symptoms occur later, sometimes weeks or months after PVP.[1,2,12] Therefore, it is generally difficult to recognize asymptomatic PCE.

PCE appears on chest radiographs as single or multiple radiographically dense opacities with a tubular or branch shape in the lung field. These findings can be alarming and may be encountered on routine chest radiographs, even in asymptomatic patients.[1,4] The presence of hyperattenuating cement emboli in the branches of the pulmonary arteries can be confirmed by chest CT.[5] One study revealed an incidence of 4.6% after a retrospective review of postprocedural chest radiographs in 65 PVP procedures.[1] Furthermore, the chest CT scans ($n = 22$ using contrast) obtained in their study did not show any PCEs that were also not visualized on the chest radiographs.[1] An echocardiogram may be of benefit to check for a secondarily elevated pulmonary artery pressure in the setting of multiple emboli or a large, single embolus in a symptomatic patient.[12] Our patient, who had undergone PVP 14 months previously, had a normal chest radiograph on admission and no evidence of PCE on the lung or soft tissue windows on chest CT. However, several hyperdense linear lesions were found in both peripheral pulmonary arteries through the bone window on chest CT. Our case indicates that patients with PCE may even have normal chest radiograph findings and that use of the bone window setting on chest CT is an important tool for diagnosing PCE.

No clear consensus has been reached regarding the optimal treatment strategy for PCE. A review of the literature is recommended according to the severity of symptoms and location of the PCE.[2] Conservative treatment or clinical follow-up and anticoagulation represent the cornerstones of treatment, but some reports have described symptomatic patients with central embolisms requiring surgical removal.[2,10] The rationale for anticoagulation is to reduce the risk of thrombus formation due to the thrombogenic nature of the cement. However, no data support the formation of thrombi on the cement.[1] Generally, anticoagulation therapy is recommended for 3 to 6 months after the occurrence of the embolism.[2] Previous studies have shown that patients with PCE have favorable outcomes even when they do not

receive specific treatment. Although early mortality has been reported in patients with acute PCE, an autopsy study revealed that none of the patients had died due to the impact of PCE.[1,2,7,10,11,13,14] Our patient showed mild symptoms with pulmonary hypertension due to chronic peripheral PCE. Therefore, we started treatment with diuretics. Although we did not use anticoagulants, no problems were noted during clinical follow-up.

This case highlights the usefulness of the bone window on chest CT in patients with normal chest radiographs and a history of PVP. PCE is being increasingly recognized, probably because of the wide therapeutic approach of PVP for symptomatic vertebral compression fractures, the increased use of CT, and the longer lifespans of patients. This report will be beneficial to all physicians who examine older patients with dyspnea.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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