Case Letter

Pulmonary cement emboli complicated by cardiogenic shock following percutaneous kyphoplasty

Sir,

Pulmonary cement emboli (PCE) are uncommon complications of percutaneous vertebral augmentation procedures, which are routinely performed to alleviate pain associated with vertebral compression fractures. Percutaneous vertebral augmentation procedures consist of injecting bone cement into the vertebral body through the pedicle under imaging guidance.[1,2] The most detrimental outcomes of this surgery pertain to the systemic leakage of cement.[1,3] PCE occur when bone cement passes through the valveless venous circulation and ultimately reaches the pulmonary artery.[1,4] While most cases of PCE are asymptomatic, patients can present acutely with severe symptoms. We present the fatal case of a 61-year-old male who developed PCE complicated by cardiogenic shock within 56 hours of undergoing percutaneous kyphoplasty. Our aim is to raise the awareness toward a potentially life-threatening complication of percutaneous vertebral augmentation procedures that is not well-documented outside of orthopedic literature.

Our case depicts a 61-year-old male who presented to the emergency department (ED) with progressively worsening back pain following a mechanical fall 6 weeks prior. Physical examination was remarkable for tenderness along the thoracic spinous process without any focal neurologic deficits. Magnetic resonance imaging of the thoracic and lumbar spine revealed a T12 burst fracture with ligamentous injury and bony retropulsion. The patient underwent an uncomplicated percutaneous kyphoplasty of T11, T12, and L1 vertebrae. His postoperative hospital course was unremarkable, and he was discharged to a nursing home 48 hours following the surgery.

Eight hours after discharge, the patient returned to the ED due to acute onset dyspnea. At the time of initial presentation, he was tachypneic, tachycardic, hypotensive, and hypoxic, despite supplemental oxygenation through a nonrebreathe mask. His physical examination was notable for decreased breath sounds bilaterally with crackles at the bilateral lung bases and increased work of breathing with accessory muscle use. Chest radiograph revealed hyperdense perihilar opacities [Figure 1]. Computed tomography angiography (CTA) of the chest reported enlargement of the main pulmonary artery with extensive hyper-densities in the right main and bilateral segmental pulmonary arteries, along with bilateral dependent atelectasis [Figure 2]. A stat two-dimensional echocardiogram showed evidence of right heart strain with dilation of the right atrium and right ventricle.

In the ED, the patient was intubated due to concern for impending respiratory failure and started on vasopressors for cardiogenic shock. He was admitted to the intensive care unit for further care. Given that the PCE were nonobstructive and the patient was hemodynamically unstable, the risks of thrombectomy were felt to outweigh any potential benefit of the procedure. He remained hypotensive despite utilizing three vasopressors at maximal doses. The patient suffered from three cardiac arrests, which proved to be fatal.

This case illustrates that patients presenting with symptoms of dyspnea, chest pain, chest tightness, palpitations, cough, or hemoptysis in the appropriate clinical setting should raise concern for PCE. It is important to note that unlike thromboembolic emboli, cement emboli can be well visualized on chest radiograph because bone cement has a higher density than that of the lung parenchyma.[5] On chest radiograph, solitary or multiple, tubular or branching, dense opacities should raise suspicion for PCE and warrant additional imaging.[1,5,6] Noncontrast CT of the chest has a higher sensitivity for PCE and commonly visualize PCE as branching, highly attenuated areas along the course of the pulmonary artery.[1,3]

Figure 1: Chest radiograph taken at time of hospital re-admission depicting hyperdense perihilar opacities (arrows), which are characteristic of pulmonary cement emboli
There are no standardized therapeutic guidelines for PCE. Asymptomatic patients and symptomatic patients with peripheral emboli are typically managed conservatively. The mainstay of treatment for central pulmonary emboli is removal through surgical or interventional radiological approaches. The role of anticoagulation following PCE is disputed, however, most clinicians agree with the immediate administration of unfractionated heparin, followed by anticoagulation with warfarin for 3–6 months. Currently, there are no set criteria for the surveillance of PCE and no evidence to support routine postprocedure chest imaging. Patients undergoing cement augmentation procedures should have close follow-up. Health-care providers should have a low threshold to evaluate any patient presenting with postoperative dyspnea with a chest radiograph or CT.

In summary, we depict the case of a middle-aged male who passed away after developing PCE to the pulmonary arteries mere hours following percutaneous kyphoplasty. This case is unusual in that PCE seldom cause clinical symptoms and even more rarely result in death. This case presentation adds to the growing body of the literature on PCE and can help establish screening criteria and guidelines for intervention. Familiarization of this condition among medical providers across specialties, including emergency medicine, critical care, and pulmonology, can help prevent similar adverse outcomes.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal.

The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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