

# Cement Pulmonary Embolism: A Common and Potentially Serious Complication of Vertebroplasty

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# Cement Pulmonary Embolism: A Common and Potentially Serious Complication of Vertebroplasty

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## Introduction:

Vertebroplasty is a surgical intervention used to treat symptomatic vertebral compression fractures that are refractory to conservative medical therapy. The procedure involves injection of a cement polymer, commonly polymethylmethacrylate (PMMA), into the vertebral body, allowing for improved stability and pain relief. First introduced in the 1980's, vertebroplasty has become an increasingly recommended therapeutic intervention due to its high efficacy. It has been reported that 90% of patients with vertebral lesions from either osteoporotic compression fractures or hemangiomas and 70% of those from multiple myeloma or metastatic disease have experienced significant relief of their pain after vertebroplasty.<sup>1</sup>

A common complication of this procedure is cement leakage into the surrounding tissues, which one study using post-procedure CT on all patients demonstrated occurred in 80%.<sup>2</sup> If the injected PMMA gains entry into the venous system, embolization to the pulmonary vasculature can occur.

Here we present a case of a 50-year-old female who developed multiple symptomatic bilateral cement pulmonary emboli (PE) following vertebroplasty of a symptomatic osteoporotic vertebral compression fracture.

## Case Presentation:

A 50-year-old female with a history of osteoporosis, systemic lupus erythematosus and pulmonary embolism on chronic anticoagulation presented with a three-week history of mid to low back pain, difficulty urinating and bilateral leg weakness. Medications included prednisone and warfarin.

Physical exam was significant for mildly elevated blood pressure, a cushingoid appearance, tenderness to palpation around the T12 vertebral body, hyperreflexia in the legs, and extensor plantar response on the left.

MRI of the thoracic and lumbar spine demonstrated acute to subacute severe vertebral compression deformity of T12 with bony retropulsion resulting in cord compression. Remote compression deformities were seen at L1, L4 and L5.

She subsequently underwent posterior spinal fusion from T9-L3, vertebroplasty of the vertebral bodies of T9-11 and L1-3 and posterior vertebral body osteotomy at T12 in order to reduce her spinal deformity and decompress the cord. No immediate complications were noted.

Four hours post-operatively, she developed dyspnea and was found to have fever, tachycardia and hypoxia, with the following vital signs: T 101.7 F, P 120, RR 14, BP 118/64, SpO2 95% on 4L NC.

Chest x-ray revealed opaque embolic material in the smaller pulmonary vessels bilaterally (Figure 1). CT of the chest with contrast demonstrated areas of very high attenuation within several pulmonary artery branches with an appearance consistent with cement pulmonary emboli (Figures 2a and 3a).

Anticoagulation was initiated with unfractionated heparin and she was bridged to

warfarin. Her fever and tachycardia resolved in the next two and four days, respectively; however, she remained dependent on supplemental oxygen until post-operative day 11. On post-operative day 13, she was discharged to inpatient rehabilitation in stable condition.

## Discussion:

**Introduction:** Several studies have looked at the incidence of cement leakage and pulmonary embolism in patients undergoing vertebroplasty. They have shown that when chest x-ray or chest CT was obtained on all patients post-vertebroplasty regardless of symptoms, the incidence of asymptomatic cement leakage and embolization was quite high, with up to 26% of patients demonstrating evidence of cement pulmonary emboli on chest CT.<sup>3</sup> Thus, because symptoms of cement PE are often mild or absent and post-procedure imaging of the chest is not routinely performed, this condition is believed to be often missed in clinical practice. If left undetected and untreated, cement pulmonary emboli could potentially cause not only acute cardiopulmonary problems, but potentially irreversible pulmonary parenchymal damage if extensive.

**Pathogenesis:** Vertebral bodies are highly vascularized entities and form a valveless network with the paravertebral and extradural venous plexuses. Vertebral compression fractures decrease bony hindrance of the venous drainage and facilitate migration of cement fragments into the systemic venous circulation. Tozzi et al suggested three factors that appear to play a role in the development of cement PE: insufficient polymerization of the polymethylmethacrylate at the time of injection, needle positioning with respect to the basivertebral vein and overfilling of the vertebral body.<sup>4</sup>

**Clinical Features:** While the signs and symptoms can be similar to those of thromboembolic PE, including hypoxemia, dyspnea, tachypnea, hemoptysis, chest pain, palpitations, arrhythmia, low-grade fever and hypotension, the majority of affected patients are asymptomatic. Only an estimated 1% of cases with osteoporotic

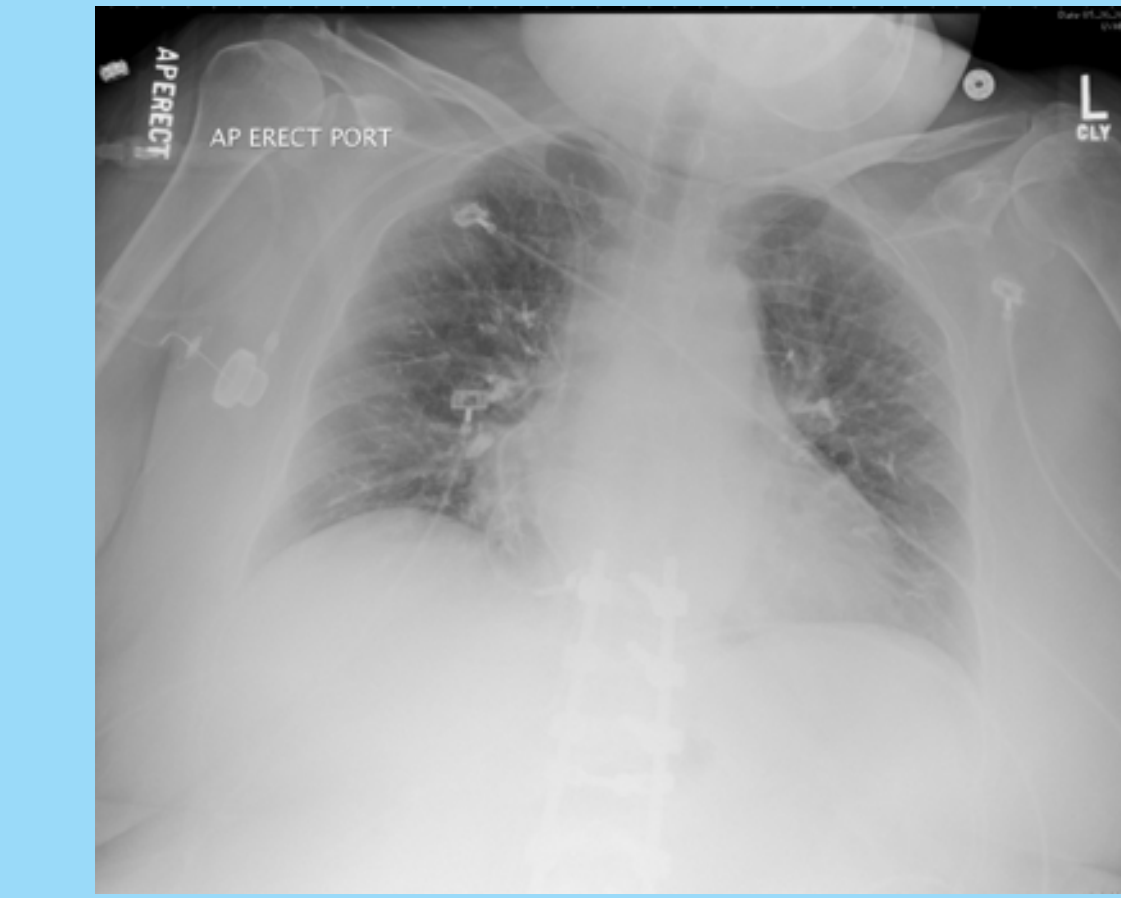


Figure 1. Portable chest x-ray: Opaque embolic material is seen in small pulmonary artery branches bilaterally.

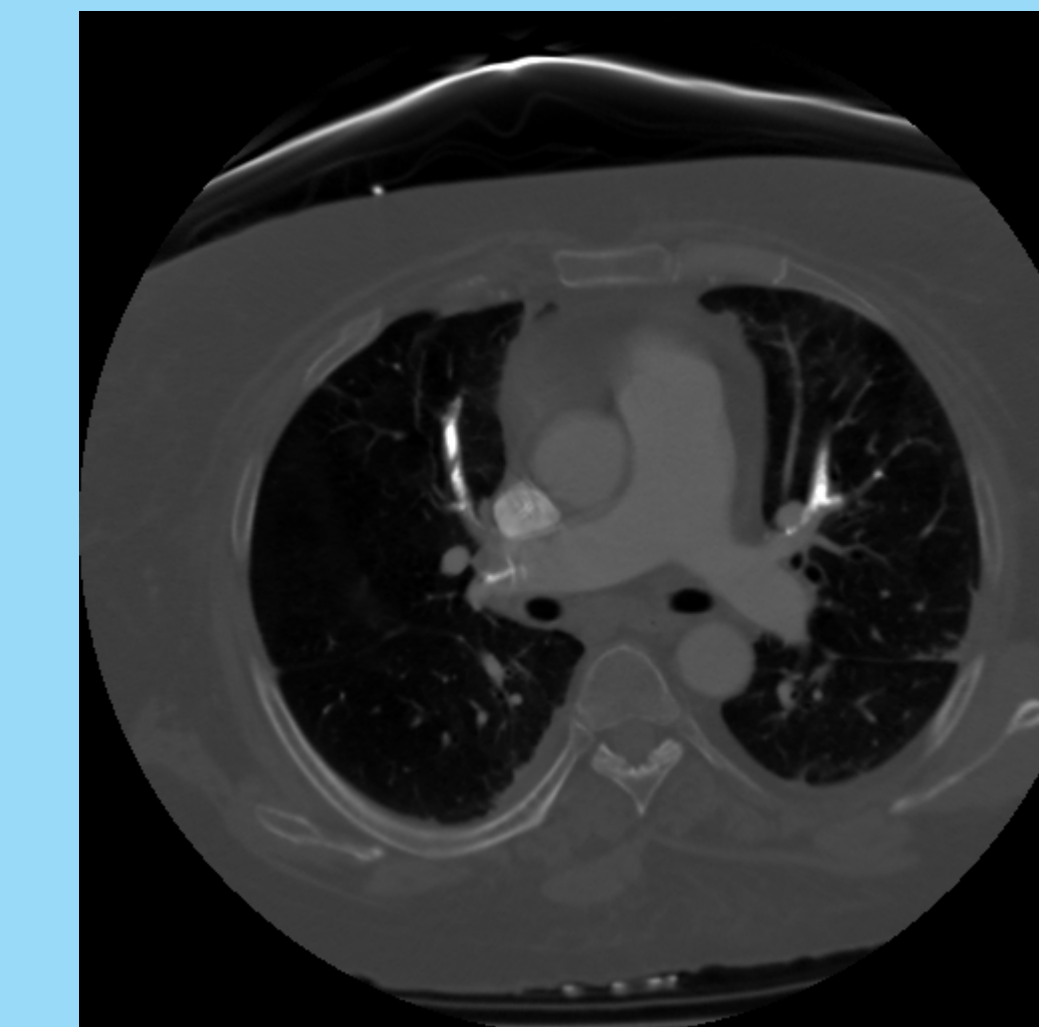


Figure 2a. Chest CT with contrast- bone window: Areas of high attenuation within pulmonary artery branches bilaterally consistent with cement pulmonary emboli.

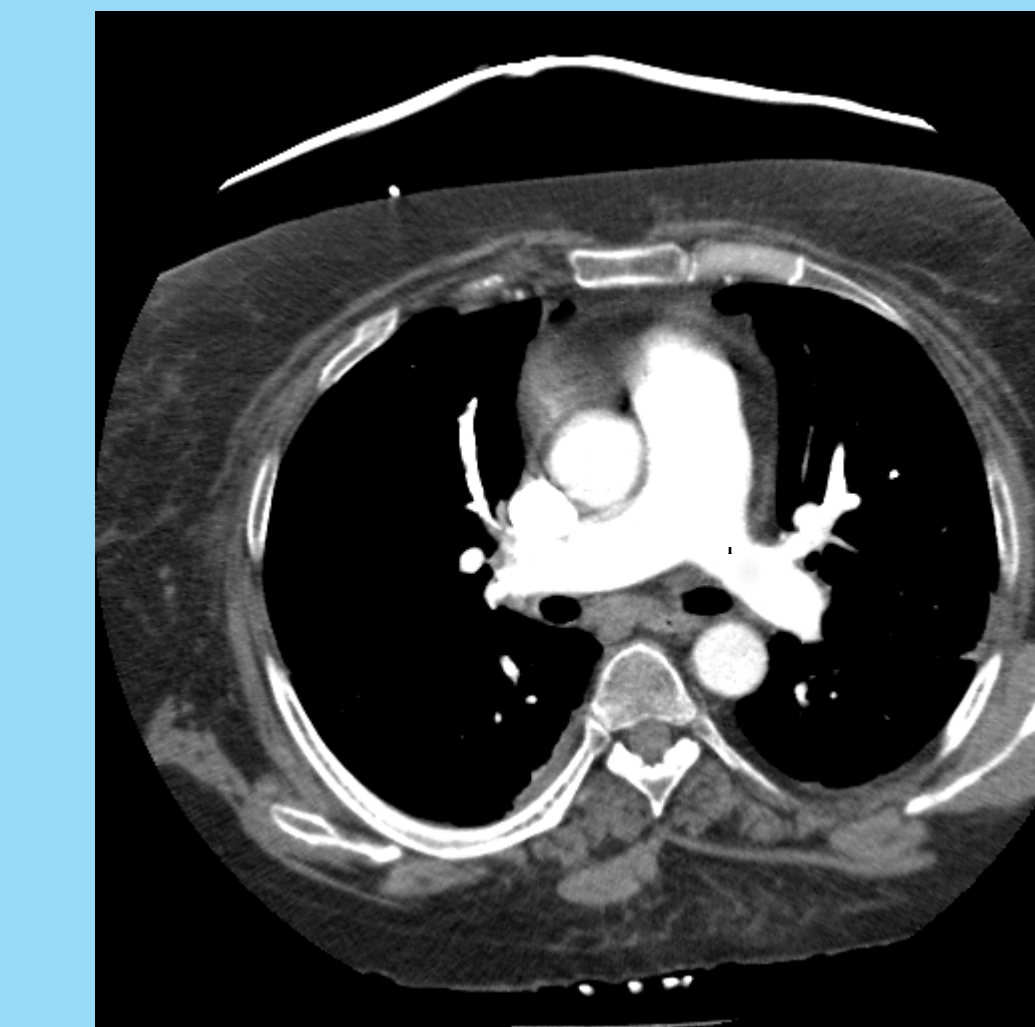


Figure 2b. Chest CT with contrast- mediastinal window: cement pulmonary emboli not well-visualized.

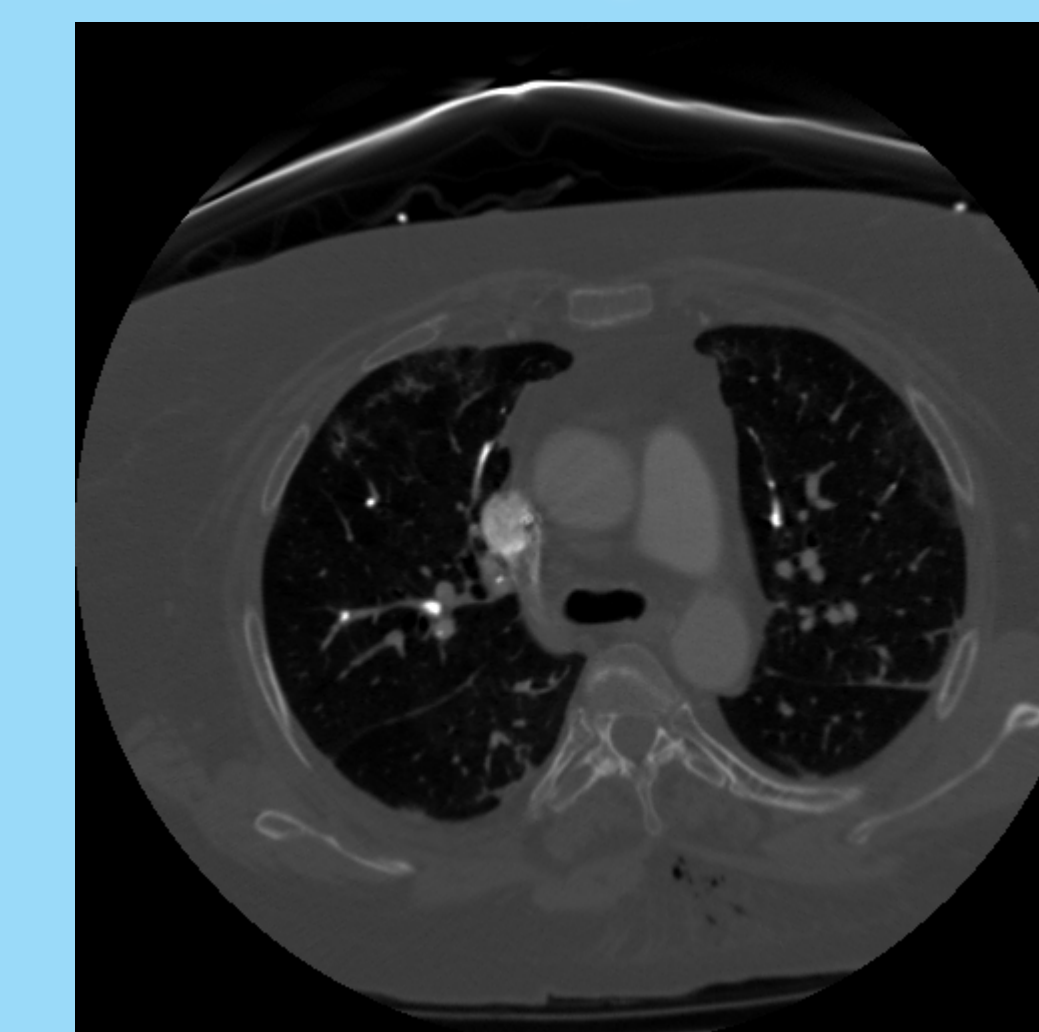


Figure 3a. Chest CT with contrast- bone window: Areas of high attenuation within pulmonary artery branches bilaterally consistent with cement pulmonary emboli.

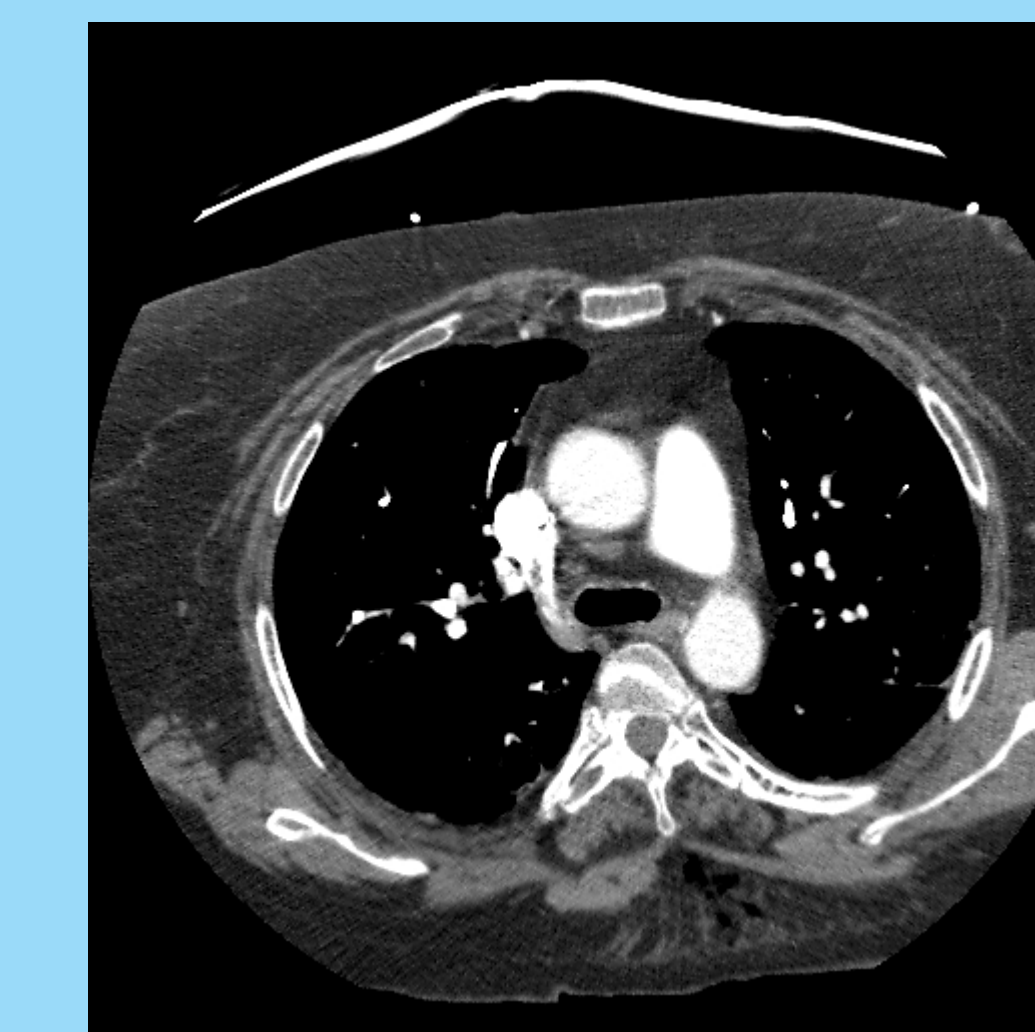


Figure 3b. Chest CT with contrast- mediastinal window: cement pulmonary emboli well-visualized.

compression fractures and 2-5% of those with osteolytic metastases will be symptomatic.<sup>5</sup> While less common, severe and fatal cases have been reported.

**Diagnosis:** Cement PE can be diagnosed by detection of radiopaque material within the pulmonary vasculature on chest x-ray (Figure 1) or by areas of high attenuation on chest CT. Unlike thrombotic PE, cement emboli are best visualized in the bone window of CT due to their density, particularly if intravenous contrast is used (Figures 2a, 2b, 3a, 3b).

To date, there have been no guidelines published on the detection and management of cement PE. Routine post-procedure screening chest x-rays to screen for this complication may be of benefit, as prompt initiation of anticoagulation may prevent superimposed thrombus formation and progressive arterial occlusion in affected patients.

**Treatment:** Recommended acute treatment is with unfractionated heparin, with transition to low molecular weight heparin or warfarin prior to discharge. While optimal duration of anticoagulation therapy for this condition is unknown, six months may allow for sufficient endothelialization of the polymethylmethacrylate embolus. In the rare case of cement PE complicated by respiratory or right heart failure with a large central embolus, embolectomy may be necessary.

## Conclusion:

In conclusion, cement PE is a common post-operative complication of vertebroplasty that physicians should be aware of to allow for prompt detection and treatment.

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