

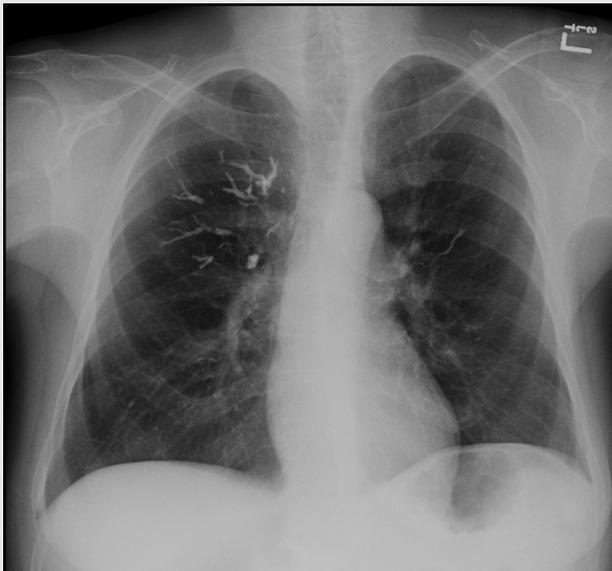
Cement emboli

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CASE

This patient is a 71-year-old woman who presented with persistent chest symptoms. She has a remote history of pulmonary tuberculosis in the 1970s diagnosed by lymph node biopsy and treated with isoniazid and rifampin for an unknown period of time. She has a 20 pack-year smoking history but quit 38 years ago. She worked in the trucking industry and developed a deep venous thrombosis (DVT) in 1991 probably secondary to prolonged trips and was treated with Coumadin for an unknown period of time. In June 2016, she underwent vertebral kyphoplasty. Shortly thereafter, she developed the sudden onset

of shortness of breath with dry cough. A V/Q scan was read as an intermediate probability for pulmonary embolus in the right upper lobe. Lower extremity ultrasound studies were negative for DVT at that time. She was started on rivaroxaban and referred for evaluation. She denied dyspnea but stated that she had a sensation of her “chest catching” intermittently. On presentation, her pulse was 91 beats per minute, her respiratory rate was 16 breaths per minute, and her SpO₂ was 97% on room air. Her lungs were clear without adventitious breath sounds. There was no lower extremity edema. Her pulmonary function tests showed normal volumes and mild impairment in diffusion capacity (DLCO 78% predicted, both corrected



This figure includes PA and lateral chest x-rays which show calcified densities with a vascular distribution in the upper lung fields. These represent the cement emboli following this patient's vertebroplasty.



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and uncorrected for alveolar volume). Chest radiograph showed branching radiopaque opacities in both upper lobes, right greater than left, and a clinical diagnosis of pulmonary cement embolism was made.

DISCUSSION

Pulmonary embolism (PE) usually occurs secondary to a DVT. There are approximately 1 million reported cases annually in the United States with 60,000 to 100,000 being fatal.¹ Ninety-five percent of all reported PEs are associated with a DVT. Nonthrombotic PEs occur less frequently and include embolization of fat, air, amniotic fluid, tumor, and foreign bodies. Iatrogenic causes of PE include migration of guidewires, vascular coils, vena cava filters, fragments of intravascular catheters, and implanted devices. These foreign bodies require removal to prevent infection and/or thrombus formation. Identifying the size, shape, and location of foreign bodies is important, and several different radiology studies can do this. Retrieval of foreign bodies should be done using an intravascular approach when possible, but some objects are too large to retrieve by this method. Invasive open surgical retrieval is required for some objects. Projectiles, like bullets, require surgical retrieval and repair of any damaged structures. The longer objects have been lodged in the vasculature the more difficult they become to remove. Long term complications of thrombotic and nonthrombotic PE are similar and include pulmonary hypertension, right sided heart failure, and recurrent PE.

A pulmonary cement embolism (PCE) is an iatrogenic form of PE. These PEs can be caused, as in this case, by polymethylmethacrylic (PMMA), a cement used for vertebroplasty. The leakage of PMMA may occur when the cement is mixed too thin and/or when high pressure is used to inject the cement. The PMMA leaks into the vertebral vessels and then hardens in the pulmonary vasculature after traveling through the right side of the heart. Studies have reported significant variability in the frequency of PCE during this procedure; it ranges from 2-26%.² This wide range can be

explained by study designs that include screening radiology for all subjects after the procedure or screening only symptomatic subjects. Most PCE are asymptomatic and are incidental findings on chest radiography. A PCE is easily identifiable with plain chest X-ray due to the barium component added to the cement mixture. No treatment guidelines have been established. Current recommendations include standard treatment for a thrombotic PE. This includes 3-6 months of anticoagulation therapy in patients with mild to moderate symptoms. Very symptomatic patients may require surgical removal of cement or lobectomy. Massive PCE at the time of the procedure has led to fatalities in some cases. Asymptomatic patients should be monitored with routine clinical evaluations.

Keywords: cement, vertebroplasty, pulmonary emboli, iatrogenic

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