Acta Radiologica Open
4(8) 1–4

DOI: 10.1177/2058460115595660
arr.sagepub.com

Venous extravasation and polymethylmethacrylate pulmonary embolism following fluoroscopy-guided percutaneous vertebroplasty

Mina S Makary[1], Ivan L Zucker[2] and John M Sturgeon[2]

Abstract

Percutaneous vertebroplasty has gained widespread popularity and demonstrated clinical efficacy in the treatment of spinal osteoporotic compression fractures and pathologic osteolytic lesions. Despite its rapid pain relief and safety, this minimally invasive intervention has exhibited some rare complications over the past decade. In this case study, we describe a patient with an uncommon complication of polymethylmethacrylate (PMMA) cement pulmonary embolism following fluoroscopy-guided percutaneous vertebroplasty for treatment of pain associated with an osteoporotic vertebral fracture. We present this case to highlight that vertebroplasty is not risk-free and that knowledge of such potentially severe complication is necessary for prevention and optimal operative outcomes.

Keywords

Vertebroplasty, pulmonary embolism, polymethylmethacrylate (PMMA), complication

Date received: 14 February 2015; accepted: 18 June 2015

Introduction

Since its introduction by the French radiologist Galibert in 1984 for treatment of aggressive spinal hemangiomas (1–3), vertebroplasty has gained widespread popularity and demonstrated efficacy as a minimally invasive intervention for a variety of conditions. Percutaneous vertebroplasty has now shown clinical utility in vertebral body augmentation for osteoporotic vertebral compression fractures (VCFs) of the spine as well as for pathologic osteolytic lesions including myelomatous and metastatic disease (4–7). Despite its rapid pain relief, minimal invasiveness, and safety, this effective technique has exhibited some rare complications over the past decade. Case reports and some small studies have shown postprocedure complications of infection, hemorrhage, radicular pain, neurologic deficits, and embolic sequelae (7,8). In this case study, we report a patient with an uncommon complication of PMMA cement pulmonary embolism following fluoroscopy-guided percutaneous vertebroplasty for treatment of pain associated with an osteoporotic vertebral fracture.

Case report

A 47-year-old female patient with history of osteoporosis presented with severe low back pain refractory to conservative analgesic therapy. Her past surgical and family history was otherwise non-contributory, and physical examination revealed lumbar pain with palpation and paraspinal tenderness from L2 to L4. A lumbar radiograph revealed a vertebral fracture and degenerative disc disease. Magnetic resonance imaging (MRI) confirmed a compression fracture of the L2 vertebral body with 40% height loss.

[1]Department of Radiology, The Ohio State University Wexner Medical Center, Columbus, Ohio, USA
[2]Department of Radiology, Mount Carmel West Medical Center, Columbus, Ohio, USA

Corresponding author:
Mina S Makary, Department of Radiology, The Ohio State University Wexner Medical Center, 395 West 12th Avenue, 4th Floor, Columbus, Ohio 43210, USA.
Email: mina.makary@osumc.edu

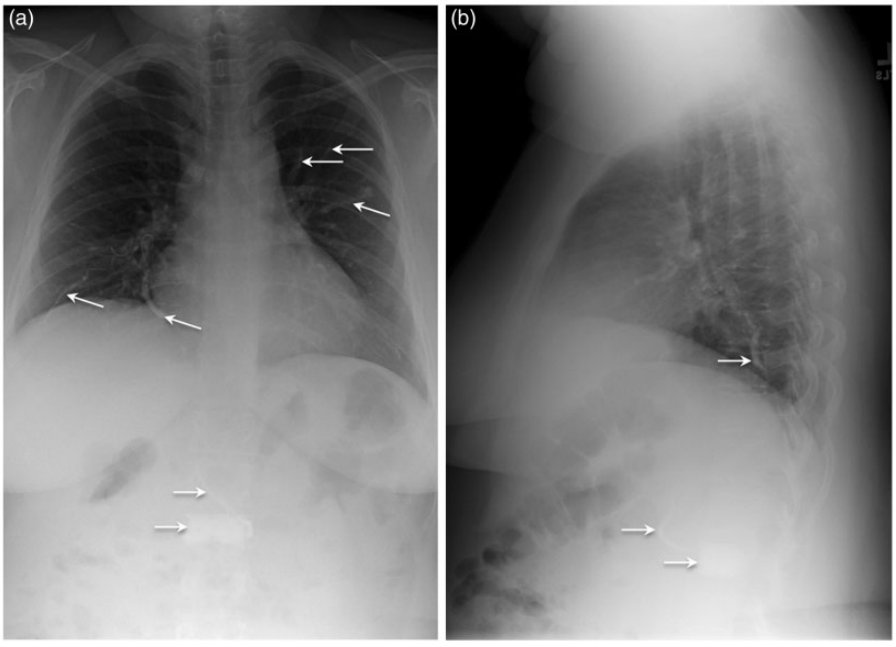

Fig. 1. Posteroanterior (a) and lateral (b) chest radiographs demonstrating multiple bilateral high-density opacities within subsegmental pulmonary arteries, representing cement pulmonary emboli. A linear density coursing superiorly lateral to the L2 vertebral body, consistent with PMMA extravasation into the left lumbar vein and the paravertebral spinous plexus, is also noted.

The patient subsequently underwent elective percutaneous vertebroplasty for vertebral body augmentation and stabilization of the compression fracture. The procedure was performed under conscious sedation and local anesthesia by a bipedicular approach with fluoroscopic guidance. Opacified PMMA cement (6 mL) was slowly injected into the L2 vertebral body until good filling in a symmetrical fashion was observed. The bones of this osteoporotic vertebra were very soft, and the needle was readily guided into the vertebral body. The patient was discharged from the radiology suite in excellent condition, and her postoperative course was uneventful.

Four years later, the patient developed a respiratory infection, and chest radiographs revealed an incidental finding of multiple dense linear and branching opacities bilaterally (Fig. 1). Non-contrast chest computed tomography (CT) confirmed the presence of tubular high-attenuation material within the right lower lobe pulmonary artery and its branch vessels as well as a small linear high attenuation focus within the anterobasilar aspect of the left lower lobe (Fig. 2). There was also high attenuation material filling a portion of the left lumbar vein adjacent to the second lumbar vertebra extending up to its junction with the inferior vena cava.

Based on the clinical course, the history of vertebroplasty, and the imaging characteristics, the diagnosis was pulmonary cement emboli following venous cement extravasation. Given that the patient has been asymptomatic without complicating pulmonary features, prophylactic anticoagulation was thought to be unnecessary. Interval surveillance imaging demonstrated no change in the position and size of PMMA to date, and the patient has done well without any long-term consequences.

Discussion

Despite its established safety and widely demonstrated efficacy (4–6), vertebroplasty does have some rare risks. The literature suggests a complication rate in the range of 1–10% (9,10). While major complications are rare, most complications are local including infection, pain exacerbation, bleeding at puncture site, transient radiculopathy, fractures, or cement leakage. Cement extravasation is the most common, comprising 30–65% of complications in patients with osteoporotic vertebral collapse, but significant adverse events occur in less than 1% of those patients (11,12). Cement leaks can have serious sequelae. For example, spinal extravasation can lead to cord and nerve injury as well as paravertebral soft tissue and intervertebral disc damage (1). Venous extravasation into the epidural and vertebral veins can cause cardiac and pulmonary cement embolization. Other severe reported complications include pulmonary hypertension, acute respiratory distress syndrome (ARDS), cardiac rupture, and death (13).

This case study highlights the uncommon, yet potentially serious vertebroplasty complication of PMMA

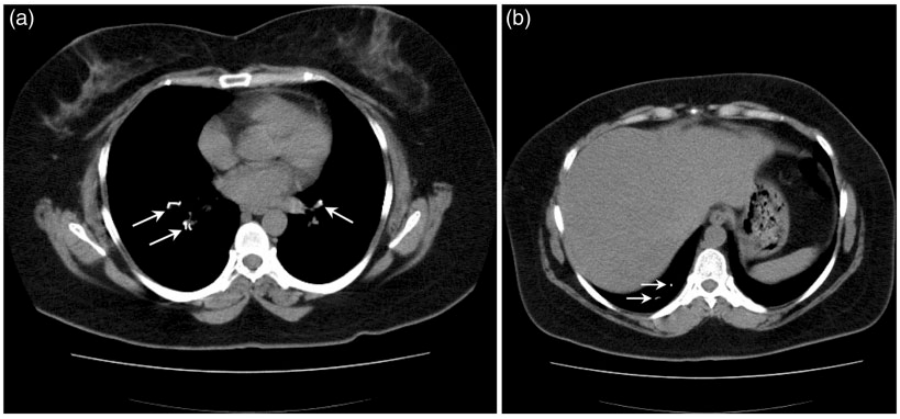

Fig. 2. (a, b) Select axial images from non-contrast chest at bone window revealing several small vermiform high-attenuation cement emboli in pulmonary artery branches at different slice levels.

cement pulmonary embolism. Of the frequent cement extravasation events, up to 24% are venous leaks, and only 4.6–6.8% lead to consequent cement pulmonary embolism according to early observational reports (2,14). A more recent large study specifically investigating post-vertebroplasty PMMA pulmonary embolism with chest CT screening determined an incidence of only 2.1% (15). Conversely, a sub-group of the Vertos II trial patients who were examined with chest CT scans had a frequency of 26% and similarly up to 23% in another study (2,16). The true incidence of pulmonary cement embolism after vertebroplasty is unknown because a mere 0.4–0.9% of the cases have symptoms (17), and asymptomatic patients are not routinely screened with postprocedure chest imaging. Another reason is that even with screening, the degree of identifying the embolic outcomes differs depending on the imaging study used; CT scans identify cement leakage more than chest radiographs by a factor of 1.5 (18).

An obvious cause for venous cement extravasation is the inadvertent needle placement into the basivertebral vein. The cement subsequently spreads through the segmental spinal veins, vena radicularis magna, azygous vein, and the accessory hemiazygous vein into the inferior vena cava, right cardiac chambers, and ultimately the pulmonary vasculature (19). Other risk factors that facilitate cement venous migration through this pathway include overfilling the vertebral body (9–15 mL), applying excess injection pressure, utilizing low-viscosity PMMA with insufficient polymerization (which we suspect in this case), and injecting at too many levels (20). Severe vertebral collapse, cortical damage, and increased vascularity are also associated with increased risk of PMMA embolic phenomena (8). Furthermore, anomalous vascular anatomy, lack of biplane fluoroscopy, and operator experience are important considerations.

Despite the potential severity of pulmonary complications, there are no established management guidelines for cement pulmonary emboli, and the current limited treatment approaches are non-evidence-based. Although pulmonary PMMA emboli are one of the most dreaded adverse events, most patients are safe and asymptomatic, and do not require any treatment besides clinical follow-up. In fact, the overall mortality of a PMMA embolus is <1% (21). It is unclear, however, if long-term consequences, such as the development of chronic pulmonary hypertension secondary to a burden of small peripheral emboli which we currently consider ''benign,'' will occur (2). In symptomatic patients, treatment options depend on the location and size of the embolus as well as the severity of symptoms. The cornerstone of therapy is anticoagulation for the purpose of reducing the risk of further thrombus formation on the embolized cement nidus. For central and symptomatic peripheral emboli, the recommended approach is initial heparinization followed by 6 months of warfarin therapy, which is similar to the treatment for venous thromboembolism. Surgical intervention and embolectomy are reserved for severe cases of massive central pulmonary emboli (22).

In conclusion, this report draws attention to the uncommon but potentially severe occurrence of pulmonary PMMA cement embolism following vertebroplasty. Patient selection should take into account this risk, especially in complex patients with multiple co-morbidities and diminished lung reserve. Unsafe techniques such as utilizing low-viscosity PMMA, overfilling the vertebral body, and applying excess injection pressure should be avoided to reduce the risk of cement extravasation. Lastly, clinicians should have a low threshold for diagnosing pulmonary PMMA embolism and providing appropriate anticoagulation therapy for symptomatic patients.

Conflict of interest

None declared.

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