An Incidental Finding of Pulmonary Cement Embolism Four Weeks After Vertebroplasty in a 50-Year-Old Man with Multiple Myeloma

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Patient: Male, 50-year-old
Final Diagnosis: Pulmonary cement embolism
Symptoms: Asymptomatic
Clinical Procedure: Vertebroplasty
Specialty: Pulmonology

Objective: Unusual or unexpected effect of treatment
Background: Vertebroplasty is a minimally invasive radiological procedure that involves injection of cement to stabilize the fractured vertebra. It has also been increasingly used to relieve pain in patients with bone-incorporated malignancies. The most frequently encountered complication of this procedure is inadvertent cement leakage, which has the potential to embolize. This report presents an incidental finding of cement embolism during fluoroscopy for a peripherally inserted central catheter (PICC) line 4 weeks after vertebroplasty in a 50-year-old man with multiple myeloma.

Case Report: Our report details the case of a 50-year-old man who presented for progressive sciatic nerve pain and was found on imaging to have an L3 fracture, spinal stenosis, lumbar spinal spondylosis, and diffusely decreased bone density, eventually diagnosed with multiple myeloma. For symptomatic relief and vertebrae stabilization, he underwent a CT-guided fluoroscopic vertebroplasty procedure. Four weeks later, during fluoroscopy for a PICC, he was incidentally found to have radiopaque opacities within the pulmonary arteries. He was diagnosed with a pulmonary embolism due to transvertebral cement leakage from his vertebroplasty. Given the central nature of his embolism, he was treated with anticoagulation and closely monitored. Throughout the treatment period, he remained asymptomatic with normal vital signs and NT-brain natriuretic peptide.

Conclusions: This report highlights the association between vertebroplasty and pulmonary cement embolism (PCE), the potential for late detection upon chest imaging even weeks after vertebroplasty, and suggests that some patients with PCE may need therapeutic anticoagulation.

Keywords: Kyphoplasty • Methylmethacrylate • Pulmonary Embolism • Vertebroplasty
Abbreviations: VP – vertebroplasty; KP – kyphoplasty; PMMA – polymethylmethacrylate; PCE – pulmonary cement embolism; VVS – vertebral venous system

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Background

Vertebral fractures can be caused by osteolytic tumors such as metastatic carcinoma and multiple myeloma [1]. Percutaneous vertebroplasty (VP) and kyphoplasty (KP) are 2 minimally invasive radiological procedures which have become standard of care in the treatment of such fractures [1-3]. Both procedures involve the injection of polymethylmethacrylate (PMMA), a viscous, cement-like polymer into the affected vertebral bodies under image guidance [1-3]. Multiple studies have documented the pain-relieving effects of these procedures, leading to the growing popularity of both VP and KP [4,5]. Nonetheless, complications can occur, most commonly from leakage of the PMMA into the surrounding tissues [6,7]. Cement leakage after VP occurs in 30-75% of cases, whereas leakage occurs less frequently after KP (8-33%) [8]. Migration of cement into the vertebral venous system can lead to pulmonary cement embolism (PCE), which has been reported in 2.1-26% of cases [9]. This report presents an incidental finding of cement embolism during fluoroscopy for a PICC line detected 4 weeks after vertebroplasty in a 50-year-old man with multiple myeloma.

Case Report

Our patient was a 50-year-old man with a past medical history significant for hypertension and obesity who presented with progressive sciatic nerve pain of the right lower extremity. Computed tomography (CT) of the spine revealed an acute-to-subacute fracture of L3 as well as other mild compressive fractures in the vertebrala, with congenital spinal stenosis, lumbar spinal spondylosis, and diffusely decreased bone density with scattered lucencies. The patient was soon thereafter diagnosed with multiple myeloma. For symptomatic relief and vertebre stabilization, he underwent a CT-guided fluoroscopic vertebroplasty procedure.

Details of the procedure were as follows: the patient was placed in the prone position and eleven-gauge parapedicular VertePort access needles were directed into the lateral aspects of the vertebral bodies. A Stryker curette was advanced through each access cannula and directed medially into the center of the vertebral bodies to displace the adjacent bone. Stryker PMMA composite was injected into the vertebral bodies under real-time fluoroscopic guidance. During the procedure, there was no reported extravasation of cement anteriorly into the ventral epidural space or into the disc spaces and there were no other identified procedural complications.

Four weeks later, the patient underwent fluoroscopy for PICC line placement. Briefly, PICC lines are used in patients who require venous access for weeks-to-months – often for long-term medication administration – as was the indication in this patient [10]. During fluoroscopy, he was incidentally noted to have radiopaque opacities within his pulmonary arteries (Figure 1). Upon further analysis, non-contrast CT of the chest demonstrated radiopaque material in the distal right main pulmonary artery extending into the right upper lobar anterior and posterior segmental arteries, the right middle lobar artery, and the right lower lobar arteries (Figure 2). Transthoracic echocardiography demonstrated a normal right atrial area, as well as normal right ventricular size and function. The patient was diagnosed with a PCE due to transvertebral cement leakage secondary to his vertebroplasty procedure 4 weeks prior.

Throughout the workup for the PCE, the patient’s vital signs remained stable. NT pro-BNP was within normal limits. At no point did he have signs or symptoms of right ventricular dysfunction. Specifically, he denied palpitations, lightheadedness, syncope, or lower-extremity swelling. Functionally, the patient was asymptomatic, denying exertional dyspnea or chest pressure.

Given the central nature of the patient’s embolism and coexisting thrombocytopenia, the vascular medicine consultants advised treatment with intermediate-dose oral anticoagulation. The patient continued to follow up as an outpatient in the oncology clinic and remained asymptomatic without further need for intervention.
Discussion

Here, we: i) highlight the procedural technique of vertebroplasty compared to similar procedures, ii) discuss potential adverse events related to the procedure, iii) detail relevant physical anatomy to better characterize how embolism may occur, iv) describe risk factors that may lead to increased incidence of embolism, v) discuss the role of imaging in diagnosis, and lastly, vi) review treatment considerations for PCE.

Vertebroplasty Versus Kyphoplasty Technique

Galibert and colleagues first described vertebroplasty for use in treatment of cervical vertebral angioma [2]. Shortly thereafter, application of the procedure was expanded to use in lytic bone disease and osteoporosis, with numerous documented effects on pain relief [3]. Briefly, vertebroplasty involves insertion of a needle between the head of the rib and the lateral aspect of the pedicle, using fluoroscopy to guide the insertion [1]. Contrast material is injected to confirm that the needle is not positioned in the path of venous flow, at which point liquid PMMA is injected into the vertebra [1].

Kyphoplasty has equivalent indications to VP, with a similar initial setup [1]. However, in kyphoplasty, after the needle is inserted, a bone tamp and balloon are introduced into the vertebral body [1]. When inflated, the bone tamp compresses the cancellous bone and expands the vertebral body, creating a cavity [1]. The balloon is thereafter deflated and removed. This technique creates a lower-pressure cavity into which the PMMA is then injected [1].

Adverse Events & PCE

Serious adverse events from both VP and KP have been reported, such as myocardial infarction, hematoma, neurologic deficits, and infection, although they are uncommon [11-13]. The most frequently encountered complication of these procedures is inadvertent cement leakage [14]. In the VERTOS II trial, a prospective, multicenter, randomized control trial comparing VP with conservative therapy in 202 patients, 43% of injected vertebrae demonstrated cement leakage immediately after the procedure [14]. Notably, all patients with identified leakage remained asymptomatic [14]. While cement leakage is also reported in KP, it occurs less frequently, presumably due to creation of a lower-pressure cavity by balloon compression and use of higher-viscosity cement [15].

Pulmonary cement embolism is a noted complication of both procedures, but happens more frequently in VP compared to KP [16]. Because patients do not routinely undergo chest radiography after the procedures and most remain asymptomatic, the true incidence of PCE is unknown. In the VERTOS II trial, follow-up CT scans 22 months after VP detected PCE in 26% of patients [14]. Similar rates of PCE after VP have been reported in other studies [17].

Anatomy

A thorough appreciation of the vertebral and perivertebral anatomy is crucial to understanding how postprocedural embolic complications occur. Within and around the vertebral column lies the vertebral venous system (VVS), a large valveless venous network that extends from the sacrum to the foramen magnum [18]. The VVS can be divided into 3 intercommunicating divisions: the internal vertebral venous plexuses, the external vertebral plexuses, and the basivertebral veins [18].

The basivertebral veins lie largely within the vertebrae and, via transverse branches, communicate with the internal and external venous plexuses [18]. Presumably, during VP the high-pressure injection of liquid cement into the collapsed vertebral cavity causes cement leakage into these basivertebral veins. The basivertebral veins in turn communicate with the external venous plexuses, which, through numerous connections by way of the ayzygos and lumbar veins, lead to the superior and inferior vena cava [18]. Thus, migrating cement may make its way through the venous system, through the right side of the heart and into the pulmonary artery. Iwanaga and colleagues...
also demonstrated that injections into the lumbar vertebral bodies, specifically, may drain more directly into the inferior vena cava system and bypass the vertebral venous plexus system of the vertebral canal, causing an alternate route for cement embolism after VP [19].

Risk Factors

Multiple groups have studied risk factors that may influence the likelihood of cement leakage from the vertebral body into the surrounding tissue after KP and VP [6,7,20-23]. Specifically, they examined factors such as the volume and viscosity of the injected cement, the intravertebral pressure, and the underlying medical diagnoses that can increase the chances of cement leakage. In a retrospective review of 71 patients after KP, Ren et al determined that the volume of PMMA injected into the vertebrae correlates closely with the complication rate. Particularly, the average injected cement volume was greater in the cement leakage group (5.34 mL) than in the group with no leakage (4.17 mL) [20].

PMMA viscosity has also been associated with leakage risk. Krueger and colleagues suggest that the material should have a viscous, “toothpaste-like consistency” before injection. Using PMMA that is too thin or liquefied may risk leakage [21].

Weikopf et al studied intravertebral pressure in vitro with cadaveric spines. Using a pressure sensor, they detected an average intravertebral pressure nearly 3 times greater in VP compared with KP, possibly leading to increased leakage. However, in KP, they noted a distinct increase in the intravertebral pressure during the final stage of stabilization, when the injected volume exceeded the filling capacity of the preformed cavity, suggesting that special attention is needed to avoid overfilling the cavities in this final filling stage, as it can lead to increased risk of complications [22].

Prior similar studies have also shown that certain underlying disease states can impact likelihood of cement leakage [23]. Mansour et al analyzed 102 patients with cancer who underwent VP, showing that those with multiple myeloma were most likely to develop PCE, followed by those with breast cancer, and then lymphoma [23]. Based on these findings, our patient may have had comparatively higher risk for PCE given his recent multiple myeloma diagnosis.

Imaging

Specific PCE patterns have been well-described on imaging. On X-ray, the presence of scattered dense opacities that form tubular branches in the lung fields are indicative of PCE [17]. On CT scan, PCE is marked by an intraluminal branching pattern with hyperattenuation of the pulmonary vasculature [17]. Given its variable reported incidence, some groups have suggested incorporation of postoperative imaging to assess for PCE.

Bliemel et al used postoperative anterior-posterior and lateral plain radiographs of the chest after KP, finding PCE in only 1 out of 94 patients [24]. Given the low incidence, they concluded that postoperative chest X-rays are not indicated for asymptomatic patients [24]. Venmans et al found CT evidence of PCE in 26% of patients treated with VP [14]. Longitudinal follow-up demonstrated that the cement emboli remained intact without any inflammatory response, and the patients remained asymptomatic. The group concluded that asymptomatic patients do not need postoperative CT scans [14].

Treatment

There are currently no standard treatment guidelines for PCE. Krueger et al conducted a systematic review on PCE following either KP or VP and suggested a therapeutic strategy based on the location of the embolus (central vs peripheral) and the presence of symptoms [21]. They recommend that asymptomatic patients with peripheral PCE should be monitored for evolution of symptoms without therapeutic anticoagulation [21]. In cases of symptomatic peripheral or central emboli, Krueger and colleagues recommend therapeutic anticoagulation with intravenous unfractionated heparin followed by 6 months of warfarin therapy to minimize further thrombotic development [21]. Continuous anticoagulation after the initial 6 months of treatment is generally contraindicated, largely due to the bleeding risks imposed on what is often an older patient population [21]. In massive central and symptomatic PCE, surgical embolectomy may be considered in select cases [21].

To the best of our knowledge, no studies have examined why some patients become symptomatic and others remain asymptomatic from PCE. Krueger et al (2009) found that 11 reports detailed 18 symptomatic PCE cases in 1430 patients [21]. In this group, symptomatic patients most commonly presented with dyspnea. Further, 25 reports detailed 62 cases of asymptomatic PCE in 3774 patients. The authors speculated that size and proximal location of the embolic fragment(s) and presence or absence of underlying heart disease can impact the severity of the clinical presentation, although symptoms and absence of symptoms have been reported with both central and peripherally located emboli [21]. Indeed, our patient remained asymptomatic despite a significant proximal embolus. Certain rare presentations of embolization after VP are likely to produce symptoms, such as lodging of emboli within the heart. Cement fragments as small as 10-20 mm have been reported to cause right ventricular perforation leading to tamponade [25]. Notably, studies have demonstrated that incidentally found asymptomatic PCE do not increase mortality risk [26]. Nonetheless, future research should examine why
some patients with PCE present with symptoms while others remain symptom-free.

Conclusions

Pulmonary cement embolism can occur after vertebroplasty due to transvertebral cement leakage and may remain unrecognized even until weeks later. Our patient with a centrally located PCE treated with oral anticoagulation remained asymptomatic 3 months after vertebroplasty. Multiple prior studies have documented similar presentations, diagnoses, and clinical courses to that of our patient, specifically identifying PCE on incidental imaging in asymptomatic patients following vertebroplasty [14,23]. We further highlight the association between vertebroplasty and PCE, offer an evidence-based explanation for how PCE occurs and factors that may increase PCE risk, and suggest that some patients with centrally located PCE, or those who are symptomatic, may be considered for short-term anticoagulation.

Declaration of Figures’ Authenticity

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