

Pulmonary cement embolism after percutaneous vertebroplasty (RCD code: II-1C.0)

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Abstract

Pulmonary cement embolism is a relatively new medical issue. The cement used in orthopedics is not even mentioned in the 2014 European Society of Cardiology (ESC) guidelines on the diagnosis and management of acute pulmonary embolism, however it will be a more frequent occurrence as minimally-invasive orthopedic procedures become more commonplace. An 81-year-old female was admitted to the emergency room with syncope. She had reported similar episodes of loss of consciousness and mild dyspnea for a few years prior to hospital admission. In 2008 the patient had undergone percutaneous vertebroplasty due to vertebral compression fractures. Her chest radiograph revealed multiple calcifications along the pulmonary vessels. Pulmonary computed tomography angiography confirmed pulmonary cement embolism. The presence of coexisting thrombus in one of the branches of the pulmonary artery was also revealed. The patient was administered enoxaparin subcutaneously and discharged. After 3 weeks repeat echocardiography showed a slight reduction in the dimensions of the pulmonary artery and its branches. Our case demonstrates that pulmonary cement embolism after percutaneous vertebroplasty may coexist with thrombotic embolus. Screening chest radiography after procedures using medical cement should be considered. Long term anticoagulation seems to be appropriate after pulmonary cement embolism. JRCD 2016; 2 (8): 266–269

Key words: pulmonary embolism, hilar calcifications, percutaneous vertebroplasty

Review of the literature

Pulmonary artery (PA) embolism is usually caused by lower extremity deep vein thrombosis (DVT). Less often, there is some other embolic material, such as fat (especially after pelvic bone fractures), air (iatrogenic) or neoplastic cells. According to the 2014 European Society of Cardiology (ESC) guidelines on the diagnosis and management of acute pulmonary embolism foreign bodies can also be the cause, for instance silicon, broken catheters or guidewires and intravascular stents [1]. In this article we discuss the case of pulmonary embolism caused by medical cement used by orthopaedic surgeons in percutaneous vertebroplasty (PV). PV is a minimally-invasive procedure invented by Galimbert et al. in 1987 [2]. Cement is injected into a fractured vertebra through a small incision to relieve pain caused by compression fractures. In the Pub Med database there are 299 results for the phrase: “pulmonary cement embolism” (as of January 20, 2016). The incidence of pulmonary cement embolism (PCE) varies

between 2.1 and 26% [3,4]. The largest percentage of procedures complicated by PCE was recorded after treatment of osteoporotic fractures. Patients are usually asymptomatic. In a systematic review of 20,000 patients who had undergone PV, PCE was found in 86 of them whilst 18 had some symptoms. The most common were dyspnea, tachypnea, tachycardia, cyanosis, hemoptysis, dizziness and excessive sweating [5]. Some patients can develop respiratory failure resulting in death [6]. The easiest way to diagnose PCE is postoperative chest radiography of the lungs, but as yet there is no recommendation to perform this exam in all patients after PV. There is also a lack of guidelines on the treatment of PCE. The authors of the publications described different therapeutic strategies in their case reports. Usually, patients were administered heparin or a coumarin. Sometimes, especially in asymptomatic cases, an observation with no treatment strategy was chosen. There are also publications describing percutaneous removal of cement from the PA via femoral vein cannulation. One case turned out to be fatal despite the successful percutaneous retrieval of a large

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Figure 1. Chest radiograph; postero-anterior view obtained on admission. Radiodense tubular, branching lines over the right hilum of the lung corresponding to the medical cement in the pulmonary arteries

cement fragment [7,8]. In a few cases surgical treatment was necessary. Rothermich et al. described the case of a 29-year-old male who had been admitted to hospital because of severe pleuritic pain and shortness of breath eight days after PV. Computed tomography (CT) had revealed massive PCE and pulmonary infarction of the right lower lobe. Due to respiratory failure thoracotomy with embolectomy and resection of the right lower lobe had been performed. Radical improvement was seen as a result of this treatment [9]. There are still no studies comparing the effectiveness of different treatment strategies. Krueger et al. tried to systematize the indications for each type of therapy. They divided patients with PCE into four groups. According to the authors, those with peripheral embolism (based on chest radiography) and without any symptoms do not need any treatment. Patients with peripher-

al but symptomatic PCE and those with central but asymptomatic PCE should be administrated anticoagulation (heparin followed by a coumarin) for 3–6 months after occurrence of the embolism. In cases of central and symptomatic embolism surgical or percutaneous removal should be considered [5].

Case presentation

An eighty-one-year-old female with a history of hypertension, diabetes and Hurthle cell thyroid cancer treated with strumectomy in 1972 was admitted to the emergency room with syncope. Years later, in 1996, spinal metastases were diagnosed and radiotherapy was performed. In 2008 the patient underwent percutaneous vertebroplasty because of pathological fractures of five vertebrae (L3 to Th11). She had reported similar episodes of loss of consciousness and mild dyspnoea for several years prior to hospital admission. The patient was obese with a body mass index (BMI) of 32.7 kg/m². On physical examination her blood pressure and heart rate were 110/50 mm Hg and 75 beats/min respectively. There was a vesicular murmur on auscultation. The haemoglobin oxygen saturation was 95%. The laboratory blood tests revealed raised D-dimers (1279H ng/ml [0 – 500]) and C-reactive protein (CRP) (17,8 H mg/l [0,0 – 5,0]). N-terminal prohormone of brain natriuretic peptide (NT-proBNP) level was 938 pg/ml [0–125] and high sensitive troponin T was 13,64 pg/ml [3,0–14,0]. The electrocardiogram (ECG) showed sinus rhythm with left axis deviation and right bundle branch block (RBBB). Chest radiograph showed several tubular high-density opacities suspected to be the medical cement along the expected course of the PAs, especially in the right lung (Figure 1). In the Emergency Room bedside echocardiography was performed revealing enlargement of the PA to 29 mm (Figure 2) and the right ventricle to 41 mm. The branches of the PA were also extended (the right one to 26 mm and the left to 23 mm). Mild tricuspid valve regurgitation was found with a maximal pressure gradient of 35 mmHg. In spite of chronic kidney disease (estimated glomerular filtration rate [eGFR] = 34,8 ml/min/1,73m²) pulmonary CT angiography was carried

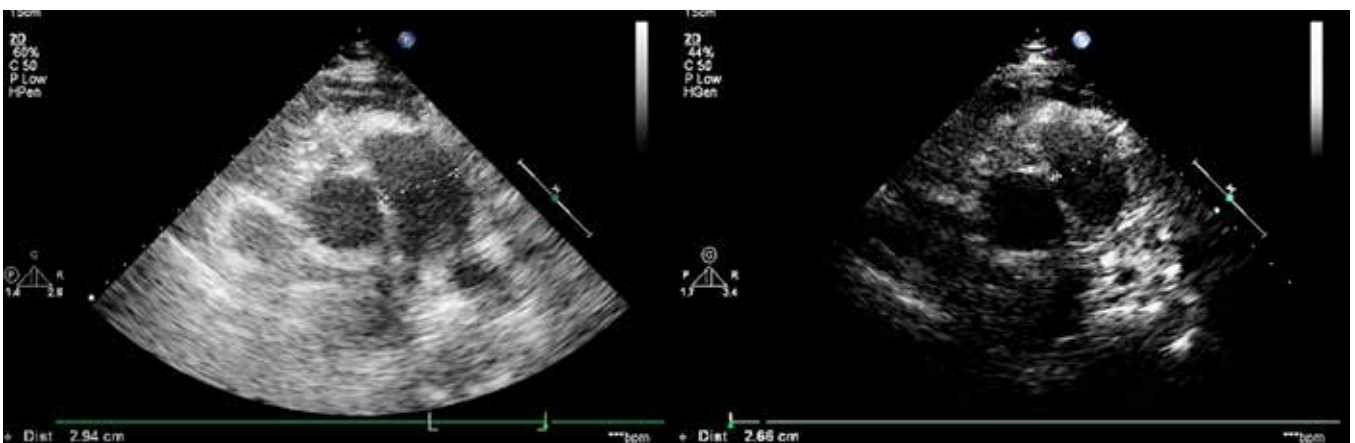


Figure 2. Transthoracic echocardiogram; left parasternal short axis view. On the left – extension of the pulmonary trunk (2,94 cm) obtained on admission. On the right – slightly smaller dimensions of pulmonary artery (2,66 cm) obtained three weeks after discharge

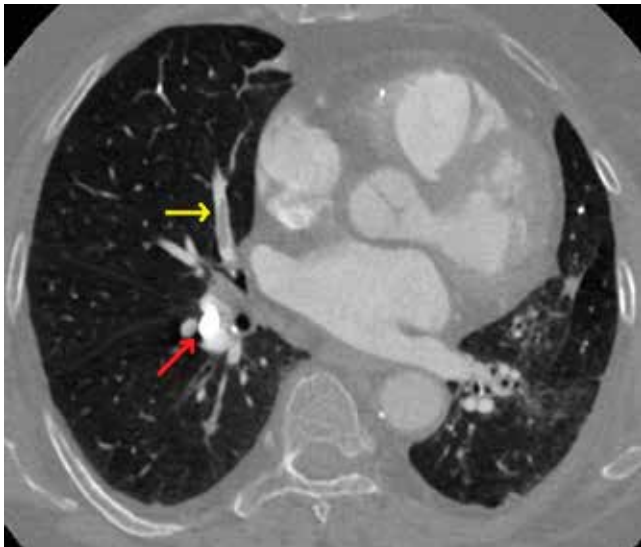


Figure 3. Pulmonary computed tomography angiogram; axial plane. Medical cement emboli in the right lower lobe artery and its branches (red arrow). Thrombus in the proximal section of the artery supplying the 5th segment (yellow arrow)



Figure 5. Pulmonary computed tomography angiogram; axial plane. Medical cement emboli in the right lower lobe artery and in the segmental arteries. Extension of the pulmonary trunk (29 mm), right pulmonary artery (28 mm), and left pulmonary artery (24 mm)



Figure 4. Pulmonary computed tomography angiogram; coronal plane. Medical cement emboli in the right lower lobe artery and in the segmental arteries

out confirming the diagnosis of PCE and showing the presence of the coexisting thrombus (Figure 3–5).

Patient management and follow-up

The patient was admitted to a department of cardiology. 24-hour Holter monitoring of the ECG and head CT scan did not show any significant pathology. Transthoracic echocardiography was carried out. The result was as follows: left ventricular diastolic diameter (LVDD): 49 mm, left ventricular systolic diameter (LVSD): 34 mm, right ventricular diastolic diameter (RVDD): 41 mm, inter-

ventricular septum diastolic diameter (IVSDD): 11 mm, interventricular septum systolic diameter (IVSSD): 14 mm, posterior wall diastolic diameter (PWDD): 12 mm, posterior wall systolic diameter (PWSD): 14 mm, right ventricular wall thickness (RVWT): 9 mm, aortic root (AoR): 30 mm, ascending aorta (AAo): 33 mm, PA: 30 mm, left PA: 23 mm, right PA: 26 mm, left ventricular ejection fraction (LVEF): 50%. No mitral regurgitation was found. Maximal pressure gradient through aortic valve was 7 mm Hg. No significant regurgitation was found. Mild tricuspid regurgitation was visualised with maximal pressure gradient of 35 mm Hg. No pericardial effusion was detected. Following consultation with a cardiothoracic surgeon the patient was considered as too high risk for surgery. Percutaneous removal of cement from the PA via femoral vein cannulation was discussed with the interventional cardiologists, however because of the stable nature of the patient and absence of severe dyspnea, conservative treatment was chosen. The patient was administered 100 mg enoxaparin once a day subcutaneously and discharged home. After three weeks she was admitted electively for follow up evaluation. The patient was stable with no dyspnea at rest. Echocardiography showed slightly smaller dimensions of the PA and its branches (trunk: 26,6 mm, right artery: 26 mm, left artery: 20 mm) (Figure 2). The estimated pressure gradient was also slightly lower (32 mm Hg). The patient reported further temporary episodes of loss of consciousness after discharge. We recommended heparin treatment and further cardiologic and neurologic assessment in an outpatient department. The 24-hour Holter ECG monitoring was repeated. Bifascicular block with intermittent first degree atrio-ventricular conduction block was recorded. In accordance with the 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy our patient was given a dual chamber pacemaker. There have been no syncopal episodes in further follow up.

During her second hospitalization the patient brought additional documentation with details of her past medical history. These showed that she had been hospitalized on the pulmonary ward in May 2009 because of a chronic dry cough and “multiple calcifications in the hilum and along bronchi of the right lung” were seen on the chest radiograph. A CT scan of the chest with contrast was performed. The lung lesions were described as longitudinal calcifications in the pulmonary tissue along the vessels mainly in the hilum of the right lung. It was about ten months after the patient had undergone PV and the calcifications were probably the emboli from the medical cement. We managed to obtain past chest radiographs of our patient. One X-ray was performed before the PV while the latter was from about fifteen months afterwards. These images clearly show the causal relationship between PV and the PCE.

Conclusions

PCE is a relatively new phenomenon but as the number of PVs performed increases cement embolism will become more common. It is crucial that physicians of different specialties are aware of PCE. Our case demonstrates that PCE can often be evaluated by respiratory physicians and that making correct diagnosis in these patients can be difficult. The problem of PCE requires commitment to proper methods of prophylaxis, and both diagnostic and treatment standards.

In order to increase the traceability of PCE it is worth considering performing chest radiograph the day after every PV procedure. This seems to be important even in primarily asymptomatic patients. Our patient is a good example that symptoms can occur a lot later on (she was hospitalized in the pulmonology department about ten months after PV). Early diagnosis may increase the chances of removal of cement emboli by percutaneous procedure before it gets covered by endothelial cells. We suggest that patients diagnosed with PCE should be periodically assessed by a cardiologist in case pulmonary hypertension and right ventricular heart failure occur. Our case demonstrates that PCE can coexist with thrombi. We cannot confirm that the cement caused thrombus formation in PAs but it is very likely that, as a foreign body, cement has prothrombotic properties. In those individuals with coexisting thrombi we suggest long-term anticoagulation no matter whether it is a central, peripheral, symptomatic or asymptomatic cement embolism.

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