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Pulmonary cement embolism: a rare complication of vertebroplasty

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Abstract

This comprehensive case study aimed to highlight the condition of pulmonary embolism (PE), which despite its relative obscurity, may be more common than initially assumed. PE frequently occurs in the Emergency Department. PE along with deep vein thrombosis are the two most prevalent forms of venous thromboembolism (VTE). VTE ranks as the third most common cardiovascular condition encountered in the Emergency Department, following heart attacks and strokes. Acknowledging the symptoms of VTE and acting on them early are important to prevent chronic manifestations and heart failure in some cases owing to persisting pulmonary hypertension. One of the most frequent causes of VTE is surgery, which often occurs owing to immobilization. However, in some cases, VTE is due to the procedure itself or due to what is used in the procedure or injected in the patient. We report the case of a patient who complained of dyspnea for some time before diagnostic steps were taken, such as computed tomography and echocardiography. This case report describes the complication of PE due to vertebroplasty, which is well described but underdiagnosed because it may not be considered. More focus on this complication during follow-up could help diagnose and treat patients more rapidly.

Keywords

Pulmonary embolism, hypertension, vertebroplasty, cement embolism, anticoagulation, surgery, venous thromboembolism

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Background

Pulmonary embolism (PE) frequently occurs in the Emergency Department. PE and deep vein thrombosis are the two most prevalent forms of venous thromboembolism (VTE). VTE ranks as the third most common cardiovascular condition encountered in the Emergency Department, followed by heart attacks and strokes.[1]

In certain circumstances, patients with similar symptoms, clinical profiles, and radiological findings with a diagnosis of PE may present with pathophysiological nuances and underlying causes that deviate from the typical scenario. We report a case of a woman who was admitted to hospital because of headaches, dyspnea, orthopnea, and swelling in both lower extremities. An investigation showed deterioration in renal function, elevated concentrations of uric acid and potassium, and a reduced hemoglobin value and platelet count. A computed tomography (CT) scan of the thorax, abdomen, and pelvis identified the presence of embolic material. Her symptoms of dyspnea started not long after her vertebroplastic operation and typical radiology findings suggested cement emboli/chronic thromboembolic pulmonary hypertension (CTEPH). This comprehensive case study aimed to highlight the condition of PE, which despite its relative obscurity, may be more common than initially assumed.

Medical history

The patient (de-identified) was a woman in her 70s who was diagnosed with chronic lymphocytic leukemia (CLL) in Binet stage 1 and remained untreated until her current presentation. Her medical history included previous cervical cancer (hysterectomy in 1974), hypertension, renal failure, psoriasis, gastric ulcer, and chronic gastritis (*Heliobacter pylori*-positive in December 2020), along with osteoporosis and a potential diagnosis of chronic obstructive pulmonary disease. Additionally, she had a history of anxiety and depression. She had smoked since the age of 16 years, and at hospitalization, she was smoking five cigarettes each day (previously 1 pack/day) and had a total of approximately 50 pack-years. The patient had a body mass index of 38 kg/m^2, height of 160 cm, and weight of 97 kg.

In the fall of 2020, she was admitted to hospital after sustaining an osteoporotic fracture of L1. The surgical intervention involved fixation of Th11–L3 and an injection of a bone substitute via a vertebral body stent, resulting in a satisfactory initial outcome. She had slight dyspnea, which was thought to be due to overweight, smoking, and immobilization.

In late June 2021, the patient was readmitted to the Oncology Department owing to the sudden onset of headaches, worsening of dyspnea, orthopnea, and swelling in both lower extremities. Her known renal failure showed signs of deterioration, along with elevated concentrations of uric acid and potassium, accompanied by mild thrombocytopenia and anemia. Laboratory results indicated a C-reactive protein concentration of 58 mg/L (reference: <6 mg/L), white blood cell count of 3.79×10^9/L (reference: 3.5–11.0 $\times 10^9$/L), platelet count of 66×10^9/L (reference: 150–400 $\times 10^9$/L), hemoglobin value of 8.4 g/L (reference: 11.7–15.3 g/L), estimated glomerular filtration rate of 30 mL/minute/1.73 m^2 (reference: 63–108 mL/minute/1.73 m^2), creatinine concentration of 163 μmol/L (reference: 45–90 μmol/L), uric acid concentration of 738 μmol/L, N-terminal pro-B-type natriuretic peptide concentration of 2227 ng/L (reference: <300 ng/L), and D-dimer concentration of 1.0 mg/L FEU (reference: <0.5 mg/L FEU). Initially, there was suspicion of spontaneous tumor lysis syndrome or exacerbation of CLL.

A CT scan of the thorax, abdomen, and pelvis was conducted to investigate the

possibility of transformation into aggressive lymphoma. This scan showed no signs of lymphoma but indicated the presence of embolic material in both lungs. Cement emboli were suspected because of the observed high density, along with an increased right/left ventricle ratio indicative of pulmonary hypertension, possibly resulting from vertebroplasty performed in Th11–L3. Additionally, enlarged lymph nodes in the liver hilum and conglomerates in the splenic hilum, consistent with CLL, were observed (Figures 1 and 2).

Consequently, the patient developed respiratory failure necessitating oxygen support. Echocardiography demonstrated well-functioning right ventricular function without signs of dilation or right heart strain. Mildly dilated atria were noted, along with evidence of severe pulmonary hypertension, and the systolic pulmonary artery pressure was estimated to be in the range of 65 to 70 mmHg.

The renal failure was diagnosed as pre-renal and managed with fluid therapy, while the hyperkalemia was treated by a glucose–insulin infusion, sodium zirconium cyclosilicate, and patiromer. Although allopurinol was initiated to reduce uric acid concentrations, it had to be discontinued owing to the development of urticaria. Low-molecular-weight heparin was administered at a reduced dose of 2500 IU twice daily to manage thromboemboli associated with renal failure. After the patient's condition stabilized, she was transferred to Oslo University Hospital Rikshospitalet 6 days later for further evaluation and treatment of the cement emboli. A subsequent echocardiogram showed satisfactory left and right ventricular function, with minor tricuspid insufficiency and only a slight increase in pulmonary circulation pressure.

The parameters measured during right heart catheterization were as follows: right

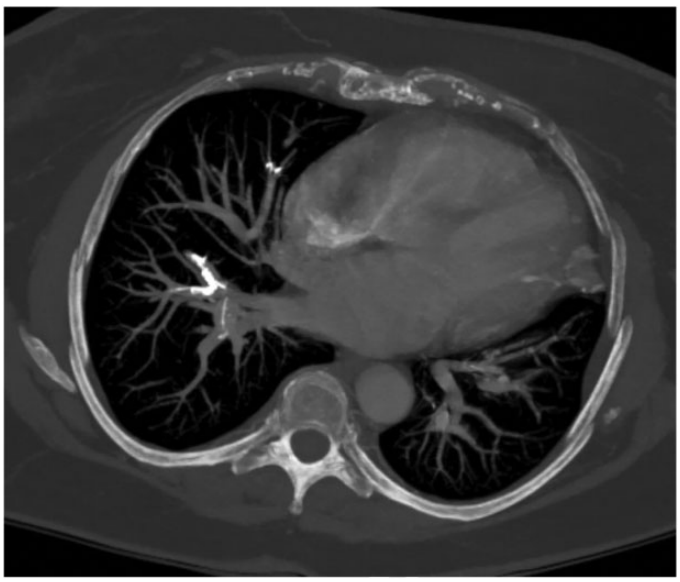

Figure 1. A computed tomography scan shows material with a higher density (depicted in white) than the contrast in various pulmonary arteries. Thrombotic material typically shows a lower density (depicted in black) than the contrast.

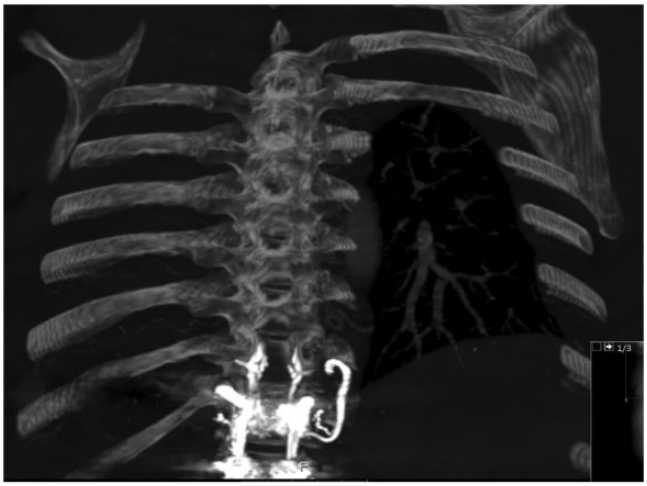

Figure 2. A computed tomography scan shows material with a higher density (depicted in white) than the contrast in the paravertebral vein.

atrial pressure was 6 mmHg, mean pulmonary artery pressure was 26 mmHg, central venous pressure was 10 mmHg, the arterial–venous oxygen difference was 34.4, the cardiac output/cardiac index (L/minute)/(L/minute/m^2) was 8.0/4.0, and pulmonary artery resistance was 2.0 Wood units. These findings indicated excellent cardiac output and normal resistance, with only moderately elevated pressure in the pulmonary circulation. Pulmonary angiography showed no dilation of proximal vessels. Peripheral perfusion appeared generally good bilaterally, with potentially small changes suggestive of subsegmental thromboembolism on the right side, although this remained unclear.

During a meeting for CTEPH, the patient's condition was thoroughly discussed. No thromboembolic substrates for balloon dilatation were identified, and several of her comorbidities were thought to have contributed to the pronounced hypoxia and dyspnea, including a potential respiratory infection. Subsequently, the patient was transferred back to Kalnes Hospital without any interventions or modifications to her medication.

After her discharge, she underwent a lung assessment and underwent a pulmonary function test, which showed a pulmonary forced vital capacity of 83% of the predicted value, forced expiratory volume in 1 s of 64% with an obstructive pattern, and total lung capacity of 59% of the expected value. Following the inhalation of ipratropiumbromid and salbutamol, there was a minimal improvement in the forced expiratory volume in 1 s by 8%. The test results led to the diagnosis of chronic obstructive pulmonary disease stage 2, and smoking cessation was recommended.

In December 2021, she attended a 1-year follow-up at the Orthopedic Department at Kalnes Hospital. During this visit, she reported considerable back pain, a reduced walking distance of 100 m, and instability in her legs, resulting in substantial back pain. No resting pain was reported. Radiographically, an X-ray showed a greater kyphosis angle than that in the postoperative state, and the rods had bent downward, with no other notable changes. She has been referred for reassessment to the operating unit in the Surgical Department at Oslo University Hospital

because of her escalating back pain, minimal walking distance, and potential complications related to cement emboli after the spinal surgery. Currently, she has experienced progression in her CLL, prompting the initiation of treatment with venetoclax.

Discussion

Percutaneous vertebroplasty is an intervention for osteoporosis-induced vertebral compression fractures, and vertebroplasty and kyphoplasty are the two most commonly used methods. This case report aimed to shed light on an underdiagnosed complication – PE, where the risk appears to be highest with vertebroplasty.

During these procedures, polymethylmethacrylate (PMMA), or bone cement, is injected into the targeted vertebra. Complications in the form of PE with bone cement are not uncommon, and have been reported in approximately 3% to 23% of patients after the procedure. This complication may lead to mechanical occlusion, resulting in symptoms, such as dyspnea and pain, typically manifesting weeks to months after the procedure. Importantly, a considerable proportion of patients are asymptomatic and incidentally detected during follow-up. Nevertheless, according to the literature, this complication can be severe, with at least six reported deaths.[2,3]

The pathophysiology of PE with bone cement involves the accidental or unfortunate extravasation of cement into the valveless venous plexus around the vertebra, subsequently entering the venous system in the chest region. PMMA rapidly solidifies upon injection, and early or rapid injection before the substrate begins to harden increases the risk of its spread into the venous system.[3] While previous studies have indicated that PMMA does not induce platelet aggregation or the formation of additional clots *in vitro*,[4] uncertainties persist regarding whether PMMA is prothrombotic by damaging the endothelium *in vivo*. This uncertainty could potentially lead to further thrombosis and exacerbation. Consequently, this may have therapeutic implications, and the use of anticoagulation could prove beneficial.

In a study that comprised 75 patients who underwent vertobroplasty, an incidence of PE of 23% (18 patients) was observed, and the majority of patients were asymptomatic.[3] The emboli were typically identified in the distal part after the third branching of the pulmonary arteries. This study also highlighted that only leakage of cement into the inferior vena cava showed an association with PE. The highest risk was observed when the procedure was performed by non-radiologists. Cement embolism has also been reported to be higher in hip arthroplasty, and is related to a higher volume of cement.[3,5]

The treatment of cement emboli lacks an established evidence-based gold standard.[6] Decisions of treatment are guided by the patient's symptoms and associated risk factors. As a general approach, asymptomatic emboli are typically monitored without intervention, while symptomatic cases are managed on the basis of the severity of symptoms and the extent of embolization. Treatment modalities include anticoagulation, corticosteroids, antibiotics, percutaneous thrombus removal, cardiopulmonary bypass, and/or surgical extraction, and are often performed in combination. Although Krueger et al. attempted to devise a treatment algorithm, surgical removal of large emboli appears to be the only unequivocal recommendation.[6] The role of anticoagulation regarding the timing and duration remains unclear. Regarding other non-thrombotic emboli, such as fat or fluid emboli, the standard approach excludes anticoagulation. However, because cement embolism may promote further thrombosis due to endothelial damage and/or foreign body effects, limited anticoagulation could

be considered, especially when surgery is not indicated.[6]

Clinicians should be vigilant for the likely underdiagnosed complication of PE, which has been reported in 3% to 23% of cases.[2,5] Routine lung examinations with chest X-rays before discharge following vertebroplasty and during follow-up for all patients are advisable. In cases of newly developed or worsening symptoms, such as chest pain and dyspnea, a thoracic CT scan should be conducted to assess the possibility of embolism. This consideration is particularly crucial because surgery and immobilization inherently increase the risk of VTE.

The reporting of this study conforms to the CARE guidelines.[7]

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Author contributions

ET conceived the idea of the study and wrote the manuscript. APTH assisted with the English and helped review the manuscript. DOS helped interpret the radiological images and helped review the manuscript. NR and APTH were responsible for interpreting the echocardiographic findings and treatment of the patient.

Data availability statement

Not applicable.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Ethics statement

This case report did not require approval by an ethics committee. The patient has read the manuscript and provided written informed consent for publication.

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