Case report

Pulmonary cement embolism complicating percutaneous kyphoplasty: A case report

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A 55-year-old woman with a history of tobacco abuse, sural vein thromboses five years ago (normal blood clotting results), and endometriosis, presented to the hospital for atypical chest pain on the right side and two episodes of presyncope within the last two weeks. She underwent three weeks before a kyphoplasty at the level of L1 after a compression fracture because of a car accident. The procedure was done under biplanar fluoroscopy control. A pedicular

1. Background

Kyphoplasty (KP) and percutaneous vertebroplasty (PVP) are commonly used for the treatment of painful vertebral fractures due to traumatic injury, osteoporosis or metastatic lesions. During these so-called mini invasive procedures, a cement augmentation by injecting polymethylmethacrylate (PMMA) enhances the strength of a pedicle screw or a vertebra and achieves significant relief of symptoms. This technique of cement vertebral augmentation is increasingly used and consequently the complications are accumulating. Among them pulmonary cement embolism (PCE) is rarely described but can lead to serious outcomes [1]. Here we report a case of PCE after kyphoplasty and discuss clinical presentation, diagnostic procedures and potential anatomical and technical conditions leading to this complication.

2. Case presentation

A 55-year-old woman with a history of tobacco abuse, sural vein thromboses five years ago (normal blood clotting results), and endometriosis, presented to the hospital for atypical chest pain on the right side and two episodes of presyncope within the last two weeks. She underwent three weeks before a kyphoplasty at the level of L1 after a compression fracture because of a car accident. The procedure was done under biplanar fluoroscopy control. A pedicular
approach was used and fracture reduction was performed using a spine jack (diameter 4.2). After fracture reduction, six ml of PMMA were injected in the vertebral body. The procedure was uneventful. In the emergency room, the patient did not have fever, dyspnea, cough or hemoptysis. Breath sounds were normal bilaterally. Blood tests showed D-Dimer 0.29 μg/ml (<0.50 μg/ml), stable troponin <5 ng/ml, Brain Natriuretic Protein 14 ng/ml. Arterial blood gases analysis in ambient air showed pH 7.45, PaO₂ 99 mmHg, PaCO₂ 36 mmHg, bicarbonate 25 meq/l. A computed tomography scan of the chest was performed which showed spontaneously linear hyperdense foreign bodies in two segmental pulmonary arteries at the level of middle lobe and right lower lobe compatible with pulmonary cement embolism (Fig. 1). Thrombotic pulmonary embolism was ruled out. Because of the clinical presentation and patient’s past history of thrombotic events and after patient’s informed consent, an anticoagulation treatment was offered for a 6-month period.

3. Discussion and conclusions

Pulmonary cement embolism consists of the migration of cement through the pulmonary arteries during or after cement augmentation of a vertebral body. The incidence is variable from 3.5% to 28.6% according to the literature because PCE is frequently asymptomatic and the diagnosis depends on the realization of systematic postoperative imaging, i.e. chest radiograph or chest computed tomography (CT) which is a more sensitive test [2]. If a post-operative chest CT is not done, it is likely that a PCE can be missed and there is an underestimation of the true incidence of cement embolization [3]. If patients are asymptomatic, the diagnosis can be made weeks or months later. When happened, the symptoms are similar to a pulmonary embolism of thrombotic cause with dyspnea, tachypnea, chest pain and, in rare clinical situations, hypotension and loss of consciousness leading to cardiorespiratory arrest and death. However, imaging findings on CT scan are different. On the CT pulmonary angiography, the cement is a high-density material (more than 1000 HU) visualized as branching, tubular or multiple opacities different from hypodense thrombotic embolus. Pulmonary cement embolism is more common after PVP than after KP. In the treatment of vertebral compression fractures, a meta-analysis comparing PVP and KP reported a prevalence of PCE in 0.9% and 0.4% respectively but without statistically significance and with no distinction between different kinds of emboli [4]. An explanation should be that lower pressures are generated in KP procedure and that balloon before cement injection might create a cavity confining the cement in the vertebral body [4]. Moreover, even no relation has been shown with PCE, high volume and low-viscosity of injected cement are risk factors of local cement leak [1]. The use of PMMA with lower polymerization temperatures reduces this complication. Newer varieties of bone cement are in developments with greater viscosity and lower stiffness properties. Conversely, the number and the level (cervical, thoracic or lumbar vertebrae) of treated vertebrae during a procedure and the etiology of the fracture (malignant versus non malignant) are not risk factors of PCE [2]. Our patient underwent a KP under biplanar fluoroscopy control in order to overcome the difficulties to view real-time lateral vertebral leakage during the uniplanar procedure which is usually done. The procedure per se was uneventful and the delay between the procedure and the symptoms (3 weeks) leads to consider the necessity of imaging control after the procedure. There is no dedicated recommendation in this field. Despite mild symptoms systemic anticoagulation was delivered for 6 months taking into account history of repeated peripheral thrombotic events. There is no clear therapeutic protocol for the management of PCE. A recent study showed that incidentally found PCE had no significant impact on mortality risk [5]. The management of symptomatic patients remains uncertain. In case of severe symptoms, from the literature, anticoagulation is usually advised depending on the location (centrally, i.e. involvement of the main pulmonary trunk and/or the right or left main pulmonary arteries) and the size of the emboli but with poor-quality evidence and decision-making may be complex. In these cases a clinical follow-up is recommended. To summarize usually the recommendation is to follow the standard treatment guidelines for the treatment of thrombotic pulmonary embolisms [1].

Ethics approval and consent to participate

Not applicable in this section.

Fig. 1. Non-contrast chest computed tomography showing cement pulmonary emboli (arrows). There is no thrombotic cause. Lung parenchyma (no shown) was normal.
Consent for publication

Appropriate written informed consent was obtained for publication of this case report and accompanying images.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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CRediT authorship contribution statement

Romain Naud: Writing - original draft, Writing - review & editing, Data curation, Formal analysis. Julien Guinde: Writing - original draft, Writing - review & editing. Philippe Astoul: Writing - original draft, Writing - review & editing.

Declaration of competing interest

The authors declare that they have no competing interests.

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