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Case Report

# Pulmonary Cement Embolism Complicated by Pulmonary Thromboembolism During Vertebral Surgery - A Case Report

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## Background

Pulmonary Cement Embolism (PCE) is a rare adverse event, consequent to cement leakage during augmentation procedures in orthopedic surgery. Together with the Bone Implantation Cement Syndrome (BCIS), it constitutes the main complication in the use of cement in orthopedic surgery [1]. Bone cement augmentation of pedicle screws bares a risk of cement leakage both into the intraspinal space, causing radicular compression symptoms, neurologic deficit, or pain, and into the epidural veins, the cava vein, the pulmonary artery, or the right atrium, potentially causing PCE and right heart failure. Furthermore, the application of bone cement can cause an anaphylactoid reaction and in severe cases ends up in circulatory collapse. In addition, heat generated by polymerization can cause ischemia, radicular irritation and radiculopathy [2]. PCE has been described as a possible major complication of augmentation vertebral procedures like kyphoplasty (PKP) and vertebroplasty (PVP) [3], but also in Cement- Augmented Pedicle Screw Instrumentation (CAPSI) of the thoracolumbar spine [4]. In most cases PCE is undiagnosed and underreported [3] and it is often described as well tolerated and asymptomatic, thus requiring only basic medical management. We present the case of a patient who developed PCE, complicated by pulmonary thromboembolism, with a favorable outcome after undergoing vertebral surgery performed in May 2019 at the "Istituto Ortopedico Rizzoli", Bologna (Italy).

## Case Presentation

A 53-year-old man (height 182 cm, weight 115 Kg, BMI 34.8) underwent vertebral surgery under general anesthesia for a severe osteoporosis on the thoraco-lumbar spine. His past medical history showed a neurosurgical intervention for cavernoma excision from the third ventricle, iatrogenic diabetes insipidus, L4-L5 posterior arthrodesis, deep vein thrombosis following the neurosurgical intervention, and allergic asthma. An extended vertebral arthrodesis T10-L5, with cement-augmented pedicle screw instrumentation, was performed with the patient in prone position, in association with vertebral augmentation procedures (vertebroplasty of T9 and kyphoplasty of L4), implying the use of Polymethylmethacrylate (PMMA, bone cement). Total intravenous anesthesia was administered (propofol and remifentanil for induction and maintenance of anesthesia, check of anesthesia depth with bispectral index, control of the integrity of motor and sensory nerve pathways whit somatosensory evoked potentials and motor evoked potentials).

Cardiovascular monitoring was performed with EV-1000™ platform (Edwards Lifesciences) in order to manage the fluid therapy according Goal-Directed Fluid Therapy (GDFT) protocol. It was based on Stroke Volume Variation (SVV) with the aim of optimizing the fluid and transfusion therapy. At the beginning of surgery, vital signs were normal (HR 70, IBP 100/65 mmHg, SV 115, SVV 10%) and the prone position was well tolerated; the first Arterial Blood Gas Analysis (ABG) showed slightly altered gas exchanges with a $paO_2$ of 91 mmHg ($FiO_2$ was set at 0.5), $paCO_2$ 46 mmHg and Hb 13.3 g/dL. Immediately after the use of PMMA for augmentation procedures, a sudden hemodynamic instability occurred: arterial blood pressure reached the lowest value (50/35 mmHg) and a new ABG showed hypoxemia ($paO_2$ 66 mmHg) and an initial increase of $paCO_2$ (50 mmHg). The anesthesiologist administered first a rapid fluid resuscitation with crystalloids and

colloids and boluses of ephedrine, according the GDFT protocol. A rapid optimization of ventilation (increase of the tidal volume and $FiO_2$) was carried out. Unfortunately, Mean Arterial Pressure (MAP) remained under 60 mmHg, making a rapid supination of the patient necessary in order to allow a better cardiovascular and ventilatory management.

During resuscitation, vasopressor drugs were firstly used in boluses, but then norepinephrine in continuous perfusion was needed as cardiovascular support in order to reach a normal MAP (highest dosage: 0.1 mcg/Kg/min). Recruitment procedures of the lungs were also needed to obtain a satisfactory oxygen saturation. During resuscitation, analgesia, hypnosis and myoresolution were guaranteed with fentanyl, propofol and rocuronium. Furosemide was administered for diuresis contraction. A chest radiography (X-Ray) was performed in the operating room revealing left lower lobe atelectasis. After stabilization, $paO_2$ was 100mmHg with $FiO_2$ of 60%, $paCO_2$ 50 mmHg, and MAP was 65mmHg with minimal norepinephrine support. The surgeons ended the procedure and the patient was transferred to the Intensive Care Unit (ICU).

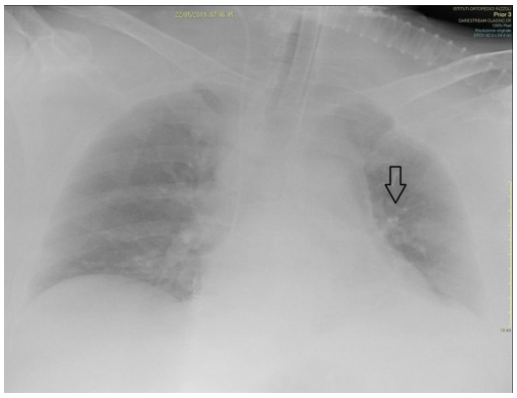

**Figure1:** Hyperdense imagine in the parahilum region of left lung suggestive of pulmonary cement embolism, detected on postoperative chest X-ray (arrow).

In the ICU, vital functions were supported with mechanical ventilation and norepinephrine infusion. An echocardiographic study was performed and showed a moderate dilatation of the right ventricle with a slight global hypokinesia and indirect signs of right overload. A new chest X-Ray revealed a hyperdense image in the parahilum region of the left lung, suggestive of PCE. Once vital signs were stable, a CT angiography was performed: cement emboli in the left upper parahilum region and in the left lung parenchyma were confirmed. Given the patient's critical state, a cardiac surgery consultation was required to evaluate the indication for embolectomy. The operation was considered unnecessary by the cardiac surgeons due to the small size and peripheral location of the emboli.

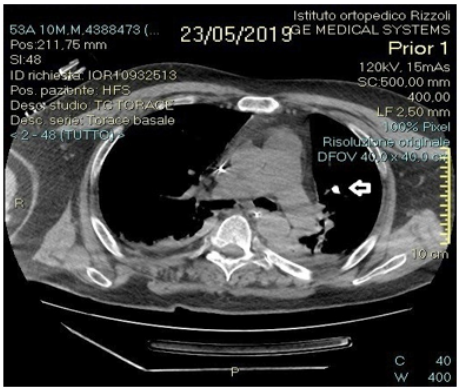


**Figure 2:** Pulmonary cement embolism detected on axial computed tomography (arrow).

The post-operative period was further complicated by the appearance of negative T wave and repolarization anomalies in the electrocardiogram. Therefore, an antiplatelet therapy was started. A new endoluminal defect, compatible with pulmonary embolism, was detected whit CT-scan in the vessels to the dorsal segment of the upper right lobe and posterior basal portion of the left lower lobe. Anticoagulant therapy with low molecular weight heparin was rapidly converted from a prophylactic to a therapeutic regimen. Despite complications, the patient was weaned from norepinephrine and from mechanical ventilation after ten days of hospitalization: the last echocardiography showed normal global kinesis of both ventricles. The patient was discharged from the ICU in a good neurological status with an anticoagulation therapy with coumadin prescribed for six months.

## Discussion

The vertebral body cement augmentation procedures, precisely PKP and PVP, are routine procedures in the management of painful vertebral fractures. They are considered safe but they are not risk free. In the last years many cases of major complications after PKP and PVP have been reported, with PCE being one of them, even though it is often well tolerated, and there are only few reported cases of death caused by it [4,5]. CAPSI of the thoracolumbar spine is indicated for the treatment of osteoporotic fractures or in cases of degenerative spine diseases associated with osteoporosis or osteopenia. From recent data, it seems that CAPSI has a considerable possibility to lead to symptomatic PCE: some cases of death due to fulminant PCE, cases of relevant intraoperative hemodynamic reactions due to anaphylactic shock and cases of intraoperative cardiopulmonary resuscitation have been described [3]. Only few studies report the incidence of PCE [3,6,7] varying according to the sensitivity of the imaging employed to diagnose the emboli: with post-operative chest X-Ray, the incidence varies from 1% to 6.8%; using chest CT, it arises to the range 2.1% to 26%.

However, it appears that the diagnosis of PCE is underestimated and nowadays there are no guidelines to help the diagnostic process or its management [8]. In literature, the technical means to decrease the incidence of cement leakage and thus PCE indicate the use of bone cement of viscous, toothpaste-like consistency, and the interruption of injection as soon as cement leakage is noticed or suspected in intraoperative fluoroscopy [3]. Furthermore, the anesthesiologist is forewarned to look out for any hemodynamic instability, that is a sudden drop of arterial blood pressure, oxygen saturation or end tidal carbon dioxide, as possible indicators for PCE. Risk factors for development of PCE have not been clearly defined yet. Some authors have found a correlation with the total number of vertebral levels treated [3,9]. Others affirm that malignant lesions (bone metastasis from carcinoma) and some types of fracture (the ones which lead to great cortical destruction) cause a major risk of cement leakage [3,10].

Despite the absence of guidelines regarding the diagnosis of PCE, some authors recommend a routine post-operative chest X-Ray within 24 hours from surgery, while obtaining a chest CT is advisable when there are some clinical evidences such as respiratory symptoms and unstable vital signs [2,11]. Moreover, the management of PCE has not been universally established yet and it is based on the patient's clinical conditions and on the embolism severity: asymptomatic patients with signs of peripheral PCE should not be treated, while patients with symptomatic peripheral or asymptomatic central PCE deserve anticoagulation therapy (situation comparable to thrombotic pulmonary embolism); finally, patients with symptomatic central embolism should be evaluated for surgical treatment with embolectomy [2]. There are however authors who employ anticoagulation therapy even in asymptomatic PCE [3]. According to other sufficient evidences, PMMA is not thrombogenic in vitro [12], and its thrombogenicity in vivo is still under discussion.

There is also a case report of a patient with a large cement embolus that was removed 4 years after kyphoplastic with no finding of thrombus formation on the removed foreign body [13]: these authors' opinion is that cement emboli might indirectly cause thrombi by the generation of a turbulent blood flow inside pulmonary vessels. They also suggest to start a coumadin therapeutic regimen only if the right atrium is involved. In our opinion, the pulmonary thromboembolism that has complicated the surveyed case is not a direct consequence of the PCE but rather caused by the patient's comorbidities like obesity and long bedrock, and by the pro-inflammatory state that the critical condition, deriving from the intervention, involved. In fact, the patient had suffered from deep vein thrombosis following the previous neurosurgical intervention. The important respiratory and circulatory complications that the patient suffered during surgery are probably due to an anaphylactoid reaction, which have to be considered because the patient had a relevant systemic reaction compared to the cement embolus dimensions.

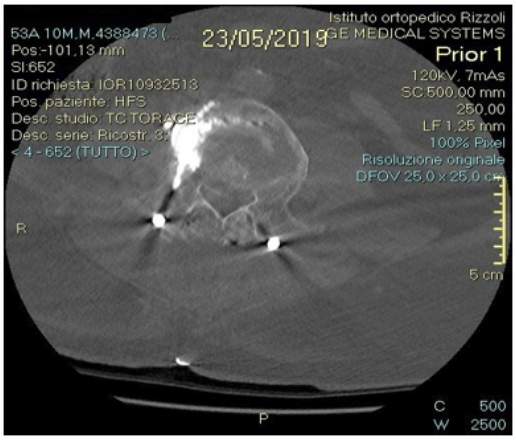


**Figure 3:** Sickle-shaped area of cement in the right paravertebral level of the lumbar vertebrae.

## Conclusion

An identification strategy for patients at risk of developing PCE has not been developed yet. From the recent literature emerges that patients who are subjected to CAPSI, PKP or PVP on multiple thoracolumbar vertebral levels have more chances to develop PCE, as well as patients suffering from spinal metastases. Patients with cardiopulmonary diseases seem to be at risk for symptomatic events, which may be due to the lack of compensatory mechanism. The need for a long-lasting anticoagulant therapy in patients with symptomatic PCE is still to be clarified. Even small cement emboli can carry extensive intraoperative systemic reactions that can be explained by the triggering of anaphylactoid reactions. The use of cement augmentation must be performed on a strict indication, especially in patients with risk factors. During surgery, raising awareness of the risks of cement augmentation and increase alertness of both surgeon and anesthesiologist might reduce the amount of cement extravasation and, most of all, attenuate the clinical consequences.

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