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**Intracardiac, pulmonary cement embolism in a 67-year-old female after cement-augmented pedicle screw instrumentation: A case report and review of literature**

Liang TZ *et al*. Intracardiac, pulmonary cement embolism after CAPSI

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**Abstract**

BACKGROUND

We report a case of pulmonary and intravenous cement embolism after cement-augmented pedicle screw instrumentation in treating spondylolisthesis underlying osteoporotic bone, which was successfully managed by conservative treatment. We describe the treatment and outcome of the patient, hoping to shed light on the management of bone cement embolism.

CASE SUMMARY

A 67-year-old female suffered from progressive low back pain and numbness in lower extremities for 30 years. She was diagnosed with L4 and L5 spondylolisthesis, spinal stenosis, and osteoporosis. The patient underwent spinal canal decompression, an interbody fusion of L4/5 and L5/S1, cement-augmented pedicle screw instrumentation in L4-L5 segments, and regular pedicle screw in S1 segments. Three days postoperatively, a sudden drop in oxygen saturation occurred. Computerized tomography scan confirmed pulmonary and intravenous embolism. The patient was treated conservatively by continuous low-flow oxygen inhalation, anti-coagulation, and antibiotic therapy for 1 mo and continued anticoagulation treatment for 6 mo. The patient showed no further symptoms in a 30-mo follow-up.

CONCLUSION

Intracardiac, pulmonary cement embolism after cement-augmented pedicle screw instrumentation is extremely rare. Careful clinical and radiographic evaluation is required in multiple sites of bone cement embolism. Conservative treatment may be a primary consideration in scattered emboli without life-threatening conditions, but a clinical decision should be made on an individualized basis.

**Key Words:** Osteoporosis; Polymethyl methacrylate cement; Pulmonary embolism; Cement-augmented pedicle screw instrumentation; Conservative treatment; Case report

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**Core Tip:** Intracardiac and pulmonary cement embolism event after cement-augmented pedicle screw instrumentation is an extremely rare complication in orthopedics surgery. In our case, the patient developed no symptoms except for rapid decrease in oxygen saturation to 70%. The case presents evidence for treating cement embolism cases without severe life-threatening complications, which include close monitoring, oxygen inhalation, anti-coagulation, and antibiotic therapy. This case improves our understanding of bone cement embolism and informs optimization of the duration and type of anticoagulant drugs to be used when the complication occurs.

**INTRODUCTION**

Currently, pedicle screw instrumentation is a common and necessary approach in spinal surgery. With a surge in the aging population, degenerative lumbar disease and osteoporosis have become increasingly common. When dealing with osteoporotic patients, the strength of internal fixation is reduced, thus leading to increased intraoperative bleeding and higher postoperative revision rate and failure rate. A study performed by Wang *et al*[1] showed that osteoporosis is a risk factor for longer instrumented posterior spinal fusion[1]. Longer instrumented extending to the adjacent segment may lead to several complications such as proximal junctional kyphosis that undermine the effectiveness of surgery[1,2]. To enhance the anchoring strength, several techniques have been used, including bicortical screws, tricortical screws, expandable screws, iliac screws, and iliosacral screws[3,4]. While it is not recommended to extend the extra fixation segment, the strength of bicortical or tricortical screws are also limited and may lead to internal fixation failure in an osteoporotic patients[5].

Cement-augmented pedicle screw instrumentation (CAPSI), first reported in the 1970s, has been widely applied in spinal surgeries for osteoporotic patients[6]. Generally, the indications for CAPSI include osteoporotic spine and revision surgeries[7,8]. Bone cement augmentation has complications of leakage into the intraspinal space and venous system and may lead to neurological deficits, pulmonary cement embolism (PCE), or intracardiac cement embolism (ICE)[9,10]. Compared to vertebroplasty, only a few studies focused on embolism events caused by CAPSI[11]. PCE and ICE caused by cement leakage into the venous system after CAPSI have been rarely reported. However, the incidence of cement embolism is increasing, and the treatment of severe multiple organ embolism remains controversial. It is estimated that the risk of all cement leakage was around 21.8%, while the incidence for symptomatic pulmonary embolism was between 0.2%-1.4%[12]. In this article, a case of conservative treatment for intracardiac and pulmonary cement embolism after CAPSI and a 30-mo uneventful outcome status is reported.

**CASE PRESENTATION**

***Chief complaints***

Low back pain for 30 years, worsening with numbness of both lower limbs for 3 mo.

***History of present illness***

A 67-year-old woman initially presented with the symptoms of chronic low back pain for 30 years. The patient received pain relief treatment, but she was not examined systematically because of poor compliance. The pain was then exacerbated and was accompanied by numbness in the lateral thigh of both sides for 3 mo. Therefore, the patient came to our hospital for a comprehensive examination.

***History of past illness***

The patient was diagnosed with hypertension for 10 years. The highest blood pressure level was around 150/90 mmHg. The patient did not have dizziness, vertigo, or blurred vision. She began to take metoprolol 47.5 mg once a day and yielded a good control of blood pressure level. The patient had no record of vascular diseases or embolism.

***Personal and family history***

Her family members had no records of thrombotic disease.

***Physical examination***

A physical examination revealed a step sign on the L5 and S1 spinous process with tenderness on the paraspinal muscles of L4-S1. The Laseque sign was negative for both limbs of the patient. Sciatica was not induced by bilateral femoral nerve stretching test of both lower limbs. No obvious scoliosis and kyphosis were observed in the patient.

***Laboratory examinations***

Prothrombin and partial thromboplastin times were normal, and D-dimers were slightly increased at 632 ng/mL. The blood analysis showed a hemoglobin level of 133 g/L, a platelet count of 247 ×109/L, and a white count of 5.77 × 109/L, which were all within the normal range. Three days after the surgery when the drop of oxygen saturation occurred, blood analysis revealed a leukocytosis 14.27 × 109/L, with predominant neutrophils (91.6%) with decreased hemoglobin at 96 g/L and normal platelet count. D-dimers were significantly increased at 7669 ng/mL (referenced range: 68-494 ng/mL). Creatine kinase was increased slightly at 219 U/L (referenced range < 190 U/L). Arterial blood gas revealed an elevated pH 7.479 and reduced oxygen partial pressure at 58.6 mmHg. The blood biochemistries, electrocardiogram, and urine analysis were normal.

***Imaging examinations***

The T-value measured by dual-energy X-ray absorptiometry was -3.21 SD, which indicated osteoporosis of the lumbar spine. The average Hounsfield unit value of the L4 vertebrae was 98.82, while the average Hounsfield unit value of the L5 vertebrae was 98.77 (Supplementary Figure 1). A preoperative radiograph and computed tomography (CT) scan showed L4 and L5 isthmic spondylolisthesis. The magnetic resonance imaging scan indicated anterior L4 spondylolisthesis complicated with spinal canal stenosis of the L4-S1 segment. Bilateral nerve root was compressed significantly. After surgery, chest CT was performed to evaluate the severity of embolism. Multiple columnar high-density shadows in pulmonary arteries of multiple segments of right pulmonary arteries, anteromedial basal segment, and basal lateral segment of left lower lobe and azygos vein were observed.

***Surgery procedure***

The patient was anesthetized and placed in a prone position. Under fluoroscopic guidance, 6.5 mm fenestrated pedicle screws were inserted in L4 and L5, and regular pedicle screws were inserted in S1. For cement augmentation, 2 mL of high-viscosity polymethyl methacrylate bone cement was injected into each pedicle screw gently 4 min after mixture without application of high-pressure injection system. An L4 and L5 Laminectomy and decompression were performed, and the nerve roots were released. A standard posterior lumbar interbody fusion was performed of the L4/5 and L5/S1 segments.

***Postoperative course and conservative treatment***

The patient’s condition was stable after surgery. The low back pain and numbness in the lower extremities were relieved immediately. Her oxygen saturation remained normal at 99%. However, on the third day after surgery, the patient presented with a sudden decrease in oxygen saturation to 70%. X-ray of lumbar vertebrae showed leakage of cement from the venous system (Figure 1). The patient did not have a cough, hemoptysis, or shortness of breath, and there were no signs of chest pain or palpitation. The oxygen saturation raised to 85% after low-flow oxygen inhalation. A blood gas analysis revealed a pH 7.479, partial pressure of carbon dioxide = 41.0 mmHg, and oxygen partial pressure = 58.6 mmHg. Blood D-dimer was raised from 631 ng/mL 1 d preoperative to 7669 ng/mL 3 d postoperative. Spirometry revealed small airway dysfunction without diffusion impairment. The pulmonary function test showed that the diffusion function was normal, but small airway dysfunction existed. Chest CT confirmed the presence of multiple columnar high-density shadows in the pulmonary arteries of multiple segments of right pulmonary arteries, anteromedial basal segment, and basal lateral segment of left lower lobe and azygos vein (Figure 2). The bone cement embolism was located within the right and left lower lobe of the lung. The CT scan also revealed exudation and inflammation of both lungs. The electrocardiograph was normal, and the molecular markers of myocardial infarction including creatine kinase (219 U/L), lactate dehydrogenase (293 U/L), and myoglobin (78.4 ug/L) were slightly elevated. The Pulmonary Embolism Severity Index and simple Pulmonary Embolism Severity Index score for the patient were 20 and 1, respectively. Because the patient exhibited no significant hemodynamic disorder or signs of cardiac failure, she was treated with continuous low-flow oxygen inhalation and 5000 IU/d low molecular weight heparin. Blood oxygen was restored in 2 h. The patient developed the symptoms of fever and her white blood cell count elevated to 12.4 × 109/L. Meanwhile, plain radiography indicated diffuse exudation in the lungs.

**FINAL DIAGNOSIS**

The final diagnosis of the presented case is intracardiac, pulmonary, and venous bone cement embolism after cement-augmented pedicle screw instrumentation.

**TREATMENT**

To treat plausible infection, 500 mg/d of levofloxacin was used. After the commencement of cement embolism, the patient was nursed sitting up regularly and was atomized to clear airway secretions twice daily. The patient developed no further embolism, and blood oxygen was maintained above 98%. Therefore, the patient was discharged 3 wk after surgery. The patient was treated with low molecular weight heparin 5000 IU/d for 1 mo and was switched to 2.5 mg/d of warfarin for 6 mo after discharge. The coagulation function was closely monitored by international normalized ratio during warfarin administration.

**OUTCOME AND FOLLOW-UP**

Regular tests and serial cardiac and pulmonary assessments were carried out and revealed no signs of an exacerbation. She did not report further discomfort in the 30-mo follow-up (Figure 3).

**DISCUSSION**

In the case we presented, the patient was diagnosed with L4-L5 isthmic spondylolisthesis, spinal canal stenosis, and osteoporosis. The patient developed symptoms such as decreased blood oxygen saturation after surgery, and imaging confirmed the occurrence of bone cement embolism in multiple organs. The patient has obtained good clinical outcome under conservative treatment. In a randomized, controlled trial, Ghogawala *et al*[13] suggested lumbar laminectomy plus fusion may yield better clinical improvement compared with laminectomy alone[13]. To improve the quality of life and prevent internal fixation failure, we decided to perform CAPSI in L4 and L5 segments. CAPSI was first described in 1975, and it has been widely used to increase screw holding power and resist pull-out force, especially in osteoporotic patients.

Bone cement leakage is a common complication of a bone cement-augmented pedicle screw. Two major complications underlying CAPSI are PCE and perivertebral cement leakage (PCL). It is estimated that the risk of PCE in an augmented pedicle screw insertion is approximately 1.5%-7.9%[14]. Janssen *et al*[11] reported the risk of cardiac cement embolism following percutaneous vertebroplasty was approximately 3.9%. Approximately 71% of patients with underlying CAPSI may develop cement leakage, but most of them were asymptomatic[11]. In a study conducted by Ulusoy *et al*[15], the distribution of intravenous leakage locations in PCL-positive cases was 10.8% in both the azygos vein and inferior vena cava. In PCE-positive patients, emboli in segmentary and subsegmental arteries were detected in 52% of patients, while 48% had emboli detected in the lobar and main pulmonary arteries[15]. In this case, the intracardiac, pulmonary, and vein cement embolism were all developed after CAPSI.

Very few studies have focused on the risk factors of an embolism following CAPSI. Known risk factors for PCE include thoracic spine instrumentation, spinal metastases, more instrumented levels, and more total cement volume[16]. Risk factors of symptomatic PCE include more than 10 instrumented levels, previous cardiopulmonary disease, and a total cement amount of more than 30 mL[15,17]. Besides, large-scale and multi-center retrospective studies should be conducted to determine risk factors of PCE and ICE after CAPSI. In the case we presented here, the patient did not present with the risk factors mentioned above. However, the patient was diagnosed with osteoporosis, which might also be considered as a risk factor for CAPSI.

Most cardiopulmonary cement embolisms are asymptomatic, but in some cases, cement embolisms can lead to severe clinical outcomes[18,19]. More devastating clinical outcomes may occur when PCL enters the lumbar venous plexus, azygos vein, inferior vena cava, the right heart, and eventually the pulmonary arteries[20-26]. Symptoms such as dyspnea, oxygen desaturation, tachypnea, or cardiopulmonary arrest seem to begin a few days or immediately after surgery (Figure 4 and Table 1). Life-threatening conditions including acute respiratory distress, pericardial effusion, and fatal cardiac perforation may occur[15,27,28]. Most PCE and ICE patients develop symptoms during hospitalization[29], but in some cases, it may take more than 5 years before the symptoms emerge[30].

Many studies have reported removing the central embolism caused by large emboli through surgery. Open surgery may be considered when the patient develops life threating conditions such as progressive dyspnea, chest pain, hemodynamic disruption, cardiac perforation, or tamponade[31]. After careful evaluation of the location and the shape of the cement emboli, percutaneous retrieval is an approach that is considered to extract the embolus. There is a viable algorithm and indication of reverting to surgery in treating symptomatic central bone cement embolization. After careful evaluation of clinical presentation, cement size and location, and vital signs, the decision of interventional radiology or either open/minimal invasive cardiac surgery can be made[9]. Sometimes, the embolus may be difficult to extract, and forced removal may cause extra damage. In this case, the symptomatic cardiopulmonary embolism was treated using conservative treatments under close monitoring. It is not practical to remove all the sparse and fragile emboli through catheter-based retrieval. Therefore, in the case of mild clinical manifestation with stable hemodynamic status, conservative treatment including anticoagulation, continuous low-flow oxygen inhalation, and antibiotics can be an option. Some preventive measures have been used to avoid embolic complications of CAPSI (Table 2).

Baroud *et al*[32] suggested the use of high-viscosity bone cement and longer mixing time[32], while lower injection pressure and intermittent injections have also been recommended[33]. Close monitoring under consecutive fluoroscopy can detect early leakages.

**CONCLUSION**

Spine surgeons should pay more attention to the occurrence of cement embolism when treating an osteoporotic or metastatic spine tumor through CAPSI. In this case, the patient developed intracardiac, pulmonary, and intravenous embolism without significant clinical symptoms. The leakage may be caused by the injection of bone cement into paravertebral veins. The patient was treated with continuous low-flow oxygen inhalation, anticoagulation, and antibiotics, and the prognosis was acceptable in a 30-mo follow-up. When making clinical decisions, conservative treatment may be considered in patients without significant symptoms and when the embolus is small. Despite the rare complications, CAPSI is generally a reliable and safe surgical procedure. Nevertheless, more studies are needed to establish a decision-making algorithm regarding CAPSI related embolism events.

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**Footnotes**

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**Figure Legends**



**Figure 1 Internal fixation of a 67-year-old patient with osteoporotic bone.** A: Preoperative (March 15, 2017) lumbar spine plain radiograph lateral view; B: Anteroposterior view; C: Postoperative lumbar spine plain radiograph lateral view at 3 d (March 25, 2017); D: Anteroposterior view showing the leakage of bone cement through the venous system (high-density line indicated by the red arrow) and the extent of cement augmentation of the lumbar spine.



**Figure 2 Bone cement entering the vascular system.** A-C: Postoperative computerized tomography scan showing the bone cement emboli in the pulmonary, cardiac, and venous system at 6 d (March 30, 2017). Cement is indicated by the red arrow; and D: Three-dimensional reconstruction of the thoracic showing the distribution of the emboli.



**Figure 3 Radiography examination after indicated time of embolization.** A: Preoperative chest radiography at 2 d; B: Postoperative chest radiography at 12 mo (March, 2018); C: Postoperative chest radiography at 24 mo (March, 2019); D: Postoperative computerized tomography scan at 30 mo (September, 2019).



**Figure 4 Timeline flow diagram of the case.** CT: Computerized tomography; LMWH: Low molecular weight heparin.

**Table 1 A timeline of the patient**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Date** | **Oxygen saturation** | **Platelets, 109/L** | **WBC, 109/L** | **Hemoglobin, g/L** | **D-dimer, ng/mL** | **Clinical intervention** |
| March 13, 2017 | 99% | 172 | 6.75 | 121 | 631 | Admission |
| March 23, 2017 | 99% | - | - | - | - | Lumbar surgery |
| March 24, 2017 | 99% | 158 | 14.27 | 96 | 7698.90 | Vital signs monitering |
| March 25, 2017 | 70% | 113 | 12.4 | 84 |  | Pulmonary expert consultation |
| March 26, 2017 | 85% | 121 | 8.90 | 85 |  5667 | Levofloxacin 500 mg qd+ LMWH 5000 IU qd |
| April 1, 2017 | 98% | - | - | - | - | Levofloxacin 500 mg qd+ LMWH 5000 IU qd |
| April 14, 2017 | 98% | - | - | - | 4638 | Discharged |

WBC: White blood cell; LMWH: Low molecular weight heparin.

**Table 2 Summary of reported cardiopulmonary embolism caused by cement-augmented pedicle screw instrumentation**

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Ref.** | **Sex** | **Age** | **Vertebrae augmented** | **Time of embolism detected Post-op** | **Clinical presentation** | **Treatment** | **Outcome** |
| Rasch *et al*[20], 2010 |  | 55 | L3/4 | 2 d | Tachycardia, dyspnea and oxygen desaturation | Pulmonary arteriotomy and cement removement | Discharged without severe complication |
| Tang *et al*[21], 2020  | F | 73 | L3-L5 | 6 d | Dyspnea, blood oxygen desaturation | Oxygen inhalation and anticoagulation treatment | Uneventful recovery in 2 yr follow-up |
| Akinola *et al*[22], 2010  | M | 76 | L3-L5 | Immediate | No symptoms | Anticoagulation treatment for 6 mo | Uneventful recovery in 6 mo follow-up |
| Zheng *et al*[23], 2013  | F | 47 | T1-T4, T9 | 1 h | Dyspnea, low blood pressure, unconsciousness  | NA | Death |
| Rahimizadeh *et al*[24], 2020 | F | N.A. (middle aged) | L3 | 1 d | Cardiopulmonary arrest | Resuscitation, anticoagulation treatment  | Uneventful recovery in 12 mo follow-up |
| Röllinghoff *et al*[25],2010 | F | 69 | T8-L1 | NA | No symptoms | NA | Uneventful recovery in 18 mo follow-up |
| Tonolini *et al*[26], 2012  | F | 75 | L1/L3 | Immediate | Dyspnea with tachypnea and bilateral hypoventilation | Anticoagulation treatment | Discharged without severe complication |

NA: Not applicable.