SCIENTIFIC ARTICLE

Pulmonary Cement Embolism Associated with Percutaneous Vertebroplasty or Kyphoplasty: A Systematic Review

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Therapeutic vertebral cement augmentation for the treatment of painful skeletal diseases, although widely applied for more than several decades, still has not thoroughly resolve the problem of cement extravasation. Based on a review of literature published, the present study was to provide a systematic review of the current understanding of pulmonary cement embolism (PCE) associated with percutaneous vertebroplasty (PVP) or percutaneous kyphoplasty (PKP), and to summarize the incidence, clinical features, prophylaxis and therapeutic management of PCE after vertebral cement reinforcement. The reported incidence of PCE ranges widely, from 2.1% to 26%. Asymptomatic PCE is a common condition without permanent clinical sequelae. Nevertheless, it is emergent once a symptomatic PCE is presented. Close attention and effective pre-measures should be taken to avoid this catastrophic complication.

Key words: Complication; Inferior vena cava; Percutaneous kyphoplasty; Percutaneous vertebroplasty; Pulmonary embolism

Introduction

Percutaneous vertebroplasty (PVP) and percutaneous kyphoplasty (PKP) are both radiological, percutaneous puncture procedures, entailing bone cement injection into a destroyed vertebral body with the aim of pain relief and bone reinforcement of the vertebrae. Due to its minimal invasion and immediate pain relief, PVP/PKP is steadily gaining popularity in the treatment for painful tumor infiltration diseases such as aggressive haemangioma, metastatic carcinoma and multiple myeloma[1–5], and for patients who have intractable pain due to osteoporotic thoracolumbar fractures[6–11]. Despite the expanding utilities of these procedures, a growing number of reports about dreadful complications are documented in literature. Of particular concern is the bone cement leakage[8,12,13]. Cement escaping posteriorly into the spinal canal can cause spinal canal stenosis or cord compromise[14–19]; and cement escaping into the intervertebral foramina can lead to nerve root compression[20,21]. Additionally, escaping cement in the perivertebral venous system and inferior vena cava (IVC)[22–26] can drift down toward the right cardiac chambers or the pulmonary circulation with catastrophic results, such as cardiopulmonary failures[23,27,28], impaired renal function[23,29], paradoxical cerebral embolism[30], and even death[13,27,31–33]. In this paper, a comprehensive review to assess the rate, clinical features, pathophysiology, prophylaxis and management of cardiopulmonary cement efforts associated with PVP or PKP was performed.

Methods

Search Strategy

An experienced librarian performed a comprehensive literature search. Studies were searched through the electronic bibliographic databases of Springer-Link, Ovid-Medline and PubMed from January 1999 to December 2010. The following search terms were used: percutaneous vertebroplasty,

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percutaneous kyphoplasty, complications and pulmonary embolism.

Study Selection

The search was limited to identifying studies published in English. Considering only a limited number or no randomized controlled trials were expected, also case reports or non-randomized controlled and observational studies were included. In addition, the reference sections of all included full text studies were inspected to identify supplementary relevant studies. Two review authors independently scrutinized all titles and abstracts yielded for eligibility according to the following inclusion criteria: (i) Inclusion being made of case reports, experimental or observational designs; (ii) Studies had to pertain to the surgical intervention of PVP or PKP for the treatment of osteoporotic and/or tumorous origin; (iii) Studies were required to include quantitative information relating to at least one of the following primary interest variables: complications, cement leakage, pulmonary embolism, pathophysiology, manifestation and management; and (iv) Studies involving pulmonary emboli produced in hip arthroplasty or fat emboli associated with long bone fracture were excluded.

Data Extraction

In order to assess the rate, clinical features, pathophysiology, management and prophylaxis of pulmonary cement efforts associated with PVP or PKP, the results of all relevant statement were extracted from the original studies.

Results

Search and Selection

Of 721 relevant articles being screened in detail, 116 full text articles were retrieved. Reviewing the reference sections of all included full text studies resulted in seven additional references. After scrutinizing all 123 articles, a total of five observational studies[34–38], three non-randomized controlled experimental animal trials[39–41] and 32 case reports[22–33,42–60] were indentified. No randomized controlled trial was identified. As a few of the papers were observational studies, methodological quality and meta-analysis of the included studies was not assessed.

Outcome

Five observational studies consist of three retrospective studies[34–36] and two prospective studies[37,38]. Fifty-one cases in all with cement pulmonary embolism were noted in the observational studies. Among these 51 cases, 50 cases were secondary to PVP and one case was following PKP. In the 32 case reports[22–33,42–60], 35 patients (34 following PVP and 1 following PKP) were diagnosed with pulmonary cement embolism (PCE), 30 were symptomatic[22–33,42–50,52–58,60] and five were asymptomatic[22,42,48,51,59]. The entire development of cement pulmonary embolism was presented, including the clinical manifestation, diagnostic methods, emergent management and outcome. Table 1 summarizes a description of the case reports of PCE. To date, five lethal cases of pulmonary embolism after percutaneous vertebroplasty have been reported[13,27,31–33] (Table 2).

Rate of Pulmonary Cement Embolism Following PVP or PKP

The rate at which cement embolizes into the cardiopulmonary circulation during vertebral reinforcement is uncertain, as patients are not routinely screened with chest imaging before and after the procedure. This is especially true when the PCE is asymptomatic. In the five observational studies, rate of PCE following PVP/PKP was discussed (Table 3). As shown in the table, the observed incidence in prospective studies with stand postprocedual chest CT scan is much higher than that in retrospective studies. That is to say that the rate of cement pulmonary embolism would commonly be underestimated.

Clinical Features

Concerning the cases of symptomatic cardiopulmonary efforts cited above, common clinical features range from precordial chest pain and tightness[24,26,44–46,49], mild to severe dyspnea[25,43,45,46,48], cyanosis, palpitation[44], and acute respiratory distress syndrome (ARDS)[23,27]. Physical examination is remarkable for tachypnea, hypotension[33,46,49], irregular cardiac rhythm[32,49] or cardiac arrest[32]. Laboratory value may show an increased plasma D-dimer level[25,42], oxygen desaturation (low SpO_2)[32,33,49], hypocapnia[33] and unstable pulmonary arterial pressure[14,32]. Pulmonary ventilation perfusion scans show ventilation perfusion mismatch with reduced blood flow to the embolized lobe[25,43]. Commonly, the vascular cement extravasation is not noticed during or immediately after the PVP/PKP procedure, resulting in a delay for the diagnosis and relevant management of pulmonary embolism[24]. Therefore, the role of chest radiography and other imaging methods in the diagnosis of polymethylmethacrylate (PMMA) pulmonary embolism has yet to be decided. (i) Chest radiography: The cement used is of such high density compared with the lung tissue, that the visualization of cement emboli on conventional chest radiograph is quite striking[51]. Some researchers conclude that the risk of pulmonary embolism of PMMA might be underestimated. Thus, a routine chest radiography was proposed following every vertebroplasty, in order to detect pulmonary PMMA embolism earlier and thereby prevent serious delayed cardiopulmonary failures[27,42,48,50]. (ii) Echocardiography: Transesophageal echocardiography is a safe and non-invasive modality to evaluate hemodynamic instability and to reveal echogenic linear material in the cardiac chambers[52,61]. (iii) CT: Computerized tomographic angiography enables the locations, the lengths, and the number of pulmonary cement pieces to be clearly visualized[45]. Multidetector CT (MDCT) accurately shows the cement material in the cardiac chambers, which is not detected on echocardiograph[24,46,57]. (iv) Pulmonary angiography: Pulmonary angiography is a characteristic test for pulmonary embolism, which facilitates earlier diagnosis. Nevertheless, there is no report about pulmonary angiography

TABLE 1 Case reports of pulmonary cement embolism (PCE) after vertebral augmentation in retrieved literature

Author/Publication date	Operation level and operation indication	Clinical manifestation	When manifestation presented/when PE was detected	Cement migration	Management	Outcome
Tozzi *et al.*[23] (2002)	T11 fracture (PVP)	Developed respiratory distress, renal failure, a mean pulmonary artery pressure of 48 mm Hg, right cardiac failure	At the end of the PVP procedure	Perivertebral venous system, IVC, both Pas	Noninvasive ventilatory support, inotropic agents, heparin, pulmonary embolectomy	Recovered from respiratory and cardiac failure
Jang *et al.*[48] (2002)	T6, T9, T10, T11, L1, L2 CF (PVP)	Chemical odor of solvent in the patient's mouth and nausea, mild dyspnea, chest discomfort	During cement injection	PA	Delayed injection, oxygen inhalation and anticoagulants	Dyspnea regressed
Katrien *et al.*[43] (2003)	T11 CF (PVP)	Mild dyspnea, reduced blood flow to the right lower lobe	2 days after the procedure	Right PA	Intravenous heparin, catheter procedure and open-heart operation, oral anticoagulant therapy	Uneventful recovery
Bernhard *et al.*[51] (2003)	T10, 11, L1 CFs (PVP)	Denied any respiratory symptoms	6 months later	Both lungs	—	Discharged
Kim *et al.*[24] (2005)	T8 fracture (PVP)	Severe chest pain, hemopericardium, cardiac perforation	7 days after the PVP	Azygous vein, RA and RV, PA	Open heart surgery for hemopericardium and cement removal	Discharged
Seo *et al.*[57] (2005)	L1, 2 CFs (PVP)	Palpable mass on the subareolar of the left chest wall	2 years after the PVP	IVC, RA, right PA	Right atriotomy and inferior vena cavotomy	—
Chung *et al.*[29] (2006)	L1 CF (PVP)	Impaired renal function	During operation	Both renal vessels	Treated conservatively	Discharged with BUN to Cr returned to normal
Freitag *et al.*[49] (2006)	L1-L5 OVCFs (PVP)	A sudden onset of arrhythmia, hypotension (BP, 65/30 mm Hg), oxygen desaturation (SpO2, 91%)	During cement injection	Left inferior lobe PA	Stopped injection, intravenous unfractionated high-dose heparin, further observation in ICU	Discharged on oral anticoagulant
Abdul *et al.*[42] (2007)	T11, 12 OVCFs (PVP)	Dyspnea, chest pain, increased D-dimers level, ventilation-perfusion mismatch	3 days after discharge	Paravertebral venous system, both lungs	Low-molecular-weight heparin, Enoxaparin and Anti-Xa	Rapid resolution of dyspnea and chest pain
Lim *et al.*[25] (2007)	L2, 5 CF (PVP)	Mild dyspnea and edema lasting 4 weeks, ventilation-perfusion mismatch	5 years after the procedure	IVC, right hepatic vein, RA, left PA	Anticoagulation, open-heart surgery for atrial thrombectomy	An uneventful recovery
Lim *et al.*[45] (2008)	T12, L1 CF (PVP)	Chest pain, dyspnea, cardiac perforation, pericardial effusion	2 months after T12 PVP and 9 days after L1 PVP	Right upper lung and RV	Pericardial collection aspiration, cement removal, repair of right ventricular wall	Experienced no sequelae
Son *et al.*[26] (2008)	L1, 2, 4 CF (PVP)	Chest pain and tightness, hemopericardium, severe tricuspid regurgitation, cardiac tamponade	5 days after the procedure	ICV, RV	Emergent operation for cement removal and suture of perforated right ventricle, tricuspid annuloplasty	Recovery without any complication and discharged
Cadeddu *et al.*[52] (2009)	T12, L2 OVCFs (PVP)	Asymptomatic	2 years after the procedure	RA and RV, left PA	Right cardiac catheterism	Failed removal of cement
Braiteh *et al.*[44] (2009)	L3 CF (PVP)	Intermittent precordial chest pain and palpitation	5 months after the operation	RA and RV	Endovascular procedure for foreign body retrieve	Symptoms relieved
Caynak *et al.*[46] (2009)	T4, 9 CF (PVP)	Progressive dyspnea, chest discomfort, hemodynamic unstability, cardiac tamponade by pericardial collection	2 months after operation	Azygous veins, both PAs, RA and RV	Anticoagulation, pulmonary physiotherapy, emergent operation for hemorrhagic fluid drain and cement particles removal	Favorable outcome
Radcliff *et al.*[60] (2010)	L2 CF (PKP)	Shortness of breath symptoms with productive cough	28 days after PKP	Right PA	Conservative management	Discharged with no further exacerbation

BUN, blood urea nitrogen; CF, compression fracture; IVC, inferior vena cava; OVCF, osteoporotic vertebral compression fracture; PA, pulmonary artery; PVP, percutaneous vertebroplasty; RA, right atrium; RV, right ventricle.

TABLE 2 Five lethal cases of pulmonary cement embolism (PCE) after vertebral augmentation (VA) with polymethylmethacrylate (PMMA)

Author/Cement type	Operation level and operation indication	Clinical manifestation	When manifestation presented/when PE was detected	Cement migration	Management	Outcome
Chen *et al*.[21]/ PMMA with tungsten	L2, L4 OVCFs	A sudden onset of bradycardia, hypotension (BP, 64/30 mm Hg), oxygen desaturation (SpO2, 70%) and hypocapnia, right heart outlet obstruction and failure	At the time of skin closure	RA, RV	Resuscitation for one hour (cardiac massage and intravenous injection of 10 mg epinephrine)	Died from bone cement implantation
Yoo *et al*.[27]/ PMMA with barium	L5 OVCF	Arthralgia, myalgia, fever, ARDS	3 days after operation	Right interlobar PA	Supplemental oxygen via face mask, tracheal intubation and mechanical ventilation, intravenous infusion of heparin, pulmonary embolectomy	Died 20 days after PVP
Monticelli *et al*.[32]/–	T12, L1, 2 fractures	Shock, low blood oxygenation and low pulmonary arterial pressure, cardiac arrest	Approximately 15 min after procedure	Paravertebral venous plexus, PAs	Extended cardiopulmonary resuscitation but failed, forensic autopsy	Died
Stricker *et al*.[31]/PMMA	L1–4 OVCFs	Severe chest pain, Restless, tachypnea, tachycardia, hypertension, oxygen desaturation, loss of consciousness, pulseless electrical activity	During the last injection of PMMA	IVC, Right PA	Mask ventilation, positive pressure ventilation, repeated intravenous boluses of noradrenaline and adrenaline	Died
Barragan *et al*.[13]/PMMA with tungsten	–Spinal metastases	Ventilatory and hemodynamic symptoms	—	—	Oral anticoagulants	Died 8 days after PVP

ARDS, acute respiratory distress syndrome; CF, compression fracture; IVC, inferior vena cava; OVCF, osteoporotic vertebral compression fracture; PA, pulmonary artery; RA, right atrium; RV, right ventricle.

being used for the diagnosis of pulmonary embolism, as it is costly, invasive and complicated.

Discussion

Following encouraging clinical outcome, particularly with respect to pain relief, indications for vertebral cement reinforcement were extended and comprise at present the palliative treatment of spinal tumor infiltration such as aggressive haemangioma, myeloma, metastatic osteolytic diseases, and more recently, osteoporotic vertebral compression fractures as well. However, the result of our review shows that PVP/PKP is a high-risk technique.

During the PVP procedure, bone cement (PMMA) leakage into the paravertebral or extradural venous plexus is a well-described local complication when cement is injected into the vertebral body under high pressure via a small needle (11–13 gauge). Moreover, a needle inadvertently placed in the basivertebral vein or an overfilling of cement in the vertebral body can facilitate cement migration into the perivertebral venous plexus[23], then through the hemiazygous vein, the azygous vein, and then the IVC. It is in these locations where polymerization of the cement embolus occur[22–24].

If it is fluid or injected too quickly, bone cement may enter into the right atrium (RA) and right ventricle (RV) via IVC, and firmly stick to the RA/RV free wall, due to the material's long and stiff nature[24,26,43–46,61]. A right atrial thrombus occasionally obstructs blood flow through the tricuspid

TABLE 3 Rate of pulmonary cement embolism (PCE) in five observed studies

Authors	Study	Number of cases	Number of sessions	Number of PCE	Rate of PCE (%)
Choe *et al*.[34]	Retrospective	62	65	3	4.6
Duran *et al*.[35]	Retrospective	73	128	5	6.8
Venmans *et al*.[36]	Retrospective	299	532	11	2.1
Kim *et al*.[37]	Prospective	75	78	18	23.0
Venmans *et al*.[38]	Prospective	54	60	14	26.0

valve with the result of hemopericardium and severe tricuspid regurgitation[24,26,44], and leading to cardiac tamponade or cardiac perforation[26,45,46].

Infrequently, cement drifts down the blood stream toward the pulmonary arterial circulation and obstructs the orifice of the local lobar branch[23,27,28,34,35,37,47–49]. In an animal model trial[41], calcium phosphate cement (CPC) was found to result in a more severe increase in pulmonary arterial pressure compared to PMMA, due to its disintegration property. The formation of anaphylactic toxins of PMMA may cause direct cellular injury by increasing cell permeability through releasing histamine and platelet-activating factors, and by stimulating neutrophil adherence and superoxide production. Pulmonary cement emboli also lead to an increase of capillary permeability, ultimately predisposes dyspnea and even acute respiratory distress syndrome, causing similar pathophysiological consequences to other pulmonary embolisms[27]. On the other hand, bone is replaced by fat tissue in osteoporotic vertebral bodies, the percutaneous injection of acrylic bone cement may increase intramedullary pressure and thereby, contents of fat or bone marrow shift into the paravertebral venous circulation with a potential to contribute to the clinical presentation of pulmonary embolism[33,42]. Based on the result of the experimental study, Krebs *et al.*[39,40] contended that cement embolus is not the sole factor in triggering the formation of pulmonary vessels occlusion.

Considering PCE predispose a great contribution of cardiopulmonary dysfunction, it is crucial to pay close attention and take effective premeasures to avoid this mortal complication.

Monitoring Equipment

Meticulous monitoring of the cement flow during the PVP or PKP procedure is critical. Uniplanar fluoroscopy is used commonly, which does help to detect the posterior vertebral leakage, but real-time detection of cement from lateral vertebral leakage remains difficult owing to the overlap of the intravertebral cement. Biplanar fluoroscopy allows a simultaneous detection of the vertebrae in two projections and cement extravasation into the perivertebral veins or the spinal canal[42]. Braak *et al.*[62] recently attempted to use real-time 3-dimensional (3D) fluoroscopy guidance in needle interventions. They testified that it is a new, promising, and feasible technique providing high accuracy. Nevertheless, fluoroscopic visualization is usually obscured by shoulders in the lateral projection in the cervical and high thoracic areas. Bayley *et al.*[63] recently shared a modified but simple position that significantly improves the fluoroscopic lateral imaging of the upper thoracic spine. They had accessed vertebrae between T1 and T5 successfully with this technique, and confirmed that it is a safe practice of kyphoplasty, vertebroplasty and biopsy throughout the upper thoracic spine[23]. Computed tomography becomes very effective by ensuring an accurate puncture and by allowing easy real-time monitoring of the cement injection, even in the high spinal level[64,65]. The disadvantage is plane-limited registration, which may lead to cement leakage being undetected between slices[42].

Opinions differ regarding the utility of venography in decreasing cement extrusion during PVP. Some authors[45,48,66] recommended an antecedent vertebral venography to identify a direct shunt from the needle tip to the venous plexus. The opposite viewpoint contends that venography does not exactly predict venous leakage, due to the differences in the viscosity and flow characteristics of the contrast material and cement[67–69]. Additionally, the opacification of the contrast agent could hinder visualization for cement injection. This obstacle is exemplified during an injection into necrotic cavities in cases of vertebral osteonecrosis or Kümmell's disease, or during an injection through the endplates to the intervertebral discs[70]. Do[71] pointed out that venography help novice or inexperienced operators to perform vertebroplasty in a safer manner, but for those who are adept at the performance of vertebroplasty, venography may represent a superfluous step.

Bone Cement Substitutes

Polymethylmethacrylate currently represents the standard in augmentation materials[12]. The mixing of PMMA with barium or tungsten quantity to 30% by weight for opacification is essential for good visualization of extraosseous cement leakage and for timely discontinuation of the cement injection[72]. Owing to its nondegradable and toxic property, however, attempts have been increasingly made in recent years to explore the alternative biomaterials that are more suited for PVP and PKP[73–75]. Calcium phosphate cement (CPC) and polypropylene fumarate (PPF) are now being investigated as prospective alternatives for vertebral augmentation due to the cytocompatibility and osteoconductive behavior[74,75], but it seems that the no alternative bone cements could deny any extrusion for their native injectable aspect. Preclinical studies demonstrated that high-viscosity cements significantly decrease the incidence of leakage compared with low-viscosity cements. In Georgy's study[76], he introduced new high-viscosity cement (Confidence spinal cement system) used for vertebroplasty. The result confirmed prior observations that the high-viscosity confidence cement results in a leakage rate comparable with that of kyphoplasty.

Cement Injection Techniques and Cement Delivery System

As a new technique, percutaneous balloon kyphoplasty is taking over the role of PVP[8]. Multiple literature sources agree that the risk of cement extravasation in PKP is reduced because the inflated balloon creates a void within the vertebral body, into which cement is injected under low pressure[16,50,76–78]. Vertebral body with cavity facilitates injection under low pressure because cement usually seeks the potential space preferentially[63]. As shown in previous studies[34,37,79], a higher frequency of vascular cement seepage was noted in cases of vertebral body without cavity. On the other hand, injecting the cement in stages may be a good strategy to avoid a relevant extravasation[42,80]. Try to inject a small volume of PMMA, and then stop the injection, even if leakage is not detected, and proceed with the augmentation about 20 to 30 s later when the endangered

veins are occluded. Any injection should be delayed immediately as long as venous leakage is detected under monitoring images[42].

Vesselplasty is another advanced device for cement injection[81]. Instead of using a balloon to create a cavity, this procedure uses a polyethylene terephthalate (PTE) artificial container (Vessel-X, A-Spine Holding Group Corporation) for restoring the height of the vertebral body and for containing the bone void material. Container is introduced into the vertebra in its reduced configuration and, once positioned within the vertebra, is expanded simultaneously by the injection of PMMA. Then, owing to the porous structure comprising the fibers of the PET vessel, a small amount of bone cement permeates through its wall and interdigitates within the vertebral body to increase its stability. At the end of the procedure, vessel and the bone void filler material can be left behind at the predetermined injection area. Theoretically, this new technology may effectively avoid the problem of cement leaking into the cardiopulmonary circulatory system. Catheter fabric kyphoplasty (CFK)[82] is the latest breakthrough cement delivery system in which an expander made up of fabric was left behind in the vertebra until the cement solidified. It serves as an external coat that would thoroughly prevent cement migrating into the adjacent tissues. Unlike the Vessel-X procedure, fabric expander can be removed after cement consolidated. The results of the preliminary trials prove that Vesselplasty and CFK offer statistically significant benefits in the improvement of pain, mobility, and cement seepage[81,82].

Therapeutic Management

Therapeutic management of pulmonary cement embolism depends on the clinical presentation of the patient[42]. Choes *et al*.[34] contended that the incidental finding of cement emboli in an asymptomatic patient should not alter medical treatment. In Venmans' study[36], all 11 patients with venous PMMA migration toward the lungs remained asymptomatic during 1-year follow-up. Repeat CT scanning after 1 year demonstrated unchanged pulmonary PMMA deposits without late reactive changes. This finding coincided with the notion that plugging a small percentage of arterial pulmonary vessels does not result in any respiratory symptoms[51]. Yoo *et al*.[27] proposed that a conservative treatment rather than a surgical removal may be recommended except when the obstruction is extensive enough to cause immediate cardiopulmonary changes. There are cases of severe pulmonary cement embolism following PVP presented in the English literature. In 1999, Padovin *et al*.[47] first described a case of symptomatic pulmonary embolism in a 41-year-old patient with Langerhans' cell histiocytosis (LCH). Symptoms of right chest pain, tachypnea, tachycardia, and hemoptysis arose immediately after PVP, and supplemental oxygen, anticoagulation and anticonvulsion produced a good result. Zaccheo *et al*.[28] reported a 77-year-old woman developed acute respiratory failure after multilevel percutaneous vertebroplasty. Mechanical ventilatory support and anticoagulation with low-molecular-weight heparin and warfarin enabled a recovery from pulmonary embolism. Concerning the management of semiotic PCE in case reports, we generalized an emergency treatment principle that if the patient presents with sudden dyspnea, and multiparameter monitor demonstrates hypotension, arrhythmia, and oxygen desaturation, any cement injection should be terminated and the patient should be immediately returned to a supine position[33,49]. Supplemental oxygen[23,27,48] can be administered via facemask to maintain Pa O_2 >60 mm Hg and Sa O_2 >90%, a tracheal intubation and mechanical ventilation is to be performed if there is no improvement[27]. Since the presence of intravascular acrylic material leads to the activation of the coagulation system, therapy with anticoagulation such as low-molecular-weight heparin should be administered immediately to reduce the danger of pulmonary infarction progression in the absence of contraindications[23,42,48,49]. In cases with saddle cement emboli at the cardiac chambers causing multiple cardiac perforations[26,45] or right heart failure[23,26,46], or at the pulmonary artery levels causing immediate respiratory distress[27], conservative treatments may fail to yield a good result. Emergent surgical intervention such as catheterism via femoral vein approach, open embolectomy from cardiopulmonary circulation or a hybrid technique combining an interventional catheter procedure with an open heart operation can be scheduled to remove the foreign acrylic cement[22–26,43,45,52].

Given that all the related studies have suggested that intracardiac thrombus and pulmonary thromboembolism can occur as a late complication due to bone cement seepage, interest in discovering new cement alternatives and advanced injection device, seems urgent.

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