CT features and risk factors of pulmonary cement embolism after vertebroplasty or kyphoplasty in patients with vertebral compression fracture: a retrospective cohort study

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Background: Pulmonary cement embolism (PCE) caused by cement leakage is one of the complications of percutaneous vertebroplasty (PVP) or percutaneous kyphoplasty (PKP). The aim of our study was to explore the imaging features on computed tomography (CT) and analyze the risk factors of PCE in patients with a vertebral compression fracture to compare the incidences of PCE caused by PVP and PKP.

Methods: In this single-center, retrospective study, 373 patients (96 males and 277 females; mean age 76.2±9.4 years) from January 2017 to December 2020 who underwent PVP or PKP for treatment of vertebral compression fracture in the China-Japan Friendship Hospital were retrospectively included. Their clinical data were recorded, and their postprocedural chest CT scans were reviewed and evaluated for PCE.

Results: Of the 373 patients, 258 patients underwent PVP while the other 115 underwent PKP. PCE was found on the postprocedural chest CT scans in 64 patients (17.2%), including 47 patients with PVP and 17 patients with PKP. The incidence of PCE of PVP and PKP was similar (χ^2=0.660; P=0.460). The typical CT findings of PCE were multiple linear or branching radiopaque densities in pulmonary arteries. The upper lobes of bilateral lungs were the most frequently involved. In addition, postprocedural chest CT demonstrated that 103 cases had cement emboli in the azygos vein, and 8 cases had cement emboli in the inferior vena cava. Binary logistic regression analysis demonstrated that PVP or PKP in the T9 vertebra [odds ratio (OR) =4.222; 95% CI: 1.490–11.966] and cement emboli in the azygos vein (OR =7.647; 95% CI: 3.937–14.856) or the inferior vena cava (OR =42.701; 95% CI: 7.525–242.302) were the risk factors of PCE.

Conclusions: The incidence of PCE during PVP or PKP was 17.2%. Postprocedural chest CT clearly showed PCE as branching hyperdense or radiopaque lesions confined within the pulmonary artery courses.

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PVP or PKP in the T9 vertebra and cement emboli in the azygos vein or the inferior vena cava were risk factors for PCE.

Introduction

For patients with vertebral compression fractures, percutaneous vertebroplasty (PVP) or percutaneous kyphoplasty (PKP) are currently considered minimally invasive treatment options because they can significantly alleviate pain symptoms and prolong the survival time of patients as compared with nonsurgical treatment (1-6). In an attempt to correct vertebral deformity and control cement distribution, PKP uses orthopedic inflatable bone tamps before bone cement injection, while PVP is similar, using needles to deliver bone cement without orthopedic balloons. However, the incidence of complications after PVP has been reported to be between 1% and 10% (7-9). In a large retrospective study, 69.1% of patients had postoperative cement leakage, and 0.72% had clinical complications (10). Pulmonary cement embolism (PCE) caused by cement leakage is one of these complications. Wang *et al.* (11) reported the incidence of PCE during PVP to be 1.9% to 26%. Krueger *et al.* (12) reported the incidence of PCE after PVP and PKP ranging from 3.5% to 23% for osteoporotic fractures. Most existing studies on this subject consist of case reports using conventional chest radiograph. However, there are few reports on the computed tomography (CT)-derived imaging characteristics of PCE, the relative incidences of PVP- *vs.* PKP-induced PCE, and the specific risk factors involved, especially the risk of each vertebra for PCE. Zou *et al.* (13) reported that patients who underwent PVP had a significantly increased PCE risk than did those who underwent PKP. In contrast, a randomized control trial indicated that PKP had fewer cement leakages than did PVP and had a similar incidence of cement embolism (14). The difference in PCE caused by PVP and PKP remains unknown. Therefore, the purpose of our study was to summarize the characteristics of PCE on chest CT, compare the incidence d of PCE between PVP and PKP, and analyze the incidence and risk factors of PCE. We present the following article in accordance with the STROBE reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-22-569/rc).

Methods

Study cohort and design

This single-center, retrospective cohort study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the institutional ethics board of the China-Japan Friendship Hospital (No. 2022-KY-048), and individual consent for this retrospective analysis was waived. First, we retrospectively searched the electronic medical record system of China-Japan Friendship Hospital from January 2017 to December 2020. We screened 472 cases (157 males and 315 females; mean age, 78.0±9.4 years) with vertebral compression fracture or PVP or PKP. Based on the patients' information, we only included the patients with preoperative vertebral X-ray and chest CT images and a postprocedural chest CT (including a dedicated chest CT with or without intravenous contrast, low-dose chest CT, or CT pulmonary angiography) through the Picture Archiving and Communication Systems (PACS; Carestream Health). A total of 99 patients were excluded because there was no postprocedural chest CT in our PACS, and then 373 patients were finally included in this research. Each patient's demographic and clinical information, including operation type, operation level, the number of involved vertebrae, and the time interval from the operation to the first postoperative chest CT, were collected by 3 residents. The detailed flowchart of patient screening is shown in *Figure 1*.

Image analysis

Two radiologists with 6 years of experience reviewed chest CT images together. They determined whether PCE could be diagnosed and the distribution of cement emboli. If there
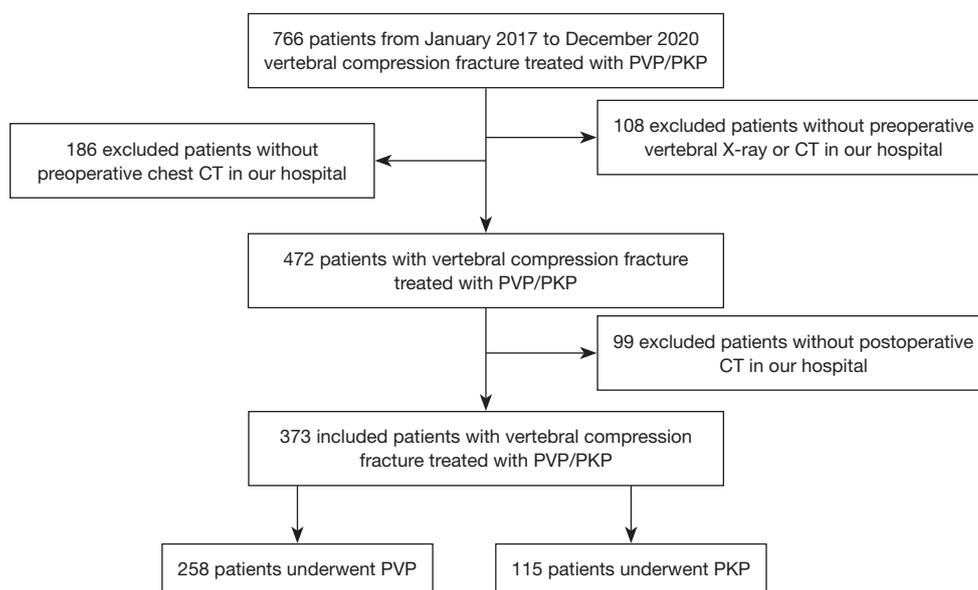
Figure 1 The detailed flowchart of the patient screening process. PVP, percutaneous vertebroplasty; PKP, percutaneous kyphoplasty; CT, computed tomography.

was a disagreement of opinion, the final diagnosis was made by a third chest radiologist with 15 years of experience.

PCE was diagnosed on the basis of a history of PVP or PKP and chest CT findings. PCE was defined as tubular, linear, or branching radiopaque densities with an attenuation >500 HU located along the course of the pulmonary artery on chest CT (15,16). Central PCE involved the main, right, and left as well as the lobar pulmonary arteries, while peripheral PCE only involved segmental and subsegmental pulmonary arteries.

Statistical analysis

The clinical and imaging data were analyzed using SPSS 26.0 (IBM Corp., New York, United States). Continuous variables are presented as mean ± SD. Comparisons between groups were performed using the t test or χ^2 test. Univariate and binary logistic regression analysis was performed on possible risk factors for the occurrence of PCE. A 2-tailed P value <0.05 was considered statistically significant.

Results

The demographics and clinical information

Of the 373 patients (96 males, 277 females; mean age 76.2±9.4 years, age range 31–97 years), 258 patients

underwent PVP, and the remaining 115 patients underwent PKP, involving a total of 472 vertebrae. After PVP or PKP, 274 patients underwent noncontrast chest CT, 19 patients underwent low-dose chest CT, 67 patients underwent high-resolution CT (HRCT), and 13 patients underwent CT pulmonary angiography (CTPA). PCE was detected using the postprocedural chest CT in 64 cases (16 males and 48 females; mean age 75.8±11.0 years) with an incidence of 17.2%. The time interval between PVP/PKP and the first chest CT after PVP/PKP was 2 to 1,482 days (median 412 days; 95% CI: 111–802 days). CTPA was performed in 13 cases within 2 days after PVP, in whom 4 and 2 patients were respectively diagnosed with PCE and acute pulmonary thromboembolism (APE). *Figure 2* shows the distribution of surgical sites and the number of patients. *Table 1* shows the demographics and clinical information of all enrolled patients.

CT findings of PCE

Noncontrast chest CT indicates cement emboli as branching hyperdense or radiopaque lesions confined within the pulmonary artery courses (*Figure 3*). CTPA can also clearly show the linear hyperdense cement emboli in the pulmonary artery (*Figure 4*). There were 11 cases with PCE with the central type and 53 cases with the peripheral type.
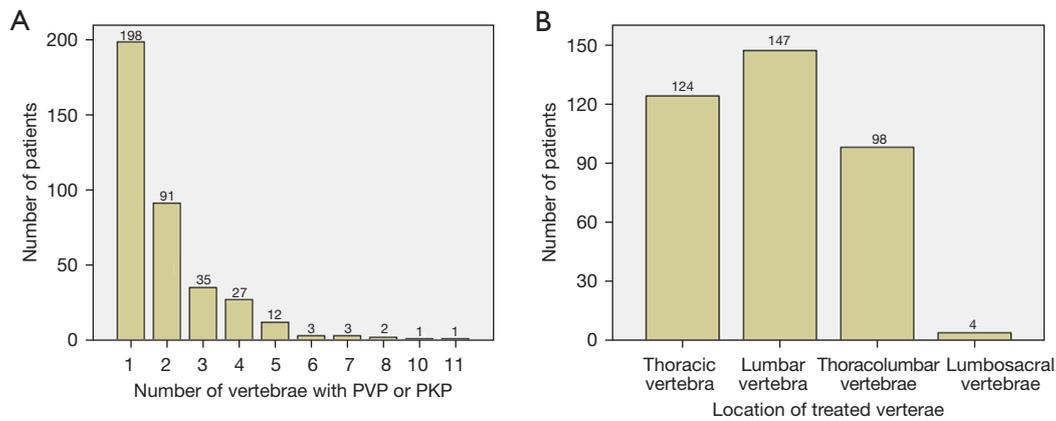
Figure 2 Numbers of patients with PVP and PKP and the distribution of the treated vertebrae. (A) The number of patients according the number of vertebrae treated with PVP or PKP differed. A total of 198 patients were treated with PVP or PKP in 1 vertebra, and 91 patients were treated with PVP or PKP in 2 vertebrae. (B) The number of patients and the distribution of the treated vertebrae with PVP or PKP. A total of 124 patients were treated with PVP or PKP in the thoracic vertebrae. PVP, percutaneous vertebroplasty; PKP, percutaneous kyphoplasty.

Table 1 The demographics and clinical information of all enrolled patients

Clinical information	Patients with pulmonary cement embolism			P value
	Sum (N=373)	Positive (n=64)	Negative (n=309)	
Gender				0.882
Male	96	16	80	
Female	277	48	229	
Age (years), mean ± SD	76.2±9.4	75.8±11.0	76.3±9.1	0.708
Puncture method				0.406
Percutaneous vertebroplasty	258	47	211	
Percutaneous kyphoplasty	115	17	98	
Location of treated vertebrae				0.365
Thoracic	124	24	100	
Lumbar	147	19	128	
Thoracolumbar	98	20	78	
Lumbosacral	4	1	3	
Treated vertebrae				
T1 vertebra	0	0	0	
T2 vertebra	1	0	1	
T3 vertebra	1	0	1	
T4 vertebra	2	1	1	
T5 vertebra	9	2	7	
T6 vertebra	11	5	6	

Table 1 (*continued*)

Table 1 (continued)

Clinical information	Patients with pulmonary cement embolism			P value
	Sum (N=373)	Positive (n=64)	Negative (n=309)	
T7 vertebra	22	8	14	
T8 vertebra	28	7	21	
T9 vertebra	29	13	16	
T10 vertebra	27	10	17	
T11 vertebra	76	21	55	
T12 vertebra	137	28	108	
L1 vertebra	148	23	125	
L2vertebra	103	27	76	
L3 vertebra	53	13	40	
L4 vertebra	52	11	41	
L5 vertebra	29	7	22	
Sacral vertebra	3	1	2	
Number of treated vertebrae	730	177	553	0.001
Cement leakage in the azygos vein				<0.001
Positive	103	39	64	
Negative	270	25	245	
Cement leakage in the inferior vena cava				<0.001
Positive	8	6	2	
Negative	365	58	307	
Cement leakage in the renal vein				
Positive	1	1	0	
Negative	372	63	309	

SD, standard deviation.

Cement embolisms were mainly involved in the anterior segment of the upper lobe of bilateral lungs; there were 39 cases in the anterior segment of the upper lobe of the right lung and 23 cases in the anterior segment of the upper lobe of the left lung. Cement embolisms were also present in the apical segment of the upper lobe of the right lung (20 cases), the upper lingual segment of the upper lobe of the left lung (18 cases), the posterior segment of the upper lobe of the right lung (16 cases), and the medial segment of the middle lobe of the right lung (17 cases). CT further showed that 103 cases had cement emboli in the azygos vein (*Figure 5*), and 8 cases had cement emboli in the inferior vena cava (*Figure 6*).

The diameter of the main pulmonary artery trunk in the PCE group was 27.6±4.8 cm, and there was no significant difference between the PCE and non-PCE groups (27.9±4.8 cm; t=0.454; P=0.650). The ratio of the main pulmonary artery diameter to aorta diameter in the PCE group (0.78±0.13) was comparable to that of the non-PCE group (0.79±0.13; t=0.905; P=0.366).

Risk factors of PCE

There was no significant gender difference between the PCE and non-PCE groups (χ^2=0.022; P=0.882). The age in the PCE and non-PCE groups was comparable (t=0.375; P=0.708). The patients who underwent PVP

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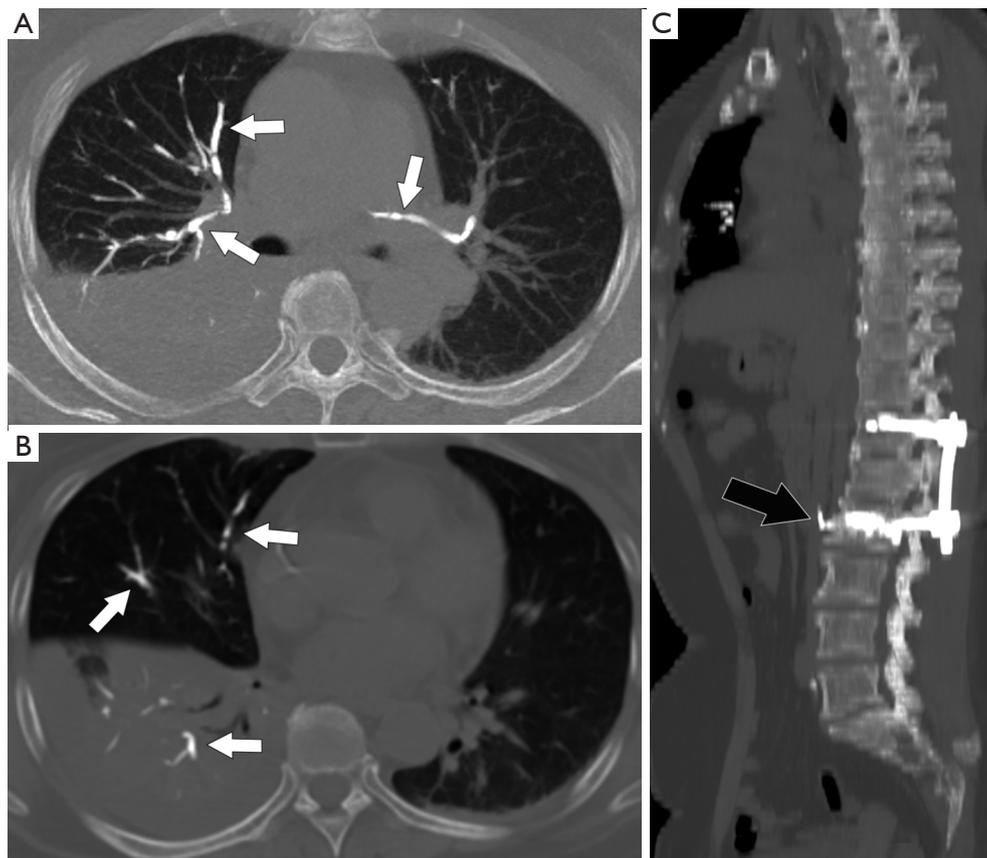
Figure 3 PCE in a 57-year-old female patient after PVP. (A,B) The pulmonary artery cement emboli appear as multiple linear, branched, high-density opacities in both lungs (white arrows) in noncontrast chest CT. (C) The sagittal image in maximum intensity projection of noncontrast chest CT showing cement leakage into the azygos veins (black arrow). PCE, pulmonary cement embolism; PVP, percutaneous vertebroplasty; CT, computed tomography.
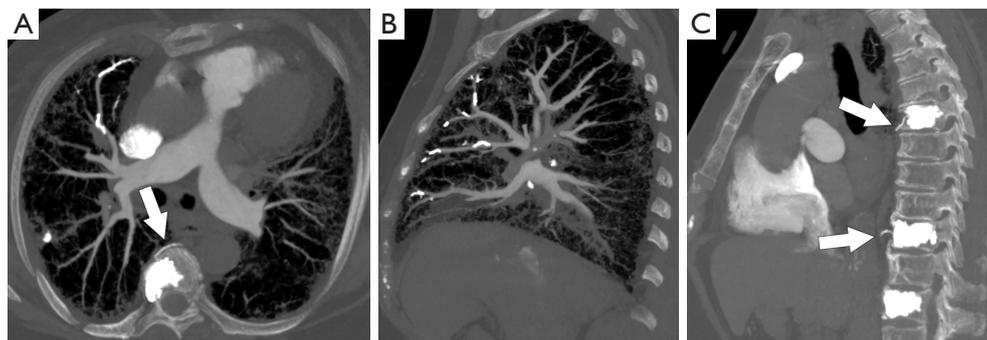
Figure 4 PCE in an 80-year-old female patient after PVP. (A) The transversal image of CTPA showing multiple linear and branched high-density cement emboli in the upper and middle lobe of the right lung and ascending lumbar vein (white arrow). (B) The sagittal image in maximum intensity projection of CTPA showing multiple linear and branched high-density cement emboli in the upper and middle lobe of the right lung. (C) The sagittal image in maximum intensity projection of CTPA showing the leakage of cement emboli into the ascending lumbar vein (white arrows). PCE, pulmonary cement embolism; PVP, percutaneous vertebroplasty; CTPA, computed tomography pulmonary angiography.
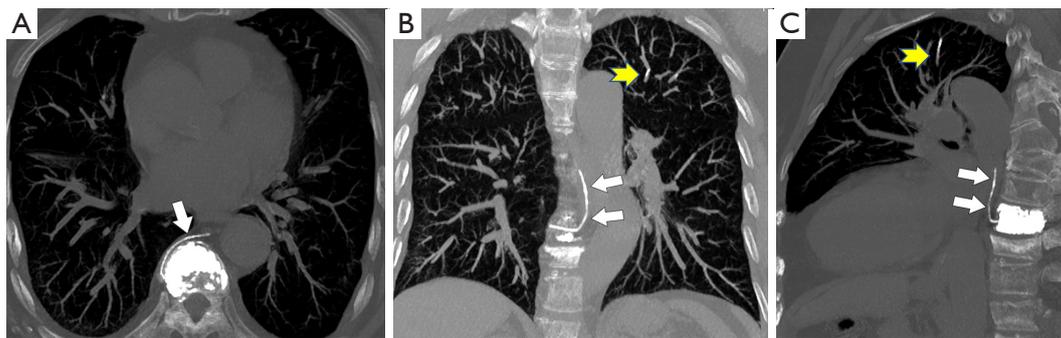
Figure 5 PCE in a 71-year-old female patient after PKP. (A) The transversal images (A), the coronal image (B), and the sagittal image (C) of noncontrast CT showing the leakage of cement emboli into the azygos vein (white arrows). (B,C) PCE in the upper lobe of the left lung (yellow arrowheads). PCE, pulmonary cement embolism; PVP, percutaneous vertebroplasty; PKP, percutaneous kyphoplasty; CT, computed tomography.
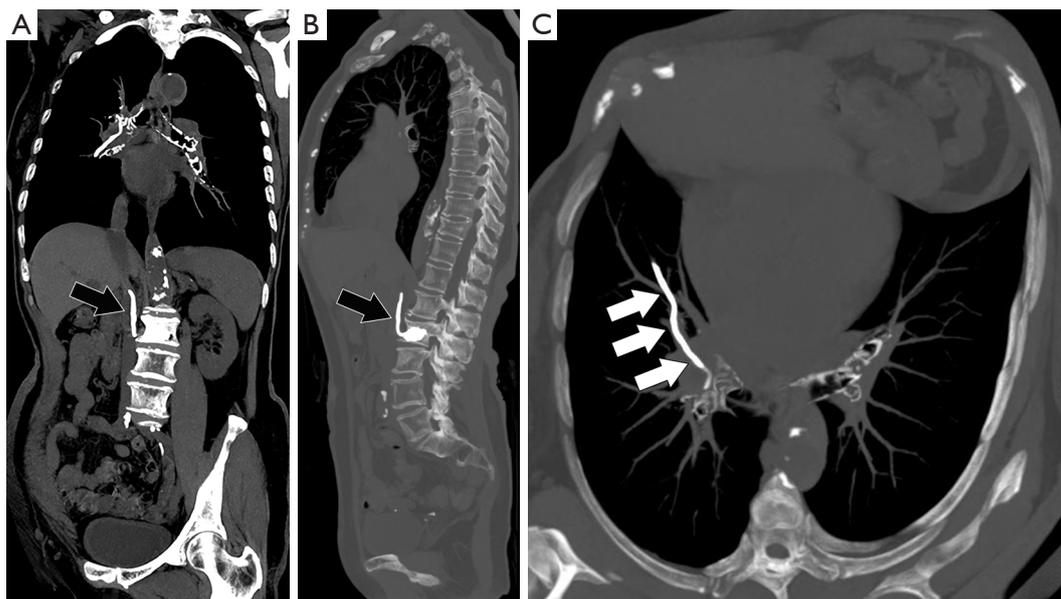
Figure 6 PCE in a 78-year-old female patient after PVP. (A) The coronal image and (B) sagittal image in maximum intensity projection of noncontrast CT showing cement leakage in the inferior vena cava (black arrows). (C) The sagittal image showing long linear cement emboli in the middle lobe of the right lung (white arrows). PCE, pulmonary cement embolism; PVP, percutaneous vertebroplasty; CT, computed tomography.

and PKP between the PCE and non-PCE groups were similar (χ^2=0.660; P=0.460). As shown in *Table 1*, there was no significant difference in the distribution of the treated vertebrae (thoracic vertebra, lumbar vertebra, thoracolumbar vertebrae, lumbosacral vertebrae) between the PCE and non-PCE groups (χ^2=3.175; P=0.365). However, for each vertebra, PVP or PKP on T6 (χ^2=6.385; P=0.026), T7 (χ^2=6.067; P=0.021), T9 (χ^2=16.937; P<0.001), T10 (χ^2=8.092; P=0.008), T11 (χ^2=7.366; P=0.007), and L2

vertebrae (χ^2=8.209; P=0.004) in the PCE and non-PCE groups were significantly different. Between the PCE and non-PCE groups, there was also a significant difference in the number of treated vertebrae (χ^2=23.71; P=0.001), azygos vein emboli (χ^2=41.93, P<0.001), and inferior vena cava cement emboli (χ^2=14.29; P<0.001). Binary logistic regression analysis (*Table 2*) showed that PVP or PKP on the T9 vertebra [odds ratio (OR) =4.222; 95% CI: 1.490–11.966], azygos vein emboli (OR =7.647; 95% CI: 3.937–

Table 2 Risk factors of pulmonary cement embolism after PVP or PKP

Factors	P value	OR	95% CI	
			Lower limit	Upper limit
T6 vertebra	0.059	4.322	0.947	19.723
T7 vertebra	0.287	1.996	0.599	7.119
T9 vertebra	0.007	4.222	1.490	11.966
T10 vertebra	0.250	1.990	0.616	6.429
T11 vertebra	0.121	1.862	0.849	4.083
L2 vertebra	0.088	1.998	0.902	4.428
Number of treated vertebrae	0.917	0.985	0.746	1.301
Cement emboli in the azygos vein	<0.001	7.647	3.937	14.856
Cement emboli in inferior vena cava	<0.001	42.701	7.525	242.302

PVP, percutaneous vertebroplasty; PKP, percutaneous kyphoplasty; OR, odds ratio; CI, confidence interval.

14.856), and inferior vena cava cement emboli (OR =42.701; 95% CI: 7.525–242.302) were risk factors for PCE.

Discussion

In our study, the incidence of PCE from PVP or PKP was 17.2%, and the incidence of PCE after PVP compared to that after PKP was similar. Postprocedural chest CT clearly indicated PCE as branching hyperdense or radiopaque lesions confined within the pulmonary artery courses. Cement emboli were mainly distributed in the upper lobes of both lungs. PVP or PKP in the T9 vertebra and cement emboli in the azygos vein or the inferior vena cava were risk factors of PCE after PVP and PKP.

Cement in the vertebra leaks into paravertebral venous plexus, from the azygos vein into the superior vena cava, or through the left renal vein and inferior vena cava, so as to enter the right atrium, right ventricle, or pulmonary artery, resulting in PCE (17-21). PCE has been extensively reported as a complication of PVP or PKP techniques, with the majority of the related literature being the form of case reports in which chest radiography was used for evaluation. Choe *et al*. (22) reported PCE is confirmed radiographically in 4.6% of patients after PVP or PKP. Another study retrospectively reviewed postprocedural chest radiographs, and 5 of 73 patients (6.8%) had PCE (8). Using follow-up CT, Venmans *et al*. (23) reported the incidence of PCE after PVP to be 26% (95% CI: 16–39%). In osteoporotic vertebral compression fractures, PCE was detected with CT in 23% of PVP sessions, developed in the

distal to third-order pulmonary arteries, and was related to leakage into the inferior vena cava (16,24). In a systematic review, the incidence of PCE ranged widely, from 1.9% to 26% (11). In our study, the incidence of PCE from PVP and PKP was 17.2%. However, a chest radiograph or CT is not mandatory after PVP or PKP, so the true incidence of PCE may be underestimated (8,18). In cases of asymptomatic patients with peripheral PCE, Krueger *et al*. (12) recommended no treatment besides clinical follow-up. However, in cases of symptomatic emboli, they recommended proceeding according to the guidelines regarding the treatment of thrombotic pulmonary emboli, which included initial heparinization and a subsequent 6-month coumarin therapy. In our study, the median time interval between PVP or PKP and a chest CT scan after PVP or PKP was 412 days (95% CI: 111–802 days), indicating that asymptomatic PCE was a common condition during PVP and PKP and that asymptomatic patients did not receive any special treatment. Moreover, although PVP and PKP are two different percutaneous puncture procedures aimed at relieving pain rand reinforcing the bone of the vertebrae, our results found that the incidence of PCE from PVP and PKP was similar. This finding is consistent with the reports of Dohm *et al*. (14). However, Zou *et al*. (13) reported that patients who underwent PVP had a significantly increased risk of PCE compared to those who underwent PKP.

PCE on noncontrast CT is shown as linear, branching hyperdense, or radiopaque lesions in the pulmonary artery course. In contrast, the intensity of cement is higher

than that derived with an iodine contrast agent. Thus, CTPA also can clearly display the high-density cement emboli in the pulmonary artery. However, cement emboli may be underestimated due to the influence of high-intensity contrast agents in the pulmonary artery (15,16). Density measurement can also be helpful to overcome this drawback, as contrast agents usually have lower attenuation than do cement emboli. Kim *et al.* (16), Venmans *et al.* (23), and Cianciulli *et al.* (25) all reported cement emboli that were small and scattered in peripheral portions of the lungs without specific lobar distribution and with no cement deposits in the heart and large vessels. In contrast, our study had 11 cases with central-type PCE and 53 cases with peripheral-type PCE. Cement emboli were mainly involved in the anterior segment of the upper lobe of both lungs. Patients underwent PVP or PKP in the prone position and remained bed in the prone position after surgery. Therefore, we speculate that the prone position may be the main reason for the specific distribution of PCE.

In a comparable study with the use of CT to detect PCE, Kim *et al.* (16) found cement leakage to the inferior cava vein as the only relevant risk factor. Hsieh *et al.* (10) reported a statistically significant correlation between the leakage of cement into the inferior vena cava and PCE, while Choe *et al.* (22) reported an association between PCE and paravertebral venous cement leakage. Venmans *et al.* (23) indicated that cement in the azygos vein on postprocedural CT of the treated vertebrae to be the only risk factor for PCE. In the study by Fadili Hassani *et al.* (19), a higher number of treated vertebrae during the PV procedure was an independent risk factor for the occurrence of intracardiac cement embolism. Zou *et al.* (13) indicated that patients with a thoracic fracture have a significantly increased risk of PCE than do those with a thoracolumbar fracture. In our study, we first analyzed the risk of each vertebra for PCE and found PVP or PKP in the T9 vertebra to be a risk factor for PCE. It is possible that the middle thoracic spine, including the T9 vertebra, is a more common vertebra of PCE because it is in close proximity to the inferior vena cava and the vena cava. However, the specific mechanism of PCE from PVP or PKP in the T9 vertebra remains unclear. A greater number of treated vertebrae was also found in the PCE group. Moreover, PVP or PKP in the T9 vertebra and cement emboli in the azygos vein or the inferior vena cava were found to be risk factors for PCE. Fadili Hassani *et al.* (19) reported the incidence of intracardiac cement embolism during PVP was low (3.9%), and concomitant pulmonary artery cement leakage was detected in 86.1% of the cases examined. However, no intracardiac cement embolism was found in our study, and only 1 case with cement leakage in the renal vein was found. Moreover, in cases of asymptomatic patients after PKP/PVP, we believe that chest CT is not mandatory to diagnose or exclude PCE. In cases of symptomatic patients after PKP or PVP, noncontrast CT would be the best method for evaluating PCE. For patients with suspected pulmonary thromboembolism after PKP or PVP, contrast-enhanced CT can clearly demonstrate PCE and differentiate PCE from a pulmonary thromboembolism.

Limitations

There are several limitations to our study. First, we employed a single-center, retrospective design, and only a relatively small number of patients who underwent chest CT after PVP or PKP were included; therefore, the incidences of PCE after PVP or PKP might have been underestimated. Although the incidences of PCE after PVP and PKP were similar, the effect of the volume of cement and the time from the operation to PCE were not analyzed. Additionally, the time interval of PVP or PKP to postoperative chest CT scan was 2 to 1,482 days (median, 412 days; 95% CI: 111–802), indicating that most cases of PCE were asymptomatic. Ultimately, the postoperative clinical symptoms or biomarkers that can suggest PCE remain unknown.

Conclusions

In this retrospective study, the incidence of PCE from PVP or PKP was 17.2% while the incidences of PCE after PVP and PKP were similar. PVP or PKP in the T9 vertebra and cement emboli in the azygos vein or the inferior vena cava were risk factors for PCE.

Acknowledgments

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-22-569/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-22-569/coif). All authors report that this work was supported by National High Level Hospital Clinical Research Funding (No. 2022-NHLHCRF-LX-01); Elite Medical Professionals Project of China-Japan Friendship Hospital (No. ZRJY2021-BJ02); Medical and health science and technology innovation project of Chinese Academy of Medical Science (No. 2021-I2M-1-049) and the National Natural Science Foundation of China (No. 81871328). The authors have no other conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and was approved by the institutional ethics board of the China-Japan Friendship Hospital (No. 2022-KY-048). Individual consent for this retrospective analysis was waived.

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