Retrospective Study

# Intravertebral Vacuum Cleft and Its Varied Locations within Osteoporotic Vertebral Compression Fractures: Effect on Therapeutic Efficacy

Weibo Yu, MD[1], Xiaobing Jiang, PhD[2], De Liang, MM[2], Zhensong Yao, MD[2], Ting Qiu, MB[2], Linqiang Ye, MD[2], Shuncong Zhang, MD[2], and, Daxiang Jin, MD[2]

From: [1]Tongde Hospital of Zhejiang Province, China; [2]The First Affiliated Hospital of Guangzhou University of Chinese Medicine, China

Address Correspondence:
Xiaobing Jiang, PhD
The First Affiliated Hospital of Guangzhou, University of Chinese Medicine
Airport Rd. 16
Guangzhou 510405 China
E-mail:
yuweibo0530@163.com

Disclaimer: There was no external funding in the preparation of this manuscript.
Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.

Manuscript received: 05-18-2016
Revised manuscript received: 09-25-2016
Accepted for publication: 01-13-2017

Free full manuscript:
www.painphysicianjournal.com

**Background:** Previous studies have reported a high incidence of re-collapse of the augmented vertebrae after percutaneous vertebral augmentation (PVA) for osteoporotic vertebral compression fractures (OVCFs) with intravertebral vacuum cleft (IVC) during long-term follow-up. Previous IVC might be considered an important predisposing factor for re-collapse, but the prior studies could not find a significant correlation.

**Objective:** To determine the incidence and distribution characteristics of IVCs and to further assess IVCs in their varied locations. To assess the long-term therapeutic efficacy of PVA for OVCFs with IVC.

**Study Design:** A retrospective cohort study.

**Setting:** Department of spinal surgery, an affiliated hospital of a medical university.

**Methods:** A retrospective review was performed on 594 patients who underwent PVA to treat OVCFs from January 2010 to December 2013. Eighty-two patients with the IVC sign were enrolled in the study. The follow-up period was a minimum of 2 years. The difference between IVC and non-IVC patients was compared. Comparisons of the radiological and clinical findings at varied IVC locations were made pre-operatively and post-operatively (immediate, at one year, and at 2 years).

**Results:** IVC incidence correlated with older patient age and severe demineralization. Other baseline parameters showed no significant differences. The rate of cement leakage and vertebral fracture was significantly lower in the IVC groups than in the non-IVC groups intraoperatively. There was no significant difference in the incidence of cement leakage or adjacent vertebral fractures between the 3 IVC groups. In the immediate postoperative period, all patients benefited from significant improvement in vertebral body height and kyphotic angle correction. However, significant re-collapse was observed at the 2-year post-operative follow-up for the IVC patients when compared to the non-IVC patients. Among the 3 IVC groups, the most severe re-collapse was observed with inferior endplate IVCs. Superior endplate IVCs and IVCs extending to both endplates demonstrated only mild re-collapse at the 2-year follow-up.

**Limitation:** Due to the infrequency of this process, the number of patients with IVCs was small.

**Conclusion:** PVA treatment was initially effective in all patients with OVCFs. However, significant re-collapse of the augmented vertebrae with IVCs, especially those with inferior endplate IVCs, was found with long-term follow-up.

**Key words:** Intravertebral vacuum cleft, percutaneous vertebral augmentation, osteoporotic vertebral compression fractures, affected vertebrae, augmented vertebrae

**Pain Physician 2017; 20:E979-E989**

Osteoporosis is common in the elderly and is becoming the leading cause of vertebral compression fractures as the size of the geriatric population increases (1). Osteoporotic vertebral compression fractures (OVCFs) can lead to spinal kyphotic deformities, can be the cause of back/lumbar pain and balance impairments, and reduce patients' quality of life (2,3).

The development of an intravertebral vacuum cleft (IVC) in OVCFs (incidence of 10% – 48% [4,5]) has been reported as an important risk factor for severe vertebral collapse, progressive kyphosis, intractable back pain, and neurologic deficits (6,7). IVC refers to a prominent radiolucency (gas containing), located centrally or adjacent to the vertebral body endplates as seen on computed tomography (CT) or plain radiographs (4,8). On magnetic resonance imaging (MRI), an IVC usually shows as low signal intensity on T1-weighted images and high or low signal on T2-weighted images, which mainly depends on whether fluid or gas fills the cleft, respectively (9,10).

To restore spinal stability and obviate severe pain, percutaneous vertebral augmentation (PVA) has been widely recommended and has also achieved good outcomes with initial short-term follow-up (11-13). However, previous studies (14) have reported a high incidence of re-collapse of the augmented vertebrae at long-term follow-up. They believed that having had an IVC might be an important predisposing factor for re-collapse, but they could not find a significant relationship between an IVC and re-collapse. Moreover, in conjunction with the previous reports (4,8), the varied locations of an IVC include adjacent to the superior endplate, adjacent to the inferior endplate, or an oblique orientation communicating with both endplates. Hence, we hypothesize that the location of the IVC may be a significant factor in re-collapse. The aim of this retrospective study is to determine the incidence and distribution characteristics of the IVC sign in OVCFs, and to demonstrate the impact that the location of the IVC has on the long-term therapeutic efficacy of PVA.

## Methods

### Patients

To determine whether the location of the IVC is a significant factor in re-collapse post PVA, 834 consecutive patients who had undergone PVA for the treatment of OVCFs were investigated between January 2010 and December 2013. The inclusion criteria were as follows: (1) a single-level osteoporotic vertebral fracture and no previous adjacent osteoporotic vertebral fracture; (2) a thoracolumbar (T11-L1) location of the affected vertebrae; (3) treatment with single-level PVA (percutaneous vertebrolplasty (PVP) or percutaneous kyphoplasty (PKP)) via bilateral portals; (4) a follow-up period of at least 2 years; (5) no additional history of trauma after surgery; (6) no complication after surgery, including leakage of polymethylmethacrylate (PMMA) into the spinal canal, or postoperative neurologic deficit et al; and (7) regular radiologic follow-up studies and continuation of osteoporotic medications throughout the follow-up period. Exclusion criteria were severe trauma, known malignancies, neoplastic fractures, and spinal infections. A total of 594 patients were enrolled in our study (M/F = 140:454). The average age of the patients was 73.1 years (range 60 – 90 years).

### PVA

All of the PVA procedures were performed by 3 spinal surgeons with greater than 10 years' experience. Each of the surgeons received standard training in PVP or PKP. The PVA technique was adopted by using a transpedicular approach (bipedicular needle insertion) in an extended posture under local anesthesia (1% lidocaine) in all cases according to methods used in previous reports (11-13,15). During the operation, 11 to 13 gauge bone biopsy needles were inserted parallel, or in a slightly descending course, through the pedicle until the needle tip was optimally positioned in the IVC area. Then, the stylet was removed from the trocar and PMMA powder with sterile barium sulfate (Tianjin Synthetic Material Research Institute, Tianjin, China) was injected in the cleft for complete filling of the cleft, maximizing stabilization of the fracture fragments. PVP was carried out on 58 patients with an IVC and 354 patients without an IVC, while PKP was used on 24 patients with an IVC and 158 patients without an IVC.

After PVA, patients were encouraged to ambulate as soon as possible. Patients were prescribed back braces to be worn for 1 – 2 months and osteoporotic medications to include bisphosphonates and vitamin D or raloxifen.

### The Evaluation for the Presence and Location of an IVC

Prior to PVA, all images were reviewed in consensus by 2 experienced radiologists. On CT and/or plain radiographs of the spine, an IVC sign was identified by a linear, triangular, or irregular region of low density

(gas) within a collapsed vertebral body. On MRI, an IVC usually shows as low signal intensity on T1-weighted images and high (fluid containing) or low signal (gas containing) on T2-weighted images (9,10). Additionally, a peripheral zone of hypointensity can be seen surrounding the hyperintensity on T-2 weighted images (4,8-10). In conjunction with previous reports of IVC locations (8,10), the varied locations of an IVC within the affected vertebral body were categorized and correlated with findings at long-term follow-up. In our study, 82 patients (M/F = 20:62; average age, 75.21 years) were identified with an IVC. The distributions of the location of the IVCs were as follows: adjacent to the superior endplate in 58 patients, adjacent to the inferior endplate in 12 patients, and extending to both endplates in 12 patients (Fig. 1).

### Data Collection of Radiological Parameters

We retrospectively measured preoperative and postoperative (immediately, one year, and 2 years) radiological parameters including vertebral height loss and kyphotic angle. The vertebral height loss and kyphotic angle were measured according to previous studies described by Ha and Kim (16) and Linn et al (8). Vertebral height was measured at the point of maximal collapse of the affected vertebrae. The vertebral compression rate (CR) at each follow-up period was measured as the rate of vertebral height of the

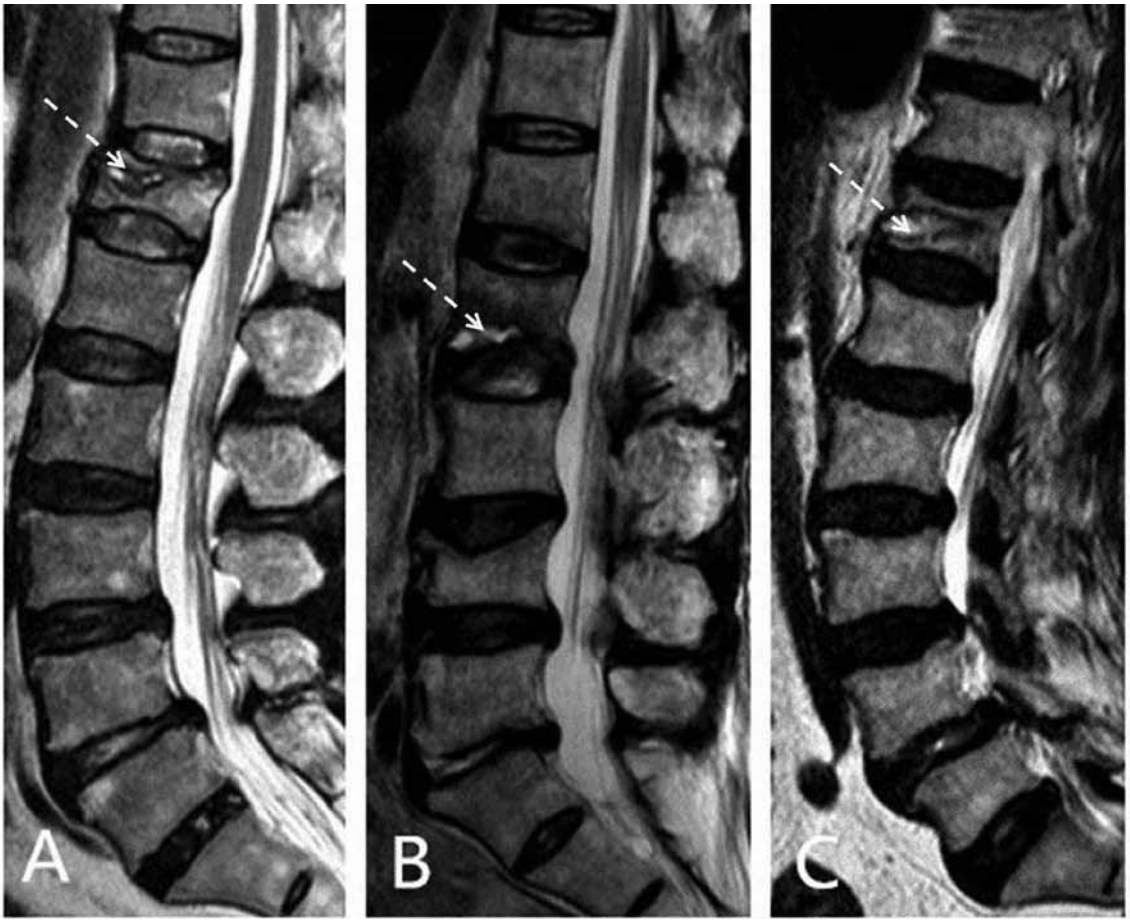


*Fig. 1. Distribution of the location of the IVCs on the sagittal T2-weighted (T2W) images. A: Sagittal T2W MRI demonstrates a small amount of fluid within an L1 superior endplate intravertebral cleft; B: Sagittal T2W MRI demonstrates fluid within an L2 inferior endplate intravertebral cleft. Chronic anterior compression fracture of L4; C: Sagittal T2W MRI demonstrates fluid within T12 extending to both endplates consistent with an intravertebral cleft. There is lumbarization of S1. IVC = intravertebral vacuum cleft.*

augmented vertebrae to the mean vertebral height of the upper and lower vertebrae. Reduction rate (RR) was calculated using the difference between preoperative and postoperative CR. The progressive height loss (PHL) was calculated using the difference between immediate postoperative and last follow-up period. The kyphotic angle (KA) was measured using Cobb's method between adjacent vertebrae, which was the angle between the upper endplate of the upper vertebral body and the lower endplate of the lower vertebrae. Reduction angle (RA) was calculated using the difference between the preoperative and postoperative KA. Progressive angle (PA) was calculated using the difference between the immediate postoperative and last follow-up period. Additionally, cement leakage was also assessed.

### Data Collection of Clinical Parameters

Several preoperative clinical parameters, including age, gender, bone mineral density (BMD), visual analog scale (VAS) score of back pain, and Oswestry Disability Index (ODI) were evaluated. Meanwhile, immediately postoperative, one-year, and 2-year VAS scores and ODI indices were compared.

### Statistical Analysis

SPSS 11.0 statistical software (SPSS, Inc., Chicago, IL, USA) was used for analysis. Comparisons were made before and at each postoperative follow-up appointment. Qualitative characteristics of groups were assessed using the Mann-Whitney U-test and t-test. Chi-squared tests were performed for categorical variables. A $P < 0.05$ was considered statistically significant.

## Results

The IVCs were observed in 82 out of 594 patients who had single OVCF in the thoracolumbar vertebrae (13.8% incidence). The primary location of the IVCs was adjacent to the superior endplate (70.7% incidence). There was no significant difference between the IVC and non-IVC patients in regard to baseline parameters including gender, VAS, ODI, vertebral height, and KA (Table 1). However, the patients in the IVC groups had lower BMD and an older patient age than did patients in the non-IVC group ($P < 0.05$, Table 1).

Although the BMD of the IVC patients was still lower than that of the non-IVC patients, Table 2 shows that the BMD had improved in all patients at the 2-year postoperative follow-up. In the immediate postoperative period, all patients benefited from significant improvement in vertebral body height and KA correction ($P < 0.05$). There was no significant difference in vertebral body height and KA between the IVC groups and the non-IVC group. However, significant re-collapse was observed at the 2-year postoperative follow-up for the IVC groups compared to the non-IVC group. Among the 3 IVC groups, the most severe re-collapse was observed with inferior endplate IVCs. The mean rate of vertebral compression and KA was 0.65 (SD 0.11) and 17.65 (SD 9.93), respectively, for the inferior endplate IVC group at the 2-year postoperative follow-up. In contrast, superior endplate IVCs and IVCs extending to both endplates only demonstrated mild re-collapse at the 2-year postoperative follow-up. The mean rate of vertebral compression and KA was 0.77 (SD 0.10) and 13.59 (SD 8.79) for the superior endplate IVC group; the mean

Table 1. *Baseline characteristics of patients in this study.*

| Patient characteristics | IVC group | | | Non-IVC group |
|---|---|---|---|---|
| | Adjacent to the superior endplate | Adjacent to the inferior endplate | Extending both endplates | |
| Number of patients | 58 | 12 | 12 | 512 |
| Age (years)a | 74.63 ± 9.96* | 75.86 ± 10.34* | 78.45 ± 11.23* | 72.23 ± 8.89* |
| Gender of female (%) | 46 (79.3) | 10 (83.3) | 6 (50.0) | 392 (76.5) |
| BMDa | -4.34 ± 0.94* | -4.35 ± 0.74* | -4.44 ± 1.06* | -3.34 ± 0.78* |
| vertebral heighta | 0.76 ± 0.10 | 0.75 ± 0.13 | 0.64 ± 0.22 | 0.77 ± 0.13 |
| kyphotic anglea | 13.76 ± 6.54 | 13.557.40 | 15.75 ± 5.61 | 13.33 ± 5.56 |
| VASa | 8.27 ± 0.36 | 8.14 ± 0.45 | 8.35 ± 0.56 | 8.27 ± 0.36 |
| ODIa | 83.77 ± 9.76 | 83.10 ± 7.62 | 84.67 ± 6.44 | 83.06 ± 4.79 |

IVC intravertebral vacuum cleft, BMD bone mineral density, VAS visual analogue scale, ODI Oswestry Disability Index
a Quantitative variables were expressed as mean ± SD
*$P < 0.05$ the comparison between IVC and non-IVC groups

Table 2. *The postoperative BMD and radiological parameters of patients within 2 years follow-up in our study.*

| | | IVC group | | | Non-IVC group |
|---|---|---|---|---|---|
| | | Adjacent to the superior endplate | Adjacent to the inferior endplate | Extending both endplates | |
| BMD | Preoperative | -4.34 ± 0.94 | -4.35 ± 0.74 | -4.44 ± 1.06 | -3.34 ± 0.78 |
| | After 1 year | -4.13 ± 0.76 | -4.11 ± 0.76 | -4.23 ± 0.93 | -3.18 ± 0.62 |
| | After 2 years | -4.01 ± 0.83 | -3.97 ± 0.69 | -4.01 ± 0.78 | -3.070.66 |
| CR | Preoperative | 0.76 ± 0.10 | 0.75 ± 0.13 | 0.64 ± 0.22 | 0.76 ± 0.10 |
| | After 1st day | 0.85 ± 0.09* | 0.85 ± 0.11* | 0.84 ± 0.14* | 0.85 ± 0.07* |
| | After 1 year | 0.79 ± 0.10 | 0.68 ± 0.10 | 0.81 ± 0.11 | 0.82 ± 0.10 |
| | After 2 years | 0.77 ± 0.10 | 0.65 ± 0.11* | 0.80 ± 0.12* | 0.79 ± 0.11* |
| KA | Preoperative | 13.76 ± 6.54 | 13.55 ± 7.40 | 15.75 ± 5.61 | 13.33 ± 5.56 |
| | After 1st day | 9.20 ± 7.36* | 9.56 ± 6.97* | 12.81 ± 6.44* | 9.30 ± 6.93* |
| | After 1 year | 12.41 ± 8.32 | 16.50 ± 8.61 | 13.95 ± 6.82 | 10.567.56 |
| | After 2 years | 13.59 ± 8.79 | 17.65 ± 9.93 | 14.18 ± 7.86 | 11.716.74* |

BMD bone mineral density; CR the rate of vertebral compression; KA the kyphotic angle, IVC intravertebral vacuum cleft
Data was shown as mean ± standard deviation
*$P < 0.05$ compared to the preoperative measurement

Table 3. *The comparison of radiological parameters and common complication of patients in our study.*

| | IVC group | | | Non-IVC group |
|---|---|---|---|---|
| | Adjacent to superior endplate | Adjacent to inferior endplate | Extending both endplates | |
| Reduction rate | 0.09 ± 0.07 | 0.10 ± 0.05 | 0.15 ± 0.13 | 0.09 ± 0.05 |
| Progressive height loss | 0.08 ± 0.07* | 0.19 ± 0.11* | 0.04 ± 0.04* | 0.06 ± 0.05* |
| Reduction angle | 4.29 ± 4.56 | 4.00 ± 3.30 | 2.94 ± 3.53 | 4.11 ± 3.98 |
| Progressive angle | 7.39 ± 5.26* | 13.10 ± 5.84* | 6.37 ± 4.13* | 3.47 ± 4.34* |
| Cement leakage (%) | 4 (6.9)* | 1 (8.3)* | 2 (16.7)* | 89 (17.4)* |
| Adjacent vertebral fracture (%) | 3 (5.2)* | 1 (8.3)* | 2 (16.7)* | 102 (19.9)* |

Data was shown as mean ± standard deviation
*$P < 0.05$ the comparison among all groups

rate of vertebral compression and KA was 0.80 (SD 0.12) and 14.18 (SD 7.86) for the group with the IVCs extending to both endplates.

Table 3 further demonstrates the change of the rate of vertebral compression and KA at the 2-year postoperative follow-up. The RR and RA showed no significant difference in all patients ($P > 0.05$). However, PHL and PA of the IVC groups was greater than that of the non-IVC group. Among the 3 IVC groups, PHL and PA was the greatest for the inferior endplate IVC group ($P < 0.05$). Progression of re-collapse of the augmented vertebrae in the inferior endplate IVC group was confirmed by imaging analysis and serial follow-up (Fig. 2).

Table 3 also demonstrated 2 common complications including cement leakage and adjacent vertebral fractures. The rate of cement leakage in the IVC groups was significantly lower than that in the non-IVC groups intraoperatively ($P < 0.05$, 8.5% versus 17.4%). The rate of cement leakage showed no significant difference in the 3 IVC groups ($P > 0.05$). Similarly, the incidence of adjacent vertebral fractures in the IVC groups was also significantly lower than that in the non-IVC groups intraoperatively ($P < 0.05$, 7.3% versus 19.9%). The incidence of adjacent vertebral fractures showed no significant difference between the 3 IVC groups ($P > 0.05$).

Table 4 shows the change of VAS and ODI scores.

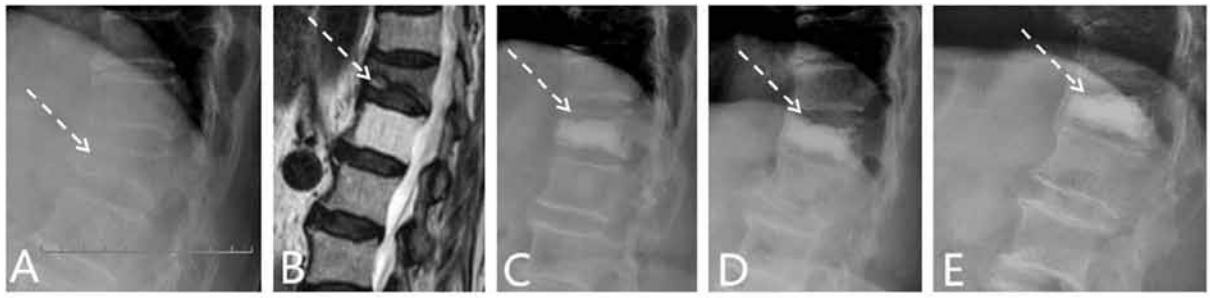


*Fig. 2. Lateral radiograph and sagittal T2W MRI demonstrate progressive re-collapse of an augmented vertebrae in a 76-year-old woman with a T12 compression fracture. A: Lateral radiograph demonstrates a T12 compression fracture. B: T2W MRI demonstrates an inferior endplate IVC with internal high/fluid signal. There was a hemangioma in L1; C: Immediate postoperative lateral radiograph shows well-deposited cement and re-expansion of the compressed vertebrae. A severe re-collapse developed at the one-year follow-up D: and 2-year follow-up E.*

Table 4. *The VAS and ODI values of patients within 2 years follow-up in our study.*

| | | Preoperative | Postoperative 1st day | Postoperative 1st year | Postoperative 2nd year |
|---|---|---|---|---|---|
| VAS | Adjacent to superior endplate | 8.27 ± 0.36 | 1.78 ± 1.04* | 2.28 ± 1.45 | 2.37 ± 1.45*# |
| | Adjacent to inferior endplate | 8.14 ± 0.45 | 1.72 ± 0.98* | 2.79 ± 1.42 | 4.15 ± 1.12*# |
| | Extending both endplates | 8.35 ± 0.56 | 1.66 ± 0.76* | 1.78 ± 1.01 | 1.81 ± 0.93*# |
| | Non-IVC group | 8.27 ± 0.36 | 1.60 ± 0.94* | 1.79 ± 0.89 | 2.01 ± 0.78*# |
| ODI | Adjacent to superior endplate | 83.77 ± 9.76 | 27.30 ± 7.40* | 29.36 ± 4.75 | 31.12 ± 4.93*# |
| | Adjacent to inferior endplate | 83.10 ± 7.62 | 26.09 ± 6.03* | 34.11 ± 8.70 | 41.03 ± 8.90*# |
| | Extending both endplates | 84.67 ± 6.44 | 27.33 ± 7.97* | 29.24 ± 3.86 | 32.54 ± 4.23*# |
| | Non-IVC group | 83.06 ± 4.79 | 26.73 ± 4.98* | 27.88 ± 4.87 | 28.50 ± 5.01*# |

VAS visual analogue scale, ODI Oswestry Disability Index, IVC intravertebral vacuum cleft
Data was expressed as mean ± SD
*$P < 0.05$ compared to the preoperative measurement
#$P < 0.05$ the comparison among all groups at the same follow-up stage

The VAS and ODI scores had the same patterns for all patients as compared to radiologic parameters. In the immediate postoperative period, the VAS and ODI scores of all patients decreased significantly ($P < 0.05$). There was no significant difference between the 3 IVC groups ($P > 0.05$). However, at the 2-year postoperative follow-up, the VAS and ODI scores in the IVC groups were significantly higher than that in the non-IVC group ($P < 0.05$). Among the 3 IVC groups, the VAS and ODI scores were the highest for the inferior endplate IVC group at the 2-year postoperative follow-up ($P < 0.05$).

## Discussion

IVC is considered to be a sign of avascular necrosis of the vertebral body (4,5). Maldague et al (17) first associated avascular necrosis with the IVC sign in 1978 by following up 10 patients with an IVC sign. They collected the histological data of one patient and radiologic data of 6 patients, and presumed that the IVC was a specific sign of local bone ischemia. Anatomic studies conducted by Ratcliffe (18) also suggested that the presence of the IVCs correlated with avascular necrosis. The vertebral ventral zone in the thoracolumbar region is only supplied by anterior peripheral and metaphyseal arteries. When OVCFs take place, these weak arteries are compromised. Furthermore, an insufficient revascularization and bone fracture healing process occurs (19,20). In addition, Dupuy et al (21) also conducted a study by observing the substance compositions in the IVC area during CT-guided biopsies. They found that the substances in the IVC area were composed of ne-

crotic cancellous bone, hyaline cartilage with fractured callus, and fluid collections, all of which were associated with underlying avascular necrosis.

Prior studies (22) have reported the location of the affected vertebrae with the IVCs occurred predominantly at the thoracolumbar junction. Our finding was compatible with theirs. Based on increased motion and greater load in this area, Ito et al (7) described the evolution of an IVC as initially taking place in the weaker anterior column of the index vertebra, and gradually expanding toward the anterior wall to cause dynamic mobility. Eventually the posterior wall becomes involved causing collapse of the entire index vertebra. In our study, the most common location of the IVC within the affected vertebral body was adjacent to the superior endplate (70.7% incidence). Linn et al (8) and Maldague et al (17) had similar results, which were 62.8% and 70% in their studies, respectively.

In our study, IVC incidence correlated with older patient age and severe demineralization. Under normal conditions, vertebral fractures should undergo an active remodeling of the vertebral body. However, the affected vertebra with severe demineralization has increased bone fragility, and has decreased the ability for remodeling of the affected vertebrae. Hence, an avascular necrosis area is more likely to occur presenting with an IVC sign.

IVCs create instability within the fractured vertebral body which could be detected on the radiographs comparing neutral with flexion and extension views (23,24). The patients with IVCs usually have severe back pain and do not respond to conservative treatment such as bed rest, medication, etc. (11,25). Hence, it is necessary to treat IVCs by surgical intervention with PVA in order to restore spinal stability by restoring continuity of the vertebral body and by slowing or halting progressive collapse of the affected vertebrae (11-13). In our study, all patients demonstrated significant improvement in radiographic findings and in their clinical evaluations during the immediate postoperative period. The results conducted by previous studies (14,15) also demonstrated good outcomes during the initial short-term follow-up. However, significant re-collapse was observed at the 2-year postoperative follow-up for the IVC patients when compared with the non-IVC patients in our study. The distribution pattern of PMMA cement might be an important predisposing factor. In the IVC groups, PMMA is injected into the cleft forming a focal conglomerate of cement. This conglomerate may induce greater stress upon the already weakened surrounding trabecula (26,27). The greater the degree of demineralization, the greater the fracture risk of un-augmented trabecula with the affected vertebral body. This might explain the significant re-collapse of the 'PMMA-nonsupported' area of the affected vertebra with IVCs.

In our study, the rate of cement leakage was significantly lower in the IVC groups than that in the non-IVC groups intraoperatively. It was possible that the fibrocartilagenous membrane at the periphery of the IVC prevents cement extrusion (26,27). It was observed that the incidence of new adjacent vertebral fractures was lower in the IVC groups than that in the non-IVC group at the 2-year postoperative follow-up. The biomechanical changes reported by Kim et al (28) and Wilke et al (29) also supported our results. They found that the stiffness of the augmented vertebral body filled with a focal conglomerate of cement was also significantly lower than that interspersed throughout the trabecular bone space of cement. Hence, it might reduce the risk of a new fracture of adjacent vertebral bodies in the IVC groups.

Among the IVC groups, the most severe re-collapse was observed with inferior endplate IVCs in our study. In our series of cases with the superior endplate IVCs, the upper aspect of the affected vertebral body was normally filled with a focal conglomerate of PMMA, and the interface between cement and unsupported surrounding trabecula was routinely located in the inferior aspect of the affected vertebral body. On the contrary, when the inferior endplate IVCs were presented, the inferior aspect of the affected vertebral body was normally filled and the interface was routinely located in the superior aspect of the affected vertebral body. Based on prior biomechanical perspective (28,30), some studies have demonstrated that the stress in the superior aspect of the affected vertebral body is higher than that in the inferior aspect. For this reason, the stress of the interface might be higher when the interface is located in the superior aspect of the affected vertebral body compared with that in the inferior aspect. This might explain our results that the concentrated cement in the inferior endplate IVCs group could induce exaggerated localized stress on the surrounding trabecula thus causing severe re-collapse.

The limitation of this study was that it was a retrospective study and had only 2 years of follow-up. Due to the low incidence of this disease, the number of patients was relatively small. A prospective large

study with longer term follow-up is needed to verify the conclusion.

## Conclusion

According to our results, IVCs are not a rare phenomenon in OVCFs, and the location of the IVC is an important factor in re-collapse post PVA. PVA may not provide long-term stabilization for patients with IVCs, especially when the inferior endplate is involved. Because of significant re-collapse for the IVC patients at long-term follow-up, we strongly recommend rigorous observation and extended follow-up for these patients, especially patients with inferior endplate IVCs.

## References


1. Johnell O, Kanis JA. An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporos Int* 2006; 17:1726-1733.
2. Lamy O, Uebelhart B, Aubry-Rozier B. Risks and benefits of percutaneous vertebroplasty or kyphoplasty in the management of osteoporotic vertebral fractures. *Osteoporos Int* 2014; 25:807-819.
3. Kado DM, Huang MH, Karlamangla AS, Barrett-Connor E, Greendale GA. Hyperkyphotic posture predicts mortality in older community-dwelling men and women: A prospective study. *J Am Geriatr Soc* 2004; 52:1662-1667.
4. Libicher M, Appelt A, Berger I, Baier M, Meeder PJ, Grafe I, Dafonseca K, Noldge G, Kasperk C. The intravertebral vacuum phenomena as specific sign of osteonecrosis in vertebral compression fractures: Results from a radiological and histological study. *Eur Radiol* 2007; 17:2248-2252.
5. Matzaroglou C, Georgiou CS, Wilke HJ, Assimakopoulos K, Karageorgos A, Konstantinou D, Velissaris D, Panagiotopoulos E, Kafchitsas K. Kummell's disease: Is ischemic necrosis or vertebral "microcracking" the first step in the sequence? *Med Hypotheses* 2013; 80:505.
6. Sasaki Y, Aoki Y, Nakajima A, Shibata Y, Sonobe M, Takahashi K, Ohtori S, Nakagawa K. Delayed neurologic deficit due to foraminal stenosis following osteoporotic late collapse of a lumbar spine vertebral body. *Case Rep Orthop* 2013; 2013:682075.
7. Ito Y, Hasegawa Y, Toda K, Nakahara S. Pathogenesis and diagnosis of delayed vertebral collapse resulting from osteoporotic spinal fracture. *Spine J* 2002; 2:101-106.
8. Linn J, Birkenmaier C, Hoffmann RT, Reiser M, Baur-Melnyk A. The intravertebral cleft in acute osteoporotic fractures: Fluid in magnetic resonance imaging-vacuum in computed tomography? *Spine (Phila Pa 1976)* 2009; 34:E88-E93.
9. Kim YC, Kim YH, Ha KY. Pathomechanism of intravertebral clefts in osteoporotic compression fractures of the spine. *The spine journal* 2014; 14:659-666.
10. Malghem J, Maldague B, Labaisse MA, Dooms G, Duprez T, Devogelaer JP, Vande Berg B. Vertebral osteonecrosis: MR imaging findings and related changes on adjacent levels. *AJNR Am J Neuroradiol* 2007; 28:42-47.
11. Jang JS, Kim DY, Lee SH. Efficacy of percutaneous vertebroplasty in the treatment of intravertebral pseudarthrosis associated with noninfected avascular necrosis of the vertebral body. *Spine (Phila Pa 1976)* 2003; 28:1588-1592.
12. Becker S, Tuschel A, Chavanne A, Meissner J, Ogon M. Balloon kyphoplasty for vertebra plana with or without osteonecrosis. *J Orthop Surg (Hong Kong)* 2008; 16:14-19.
13. Yang H, Gan M, Zou J, Mei X, Shen X, Wang G, Chen L. Kyphoplasty for the treatment of Kummell's disease. *Orthopedics* 2010; 33:479.
14. Fang X, Yu F, Fu S, Song H. Intravertebral clefts in osteoporotic compression fractures of the spine: Incidence, characteristics, and therapeutic efficacy. *Int J Clin Exp Med* 2015; 8:16960-16968.
15. Zhang GQ, Gao YZ, Chen SL, Ding S, Gao K, Wang HQ. Comparison of percutaneous vertebroplasty and percutaneous kyphoplasty for the management of Kummell's disease: A retrospective study. *Indian J Orthop* 2015; 49:577-582.
16. Ha KY, Kim YH. Risk factors affecting progressive collapse of acute osteoporotic spinal fractures. *Osteoporos Int* 2013; 24:1207-1213.
17. Maldague BE, Noel HM, Malghem JJ. The intravertebral vacuum cleft: A sign of ischemic vertebral collapse. *Radiology* 1978; 129:23-29.
18. Ratcliffe JF. The arterial anatomy of the adult human lumbar vertebral body: A microarteriographic study. *J Anat* 1980; 131:57-79.
19. Fang X, Yu F, Fu S, Song H. The vacuum cleft sign: An uncommon radiological sign. *Osteoporos Int* 2005; 16:1210-1214.
20. Benedek TG, Nicholas JJ. Delayed traumatic vertebral body compression fracture; part II: Pathologic features. *Semin Arthritis Rheum* 1981; 10:271-277.
21. Dupuy DE, Palmer WE, Rosenthal DI. Vertebral fluid collection associated with vertebral collapse. *AJR Am J Roentgenol* 1996; 167:1535-1538.
22. Kim DY, Lee SH, Jang JS, Chung SK, Lee HY. Intravertebral vacuum phenomenon in osteoporotic compression fracture: Report of 67 cases with quantitative evaluation of intravertebral instability. *J Neurosurg* 2004; 100:24-31.
23. McKiernan F, Jensen R, Faciszewski T. The dynamic mobility of vertebral compression fractures. *J Bone Miner Res* 2003; 18:24-29.
24. McKiernan F, Faciszewski T. Intravertebral clefts in osteoporotic vertebral compression fractures. *Arthritis Rheum* 2003; 48:1414-1419.
25. Brown DB, Gilula LA, Sehgal M, Shimony JS. Treatment of chronic symptomatic vertebral compression fractures with percutaneous vertebroplasty. *AJR Am J Roentgenol* 2004; 182:319-322.
26. Li KC, Wong TU, Kung FC, Li A, Hsieh CH. Staging of Kummell's disease. *J Musculoskelet Res* 2004; 8:43-55.
27. Kim YY, Rhyu KW. Recompression of vertebral body after balloon kyphoplasty for osteoporotic vertebral compression fracture. *Eur Spine J* 2010; 19:1907-1912.
28. Kim MJ, Lindsey DP, Hannibal M, Alamin TF. Vertebroplasty versus kyphoplasty: Biomechanical behavior under repetitive loading conditions. *Spine.* 2006; 31:2079-2084.
29. Wilke HJ, Mehnert U, Claes LE, Bierschneider MM, Jaksche H, Boszczyk BM. Biomechanical evaluation of vertebroplasty and kyphoplasty with polymethylmethacrylate or calcium phosphate cement under cyclic loading. *Spine (Phila Pa 1976).* 2006; 31:2934-2941.
30. Adams MA, Pollintine P, Tobias JH, Wakley GK, Dolan P. Intervertebral disc degeneration can predispose to anterior vertebral fractures in the thoracolumbar spine. *J Bone Miner Res.* 2006; 21:1409-1416.