**Retrospective Evaluation** 

# Percutaneous Kyphoplasty for Kummell Disease with Severe Spinal Canal Stenosis

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Free full manuscript: www.painphysicianjournal.com **Background:** Percutaneous kyphoplasty (PKP) has been proven as an effective, minimally invasive procedure for the treatment of Kummell's disease in the early stages. However, a risk of cement leakage and further neurological damage remains during and after PKP, especially in chronic osteoporotic stage III Kummell's disease with severe spinal canal stenosis.

**Objective:** To evaluate the feasibility and efficacy of PKP for the treatment of chronic osteoporotic stage III Kummell's disease with severe spinal canal stenosis.

Study Design: A retrospective evaluation of postoperative radiographs.

Setting: Pain management clinic.

**Methods:** A retrospective study was performed on 9 patients with 11 levels managed with PKP for chronic osteoporotic stage III Kummell's disease with severe spinal canal stenosis. Clinical and radiological outcomes were assessed.

**Results:** Substantial pain relief was attained in all the patients. Both visual analogue scale (VAS) and Oswestry Disability Index (ODI) scores improved significantly from pre- to post-operation (P < 0.05), and remained unchanged at every follow-up. No neurological deterioration was found. Postoperatively, the anterior and midline vertebral body heights were significantly corrected (P < 0.05), and were sustained at the final follow-up. Similar results were seen in the correction of kyphotic angle. Neither cement leakage into the spinal canal nor further dislodging of the posterior vertebral fragments occurred. Two cases experienced subsequent fractures with one having a second PKP and the other being treated conservatively.

Limitations: Retrospective study of 9 cases with 11 levels due partly to the rarity of the disorder.

**Conclusions:** PKP is an effective, minimally invasive procedure for the treatment of chronic osteoporotic stage III Kummell's disease with severe spinal stenosis, leading to a significant relief of symptoms and improvement of functional status.

Institutional Review: This study was approved by the Institutional Review Board.

Key words: Kummell's disease, kyphoplasty, spine, osteoporosis, cement leakage, polymethylmethacrylate, neurological deficits, burst fractures

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ummell's disease, defined as delayed posttraumatic vertebral collapse, is a complication of vertebral compression fractures (VCF) (1-3). The disease was first publicly presented by Hermann

Kummell in 1895 (4). Since his original description, multiple terms have been used to describe this pathology: vertebral osteonecrosis, pseudoarthrosis of vertebral fracture, VCF nonunion, intravertebral vacuum cleft, and delayed vertebral collapse. Although the concept of Kümmell's disease is ambiguous, with the advancement of preoperative radiographic evaluations of vertebral status, in particular with the popularity of magnetic resonance imaging (MRI), more and more VCF are found to be Kummell's disease (5).

Based on the clinical symptoms, radiographs, and the MRI, the disease is divided into 3 stages as follows: Stage I minor compression on the plain radiographs with signs of osteonecrosis on the MRI, stage II vertebral body collapse with dynamic mobility, and stage III posterior cortex breakage leading to spinal canal stenosis or cord compression with or without neurological deficit (6,7). Traditionally, early stage of the disease without serious spinal canal compromise can be treated effectively by percutaneous vertebroplasty (PVP) or kyphoplasty (PKP) allowing earlier mobilization than in conventional open techniques. However, PVP has been reported to have more complications compared with PKP (8). The treatment for chronic osteoporotic stage III Kummell's disease remains controversial. In these patients with such severe osteoporosis, fixation surgery and body reconstruction have high complication rates (9-11). Treating stage III Kummell's disease can be problematic due to the high risk of cement leakage into the spinal canal with the potential for severe neurological damage. To our knowledge, there are few reports discussing PKP for chronic osteoporotic stage III Kummell's disease with severe spinal canal stenosis.

Here, we report on our experience of the treatment for chronic osteoporotic stage III Kummell's disease with severe spinal canal stenosis by PKP. The purpose

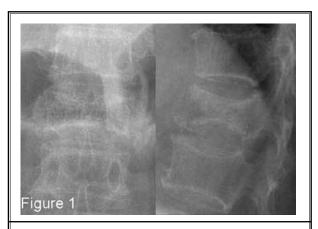


Fig. 1. Preoperative radiographs of the thoracolumbar spine demonstrate a moderately severe burst fracture of T12 with vacuum phenomenon.

of this study is to evaluate the feasibility and efficacy of PKP for the treatment of stage III Kummell's disease with severe spinal canal stenosis.

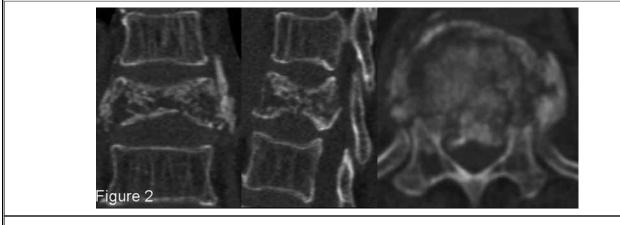
## METHODS

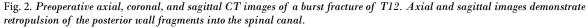
#### Patients

A total of 9 patients (11 levels) of stage III Kummell's disease with posterior wall collapse and severe spinal canal stenosis were treated with PKP from April 2005 to August 2013. Seven patients were from our institute. Two patients were from another hospital with surgery done by authors from our institute. Each patient had primary osteoporosis. The mean age of the patients was 74.8 years. Each patient had severe back pain refractory to conservative treatment with a mean duration of symptoms of 3.4 months. Furthermore, 6 patients experienced pain, numbness, and weakness of both legs. Radiographs demonstrated burst fracture with vacuum phenomenon for each patient (Fig. 1). Vertebral collapse and spinal canal stenosis were confirmed with computed tomography (CT) scan (Fig. 2). MRI demonstrated osteonecrosis with fluid signal and spinal cord compression (Fig. 3). The disease was diagnosed by comparing the result of the clinical examination with the radiologic findings. Patients with previous spine surgery, infection, or tumor were excluded. Each patient was diagnosed with osteoporosis using dualenergy x-ray absorptiometry (GE Lunar Prodigy, USA) prior to PKP. The mean T score of lumbar spine bone mineral density was -4.42. The painful levels treated by PKP were located in the thoracic and lumbar spine (Fig. 4). The possible use of PVP was discussed, but the technique was not utilized due to the concern of cement leakage. This study was approved by our Institutional Review Board.

#### Surgical Technique

The patient was informed of the possibility of paralysis and potential need for emergency surgery. A consent form was signed. PKP was performed in accordance with the procedure described by Yang et al (12). The operation was performed under general anesthesia with neurological status monitored throughout the procedure. A transpedicular approach was performed with trocar and cannula systems under biplanar fluoroscopic guidance. The balloons (Kyphon, Sunnyvale, CA, USA) were inserted through the cannulas and placed inside the anterior 3/4 of the vertebral body from a lateral view. The balloons were inflated slowly





to reduce the risk of fracture and to create a cavity for the injected cement. Polymethylmethacrylate (PMMA) cement was injected into the cavity within the vertebral body when it became doughy and could stand at the tip of the bone cement inserter. The injection process was monitored continuously under fluoroscopic control in the lateral plane. The procedure was stopped immediately if high resistance was encountered or if PMMA neared the posterior wall of the vertebral body. The amount of cement was noted. The patients were kept at bed rest for one day after the procedure (Fig. 5).

#### **Clinical Assessment**

The visual analogue scale (VAS score 0 - 10; 0 no pain; 10 the worst imagined) system was employed to evaluate back pain control. Impact on the patient's daily life was assessed using the Oswestry Disability Index (ODI) questionnaire. Clinical examinations were performed prior to surgery, postoperatively (3 days after PKP), at 6 weeks and 12 weeks after operation, and at the last follow-up (within 16 – 96 months).

#### **Radiographic Assessment**

The height of the anterior and mid portion of the necrotic vertebra body along with the kyphotic angle was measured using a standing lateral radiograph. The normal height of the necrotic vertebra was estimated from the mean of the measurements from the closest normal vertebral body cephalad and caudad to the treated level. The height of the necrotic vertebra presented with the vertebral body height ratio calculated as follows: height ratio (%) = (compromised vertebral height/mean adjacent control vertebral height) × 100.

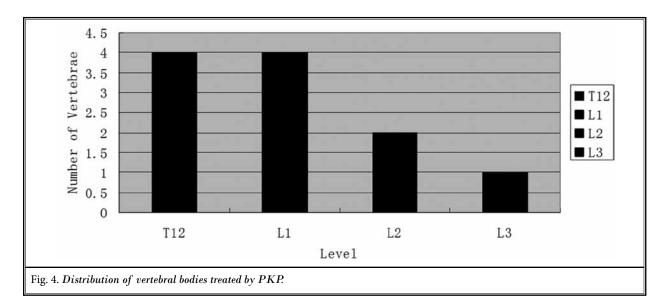


Fig. 3. Preoperative sagittal T2 weighted MRI of the thoracolumber spine demonstrates fluid within the anterior intravertebral cleft of the T12 vertebral body. Low signal marrow posteriorly suggests a fibrous or sclerotic component.

Cobb's methods were used to measure the kyphotic angle of the treated vertebrae. Cement leakage was observed on postoperative CT scans (Fig. 6).

#### RESULTS

The patients tolerated the procedure well. The operation time and hospitalization were short. The



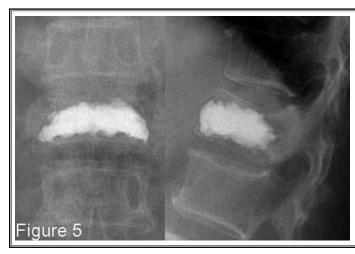


Fig. 5. Postoperative AP and lateral radiographs of the thoracolumbar spine demonstrate high-density bone cement within the anterior 2/3 of the vertebral body post PKP with improved vertebral body height.

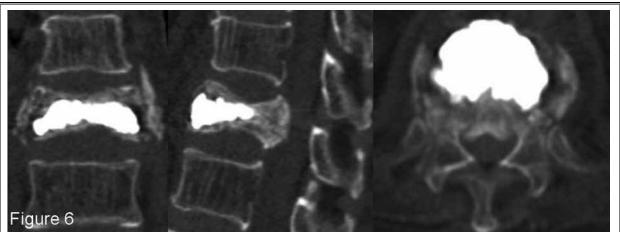


Fig. 6. Postoperative axial, coronal, and sagittal CT images of the T12 vertebral body demonstrate cement filling the anterior 2/3 of the vertebral body without cement extravasation and without further retropusion of the posterior wall fragments. Improvement in vertebral body height is not as apparent on these single slice images.

mean operative time per vertebral body level was 29.5 minutes. The mean amount of cement used per application was 4.5 mL. The mean hospitalization interval was 5.2 days. The mean follow-up period was 43.8 months. The treated level was T12 in 4 patients, L1 in 4 patients, L2 in 2 patients, and L3 in one patient. Seven patients had only single vertebral body involvement.

The mean preoperative VAS score was 8.6  $\pm$  0.5 (range 8 - 9). Substantial pain relief was achieved with each patient. The mean postoperative VAS score reduced to  $1.6 \pm 0.7$  (range 1 - 3) 3 days after the procedure. The effect of pain control was persistent. The average VAS changed slightly at every follow-up (Table 1). Similar results were seen for the ODI scores. The ODI scores decreased from 85.2 ± 3.5 before surgery to 24.5  $\pm$  4.6 3 days after operation, 26.2  $\pm$  3.7 at 6 weeks, 25.7  $\pm$  3.4 at 12 weeks, and 28.0  $\pm$  3.8 at the last followup (Table 1). Statistically significant differences were seen between the preoperative evaluation and each postoperative follow-up assessment. Post-operatively, the patients no longer experienced their preoperative neurologic symptoms. No neurological deterioration was found.

The mean postoperative anterior and mid vertebral body heights were significantly increased compared with preoperative heights (Table 2). A statistically significant improvement in the mean values for kyphotic angle was found between the preoperative and postoperative assessment (Table 2). There were no significant differences between the immediate postoperative and follow-up assessments. The post-operative CT scan demonstrated one asymptomatic intradiscal cement leak. Neither cement leakage into spinal canal or further dislodging of the posterior vertebral body fragments occurred. Two cases experienced subsequent fractures. One patient chose PKP again, while the other patient chose conservative treatment.

## Discussion

Kummell's disease is defined as delayed post-traumatic vertebral collapse and often occurs in patients with osteoporosis, extensive spondylosis, advanced age, or those undergoing long-term steroid therapy. Single vertebral body involvement is most common, which was confirmed in our study. The fractured and necrotic vertebral body typically is located in the thoracic or lumbar spine with the T12 vertebral body being the most commonly affected (2). In this study, the disease occurred in 9 patients at 11 levels including T12 in 4 cases, L1 in 4 cases, L2 in 2 cases, and L3 in one case. These findings demonstrate that the thoracolumbar junction is most susceptible to delayed vertebral collapse.

The combination of disease progression and radiographic features is critical to diagnosis and prevention of this disease. Ito et al (6) postulated that delayed vertebral collapse began with an intravertebral cleft appearing anteriorly within the vertebral body after injury. The clefts were enlarged over time and led to collapse of the posterosuperior wall of the affected vertebra, resulting in spinal canal stenosis and subsequent

Table 1. Mean	improvement i	in	VAS	and	ODI.

	Pre-PKP	3 Days after PKP	6 Weeks after PKP	12 Weeks after PKP	at Last Follow-up	
VAS	$8.6 \pm 0.5$	$1.7 \pm 0.7^{*}$	$1.8 \pm 0.7^{*}$	$1.9 \pm 0.6^*$	$2.2 \pm 0.7^{\star}$	
ODI (%)	85.2 ± 3.5	$24.5\pm4.6^{*}$	$26.2 \pm 3.7^{*}$	$25.7 \pm 3.4^{*}$	$28.0\pm3.8^{*}$	

VAS indicates visual analogue scale.

ODI indicates Oswestry disability index.

PKP indicates percutaneous kyphoplasty.

\*P < 0.05 compared to preoperative value.

Table 2. Mean ratios of	f anterior,	middle vertebral	height	and kyphotic angle.
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	Pre-PKP	3 Days after PKP	6 Weeks after PKP	12 Weeks after PKP	at Last Follow-up
На	$49.3\pm24.8$	75.3 ± 15.8*	74.9 ± 15.7*	74.3 ±1 5.6*	73.8 ± 15.5*
Hm	$48.4 \pm 16.6$	69.6 ± 15.4*	68.7 ± 15.1*	$67.9 \pm 14.9^{*}$	$67.3 \pm 15.0^{*}$
Kyphotic angle (°)	19.7 ± 13.8	6.6 ± 2.7*	7.5 ± 3.0*	7.9 ± 3.4*	8.9 ± 3.9*

Ha indicates anterior vertebral height.

Hm indicates middle vertebral height.

PKP indicates percutaneous kyphoplasty.

\*P < 0.05 compared to preoperative value.

development of neurological deficits. Cleft is an important radiographic sign associated with Kummell's disease. As radiographic examinations become common, the ability to diagnose Kummell's disease will improve.

Of the available imaging techniques, radiographs are the least sensitive method for detecting vertebral body clefts. Multi-detector, spiral CT examination with coronal and sagittal reconstructed views better demonstrates the vertebral body fractures and cleft. To date, MRI is the most sensitive and specific method for detection of Kummell's disease by demonstrating soft tissue characteristics including marrow edema and fluid within the vertebral body cleft. Chen et al (5) reported that 40 cases in 44 patients with intravertebral clefts were detected by MRI, which was significantly higher than the detection rate of x-ray and CT scanning with reformatted imaging. With increasing numbers of spinal MRIs, Kummell's disease is being diagnosed more readily, especially in elderly patients (5,13). These patients usually suffer from progressive kyphosis, back pain, and occasionally paraparesis due to spinal cord and cauda equina damage. Nakamae et al (14) reported that the incidence of delayed neurologic deficit was 19%.

Treatment for Kummell's disease remains controversial. Although early reports were centered on conservative treatment such as bracing and bed rest, more recent reports favor surgical intervention. The advantages of surgery include earlier patient ambulation and correction of the kyphotic deformity. However, extensive surgical intervention in the elderly remains challenging because of medical comorbidities or fixation complications secondary to osteoporosis. Early disease (without serious spinal canal compromise) can be treated effectively by PVP or PKP, allowing mobilization earlier than that by conventional open techniques. Bear in mind, that PVP has been reported to have a higher risk of cement leakage than PKP (8).

As for Kummell's disease with severe spinal canal stenosis, the appropriate surgical treatment should be based on the pathology of the disturbance. The clefts of the disease in older patients usually present with more severe fractures and more significant collapse and instability. Nakamae et al (14) demonstrated that intravertebral instability was the main cause of the symptoms of Kummell's disease. In addition, intravertebral instability was considered to be the predominant cause of delayed neurologic deficit. Lee et al (15) also reported a catastrophic complete conus injury caused by dynamic instability of Kummell's disease. Sudo et al (16) and Verlaan (17) suggested that the main factor causing delayed neurological deficits following vertebral collapse in the osteoporotic spine was instability of the spinal column at the fracture site rather than mechanical compression of the spinal cord by bone fragments. The control of dynamic instability is critical in management of Kummell's disease with severe spinal stenosis (14,17). In our study, the dynamic nature of the treated vertebrae was effectively stabilized when the bone cement had been deposited into the clefts. This process also brought about significant relief of symptoms.

Complications of PKP include cement extravasation, fractured transverse processes or ribs, dural tears, infection, hematomas, and subsequent VCF (18,19). The most common complication is cement extravasation, typically, into epidural, foraminal, intradiscal, and paravertebral veins (20-23). Cement leakage into the spinal canal is a devastating complication resulting in spinal cord injury. In Kummell's disease with severe spinal canal stenosis, the posterior vertebral wall is always collapsed, which increases the potential risk of cement leakage into the spinal canal and further spinal canal compromise during PVP or PKP. Concerning the higher rate of cement leakage in PVP than in PKP, our study chose PKP and no obvious cement leakage and neurological deterioration were found. Careful attention must be paid while performing cement augmentation in the presence of cord compression: 1) The PKP cannula should be placed into the cleft located normally at the anterior 2/3 of the vertebral body. The partial repositioning of the vertebra may create more room in the vertebral body for containing the injected cement without further spinal canal compromise. As patients with Kummell's disease may have had an empty space surrounded by fibrosis tissues, the cement leakage may be reduced by those fibrosis tissues. 2) The dynamic C-arm x-ray monitoring should be performed during the entire bone cement filling process. The filling process has to be stopped immediately once the bone cement reaches the lateral margin or 1/4 of the distance from the posterior wall of the vertebral body. Enforced injection should be avoided as the increased injection pressure might lead to bone cement leakage. 3) Bone cement should be infused in the sticky stage or terminal sticky stage (24-28). In order to distribute the cement more viscously, Greene et al (29) designed an eggshell technique which included another balloon placement once the cement had been inserted. Their technique reported reduced risk of cement leakage. Georgy (30) also reported that use of

radiofrequency heated cement significantly reduced the risk of cement leakage.

## CONCLUSION

PKP is an effective, minimally invasive procedure for the treatment of chronic osteoporotic stage III Kummell's disease with severe spinal stenosis, leading to a significant relief of symptoms and improvement of functional status.

### Acknowledgment

This work was supported by the National Natural Science Foundation of China (81320108018 and 31270995), Jiangsu Provincial Special Program of Medical Science (BL 201204), Jiangsu Provincial Clinical Orthopedic Center, and the Priority Academic Program Development of Jiangsu Higher Education Institutions (PAPD).

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