Recollapse of previous vertebral compression fracture after percutaneous vertebroplasty

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Abstract
Summary This study was undertaken to investigate the incidence rate, characteristics, and predisposing factors associated with recollapse of the same vertebrae after percutaneous vertebroplasty (PVP). Recollapse of the same vertebra after PVP is one of the complications of the procedure, and the incidence rate in our study was 3.21%. The most important predisposing factor was pre-operative osteonecrosis. Recollapse was not related to trauma.

Introduction PVP using polymethylmethacrylate has become a popular treatment for osteoporotic vertebral compression fracture. Recollapse of the same vertebrae after PVP has rarely been reported. This study was undertaken to investigate the incidence, characteristics, and predisposing factors associated with recollapse of the same vertebrae after PVP.

Methods Eleven patients (seven females and four males; mean age, 69.91±5.49 years), out of a total of 343 patients, developed recollapse of the same vertebra after PVP. The 11 patients who developed recollapse comprised the “recollapse group”, while the remaining 332 patients comprised the “well-maintained group”.

Results Pre-operative magnetic resonance imaging revealed that the incidence of osteonecrosis was significantly higher in the recollapse group than the well-maintained group (p<0.05). The degree of re-expansion of the compressed vertebral body after PVP was significantly higher in the recollapse group than in the well-maintained group (p<0.05).

Conclusions The most important predisposing factor for recollapse was pre-operative osteonecrosis. Recollapse was not related to trauma. Osteoporotic vertebral compression fracture with osteonecrosis or pseudoarthrosis has been regarded as a relative indication for PVP; however, the findings of this study suggest that this disease category may be a relative contraindication for PVP.

Keywords Osteoporotic vertebral compression fracture · Percutaneous vertebroplasty · Recollapse

Introduction
Percutaneous vertebroplasty (PVP) is a minimally invasive procedure consisting of the injection of polymethylmethacrylate (PMMA) into a compressed osteoporotic vertebral body for pain relief and spinal stabilization [1–4]. This procedure leads to partial or complete pain reduction in 80%–90% of all cases, and pain relief is usually observed within 72 hours of injection [5]. In spite of these excellent clinical outcomes, there may be complications such as infection, cement embolism and various other complications resulting from cement extravasation [6, 7].

Recently, adjacent vertebral compression fracture related to previous PVP or kyphoplasty using PMMA cement was reported as a complication [8–11]. Once PMMA has polymerized following mixing of the powder and liquid components, it is strong enough to bear the physical load
of the spine [12, 13]. Some biomechanical studies have shown that cement augmentation places additional stress on adjacent levels [14–16]; however, recollapse or refracture of the same vertebral body after PVP has rarely been reported, and there is some controversy about the mechanism of this recollapse [17–19]. The purpose of this study was to assess the characteristics and risk factors of patients who experience recollapse of the same vertebra after PVP.

Clinical materials and methods

We treated 343 patients with symptomatic osteoporotic vertebral compression fractures by PVP with PMMA from January 2002 to December 2006. A total of 423 compressed vertebrae were treated with PVP, and there were 271 one-level compression fractures, 64 two-level fractures, and eight three-level fractures. Patients who had pathologic vertebral compression fractures from spinal metastatic cancer, osteolytic bone tumors, and hemangioma were excluded from this study. All patients underwent plain film, radionuclide bone scanning, and magnetic resonance imaging (MRI) to define the vertebral fracture and to exclude other causes of pain such as herniated intervertebral disc. Among the various studies, MRI was the most informative in screening for acute compression fracture, in which T1-weighted images showed low signal intensity because of bone edema. Patients whose fractures did not show low signal intensity on T1-weighted imaging and hot uptake on radionuclide bone scanning were excluded from the study. We performed a vertebroplasty in patients who complained of disabling back pain refractory to conservative therapies such as bed rest, analgesics, and external bracing.

All patients participated in follow-up care via an outpatient clinic at 1 month and 4 months after PVP for evaluation of operative results. At this time we prescribed osteoporosis medication, including bisphosphonates. After this period, all patients were transferred to osteoporosis specialists for specific osteoporosis therapy, including vitamin D, calcium and hormone replacement therapy. When patients complained of back pain after vertebroplasty, we performed radiologic studies to evaluate the presence of remote or adjacent fractures and other causes of pain such as recollapse.

Recollapse of the post-PVP vertebrae was confirmed using serial follow-up plain radiographs. Among the 423 treated compressed vertebrae, we discovered 11 vertebrae displaying recollapse after PVP (Table 1), producing an incidence rate of 3.21% out of 343 patients. There were 19 vertebral compression fractures among the 11 patients, and all of the fractured vertebrae were treated by PVP. The 11 patients who developed recollapse were assigned to the

| Case Age (yrs) Time interval after vertebral compression fracture (week) Trauma Symptom History At re-collapse vertebroplasty level | Bone density (T-score) | Compliance of osteoporosis medication | Recollapse of vertebral body | Osteonecrosis in recollapse level | Symptom in recollapse level | Other medical illness History | Compliance of osteoporosis medication after vertebroplasty |
|---|---|---|---|---|---|---|---|---|
| 1 | 73, F | 86 | No Back pain | L1 | 1.5 | BDM | Good | Good |
| 2 | 76, M | 12 | No Back pain | L1 | 1.2 | BDM | Poor | Good |
| 3 | 74, F | 6 | No Back pain | T12 | 2.4 | BDM | Good | Good |
| 4 | 77, F | 6 | No Back pain | T12 | 1.8 | BDM | Good | Good |
| 5 | 70, M | 6 | No Back pain | T9, T11, L1 | 4.6 | BDM | Good | Good |
| 6 | 63, F | 111 | No Back pain | L1 | 1.8 | BDM | Poor | Poor |
| 7 | 67, F | 6 | No Back pain | L1 | 1.8 | BDM | Poor | Poor |
| 8 | 67, M | 6 | No Back pain | L1 | 1.8 | BDM | Poor | Poor |
| 9 | 64, F | 111 | No Back pain | L1 | 1.8 | BDM | Poor | Poor |
| 10 | 64, F | 6 | No Back pain | L1 | 1.8 | BDM | Poor | Poor |
| 11 | 65, M | 40 | No Back pain | L1 | 1.8 | BDM | Poor | Poor |

*Chronic obstructive pulmonary disease
“recollapse group” and the 332 remaining patients who did not develop recollapse were assigned to the “well-maintained group”.

Risk factor assessment

We retrospectively reviewed pre-operative clinical parameters such as age, sex, bone mineral density, filler material (PMMA) volume, and chemotherapy history. Additionally, we reviewed trauma history, recollapse levels, other medical history, and compliance with osteoporosis medication in recollapse patients. We defined good medication compliance as maintaining osteoporosis medication and regular hospital visits from immediate post-vertebroplasty to the time of recollapse occurrence in the recollapse group. In addition, we also retrospectively reviewed pre-operative radiological parameters, such as the level of recollapse and the presence of osteonecrosis in the vertebral body, which was defined as the collection of intravertebral fluid or the presence of conjunction with air on MRI [20]. An MRI finding of intravertebral fluid was defined as an area of hypointensity on T1-weighted images and hyperintensity on T2-weighted images, and intravertebral air was defined as an area of hypointensity or a signal intensity void on T1 and T2-weighted images [20]. The incidence of osteonecrosis was compared between the recollapse group and the well-maintained group.

The anterior and posterior heights of the fractured vertebral body were assessed in order to calculate the compression ratio (anterior/posterior height) before and after PVP. All heights were measured using Picture Archiving and Communication System (PACS) and its computer software (PathSeed™Web for Centricity 2.0, General Electric Medical Systems, Milwaukee, WI, USA). The degree of re-expansion, which is the compression ratio difference between post-PVP and pre-PVP, was calculated for all patients and compared between the recollapse group and the well-maintained group.

Statistical analysis was performed using Fisher’s exact test, a paired T-test and an independent sample T-test. P<0.05 was considered statistically significant. SPSS 13.0 for Windows (SPSS, Chicago, IL, USA) was used for statistical analysis.

Results

The mean age of the well-maintained group (78 males, 254 females) was 68.00±9.72 year and their mean T-score of bone mineral density was -3.14±0.57. The mean age of the recollapse group, which included seven females and four males, was 69.91±5.49 year and the mean T-score of bone mineral density in this group was -3.59±0.78 (Table 1 and 2). There was no statistical significance in age, sex ratio, and T-score of bone mineral density between the 11 patients in the recollapse group and the 332 patients in the well-maintained group (p>0.05, Table 2).

Incidents of recollapse after PVP were located mainly in the thoracolumbar junction (T11-L2): one in T11; four in T12; five in L1; and one in L2. The mean time interval between PVP and the occurrence of recollapse was 45.81±36.47 weeks (5–111 weeks). When recollapse occurred, there were subjective complaints of back pain, but there were no signs of spinal cord compromise. Characteristically, there was no history of trauma in the 11 patients at the time of recollapse (Table 1).

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>69.91±5.49</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>4/7</td>
</tr>
<tr>
<td>Bone mineral density (T-score)</td>
<td>-3.59±0.78</td>
</tr>
<tr>
<td>Filler material volume (ml)</td>
<td>4.27±1.40</td>
</tr>
<tr>
<td>Osteonecrosis *</td>
<td>6 of 11 patients</td>
</tr>
<tr>
<td>Compression ratio* (initial/after vertebroplasty)</td>
<td>0.601±0.171/0.741±0.165</td>
</tr>
<tr>
<td>Compression ratio difference *</td>
<td>0.140±0.153</td>
</tr>
<tr>
<td>Location of recollapse</td>
<td>Thoracolumbar junction</td>
</tr>
<tr>
<td>Alteration of filler material mass after recollapse</td>
<td>3 of 11 patients</td>
</tr>
<tr>
<td>Condensation and size reduction of PMMA</td>
<td>0 of 11 patients</td>
</tr>
<tr>
<td>Migration of PMMA into the spinal canal</td>
<td>0 of 11 patients</td>
</tr>
<tr>
<td>Poor compliance of osteoporosis medications after vertebroplasty</td>
<td>5 of 11 patients</td>
</tr>
</tbody>
</table>

*P<0.05
Risk factors for recollapse

In a retrospective review of pre-operative MRI, there were 21 compressed vertebrae with intravertebral fluid collection on T2-weighted sagittal images that were compatible with findings of osteonecrosis, and the incidence of osteonecrosis was 4.96% out of 423 vertebrae. Among the 21 compressed vertebrae with osteonecrosis, there were six cases of recollapse, and the incidence of recollapse was 28.57%. In contrast, among the 402 compressed vertebrae without osteonecrosis, there were only five cases of recollapse with an incidence of 1.24% (p<0.05, Table 3).

Thirteen out of 343 patients were administered steroid medication (3.79%). Among the 21 patient osteonecrosis group, three patients (14.29%) were administrated steroid medication. Of the 322 patients without osteonecrosis, 10 patients (3.11%) were administrated steroid medication (P>0.05). Sixteen out of 343 patients had malignant disorders and were treated by chemotherapy (4.66%). Three out of 21 patients with osteonecrosis had chemotherapy history (14.29%), and 13 out of 332 patients without osteonecrosis had chemotherapy history (3.92%) (P>0.05).

We were only able to obtain compliance information for osteoporosis medication in recollapse patients. Compliance data are unfortunately not available for the entire population. In recollapse patients, six patients had not been administrated any osteoporosis medications, three had intermittently been administrated medications, and two had been receiving regular medications when initial compression fracture was diagnosed. Six out of 11 recollapse patients maintained good compliance with osteoporosis medication after vertebroplasty (54.5%). Five out of 11 recollapse patients had poor compliance with osteoporosis medication after vertebroplasty (45.5%) (Table 1 and 2). In five patients in the recollapse group without osteonecrosis, two displayed steroid-induced osteoporosis due to ulcerative colitis and chronic obstructive pulmonary disease. One patient suffered from hypothyroidism and demonstrated poor compliance with osteoporosis medications, and two patients had severe osteoporosis with T-scores on BMD of -5.3 and -3.8 (Table 1).

Compressed vertebrae may be re-expanded with PVP using PMMA. In the recollapse group, the initial mean compression ratio was 0.601±0.171 and increased to 0.741±0.165 after vertebroplasty (p<0.05). In the well-maintained group, the initial mean compression ratio was 0.611±0.106 and increased to 0.648±0.095 after vertebroplasty (p<0.05, Table 2). The mean ratio differences of compressed vertebrae were 0.140±0.153 in the recollapse group and 0.037±0.046 in the well-maintained group. The degree of re-expansion of the vertebral body after PVP was significantly higher in the recollapse group than the well-maintained group (p<0.05, Table 2). In the 21 compressed vertebrae with osteonecrosis, the initial mean compression ratio was 0.568±0.121 and increased to 0.670±0.122 after vertebroplasty (p<0.05). In the 402 compressed vertebrae without osteonecrosis, the initial mean compression ratio was 0.604±0.108 and increased to 0.639±0.099 after vertebroplasty (p<0.05, Table 3). The degree of re-expansion of the vertebral body after PVP was significantly higher in patients with osteonecrosis than in patients without osteonecrosis (p<0.05, Table 3).

The mean volume of injected PMMA cement was 4.27±1.40 mL in the recollapse group and 4.29±0.98 mL in the well-maintained group, revealing no statistically significant difference (P>0.05, Table 2). After recollapse had occurred, the filler material was morphologically altered in three cases (27.3%). Diffusely injected PMMA was condensed and reduced in size in the vertebral body (Fig. 1). However, when recollapse occurred, PMMA cement masses did not migrate into the spinal canal, and symptoms of neurological compromise did not develop in any of the recollapse cases (Table 2).

Discussion

Vertebroplasty using PMMA is widely accepted as a safe and effective treatment for focal back pain caused by osteoporotic vertebral compression fracture [1–4]. Adjacent vertebral compression fracture related to previous PVP or kyphoplasty using PMMA cement has been reported as a complication [8–11], and may be due to additional stress on the adjacent levels caused by the cement augmentation [14–16]. Recollapse or refracture of the same vertebral body after PVP has been reported only rarely [17–19]. Recollapse, namely further pathological fracturing of a treated vertebral body after PVP, is compounded by factors such as increased stress on the adjacent vertebrae and its own bony structure, and lack of vertebral body maintenance due to osteoporosis. Thus, it is important to understand the factors that contribute to recollapse.

Table 3 Relationship between osteonecrosis and re-expansion of vertebral body

<table>
<thead>
<tr>
<th></th>
<th>Compressed vertebrae with osteonecrosis</th>
<th>Compressed vertebrae without osteonecrosis</th>
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<tbody>
<tr>
<td>Total</td>
<td>21</td>
<td>402</td>
</tr>
<tr>
<td>No. of recollapse (%)</td>
<td>6 (28.57%)</td>
<td>5 (1.24%)</td>
</tr>
<tr>
<td>Initial compression ratio</td>
<td>0.568±0.121</td>
<td>0.604±0.108</td>
</tr>
<tr>
<td>Compression ratio after vertebroplasty</td>
<td>0.670±0.122</td>
<td>0.639±0.099</td>
</tr>
<tr>
<td>Compression ratio difference *</td>
<td>0.102±0.131</td>
<td>0.036±0.043</td>
</tr>
</tbody>
</table>

*P<0.05
vertebra, may result in the development of a more serious mode of fracture that compromises the spinal cord. In this retrospective study of recollapse after PVP, recollapse occurred in 11 cases out of 343 patients; an incidence rate of 3.21%.

For this study, 5 out of 11 recollapse patients had poor compliance with osteoporosis medication after vertebroplasty. And, 9 out of 11 recollapse patients had not been on regular osteoporosis medications before the initial compression fracture occurred. We hypothesized that bisphosphonate therapy was important for the prevention of recollapse and new fractures. We therefore emphasized the importance of osteoporosis medications to our patients. In Korea, because of the strict criteria of the national health insurance system, it is not possible for osteoporosis patients to continuously receive bisphosphonate treatment.

The causes of recollapse may be explained by two factors, namely, the vertebral factor and the filler material factor. The results of this study suggest that the vertebral factor, or the pre-operative status of compressed vertebrae, was more important than the filler material factor.

With regard to the vertebral factor, osteonecrosis of the involved vertebrae was the most important predisposing factor for recollapse (Fig. 2). Post-traumatic osteonecrosis of a vertebral body occurring in a delayed fashion first described by Kummell in 1895 [20, 21]. The pathophysiology of post-traumatic osteonecrosis is not completely understood and a variety of theories have been proposed to explain this phenomenon. The anterior one-third of the vertebral body may represent a “watershed” zone due to the characteristics of the blood supply. Fractures in this area may lead to vascular disruption of medullary arterioles leading to avascular osteonecrosis [22]. Osteoporotic bone may be more susceptible to the development of post-traumatic osteonecrosis due to pre-existing microfractures [21]. Characteristic MRI finding of osteonecrotic vertebral fractures is the collection of intravertebral fluid or presence of conjunction with air [20]. In this study, there were 21
compressed vertebrae with osteonecrosis, and the incidence rate was 4.96% out of a total of 423 vertebrae. On T2-weighted sagittal MRI, we found intravertebral fluid collection. The incidence rate of vertebra recollapse was significantly higher in patients with osteonecrosis than in patients without osteonecrosis (28.57% vs. 1.24%). We hypothesize that the patterns of preoperative vertebral venography may be related to osteonecrosis. In our experience, a cystic filling pattern of contrast media, rather than an interdigitation filling pattern, is frequently seen in patients with osteonecrosis (Fig. 3). Therefore, we reasoned that patients who demonstrate the cystic filling pattern need to be closely followed-up and should be evaluated for recollapse as well as maintain strict bisphosphonate therapy after PVP.

With this type of osteonecrosis, the degree of compression may progress with time. Based on our observations, we feel that the timing of PVP is of great importance. If PVP were to be performed during the early phase of osteonecrosis, the vertebral body would continue to collapse, because osteonecrosis of the involved vertebrae would progress, which would eventually weaken the structural stiffness of the vertebral body and result in the collapse of the remainder of the vertebral body. In this study, recollapse occurred mainly at the PMMA-unsupported portion of the vertebral body. There have been reports about the effectiveness of PVP in patients with Kummell osteonecrosis in which there may be re-expansion of compressed vertebrae after PVP [23, 24]. When PMMA is injected into compressed vertebrae with osteonecrosis, a solid lump, rather than a contiguous bone interdigitation, is usually produced (Fig. 2). This can lead to re-expansion of the compressed vertebrae by volume effect. In this study, the re-expansion rate of vertebral bodies was significantly higher in recollapse patients than in well-maintained patients. We assume that this type of volume-pressure effect produced by a solid lump rather than contiguous bone interdigitation might aggravate the process of osteonecrosis. Re-expansion of a vertebral body by PVP may not always be beneficial. Thus, over-expansion of a vertebral body after PVP may be a predisposing factor for recollapse.

Another vertebral factor that needs to be considered is the re-expansion effect by PVP itself. In this study, the re-expansion effect by PVP was significantly higher in patients with osteonecrosis than in patients without osteonecrosis. However, the results from our study led us to conclude that not all patients showing re-expansion after PVP have a high risk for developing recollapse. Even in patients without osteonecrosis, re-expansion is closely related to symptom onset duration [5]. When symptom onset duration is less than 8 weeks, re-expansion may be higher than in chronic cases and in patients whose symptom onset duration is greater than 8 weeks [5]. Re-expansion is closely related to osteonecrosis of compressed vertebrae [5]. In other words, re-expansion itself is not a high risk factor for the development of recollapse and, in some cases, osteonecrosis is beneficial for sagittal deformity correction by the re-expansion effect. In the natural course of osteoporotic vertebral compression fracture, there may be physiologic restabilization by bony union, spur formation, and ligament hypertrophy. In some patients, there may be a failure of restabilization, resulting in pseudoarthrosis and instability. Several articles have reported that PVP is effective for the treatment of intravertebral vacuum phenomenon, osteonecrosis, or pseudoarthrosis in osteoporotic vertebral compression fractures [23, 25, 26]; however, with the wide range of possibilities for the natural course of the disease, it seems likely that osteonecrosis may have adverse effects such as pseudoarthrosis and instability, and may be a high-risk factor for the development of recollapse after PVP.

As for the filler material factor, PMMA itself could induce bony structural weakness and osteonecrosis, and as such, recollapse may occur in the same vertebral body after PVP. Aseptic loosening is one of the major complications in cemented joint reconstruction, which is a multifactorial phenomenon involving interfacial failure, bond failure, bone remodeling, and cement failure [27]. The most commonly discussed cause of this phenomenon is thermal necrosis. In the histologic sections of the PMMA augmented group, bone necrosis was found mainly at the periphery of the PMMA cement, probably because of the exothermic reaction during polymerization [28–31]. Another filler material consideration that should be taken into account is mismatch of elastic modulus, stiffness, and mechanical strength at the cement-bone interface [27]. The interposition

![Preoperative venography showing a cystic filling pattern of contrast media in the compressed vertebral body](image-url)
of fibrous tissues between prosthesis and bone causes excessive stress concentrations at the cement-bone interface and micro-motion between these materials [27]. PMMA is not biodegradable, and as such cannot be replaced by new bone formation, resulting in the outer fibrotic wall becoming a radiolucent zone associated with future loosening. The above mentioned adverse effects of PMMA, namely thermal necrosis and outer fibrotic wall formation, the latter which is more severe as a solid lump than as a contiguous bone interdigitation, may aggravate the process of osteonecrosis and may result in recollapse after PVP.

As a result of our findings in this study, we felt that there was a need for close follow-up of patients with risk factors for recollapse. Therefore, we informed osteoporosis specialists of our results when patients were transferred to these specialists.

Conclusions

Recollapse, which is further pathological fracturing of a treated vertebra, may result in the development of a more serious mode of fracture involving canal compromise. The incidence rate of recollapse in this study after PVP was 3.21%. The most important predisposing factor was preoperative osteonecrosis. Over-expansion of the vertebral body after PVP may be another predisposing factor for recollapse.

The biomechanical characteristics of PMMA and morphology of the injected PMMA may be other causes of recollapse. In our series, fortunately, there were no neurologically compromised patients after recollapse or refracturing of the treated vertebrae. Osteoporotic vertebral compression fracture with osteonecrosis, which manifests as an intravertebral vacuum phenomenon, intravertebral fluid collection, or pseudoarthrosis, has been regarded as a relative indication for PVP; however, the findings of this study suggest that this disease category may be a relative contraindication for PVP.

Conflicts of interest None.

References