ORIGINAL ARTICLE

Analysis of intradiscal cement leakage during percutaneous vertebroplasty: Multivariate study of risk factors emphasizing preoperative MR findings

Suk-Joo Honga, Seunghun Leeb,*, Joon Shik Yoonc, Ju Han Kimd, Youn-Kwan Parkd

aDepartment of Radiology, Korea University Guro Hospital, 152-703 Seoul, Republic of Korea
bDepartment of Radiology, Hanyang University Hospital, 17 Haengdang-dong, Sungdong-gu, 133-792 Seoul, Republic of Korea
cDepartment of Rehabilitation Medicine, Korea University Guro Hospital, 152-703 Seoul, Republic of Korea
dDepartment of Neurosurgery, Korea University Guro Hospital, 152-703 Seoul, Republic of Korea

Available online 27 September 2013

KEYWORDS
Spine; Vertebroplasty; Bone cement; MRI

Summary

Objective: Previous reports have shown that intradiscal cement leakage during percutaneous vertebroplasty (PVP) is related to several risk factors. The purpose of this study was to evaluate preoperative MRI scans for such risk factors.
Methods: The study retrospectively analyzed 136 patients (aged 43–93 years; 234 vertebral bodies) with osteoporotic compression fractures. All patients underwent both MRI and PVP. There were 28 men (20.59%) and 108 women (79.41%). Age, gender, bone mineral density (BMD) score, endplate cortical disruption, abnormal T2-weighted hyperintensity in adjacent discs, presence of Kümmell’s disease, linear body fracture with extension to endplate, level of treated vertebral body and injected cement volume were considered risk factors for intradiscal cement leakage.
Results: Of the 234 vertebral bodies, 55 bodies from 42 patients with no endplate cortical disruption showed no adjacent intradiscal cement leakage. Of 179 bodies from 95 patients with endplate cortical disruption, 54 (30.17%) showed intradiscal cement leakage. Of the other possible risk factors, abnormal T2 hyperintensity in adjacent discs was significantly related to intradiscal cement leakage ($P=0.016$). The other possible factors (age, gender, BMD score, Kümmell’s disease, linear body fracture extending to the endplate, level of treated vertebral body and injected cement volume) were not related to intradiscal cement leakage.

* Corresponding author. Tel.: +82 2 2290 9163; fax: +82 2 2293 2111.
E-mail addresses: radsh@hanyang.ac.kr, radsh@medimail.co.kr (S. Lee).

0150-9861/$ – see front matter © 2013 Elsevier Masson SAS. All rights reserved.
http://dx.doi.org/10.1016/j.neurad.2013.07.004
Introduction

Percutaneous vertebroplasty (PVP), introduced by Galibert et al. [1] more than a quarter of a century ago, has since been used to treat patients with osteoporotic vertebral compression fractures, vertebral metastatic cancer [2,3], myeloma [4] and hemangioma [1], all of which cause patients severe pain. A success rate of 90–95% has been claimed for the management of osteoporotic vertebral compression fractures with the treatment [5,6]. PVP has also been used in cases of Kümmell’s disease, where an intravertebral fluid and/or air cavity is found in the presence of osteoporotic compression fracture [7].

However, several complications associated with PVP have been reported in the treatment of osteoporotic compression fractures, including compression fractures in the adjacent vertebrae, chemical or thermal injury to neural structures, nerve compression, pulmonary embolism and leakage into paraspinal soft tissue. Of these complications, intradiscal cement leakage has been reported to be a risk factor for adjacent vertebral compression fractures [8,9]. In previous studies, other risk factors were also reported for intradiscal cement leakage. Mirovsky et al. [10] suggested that cement extravasation into the disc space was always found to occur through a fractured endplate or a vacuum cleft. Hiwataki et al. [11] reported that a cortical endplate defect and an increased T2-weighted signal intensity in the adjacent disc were related to cement leakage into the adjacent disc space during vertebroplasty. However, there are different results and various opinions concerning the possible preoperative risk factors for intradiscal cement leakage [11,12]. In our present study, several clinical and preoperative magnetic resonance imaging (MRI) risk factors were analyzed in relation to intradiscal cement leakage.

Material and methods

Case selection

Institutional review board approval was obtained for this retrospective study (IRB No. GR09145-001), whereas informed consent from patients was not required. There was also no financial relationship between the investigators and study subjects.

The study retrospectively analyzed 136 consecutive patients [28 men (20.59%), 108 women (79.41%); mean age: 71.06 years; age range: 43–93 years] with vertebral body compression fractures caused by osteoporosis (234 vertebral bodies, involving one cervical spine, 114 thoracic spines and 119 lumbar spines). Specifically, the locations and numbers of treated vertebral bodies were: C4 (n = 1); T3 (n = 1); T4 (n = 1); T5 (n = 6); T6 (n = 6); T7 (n = 14); T8 (n = 14); T9 (n = 12); T10 (n = 8); T11 (n = 16); T12 (n = 36); L1 (n = 47); L2 (n = 27); L3 (n = 15); L4 (n = 18); and L5 (n = 12). All patients underwent preoperative MRI and PVP at a single institution between January 2003 and February 2009. The indication for PVP was compression fracture causing intractable back pain not relieved by conservative treatment. In 173 cases (from 95 patients) before the vertebroplasty procedure, bone mineral density (BMD) of the lumbar spine and femoral neck was measured by bone densitometry (Discovery A, Hologic Inc., Bedford, MA, USA).

Vertebroplasty procedures

In all patients, intravenous 50-mg pethidine hydrochloride (Jeil Pharmaceutical Co., Seoul, South Korea) was administered prior to PVP to provide analgesia. PVP was carried out in single-plane angiography units (Multistar T, Siemens Medical Solutions, Erlangen, Germany) prior to 2004 and in biplane angiography units (Axiom Artis dTA; Siemens Medical Solutions) from 2004 onwards. Patients were asked to lie in a prone position, and PVP was performed through a unilateral or bilateral transpedicular or costovertebral approach, using an 11- or 13-gauge beveled bone-biopsy needle (‘J’-type bone-marrow needle, Manan Medical Products, Wheeling, IL, USA). Under strict sterile conditions, the overlying skin was cleansed and draped. For local anesthesia, 1% lidocaine was injected into the skin, hypodermis and periosteum of the vertebral pedicles. After a small skin incision was made, the bone-biopsy needle was advanced until its tip touched the pedicle. The needle was then further advanced through the center of the pedicle and, subsequently, into the anterior third and inferior third of the vertebral body under fluoroscopy guidance. Once the needles had been placed in the vertebral body, powder—liquid polymethyl methacrylate (PMMA) bone cement (Exolinte Spine, Elmdown Limited, London, UK) was mixed to a toothpaste-like consistency. This mixture was backfilled into a 10-mL syringe and then transferred into 1-mL syringes. In addition, 1-cc syringes were used instead of an injection device or bone filler to achieve fine control of injection speed, stop-motion injection, and simultaneous filling of both the fracture cleft and surrounding bone-marrow, all of which are possible using 1-cc syringes and low-viscosity cement. Careful manual injection of PMMA using a 1-ml syringe was monitored by fluoroscopy and stopped when the PMMA reached the posterior 25% of the vertebral body. If leakages were observed, the injection was stopped immediately. This manual injection procedure generally took around 1 min. If further leakages...
were seen, the cement injection was halted completely. Following the injection, a stylet was inserted into the bone-biopsy needle, and the needle was then pulled into the posterior vertebral body. After about 5 min, the needle was removed under fluoroscopy monitoring. Any cement leakage beyond the endplate and into the disc was then looked for on the procedural and immediately postoperative lateral plain radiographs. Levels of the treated vertebrae were categorized as cervical (n = 1), T3 through T9 (n = 54), T10 through L2 (n = 134) and L3 through L5 (n = 45). The volume of injected PMMA cement was recorded for each patient. Following PVP, the patient remained supine in bed for at least 4h.

**Imaging technique**

All MRI scans were performed prior to PVP using a 1.5-T scanner (Sonata; Siemens). Axial and sagittal turbo spin-echo (TSE) T2-weighted images (TR/TE: 5100/100) and TSE T1-weighted images (TR/TE: 1180/12–15) were obtained, using a surface coil array in all patients. Typical imaging parameters were: field of view: 290 × 290 mm; matrix size: 320–512 × 210–370; slice thickness: 3 mm; intersection gap: 3.6 mm; and echo train length: 1–4.

**Image assessment**

All MRI scans and fluoroscopy images were transferred to an INFINITT Healthcare (Seoul, South Korea) picture archiving and communication system (PACS). Patients’ age, gender and BMD scores were evaluated as possible clinical risk factors. Endplate cortical disruption, abnormal T2 hyperintensity in adjacent discs, Kümmell’s disease with an intravertebral air and/or fluid cavity, linear body fracture with extension to endplate and injected cement volume were also evaluated as possible preoperative risk factors for intradiscal cement leakage. Two musculoskeletal radiologists (S. Lee, S.J. Hong) reviewed all patients’ MRI scans and fluoroscopy images in consensus. Also, one resident retrospectively reviewed all patients’ medical records for age, gender, BMD score and injected cement volume.

Endplate cortical disruption was defined as a break in the cortical endplate as seen on sagittal T1- and T2-weighted images (Fig. 1). Abnormal T2 hyperintensity in adjacent discs was defined as higher signal intensity in the adjacent disc compared with other either normal-looking or degenerated discs (Fig. 2). Kümmell’s disease was identified as the presence of either an intravertebral fluid cavity, an intravertebral air-fluid cavity or an intravertebral air cavity (Figs. 3–5). In addition, a linear body fracture extending to the endplate was identified as a linear abnormal T1/T2 hypointensity in the vertebral body contiguous with the endplate (Fig. 6). Cement leakage into the adjacent intervertebral disc spaces was defined as any cement present in the disc space beyond the cortical margin seen on fluoroscopy during the PVP procedure (Fig. 7).

![Figure 1](image1.png) **Figure 1** Sagittal T1-weighted MRI of a 66-year-old woman before vertebroplasty shows compression fracture of L1 and L2 vertebral bodies. There is disruption (white arrow) in the superior endplate of L1.

**Statistical analysis**

Using generalized linear mixed models, the following multiple covariates were analyzed to determine whether or not they were associated with cement leakage: age; gender; BMD score; endplate cortical disruption; abnormal T2 hyperintensity in adjacent discs; Kümmell’s disease; linear body fracture with extension to endplate; level of treated vertebral body; and injected cement volume. SAS version 9.1 software (SAS Institute Inc., Cary, NC, USA) was used for all statistical analyses, and a p value < 0.05 was considered statistically significant.

![Figure 2](image2.png) **Figure 2** T2-weighted sagittal MRI of a 67-year-old woman with T12 compression fracture before vertebroplasty shows high signal intensity in the intervertebral disc (white arrow) adjacent to the fractured T12 body. Other intervertebral discs show diffuse low signal intensity related to degeneration.
Results

Of the 234 vertebral bodies from our 136 patients, 55 from 42 patients with no endplate cortical disruption also showed no adjacent intradiscal cement leakage. Of these 55 vertebral bodies, only nine cases showed high signal intensity T2-weighted changes in adjacent discs (16.36%). In contrast, of the 179 vertebral bodies from 95 patients with endplate cortical disruption, there were 54 (30.17%) cases with intradiscal cement leakage. However, of these 179 cases, six had no preoperative BMD scores or injection cement volume in their records, so only 173 cases with endplate disruption were included in the analysis. The mean time interval between fracture and PVP was 73.19 days (median: 30 days, range: 2–1095 days). In most of our cases (99.3%), PVP was performed within a year.

There were 151 cases with high signal intensities in adjacent discs on T2-weighted MRI out of 173 vertebral bodies (87.28%), 40 cases with linear body fractures extending to endplate (23.12%) and 109 cases free of Kümmell’s disease (63.01%). Of the 64 cases with Kümmell’s, 20 cases had intravertebral air cavities, 39 had intravertebral fluid cavities and five had intravertebral air-fluid cavities. There was a greater overall prevalence of intravertebral fluid cavities in cases with Kümmell’s disease.

On univariate analysis, two factors—high signal intensity in the adjacent disc on T2-weighted scans \(P=0.006\), odds ratio (OR): 4.39] and injected cement volume \(P<0.0001\),
between intradiscal cement leakage and adjacent vertebral body fractures. Several earlier studies also discussed the risk for cement leakages during PVP. Hiwatashi et al. [11] suggested that cortical defects in the endplate and increased T2 signal intensities in the adjacent discs may be predictors of cement leakage into the adjacent disc space during vertebroplasty. Koh et al. [12] reported that a vacuum or cystic areas in fractured vertebral bodies, as seen on MRI, were associated with a lower risk of cement leakage, while cortical disruption of vertebral endplates was associated with a higher risk of leaks into the disc space. Mirovsky et al. [10] reported that a vacuum cleft, which is the result of a vertebral fracture, was the reason for cement leakage, while Cotten et al. [4] reported that intradiscal cement leakages were associated with cortical fractures or osteolysis of endplates. Nakano et al. [14] found that the patient’s age, gender and BMD, and a short time interval between injury and vertebroplasty, could be associated with an increased risk of cement leakage.

Compared with previous studies, our present evaluation has demonstrated no intradiscal cement leakage without endplate cortical disruption. This finding supports the idea of the latter as a necessary factor for intradiscal cement leakage, as reported by Mirovsky et al. [10]. In addition, our present study has shown that high signal intensities in the adjacent disc on T2-weighted MRI are also an important risk factor for intradiscal cement leakage during PVP.

Collapse was significantly more severe when air, rather than fluid, was present [15]. Indeed, the sequential change from fluid to air may be an indication that the stage of vertebral osteonecrosis is relatively late when intravertebral air is present. In our present study, injection of cement into an intravertebral cavity during PVP was associated with leakages into the intervertebral disc surrounding the cavity in only 15 cases. This suggests that all three types of Kümmel’s disease (with intravertebral air cavities, fluid cavities and air-fluid cavities) were not significantly associated with leaks. In addition, the cause of cement leakages from vertebral bodies with clefts was primarily endplate damage, which can be severe in vertebral bodies with clefts, thereby allowing cement to leak into the intervertebral disc via the endplate damage.

### Discussion

Intradiscal cement leakage is usually asymptomatic, but may have long-term mechanical consequences for the adjacent vertebral bodies [12,13]. According to previous studies [8], there are two opposing views regarding the relationship

---

Table 1  Patients’ MRI findings and clinical records in the assessment of intradiscal cement leakages.

<table>
<thead>
<tr>
<th>Cement leakage</th>
<th>Odds ratio</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Univariate study</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endplate disruption&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Not measurable</td>
<td>Not measurable</td>
</tr>
<tr>
<td>Abnormal T2 hyperintensity in adjacent disc</td>
<td>4.39</td>
<td>0.0059</td>
</tr>
<tr>
<td>Kümmell’s disease</td>
<td>1.84</td>
<td>0.1217</td>
</tr>
<tr>
<td>Linear body fracture with extension to endplate</td>
<td>1.41</td>
<td>0.0829</td>
</tr>
<tr>
<td>Injected cement volume</td>
<td>0.70</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Level of treated vertebral body</td>
<td>1.54</td>
<td>0.1547</td>
</tr>
<tr>
<td>Bone mineral density (T score)</td>
<td>1.016</td>
<td>0.8409</td>
</tr>
<tr>
<td><strong>Multivariate study</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Injected cement volume</td>
<td>0.85</td>
<td>0.1258</td>
</tr>
<tr>
<td>Abnormal T2 hyperintensity in adjacent disc</td>
<td>2.34</td>
<td>0.0158</td>
</tr>
</tbody>
</table>

<sup>a</sup> Given no intradiscal cement leakage without endplate disruption, neither odds ratio nor P value could be calculated.
observations were based on fluoroscopy images with no computed tomography (CT) scans, as performing both CT and MRI is not our usual practice in clinical settings.

In conclusion, the present study has demonstrated no adjacent intradiscal cement leakage without endplate cortical disruption. Also, abnormal T2 hyperintensities in the adjacent discs may be related to intradiscal cement leakages, but only when there is also endplate disruption. This suggests a need to delay PVP under such conditions. There may also be a need for more careful cement injection to reduce the possibility of intradiscal cement leakages during PVP.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References


