Percutaneous vertebroplasty and balloon kyphoplasty are minimally invasive techniques that use acrylic cement for stabilization of compression fractures of the spinal column due to osteoporosis, malignancy, and trauma. Either PVP or BKP may be indicated when a trial of conservative therapy does not improve the patient’s symptoms or for intractable pain and a progressive, slow-developing deformity in specific vertebral body fractures without posterior vertebral wall incompetence. Both procedures involve injection of cement (for example, PMMA) into the affected vertebral body to provide immediate structural support. Balloon kyphoplasty is also intended to minimally correct kyphotic deformity through the use of a balloon tamp to distract vertebral endplates prior to cement injection. Both procedures are generally well tolerated and produce limited side effects. However, cement extravasation is not uncommon and has been reported in 31%–96% of PVP cases and 7%–25% of BKP cases. While generally asymptomatic, on rare occasions extravasation is responsible for severe complications. Cement leakage can result in severe neurological deficits or a cement embolism to the lungs and other organ systems.

**Neurological deficit due to cement extravasation following a vertebral augmentation procedure**

**Case report**


Rothman Institute, Thomas Jefferson University, Philadelphia, Pennsylvania

The authors endeavor to highlight the surgical management of severe neurological deficit resulting from cement leakage after percutaneous vertebroplasty and to systematically review the literature on the management of this complication.

A patient presented after a vertebroplasty procedure for traumatic injury. A CT scan showed polymethylmethacrylate leakage into the right foramina at T-11 and L-1 and associated central stenosis at L-1. He underwent decompression and fusion for removal of cement and stabilization of the fracture segment. In the authors’ systematic review, they searched Medline, Scopus, and Cochrane databases to determine the overall number of reported cases of neurological deficit after cement leakage, and they collected data on symptom onset, clinical presentation, surgical management, and outcome.

After surgery, despite neurological recovery postoperatively, the patient developed pneumonia and died 16 days after surgery. The literature review showed 21 cases of cement extravasation with neurological deficit. Ultimately, 15 patients had resolution of the postoperative deficit, 5 had limited change in neurological status, and 2 had no improvement.

Cement augmentation procedures are relatively safe, but certain precautions should be taken to avoid such complications including high-resolution biplanar fluoroscopy, considering the use of a local anesthetic, and controlling the location of cement spread in relationship to the posterior vertebral body. Immediate surgical intervention with removal of cement provides good results with complete recovery in most cases.

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**Key Words**
• vertebroplasty • kyphoplasty • decompression • neurological deficit

Abbreviations used in this paper: BKP = balloon kyphoplasty; PMMA = polymethylmethacrylate; PVP = percutaneous vertebroplasty.
The purpose of this article was to report a case of spinal cord compression resulting from PVP treatment for traumatic osteoporotic compression fractures. Additionally, we performed a qualitative systematic review of the literature to evaluate the nature of symptoms, clinical course, and various management options use after a neurological deficit develops due to cement leakage.

**Case Report**

This 84-year-old man presented with difficulty walking and progressive weakness in both lower extremities. He was tachypneic, with a respiratory rate of 24 bpm. On examination, he exhibited Grade 0/5 strength throughout his right lower extremity and between Grade 2/5 and 3/5 strength throughout his left lower extremity (numerical grades refer to the Medical Research Council scale [grade range 0–5]); he also had loss of sensation to light touch. Bowel and bladder tone were intact. He reported having undergone a vertebroplasty procedure after a fall he suffered while on vacation in Turkey the previous week. After returning home from his vacation, he was admitted to the hospital for the aforementioned examination and also received pain control and treatment for a urinary tract infection. A progressive neurological deficit prompted the patient’s transfer to a tertiary spine center, where CT scanning of the spine revealed compression deformities of the T-12 and L-1 vertebral bodies and confirmed evidence of a prior vertebroplasty. Extensive cement extravasation was present in the central canal and right foramina between T-11 and L-2 with severe canal stenosis and cord compression at L-1 (Fig. 1). Computed tomography scanning of the lung demonstrated multiple cement emboli in the pulmonary vasculature and bibasilar atelectasis with a small left pleural effusion (Fig. 2).

The patient underwent a laminectomy and facetectomies involving the T11–L2 levels and a right T12–L1 transpedicular decompression to relieve PMMA compression of the thecal sac. The exposed cement was carefully separated from the thecal sac using a nerve hook, Penfield dissector, and osteotome, and all exiting nerve roots were decompressed of all visible cement. A partial corpectomy of L-1 was performed to remove cement compressing the ventral thecal sac. After thorough decompression, posterior instrumentation was placed between T-10 and L-2 along with morselized autograft (Fig. 3). The patient's neurological examination status improved by 1 motor score in both extremities immediately after surgery, and he was admitted to the intensive care unit.

On postoperative Day 16 the patient demonstrated progressive difficulty breathing, and imaging suggested worsening of preoperative atelectasis. The patient was placed on bilevel positive airway pressure ventilation and aggressive pulmonary toilet was initiated. Two weeks later, the patient developed difficulty swallowing and a high fever leading to a diagnosis of pneumonia with sepsis; the patient died 16 days after surgery.

**Qualitative Systematic Review**

**Methods**

Our systematic review search strategy included the following electronic databases: MEDLINE, Scopus, and
Cement leak after vertebroplasty

Cochrane register of trials (1970 to June 2012). The following combination of key words were used: 1) “Vertebroplasty” [MeSH], 2) “Kyphoplasty” [MeSH], 3) “cement leak,” 4) “Cement extravasation,” 5) “Neurological deficit,” 6) “Radiculopathy,” 7) “Paraplegia,” and 8) “Compression.” Bibliographies from obtained articles and review articles were also hand searched for additional references. Systematic reviews of published studies were included to provide qualitative information regarding the prevalence of cement leakage and the reported incidence of neurological complications in the literature (Fig. 4). Case studies were analyzed for age, sex, primary procedure performed, indication, number of levels during the primary procedure, time after primary procedure when cement leakage was identified, neurological symptoms at presentation of complication, level of cement spread, location of cement spread, associated complications, surgical procedure performed, neurological recovery status, and associated events. Articles were excluded if they did not provide details about the nature of the neurological deficit, location of cement spread, surgical method of removal, or neurological examination at follow-up. Reports of neurological deficits after PVP/BKP thought to be related to causes other than cement extravasation were also excluded.

Results

A total of 15 articles were found in the literature reporting 21 individual cases of patients with neurological deficits following cement extravasation (Table 1). The majority of patients were female (18 cases [85.7%]), and the average age of all cases reported was 71 ± 12 (± SD). Osteoporotic compression fractures were the most common initial presentation (17 cases [80.1%]) followed by vertebral body malignancy (metastasis/myeloma in 4 cases [19%]). Nineteen patients underwent PVP and 2 underwent BKP. Twelve cases involved augmentation of a single vertebral body level and 9 involved multiple-level vertebral body augmentation. Following either PVP or BKP, 18 patients reported deteriorating neurological status immediately after the procedure and 2 reported symptom presentation at 1 and 3 days postprocedure. Eighteen patients reported direct extravasation or injection of cement into the neural canal (epidural, intradural, or foraminal spaces), 1 reported neural element compression due to leakage through the anterior and posterior epidural sinuses, 1 reported extraforaminal spread, and 1 reported leakage into the intervertebral disc causing disc herniation and neural element compression.

Fig. 2. Axial CT image of the chest showing PMMA emboli within a pulmonary arterial branch (arrow).

Fig. 3. Anteroposterior radiograph (A), axial CT scans of T-12 (B) and L-1 (C), and midsagittal CT image (D) of the lumbar spine after removal of epidural bone cement, decompression, and placement of instrumentation.
Surgical intervention was performed in 18 cases for removal of PMMA, while observation was used in other 3 patients. In one report, surgical intervention provided clinical benefit even when performed 4 months after symptom onset.29 Five of 18 patients who underwent surgical removal of the PMMA experienced limited change in neurological status immediately after the decompression surgery and at long-term follow-up. Two patients undergone multiple decompression procedures for cement removal. Of the 3 patients who were treated nonoperatively, 2 had no neurological improvement and 1 patient displayed neurological recovery after steroid treatment.

Discussion

A vast quantity of literature has been published on the efficacy of PVP/BKP in terms of pain relief, quality of life improvement, and a low associated rate of complications compared with nonoperative treatment for vertebral compression fractures.10,12,16,17,19,21 Balloon kyphoplasty has been credited with achieving better pain reduction, decreased cement leakage, and better deformity correction than PVP because a cavity is created by the bone tamp within the vertebral body before cement injection.4,32,34 Recently, 2 double-blind randomized controlled trials concluded that for an osteoporotic compression fracture, a PVP procedure is no more effective at pain reduction than a sham procedure.28 A BKP procedure, while considered to be more effective at pain reduction than a PVP, lacks support from high-quality published reports. Despite the aforementioned controversies, enthusiasm for these procedures has not diminished, and the indications have expanded. Consequently, such procedures are still common in current orthopedic practice.29 A grave complication of such procedures is cement extravasation resulting in neural element compression (Table 2). Our report illustrates a neurological complication of PVP in which cement leakage into the spinal canal resulted in paraplegia requiring surgical decompression.

During a PVP/BKP procedure, cement may leak into the spinal canal and neural foramina from various routes (for example, medial pedicular wall compromise, fractured posterior wall of vertebral body, endplate fractures, basivertebral foraminal spread, and leakage through the internal spinal venous plexus).21,26 Intradural spread may occur when durotomy is caused by the injection needle.11,42,46 Nerve and spinal cord injury is thought to result from direct mass effect—possibly a thermal effect of the settling cement and the pull of gravity from the tethering of nerve fibers (especially intradural spread) (Table 3).11,27,30,48 Symptomatology depends on the location of extravasated cement, the amount of extruded cement, and rate of cement injection.29 Cement leakage into the disc is fairly common and is reported to be associated with the development of adjacent fractures89 and, in some cases, disc herniation with radiculopathy.41

Cement leakage, if it is going to result in a neurological deficit, often causes immediate symptoms. However, Cosar et al.13 and Ross and Fineman80 both documented a case involving the delayed onset of neurological symptoms without a deficit in the immediate postoperative period. When a deficit becomes evident, surgical decompression should be performed immediately, taking into consideration age, comorbidities, and whether evidence of neurological element compression is present, particularly since 2 of the 3 reports of nonoperative management included in this review reported no improvement of symptoms on final follow-up4,29 and since the third report elected against surgery due to the extentiveness of cement spread and uncertain clinical benefit of incomplete decompression.11 Surgical decompression must be carefully considered given the advanced age and likelihood for substantial comorbidities in this patient population, as demonstrated by our case in which the patient developed a postoperative nosocomial infection. Delayed presentation for surgery should not be a criterion to defer surgical intervention, as Wu et al.50 reported neurological improvement even when surgery was performed 4 months after the development of symptoms.

Most patients included in the review were treated with a wide laminectomy to facilitate surgical decompression and cement especially in the lower thoracic and lumbar levels. Based on our anecdotal experience, we believe if the cement spread is minimal and is centrally located in the spinal canal, an anterior approach should be sufficient. This is especially true for cement spread in the upper thoracic spine due to a relatively small canal in these segments. Ratliff et al.39 successfully demonstrated cement removal from the upper thoracic spine via an anterior-only approach. In cases of circumferential
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<table>
<thead>
<tr>
<th>Authors &amp; Years</th>
<th>Age (yrs), Sex</th>
<th>Op Type</th>
<th>Indication</th>
<th>No. of Levels</th>
<th>Time of Symptom Presentation</th>
<th>Neurological Symptoms†</th>
<th>Cement Spread</th>
<th>Associated Complication</th>
<th>Time of Surgical Procedure(s)</th>
<th>Procedure Performed</th>
<th>Neurological Recovery Status†</th>
<th>Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lee et al., 2002</td>
<td>66, F</td>
<td>PVP</td>
<td>OCF</td>
<td>T-11, L1–2</td>
<td>immediate</td>
<td>complete deficit at T-11</td>
<td>T-11</td>
<td>AP epidural veins</td>
<td>immediate</td>
<td>T10–L2 lam</td>
<td>inconclusive</td>
<td>(died)</td>
</tr>
<tr>
<td>Ratliff et al., 2001</td>
<td>50, F</td>
<td>PVP</td>
<td>malig</td>
<td>T-1</td>
<td>immediate</td>
<td>lt arm motor &amp; sensory deficit</td>
<td>C8–T1</td>
<td>epidural, neuro-foram</td>
<td>immediate</td>
<td>ant decompress C8–T1 w/ Harms cage</td>
<td>complete rec</td>
<td>(6-mo FU)</td>
</tr>
<tr>
<td>Harrington, 2001</td>
<td>66, F</td>
<td>PVP</td>
<td>OCF</td>
<td>T10–11, L-1</td>
<td>immediate</td>
<td>lt &gt; rt leg weakness</td>
<td>T10–L2</td>
<td>epidural, foraminal</td>
<td>immediate</td>
<td>removal of spilled cement</td>
<td>no change (12-mo FU)</td>
<td>NA</td>
</tr>
<tr>
<td>Wenger &amp; Markwalder, 2002</td>
<td>80, F</td>
<td>PVP</td>
<td>OCF</td>
<td>T-12</td>
<td>immediate</td>
<td>aggravayed pre-existing paraparesis</td>
<td>T-12</td>
<td>epidural, basivertebral veins</td>
<td>immediate</td>
<td>NA</td>
<td>no change</td>
<td>NA</td>
</tr>
<tr>
<td>Shapiro et al., 2003</td>
<td>64, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L-2</td>
<td>immediate</td>
<td>back pain, lt sciatica (L2–4)</td>
<td>L-2</td>
<td>epidural, intradural cement in intrathecal space</td>
<td>12 hrs</td>
<td>L-2 lam, midline durotomy, L1–3 LMF</td>
<td>complete rec (12-wk FU)</td>
<td>needle tract cause of dural tear</td>
</tr>
<tr>
<td>Lopes &amp; Lopes, 2004</td>
<td>82, F</td>
<td>PVP</td>
<td>OCF</td>
<td>T-6</td>
<td>immediate</td>
<td>paraplegia</td>
<td>T5–6</td>
<td>epidural</td>
<td>7 hrs</td>
<td>T5–7 lam</td>
<td>complete rec</td>
<td>(1-mo FU)</td>
</tr>
<tr>
<td>Teng et al., 2006</td>
<td>54, F</td>
<td>PVP</td>
<td>malig</td>
<td>T-8, T-10, L-1</td>
<td>immediate</td>
<td>bilat weakness below T-6, urinary incontin</td>
<td>T7–L1 w/ hematoma</td>
<td>epidural, neuro-foram postural hypotension</td>
<td>1) immediate, 2) 5 days, 3) 62 days</td>
<td>T8–10 lam, T-6, T-7, L-1 lam, 3) lam of T-11, T-12, L-2, repeat 2nd op, T8–L2 neurolysis</td>
<td>paraplegia w/ bowel/urinary incontin (13-mo FU)</td>
<td>yellow dura mater (thermal effect of injury)</td>
</tr>
<tr>
<td>Teng et al., 2006</td>
<td>79, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L-2</td>
<td>immediate</td>
<td>bilat lower-limb paralys, urinary incontin, constipation</td>
<td>L-2</td>
<td>epidural, intradural</td>
<td>immediate</td>
<td>L-1, L-2 lam</td>
<td>no change (3-yr FU)</td>
<td>needle tract (low signal intensity T2)</td>
</tr>
</tbody>
</table>

(continued)
**TABLE 1: Reported cases of neurological deficits after cement extravasation** (continued)

<table>
<thead>
<tr>
<th>Authors &amp; Years</th>
<th>Age (yrs), Sex</th>
<th>Op Type</th>
<th>Indication</th>
<th>No. of Levels</th>
<th>Time of Symptom Presentation</th>
<th>Neurological Symptoms†</th>
<th>Level</th>
<th>Location</th>
<th>Associated Complication</th>
<th>Time of Surgical Procedure(s)</th>
<th>Procedure Performed</th>
<th>Neurological Recovery Status†</th>
<th>Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Teng et al., 2006</td>
<td>68, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L-1</td>
<td>immediate</td>
<td>bilateral lower-limb weakness, bowel/urinary incontinence</td>
<td>T12–L2</td>
<td>epidural, neuroforam</td>
<td>NA</td>
<td>1) immediate, 2) 30 days, 3) 88 days</td>
<td>1) T12–L1 laminotomy, 2) repeat of 1st op, 3) T11–L1 laminotomy, T12–L2 neurolysis</td>
<td>little change (4.75-yr FU)</td>
<td>arachnoid fibrosis (thermal effect)</td>
</tr>
<tr>
<td>Chen et al., 2006</td>
<td>90, F</td>
<td>PVP</td>
<td>OCF</td>
<td>T12–L1</td>
<td>immediate</td>
<td>Grade 2 weakness in both legs</td>
<td>T7–L4</td>
<td>epidural, intradural</td>
<td>postoperative ileus</td>
<td>NA</td>
<td>none</td>
<td>no change (4-mo FU)</td>
<td>intradural spread can occur up &amp; down canal</td>
</tr>
<tr>
<td>Wu et al., 2007</td>
<td>66, M</td>
<td>PVP</td>
<td>OCF</td>
<td>T12–L3; 10 mos later T7–10</td>
<td>immediate</td>
<td>bilateral lower-limb weakness, paresthesias</td>
<td>T7–8</td>
<td>epidural, neuroforam</td>
<td>paravertebral vessel leakage</td>
<td>4 mos</td>
<td>lam of T6–8, rt T6–8 facetectomy, rt T-7 pedicle substract w/hardware</td>
<td>motor &amp; sensory recovery (2-yr FU)</td>
<td>delayed op mgmt can still be effective</td>
</tr>
<tr>
<td>Sabuncuoglu et al., 2008</td>
<td>49, M</td>
<td>PVP</td>
<td>malign</td>
<td>T-12</td>
<td>immediate</td>
<td>right limb weakness (Grade 1/5)</td>
<td>T12–L1</td>
<td>epidural, intradural</td>
<td>NA</td>
<td>immediate</td>
<td>T12–L1 laminotomy</td>
<td>right leg improvement (Grade 4/5) (3-mo FU)</td>
<td>urgent decompression to reduce thermal injury</td>
</tr>
<tr>
<td>Cosar et al., 2009</td>
<td>79, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L-5</td>
<td>immediate</td>
<td>bilateral Grade 2/5 dorsiflexion in feet</td>
<td>L-4, L-5</td>
<td>subdural</td>
<td>NA</td>
<td>immediate</td>
<td>L-5 laminotomy</td>
<td>complete recovery, 5/5 motor force</td>
<td>NA</td>
</tr>
<tr>
<td>Cosar et al., 2009</td>
<td>72, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L-1, L-4</td>
<td>immediate</td>
<td>right lower-limb weakness</td>
<td>L-4, L-5</td>
<td>neuroforam</td>
<td>NA</td>
<td>immediate</td>
<td>right L-4 laminotomy</td>
<td>none</td>
<td>NA</td>
</tr>
<tr>
<td>Cosar et al., 2009</td>
<td>50, M</td>
<td>PVP</td>
<td>malign</td>
<td>T-7, T-10</td>
<td>immediate</td>
<td>bilateral weakness, paraplegia (12 hrs)</td>
<td>T4–10</td>
<td>epidural</td>
<td>NA</td>
<td>12 hrs</td>
<td>multilevel decompression laminotomy</td>
<td>no change, paraparesis</td>
<td>NA</td>
</tr>
<tr>
<td>Cosar et al., 2009</td>
<td>74, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L3–5</td>
<td>1 day</td>
<td>mild pain right leg</td>
<td>L-3, L-4</td>
<td>neuroforam</td>
<td>subligamentous leakage during PVP</td>
<td>NA</td>
<td>steroid treatment</td>
<td>pain resolved after 6 hrs</td>
<td>NA</td>
</tr>
<tr>
<td>Sonmez et al., 2010</td>
<td>74, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L-4</td>
<td>immediate</td>
<td>back pain, right leg pain</td>
<td>L-4–5 disc space</td>
<td>annulus extrusion of disc fragment on right L-5 nerve root</td>
<td>2 wks</td>
<td>microdiscectomy</td>
<td>complete recovery (3-mo FU)</td>
<td>injury cause: endplate &amp; vacuum cleft</td>
<td>(continued)</td>
</tr>
</tbody>
</table>
TABLE 1: Reported cases of neurological deficits after cement extravasation* (continued)

<table>
<thead>
<tr>
<th>Authors &amp; Years</th>
<th>Age (yrs), Sex</th>
<th>Op Type</th>
<th>Indication</th>
<th>No. of Levels</th>
<th>Time of Symptom Presentation</th>
<th>Neurological Symptoms†</th>
<th>Level</th>
<th>Location</th>
<th>Associated Complication</th>
<th>Time of Surgical Procedure(s)</th>
<th>Procedure Performed</th>
<th>Neurological Recovery Status†</th>
<th>Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ross &amp; Fineman 2010</td>
<td>81, M</td>
<td>PVP</td>
<td>OCF</td>
<td>L-2</td>
<td>3 days</td>
<td>rt leg pain, numbness</td>
<td>L-2</td>
<td>epidural, intradural</td>
<td>NA</td>
<td>3 days</td>
<td>lam, arthrodesis of L1–3</td>
<td>complete rec (9-mo FU)</td>
<td>delayed presentation</td>
</tr>
<tr>
<td>Park et al., 2010</td>
<td>88, F</td>
<td>BKP</td>
<td>OCF</td>
<td>L3–4</td>
<td>immediate</td>
<td>leg weakness (Grade 3) below L-3</td>
<td>L2–4</td>
<td>epidural</td>
<td>NA</td>
<td>2 days</td>
<td>decompress L3–4, partial L-2</td>
<td>improved to Grade 4 bilat (6-wk FU)</td>
<td>pedicle violation caused spread</td>
</tr>
<tr>
<td>Park et al., 2010</td>
<td>77, F</td>
<td>BKP</td>
<td>OCF</td>
<td>L-1</td>
<td>immediate</td>
<td>weakness in lt thigh (muscle strength Grade 3)</td>
<td>L-1</td>
<td>epidural</td>
<td>NA</td>
<td>immediate</td>
<td>decompress L-1, T12–L2 hardware</td>
<td>muscle strength Grade 5 (1-mo FU)</td>
<td>incompletely transforaminal caused partial medial wall compromise</td>
</tr>
<tr>
<td>Chen et al., 2007</td>
<td>78, F</td>
<td>PVP</td>
<td>OCF</td>
<td>L-2, L-5</td>
<td>?</td>
<td>rt leg weakness (Grade 3)</td>
<td>L2–3</td>
<td>rt extra-neuroforam</td>
<td>L4–5 disc, L-4 fracture</td>
<td>2 wks</td>
<td>L3–5 posterior fusion</td>
<td>complete rec at 6 days</td>
<td>lat wall perforated by needle</td>
</tr>
</tbody>
</table>

* ant = anterior; AP = anteroposterior; decompress = decompression; facetect = facetectomy; FU = follow-up; incontin = incontinence; lam = laminectomy; LMF = lateral mass fusion; malig = malignancy; mgmt = management; NA = not available; neuroforam = neuroforaminal; OCF = osteoporotic compression fracture; rec = recovery; substract = subtraction; ? = uncertain.
† In cases where a numerical grade is given, the grade refers to the Medical Research Council scale (grade range 0–5).

TABLE 2: Cement extravasation with neurological injury rate*

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Time frame of Studies</th>
<th>Indication/Technique</th>
<th>No. of Studies</th>
<th>Cement Leakage Reported</th>
<th>Neurological Injury Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bouza et al.</td>
<td>2006</td>
<td>1984–2005</td>
<td>VCF/BKP</td>
<td>26</td>
<td>Overall 7.13% (134/1742)</td>
<td>Symptomatic 1.50%</td>
</tr>
<tr>
<td>Bouza et al.</td>
<td>2009</td>
<td>2003–2008</td>
<td>malig/BKP</td>
<td>7</td>
<td>41 cases</td>
<td>0 cases</td>
</tr>
<tr>
<td>Chew et al.</td>
<td>2011</td>
<td>no restriction (up to 2010)</td>
<td>malig/PVP</td>
<td>30</td>
<td>4.2%–13.5%</td>
<td>2%</td>
</tr>
<tr>
<td>Eck et al.</td>
<td>2008</td>
<td>no restriction (up to 2006)</td>
<td>VCF/PVP</td>
<td>103</td>
<td>19.7% (1838/9330)</td>
<td>1.6% (65/4125)</td>
</tr>
<tr>
<td>Hulme et al.</td>
<td>2006</td>
<td>no restriction (up to 2005)</td>
<td>VCF/PVP</td>
<td>47</td>
<td>41% (32%–52%)</td>
<td>3.90%</td>
</tr>
<tr>
<td>Ploeg et al.</td>
<td>2006</td>
<td>1984–2005</td>
<td>VCF/PVP</td>
<td>15</td>
<td>9% (2.6%–16%)</td>
<td>2.20%</td>
</tr>
<tr>
<td>Taylor et al.</td>
<td>2006</td>
<td>1983–2004</td>
<td>VCF/PVP</td>
<td>57</td>
<td>40% (614/1551)</td>
<td>3.0% (8/275)</td>
</tr>
<tr>
<td>Taylor et al.</td>
<td>2007</td>
<td>2004–2006</td>
<td>VCF/BKP</td>
<td>13</td>
<td>8.0% (90/1111)</td>
<td>0.00%</td>
</tr>
</tbody>
</table>

* para = paraparesis; radic = radiculopathy; SCC = spinal cord compression; VCF = vertebral compression fracture.
spread, a posterior lateral transpedicular approach with or without a corpectomy may be used for greater access to the ventral epidural space. Removal of cement from the spinal canal requires a careful approach to avoid excessive thecal retraction, compression, or iatrogenic violation during central, lateral recess, foraminal, or lateral recess decompression. Often times, the cement does not adhere to the dura, with a plane present between the cement and the dura itself. The key is to ensure that plane with a micro-nerve hook, micro-Penfield dissector, and a microcurette and then to try to develop a surgical quarter in which you could remove the cement without causing undue pressure on the neural elements. An osteotome can be used for removal of large cement pieces. A high-speed bur with different bur tips depending on the size of the cement is useful for shaving off cement pieces adherent to the dura. Copious irrigation must be used to remove cement shavings and reduce thermal injury. Intradural spread of cement has been shown to track along nerve fibers leading to paraplegia.11 Shapiro et al.42 demonstrated that adherent PMMA can be successfully removed from both external and internal dural surfaces. Intradural PMMA removal requires a durotomy and careful cement separation from involved nerve rootlets. The placement of instrumentation should be considered when the index vertebral body injury is unstable, the decompression procedure is likely to cause iatrogenic destabilization, existing deformity must be corrected, or there is likelihood of worsening deformity, especially in the presence of severe underlying osteoporosis.51

Most patients included in the review demonstrated improvement in neurological status after surgical intervention, except for the report of Teng et al.46 in which multiple surgical procedures were unsuccessful in improving neurological function. The reason may have been development of iatrogenic destabilization after the initial isolated decompression cement removal procedure.

Certain interventions prior to PVP/BKP may decrease the risk of neurological injury. Most authors recommend that cement should have the consistency of toothpaste before injection,3 and careful review of imaging studies is necessary to ensure that the posterior cortical wall is intact.19 During needle advancement, the needle tip should not cross either the medial or lateral border of the pedicle on the anteroposterior view.26 Complete filling of the cancellous space radiographically is unnecessary for good results, and cement injection should stop when cement reaches the posterior one-fourth of the vertebral body to prevent cement extravasation into the spinal canal.31 Some authors recommend performing these procedures with patients under local anesthesia to enable early detection of neurological compromise,26 while others recommend general anesthesia for cardiorespiratory management, improved ability to obtain static images, and earlier intervention should clinically significant extravasation be suspected intraoperatively.34

Obvious limitations of our review include the nature of the articles included, which represent relatively low levels of evidence. In many instances, case reports did not include all the information that we sought to extract. However, complications may be significantly underreported, making all descriptions of extravasation leading to neurological injury valuable lessons for practicing surgeons. Nevertheless, we encourage higher-quality investigations to provide greater insight into the clinical course and optimal management of patients presenting with a neurological deficit after cement extravasation.

Conclusions

Serious neurological complications may occur after vertebral augmentation procedures. Surgeons need to be aware of possible risks associated with cement leakage and be prepared for surgical management of these complications to optimize patient outcome.

Disclosure

Dr. Albert owns stock in K2M, Vertech, In Vivo Therapeutics, Paradigm Spine, PearlDiver, Biometrix, Breakaway Imaging, Crossroot, Invuity, Pioneer, Gentis, ASIP, PMIG, and Facet-Link Inc.; he is a consultant for DePuy; he is also a board member of United Healthcare; receives royalties from DePuy and Biomet, is a member of the Cervical Spine Research Society, and is past chairperson of the International Meeting on Advanced Spine Techniques.

Author contributions to the study and manuscript preparation include the following. Conception and design: Sidhu, Kepler, Vaccaro. Acquisition of data: Sidhu, Savage. Analysis and interpretation of data: Sidhu, Savage. Drafting the article: Sidhu, Savage. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Sidhu. Administrative/technical/material support: Sidhu, Kepler, Albert, Vaccaro. Study supervision: Sidhu, Albert, Vaccaro.

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Cement leak after vertebroplasty


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