

## Spinal subdural hematoma from a ventral dural puncture after percutaneous vertebroplasty: illustrative case

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**BACKGROUND** Percutaneous vertebroplasty (PVP) is a common procedure, but cement leaks are not uncommon. Leakages do not always have consequences, but rarely complications do occur. Spinal subdural hematomas (sSDHs) are rare and even rarer presented as a complication after PVP. The best management for sSDH is, therefore, difficult to decide.

**OBSERVATIONS** The patient first received PVP for acute low back pain after falling. Cement leakages were noted after the procedure, but a sudden new-onset leg weakness only developed later. An emergency lumbar computed tomography scan showed cement leakages anterior to the dural sac; lumbar magnetic resonance imaging revealed a subdural spinal hematoma, and a decompressive laminectomy was performed. During the operation, a small cement mass in the shape of a horn was seen and was believed to have caused the sSDH. Postoperatively, the patient recovered to leg strength 5/5.

**LESSONS** PVP is considered a low-risk procedure, and cement leaks rarely give rise to complications. However, when leakages present anterior to the dural sac, they may cause dural tear and possible sSDH, regardless of size. This possibility draws attention to keeping awareness of such rare but possible complications after routine PVP procedures. Timely intervention for sSDH is necessary to ensure meaningful recovery.

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**KEYWORDS** spinal subdural hematoma; vertebroplasty; cement leakage; ventral dural tear

Percutaneous vertebroplasty (PVP) is a common and efficient procedure for treating vertebral compression fractures. Although often perceived as a minimally invasive low-risk procedure, postoperative complications caused by cement leakages are not uncommon and can lead to either a mild local leakage mass or serious systemic embolic events.<sup>1–5</sup> Spinal subdural hematoma (sSDH) after PVP is a rare complication.<sup>6–8</sup> Clinically, sSDHs can manifest as various nonspecific spinal symptoms and signs, which makes diagnosis difficult. Because no pathognomonic signs of SDHs have been described<sup>9</sup> and no standard treatment guideline is available, its management commonly takes reference from treatments of other types of spinal hematomas. However, cases of acute progressive neurological deficit require more aggressive treatment intervention. Herein, we present a case of an sSDH that developed after PVP and a discussion of the current understanding of the disease's pathogenesis, with an emphasis on the role of cement leakage and dural tear.

### Illustrative Case

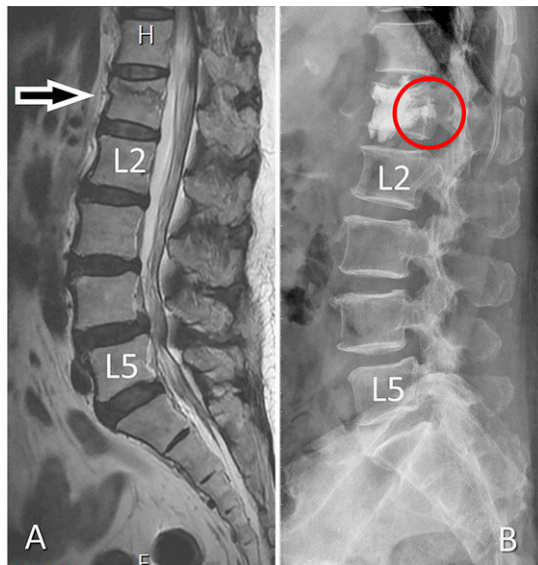
A 70-year-old woman presented to the outpatient department with acute low back pain after falling from a trestle ladder. An L1 lumbar compression fracture was diagnosed (Fig. 1), and elective PVP was performed with high-viscosity bone cement. The patient's back pain improved, with a decrease of the visual analog scale score from 6 to 1. Postprocedural radiographs (Fig. 2) revealed cement extravasation through the left anterolateral and superior and inferior cortices, but no immediate neurological deficit was noted, and the patient was discharged the following day. However, within 12 hours of discharge, the patient developed new-onset sudden acute bilateral leg weakness and returned to the emergency department for reassessment. Physical examination revealed a 2/5 paraparesis. An emergency lumbar computed tomography (CT) scan showed cement extravasation into the paraspinal spaces; the axial cuts showed a cement mass inside the canal, anterior to the neural sac (Fig. 3). Lumbar magnetic resonance

**ABBREVIATIONS** CSF = cerebrospinal fluid; CT = computed tomography; PVP = percutaneous vertebroplasty; sSDH = spinal subdural hematoma.

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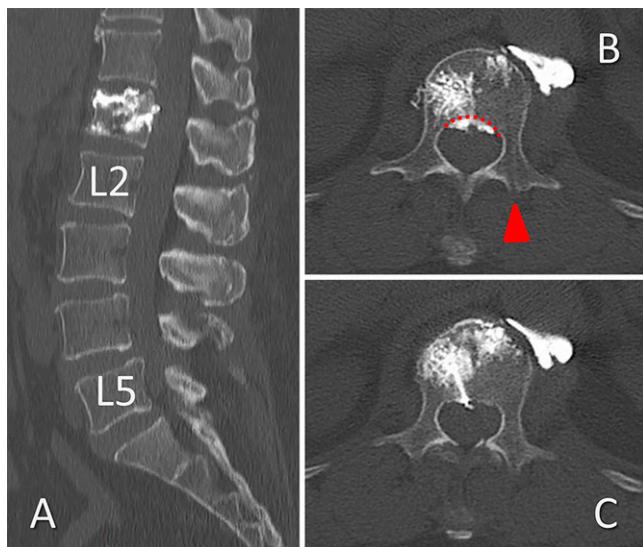
**FIG. 1. A:** Lumbar magnetic resonance imaging (MRI) showing an AO-A1 type wedge deformity and a transverse fracture through to the posterior cortex but not quite yet a burst pattern (arrow). **B:** Radiograph after cement vertebroplasty showing cement leakage material at the posterior cortex and maybe inside the canal as well (red circle).



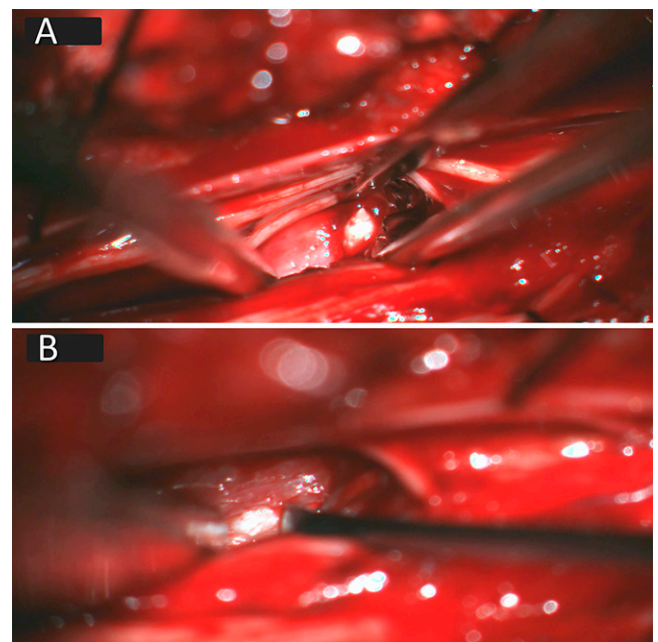
**FIG. 3. MRI** after the cement vertebroplasty. **A:** T2 heterogeneous signal changes spanning the L1–3 levels (arrows). **B and C:** Axial cuts of the lesion ventral to the neural elements, consistent with hematoma formation (arrows).

imaging revealed a T1-hyperintense intradural extramedullary lesion extending from L1 to L3 vertebral segments with compression to the cauda equina (Fig. 4). A diagnosis of L1–L3 subdural spinal hematoma was made, and decompressive laminectomy from L1 to L3 was performed to remove the subdural hematoma.

After the laminae and ligamentum flavum were removed, the dura was inspected under direct microscopic vision, which revealed no dural puncture on the dorsal or lateral aspects. Moreover, no epidural clot was found. The dura was then opened at the midline to evacuate the subdural hematoma. After the clot was cleared, a



**FIG. 2. CT** scans after cement vertebroplasty. **A:** Sagittal view confirming leakage within the spinal canal at L1. **B and C:** Axial cuts of cement leakage presence in paraspinous spaces and through the posterior cortex. The leakage material was within the concave contour of the posterior cortex and between the pedicles (red dotted line), which did not appear as massive on the sagittal view but presented with possible compression. The single trocar track (red arrowhead) was identified as the hypodense line through the pedicle, on which no breach was noted.



**FIG. 4. Intraoperative** images. **A:** Subdural clots interspersed between the rootlets. Ventral to the rootlets was a “spiky” cement leakage through the dura. **B:** After removal of the leakage, a hole in the ventral dura was clearly seen (at the tip of the microdissector).

cement mass in the shape of a horn was observed protruding from the ventral dura. The cement mass was removed, and a small dural opening was noted. It was sealed with a piece of triangle-shaped artificial dura substitute. After generous irrigation to ensure adequate hemostasis, the dura was closed in a water-tight fashion using a running Prolene suture. Additional stabilization with cement-injected fenestrated screw fixation was performed. Postoperatively, the patient recovered to achieve a leg strength of 5/5, with negligible residual weakness in the left leg. At the 3-month outpatient follow-up, the patient had regained considerable motor strength and could walk independently without assistance.

## Discussion

PVP is a common procedure for vertebral body compression fractures. Although cement leaks are not uncommon, sSDHs after PVP are rare.<sup>10-12</sup> Because of the rarity, few cases have been reported.

## Literature Review

We reviewed a total of eight cases, including cases reported previously and our case (Table 1). Trauma at an age of >60 years was the most prevalent among the cases; only one case involved an 18-year-old patient. The most common fracture pattern was an

AO-A1 wedge caused by low-energy falls. Six patients were considered to have acute onset that occurred immediately after their PVP or within days after their PVP, and two patients had delayed onset, presenting at 2 to 3 weeks. Six patients had paraparesis and two had single-leg paresis, and all but one had only radicular pain. Of the patients with paraparesis, two also had urinary and bowel dysfunction, suggesting a more severe presentation; of them, one case had acute onset (within 12 hours) and the other had onset 2 weeks after the PVP, making the association between time of onset and disease severity unclear. Almost all patients presented with an sSDH in the anterior of the canal. Two cases had short sSDHs (covering only two segments), but lesion length did not appear to be associated with clinical presentation or the decision to operate. Decompressive laminectomy of various extents was performed, but in all operative cases, evacuation of the subdural hematoma was attempted. Of the two cases treated conservatively, one was placed on steroid therapy and the other was prescribed physical therapy and rehabilitation. Furthermore, two cases had arachnoiditis presenting as refractory back pain, which occurred months later; one case had persistent sensory dysfunction and two cases exhibited a single-leg strength of 4/5. However, all patients, regardless of treatment, exhibited favorable motor recovery.

**TABLE 1. Summary of cases of spinal subdural hematoma after percutaneous cement vertebroplasty**

Case No.	Authors & Year	Age (yrs)/ Sex	Cause of Compression Fracture	Fracture Level, Type	Onset Time of sSDH After PVP	Clinical Presentation	sSDH Location & Length (segments)	Treatment for sSDH	Motor Recovery
1	Cosar et al., 2009 <sup>2</sup>	18/M	Motor vehicle accident	L2-4, A1	12 hrs	Back pain & progressive 2/5 paraparesis	Anterior to T1-L2 (14)	DL & sSE	5/5 strength
2	Cosar et al., 2009 <sup>2</sup>	75/F	Elective PVP	L1, A1	12-24 hrs	Paraparesis & fecal/urinary dysfunction	Anterior to T10-L3 (6)	DL & sSE	5/5 strength
3	Lee et al., 2012 <sup>14</sup>	40/F	Stair fall	T11-12, A1	2-3 wks	Back pain with progressive leg dysesthesia; no paresis	Dorsal of T11-L4 (6)	Intravenous dexamethasone	5/5 strength
4	Mattei et al., 2015 <sup>15</sup>	49/F	Stair fall	T8, A2	Immediately after PVP	Acute left leg 3/5 paresis & dysesthesia	Anterior of T7 to lower C (>8)	Physical therapy & rehabilitation	At least 4/5 strength
5	Tropeano et al., 2017 <sup>8</sup>	63/M	Oncological fracture	L1 & L3, A2	2 wks	Paraparesis (left 1/5; right 3/5), & dysesthesia & urinary dysfunction	Anterior of L2-3 (2)	DL & sSE	5/5 strength
6	Wang et al., 2018 <sup>5</sup>	64/F	Ground-level fall	T12, A1	Immediately after PVP	Acute right leg 3/5 paresis & dysesthesia	Anterior of T4-12 (9)	DL & sSE	5/5 strength
7	von der Brelie, et al. 2019 <sup>4</sup>	63/M	Elective PVP	T12, A1	40 hrs	Acute (0/5) paraplegia & dysesthesia, & fecal dysfunction	Anterior of T8-L3 (8)	DL & sSE	4/5 strength
8	Present study	70/F	Fall from trestle ladder	L1, A1	<12 hrs	Back pain, acute 2/5 paraparesis, & dysesthesia	Anterior of L1-3 (2)	DL & sSE	5/5 strength

DL = decompressive laminectomy; sSE = spinal subdural hematoma evacuation.

## Observations

The underlying pathogenesis of sSDHs is hypothesized to result from a sudden shift in the pressure difference between the spinal basivertebral system (i.e., epidural veins) and paraspinous intraabdominal/intrathoracic pressure.<sup>13–15</sup> A sudden drop in the cerebrospinal fluid (CSF) pressure may also cause a negative gradient, which affects the epidural veins or intradurally the radiculomedullary veins. Subarachnoid hemorrhage caused by head trauma may also migrate through the CSF to the spine and may present as an sSDH.<sup>14</sup> These speculations are more relevant to spontaneous sSDHs, but sSDHs after PVP may have a different pathogenesis.

PVP is a percutaneous procedure and involves the risk of trocar malpositioning and dural puncture.<sup>10,16</sup> A dural tear after PVP is a potential cause of an sSDH. Another mechanism by which PVP may cause a dural tear involves cement leakage. When cement injection is not well monitored, cement may migrate toward the posterior one-third of the vertebra or overtly into the canal. When the leakage material comes in contact with the dura, the heat produced from cement curing can pose a tear risk, regardless of the size of the leakage.

Although cases of sSDH after PVP with the absence of an epidural clot but with subdural hematoma interspersed between the rootlets (lumbar lesions) have been reported, no report has described cement leakage puncturing the dura through a tear. Although the pathogenesis remains unclear, our case demonstrated that even a small leakage can tear the dura and create an entrance for epidural blood to ingress into the subdural space, causing sSDH. A similar mechanism has also been observed by Cosar et al.<sup>2</sup> Our patient experienced sudden paraparesis after PVP even while performing normal daily activity such as sitting down and walking. Similar findings were observed by Lee et al.<sup>14</sup> in a patient who reportedly presented with an sSDH without obvious trauma from external forces (i.e., sudden onset while walking on a flat road weeks after PVP). These findings suggest that a leakage inside the canal can cause a dural tear, which may result from dynamic spine motioning, even during daily activity, regardless of the time after PVP.

Our case is unique in that the patient received a unilateral transpedicular cement vertebroplasty under live fluoroscopic monitoring. During the procedure, a single trajectory was used and no malpositioning of the trocar needle was noted. Although leaks were noted on the postprocedural radiograph, the patient did experience symptomatic relief. The interval from PVP to when the acute motor deficit developed suggested that the sSDH occurred sometime after the PVP. The leakage first appeared to occupy slightly less than one-fourth of the canal anteriorly, with mild sac compression and probably no myelopathic effect on the neural elements. However, with the sharp protrusion as seen on the lumbar axial CT scan and direct visual during the surgery, we confirmed that the resultant dural tear was simply caused by the material leakage. Such posterior leakages, when present as small masses or obscured by superimposed pedicles on lateral radiographs, are difficult to detect.<sup>9,17</sup> Strategies using surrogate imaging markers to stratify the risk of a leak have been developed; however, when these markers are not clear, determining a leak becomes challenging.<sup>17</sup> This case did not have a lamina fracture or an overt angulated spinal deformity compromising the canal with stenosis. However, similar to the other cases, no epidural hematoma was present. In contrast to the case reported by Lee et al., this patient's leakage mass was much smaller but had a sharp, spiky shape. We believe that size alone

may contribute partly to the post-PVP dural tear, but certain morphology should also be considered a risk.

Intradurally, many clots were found interspersed between the rootlets, resulting in compression of the neural elements. Although two cases from previous reports were treated conservatively and exhibited favorable motor recovery, in the acute setting of an sSDH and probable progression, most surgeons seemed to prefer a timely decompressive operation to evacuate the hematoma and save motor function.

## Lessons

This case demonstrates the rare sSDH as a complication after cement PVP. Although definitive clinical sequelae of sSDH cannot be predicted, their presentation as nonspecific and varying deficits can often be confusing and delay timely diagnosis. When presented after procedures such as PVP, which is usually a low-risk procedure, a comprehensive assessment should be implemented to exclude sSDH. Although complications of cement leaks are not common, when present anterior to the dural sac (or inside the ventral dura), even small leakages may cause dural tears.<sup>7</sup> Because the exact mechanism of this is yet to be fully elucidated and clinical assessment with conventional imaging technologies may be inconclusive, clinicians are required to rely on a high index of suspicion. Moreover, sSDH as a complication can occur any time after PVP. When sSDH presents with sudden-onset motor deficit after PVP,<sup>10</sup> this should prompt clinicians to consider all probabilities to ensure timely detection and treatment.

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### Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

### Author Contributions

Conception and design: all authors. Acquisition of data: Yang, Liu. Analysis and interpretation of data: Yang, Liu. Drafting the article: Yang. Critically revising the article: Yang, Liu. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Lin. Administrative/technical/material support: Yang, Liu. Study supervision: all authors.

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