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CASE REPORT

Spinal Epidural Hematoma After Percutaneous Kyphoplasty: Case Report and Literature Review

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Objective: To present the case of a patient on long-term anticoagulants who developed acute spinal epidural hematoma (SEH) after percutaneous kyphoplasty (PKP) without signs of major cement extravasation to the spinal canal.

Methods: A 64-year-old woman with long-term oral antiplatelet drugs underwent the L1 PKP. Immediately after the operation, the back pain improved significantly without neurological deficit. However, 12 hours later, she developed progressive weakness of the bilateral lower limbs. No intraspinal cement leakage was obvious on the postoperative lumbar radiograph and computed tomography.

Results: An emergency MRI examination revealed a high signal aggregation in front of the spinal cord from T12 to L1, indicating spinal cord compression. The SEH was verified and removed during the laminectomy from T12–L1. Following the decompression surgery, the neurological deficit of the lower limbs improved. On follow-up after 6 months, the muscle strength of the bilateral lower limbs had returned to normal.

Conclusion: For the patient with long-term oral antiplatelet drugs or coagulation malfunction, the transpedicle approach or that via the costovertebral joint with a smaller abduction angle is recommended to reduce the risk of injury to the inner wall of the pedicle. For progressive aggravation of neurological dysfunction after surgery, SEH formation should be suspected despite the absence of intraspinal bone cement leakage. Secondary emergency decompression should be considered to avoid permanent damage to spinal cord nerve function caused by continuous compression.

Keywords: spinal epidural hematoma, percutaneous vertebroplasty, percutaneous kyphoplasty, spinal cord compression, coagulation malfunction

Introduction

Percutaneous kyphoplasty (PKP), a minimally invasive procedure, is widely used for treatment of pain owing to osteoporotic vertebral compression fractures (OVCF). However, it has many serious clinical complications, including cement intraspinal canal leakage, that causes spinal cord compression,[1–4] pulmonary or renal vascular embolism,[5–8] infection,[9] and adjacent vertebral body fractures.[10] As an emergency complication, spinal epidural hematoma (SEH) is clinically rare after PKP or percutaneous vertebroplasty (PVP), and has been reported in seven studies.[11–17] In this report, we present the case of a patient on long-term anticoagulants who developed acute SEH after PKP without signs of major

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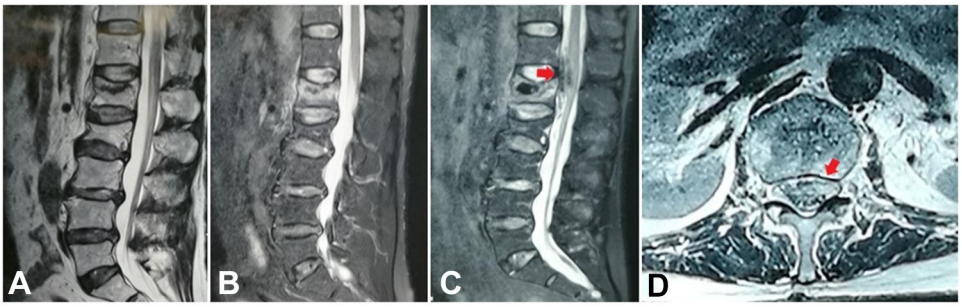

Figure 1 Although the L1 and L2 vertebra are wedge shaped at the T2 weighted imaging of magnetic resonance imaging (MRI) (**A**), the high signal of the short time inversion recovery (STIR) sequence was only seen in the L1 vertebral body (**B**). Twelve hours after the PKP, a high signal aggregation in front of the spinal cord from T12 to L1 was detected at the sagittal ((**C**), red arrow) plane of MRI examination, which gathered in the left part of the spinal canal at the axial plane of MRI ((**D**), red arrow).

cement extravasation to the spinal canal. Further, we comprehensively review the relevant literature on the possible pathogenesis of SEH.

Case Presentation

A 64-year-old woman with severe back pain was admitted to the primary hospital. Six months ago, she underwent cardiac stent implantation for myocardial infarction and was prescribed oral aspirin (100 mg/daily) and clopidogrel (75 mg/daily). The prothrombin time (PT) and activated partial thromboplastin time (APTT) were within the normal range on preoperative laboratory examination. The lumbar magnetic resonance imaging (MRI) showed a L1 and L2 compression fracture; the high signal of the short time inversion recovery (STIR) sequence was seen in the L1 vertebral body (Figure 1A and B). She had no neurological symptoms, and she underwent the L1 PKP. The puncture, balloon dilatation, and cement injection were performed using intraoperative fluoroscopy (Figure 2A–F). Immediately after the operation, the back pain improved significantly without neurological deficit. However, 12 hours later, she developed progressive weakness of bilateral lower limbs. No intraspinal cement leakage was obvious on the postoperative lumbar radiograph (Figure 3) and computed tomography (CT) (Figure 4). The patient was transferred to our hospital and underwent an emergency MRI examination, which revealed a high signal aggregation in front of the spinal cord from T12 to L1, indicating spinal cord compression (Figure 1C and D). Physical examination revealed grade III strength in the bilateral iliopsoas, quadriceps femoris, and tibialis anterior muscles. We suspected postoperative SEH and performed laminectomy and decompression from T12–L1. Following excision of SEH and decompression of the spinal cord, the neurological deficit of the lower limbs improved. On follow-up after 6 months, the muscle strength of the bilateral lower limbs had returned to normal.

Discussion

PVP and PKP are often used to treat OVCF. They have been applied successfully even in burst fractures without neurological deficit.[18] We describe the case of a patient on long-term anticoagulants, who developed acute SEH after PKP, despite the absence of major cement extravasation into the spinal canal, and review the possible pathogenesis.

SEH is associated with idiopathic, iatrogenic, traumatic, and coagulation diseases, and is relatively rare in clinical practice. SEH may cause severe acute spinal cord injury (SCI). Domenicucci et al[19] reviewed 1,010 SEH cases in 16 years and concluded that 18% of the significant cases were iatrogenic (spinal puncture), while 29% were non-iatrogenic and caused by factors such as clotting, trauma, and pregnancy. However, iatrogenic SEH after PKP or PVP has been reported in seven previous studies[11–17] (Table 1). Wang et al[11] suspected that SEH after PKP was caused by direct injury from intraoperative puncture. This finding was supported by three other studies.[12–14] Mattei et al[15] suggested that venous congestion plays a pivotal role in the etiology of SEH. von der Brelie et al[16] attributed the development of SEH after PVP in a patient with long-term oral aspirin to coagulation malfunction owing to aspirin.

When the needle arrived at the posterior wall of the vertebral body in the sagittal plane of fluoroscopic view,

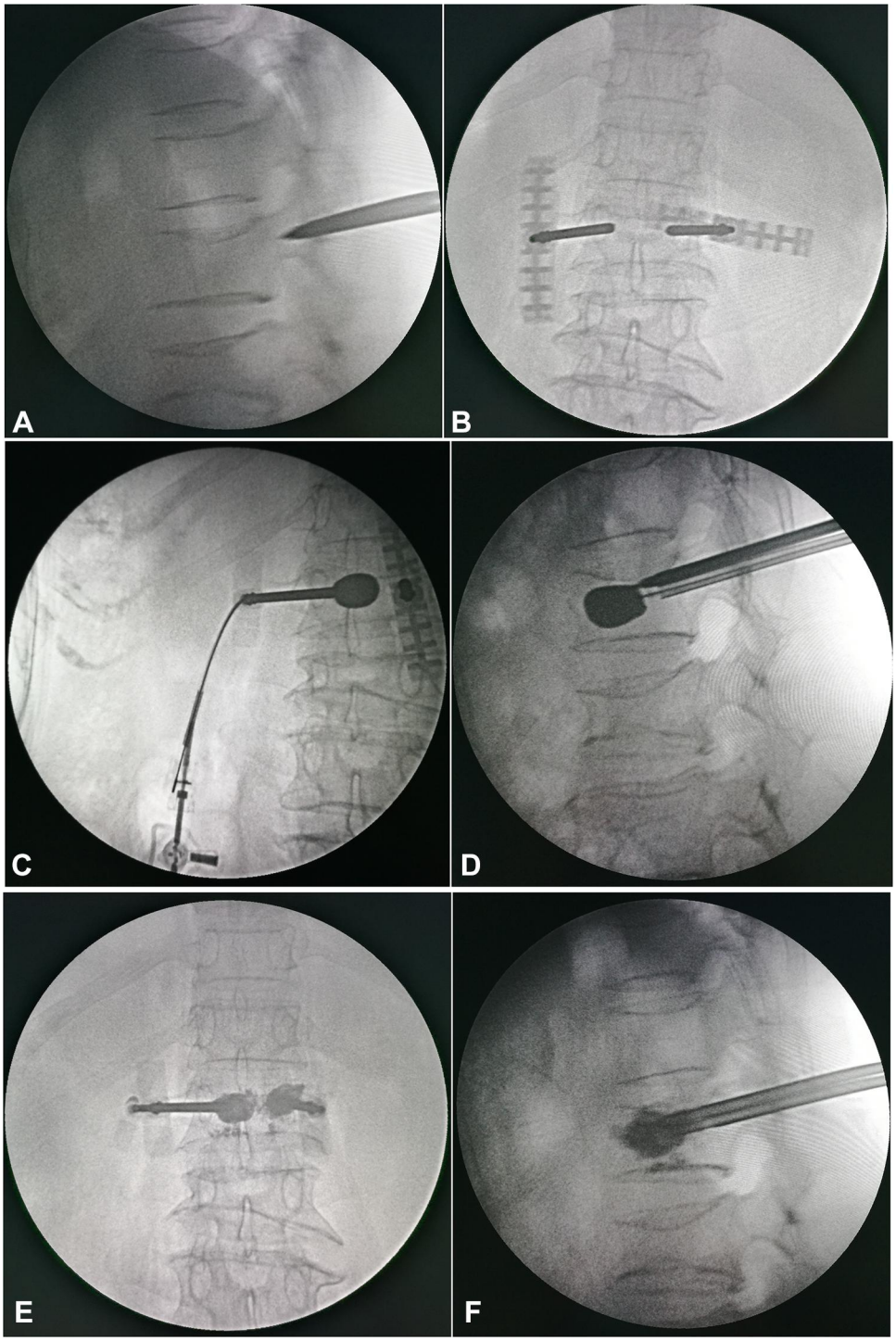

Figure 2 At the biplane monitoring, the puncture (**A, B**), balloon dilatation (**C, D**), and cement injection (**E, F**) were performed stepwise. Notably, when the needle arrived at the posterior wall of the vertebral body in the sagittal plane of fluoroscopic view, the intraoperative puncture revealed extension of the puncture needle beyond the inner edge of the projection of the pedicle in the coronal plane, suggesting a large abduction angle of the needle (**A, B**).

the intraoperative puncture revealed an extension of the puncture needle beyond the inner edge of the projection of the pedicle in the coronal plane, suggesting a large abduction angle of the needle (Figure 2) and increased risk of perforation of the inner wall of the pedicle. Postoperative CT also revealed dispersion of the bone cement on the left side of the vertebral body along the puncture trajectory whose extension line is medial to the inner wall of the

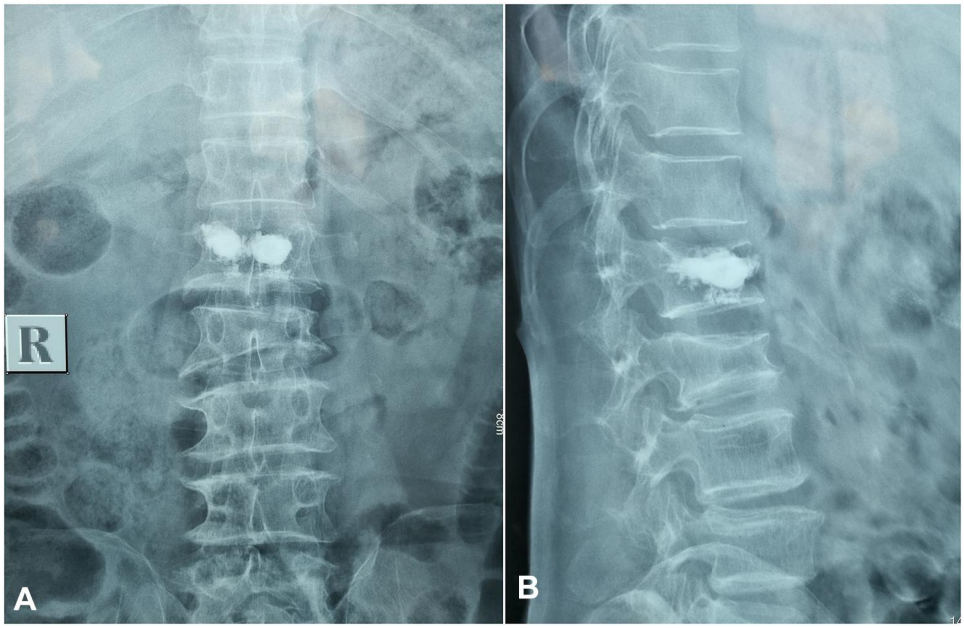

Figure 3 No intraspinal cement leakage was obvious on the anteroposterior (**A**) and lateral (**B**) view of the postoperative lumbar radiograph.

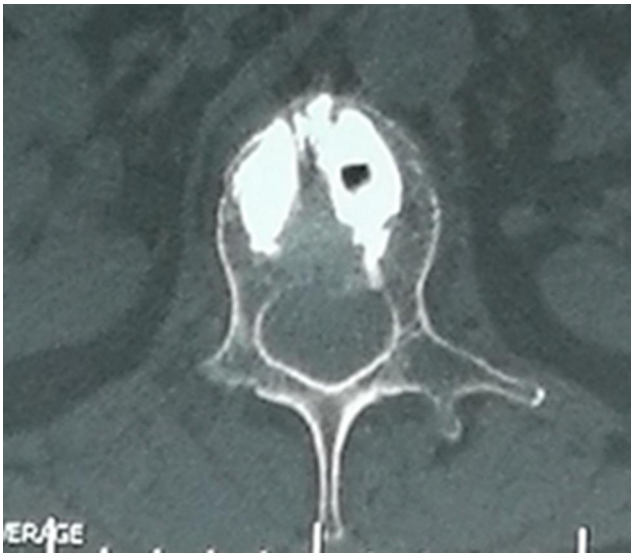

Figure 4 While there was no obvious intraspinal cement leakage on the postoperative lumbar computed tomography, there is visible dispersion of the bone cement on the left side of the vertebral body along the puncture trajectory, whose extension line is medial to the inner wall of the pedicle.

pedicle (Figure 4). Owing to the significant buffer space surrounding the spinal cord at the level of L1, the puncture needle did not damage it, despite it's large puncture angle or slightly perforated inner wall of the pedicle. Thus, the patient did not experience nerve dysfunction immediately after the operation. Owing to stent implantation, long-term oral aspirin and clopidogrel were administered, which had inhibitory effects on platelet aggregation. Despite normal preoperative PT and APTT, a lack of effective blood clotting resulted in flow of blood from the sinus of the vertebral body through the hole in the inner wall of the pedicle along the pressure gradient, causing SEH and acute compression of the spinal cord. Therefore, the patient showed a gradual decline in neurological function 12 hours after surgery.

Özkan[20] reported that if a patient receiving anticoagulant therapy has local or reflected pain and loss of strength and sensation, spontaneous SEH should be considered. There is a lack of consensus on the role of long-term oral antiplatelet drugs and coagulation malfunction on the risk of postoperative SEH in patients with OVCF. In this case, anexcessive puncture angle was considered to have damaged the inner wall of the pedicle, causing SEH. The transpedicle approach or that via the costovertebral joint with a smaller abduction angle is recommended to reduce the risk of injury to the inner wall of the pedicle. Thus, a bilateral procedure is theoretically better than a unilateral procedure. For progressive aggravation of neurological dysfunction after surgery, SEH formation should be suspected despite the absence of intraspinal bone cement leakage. Secondary emergency decompression should be considered to avoid permanent damage to spinal cord nerve function caused by continuous compression.

Table 1 Surgical Level, Anticoagulants, Symptoms, Cause Analysis, and Clinical Outcome for the Cases with Iatrogenic SEH after PKP or PVP

Author	Surgical Level	Anticoagulants	Symptoms	Cause Analysis	Clinical Outcome
Fang 2018[12]	T8–T12 (6 cases)	Not described	Low back pain, spinal cord injury after PKP	The piercing damage	Recovery after surgical decompression
Wang 2018[11]	T12	Not described	Weakness of both lower limbs	The piercing damage	Recovery after surgical decompression
Mattei 2015[15]	T8	Not described	Weakness of left lower limb	The congestion of venae spinales	Recovery after surgical decompression
Tropeano 2017[17]	L1–L3	Not described	Weakness of both lower limbs	Not described	Recovery after surgical decompression
von der Brelie 2019[16]	T12	Preoperative medication of Aspirin	Weakness of both lower limbs	Anticoagulation	Recovery after surgical decompression
Cosar 2009[13]	L1; L2–L4 (2 cases)	Not described	Weakness of both lower limbs	The piercing damage	Recovery after surgical decompression
Lee 2012[14]	T11–T12	Not described	Radiate pain for both lower limps	The piercing damage	Recovery after drug therapy

Abbreviations: SHE, spinal epidural hematoma; PKP, percutaneous kyphoplasty; PVP, percutaneous vertebroplasty.

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Disclosure

The authors report no conflicts of interest for this work.

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