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Case Report

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Acute Paraparesis Caused by Spinal Epidural Fluid After Balloon Kyphoplasty for Traumatic Avascular Necrosis: A Case Report

Chi Ho Kim, Pius Kim, Chang Il Ju, and Seok Won Kim

Department of Neurosurgery, College of Medicine, Chosun University, Gwangju, Korea

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Address for correspondence:
Seok Won Kim
Department of Neurosurgery, College of Medicine, Chosun University, 365 Pilmun-daero, Dong-gu, Gwangju 61453, Korea.
Email: chosunns@chosun.ac.kr

ORCID iDs
Chi Ho Kim
https://orcid.org/0000-0002-9955-5107
Pius Kim
https://orcid.org/0000-0001-5514-9257
Chang Il Ju
https://orcid.org/0000-0001-9123-2808
Seok Won Kim
https://orcid.org/0000-0002-1910-0242

Conflict of Interest
The authors have no financial conflicts of interest.

ABSTRACT

Minimally invasive procedures, such as percutaneous vertebroplasty or balloon kyphoplasty (BK), eliminate motion at the fracture site and relieve pain associated with traumatic avascular necrosis when conservative treatment fails. However, these are associated with complications, most of which are directly related to cement leakage. Herein, we report a rare case of acute paraparesis caused by spinal cord compression by epidural fluid following BK for the treatment of Kummell's disease in the absence of cement leakage. To the best of our knowledge, this is the first report describing this complication.

Keywords: Osteonecrosis; Epidural space; Complication

INTRODUCTION

The incidence of osteoporotic compression fracture (OCF) is increasing with age in the elderly population. Kummell's disease after OCF is not a rare complication and is usually related to continuous pain and neurologic deficits.

A number of theories have been proposed to explain its pathophysiological mechanisms of this disease entity and now it is also termed traumatic avascular necrosis, nonunion, all of which suggest the disruption of the healing process of fractured vertebrae.[3)]

Bone cement injection such as percutaneous vertebroplasty (PV) or balloon kyphoplasty (BK) can be adopted when conservative treatment fails for Kummell's disease. BK can correct local kyphosis and decrease cement leakage compared to PV and BK is adopted for the management for Kummell's disease. However, it is not free of complications. Here, we report a rare case of acute paraparesis following BK for treating Kummell's disease without evident cement leakage. it is important to understand its pathophysiology to ensure that necessary precautions are taken when performing BK to treat Kummell's disease.

CASE REPORT

A 72-year-old man experienced persistent back pain since he slipped down 6 months before medical consultation. He was transferred from a traditional oriental hospital to our institute due to difficulty sitting. Physical examination revealed marked tenderness at the L1 level and increased pain specially during flexion and extension. The pain relieved only when he lay still in bed. The neurological examination and laboratory findings were within normal limits.

Computed tomography (CT) showed a compression fracture of the L1 vertebra with an intravertebral vacuum cleft (IVC) (**FIGURE 1A & B**). Magnetic resonance imaging (MRI) of the thoracolumbar spine revealed an IVC filled with gas (**FIGURE 1C & D**).

He had severe osteoporosis, with a mean T-score of −3.40 on bone densitometry. There was no evidence of a soft tissue mass or a paravertebral abscess; therefore, he was considered to have a benign OCF with traumatic avascular necrosis, named Kummell's disease. BK was performed because the intractable back pain was not relieved by conservative treatment, and approximately 6 mL of bone cement was carefully injected into the vacuum space of the vertebral body under C-arm fluoroscopy through both right and left sides.

Postoperative radiographs showed that the bone cement was well-localized in the cavitary lesion and filled the L1 vertebral body (**FIGURE 2A & B**). At the time of discharge, 2 days after BK, the pain was significantly relieved and the patient was able to ambulate independently wearing a brace.

However, the patient was referred to the emergency room because of progressive bilateral weakness of the lower extremities 3 weeks after BK. He was unable to walk independently. The back pain was aggravated again; Moreover, neurological examination revealed weakness in both thighs and legs and numbness in the anterior aspect of both thighs and he was unable to flex his hips. Simple radiographs and CT scans of the spine showed that the cement was still contained within the L1 vertebral body without any leakage. However, simple radiographs revealed the separation of bone cement (**FIGURE 2C & D**). Bone cement did not adhere to the endplate of the fractured vertebra with a disrupted posterior cortical wall (**FIGURE 3A & B**). MRI of the spine revealed a canal stenosis caused by spinal epidural fluid compressing the

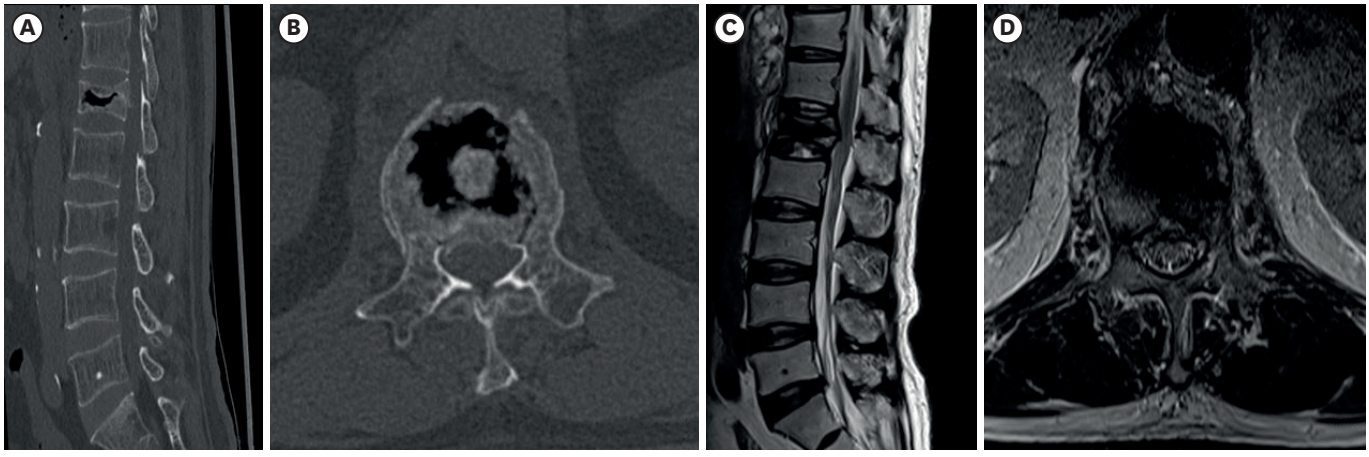

FIGURE 1. Imaging studies obtained before balloon kyphoplasty. (A, B) Sagittal and axial computed tomography scans reveal L1 compression fracture with an intravertebral cleft. (C, D) T2-weighted sagittal and axial magnetic resonance images show an acute compression fracture.

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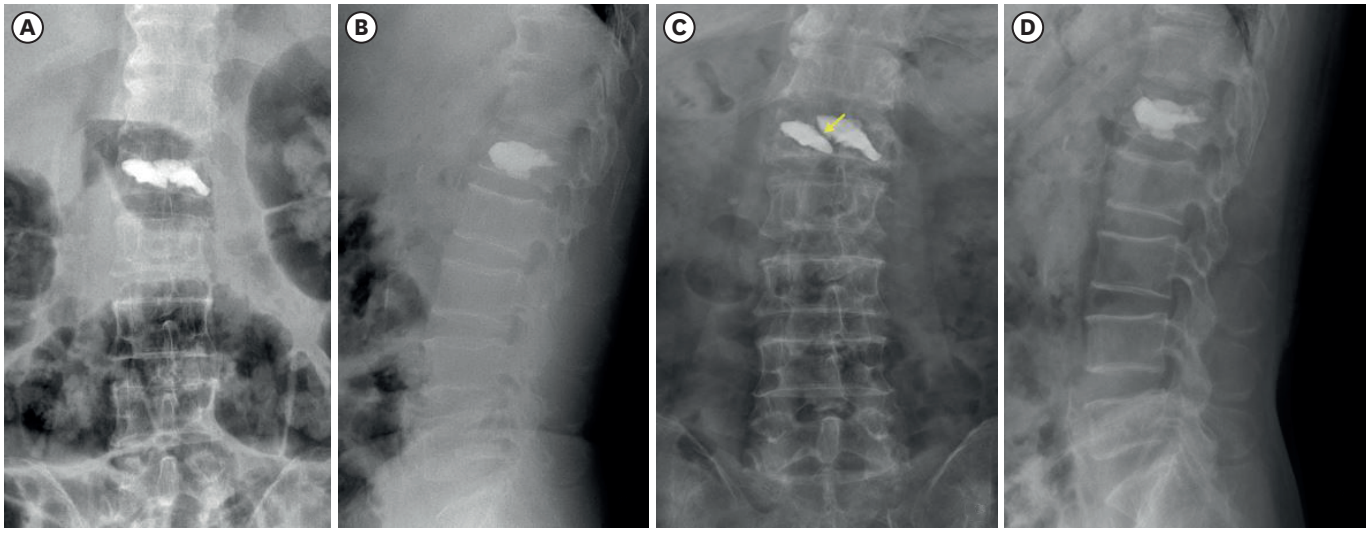

FIGURE 2. (A, B) Simple radiographs right after balloon kyphoplasty show effective cement filling of fractured cavity. (C, D) Simple radiographs taken 3 weeks after balloon kyphoplasty reveal the separation of bone cement (arrow).

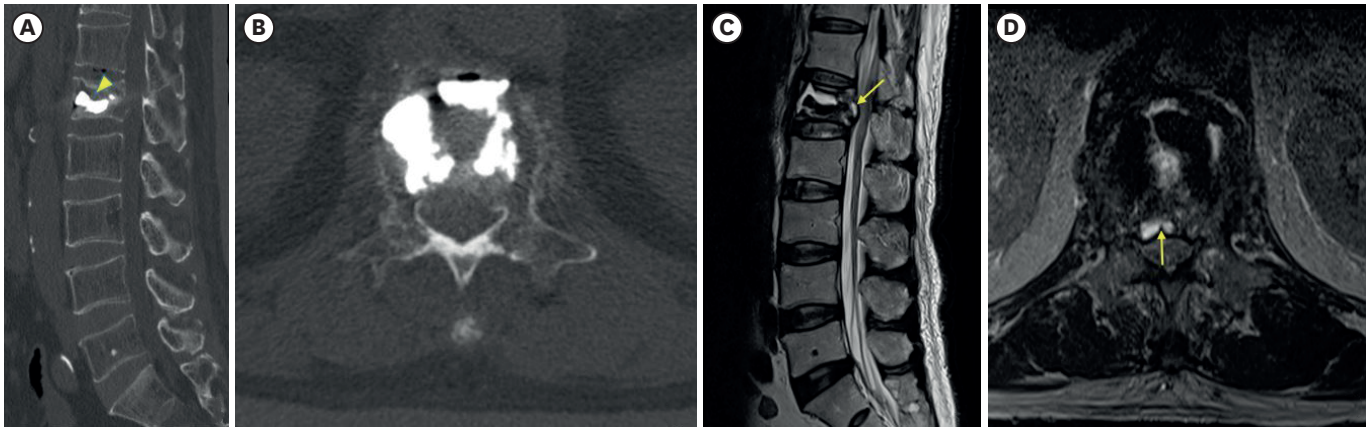

FIGURE 3. (A, B) Sagittal and axial computed tomography scans 3 weeks after balloon kyphoplasty reveal bone cement not adhering to the endplate of the fractured vertebra (arrowhead) and a disrupted posterior cortical wall without cement leakage into the spinal canal. (C, D) T2-weighted sagittal and axial magnetic resonance images taken 3 weeks after balloon kyphoplasty show high signal intensity seen as spinal epidural fluid, which is connected to fluid with intravertebral body (arrows).

spinal cord, which was connected to an IVC filled with fluid despite the BK (**FIGURE 3C & D**). Surgical stabilization by bone cement augmented percutaneous screw fixation from T12 to L2 was performed without decompression. Motor power improved to normal strength with a decrease of paresthesia 4 days postoperatively, and he was again able to walk independently 3 weeks postoperatively. Back pain and neurological deficits were completely resolved 24 months postoperatively, and the patient could walk without any difficulty (**FIGURE 4**).

DISCUSSION

The incidence of osteoporotic vertebral compression fractures increases with age, and BK is increasingly adopted for the treatment of OCFs. BK not only relieves pain immediately, but also improves sagittal balance, reduces extravasation rate by low-pressure injection, and increases viscosity during injection.[6]

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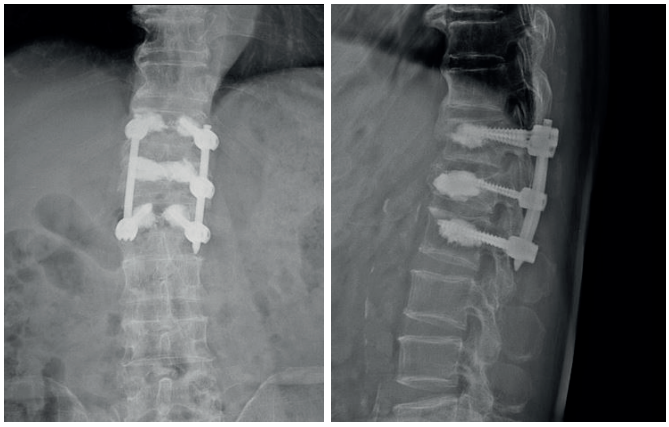

FIGURE 4. Simple radiographs taken 24 months after bone cement augmented percutaneous screw fixation show good surgical stabilization.

Kummell's disease is a spinal disorder characterized by a delayed post-traumatic collapse of the vertebral column. Moreover, it is thought to be pseudo-arthrosis in which IVCs represent a fracture non-union and mobility.[5] When conservative treatment fails, bone cement augmentation, such as PV or BK, can be effective minimally invasive procedures to relieve pain and enable patients with Kummell's disease to mobilize. Furthermore, IVC is completely filled to maximize fracture stabilization. However, the injection of viscous cement at low pressure decreases the penetration of cement into the microstructure of cancellous bone and its interdigitation with the surrounding hollow trabecular bone in Kummell's disease.[7]

Kummell's disease with spinal epidural fluid is a unique complication that has been rarely reported since first reported in 2008.[4] The integrity of the posterior cortex is an important factor to consider in the formation of epidural fluid, as any defects in it may lead the epidural fluid to be too weighty to bear because of the non-union and mobility. The treatment of Kummell's disease with formation of spinal epidural fluid depends on the presence of neurological deficits, severe back pain, and the ability to withstand surgery. Kim et al.[2] suggested that the fluid, including the hemorrhage inside the IVC, may be pushed out under pressure into the epidural space during daily motion. The connection between the IVC and epidural space causes subacute or chronic formation of epidural fluid. Bone cement augmentation using PV or BK could be effective in treating spinal epidural fluid or hematoma. Filling the IVC with bone cement can block the connection between the intravertebral cleft and the epidural space and aid in the spontaneous resolution of the hematoma over time. Bai et al.[1] also reported that bone cement augmentation using PV not only alleviate pain effectively, restore vertebral body height, and reconstruct spinal stability, but also block the connection between the IVC and the epidural space, by filling the IVC with bone cement, and help in the spontaneous absorption of the spinal epidural hematoma.

However, our patient had acute paraparesis despite the cement augmentation performed in BK. Less interdigitation by BK was not able to block the connection between IVC and epidural space. Therefore, Kummell's disease with posterior cortical disruption occurs, and surgical stabilization via fusion should be considered. Moreover, the spinal epidural fluid and its potential to spread along the epidural space through the operated level should be considered in patients with acute paraparesis who do not show cement dislodgment or leakage.

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CONCLUSION

We report a rare case of spinal epidural fluid associated with Kummell's disease, despite undergoing BK. Although the bone cement augmentation in BK can theoretically block the connection between the IVC and epidural space, it can cause a delayed spinal epidural fluid, resulting in acute paraparesis. Although rare, the possibility of spinal epidural fluid formation after performing BK for treating Kummell's disease should be considered.

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