

Case Report

Minimally invasive transforaminal endoscopic treated neurologic deficit after percutaneous kyphoplasty-a case report

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Abstract: Although iatrogenic neurologic injuries are infrequent after percutaneous kyphoplasty (PKP), the consequence is ruinous. Previous reports confirmed that acute neurologic deficit could mainly be attributed to cement extravasation into the spinal canal, resulting in the compression of spinal cord and nerve root. Herein, we present a case of using percutaneous transforaminal endoscopy to relieve nerve compression which caused by the fracture of superior articular process after PKP. A 67-year-old male patient was admitted to our hospital with leg radiculopathy and weakness of extensor digitorum longus three weeks after PKP. CT detected the fracture of superior articular process of L4, and confirmed that fracture fragment compressed nerve root of L4. The patient was treated using a minimally invasive transforaminal endoscopic approach, with an indirect decompression of the nerve root performed by removing the posterior border of the L4 vertebra, along the direction of the affected nerve root. The approach was successful, with the patient remaining symptom free at 1-year post-surgery. We propose that transforaminal endoscopic surgery can provide a suitable alternative for the treatment of spinal cord and/or nerve root compression after PKP in selected cases.

Keywords: Vertebroplasty, neurologic deficit, percutaneous endoscopic

Introduction

Percutaneous vertebroplasty (PVP) and percutaneous kyphoplasty (PKP) are widely performed for the treatment of the pain due to vertebral compression fractures associated with osteoporosis [1]. Although these procedures are considered to be relatively safe, occurrence of complications have been reported, including acute neurological deficits, cement leakage, infection, epidural hematoma, rib fractures, pulmonary embolism, systemic cement toxicity, and adjacent vertebral body fracture [2-9]. Of these, acute neurological deficits, principally due to cement extravasation into the spinal canal or into the paravertebral vascular system, are the most common [3, 10, 11]. Moreover, the increased intra-vertebral pressure and expansion of the vertebral body that occurs with cement filling for reconstruction can cause a retropulsion of the bony fragments of pre-existing or newly formed fractures, which can

compress the spinal cord and/or nerve roots [3, 10, 11]. In this case report, we describe the occurrence of a fracture of the superior articular process after PKP, which compressed the nerve root of L4, resulting in neurologic deficits. This fracture was difficult to diagnosis considering the absence of radiographic evidence of cement leakage into the spinal canal and of previous reporting of such fractures in the clinical literature. The patient was transferred to our hospital for assessment of the neurological deficit and treatment.

Case report

Initial history

The patient was a 67-year-old male who had undergone PKP for treatment of an osteoporotic fracture of L4 at a local hospital. The patient did not have a previous history of neurological deficit. Immediately following the procedure,



Figure 1. X-ray demonstrated good filling of the L4 vertebral body, and no cement leakage was observed within spinal canal. A. Anteroposterior radiographs; B. Lateral radiographs.

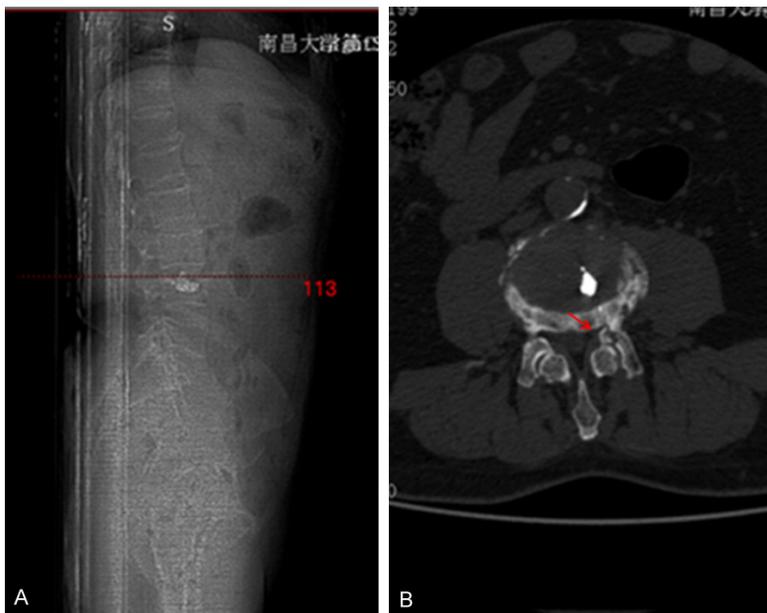


Figure 2. CT detected traveling nerve root of L4 (red arrow) was compressed due to the fracture of superior articular process of L4. A. Locator; B. Coronal CT.

the patient began complaining of left leg pain. Radiographs obtained at that time (**Figure 1**) showed good filling of the L4 vertebral body, with no evidence of cement leakage into the spinal canal. Bed rest was advised for 1 week, with Flurbiprofen Axetil (a non-steroidal anti-inflammatory drug) prescribed for pain relief.

This conservative treatment significantly improved the left leg pain and the patient was discharged from hospital 1 week after PKP. Within 5 days of discharge, symptoms re-appeared, with aggravation of the pain during walking and change in position, from lying to sitting. The patient was admitted to our hospital with left leg radiculopathy, 3 weeks after the PKP procedure.

Physical examination

Salient findings included pain in the distribution of L4 and weakness of the left extensor digitorum longus. Using a 10 cm visual analog scale (VAS), the patient reported the intensity of his leg pain to be 8/10, with an Oswestry disability index (ODI) score 72, where a higher score on both the VAS and ODI is indicative of worse symptoms and disability, respectively.

Computed tomography (CT) imaging was performed, revealing a fracture of superior articular process of L4 with an associated lateral recess stenosis and L4 nerve root compressions (**Figure 2**). The fracture was deemed to have resulted from an inadequate puncture trajectory through the vertebral body for cement filling. Surgical decompression was recommended to the patient, based on previous reports of wide laminectomy being the most effective treatment to obtain

satisfactory clinical outcomes in most cases of neurological deficit after PKP [3, 10, 11].

Surgical procedure

In this case, considering the severity of osteoporosis, an open laminectomy, with or without

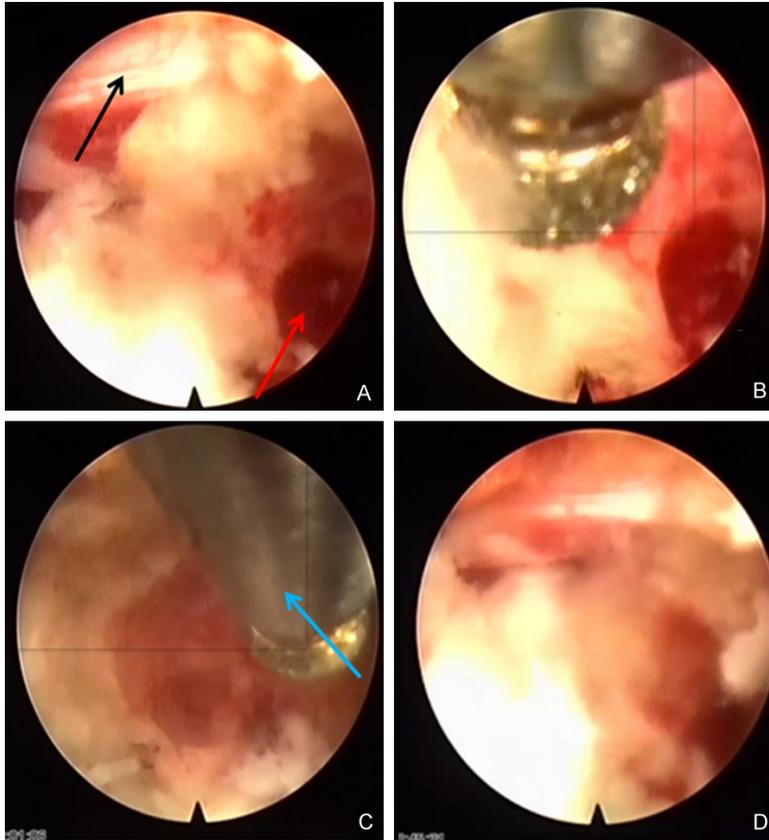


Figure 3. The magnification is 20 times the original size. A. Traveling nerve root of L4 (black arrow) was exposed and a part of posterior border of L4 vertebrae was removed (red arrow). B. Further removing the posterior border of L4. C. A 3.2-mm flexible drill (blue arrow) decompressed the nerve root. D. An endoscopic view confirmed the decompressed L4 nerve freely in the epidural space.

to the midline. The direction and depth of entry to reach the target region of the compressed L4 nerve root were determined based on calculations on three-dimensional reconstruction of CT images obtained for diagnosis. An 18-gauge needle was placed between the L4 nerve root and the posterior border of L4. Subsequently, a 6-mm skin incision was performed and a cannula was placed at the level of L3/L4, under fluoroscopic guidance. A 3.2-mm flexible drill was used to remove part of the posterior border of the L4 vertebra, along the direction of the affected nerve root. The freed position of the decompressed L4 nerve root in the epidural space is shown in **Figure 3**. Before terminating the procedure, the patient was asked to confirm relief of his radicular pain. The working channel and scope were removed, pressure was held on the 6-mm incision for 5 min and the wound was closed with a single interrupted suture.

instrumented fusion, was not considered to be a feasible approach, carrying a high risk of further fracture and destabilization of the spinal segment. Therefore, a minimally invasive transforaminal endoscopic approach was used to decompress the L4 nerve root.

Non-steroidal anti-inflammatory drugs (Parecoxib Sodium, 40 mg) were administered as a single intra-muscular dose 30-min prior to surgery, with repeated doses administered during the surgery for pain relief. The patient was placed in a prone position on a radiolucent operating table. The surgery was performed under local anesthesia (8-10 mL of 1% lidocaine), allowing the patient to communicate with the surgeon throughout the procedure. The Joimax TESSYS endoscopic system was used for the procedure. Percutaneous entry was established through the skin, 11 cm lateral

Postoperative course

A repeat CT performed at 1-week postoperatively confirmed absence of any compression on the L4 nerve root (**Figure 4**). Preoperative leg visual analog scale (VAS) score was 8 point, and Oswestry disability index (ODI) score was 72 point. VAS and ODI score was 2 and 14 at one week postoperative follow-up, and 0 and 4 at one year postoperative follow-up. The VAS and ODI scores improved from preoperative levels, and there was no occurrence of postoperative complications. At 1-year after the endoscopic procedure, the patient had no clinical symptoms of L4 compression and had returned to his activities without pain. Publication of this case report was approved by the Medical Ethics Committee of our Hospital, and informed consent was obtained from the patient.



Figure 4. One week postoperatively, CT confirmed that a part of posterior border of L4 vertebrae were successfully removed and traveling nerve root of L4 (red arrow) was completely exposed. A. Locator; B. Coronal CT.

Discussion

Cement extravasation into the spinal canal, causing compression of the spinal cord and/or nerve roots and ischemic injury, is a well-recognized complication of PKP resulting in acute neurological deficit. In this case, we further demonstrate that fracture of the superior articular process is also a potential cause of acute neurological deficit after PKP. As the fracture is not discernable on plain radiographs and is not associated with cement extravasation into the spinal canal, this fracture can be easily omitted as a cause of neurological symptoms. Our case also demonstrates that open laminectomy, which is recommended for decompression of the spinal cord and/or nerve roots after PKP [3, 10-12], may not be suitable for patients with severe osteoporosis, with the risk of further fracture, destabilization of the spinal segment and further deformity in presence of severe osteoporosis. We describe the satisfactory clinical outcomes that we obtained using transforaminal endoscopic surgery to decompress the L4 nerve root in our patient. To our knowledge, this is the first case describing the successful use of endoscopic surgery to treat a nerve root compression after PKP.

Removal of the superior articular process of L4 would have directly decompressed the L4

nerve root. However, observation of the fracture indicated incomplete healing. Therefore, removal of the superior articular process with drilling carried the risk of displacing the fracture fragment and, therefore, of further iatrogenic neurological injury. To minimize this risk, we partially removed the posterior border of L4 vertebra to indirectly decompress the nerve root. The patient's pain improved immediately after surgery, with no further complications and the patient remaining pain free at 1-years post-surgery.

Our minimally invasive transforaminal endoscopic surgery could also be used to treat compression of neural

structures caused by retropulsion of pre-existing fracture fragments or cement extravasation into the spinal canal after PKP. Wagner et al. [12] have reported on one case in which they used transforaminal endoscopic surgery to successfully remove cement extravasation and decompress the L2 nerve root after PKP. With continued aging of the population worldwide, the incidence of osteoporosis is increasing and, consequently, the prevalence of spinal compression fractures. As PKP is the treatment of choice to treat these fractures, endoscopic surgery could provide a feasible alternative to open laminectomy to manage acute compressions of the spinal cord and/or nerve root associated with PKP in these patients. Certainly, the limitations of transforaminal endoscopic surgery need to be recognized. Foremost, the technique is not suitable for intradural cement leakage or for large extravasation of cement into the spinal canal. The publication of future reports on the use of transforaminal endoscopic surgery for the treatment of spinal cord and nerve root is encouraged to more fully evaluate clinical outcomes and indications.

Disclosure of conflict of interest

None.

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