

Cauda equina syndrome without motor dysfunction following lumbar spinal stenosis surgery

A case report

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Abstract

Rationale: Cauda equina syndrome (CES) refers to a group of symptoms that occur when the nerves in the cauda equina become compressed or damaged. The most common etiology of CES is lumbar intervertebral disc herniation, but CES following lumbar spinal surgery is rare, especially without motor dysfunction. Herein, we illustrate a case of CES that developed as a complication of spinal surgery and to deduce its possible underlying cause.

Patient concerns: A 46-year-old man experienced lumbago, bilateral shank pain, and numbness with neurogenic claudication for 3 years due to degenerative lumbar disc herniation and spinal cord stenosis. After a thorough examination to diagnose lumbar spinal stenosis, the patient underwent bilateral decompression and pedicle screw system internal fixation with bone graft. Postoperatively, the patient showed regained strength in his bilateral shanks, and he did not complain of lumbago and shank pain, but CES occurred, which manifested as underpants-type numbness in the perineum without bladder, anal, and motor dysfunction.

Diagnoses: CES as a postoperative complication of lumbar stenosis.

Interventions: The patient underwent bilateral laminectomies, partial facetectomies, and pedicle screw system internal fixation and fusion with bone graft. Postoperatively, the patient performed adequate rehabilitation exercises and was expected to recover spontaneously.

Outcomes: The symptoms of pain and claudication resolved after 3 weeks in the hospital, but an underpants-type hypoesthesia in the perineum without motor dysfunction developed. The patient experienced full recovery from CES 6 months after surgery.

Lessons: CES as a complication of lumbar spinal surgery is very rare. Excessive sensitivity to the traction of the dural sac was, in our opinion, the most possible cause of postoperative CES in this case. When the nerve root is pulled intraoperatively, it is best not to cross the central line of the spinous process. The plane of the nerve retractor needs to be parallel to the dural sac at the pulling point to reduce the formation of shear force. Most importantly, gentle maneuver is required because sensitivity to the traction of the dural sac varies individually.

Abbreviations: CES = cauda equina syndrome, MRI = magnetic resonance imaging.

Keywords: cauda equina syndrome, complication, lumbar spinal stenosis, postoperative

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The patient provided written informed consent for this report, and his information has been anonymized.

The authors have no conflicts of interest to disclose.

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1. Introduction

Cauda equina syndrome (CES) is a group of symptoms that occur when nerves in the cauda equina (a collection of nerve roots that spread out from the bottom of the spinal cord) become compressed or damaged. These nerve roots connect the central nervous system and peripheral nervous system. Clinically, symptoms and signs of CES include low back pain, saddle anesthesia, unilateral or bilateral sciatica, and motor weakness of the lower extremities with bladder and bowel dysfunction.^[1–3] It is usually caused by the compression of the spinal cord, such as due to disc herniation,^[4] tumors, hematoma, trauma, or spinal stenosis.^[5] Ischemia can also play a significant role.^[6,7] However, it is very rare that CES occurs as a complication of lumbar spine surgery, especially without motor dysfunction. Herein, we present a case of CES that occurred as a complication of spinal surgery and deduce its possible underlying cause.

2. Case report

A 46-year-old man had a 3-year history of lumbago, bilateral shank pain, and numbness with neurogenic claudication. Neurologic examination demonstrated that numbness involved bilateral shanks. On examination, the tibialis anterior and extensor hallucis longus muscle were grade III, bilateral patellar tendon reflex were weakened, and bilateral straight-leg raising test was positive, while bilateral ankle reflexes were absent. The skin sensation of the perineum was normal. Magnetic resonance imaging (MRI) of the lumbar spine confirmed protrusion of the intervertebral disc at L3/L4 and L4/L5 levels (Fig. 1A and B), and on axial T2-weighted MRI, the L4 nerve root was compressed due to disc herniation, the herniation of the L4/L5 disc was impinging the dural sac, and the L5 nerve root was stenosed (Fig. 1C and D)

After failed conservative treatment, the patient underwent bilateral laminectomies and partial facetectomies with decompression of the L3/L4 and L4/L5 segments, pedicle screw system internal fixation, and fusion with bone graft.

Postoperatively, the patient showed regained strength of his shanks, and the pain and claudication resolved 3 weeks since admission, but he experienced underpants-type numbness in

the perineum. His urination and defecation function were normal after urinary catheter was removed, which means that his bladder and anal functions were normal. He did not show signs of motor dysfunction either. Postoperative MRI showed no obvious malposition of the pedicle screw or disc components that may compress the dural sac and nerve root. The cauda equina could not be observed well because of the artifacts made by the pedicle screw in the sagittal image (Fig. 2A and B). Axial T2-weighted MRI showed that the L4 nerve root (Fig. 2C) was released, and the spinal canal of L4/L5 level was unobstructed (Fig. 2D), which means that the decompression was adequate (Fig. 2).

X-ray and three-dimensional computed tomography evaluations revealed that the internal fixation of pedicle screw system is in the pedicle area with no displacement to the spinal canal, which means that the cauda equina was not compressed by the pedicle screw (Fig. 3).

After the CES was confirmed, the patient received the following treatment: 5.4 mg/kg·h of methylprednisolone (intravenous) for 2 days, 5 mg of dexamethasone (intravenous) every 12 h for 3 days, and 0.5 mg of mecobalamin tablets (oral) every 8 h. Meanwhile, the patient underwent physical therapy. Blood cell and differential counts, erythrocyte sedimentation rate, C-reactive protein,

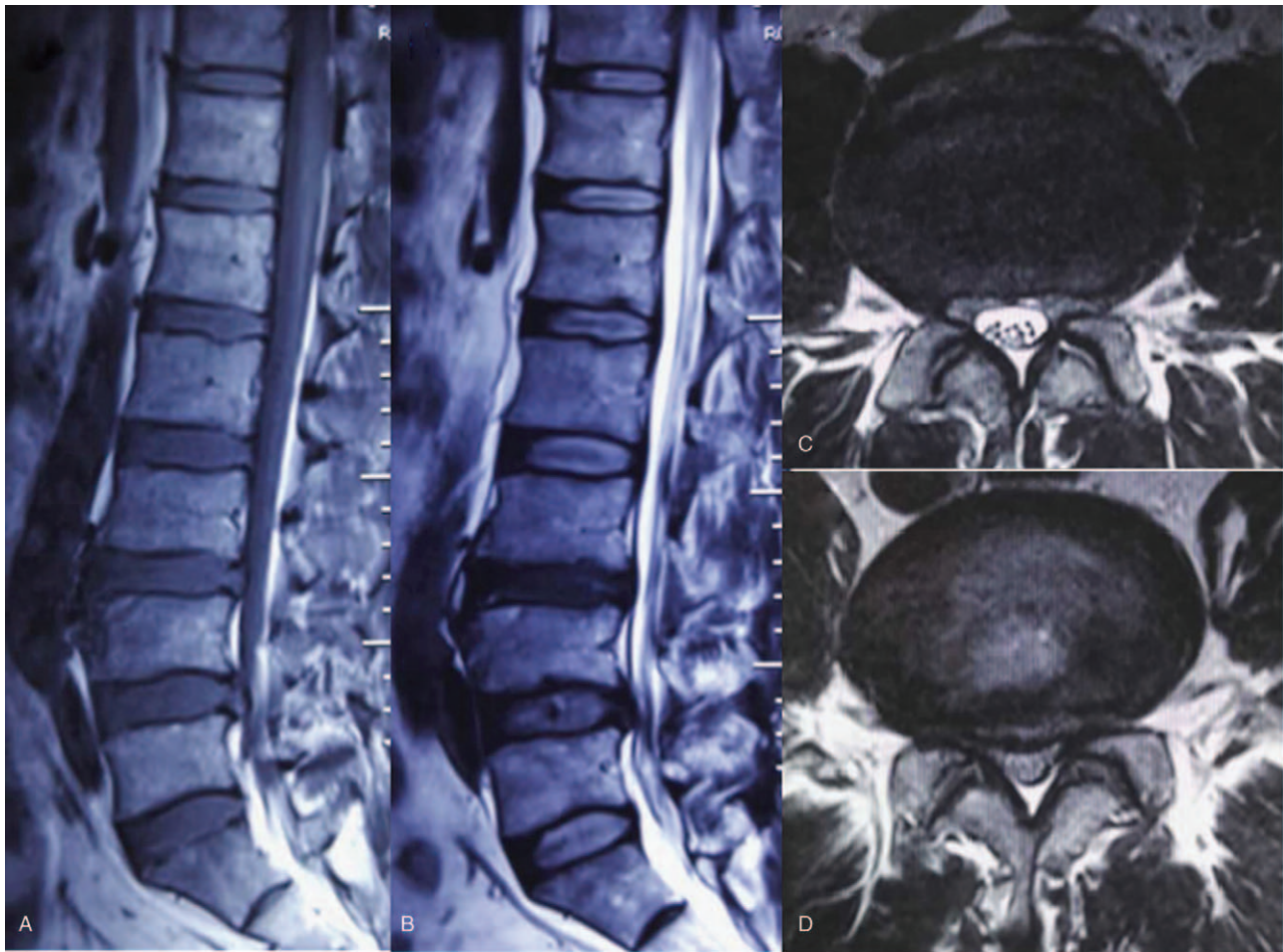


Figure 1. (A) Preoperative T1-weighted MRI. (B) Preoperative T2-weighted MRI demonstrating disc herniation of L3/L4 and L4/L5 levels that compressed the dural sac. (C) Axial T2-weighted MRI demonstrating spinal stenosis at the L3/L4 level. The L4 nerve root was compressed by the herniated disc. (D) Axial T2-weighted MRI demonstrating the herniation of the L4/L5 disc impinging the dural sac and the L5 nerve root had stenosis. MRI=magnetic resonance imaging.

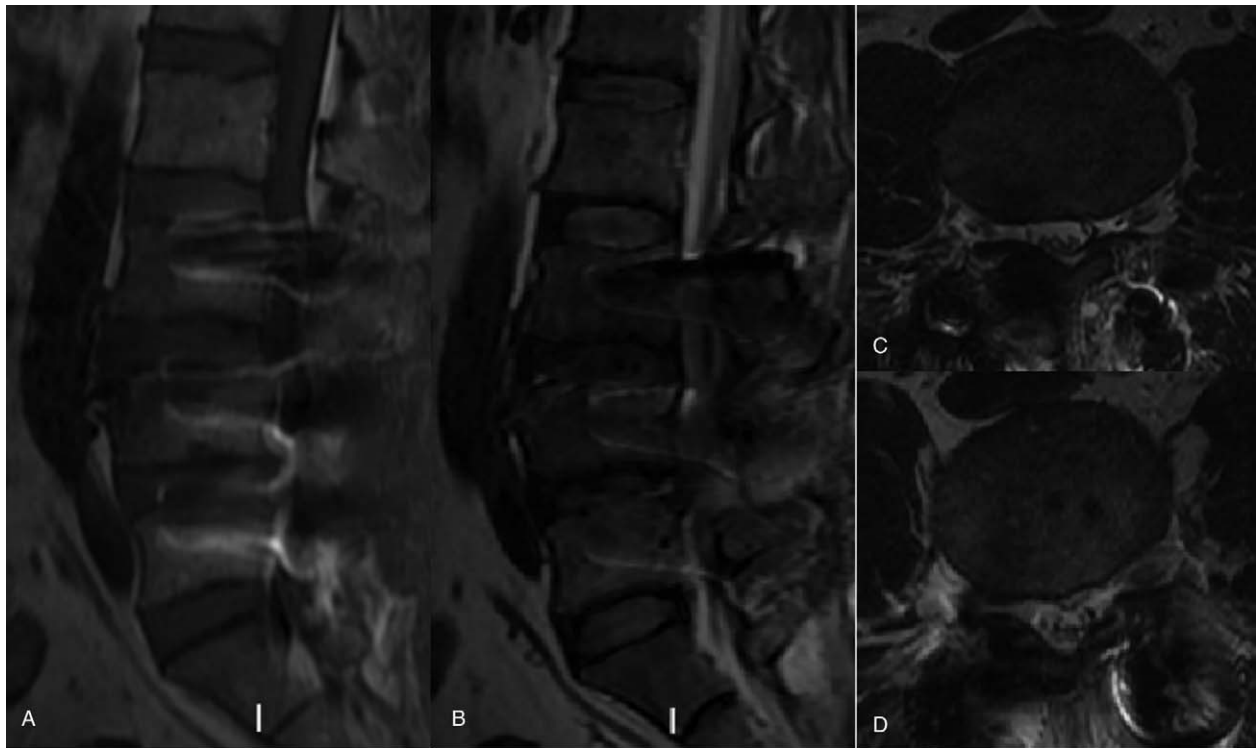


Figure 2. Postoperative MRI was performed to confirm whether the dural sac was compressed or not. (A) Postoperative T1-weighted sagittal MRI. (B) Postoperative T2-weighted sagittal MRI. (C and D) Axial T2-weighted MRI demonstrating that the spinal canal was unobstructed, and there was no compression to the nerve root and dural sac at L3/4 (C) and L4/5 segment (D). MRI=magnetic resonance imaging.

urine test, plasma electrolyte levels, and liver function panel tests were all normal. Two weeks after the operation, the patient's wound was in the primary healing stage. Six months after his operation, his numbness in the perineum was improved.

3. Discussion

Normally, CES occurs when the dural sac is compressed, such as in case of disc herniation and long period of stenosis. Immediate decompression was considered the best solution.^[8] However, few

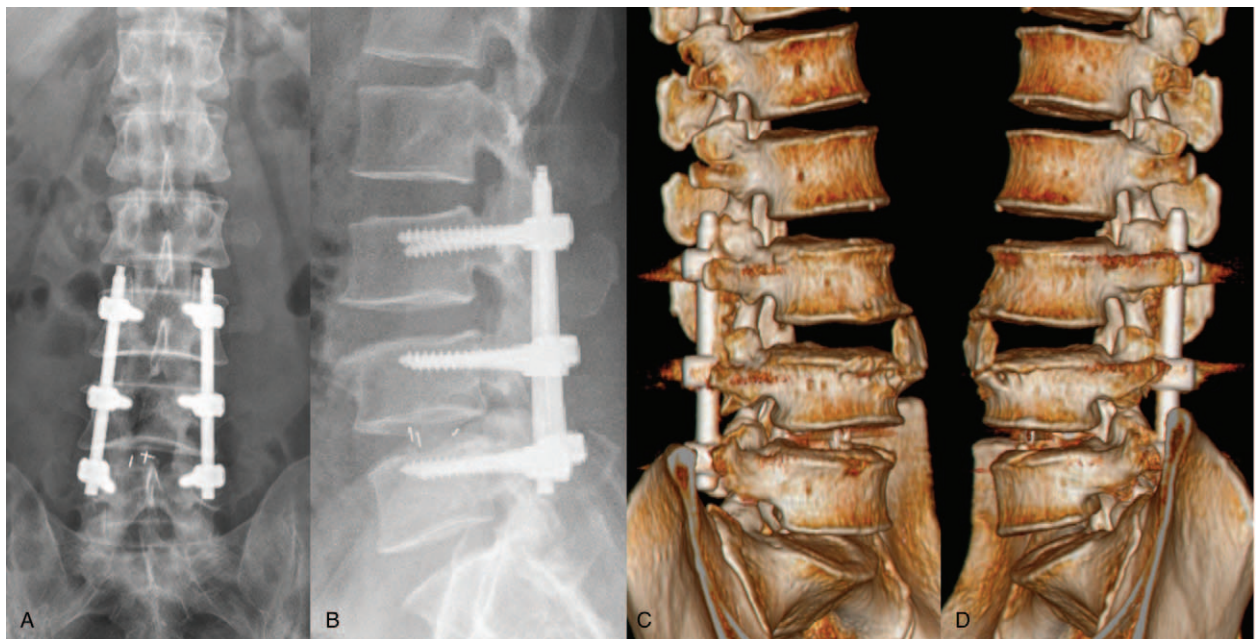


Figure 3. Postoperative X-ray (A and B) and 3D-CT (C and D) scan showing pedicle screw system internal fixation in the pedicle area with no displacement to the spinal canal. 3D-CT=three-dimensional computed tomography.

reports revealed that CES is a complication of spinal surgery, with reported incidence of 0.2%^[8] to 2.8%.^[9] To our knowledge, in the past 5 years, there is no published literature on CES as a complication of lumbar spinal surgery, and the present case is the first of its kind. Usually, numbness in the perineum and motor dysfunction are the characteristic features of CES after lumbar spinal surgery; however, in the presented case, the patient only experienced numbness.

Several factors have been suggested to cause postoperative CES, such as retained disc fragments,^[10] hematoma,^[11] epidural fat graft,^[11] anesthetic agent use,^[12] and a retained operation sponge.^[13] Vascular origin might also play an important role.^[6,7]

McLaren and Bailey^[8] reported 6 cases of CES following lumbar spinal stenosis, and all patients underwent discectomy through a keyhole interlaminar approach. Five of the 6 cases had coexisting bony spinal stenosis at the level of the disc protrusion. The bony spinal stenosis was not decompressed when discectomy was performed. All 5 patients underwent second operation to decompress the dural sac, and the resulting CES had different levels of recovery. This finding hints that inadequate decompression may have been the cause of CES after lumbar surgery.

Prusick et al^[11] reported 2 cases of postoperative CES following the placement of a free epidural fat graft. One patient underwent performed routine lumbar discectomies and developed CES on postoperative day 2, and the patient manifested urinary retention, motor weakness, and numbness in the perineum. Postoperative imaging studies showed that spinal canal compromise was caused by the fat graft. Jensen^[5] also reported 1 patient who underwent fat graft because of incidental durotomy, and the patient reported urinary retention and numbness in the perineum, but postoperative images show no remarkable injury to the spinal canal. This case suggests that if fat graft compresses the spinal cord, postoperative CES may occur.

Vascular origin might be mentioned when patients presented with symptoms consistent with CES, but there was no demonstrable compressive lesion in imaging studies. But this had some experiment support. Parke et al^[6] report a regional hypovascularity below the level of the conus medullaris. In other words, the vascular supply to the cauda equina is tenuous, which can lead to regional neuroischemia. Furthermore, Parke^[7] discusses the mechanism of ischemic neuropathy, that is, in case of pathologic disorders of the spinal cord, venous return phase is vulnerable. In the present case, venous return may have been impaired or the vascular supply to the cauda equina was insufficient.

CES as a complication of lumbar spinal surgery is very rare; thus, urgent imaging studies are necessary to identify whether it was due to a secondary compression. Mechanical compression, such as by retained disc fragments, hematoma, fat graft, or Gelfoam, can play an important role in CES following spinal surgery. The surgeon must try to avoid occurrence of these secondary compressions. Inadequate decompression during spinal surgery may lead to CES; thus, full awareness of the patients' condition preoperatively can help avoid this pitfall.

As a summary, the patient who suffered from lumbago, bilateral shank pain, and numbness with neurogenic claudication underwent spinal surgery, which improved his symptoms; however, CES occurred, presenting as an underpants-type

numbness in the perineum. Bladder and anal functions were normal, and there was no motor dysfunction. After 6 months of systematic physical and neurotrophic treatment, the numbness in the saddle area was improved well. Inadequate decompression and residual compression factor was reported to be the main causes of postoperative CES. However, postoperative imaging examination did not show residual compression and decompression was adequate. Excessive sensitivity to the traction of the dural sac was, in our opinion, the most possible cause of postoperative CES in the present case. To avoid this complication, when the nerve root was pulled intraoperatively, it is best not to cross the central line of the spinous process and the plane of the nerve retractor needs to be parallel to the dural sac at the pulling point to reduce the formation of shear force. Moreover, gentle maneuver is required because each patient has different sensitivities to traction of the dural sac.

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