

Postoperative Cauda Equina Syndrome in Trivial Lumbar Congenital Kyphosis: A Case Report

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Abstract- A 25-year old man presented with chronic low back pain for about 5 years due to mild congenital lumbar kyphosis (L1-L3 25° with congenital posterior wedge vertebra L2). Preoperative neurologic examination was normal. After posterior spinal fusion and instrumentation with moderate curve correction, the patient gradually developed the symptoms and signs of cauda equina syndrome due to intraoperative L2-3 disc herniation. After 5 days the patient underwent posterior decompression surgery and on the latest follow up visit at 2 years later, nearly all the motor power was recovered but the patient complained of occasional urinary incontinence and residual right leg paresthesia. In surgical treatment of congenital kyphosis, much attention should be paid to the presence of contemporaneous asymptomatic disc herniation.

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Introduction

Postoperative neurologic injury always is a fearful complication that may occur with nearly every spinal interventional surgery. This complication is more common after complicated or revision spinal surgeries and maybe caused by technical or tactical (planned) errors. The first is most commonly occurred when the neural elements are injured by inappropriate instrumental insertion, handling or direct neural injury due to inappropriate surgical technique. These happenings are usually occurred intraoperatively but tactical failures occurred when the whole surgical planning is incorrect. Although neurologic deficit associated with congenital kyphosis is not an uncommon entity (1-5) especially in unstable type 1 lesions (6), intraoperative lumbar disc herniation during posterior spinal fusion for correction of trivial congenital lumbar kyphosis has not previously been reported in the literature. In this study, we report a case of a 25 years old man with trivial congenital lumbar kyphosis who developed intraoperative lumbar disc herniation during posterior lumbar fusion and instrumentation for kyphosis correction.

Case Report

A 25-year old man who was a medical student presented

with chronic low back pain for about 5 years. He complained from pain that was aggravated after prolong standing and walking. He had no history of significant previous trauma or underlying disease. In physical examination, he had normal standing posture without any apparent scoliosis, kyphosis or truncal imbalance. Posterior midline stigmata (*e.g.* nevus, hemangioma) were not present and neurologic examination was completely normal. Plain radiography and magnetic resonance imaging (MRI) were shown in figure 1 and 2.



Figure 1. Preoperative standing anteroposterior, lateral and supine fulcrum hyperextension lateral radiographs of lumbosacral spine.

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Cauda equina syndrome after kyphosis correction

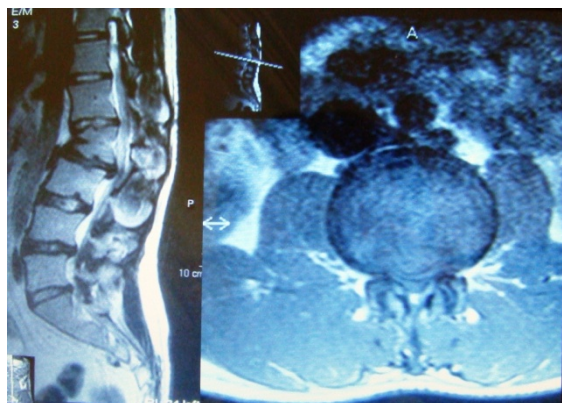


Figure 2. Preoperative MRI of the lumbosacral region.

Standing preoperative radiographs revealed mild congenital lumbar kyphosis (L1-L3 kyphosis: $+27^\circ$ with congenital L2 posterior wedge vertebra) that was corrected to $+15^\circ$ on supine fulcrum hyperextension lateral film. Preoperative MRI was initially reported as normal.

Due to the refractory pain and trivial severity of the flexible kyphosis, the posterior only spinal surgery was suggested. After the patient signed the informed consent, the posterior spinal correction, arthrodesis and instrumentation from T12 to L3 were carried out. All the pedicular screws were inserted under biplane fluoroscopic control and the kyphosis was moderately corrected to $+7^\circ$ (Figure 3). Because of the tiny severity, great flexibility and lumbar location of the deformity, motor evoked potentials (MEP) and somatosensory evoked potentials (SSEP) were not carried out but wake-up test was performed that it was normal bilaterally (spontaneous movement of both lower extremities). The duration of the surgery was 110 minutes without any significant intraoperative complication. The amount of blood loss was 340 milliliter and blood transfusion was not necessary at all.

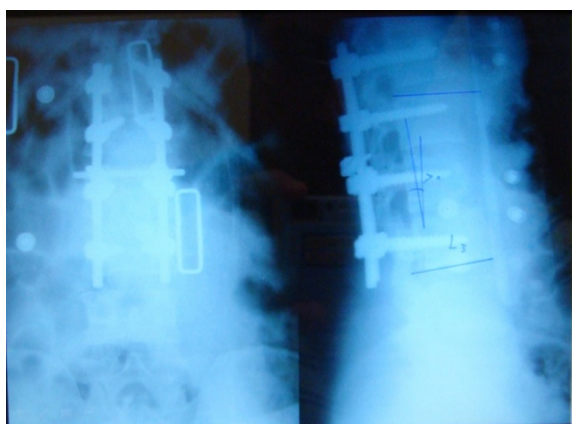


Figure 3. postoperative anteroposterior and lateral standing radiographs of lumbosacral spine.

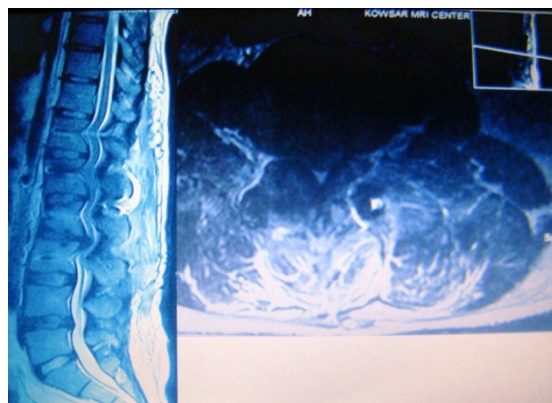


Figure 4. Postoperative MRI showed a L2-L3 sequestered disc that completely obstructed the central spinal canal and cause cauda equina syndrome.

Immediately after surgery, the patient complained from right leg paresthesia. It was thought to be related to the compression of lateral cutaneous nerve of thigh during prone positioning of the surgery and the patient was discharged 2 days after operation with rigid lumbosacral orthosis. 2 days later, he returned due to fall while walking. He reported that the cause of falling was the severe weakness of the lower extremities muscles. In physical examination, motor power of the right lower extremity was diminished to about 2/5 in all the muscles distal to the knee and 3/5 in the proximal. Motor power of left lower extremity was also diminished (4/5 in proximal and distal muscles). The patient had diminished sensation to pinprick and light touch in the saddle area and both legs especially in the right side. He complained of urinary retention and bowel incontinence. The patient was admitted again and complete bed rest and corticosteroid therapy were ordered and radiography and MRI scanning requested.

Plain radiography was normal but MRI scanning revealed relative severe L2-L3 disc herniation without any apparent epidural hematoma in the operated field (Figure 4).

When we retrospectively reviewed MRI scans and plain radiographs of the patients before and after surgery, we noticed that in addition to congenitally stenotic spine (short pedicles in plain radiography, central and lateral recess spinal stenosis throughout the lumbar spine in MRI), a subclinical herniated L2-L3 intervertebral disc (protrusion stage) was also present before surgery that after operation with forceful kyphosis correction, it was deteriorated to the extrusion stage and cause full-blown cauda equina syndrome.

By postoperative day 5, the patient was operated again and complete posterior decompression (from pedicle to pedicle) was carried out with total L2 and L3

laminectomy and L2-3 bilateral discectomy.

After the second operation, the patient self perception of leg weakness and paresthesia was improved significantly and on the 4th day, he could stand and walk with aid. On the latest follow up visit at 2 years later, nearly all the motor power of both lower extremities muscles was recovered to more than 4⁺/5 but the patient still complained of occasional urinary incontinence and residual right leg paresthesia. The radiologic correction loss was about 4 degrees.

Discussion

Although the prevalence of congenital kyphosis is less than congenital scoliosis, coincidence of neurologic deficit is more common. Progressive neurologic deficit because of the compression of the spinal cord usually occurred only in unstable type 1 lesion (7).

According to our knowledge, postoperative cauda equina syndrome in mild lumbar congenital kyphosis of an adult case has not been reported previously. Recently, a similar case with intraoperative disc herniation during the surgical treatment of Scheuermann's kyphosis was reported by Llado *et al.*, but that case not only had a different underlying disease, but also developed a thoracic (not lumbar) disc herniation (8). In Scheuermann's kyphosis, according to the recent review of the SRS database, Incidence of acute neurologic complication from spinal cord injury is 1.9% (9). Apparently the age of the patient or the approach of the surgery does not have any remarkable effect on this incidence and most of these complications are attributed to hypoperfusion but postoperative MRI was not carried out routinely.

In other study on 7885 cases with spinal deformity operated mostly with posterior only fusion, the overall incidence of neurologic complication was 0.725% (10). In this research, Mac Ewen *et al.* reported that the patients at risk for this complication are those with preexisting neurologic deficit, kyphosis, congenital scoliosis, and great severity of the curves. In this study, there was not any implication for intraoperative disc herniation induced neurologic deficit.

The major failure of our study was the lack of motor evoked potentials and somatosensory evoked potential monitoring during the surgery for spinal deformity correction. Although the fate of this patient was not so devastating, the next one may not be so lucky. In

conclusion, in surgical treatment of congenital kyphosis, much attention should be paid to the presence of concurrent asymptomatic disc herniation.

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