Pure Conus Medullaris Syndrome without Lower Extremity Involvement Caused by Intradural Disc Herniation at L1/2: A Case Report

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Abstract:

Introduction: Conus medullaris syndrome (CMS) is a rare pathology. The conus medullaris is located at the end of the spinal cord and continues to the cauda equina. Conus medullaris lesions can cause variable symptoms and neurological deficits, usually involving the lower extremities; CMS that does not affect the lower limbs is extremely rare. No reports have described isolated CMS caused by intradural disc herniation (IDH). This report describes a case of CMS without lower extremity involvement associated with IDH at L1/2.

Case Report: A 52-year-old man with a 10-year history of lower back pain complained of dysuria and lumbago with no leg symptoms at his first visit to the urology department. Neurological examination revealed mild perineal hypoalgesia; however, motor function and lower extremity sensation were normal with except for left ankle dorsiflexion weakness (manual muscle test, 4/5). Magnetic resonance imaging revealed conus medullaris compression by a mass, continuous with the L 1/2 disc, and severe spinal canal stenosis at vertebral levels L3/4 and L4/5. Postmyelographic computed tomography indicated direct conus medullaris compression by an intradural and extramedullary mass continuous with the L1/2 disc. Without recovery of his dysuria, the patient underwent surgery, including partial laminectomy of the L1/2, incision of the dura mater, and removal of the herniated disc. Immediately after surgery, his dysuria completely resolved. More than one year post-operatively, the patient remained active with no change in his neurological condition.

Conclusions: Although CMS without lower limb symptoms is extremely rare, we experienced an isolated case of CMS associated with IDH causing direct conus medullaris compression. Without lower extremity involvement, the CMS diagnosis was relatively easy. Surgical treatment for CMS without lower extremity involvement caused by IDH was effective. **Keywords:**

conus medullaris syndrome, intradural disc herniation, lumbar spine, disc degeneration, urinary disturbance, dural degeneration, lower back pain, cauda equina syndrome

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Introduction

Conus medullaris syndrome (CMS) without lower extremity involvement is extremely rare. Anatomically, the conus medullaris (CM) is located at the terminal spinal cord and surrounded by the nerve rootlet^{1,2)}. Therefore, CM lesions due to compression commonly cause lower extremity symptoms³⁻¹¹⁾. We describe a case of CMS caused by intradural disc herniation (IDH) of the L1/2 disc.

Case Report

A 52-year-old man who had been a truck driver for >30 years and experienced lower back pain for the past 10 years complained of acute-onset dysuria and lower back pain but no leg symptoms at his first visit to the urology department. Neurological examination revealed mild perineal anesthesia, but lower extremity motor function and sensation were normal except for left ankle dorsiflexion weakness (manual muscle test, 4/5). The bilateral straight leg raise test and femoral nerve stretch test were negative, and deep tendon reflexes were absent. Plain radiography showed severe spon-

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dylotic changes (Fig. 1). Magnetic resonance imaging (MRI) revealed CM compression with a mass extending from the discontinuous boundary line behind the L1/2 intervertebral disc and severe lower lumbar spinal canal stenosis (LSCS). The mass showed low intensity on T1 imaging and isointensity to high intensity on T2 imaging (Fig. 2A-D). Computed tomography myelography indicated that an intradural and extramedullary mass, continuous with the L1/2 disc, was



Figure 1. Lateral-plane roentgenogram showing severe degenerative changes.

compressing the CM (Fig. 3A-C).

Pure CMS associated with IDH at the L1/2 disc was suspected. Without recovery of urinary function, the patient underwent surgery. A partial laminectomy of L1/2 revealed no abnormality in the epidural space, but dura tightness was found. The cartilaginous endplate fragment, and other fragments of the nucleus pulposus within the nerve rootlet, were removed via a dura mater incision (Fig. 3D). Immediately postoperatively, the patient's urinary disturbance and perineal anesthesia completely resolved with no change in his leg muscle strength. More than one year postoperatively, the patient remained active with no change in his neurological condition. A postoperative MRI indicated the IDH compressing the CM at L1/2 had disappeared (Fig. 4).

Discussion

CM compression from outside the dura mater causes volume-dependent neurological symptoms; a large volume of material is unlikely to compress only the CM without the nerve rootlet, and a very small volume is likely to be asymptomatic. Therefore, direct compression by a relatively small volume of soft material may be all that is necessary to influence the CM. Additionally, the threshold of compression-related symptom expression may be lower in the CM than in the nerve rootlet.

IDH comprises only 0.26% to 0.30% of lumbar disc surgeries, and occurrence at the L1/2 level is extremely rare^{3,12,15)}. Although IDH's etiopathogenesis is unknown, previous reports indicated adhesion of the anterior dura and posterior longitudinal ligament (PLL) facilitate disc herniation into the dural sac^{4,6,13-16}. Adhesion causes include chronic

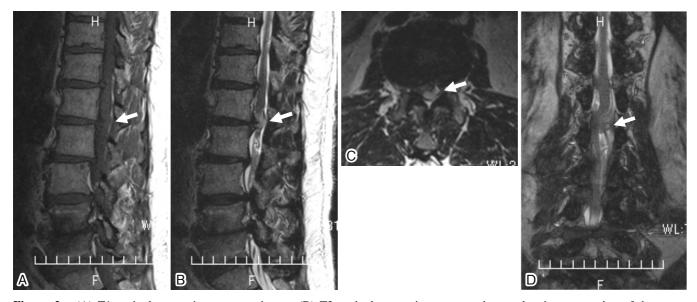


Figure 2. (A) T1 sagittal magnetic resonance image. (B) T2 sagittal magnetic resonance image showing occupation of the conus medullaris region with a mass continuous from discontinuity of the boundary line behind the L1/2 intervertebral disc and severe lower lumbar spinal canal stenosis. (C) Axial magnetic resonance image at L1/2 showing occupation of the mass from the left side to the dorsal side of the spinal canal. H, head; F, foot. (D) Coronal magnetic resonance image showing compression of the conus medullaris from the left side by the mass. The white arrows indicate the mass with low intensity on T1 imaging and isointensity to high intensity on T2 imaging.

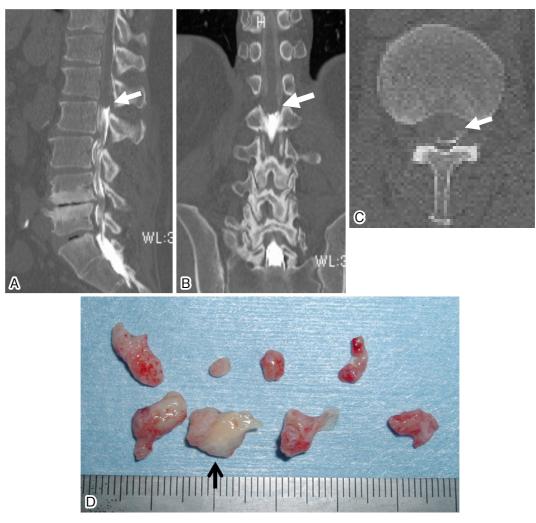


Figure 3. (A) Sagittal computed tomography myelography image showing block of contrast medium and cup-like shape. (B) Coronal computed tomography myelography image showing block of contrast medium and cup-like shape. (C) Axial computed tomography myelography image showing a half ring-like shape. (D) Photograph of herniated fragments collected during surgery. The black arrow shows a fragment of the cartilage endplate. Other fragments were soft nucleus pulposus. Scale bar = 1 mm.

inflammation and degenerative changes. In the present case, the degenerative lumbar spine and history of lower back pain were consistent with previous reports^{3,4,6,7,12-16}. Moreover, the cartilage endplate in the herniated disc is thought to have penetrated the dura and PLL.

CM lesions usually cause leg symptoms and are thus difficult to distinguish from cauda equina syndrome (CES)¹⁸). In this case, the MRI showed lower LSCS. However, there were no usual symptoms in the lower extremity (e.g., pain, paresthesia, or gait disturbance) or obvious neurological deficits associated with CES, which most commonly occurs acutely by a massive prolapsed disc herniation¹⁸) or IDH^{4,6,12-17}). Additionally, it is far more anatomically difficult for lower LSCS than IDH in the CM region to selectively cause urinary disorder and perineal analgesia. Therefore, together with the acute onset, it was reasonable to diagnose pure CMS caused by L1/2 IDH in the present case. Despite the improvement in the patient's micturition disorder postoperatively, his muscular weakness did not improve; this is inconsistent with past reports showing that patients with CMS or CES due to IDH with improved urinary function also exhibit improved lower limb function^{3,4,6,13-15)}. Therefore, the muscular weakness was thought to have worsened due to chronic compression by the lower LSCS.

Preoperative diagnosis of IDH is difficult, and the diagnosis is often only made during surgery¹⁴; however, multiple image analyses should enable some form of preoperative assessment^{4,6,7,12,15,16,19-23}. Therefore, it is crucial to fully consider IDH. As in past reports^{3,4,6,8}, we obtained excellent results with surgical decompression for CMS due to IDH in the present case. Thus, surgery should be promptly performed if no improvement occurs following conservative treatment.

Conflicts of Interest: The author declares that there are no relevant conflicts of interest.

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Figure 4. Postoperative magnetic resonance image shows disappearance of the intradural disc herniation compressing the conus medullaris at L1/2.

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