



## Case Report

# Lumbar spinal stenosis in a patient with complex spinal dysraphism caused by a supplementary midline muscle: A case report

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## ABSTRACT

**Background:** The clinical tethered cord syndrome (TCS) can become symptomatic during adulthood, known as adult tethered cord syndrome (ATCS). Distinguishing ATCS from neurogenic claudication attributed to lumbar spinal stenosis may pose a clinical challenge.

**Case Description:** A 66-year-old male with an underlying complex occult spinal dysraphism (OSD) presented with new onset of lower back and bilateral leg pain plus neurogenic claudication. Magnetic resonance imaging documented OSD, and lumbar spinal stenosis (LSS) attributed to a supplementary midline muscle. Following decompressive surgery for LSS without untethering the ATCS, the patient's symptoms resolved.

**Conclusion:** A patient with OSD and ATCS with LSS due to a supplementary midline muscle presented with new onset of neurogenic claudication. Surgical decompression of the LSS by removing the supplementary midline muscle resolved patients' symptoms.

**Keywords:** Decompressive surgery, Neurogenic claudication, Spinal dysraphism, Spinal stenosis, Supplementary midline muscle, Tethered cord

## INTRODUCTION

Clinically, occult spinal dysraphism (OSD) may be associated with the clinical tethered cord syndrome (TCS) which includes pain, symptoms of motor and sensory loss in the lower extremities, and bladder dysfunction.<sup>[1]</sup> Although less common, TCS can become symptomatic during adulthood (ATCS).<sup>[2]</sup> As the signs and symptoms of ATCS (adult tethered cord syndrome) are relatively nonspecific, distinguishing ATCS and neurogenic claudication in the case of concurrent lumbar spinal stenosis, or vice versa, may pose a clinical challenge. Here, we describe a patient referred to our center with a complex OSD with the complaints of pain and muscle weakness exacerbated by walking with magnetic resonance imaging (MRI) revealing a lumbar spinal stenosis (LSS) due to a supplementary midline muscle.

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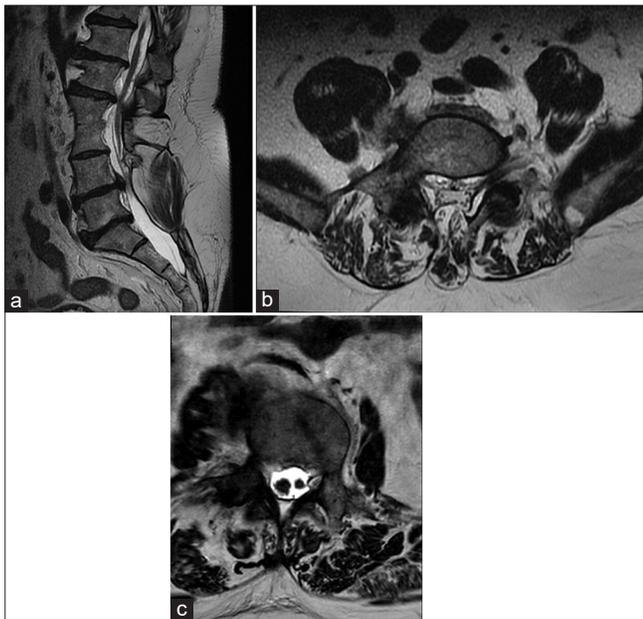
## CASE REPORT

### Clinical presentation

A 66-year-old male presented with neurogenic claudication characterized as lower back and bilateral leg pain. The patient had a known complex OSD that included ATCS and multiple other congenital anomalies. Consequently, the left leg was shortened and atrophied. Further neurological examination revealed diffuse weakness 4/5 in both legs, absence of the left patellar and Achilles responses, and an unsteady gait.

### Imaging

MRI of the lumbar spine revealed OSD in combination with spondylotic changes [Figure 1]. Fusion of the L2 and L3 vertebrae, hemivertebrae of L4 and L5, unfused spinous process L2, an interspinous lipoma at the level of L3, split cord malformation (type 2), and a tight filum were observed in relation to OSD. A supplementary midline spinal muscle at L5 extended into the spinal canal through the hemivertebrae of L4 and L5. The study also showed a herniated disc at level L4/L5 and hypertrophy of facet joints and ligamentum flavum contributing to significant stenosis/spondylosis from L4/L5.



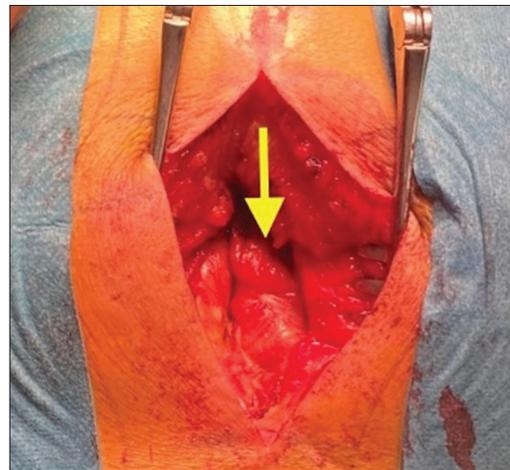
**Figure 1:** (a) Preoperative sagittal MRI T2 scan showing fusion of the L2 and L3 vertebrae, hemivertebrae of L4 and L5, unfused spinous process L2, an interspinous lipoma at the level of L3, and a tethered cord. (b) Preoperative axial MRI T2 scan L4-L5. The midline muscular abnormality causes spinal cord compression. Furthermore, a left foraminal stenosis is seen. (c) Preoperative axial MRI T2 scan L1 showing diastematomyelia of the spinal cord.

### Surgery

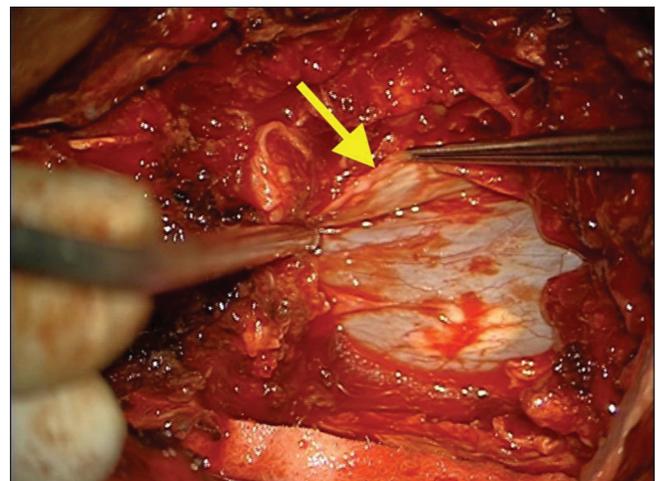
The spinal canal was approached from the left side through a L4/L5 hemi-laminectomy [Figure 2]. The fascia of the midline muscle (i.e., attached to the dura) had to be carefully dissected away from the underlying dura [Figure 3]. Decompression of the spinal canal was then completed following a full myomectomy and flavectomy on the right side down to the L5 level.

### Outcome

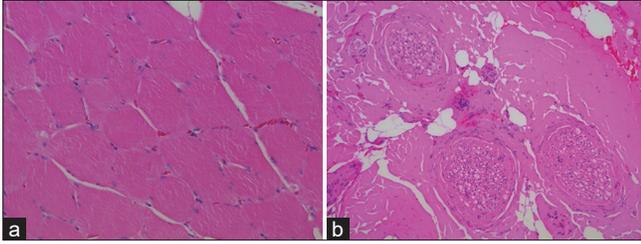
Postoperatively, the patient experienced transient complaint of a left-sided lateral femoral cutaneous nerve entrapment syndrome, which resolved spontaneously. The patient was discharged 2 days later, and 2 months later reported a significant



**Figure 2:** Intraoperative photo. Exposure of the muscular abnormality (arrow).



**Figure 3:** Intraoperative microscopic photo. After extraction of the muscular abnormality, the dura is exposed. The internal fascia sheet (arrow) is detached from the dura.



**Figure 4:** Histological aspect of excised tissue with hematoxylin and eosin (HE) stain. (a) Muscle tissue HE  $\times 200$ . (b) Muscle tissue with nerve fibers HE  $\times 100$ .



**Figure 5:** (a) Postoperative sagittal MRI T2 scan. (b) Postoperative axial MRI T2 scan L4-L5. The midline muscular abnormality is removed, showing adequate decompression of the spinal canal.

reduction in neurogenic claudication. The pathology report confirmed that the tissue removed was normal muscle tissue [Figure 4]. Postoperative MRI at 3 months revealed adequate decompression of the spinal canal [Figure 5].

## DISCUSSION

The patient in this report was known to have complex surgically untreated OSD in conjunction with multiple other congenital anomalies including ATCS and a hemivertebrae at the L4/L5 level. Due to direct muscle adherence to the dura through the L4/L5 hemivertebral defect, the patient had developed new symptoms of neurogenic claudication. Following the lysis of these adhesions and decompression of the L4/L5 lumbar canal, the patient's symptoms significantly improved (i.e., within 2 postoperative months).

## Etiology of supplementary midline muscle

The developmental mechanism of the supplementary midline muscle in our patient remained unclear. The muscle was located in midline at L5 and extended in the spinal canal through the hemivertebrae of L4 and L5. Possibly, during the development of the spine, the hemivertebrae provided extra space, which was then filled up by the supplementary muscle, or vice versa, the muscle was obstructive for the vertebrae to correctly develop.

## CONCLUSION

A 66-year-old male with OSD and multiple congenital anomalies (i.e., including a hemivertebrae at L4/L5) presented with new onset of neurogenic claudication and symptoms/signs of lumbar stenosis. As the MR revealed adherence of the midline musculature to the dura through the L4/L5 hemivertebral defect, the patient's stenosis was relieved following a lysis of these adhesions and canal decompression at the L4/5 level.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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