



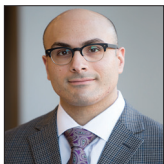
## Case Report

# Spinal cord ischemia/infarct after cauda equina syndrome from disc herniation – A case study and literature review

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## ABSTRACT

**Background:** Spinal cord infarction is rare and occurs in 12/100,000; it represents 0.3%–2% of central nervous system infarcts. Here, we present a patient who developed recurrent bilateral lower extremity paraplegia secondary to spinal cord infarction 1 day after a successful L4-5 microdiscectomy in a patient who originally presented with a cauda equina syndrome.

**Case Description:** A 56-year-old patient presented with an acute cauda equina syndrome characterized by severe lower back pain, a right foot drop, saddle anesthesia, and acute urinary retention. When the lumbar magnetic resonance imaging (MRI) revealed a large right paracentral lumbar disc herniation at the L4-L5 level, the patient underwent an emergency minimally invasive right-sided L4-5 discectomy. Immediately, postoperatively, the patient regained normal function. However, 1 day later, while having a bowel movement, he immediately developed the recurrent paraplegia. The new lumbar MRI revealed acute ischemia and an infarct involving the distal conus medullaris. Further, workup was negative for a spinal cord vascular malformation, thus leaving an inflammatory postsurgical vasculitis as the primary etiology of delayed the conus medullaris infarction.

**Conclusions:** Acute neurologic deterioration after spinal surgery which does not neurologically correlate with the operative level or procedure performed should prompt the performance of follow-up MR studies of the neuraxis to rule out other etiologies, including vascular lesions versus infarctions, as causes of new neurological deficits.

**Keywords:** Complication spine surgery, Disc herniation, Spinal cord infarct, Spinal cord ischemia

## INTRODUCTION

Spinal cord infarctions are rare and occur in approximately 12/100,000 patients,<sup>[8]</sup> accounting for only 0.3%–2% of all central nervous system infarcts.<sup>[6]</sup> Postoperative spinal cord infarctions are even more infrequently encountered and require a high index of suspicion for a spinal dural arteriovenous fistula (AVF) as noted in this case when a middle-aged male who originally presented with a cauda equina syndrome due to an L4-L5 disc herniation, acutely developed recurrent paraparesis, 1 day postoperatively following a Valsalva maneuver.

## CASE DESCRIPTION

A 56-year-old patient presented with an acute cauda equina syndrome characterized by severe lower back pain, saddle anesthesia, and acute urinary retention. Neurologic examination showed diffuse weakness

in the right leg, more so in the distal leg muscle groups and poor rectal tone.

Medical history was significant for obesity (body mass index = 34.5), type 2 diabetes mellitus, hyperlipidemia, hypertension, peripheral vascular disease (unspecified), obstructive sleep apnea, chronic kidney disease (Stage II thought to be secondary to diabetic nephropathy), and polyarticular arthritis.

The lumbar magnetic resonance imaging (MRI) demonstrated an acute right L4-L5 paracentral disc herniation contributing to the canal and right lateral recess stenosis [Figure 1]. He underwent an uneventful right-sided L4-L5 microdiscectomy. During surgery, the mean blood pressure (MBP) was maintained between 69 and 111 mmHg (mean MBP = 88.7 mmHg and median MBP = 82 mmHg).

The patient tolerated the procedure well, and postoperatively, his right lower extremity weakness markedly improved. However, 1 day postoperatively, when moving his bowels (e.g., Valsalva maneuver), he acutely developed sudden bilateral lower extremity paralysis. The emergent postoperative lumbar MR showed a subtle increased T2 signal at the L1-L2 level involving the conus medullaris; autoimmune, inflammatory, and hypercoagulation workups proved negative, but the remaining differential diagnoses included ischemia versus posttraumatic/inflammatory changes [Figure 2]. Despite the postoperative lumbar MRI scan showing no recurrent disc herniation, the patient underwent a “reexploration” of the wound that did not result in any neurological improvement. The patient did not demonstrate any further significant improvement in his neurologic deficits and was ultimately discharged to an acute rehabilitation facility with persistent severe paraparesis. Several weeks later, the follow-up MRI revealed a low T2 signal within the conus, consistent with/suggestive of hemorrhage [Figure 3].

## DISCUSSION

Our patient initially presented with a cauda equina syndrome due to an L4-L5 disc herniation. Postoperatively, he acutely neurologically improved. Nevertheless, when having a bowel movement (e.g., acute Valsalva maneuver), he suddenly became paraparetic. When the postoperative MR documented ischemia involving the conus medullaris and reexploration of the prior laminectomy site prove nondiagnostic, the conclusion was that the patient had sustained a conus medullaris infarct. Etiologies of a conus medullaris syndrome variously include neoplasm, autoimmune/inflammatory, degenerative/compressive pathology, arteriovenous malformation (AVM), dural AVE, and infarct.

### Demographic parameters for infarcts resulting in conus medullaris syndrome

Typically, patients with spinal/conus medullaris infarctions are males, in their early sixties, whom present with significant



**Figure 1:** Pre-operative lumbar MRI showing a right paracentral disc herniation at L4-L5 causing nerve root compression (arrow). Notice the patient's significant bladder distension (\*).



**Figure 2:** Sagittal T2 image of the post operative lumbar MRI showing the conus medullaris is expanded and there is new subtle increased signal. This is remote from the area of the surgery (arrow).



**Figure 3:** Sagittal views from late post operative lumbar MRI (2 weeks post surgery). T2 (left), STIR (right) showing hemorrhagic conversion in the conus medullaris with cavitation from the hemorrhage (arrow).

motor deficits and dissociated sensory loss.<sup>[4]</sup> Typically, they present after Valsalva maneuvers which transiently increase intravascular pressure within the spinal cord, thus precipitating hemorrhage into the conus and/or AVMs.<sup>[1,8]</sup> Patients with spinal cord infarctions often have hypertension, diabetes mellitus, peripheral vascular disease, and dyslipidemia<sup>[4]</sup>; however, many thoracic/thoracolumbar spinal infarctions are idiopathic.<sup>[5,7]</sup>

While the cervical spinal cord is the very well vascularized, supply to the midthoracic portion is most fragile. In this section, blood supply is merely provided through the artery of Adamkiewicz. Hypoperfusion, atherosclerosis, iatrogenic occlusion, vasculitis/vasculopathies, thromboembolic occlusion, trauma, infection, or inflammatory conditions have also been associated with spinal cord infarction [Table 1].

### Etiology of spinal cord infarction

The most frequent identifiable etiology of spinal cord infarction is occult spinal vascular malformations including AVM (most commonly found at the level of the conus medullaris) and dural AVF.<sup>[1]</sup> Either of these may not be readily apparent on the MRI and may warrant angiography.<sup>[3,5,8]</sup> In this patient, workup for an AVM, including MR angiography of the thoracic spine and contrast-enhanced computed tomography of the abdomen and pelvis, was negative. Due to the complete and continued paralysis, it was determined that formal spinal angiography would not improve the patient's outcome.

**Table 1:** Causes of spinal cord infarction.

Aortic disease
Aortic dissection
Aortic aneurysm surgery
Vertebral artery dissection
Embolism
Arterial or cardiac embolism
Fibrocartilaginous embolism
Trauma
Surfer's myelopathy
Hypercoagulable states
Sickle cell disease
Antiphospholipid syndrome
Protein S deficiency
Malignancy
AV malformations
Radicular artery compression from a disc
Vasculitis
Infection and spinal cord abscess
Drug-induced ischemia
Cocaine
Global hypoperfusion
Cardiac arrest
Decompression sickness
Idiopathic (33%–81%)

### Imaging of conus medullaris infarction

The imaging findings here were classic for spinal infarction; they included early nonspecific edema, cord swelling, and enhancement of a hemorrhagic lesion.<sup>[9]</sup> While the differential diagnosis in our patient included neoplasm, infection, inflammatory diseases, and demyelination, the diffusion-weighted MRI sequences can help confirm the diagnosis of an acute spinal cord infarction.<sup>[10]</sup> The “owl eyes” or “snake eyes” appearance of the central cord (gray matter) with a resultant high signal further establishes the diagnosis of a spinal cord infarction. This original MRI pattern results from edema but is later characterized by cord atrophy.<sup>[11]</sup>

### Postoperative infarction of the conus medullaris

Postoperative conus medullaris infarction is even more rare than *de novo* infarction. There were just two reports in literature by Stevens and Iovtchev that focused on occult dural AVF acutely diagnosed following lumbar discectomy resulting in acute spinal cord infarction at a site not associated with initial surgery.<sup>[3,9]</sup>

### CONCLUSIONS

The patient's initial symptoms correlated with his lumbar disc herniation. However, his secondary acute onset of paraparesis attributed to a Valsalva maneuver, pointed to an acute vascular event involving the conus medullaris ultimately attributed to an occult AVM with resultant acute ischemia/hemorrhage or inflammatory postsurgical vasculitis.<sup>[2]</sup>

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### Conflicts of interest

There are no conflicts of interest.

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