



## Case Report

# Distal *Cauda equina* syndrome: A case report of lumbosacral disc pathology and review of literature

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## ABSTRACT

**Background:** *Cauda equina* syndrome (CES) is an uncommon entity that presents acutely with all or some of the following symptoms; urinary incontinence from retention, fecal incontinence from loss of sphincter tone, saddle area hypoesthesia or anesthesia, and acute or progressive weakness in one or both lower extremities. The protean symptomatology is often mixed and is vulnerable to confounding comorbidities making the accurate and timely diagnosis of this syndrome uniquely challenging. Here, we present the case of a man who developed isolated sacral nerve dysfunction from CES in the midst of a diabetic crisis.

**Case Description:** A 53-year-old male with a long history of uncontrolled Type 2 diabetes presented with acute-onset urinary and fecal incontinence, scrotal anesthesia, and a 3-day history of lower back pain with intermittent bilateral leg pain. This patient displayed no objective changes in leg strength, sensation, or reflexes. In addition, the patient tested positive for cocaine and had a blood glucose level of 800 mg/dL which confounded his clinical picture. The patient underwent bilateral laminectomies from L4–S1 with the removal of a large sequestered disc fragment from the S1–S2 disc space within 8 h of presentation with reasonable recovery.

**Conclusion:** Highly variable presentations often confound the accurate and timely diagnosis of CES with severe implications on quality of life. Despite the limited functional recovery seen after surgical decompression, urgent or emergent intervention is paramount for treatment. Our patient's presenting symptomatology and comorbidities highlight the need for practitioners to maintain a high index of suspicion in anyone with incontinence and back pain, regardless of distractors and even in the absence of other anticipated motor or sensory findings.

**Keywords:** Cauda, equina, herniation, neurosurgery, syndrome

## INTRODUCTION

*Cauda equina* syndrome (CES) is an uncommon, but well-known surgical entity that is often seen in primary care clinics and emergency departments. It characteristically presents acutely with some or all of the following symptoms: urinary retention with or without overflow incontinence, fecal incontinence from loss of anal sphincter tone, saddle area hypoesthesia or anesthesia, and acute or progressive weakness in the

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muscles of one or both lower extremities.<sup>[17,27,28]</sup> Although the time frame for surgical decompression has not been clearly delineated, it is often considered a surgical emergency and the presence of a true CES is typically treated within 24–48 h if possible.<sup>[3,13,51]</sup> The incidence of CES is approximately 0.04% (1:2500) in all patients who present with low back pain and only 1–2% of patients with lumbar disc herniations which is the most common cause, though the etiology of CES is diverse and can be caused by tumor, trauma, and infections.<sup>[1,2,5,6,18,21,24,25,33,39,43,45,50,52]</sup> The documented incidence appears to be on the rise, however, and given the legal implications surrounding the diagnosis and misdiagnosis of this entity, the true clinical syndrome may be overreported in literature.

Despite CES being well established among spine surgeons, there are several variables that can often contradict the suspected diagnosis and lead to delays in treatment. These can include inconsistent portions of the reported history, the physical examination, and even lumbar imaging.<sup>[7]</sup> Given the physical and legal ramifications, ruling out CES becomes the clinical responsibility of the treating physician which can be both challenging and fraught with peril. We suggest that a high level of suspicion should be maintained when caring for any patient presenting with urinary or fecal incontinence and acute or chronic back pain, even in the absence of other associated motor or sensory findings. We now report an atypical presentation of distal CES caused by a large, sequestered, caudally migrated L5–S1 herniated disc fragment.

## CASE REPORT

A 53-year-old male with a long history of uncontrolled Type 2 diabetes presented to the emergency department after recent cocaine ingestion with newly discovered scrotal numbness and several episodes of urinary and fecal incontinence. His finger-stick blood glucose was measured to be 800 mg/dL before consultation and his hemoglobin A1C was >18.5%. He endorsed mild-moderate low back pain for the previous 3 days with intermittent bilateral leg pain that was nondermatomal in distribution but denied any leg weakness or difficulty ambulating. His physical examination confirmed that his motor strength was full and his sensation to light touch was intact in all lower extremity muscle groups and tested dermatomes bilaterally. He also had normal patellar and Achilles deep tendon reflexes bilaterally. He did have perineal anesthesia to light touch and pinprick testing, as well as poor rectal tone. The patient had already received a Foley catheter before consultation without precatheter bladder volume assessment or documented initial urinary output.

He underwent an emergent lumbar magnetic resonance imaging which demonstrated mild-moderate multilevel degenerative spondylosis in the setting of congenital canal stenosis with significant lateral recess stenosis at L4–5 and a centrally herniated nucleus pulposus (HNP) at L5–S1 [Figure 1]. After discussing the risks and benefits of surgical decompression, the patient elected to proceed and was brought to the operating room for surgery within 8 h. Bilateral posterior laminectomies were performed at

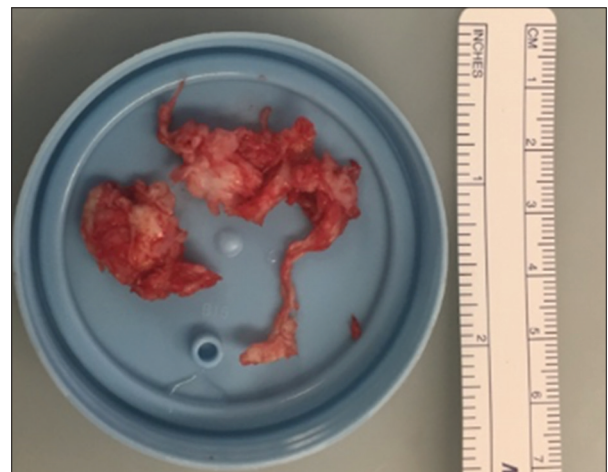
L4 and L5. The patient was noted to have severe stenosis caudal to the S1 lamina intraoperatively, so this was also removed bilaterally and revealed a large sequestered herniated disc fragment [Figure 2] posterior to the S1–S2 disc space [Figure 3] causing severe sacral nerve compression. Although the fragment was not sent for microscopic evaluation, it was of typical appearance, texture, and size of an HNP, and there was a clear annular defect at L5–S1 consistent with its presumed origin. The patient did well after surgery with regard to his back pain, and his perineal sensation and bowel continence improved significantly during his hospitalization. He was discharged home with a Foley catheter and outpatient urology follow-up on postoperative day 5.

## DISCUSSION

First described by Mixter and Barr, in 1934, the most common etiology of *C. equina* compression is an HNP.<sup>[36]</sup> Symptoms are a



**Figure 1:** Lumbar magnetic resonance imaging demonstrating mild-moderate multilevel degenerative disease in the setting of congenital canal stenosis with significant lateral recess stenosis at L4–5 and a herniated nucleus pulposus at L5–S1.



**Figure 2:** Sequestered herniated disc fragment.



**Figure 3:** Radiograph demonstrating herniated disc fragment posterior to the S1–S2 disc space.

direct result of nerve root dysfunction and most authors would agree that micturition impairment should be present to diagnose CES.<sup>[13,30,41,47]</sup> Symptoms that support the diagnosis of CES include decreased anal sphincter tone, reduced sensation in the sacral dermatomes (saddle area), and sexual dysfunction.<sup>[3,5,28,35,42]</sup> It should be noted that symptoms of sciatica (neurological deficits in one or both lower extremities including changes in reflexes, motor function, or sensation) are more common<sup>[27,28]</sup> at the time of CES presentation and the prevalence of it has been quoted to be 97%, while micturition dysfunction was seen in 92%, saddle anesthesia in 93%, and defecation dysfunction found in 74% of patients.<sup>[28]</sup> However, many studies have identified discrepancies between physical examination findings, imaging findings, and accurate diagnosis highlighting the protean presentation and need for vigilance.<sup>[8,16,20,40,45]</sup>

It is estimated that fewer than 1 in 2000 of patients who present with acute severe lower back pain will be diagnosed with CES<sup>[49]</sup> and up to 19–41% of patients who are referred for CES evaluation have essentially negative imaging studies.<sup>[7,8]</sup> In fact, patients presenting to the emergency department with isolated low back pain rarely require urgent or emergent neurosurgical intervention,<sup>[11,14,31]</sup> yet there is a myriad of CES causes that can present with subacute or vague pain and symptomatology. Some well-documented etiologies include lesions such as intracanalicular hemorrhages,<sup>[12,25]</sup> tumors,<sup>[26,44,48]</sup> cysts,<sup>[37,53]</sup> lipomatosis,<sup>[52]</sup> infection,<sup>[11]</sup> and even perineural spread of endometriosis.<sup>[46]</sup> Moreover, inflammatory and degenerative conditions that lead to stenosis such as ankylosing spondylitis,<sup>[29]</sup> Paget's disease,<sup>[39]</sup> osteoporotic stress fracture,<sup>[38]</sup> and hypertrophic degenerative stenosis<sup>[24]</sup> have also been well recognized in literature. While most pathologies have distinct imaging characteristics, there are various neoplastic causes of CES that has masqueraded as simple herniated discs including Ewing sarcoma,<sup>[22]</sup> osteosarcoma,<sup>[9]</sup> and even intradural metastatic cancer.<sup>[48]</sup> The vast etiological umbrella that underlies CES warrants a thorough workup on a case-by-case

basis and should any doubt exist regarding intraoperative findings during the requisite surgical intervention, we would advocate that a specimen be provided for pathological analysis to confirm the etiology.

Despite CES being considered a surgical emergency with decompression often occurring in an expedient fashion, the postoperative resolution of deficits is historically quite poor.<sup>[19]</sup>

A meta-analysis by Korse *et al.*, in 2017, found that at postoperative follow-up, the prevalence of residual sciatica was 48%, micturition dysfunction was 48%, altered saddle sensation was 57%, defecation dysfunction was 42%, and sexual dysfunction was 53%.<sup>[28]</sup> This emphasizes the point that assessing patients with this constellation of symptoms must be taken seriously as the impact on long-term quality of life is quite profound even with proper treatment. Moreover, comorbidities such as hyperglycemic neuropathy in a diabetic may have elements of these symptoms at baseline and can act as a distractor in cases of suspected CES as in our patient.<sup>[15]</sup> Indeed, CES diagnosis often becomes a mounting medicolegal issue with at least one study documenting that in 96% of cases of CES brought to a malpractice insurance organization, 65% progressed to claims by the affected patients, and 48% of these cases resulted in payment of compensation for damages.<sup>[34]</sup> It must be noted that rare cases of CES resulting from iatrogenic causes such as retained surgical cottonoids,<sup>[4]</sup> epidural steroid injections,<sup>[10]</sup> and even a pathological engorgement of the epidural venous plexus after elective lumbar microdiscectomy<sup>[32]</sup> have been reported in literature with additional consequences. Furthermore, studies have shown that a delay in treatment typically results in adverse legal decision highlighting the requisite expedited intervention.<sup>[19,23]</sup>

## CONCLUSION

As in our patient with clinical distractors and undocumented bladder dysfunction before consultation, the diagnosis of CES often becomes difficult to specify and confirm, so we ultimately advocate for physicians to maintain a high index of suspicion for CES in any patient presenting with concern for sacral nerve dysfunction.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patients understand that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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