

**A rare case report on cauda equina syndrome****Sai Charitha Sreeram\*, Talari Sampurna, P. Lakshmi**

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**ABSTRACT**

Cauda Equina Syndrome (CES) is a rare but serious neurologic condition in which neurological dysfunction affects the lumbar and sacral nerve roots within the vertebral canal. CES is a clinical entity consisting of low back ache, bilateral leg pain with motor and sensory deficits, genitourinary dysfunction, saddle anaesthesia and faecal incontinence. It is a rare condition with a prevalence in the general population estimated between 1:100000 and 1:33000. The presentation of CES may vary, especially in the early stages of compression. Magnetic Resonance Imaging (MRI) is the accepted gold standard modality of choice for diagnosing CES which identifies potential mimics such as aortic dissection or spinal infarction. Here we discuss a rare and unique case of CES with atypical presentation, knowledge of which is essential for early diagnosis, treatment and prevention of permanent neurological dysfunction.

**Keywords:** Cauda equina, Genitourinary dysfunction, Faecal incontinence, Saddle anaesthesia

**INTRODUCTION**

Cauda equina syndrome (CES) is a rare syndrome that has been described as a complex of symptoms and signs - low back pain, unilateral or bilateral sciatica, motor weakness of lower extremities, sensory disturbance in saddle area, and loss of visceral function resulting from compression of the cauda equina. CES occurs in approximately 2% of cases of herniated lumbar discs and is one of the few spinal surgical emergencies.<sup>1</sup>

CES may be divided into complete or incomplete. In complete cauda equina syndrome patients present with saddle anaesthesia and retention or incontinence of bladder or bowel. In incomplete CES there is saddle anaesthesia but bladder and bowel dysfunction has not progressed to full retention or incontinence. Bladder or bowel symptoms that these patients may report include loss of urgency or altered urinary sensation.<sup>2</sup> The

commonest cause of cauda equina syndrome in our practice and the focal causative condition in the literature is compression arising from large central lumbar disc herniation at the L4/5 and L5/S1 level.<sup>3</sup>

Magnetic Resonance Imaging (MRI) is the accepted gold standard modality of choice for diagnosing CES which identifies potential mimics such as aortic dissection or spinal infarction.<sup>4</sup> Myelography and CT Myelography is used as an alternative for patients not suitable for MRI but have the disadvantage of being invasive techniques.<sup>5</sup> Most cases of cauda equina syndrome are caused by herniation of the lumbar disc, for which the surgery indicated is decompression at the level of the herniation, usually involving discectomy. Early surgery within 24hours is required to relieve pressure from the nerves in the cauda equina and to remove the offending elements. Treatment depends on the underlying cause with wide laminectomy and extensive decompression being the

accepted surgical technique for a large lumbar disc herniation.<sup>6</sup>

## CASE REPORT

A 45-years-old man, he was brought to the hospital with chief complaints of weakness and loss of sensation of both lower limbs which is gradually progressive since 1 year and low back pain since 1 year. He had a long history of back pain presents with a recent increase in pain and urinary incontinence. He had decreased power in the lower limbs, saddle anaesthesia and decreased anal tone. He had a history of cotton-wool sensation of ground since 1 year and abnormal bowel & bladder involvement since 3 months. His history of present illness states that low back pain which was more on right side since 3 months, asymmetrical onset. Past history reveals that the patient had Bed sores - ulcers over buttock region.

At the time of admission, he was conscious and coherent. Laboratory data included Routine haemogram revealed haemoglobin 10.8 gm%, platelets 1.78 lakhs/cumm packed cell volume 32%, total leukocyte count 7,400 cells/mm<sup>3</sup>, urinalysis, liver and renal functions, with normal triglyceride, cholesterol, and uric acid levels. Serum creatinine 0.9mg/dl, serum electrolytes - sodium 137mmol/l; potassium 3.8mmol/l; chloride 92mmol/l. MRI scan revealed spinal arterio venous malformation with feeding vessels originating from the internal iliac artery. From the above examinations, the patient was diagnosed as CAUDA EQUINA SYNDROME and was on treatment with Tab. Paracetamol 500mg PO TID, Inj. Optineuron 1amp IM OD, Tab. B-complex PO OD, Tab. Cefixime 200mg PO BD, Tab. Pantop 40mg PO OD, Tab. Metrogyl 400mg PO TID and Inj. Tramadol 2cc IM OD. And the patient was discharged after 13 days.

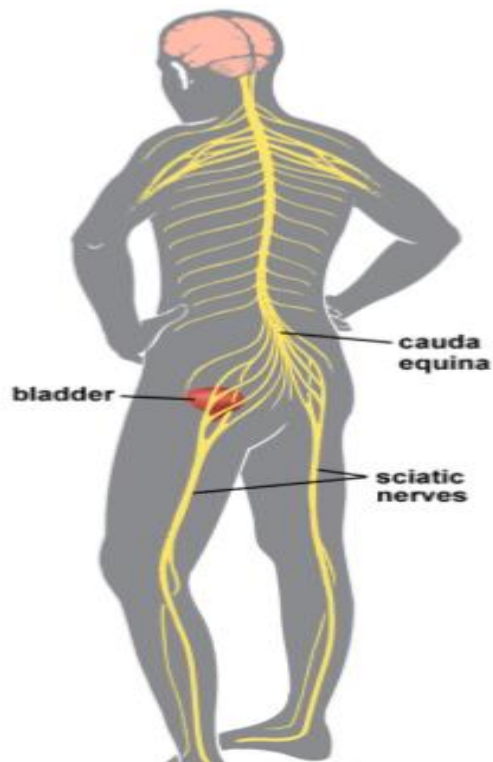


**Figure 1: MRI Scan showing compression on Cauda equine.**

## DISCUSSION

Cauda equina syndrome is a serious neurological condition which results from the dysfunction of multiples

sacral and lumbar nerve roots in the lumbar vertebral canal. Such root dysfunction can cause a combination of clinical features, but the term cauda equina syndrome is used only when these include impairment of bladder, bowel, or sexual function, and perianal or “saddle” numbness.<sup>7-9</sup>



**Figure 2: The cauda equine is a bundle of spinal nerves in the lower back.**

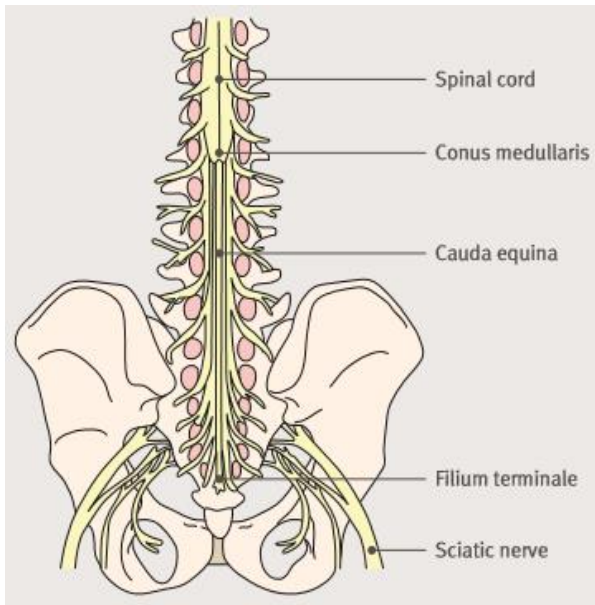
Branches from these nerves go to the bladder, colon and pelvic organs.

The nerves continue through the pelvis and down the back of each leg as the sciatic nerves.

The cauda equina consists of nerve roots distal to the conus medullaris.<sup>10,11</sup> The dorsal root consists of afferent fibers for transmission of sensation, and the ventral root provides motor fibers for the efferent pathway.<sup>11,12</sup>

Orientation within the cauda equina is unique and specific. The most posterior neural elements within the sac are the fifth sacral nerve roots. In a cranial direction, the fifth sacral nerve roots progress anteriorly from the fourth through the first sacral vertebra. The most anterior element at the fifth lumbar and first sacral disc level is the first sacral nerve root. The fifth lumbar nerve root enters anterolaterally between the fourth and fifth lumbar vertebrae-displacing the first sacral nerve root more posteriorly. Each subsequent nerve root continues this displacement, with one root added in the cephalad direction at each disc level. The motor fiber components

are anteromedial, and the larger sensory components are posterolateral.<sup>11,13-15</sup>



**Figure 3: Anatomy of the lower lumbar and sacral spine showing the cauda equine.**

CES may be divided into complete or incomplete. In complete cauda equina syndrome patients present with saddle anaesthesia and retention or incontinence of bladder or bowel. In incomplete CES there is saddle anaesthesia but bladder and bowel dysfunction has not progressed to full retention or incontinence. Bladder or bowel symptoms that these patients may report include loss of urgency or altered urinary sensation.<sup>2</sup>

The commonest cause of cauda equina syndrome in our practice and the focal causative condition in the literature is compression arising from large central lumbar disc herniation at the L4/5 and L5/S1 level.<sup>3</sup> Numerous other less common causes of cauda equina syndrome have been reported - for example, spinal injury with fractures or subluxation. Spinal neoplasms of metastatic or primary origin can cause compression, usually accompanied by marked pain and often as part of a chronic condition. Infective causes with abscess formation or bony involvement, either within the spinal canal or impinging on it, may also cause cauda equina syndrome.<sup>16</sup>

The spine is the most commonly affected skeletal site for tuberculosis, and Pott's paralysis is well documented.<sup>17</sup> Multiple other pathologies can damage the anatomical structures involved. An extensive list of causes is given in the Table 1.

CES is defined by impairment of bladder, bowel and sexual function with perianal and saddle numbness.<sup>7</sup> A thorough history should include any obvious precipitants relating to the aetiologies listed above such as trauma, underlying malignancy or recent surgery. Saddle

anaesthesia and bladder, bowel or sexual dysfunction are the key clinical findings to discriminate between CES and sciatica, which can also present with low back pain and radiculopathy.<sup>2</sup> The autonomic dysfunctions may be present in various combinations. The psychosocial aspect or presence of back pain and other urological dysfunction may inhibit the patient to volunteer sexual dysfunction.<sup>18</sup>

**Table 1: Aetiology of CES lesions.**

Causes	Lesions
Congenital	Spinal dysraphism
	Vertebral body malformations
	Dwarfing syndromes
	Congenital tumours
Acquired	Trauma Spinal fracture or dislocation
	Infective Bacterial abscess tuberculosis
	Neoplastic Primary tumour secondary metastases
	Degenerative Spondylolisthesis spinal stenosis disc prolapse
	Inflammatory Rheumatoid arthritis ankylosing spondylitis
	Vascular AV malformation epidural/subdural haematoma
	Iatrogenic Secondary to surgery

Magnetic Resonance Imaging (MRI) is the accepted gold standard modality of choice for diagnosing CES. This clearly depicts the soft tissue pathology and delineates the level. It can also identify potential mimics such as aortic dissection or spinal infarction, presence and site of compression of the cauda equine. Disadvantages include lack of 24-hour availability and contraindications such as pacemakers and poor patient tolerance due to claustrophobia.<sup>4,19</sup>

Magnetic resonance imaging (MRI) do not define bone as clearly as computed tomography (CT) but is better at showing soft tissues such as intervertebral disc, ligamentum flavum, dural sac, and nerve roots.<sup>19</sup> Plain films are generally unhelpful in the investigation of a herniated disc but can provide valuable information in the setting of acute trauma. Myelography and CT Myelography can be used as an alternative for patients not suitable for MRI but have the disadvantage of being invasive techniques.<sup>5</sup> Inflammatory markers and Cerebrospinal fluid (CSF) studies should be performed when an inflammatory or infectious aetiology is being considered. Saddle anaesthesia and urinary retention greater than 500 mL may be the best positive predictive indicators for CES.<sup>20-22</sup> Clinical diagnosis of cauda equina syndrome even by resident neurosurgeons has a 43% false positive rate, So accurate confirmatory imaging is important.<sup>23</sup>

The most important issue regarding CES is timing. When a patient has clinical features of cauda equina syndrome and an MRI scan shows a potentially reversible cause of pressure on the cauda equina then current consensus recommends surgical decompression.<sup>3</sup> Most cases of cauda equina syndrome are caused by herniation of the lumbar disc, for which the surgery indicated is decompression at the level of the herniation, usually involving discectomy. The operation can be very demanding technically, and great care is needed to avoid causing further damage to nerve roots or tearing tightly compressed dura. Incomplete cauda equina syndrome requires emergency surgery to try to stem the deterioration in bladder function.<sup>24,25</sup>

Recently published guidelines for the management of patients with back pain and neurological signs recommend urgent surgical referral for suspected cauda equina syndrome.<sup>26</sup> A review that is widely quoted suggests that intervention less than 48 hours after the onset of symptoms will produce a better outcome than intervention delayed for longer than this.<sup>3</sup> Persisting cauda equina syndrome has a devastating effect on personal and social life, and its mismanagement is one of the commonest causes for litigation in spinal surgery. Most patients are young to middle aged and in work before they develop cauda equina syndrome, so the size of claims is large. The presence of residual symptoms means that many of these patients are unable to work and have genitourinary and bowel symptoms. The timing of surgery remains a contentious issue and it is agreed that early decompressive surgery should be performed to reduce the risk of long term neurological dysfunction.<sup>27</sup>

The aetiologies that are previously discussed such as spinal cord malignancy or aortic dissection are treated by addressing the underlying cause i.e. radiotherapy or vertebroplasty in the case of malignant cord compression. The role of surgery is to relieve pressure from the nerves in the cauda equina and to remove the offending elements. Treatment depends on the underlying cause with wide laminectomy and extensive decompression being the accepted surgical technique for a large lumbar disc herniation.<sup>28</sup>

Treatment with high-dose steroids (dexamethasone 4-100 mg IV) may provide pain relief and improved neurologic function (by reducing edema) while awaiting diagnostic studies and surgical decompression.<sup>29</sup> But here only Tramadol & paracetamol were given to reduce pain and vitamin supplements were also given (B-complex and optineuron).

The outcome for patients with CES is determined primarily by their symptoms at presentation. Patients who can ambulate at presentation will generally remain ambulatory. Patients who present with paresis but are ambulatory with assistance have approximately a 50% chance of walking again, and as many as 79% of patients

presenting with urinary retention will continue to require a urinary catheter after treatment.<sup>30</sup>

The clinical outcome in this patient who has Cauda equina syndrome with retention is poor anyway and bears no relation to timing of surgery. In this case, the patient was unable to walk and there is also no chance of recovery even after the surgery was done, because he was suffering with these complaints since 1 year.

## CONCLUSION

Cauda equina syndrome is rare, but devastating if symptoms persist. Clinical diagnosis is not easy and even in experienced hands is associated with a 43% false positive rate. The investigation of choice is magnetic resonance imaging. There are numerous causes of CES, however degenerative disc disease with resultant prolapse remains the most common cause. Once urinary retention has occurred the prognosis is worse. Early decompression, preferably within 24 or 48 hours, for all patients with CES, provided the patient's medical condition and the presence of qualified support staff allow for it. Good retrospective evidence supports urgent surgery especially in early cases. The timing of surgery remains a contentious issue and it is agreed that early decompressive surgery should be performed to reduce the risk of long term neurological dysfunction and for improved chance of recovery.

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