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Case Report

Indeterminate cauda equina syndrome: A case report

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ABSTRACT

Introduction: The presentation of cauda equina syndrome (CES) varies from its classical presentation, especially in its early stages of compression. We present a case of lumbar disc prolapse causing CES in an uncharacteristic way, knowledge of which is essential for orthopaedicians to diagnose this condition early and prevent neurological complications. **Case report:** A 32-year-old male patient presented to us with complaints of inability to lift his left ankle and numbness over his left leg and ankle for 14 days. Clinical examination showed involvement of left L3, L4, L5 and S1 nerve roots as evidenced by weakness of quadriceps, extensor hallucis longus, extensor digitorum longus muscles and tendo achilles. Knee jerk was absent. The opposite lower limb was normal and there was no evidence of bowel bladder involvement or saddle anaesthesia. The MRI showed L2 L3 posterocentral disc prolapse compressing the cauda equina. The patient underwent laminectomy and discectomy. Post-operatively, the patient showed significant improvement in his sensory symptoms with complete recovery of motor power in 12 weeks.

Discussion: In contrast to the classical presentation of CES, several case series have been reported with varied clinical manifestations like unilateral leg symptomatology, unilateral or bilateral saddle anaesthesia with or without leg symptoms and CES with complete absence of signs and symptoms in the lower limbs. The disc prolapse in our case at L2–L3 level has compressed the left-sided L3, L4, L5 roots with minimal compression of S1. The classical features of CES would have occurred due to the lateral shift of the cauda equina in our case but for our early diagnosis and intervention.

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1. Introduction

Cauda equina syndrome (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.^{1,2} The presentation of CES may vary, especially in the early stages of compression.^{3,4} This article discusses a case report of CES with atypical presentation, knowledge of which is essential for early diagnosis and prevention of permanent neurological dysfunction.

2. Case report

A 32-year-old male, a manual labourer by occupation, presented to the out-patient clinic with inability to lift his left ankle and numbness over his left leg and ankle during the previous 14 days. He gave a history of getting a painkiller injection at a local hospital over his left gluteal region 14 days back and his symptoms developed thereafter. He was initially diagnosed to have post-injection paralysis of sciatic nerve (common peroneal nerve component). But on subsequent scrutiny, he divulged that his symptoms did not develop immediately after the injection, as it would have been if it were a post-injection nerve palsy, rather it developed insidiously over time. He denied history of trauma or fever.

Clinical examination showed only a flicker of contraction in his left extensor hallucis longus (EHL), extensor digitorum longus (EDL) and tibialis anterior (TA) muscles. Quadriceps power was grade 2 (MRC grading). Flexor hallucis longus (FHL), flexor digitorum longus (FDL) and tibialis posterior (TP) muscles of the left side had grade 5 power. Tendo achilles and hamstrings power was grade 4. Muscles around the left hip had full power. There was sensory loss over L3, L4 and L5 dermatomes on the left side. Knee jerk was absent and ankle jerk was normal. Femoral stretch pain on the left side was

present. The patient's right lower limb was uninvolved. Both upper limbs and trunk were normal. There was no evidence of bowel bladder involvement or saddle anaesthesia.

Nerve conduction study performed over both lower limbs showed reduced amplitude in the left common peroneal nerve with absent F wave. Distal latency and conduction velocities were normal. SNAP of superficial peroneal nerves of both sides was absent. Features were consistent with bilateral axonal sensorimotor neuroradiculopathy of peroneal nerves. MRI of the lumbar spine showed L2 L3 posterocentral disc prolapse compressing the cauda equina (Figs. 1–3). MR myelogram image showed complete block at L2 L3 level (Fig. 4). The patient was planned for discectomy.

Under general anaesthesia, L2 laminectomy with L2 L3 discectomy was performed. In the immediate post-operative period, the patient showed significant improvement of his sensory symptoms. Drain was removed on the second post-operative day and sutures on the 14th day. The patient was followed up regularly on 4th, 8th and 12th week post-operatively. At 12 weeks, the patient showed full recovery of his motor weakness. The power of all muscles was grade 5 on the left side and the sensory improvement was 100%.

3. Discussion

In patients with low back pain or sciatica, the presence of one of the following features – saddle anaesthesia, urinary retention or incontinence, bowel incontinence, sensory or motor weakness in either of the lower extremities – suggests the diagnosis of CES. The most common cause for CES is lumbar disc prolapse (1–10% of cases), though it can also be caused by trauma, spinal canal stenosis, tumours, epidural haematoma/abscess, inferior vena cava thrombosis and spinal manipulation.⁵ There are two features which make cauda equina susceptible to the effects of compressive and tensile forces. First, the nerve roots have no Schwann cell covering;

Fig. 1 – Sagittal MRI sections showing disc prolapse at L2 L3 level compressing the cauda equina.

Fig. 2 – MRI showing compression of the cauda at L2 L3 level by the prolapsed intervertebral disc.

Fig. 3 – Axial section of MRI showing posterocentral disc prolapse at L2 L3.

second, cauda equina lacks a regionalised segmental blood supply with relative hypovascularity in the central portion of the nerve root, making it vulnerable to ischaemia from compression.⁶

The classical presentation of CES includes low back pain with saddle anaesthesia, bilateral variable lower limb sensory and motor disturbances and bowel bladder involvement.⁴ Contrary to the above statement, several case series have been reported with varied clinical manifestations like unilateral leg symptomatology,^{4,7} unilateral or bilateral saddle anaesthesia with or without leg symptoms and CES with complete absence of signs and symptoms in the lower limbs.^{8,9} Young reported 39% of the 470 patients operated for CES, who had no

neurological signs in the lower limbs.⁸ O'Laoire reported CES with acute bladder retention and lack of sensory deficit in 2 out of his 29 patients.⁹

Our patient had involvement of L3, L4 and L5 nerve roots on the left side. He had weakness of knee extension with only a flicker of contraction in his TA, EHL and EDL. Sensory loss was present over L3, L4 and L5 dermatomes and knee jerk was absent. Also, there was minimal involvement of S1 root; as evidenced by the power of tendo achilles viz. grade 4 compared to the normal side. S2, S3, S4 roots were uninvolved both clinically and radiologically. The plausible explanation for the lack of classical features of CES in our case could be due to the arrangement of nerve fibres within the cauda: the higher nerve

Fig. 4 – MR myelogram showing complete block at the level of disc prolapse.

roots travelling more laterally and the lower sacral nerve roots travelling within the medial central aspect of the cauda. A large posterior disc herniation can impinge both the traversing root below it as well as the cauda equina through a lateral shift within the spinal canal.¹⁰ In our case, the disc prolapse at L2-L3 level has compressed the left-sided L3, L4, L5 roots with minimal compression of S1. The classical features of CES would have occurred due to the lateral shift of the cauda equina in our case but for our early diagnosis and intervention. Hence, by virtue of diagnosing our case in the very early stages of compression, the full-blown features of cauda equina were averted. Similar uncharacteristic cases of CES have been reported in the literature.^{11,12} Bartels and de Vries reported 10 patients with a specific combination of symptoms: unilateral sciatica, unilateral sensibility loss in dermatomes S1 to S5 and subjective micturition problems, which they called hemicauda equina syndrome from herniated lumbar disc.¹³

Our patient came to us after 14 days of the onset of symptoms. Laminectomy and discectomy were done on the fourth day after admission. Literature is equivocal on the timing of decompression after the onset of symptoms. Shapiro, in a retrospective case series, found 100% resolution of bladder and bowel symptoms if the decompression was done within 48 h of the onset of symptoms. This percentage dropped to 33% if the decompression was done after this time frame.^{14,15} Kostuik found no correlation between the time of onset of symptoms to surgery and extent of functional recovery in his case series.¹⁶ In our case, the decompression was done 18 days after the onset of symptoms and the patient showed complete recovery. Though there is no clear consensus regarding the urgency of surgical decompression, all authors still recommend surgery as soon as possible to maximise functional recovery, especially the bladder symptoms.¹⁷ Patients with premonitory symptoms without bladder involvement, like in our case, should be viewed seriously, assessed pertinently to intervene early precluding eventual bladder bowel involvement.

Failure to recognise CES can result in patient morbidity such as loss of bladder, bowel and sexual function with potential medico legal repercussions. It is, therefore, imperative that all orthopaedic surgeons should have a detailed knowledge of the varied clinical presentations of this condition and assess the patients with low back pain for signs of CES. In equivocal cases as the one described above, clinicians should never hesitate to investigate further with a MRI of the lumbar spine.

4. Conclusion

This case is presented for its uncharacteristic clinical presentation of a posterocentral lumbar disc prolapse causing compression of the cauda equina producing unilateral loss of sensation and motor power without bladder bowel involvement or saddle anaesthesia. This could be the premonitory features of CES and failure to recognise this atypical constellation of symptoms and signs can delay the diagnosis of CES by the orthopaedic surgeon. The eventual prognosis of this condition depends on early diagnosis, as prompt surgical intervention before bladder bowel involvement will give gratifying functional recovery.

Conflicts of interest

The authors have none to declare.

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