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Case Report

Orthopaedics Section

Conus Medullaris Syndrome Following a L1 Burst Fracture: A Rare Case Report

RS MONISH ANAND[1], B VIJAY ANAND[2], S DEVI PRASAD[3], ROBERT SEBASTIAN DIAS[4], ASHWIN MATHEW KADAVIL[5]

ABSTRACT

Thoracolumbar burst fractures are common injuries related to high-energy trauma and often lead to loss of the spinal canal and neurologic loss. Among such injuries, one rare but severe complication is Conus Medullaris Syndrome (CMS), which causes bowel and bladder dysfunction, loss of perianal sensation, and may cause motor deficits. The present case report presents an uncommon presentation of pure CMS in a 34-year-old male following an L1 burst fracture. The trauma occurred while the patient was seated in a bus, during which axial loading led to lower back pain followed by delayed onset neurological symptoms. Neurological examination revealed a deficit in reflexes and perianal sensory loss, with preserved lower-limb motor weakness. Radiological examination revealed an L1 burst fracture with severe spinal canal injury and injury to anterior ligamentous complex. The patient underwent pedicle screw fixation and posterior decompression, after which neurological recovery was gradual. Bowel control returned on Postoperative Day (POD) 8, and bladder control was regained on POD 40. The present case highlights the role of early diagnosis, advanced imaging, and customised surgical treatment in achieving the best results in thoracolumbar fracture patients on CMS. The abnormal pathogenesis and delayed development of symptoms emphasise the significance of focusing on the diagnosis and treatment of spinal trauma cases.

Keywords: Decompression, Lumbosacral region, Neurogenic, Spinal cord compression, Spinal fractures/surgery, Surgical, Urinary bladder

CASE REPORT

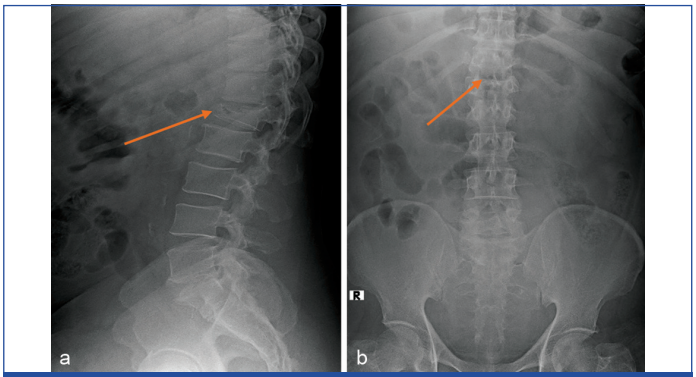
A 34-year-old male was presented to the Emergency Department with three days of lower back pain of severe nature, two days of bowel and bladder incontinence. The accident occurred when he was a backseat passenger of a bus and was forcefully lifted off the seat as the bus rolled over speed bumps, causing a heavy blow on the buttocks. The patient experienced sudden-onset lower back pain but did not seek immediate medical attention. His symptoms worsened the following day, and bowel and bladder incontinence were noticed. The patient denied loss of consciousness, vomiting, ear bleeding, or any neurological deficiency such as numbness or lower-limb weakness. His past medical history was significant for systemic hypertension for four years, well-controlled with regular follow-up and oral antihypertensive medication (Amlodipine 5 mg once daily).

Clinical Examination

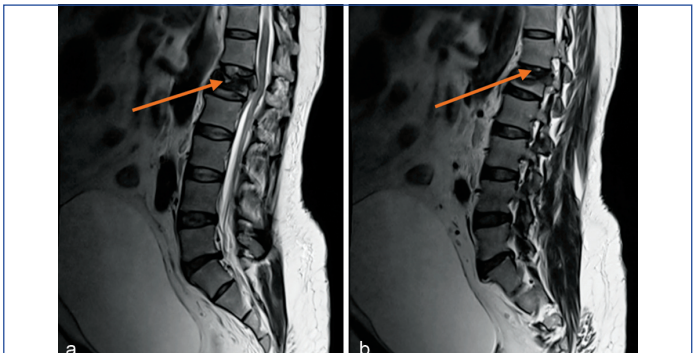
On examination, diffuse tenderness was noted from D12 to L3, accompanied by increased paraspinal muscle spasm. The passive straight-leg raise was approximately 80° bilaterally. Motor and sensory examinations did not reveal any deficits. Neurological assessment revealed absent cremasteric and bulbocavernosus reflexes, decreased perianal sensation, and absent sphincter tone. American Spinal Injury Association Impairment Scale (ASIA) charting findings included altered S4-S5 sensations and no voluntary anal contractions, confirming neurological involvement indicative of CMS [1].

Investigations

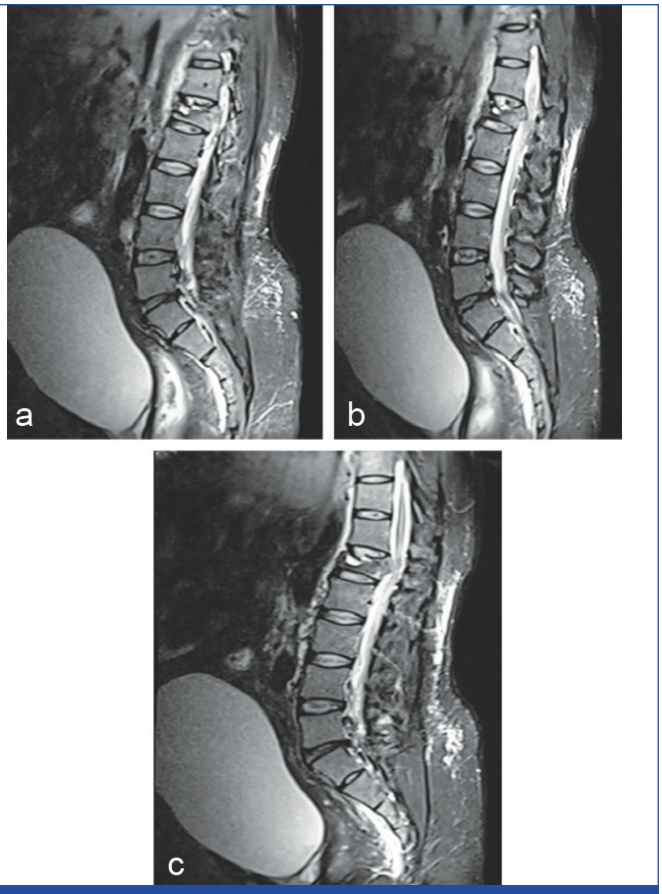
Baseline laboratory investigations, including complete blood count, renal and liver function tests, and electrolytes, were within normal limits. Plain radiographs of the lumbosacral spine [Table/Fig-1a,b] demonstrated loss of vertebral body height and retropulsion of fracture fragments. Magnetic Resonance Imaging (MRI) confirmed compression of the conus medullaris and disruption of the Posterior Ligamentous Complex (PLC) [Table/Fig-2-5]. The fracture was classified according to the Arbeitsgemeinschaft für Osteosynthesefragen (Association for the Study of Internal Fixation Spine Classification as Type A3 (incomplete burst fracture) and



[Table/Fig-1]: Preoperative X-ray of the lumbosacral spine; a) Lateral view showing an L1 burst fracture with loss of vertebral body height and retropulsion of fracture fragments into the spinal canal (marked with arrow); b) Anteroposterior (AP) view showing collapse of the L1 vertebral body and widening of the interpedicular distance (marked with arrow).

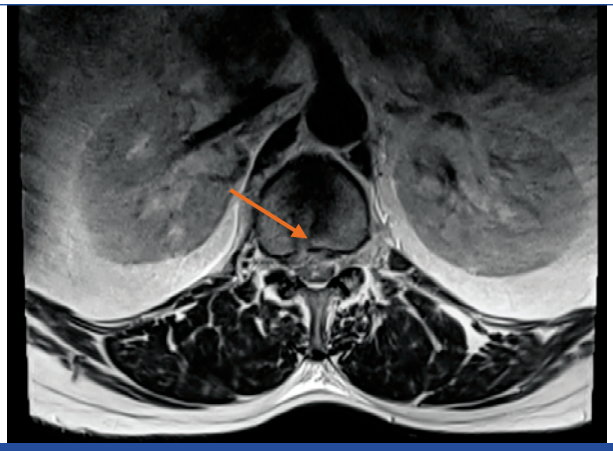


[Table/Fig-2]: Preoperative T2-weighted MRI of the lumbosacral spine; a) Sagittal view showing a burst fracture of the L1 vertebral body with more than 50% loss of vertebral height (arrow); b) Sagittal view showing retropulsion of fracture fragments into the spinal canal causing compression of the conus medullaris (arrow).



[Table/Fig-3 a-c]: Preoperative T2 Short Tau Inversion Recovery (STIR) sagittal view of the lumbosacral spine showing a burst fracture of the L1 vertebral body with more than 50% reduction in height, retropulsion of fracture fragments leading to cord compression, and focal disruption of the ligamentum flavum.

[Table/Fig-4]: Preoperative T2-weighted whole spine sagittal MRI showing an L1 burst fracture with retropulsion of fracture fragments into the spinal canal, causing conus medullaris compression and Posterior Ligamentous Complex (PLC) injury.



[Table/Fig-5]: Preoperative T2-weighted MRI axial cut at the L1 level demonstrating retropulsion of fracture fragments causing significant spinal canal compromise (arrow)

by the Denis Three-Column Classification as Type 2B [2,3]. The Thoracolumbar Injury Classification and Severity Score (TLICS) was calculated to be 7, based on fracture morphology (2 points for burst fracture), PLC injury (3 points), and neurological deficit due to CMS (2 points), thereby justifying the need for surgical intervention [4].

The patient was diagnosed with an L1 burst fracture with associated CMS. Differential diagnoses considered included cauda equina syndrome, lumbar disc herniation with severe canal stenosis, spinal cord infarction, intramedullary or extramedullary tumours, and infective or inflammatory causes such as tubercular spondylitis or epidural abscess. However, the acute traumatic mechanism, characteristic imaging features, and neurological correlation confirmed the diagnosis of an L1 burst fracture with conus medullaris compression.

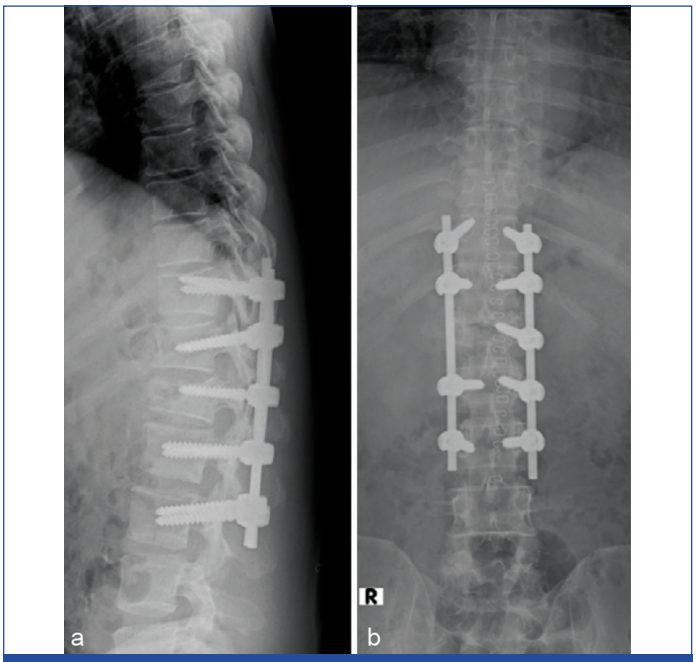
Surgical Management

The patient underwent posterior stabilisation of the thoracolumbar segment (D11-L3) under general anaesthesia. After positioning the patient prone and after standard sterile preparation and draping, a midline posterior approach was performed, with levels confirmed using a needle and C-arm. Skin and subcutaneous tissue were retracted, and subperiosteal dissection exposed the posterior elements from D11 to L3. Pedicle screws were inserted at D11, D12, L2, and L3 on the left side, followed by screw placement at L1, L2, and L3 on the right side under fluoroscopic guidance. Rods were contoured and secured to achieve reduction and restore alignment of the thoracolumbar junction. Intraoperatively, retropulsed fracture fragments were visualised causing significant compression of the conus medullaris, along with evidence of PLC disruption.

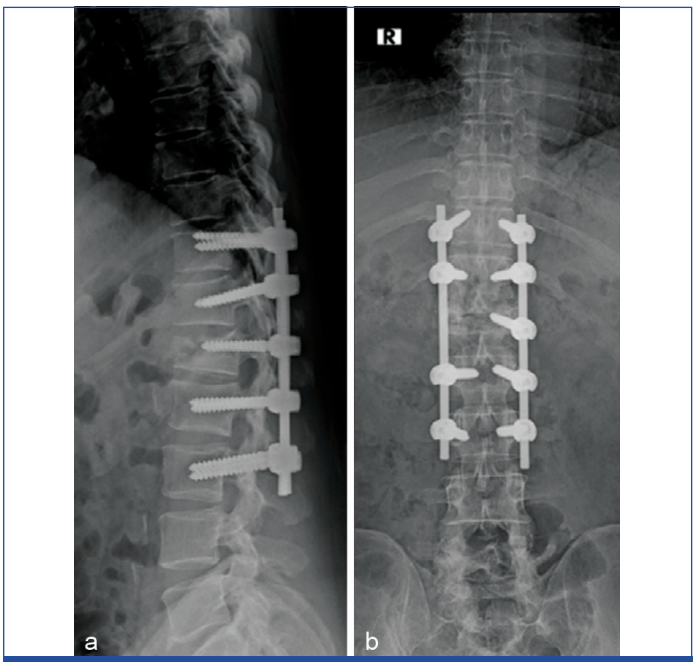
A meticulous L1 laminectomy was performed for spinal canal decompression, ensuring adequate clearance of the neural elements. Haemostasis was secured, and bone grafts were placed posterolaterally to promote fusion. The wound washing was done using two litres of normal saline and closed in layers after securing a drain tube. Satisfactory stabilisation of the affected segment was confirmed on postoperative X-rays. The intraoperative and postoperative periods were uneventful. Postoperative recovery was closely monitored, with serial X-rays confirming satisfactory stabilisation of the thoracolumbar segment [Table/Fig-6]. The patient received intravenous antibiotics for five days, analgesics for pain relief, and muscle relaxants as needed. He was advised strict bed rest initially, followed by gradual mobilisation with the support of a thoracolumbar brace. Wound care instructions, bowel regimen, and bladder training techniques were explained to the patient and caregivers.

The patient was mobilised on the second POD 2 with physiotherapy guidance. Neurological recovery was assessed clinically through serial ASIA charting, perianal sensory testing, and evaluation of anal sphincter tone and voluntary contractions. Recovery was gradual but

steady, with bowel control regained by POD 8 and bladder control by POD 40 through regular bladder training. At one-month follow-up, the patient demonstrated significant neurological improvement, with his ASIA score improving from Grade B at presentation to Grade D, indicating marked recovery in sensory and reflex function. Follow-up X-ray at one month postoperatively showed satisfactory stabilisation and alignment of the thoracolumbar spine [Table/Fig-7].



[Table/Fig-6]: Immediate postoperative X-ray showing successful stabilisation of the thoracolumbar spine (D11-L3) using pedicle screw fixation: a) Lateral view demonstrating restoration of vertebral alignment and sagittal profile; b) AP view showing accurate screw placement and construct stability.



[Table/Fig-7]: Follow-up X-ray at one month postoperatively showing satisfactory stabilisation and alignment of the thoracolumbar spine: a) Lateral view demonstrating maintained vertebral alignment and sagittal profile; b) AP view showing well-positioned pedicle screws.

DISCUSSION

The present case presentation is uncommon because the patient presented with pure CMS manifesting solely as bowel and bladder dysfunction without any motor weakness of the lower limbs following an L1 burst fracture-an extremely rare occurrence. Typically, thoracolumbar burst fractures present with cauda equina syndrome or mixed neurological deficits, whereas isolated involvement of the conus medullaris is unusual [5]. The mechanism of injury in the present case, involving axial loading while seated during a bus ride rather than a typical fall from height or road traffic accident, further contributes to its exceptional nature.

Thoracolumbar burst fractures are common, particularly in young males subjected to high-energy trauma. These injuries were most frequently reported in the 15-29-year age group before 2000, but the current median age has shifted to around 35 years, which aligns with the 34-year-old patient in this case [6]. Studies by Alawad MO et al., and Grivna M et al., confirm this epidemiological trend, showing that spinal injuries predominantly affect young, active males and are frequently associated with axial-loading mechanisms such as falls from height, motor vehicle collisions, and workplace accidents [7,8]. These findings highlight the vulnerability of this demographic to high-impact trauma and explain the predominance of axial load-related fractures in epidemiological series.

Radiographic imaging remains pivotal in diagnosing and planning management for thoracolumbar fractures. In this case, X-rays revealed classic burst fracture characteristics, including vertebral height loss and retropulsion of bony fragments. These findings align with the work of Denis F who emphasised that thoracolumbar stability depends on the integrity of all three spinal columns-anterior, middle, and posterior-and demonstrated that burst fractures typically involve disruption of the anterior and middle columns, often accompanied by retropulsion into the spinal canal [3].

MRI further confirmed compression of the conus medullaris along with disruption of the Posterior Ligamentous Complex (PLC). Cahueque M et al., highlight the importance of MRI in assessing soft-tissue trauma and spinal canal compromise, which is essential for determining prognosis and guiding surgical planning [9]. The fracture was classified using both the AO Spine Classification and the Denis Three-Column Theory [2,3]. According to the AO Spine Classification, the injury was categorised as a Type A3 injury, which represents a Type A burst fracture involving the anterior and middle columns without disruption of the outer tension band. Using the Denis Three-Column Theory, it was classified as Type 2B, including the collapse of the anterior and middle columns with retropulsion into the spinal canal, was diagnosed using the Three-Column Theory of Denis [3].

The reason for surgical intervention was the TLICS of 7, reflected the severity of the fracture morphology, PLC injury, and neurological deficit. According to Vaccaro AR et al., and Oner FC et al., the TLICS system is also a valid instrument of treatment decision-making, which considers fracture morphology, neurological status, and Posterior Ligamentous Complex (PLC) integrity [10,11]. A high score in this case indicated significant injury severity and warranted operative management.

The patient underwent pedicle screw repair and de-spinal canal decompression. The method is mostly considered as an effective means of restoring stability and offloading neuropathy in thoracolumbar burst fractures [12]. It can be applied specifically in the event of neurological impairments as well as in instances where the PLC is destroyed [10,11]. The midline posterior approach provided adequate exposure for safe and complete decompression of the conus medullaris, with minimal intraoperative risk. Posterior fixation is quite effective, especially effective in acute trauma cases, facilitating early mobilisation, pain relief, and the nervous system recovers. Pedicle screws offered good fixation, a requirement of successful fusion, maintenance of alignment, and prevention of progressive deformity [11,13-16].

In the present case, the patient showed a stable neurological recovery, with bowel and bladder functions regained on postoperative days 8 and 40, respectively aligning with findings revealed by Tanaka S et al., who described a patient with an L1 compression fracture who developed urinary incontinence without lower limb weakness, and who regained vesicorectal function one year after decompression [17]. Similarly, Wang H et al., reported a case of traumatic CMS

due to an L1 burst fracture, in which early surgical decompression led to progressive improvement in urinary continence within six weeks [18]. These reports, along with the present case, emphasise that timely surgical decompression is crucial for improving bowel and bladder function and for achieving favourable neurological outcomes in CMS.

Findings from several published case reports on CMS further support the observations in this case. Chung JY et al., (2015) reported a 36-year-old male with CMS following an L1 burst fracture who achieved complete recovery of bladder function within three months after posterior decompression and fixation [19]. Yoo DS et al., (2018) described isolated CMS secondary to an L2 burst fracture, where surgical decompression performed within 24 hours led to full recovery of sensory and autonomic function at six months [20]. Similarly, Kang MS et al., (2020) presented a case of CMS caused by an intradural disc herniation at L1-L2 level, in which removal of the herniated fragment resulted in immediate improvement of urinary dysfunction [21]. Collectively, these reports highlight that prompt diagnosis and timely decompression are pivotal in restoring bowel and bladder control and achieving favourable neurological outcomes in CMS.

The present case highlights a rare presentation of pure CMS following an L1 burst fracture, manifesting as isolated bowel and bladder dysfunction without motor weakness. Early surgical decompression and stabilisation resulted in progressive neurological recovery. The findings emphasise the importance of maintaining high clinical suspicion for conus involvement in thoracolumbar fractures and of employing timely imaging and surgical intervention to optimise functional outcomes.

CONCLUSION(S)

The present case highlights an uncommon presentation of pure CMS following an L1 burst fracture and underscores the importance of early diagnosis, advanced imaging, and individualised surgical management. The postoperative improvement in bladder and bowel function after posterior stabilisation and decompression demonstrates the effectiveness of timely intervention. A thorough understanding of spinal injury pathophysiology, careful attention to subtle or delayed neurological symptoms, and the use of structured imaging and classification systems are essential for accurate diagnosis and optimal outcomes in patients with thoracolumbar spinal trauma.

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PARTICULARS OF CONTRIBUTORS:

1. Postgraduate Student, Department of Orthopaedics, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India.
2. Professor and Head, Department of Orthopaedics, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India.
3. Professor and Unit Chief, Department of Orthopaedics, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India.
4. Assistant Professor, Department of Orthopaedics, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India.
5. Postgraduate Student, Department of Orthopaedics, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
RS Monish Anand,
Department of Orthopaedics, SRM Medical College Hospital and Research Centre, Chennai, Tamil Nadu, India.
E-mail: rsmonishanand.17@gmail.com

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