ISSN 2689-8268| Volume 8

American Journal of Surgery and Clinical Case Reports

Case Report Open Access

Isolated Lumbar 4 Burst Fracture with Neurological Deficits: A Rare Case Treated with Posterior Lumbar 3-5 Fixation and Lumbar 4 Laminectomy

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Received: 18 Feb 2025
Accepted: 28 Feb 2025
Published: 05 Mar 2025
J Short Name: AJSCCR

Keywords:

Lumbar High Energy Trauma; Burst Fracture of Lumbar Vertebra;Isolated L4 Burst Fracture; Lower Lumbar Vertebrae Fracture; Burst Vertebral Body Fracture

Citation:

Bircesu Uslu. Isolated Lumbar 4 Burst Fracture with Neurological Deficits: A Rare Case Treated with Posterior Lumbar 3-5 Fixation and Lumbar 4 Laminectomy. Ame J Surg Clin Case Rep. 2025; 8(4): 1-6

1. Abstract

Due to the unique biomechanics of the lower vertebrae, which are protected by the iliolumbar ligaments, pelvis, and strong musculature, high-energy trauma rarely results in an isolated burst fracture of the fourth lumbar vertebra (L4). Moreover, an isolated L4 burst fracture causing neurological deficits is even rarer. Rarity of these fractures resulted in limited studies on treatment approaches and lack of treatment guidelines. In this case report, we discuss the surgical approach involving posterior L3-L5 fixation and L4 laminectomy, as well as its contribution to the patient's neurological recovery. A young adolescent male involved in a high energy vehicle accident was admitted to our clinic. The patient, who had an active lifestyle and no history of bone or spinal malformation, presented with neurological deficits in both lower extremities, as well as bladder and anal sphincter dysfunction. Due to the severity of the neurological deficits, surgical intervention was deemed necessary. Posterior fixation of L3-L5 and laminectomy of L4 were performed. The patient underwent an extensive four-month rehabilitation program with an appropriate diet programme. Postoperative progress was monitored from the time of the accident until four months after surgery. The patient was discharged four months after admission.

2. Introduction

Vertebral burst fractures typically result from high-energy trauma but can also occur in patients with osteoporosis. [1-2]. These fractures are caused by axial compression forces combined with a flexion moment, leading to a kyphotic deformity in the normally lordotic spine. This results in the failure of the anterior and middle spinal columns, causing the vertebral body to collapse [2]. The collapse is often accompanied by varying degrees of spinal canal invasion, which may lead to neurological compromise [3].Burst fractures most commonly affect the thoracolumbar spine, while low lumbar spine injuries are rare, accounting for only 1.2% of all spinal injuries [3-4]. Fractures in the lower lumbar spine have distinct biomechanical and anatomical characteristics that differentiate them from thoracolumbar junction fractures [5]. Due to these differences there is no one guideline for treatment and treatment approaches remain controversial. Although most thoracolumbar and lumbar burst fractures can be managed conservatively unless they are unstable and/or cause neurological deficits, cases with neurological deficits are harder to manage due to lack of consensus [4].The primary goals of surgical treatment are decompression of spinal canal and cauda equina in order to improve neurological function, as well as spinal stabilization to alleviate pain and restore mobility while maintaining sagittal and coronal balance. Surgical decompression is generally recommended in cases of major neurological deficits, progressive neurological loss, or significant spinal canal compromise [6,7]. However, the treatment of isolated L4 burst fractures with neurological deficits remains controversial [4].

3. Case Presentation

A 17-year-old male with a history of mitral valve defect treated with 5 mg enalapril was admitted to our clinic following a high energy vehicle accident that resulted in an isolated L4 burst fracture and neurological deficits in the lower extremities. The patient had an active lifestyle and no history of previous fractures, osteomalacia, rickets, growth defects, kyphosis, scoliosis, spinal or bone malformations.A computed tomography (CT) scan of the abdomen

revealed a burst fracture of the L4 vertebral body with 50% spinal canal stenosis, as well as non-displaced fractures of both of the L4 transverse processes. Magnetic resonance imaging (MRI) showed preserved lumbar lordosis but injury to the posterior ligamentous complex.Upon admission, neurological examination revealed no deficits in the upper extremities (5/5). However, deficits were present in both lower extremities, including impaired dorsiflexion of the ankles (L4) and toes (L5), as well as plantar flexion (S1). Additionally, sensory and motor losses in voluntary anal and bladder sphincter control were observed. Given the extent of the neurological deficits, surgical intervention was decided.The patient received surgical prophylaxis with cefazolin sodium and was placed under general anesthesia in the prone position. A midline incision was made over the L3-L5 vertebrae, and the paraspinal muscles were dissected to expose the posterior elements. The L4 lamina was carefully removed to decompress the spinal canal and L3-L5 fixation was performed. Four 6.5 mm polyaxial screws, two rods, and four nuts were used for stabilization. The wound was irrigated, and the muscles and skin were closed in layers.Postoperatively, the patient was fitted with a brace, the surgical wound was monitored and surgical stitches were removed fifteen days after the operation without any complication. The patient was prescribed 1000 mg levetiracetam daily, continued to take 5 mg enalapril and given a high protein high calorie diet. Nonsteroidal anti-inflammatory drugs (NSAIDs) were also administered daily to improve treatment compliance. No complications were observed during the follow-up period. Neurological improvement in the lower extremities was not immediately apparent after surgery. Four days postoperatively, the patient was transferred to a physical therapy and rehabilitation center.At the rehabilitation center, the patient underwent a comprehensive program that included range-of-motion exercises, strengthening exercises for the upper and lower extremities, ambulation training, balance coordination, robotic rehabilitation, electrical stimulation, and pelvic rehabilitation with a physiotherapist. Psychological support was also provided for the patient in the centre.Preliminary psychological evaluation showed the need for a psychological support programme. Although the patient showed very little interest in these sessions, he was seen and evaluated regularly by a psychiatrist but no psychiatric medication was prescribed.Two weeks postoperatively, a CT scan showed proper placement of the polyaxial screws with no loosening of the rods or new fractures. Six weeks after surgery, electromyography (EMG) was scheduled to evaluate the extent of spinal nerve damage and progress. EMG revealed ongoing degeneration of both L5 nerve roots, severe degeneration of the left S1 nerve root, and moderate degeneration of the right S1 nerve root.Four months after admission and an extensive rehabilitation program, the patient was discharged. Neurological findings at discharge included voluntary contraction of the anal and bladder sphincters, minor recovery of right ankle dorsiflexion (L4) (1/5), persistent right toe dorsiflexion deficit (L5) (0/5), full recovery of right plantar flexion (S1) (5/5), improved left ankle dorsiflexion (L4) (2/5), improved left toe dorsiflexion (L5) (2/5), and improved left plantar flexion (S1) (3/5). The patient was discharged with a prescription of high protein high energy formula and 1000 mg levetiracetam, given exercises and told to follow his post discharge orthopedic check ups.

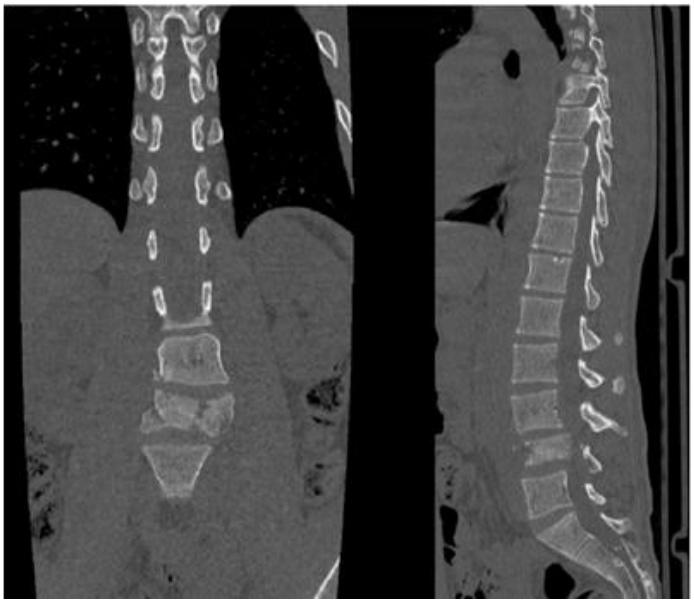

Figure 1: Preoperative CT coronal view and sagittal view respectively shows aburst fracture of L4 with more than⅓ of vertebral body height was affected.

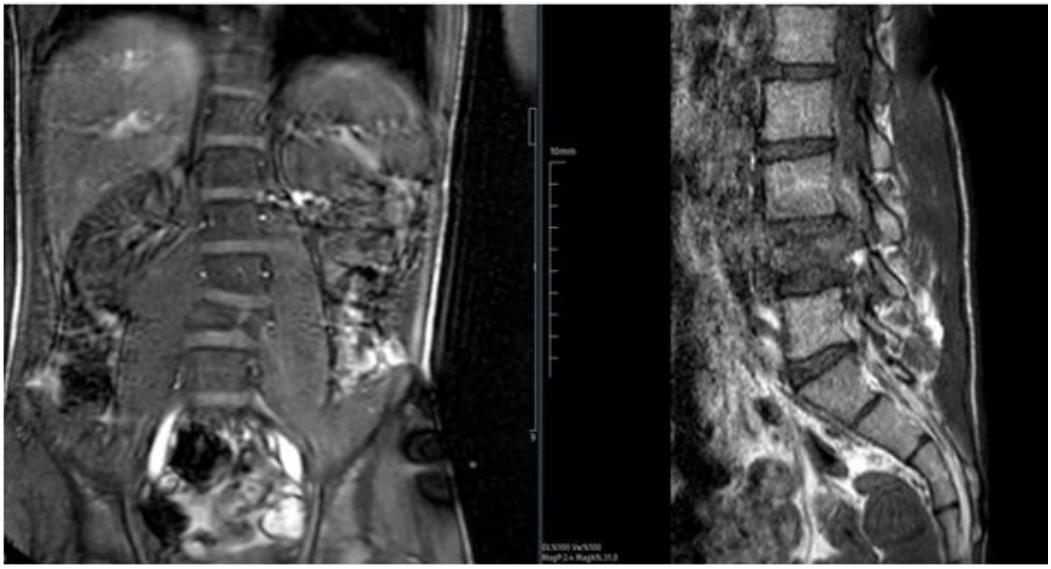

Figure 2: Preoperative MRI coronal view and sagittal view respectively showing protected lordosis of the lumbar spine and the damaged integrity of the posterior ligamentous complex.

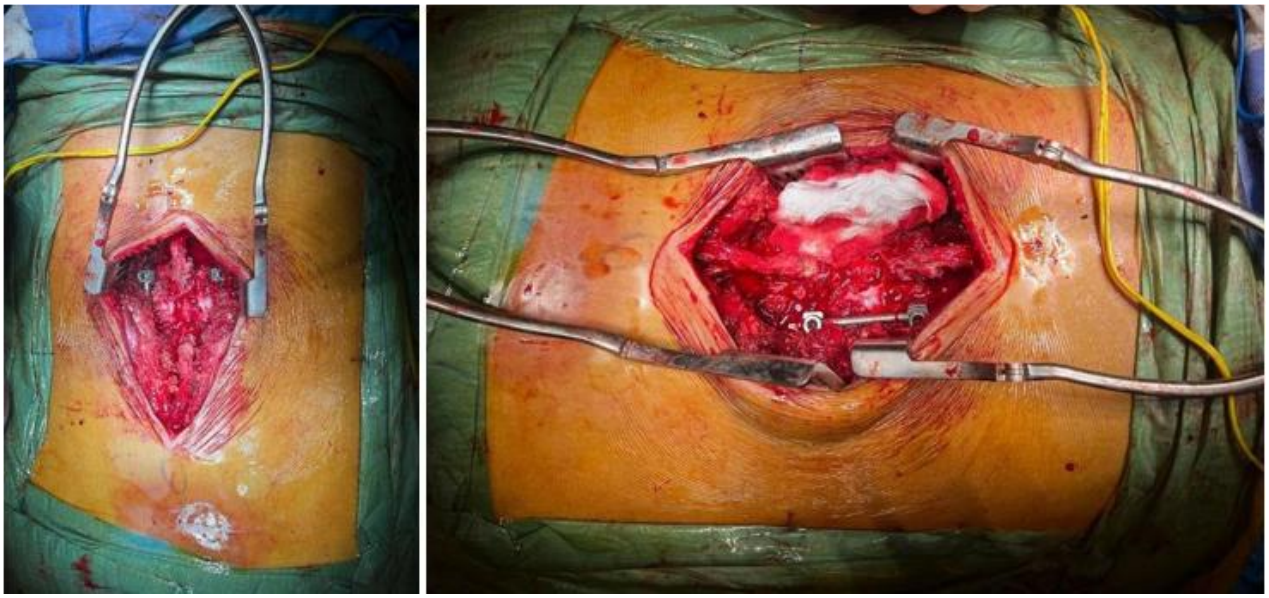

Figure 3: Intra operative pictures of the patient in proneposition,during posterior L3 and L5 fixation and laminectomy of L4 approach.

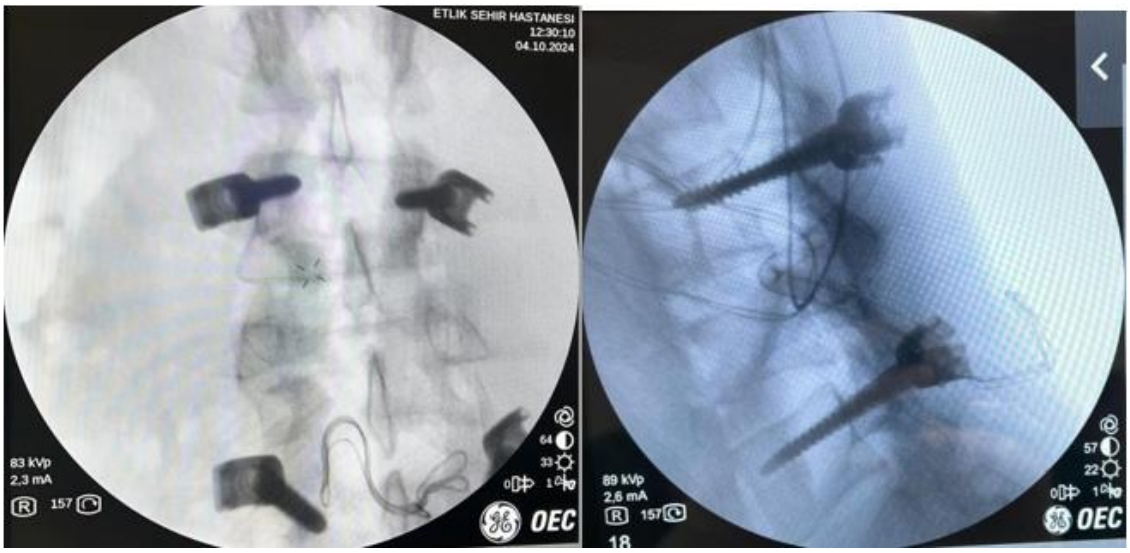

Figure 4: Intra operative fluoroscopy pictures (AP and lateral view respectively) showing four polyaxial screws.

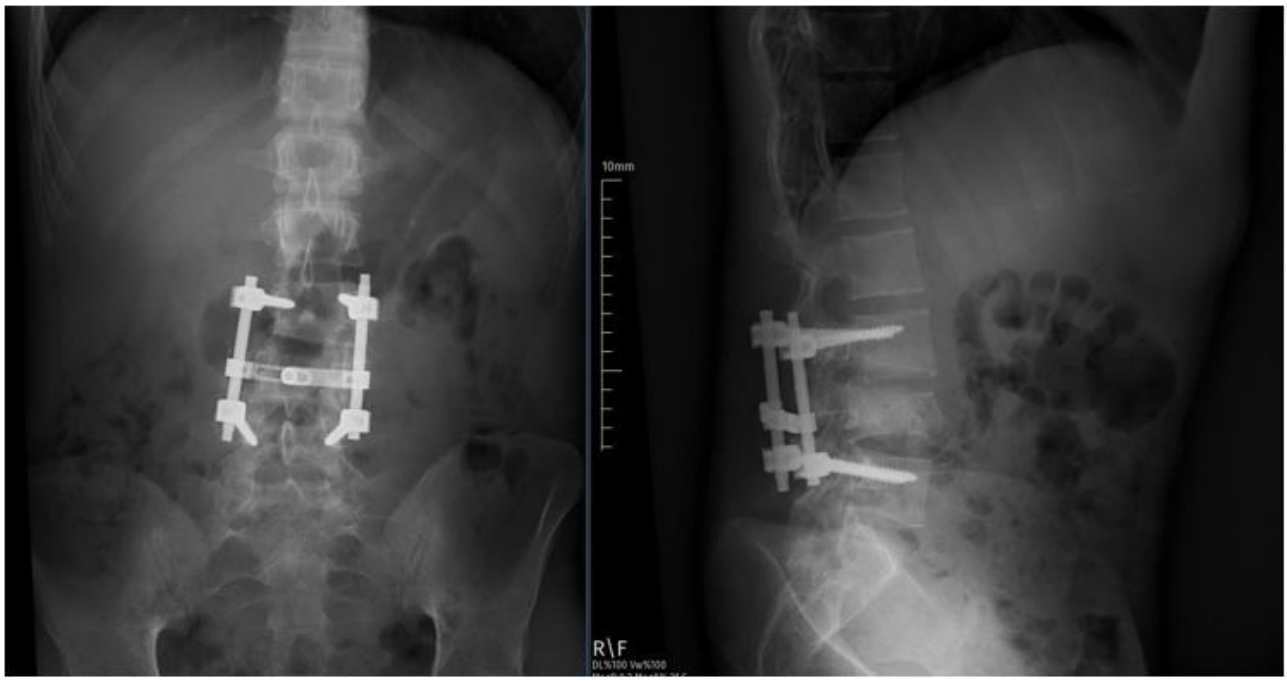

Figure 5: Post operative Xray images (AP and lateral view respectively) showing posterior L3 and L5 fixation and laminectomy of L4.

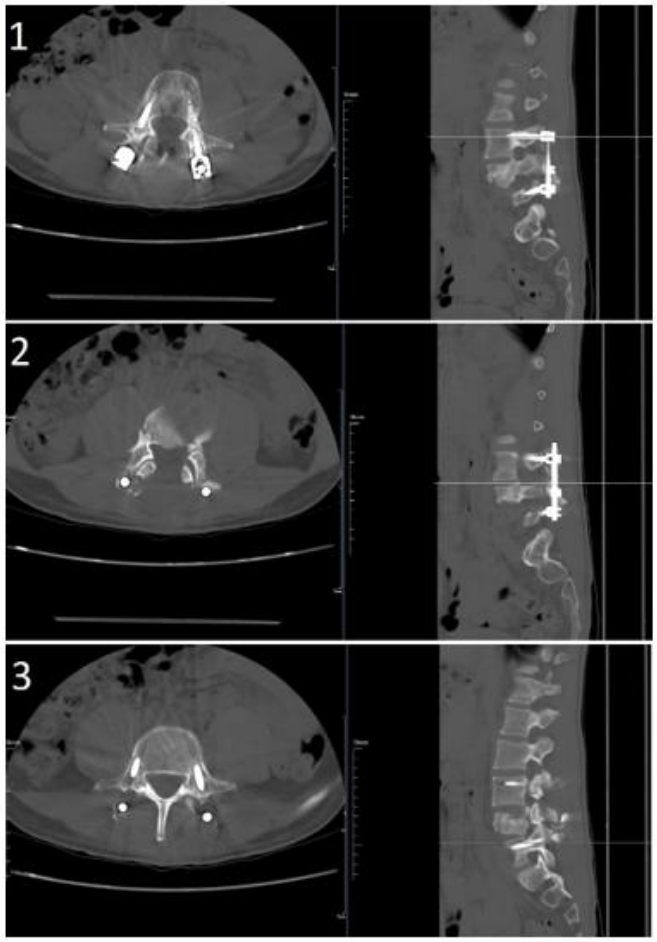

Figure 6: Post operative CT Scan (axial and sagittal view respectively) showing regained vertebral body height. Image 1 and 3showing posterior fixation of L3 and L5 respectively and Image2 showing laminectomy of L4.

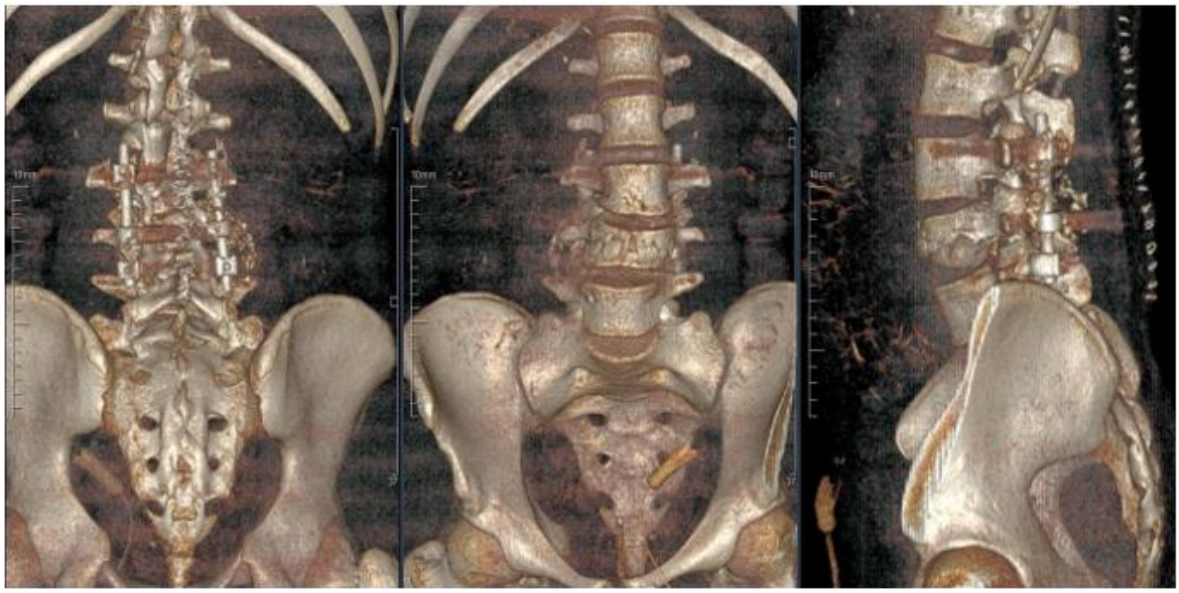

Figure 7: Postoperative Multislice 3D CT scan images (posterior, anterior and lateral views respectively) showing posterior L3 and L5 fixation and laminectomy of L4 and proper placement of the polyaxial screws with no loosening.

4. Discussion

Vertebral burst fractures are typically caused by high-energy trauma such as falling or traffic accidents but can also occur in osteoporotic patients [4]. The incidence of lumbar vertebral fractures has increased by 66.3% over the past decade. However, lower lumbar burst fractures, particularly at L4 and L5, account for only about 1% of all lumbar spine fractures [5]. The most common causes of lumbar vertebral fractures between 2010 and 2018 were falls from standing height (20.9%), falls involving stairs or steps (12.6%), and falls from ladders (8.1%). Together, these mechanisms accounted for 41.6% of all lumbar vertebral fractures during this period [8].Several classification systems, including the Denis, AO Spine, and Thoracolumbar Injury Classification System (TLICS), are used to evaluate spinal injuries and determine the need for surgical intervention [9]. In this case, the patient scored 7 on the TLICS, corresponding to group B of the Denis classification [11] and A4 of the AO classification. [12]. Although these classification systems give a general idea when surgical intervention is needed, there is no agreed consensus on when or which surgical intervention is the most suitable treatment option.The L4 and L5 vertebrae play a critical role in axial weight-bearing and maintaining lumbar lordosis [13]. The lower lumbar spine is protected by the iliolumbar ligaments, pelvis, and strong musculature, making it less susceptible to collapse or kyphosis compared to the thoracolumbar junction. Additionally, the wide neural canal in the lower lumbar region reduces the risk of cauda equina damage, potentially improving recovery rates [14]. However, these biomechanical properties also make the treatment of lower lumbar burst fractures more challenging [15,16].The rarity of L4 fractures has resulted in limited documentation in the literature. Most studies focus on thoracolumbar junction fractures, leaving the management of low lumbar burst fractures poorly defined [17-19]. Treatment strategies must be tailored to the unique characteristics of the lower lumbar spine, as recommendations for thoracolumbar trauma may not apply [20-21]. The ideal treatment for thoracolumbar and lumbar fractures remains controversial. (19, 22) Conservative management, including bed rest, bracing, and gradual ambulation, is effective for neurologically intact patients with low lumbar burst fractures [5,22]. However, surgical decompression and stabilization are clearly indicated for patients with neurological deficits at the thoracolumbar junction [5,18,23]. Whether these principles apply to the lower lumbar spine, which contains only nerve roots, remains unclear [24-25].

4.1. Treatment 0f L4 and L5 Burst Fractures often Requires an Anterior or Posterior Approach.

However, anterior approaches to these levels are challenging due to the proximity of the aorta and inferior vena cava [26]. A posterior approach with removal of retropulsed fragments can eliminate the need for anterior surgery, reducing morbidity compared to a two-stage procedure [27]. However, there is insufficient evidence to support the superiority of one surgical technique over another in terms of morbidity [7].Retrospective studies suggest that long instrumentations and fusions should be avoided in patients with low lumbar burst fractures, regardless of neurological status. Historically, long instrumentation in this region has been associated with high rates of pseudarthrosis, implant failure, and flat-back deformity, despite the benefits of early ambulation and anatomical reduction [19, 28].Even after taking all but limited studies into consideration it is not clear exact treatment approaches for the L4 burst fractures.

Limitations of the study need to be taken into consideration. Although this case report is important to show a rare situation, it is limited by its single-patient design. Larger studies are needed

to establish definitive treatment guidelines for isolated L4 burst fractures.

5. Conclusion

L4 burst fractures are typically caused by high-energy trauma, such as falls or motor vehicle accidents. Due to the protective biomechanics of the lower lumbar spine, fractures at this level are rare, accounting for a small percentage of all spinal fractures. In addition the wide spinal canal in the lower lumbar region reduces the risk of cauda equina damage. These unique biomechanics of this area make treatment challenging.The rarity of isolated L4 burst fractures has resulted in limited studies on their management and outcomes. Studies usually focus on burst fractures thoracic and upper lumbar levels of vertebrae. This case highlights a rare phenomenonpartial spinal cord injury caused by an isolated L4 burst fracture secondary to high-energy traumaand demonstrates the surgical treatment, postoperative physical therapy and four-month follow-up of such an injury.

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