J Orthop Spine Trauma. 2016 June; 2(2):e8060. doi: 10.17795/jost-8060.

Published online 2016 June 29. **Case Report**

Late Onset of Neurogenic Complications in Single Neglected Unstable L2 A4/AO Type Fracture: Two Case Reports and Review of the Literature

Papaioannou Ioannis,[1,*] Baikousis Andreas,[1] and Korovessis Panagiotis[1]

[1]Spine Unit, Department of Orthopaedics, General Hospital of Patras, Patras, Greece

[*]*Corresponding author*: Papaioannou Ioannis, Spine Unit, Department of Orthopaedics, General Hospital of Patras, Patras, Greece. Tel: +30-2610007532, Fax: +30-2610227681, E-mail: john-pane1984@hotmail.com

Received 2016 April 30; **Accepted** 2016 June 01.

Abstract

Introduction: Late-onset neurological impairment in unstable A3-A4/AO type thoracolumbar spinal fractures are rare, particularly if they are misdiagnosed as stable (25%). We present two neglected unstable burst fractures, who presented delayed trauma with neurologic deficit.

Case Presentation: Case 1: A 62-year-old male, who sought our department three months after trauma claiming neurogenic claudication, following fracture of L2.

Case 2: A 50-year old male, who had a jerky fall from his vehicle with subsequent back pain for a few days. One-month after the trauma he presented urinary retention.

Conclusions: Unstable thoracolumbar A4 /AO type fractures, if left untreated or misdiagnosed, lead to vertebral body fragments retropulsed to the spinal canal under axial and bending forces resulting in progressively increased encroachment of the canal and finally compression on to cauda equina. Early recognition of an unstable A3-A4 /AO-type thoracolumbar fracture with C/T scan is mandatory along with appropriate treatment. This should be done in all cases, even in these without any neurological deficit and these with "normal" plain X-rays, because misdiagnosis and late onset neurological deficit is always a possibility. Physicians should be aware of this complication when treating thoracolumbar spine injuries with minor or absent symptoms and physiological neurologic findings.

Keywords: Thoracolumbar A3-A4/Type Fracture, Late Onset Neurologic Deficits, Decompression, Stabilization, MRI or C/T Scan

1. Introduction

It has been reported that 10 to 20% of fractures occurring in the thoracolumbar region are A3/AO type burst fractures (1). Neurologic impairment is associated with A3/AO fractures in about half of the cases. Posterior vertebral body wall fracture in A3/AO fractures is usually missing, in plain roentgenograms taken at the emergency room in about 25% of the cases because it is hidden by the wide lumbar vertebra pedicles (2). We report on two cases with delayed appearance of neurologic impairment in neglected/misdiagnosed unstable A3-A4/AO thoracolumbar fractures.

2. Case Presentation

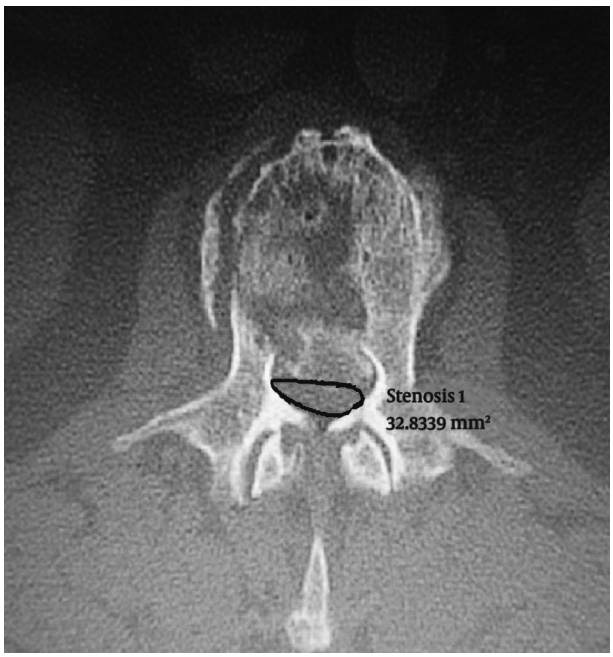
2.1. Case 1

A 62-year-old male, who worked as a builder, referred to our department with neurogenic claudication. The patient reported a fall from a 2-m height three months ago, for which he sought another physician, but no fracture was diagnosed at that time, on poor quality plain roentgenograms. On admission, the physical examination revealed motor weakness (2/5) of the hip flexors, bilaterally. Furthermore, the patient was examined by new roentgenograms and Magnetic Resonance Imaging (MRI) scan, in which the fracture was now obvious. Computerized Tomography (CT)-scan of thoracolumbar spine disclosed a A3-A4/AO type fracture of L2 vertebra with significant body comminution (LSS score 6) of the associated vertebra, and 62.5% encroachment of the spinal canal at this level (Figure 1). Since the L2 fracture was already healed in kyphotic deformation, a wide posterior decompression and stabilization with segmental pedicle screw fixation from T12 to L4 vertebra was made. Postoperative roentgenograms and CT scan showed a wide spinal decompression and excellent alignment of the lumbar spine.

2.2. Case 2

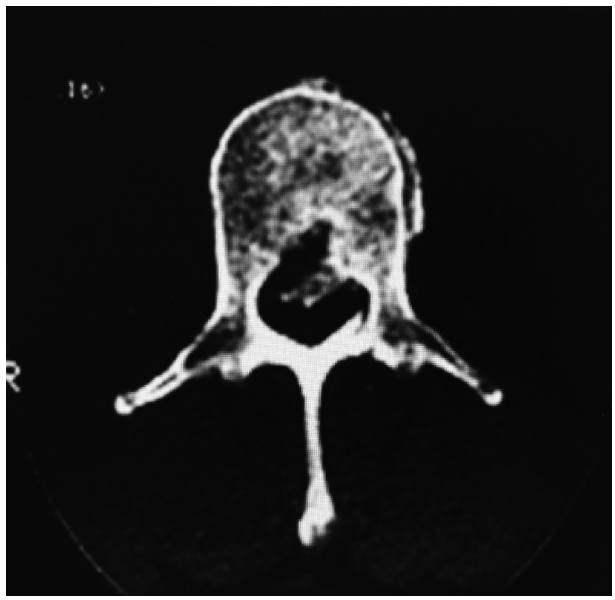
A 50-year-old male truck driver, who had jumped from his track from a 2.5-meter height on his legs one month

Figure 1. Case No. 1



Axial computerized tomography scan view showing a remarkable spinal canal encroachment of 62.5% due to retropulsion of the comminuted vertebral body fragments.

Figure 2. Case No. 2



Axial computerized tomography scan view showing a remarkable spinal canal encroachment.

before his presentation. He had for a while some back discomfort and he considered this trauma as insignificant, and had been taking painkillers. Three weeks following trauma, our patient faced micturition difficulties and thought he had some prostate disorder and visited an urologist, who inserted a bladder Foley catheter but no prostate disorder was diagnosed. On presentation, plain lateral roentgenogram of the spine revealed a A3-A/AO fracture of the L2 vertebral body and CT-scan showed a significant comminution (LSS: 7) with significant encroachment of the canal (Figure 2).

The neurological examination disclosed perianal hypoesthesia by means of saddle hypoesthesia and muscular weakness (4/5) of hip flexors. Anal sphincter was unspoiled. An anterior left-sided retroperitoneal anterior decompression at L2 and stabilization with Kaneda device and autogenous fibula grafts from L1 to L3 vertebra was made. Motor and sensor deficits recovered after four to six months and urinary retention recovered completely six months following decompression. Axial C/T scan of the spine showed wide decompression of the spinal canal on the left side.

3. Discussion

Many systems exist to classify thoracolumbar A3/AO burst fractures. Typically, a fracture is considered unstable if it is associated with neurologic deficits, high risk of deformity progression, or disruption of all three columns. While most thoracolumbar fractures can be treated nonoperatively with bed rest or brace (3), unstable fractures may need surgical stabilization, especially those with neurologic impairment (4). Good long-term results of nonoperative treatment, such as less back pain, satisfactory work status, and lack of neurologic deterioration or complications have been reported in details (5, 6).

Operative treatment may reduce the possibility for kyphotic deformity or loss of vertebral height (7). However, neurological deterioration and cauda equine in association with a lumbar fracture is a clear indication for decompression and stabilization (8). Our search of the literature indicated that delayed appearance of neurological symptoms and deficits in patients with lumbar burst fractures was negative. In cases presented in this report the initial spinal canal encroachment progressively increased, mainly for mechanical reasons, including primary instability due to the type of the fracture, A4AO/type, that involves both endplates and the posterior wall with significant comminution, and bending forces applied across the unprotected (no brace, no operation) mobile lumbar

spine. As a result of acting mechanical forces, the nucleus pulposus in adjacent segments may later herniate through the fractured or not united vertebral endplate, resulting in anterior vertebral column insufficiency, progressive collapse and finally failure or increased spinal canal encroachment and compression on to the cauda roots (9).

Due to the mechanical importance of having support in the anterior column, a load-sharing classification (LSS) was developed to grade the comminution of thoracolumbar fractures, and provide guidance about the need to provide anterior support (10). Load-sharing classification is a point system that amounts 3 to 9 degrees and grades: 1) the amount of damaged vertebral body, 2) the spread of the fragments in the fracture site and 3) the amount of corrected traumatic kyphosis. This point system can 1) predict screw breakage when short segment pedicle screw fixation are being used, 2) describe any spinal injury for retrospective studies and 3) select spinal fractures for anterior reconstruction. The crucial number is 6. Fractures with LSS > 6 require anterior reconstruction.

The theoretical reasons for the lack of accurate diagnosis in these cases was the absence of 1) significant pain, 2) neurological complaints/findings and 3) imaging findings immediately after trauma (CT-scan and MRI). Neurological damage is believed to occur during spinal trauma and is not always dependent on the amount of spinal canal encroachment in the lumbar spine. However, because of the wide spinal canal at the level of the L2 vertebra and probably initial low encroachment of its cross-sectional area by the fragments, the neurological impairment signs appeared delayed, one to three months post-injury, when there was an increase of the spinal canal encroachment with significant pressure on the cauda.

Physicians should be aware of this complication when treating thoracolumbar spine injuries with minor or absent symptoms and physiological neurologic findings. Even with suspicion of unstable burst fracture in cases with “negative” plain roentgenograms, CT or MRI should always be performed. If CT or MRI scan are not available in an institution, the patient should be advised, protected with a brace, and transferred to a tertiary hospital.

Nowadays, the accurate diagnosis of thoracolumbar injuries must be confirmed immediately on admission, regardless of the presence of neurological impairment and/or significant pain with CT-scan or MRI. Late onset of neurological deficit can occur either due to misdiagnosis or poor initial conservative treatment. In case of non neurological impaired patients, open or MIS posterior pedicle screw fixation plus restoration of the anterior column disruption with vertebroplasty/kyphoplasty in cases with LSS > 6, is recommended by the authors. If a neurological deficit occurs, surgical decompression and stabilization is the gold standard in these cases.

Footnote

Authors’ Contribution: Study concept and design, Panagiotis Korovessis, Andreas Baikousis and Ioannis Papaioannou; drafting of the manuscript, Ioannis Papaioannou and Panagiotis Korovessis; critical revision of the manuscript, Panagiotis Korovessis.

References

1. Gertzbein SD. Scoliosis Research Society. Multicenter spine fracture study. *Spine.* 1992;**17**(5):528–40. [PubMed: 1621152].
2. Ballock RT, Mackersie R, Abitbol JJ, Cervilla V, Resnick D, Garfin SR. Can burst fractures be predicted from plain radiographs?. *J Bone Joint Surg Br.* 1992;**74**(1):147–50. [PubMed: 1732246].
3. Thomas KC, Bailey CS, Dvorak MF, Kwon B, Fisher C. Comparison of operative and nonoperative treatment for thoracolumbar burst fractures in patients without neurological deficit: a systematic review. *J Neurosurg Spine.* 2006;**4**(5):351–8. doi: 10.3171/spi.2006.4.5.351. [PubMed: 16703901].
4. Lim TH, An HS, Hong JH, Ahn JY, You JW, Eck J, et al. Biomechanical evaluation of anterior and posterior fixations in an unstable calf spine model. *Spine.* 1997;**22**(3):261–6. [PubMed: 9051887].
5. Cantor JB, Lebwohl NH, Garvey T, Eismont FJ. Nonoperative management of stable thoracolumbar burst fractures with early ambulation and bracing. *Spine.* 1993;**18**(8):971–6. [PubMed: 8367784].
6. Mumford J, Weinstein JN, Spratt KF, Goel VK. Thoracolumbar burst fractures. The clinical efficacy and outcome of nonoperative management. *Spine.* 1993;**18**(8):955–70. [PubMed: 8367783].
7. Gnanenthiran SR, Adie S, Harris IA. Nonoperative versus operative treatment for thoracolumbar burst fractures without neurologic deficit: a meta-analysis. *Clin Orthop Relat Res.* 2012;**470**(2):567–77. doi: 10.1007/s11999-011-2157-7. [PubMed: 22057820].
8. Lee JY, Vaccaro AR, Lim MR, Oner FC, Hulbert RJ, Hedlund R, et al. Thoracolumbar injury classification and severity score: a new paradigm for the treatment of thoracolumbar spine trauma. *J Orthop Sci.* 2005;**10**(6):671–5. doi: 10.1007/s00776-005-0956-y. [PubMed: 16307197].
9. Oner FC, van Gils AP, Dhert WJ, Verbout AJ. MRI findings of thoracolumbar spine fractures: a categorisation based on MRI examinations of 100 fractures. *Skeletal Radiol.* 1999;**28**(8):433–43. [PubMed: 10486011].
10. McCormack T, Karaikovic E, Gaines RW. The load sharing classification of spine fractures. *Spine.* 1994;**19**(15):1741–4. [PubMed: 7973969].