



## Ascending Myelitis in Operated Burst Fracture L1 with Paraplegia Asia

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### Abstract:

A 46 years female sustained post-traumatic paraplegia with burst-fracture L1, with ASIA-A neurology, and underwent surgical decompression and stabilization. On 4<sup>th</sup> post-operative day, patient developed ascending myelopathy with neurological deterioration upto C5, along with fever, neck rigidity, high counts, with MRI showing myelitis. An aggressive antibiotic therapy was started. Blood counts, fever and neck rigidity settled with a delayed partial neurological recovery at 17 weeks. Infection could not be confirmed as the cause of the ascending myelitis even though the patient responded to antibiotics. Ascending sympathetic myelitis was a differential, which though rare, is reported.

**Key words:** Bone Marrow Diseases, Myelitis, Neurology, Paraplegia, Surgical Decompression.

### Introduction

Post-traumatic subacute ascending neurological deterioration unrelated to mechanical instability is a rare complication of spinal trauma. The most common type of neurological deterioration is a rise in the level of the lesion by one and sometimes by two segments usually due to cord edema usually occurring within the first four days, and the deterioration is almost always temporary [1].

A rare form of deterioration is when a spinal cord lesion corresponding to the bony injury ascends by several segments in the first three weeks after the original injury [1]. Sub-acute postoperative ascending myelopathy presents as neurological deterioration between day one to four weeks of initial spinal cord injury [2]. It presents a diagnostic

dilemma to the treating surgeon with emphasis on early diagnosis and prompt treatment.

### Case Report

A 46 years healthy female presented to us following a fall from height. X-rays and MRI revealed burst fracture L1 with ASIA A paraplegia [Fig.1-4]; neurologically patient had a motor level of L1 with loss of sensations below L1 dermatome. Cervical spine injury was ruled out at the time of admission. Posterior stabilization was performed using posterior pedicle screw and rod system from D11 to L3 [Fig.5]. Immediate post-operative period was uneventful with no neurological deterioration.

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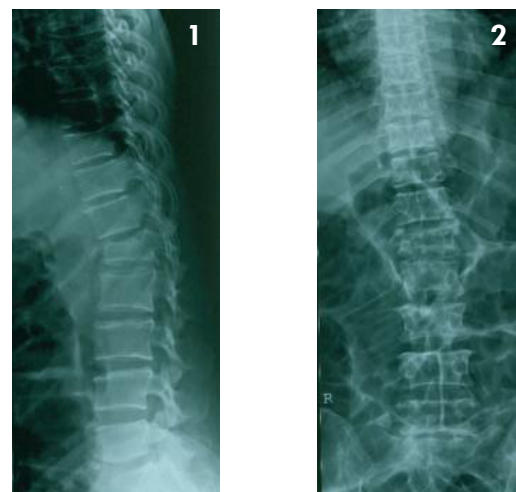
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On the 4<sup>th</sup> post-operative day, patient developed weakness of upper limbs with fever and neck rigidity. Neurological level ascended to C5. Blood investigations showed an increase in inflammatory markers. MRI showed signs of ascending myelitis, with diffuse hyper-intense signal extending from C3 upto conus [Fig.6], and no evidence of hemorrhage or syringomyelia. CSF study revealed pleocytosis with marked increase in protein levels. Gram staining was negative, and culture showed no growth.

An aggressive antibiotic therapy consisting of broad spectrum antibiotics was started, and continued for 3 weeks. Blood counts and neck rigidity settled, but upper limb neurological deficit recovered only partially. Patient was discharged for domiciliary care after 6 weeks of surgery, with no further antibiotic cover. No steroids were administered in this course of treatment. Patient was reviewed 17 weeks after the surgery, her upper limb power had dramatically improved. The MRI showed subsidence of myelitis [Fig.7]

## Discussion

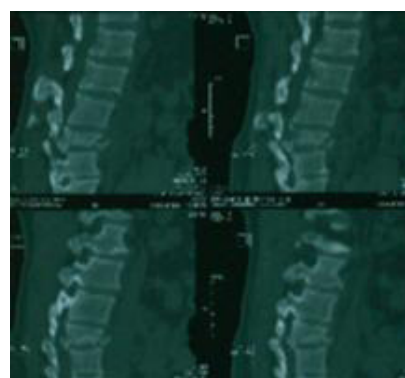
Neurological deterioration in the acute post-injury period is rare and is often the result of mechanical instability from improper immobilization or as a result of a surgical complication. The amount of energy delivered to the cord at the time of injury results in mechanical disruption of axons, interruption of normal spinal cord blood flow and initiation of several different pathways of secondary insult to an already damaged cord. The effect of this secondary insult may be acute in onset and dramatic in nature. Studies have reported similar cases in the past with rarity. Frankle [1] described eight patients with fractures of the lower thoracic spine presenting similarly within 2-11 days after injury. Belanger *et al.* [3] reported ascending neurological deficit in three patients with spine injuries starting 7-13 days after injury. One of their cases had a fracture



**Fig.(1):** Burst fracture L1. **(2):** Burst fracture L1.



**Fig.3:** MR picture of the L1 burst fracture.



**Fig.4:** CT images of the fracture.



**Fig.5:** Postoperative X-rays showing well stabilized fracture.



**Fig.6:** MR images showing signs of ascending myelitis.

L1 similar to our case. Since a majority of these patients had injury near the dorsolumbar junction Aito *et al.* [4] considered a post-traumatic thrombus of the Great artery of Adamkiewicz. In all of these cases blood counts were normal contrary to our finding of increased inflammatory markers in our patient. However the blood cultures were reported as sterile even after seven weeks.

Scmidt *et al.* [5] described sub-acute delayed ascending neurological deficit in a 35 year old lady with fracture T11/T12 with myelopathy ascending up to C5, and concluded that after a spinal cord injury factors exacerbating spinal venous hypertension and/or arterial hypotension may in some patients lead to an impaired spinal perfusion sufficient enough to produce an ischemic insult at higher levels. Most of the reported patients showed partial recovery without any specific treatment, with a repeat MRI showing decrease of the T2 weighted signal abnormality. More recently, Farooque *et al.* [6] described a similar presentation in two post-surgical decompression patients, and suggested that the possible cause of ascending neurological level in postoperative cases could be due to



**Fig.7:** MR image at 17 weeks showing resolution of myelitis.

inadvertent cord handling during decompression, which could trigger cascade of events leading to ascending myelopathy. They reported that both the patients in their series responded to steroids administered according to the NASCIS II protocol, while one showed complete recovery; the other had some residual weakness in the upper limbs.

Planner *et al.* [7] described the characteristic MR imaging findings of sub-acute progressive ascending myelopathy as (i) a central area of hyperintense signal on T2-weighted sequences, which tapers at the rostral end of the lesion, (ii) heterogeneous intramedullary signal on T1-weighted sequences, and (iii) an expanded spinal cord. Follow up imaging reveals absence of any syrinx, and the cord appears atrophic. A wide range of conservative treatments have been suggested in the literature including anticoagulation, decompression, steroids [3] and mannitol. Surgical measures like dural untethering and cordectomy have also been described [8,9]. Our patient was not administered any steroids, and gradually recovered on a three week broad spectrum antibiotic therapy, which we do not know whether was warranted or not.

## Conclusion

We were not able to confirm infection as the cause of the ascending myelitis with ascending neurological deficit, even though the patient responded to antibiotic therapy. It could still be a case of ascending sympathetic myelitis, which though rare, is reported. The purpose of this case report was to describe this interesting incident of ascending neurological deficit postoperatively in a patient who initially sustained paraplegia.

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