International Journal of Research in Orthopaedics
Shenoy S et al. Int J Res Orthop. 2025 Jan;11(1):242-246
http://www.ijoro.org

DOI: https://dx.doi.org/10.18203/issn.2455-4510.IntJResOrthop20243916

Case Report

Sub-acute post traumatic ascending myelopathy with superimposed infection in a diabetic: a case report

Shreya Shenoy[1]*, C. S. Vishnu Prasath[2], Manish R.[2], Sargunan Bala[2]

[1]Department of Orthopaedics, SKS Hospital and Postgraduate Medical Institute, Salem, Tamil Nadu, India
[2]Department of Spine Surgery, SKS Hospital and Postgraduate Medical Institute, Salem, Tamil Nadu, India

Received: 04 November 2024
Accepted: 04 December 2024

***Correspondence:**
Dr. Shreya Shenoy,
E-mail: shreyasemail2@gmail.com

ABSTRACT

Subacute posttraumatic ascending myelopathy (SPAM) is a rare entity associated with trauma to the spinal cord, usually at the level of the thoracic spine. Patient presents with neurological deterioration 4 or more levels above the site of primary injury. It usually follows an episode of fever unrelated to infection. A 43-year-old diabetic male patient with history of blunt trauma to thoracic spine with T5 flexion distraction injury with ASIA A neurology, underwent T3-T6 posterior instrumented stabilisation. Four weeks following this, he presented with signs of encephalopathy with CSF culture showing *Acinetobacter baumanii.* Though the patient had improvement with culture- sensitivity appropriate antibiotics, he developed ascending neurological deficits on day 3 of admission. On repeat MRI, he showed cord oedema up to C3. He was started on steroids and showed remarkable clinical improvement. SPAM must be kept as a differential in the diagnosis of a postoperative event with similar clinical picture.

Keywords: SPAM, Thoracic spine trauma, Neurological deficit, Post operative complications

INTRODUCTION

Five percent of all spinal trauma result in acute neurological deterioration.[1] Of these 0.4% to 1% are not attributable to the initial pathology.[2] This clinical presentation is of an ascending myelopathy in the patient which usually worsens during the first few days to weeks post operatively.[2] Occurring between day 1 to week 4 postoperatively, it is referred to as subacute post traumatic ascending myelopathy (SPAM). Initially described by Frenkel, there are very few cases of SPAM reported world over.[3,4] Most commonly reported in young adults to middle-aged patients, the epidemiology of SPAM and risk factors are yet to be deciphered.[4] The patient is reported to present with worsening neurological deterioration often ascending above the level of fixation and including higher dermatomes and myotomes, posing a diagnostic dilemma. Usually, the episode follows a fever spike in the immediate post operative period.[5] SPAM has a mortality rate of 10.34%, making it a dreaded diagnosis.[6,7] Several attempts have been made by scientists to define the phenomenon of SPAM and propose the pathomechanisms. Due to the lower incidence and few reports on the phenomenon, the aetiology and risk factors are unclear.[5] Diagnosis is often missed.

Here, we present an unusual case of SPAM in a 43-year-old diabetic.

CASE REPORT

Clinical presentation and diagnosis

A 43- year-old male patient, a native of Tamil Nadu, South India, with alleged history of fall of heavy blunt object over the mid back in August 2013 and sustained flexion distraction injury at the T5 level. Preoperatively, he had ASIA A neurology with complete motor and sensory deficits below the level of T5 (Figure 1). He was operated for the same wherein T4-T6 decompression and posterior

instrumented stabilisation was done in August 2013 (Figure 2). Perioperative period was uneventful, and he was discharged from the hospital on post operative day 5. Patient presented to us in September 2013, 4 weeks post operatively, with complaints of confusion, disorientation, drowsiness and fever for one day duration. Patient is a known case of type 2 diabetes mellitus. On clinical examination, he had a Glasgow coma scale (GCS) of 14/15 (E4V4M6), altered sensorium with raised temperature. local examination showed Upper limb power to be 5/5, while lower limb power was 0/5 (Medical research council scale). There were no signs of meningeal irritation. Patient was admitted to the intensive care unit (ICU). Infection work-up showed raised total WBC counts to15880 cells/µL, raised C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) (120 mm/hr), a haemoglobin level of 7.7 g/dl and a serum sodium of 128 mmol/L. Baseline liver and renal function tests were found to be normal. With the working diagnosis of encephalitis, patient was started on empirical antibiotics after obtaining blood and CSF for culture and on supportive treatment. On day 3 of admission, patient's GCS improved to 15/15(E4V5M6) and temperature came down to the normal level. Primary blood culture and cerebrospinal fluid (CSF) cultures came back negative. CSF was sent for the syndromic evaluation system wherein *Acinetobacter baumanii* was detected. Patient was started on meropenem as per sensitivity pattern.[8]

On day 4, patient had reduced ESR and CRP values along with return of consciousness. Though, there were signs of resolution of infection, patient developed upper limb neurological deficit. Upper limb power deteriorated from C5, C6 5/5 to 3/5 and C7, C8, T1 from 5/5 to 0/5 (Motor power scale). This prompted us to repeat a magnetic resonance imaging (MRI) for the cervico-thoracic spine. The repeat MRI showed cord oedema at the cervical level C3-T1. Considering the patient's deterioration, ascend of the neurodeficit 6 levels above the pathology, not explained by the encephalopathy, we made a diagnosis of SPAM.

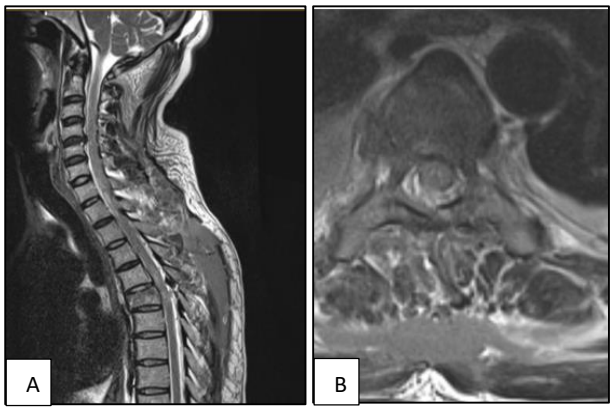

Figure 1 (A-B): Preoperative T2 weighted section of the MRI showing flexion distraction injury at the T5 level. Sagittal section and axial section showing the involvement of all three columns of the spinal cord.

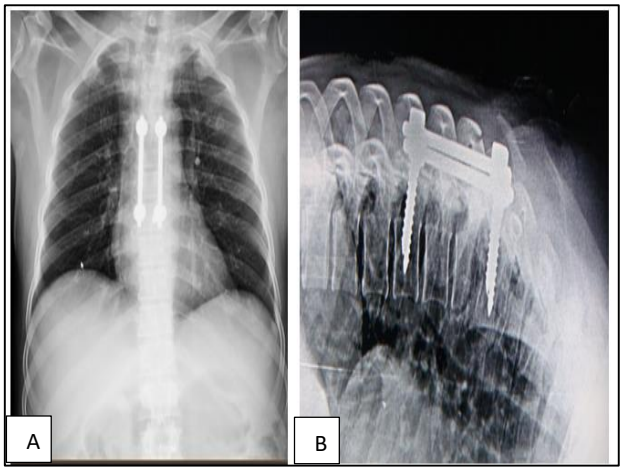

Figure 2 (A and B): Post operative radiograph showing fixation from T3-T6. Antero-posterior and lateral view showing a posterior spinal construct with pedicle screws at T3 and T6 level connected by a rod.

Management

Patient was started on Injection methylprednisolone treatment at a dose of 30 mg/kg 15-minute bolus followed by 24-hour infusion at 5.4 mg/kg/hour and on day 7, patient started showing signs of clinical improvement. On discharge patient was advised rehabilitation for upper limb and wheel-chair mobilisation.

Follow up

At 3 months follow up, patient had complete return of upper limb function and a motor power of 5/5. Patient is currently wheelchair bound and has complete function of the upper limbs.

DISCUSSION

Pathogenesis of SPAM

The patho-mechanism of development of SPAM is yet unclear.[9] However multiple theories exist in literature which attributes the ascending myelopathy to various causes. In a sick spinal cord, the presence of good blood supply is extremely important for healing. In the event of a high velocity trauma, there is incidence of arterial/venous congestion leading to decreased blood supply in the region. The vascular hypothesis put forward in the early 2000s, suggest that thrombosis rather than haemorrhage is the cause of loss of blood supply to the affected spinal region.[10] Schmidt proposed the theory of arterial hypotension and venous hypertension around the traumatic event being the major cause of hypoperfusion leading to hypo vascularity and myelomalacia.[10] Systemic hypotension during surgery and postoperative period can also lead to the onset of this phenomenon.[11] Another theory suggests the elevation of CSF pressure in the cord is a cause of ascending myelopathy.[12] Multiple studies have supported this statement with the help of imaging studies

and pressure analysis. A change in cerebrospinal fluid dynamics is the major cause of neurological dysfunction.[9,13] Moreover, the increased CSF pressure also causes ischemia to the spinal segment.[12] Patients' clinical presentation range from complete neurological worsening to partial neurological deficits. Ascending myelopathy is defined for the neurological deficits for 4 segments or above.[9] Additionally, there have been theories that infection is another precipitant of ascending myelopathy. Fever and rise in leucocyte count can activate inflammation leading to precipitation of SPAM.[2,5,14,15] Similarly, in our patient, the initial encephalitis might have been the precipitating factor. There have also been other proposed aetiologies for the condition like apoptosis, double level spinal trauma, autoimmune inflammatory reaction and meningitis, myelitis.[12,13]

The neurological defects are reversible 60% of the time. However, there have been instances of death in the cases of SPAM, up to 10% of the time.[6] The condition has been called pre-syrinx stage in the pathogenesis of spinal cord injury.[16] Some authors consider the surgical stress as an additional "second-hit" to the preexisting pathology.[6] Although the cases reported are rare, recent reviews predict the risk factors for SPAM being persistent systemic hypotension, trauma or atraumatic event at the thoracic level, orthostatic hypotension, previous CSF disorders, inadvertent early postoperative mobilisation leading to a hypotensive event.[2,10,12,17,18] Controversially, the literature also reports that patients who undergo posterior instrumented stabilisation have a lesser incidence of neurological deterioration as opposed to non-operative management, despite the secondary insult that accompanies the surgery.[19] This is because the stable environment provided by the fixation for the spinal cord, thereby preventing progression of cord oedema.

In our case, the initial trauma which rendered the patient paraplegic was the inciting event for the delayed onset ascending myelopathy. Though surgical stress was present, the stabilisation of the spinal cord facilitated healing of the cord. Following this, the patient developed an infection which most likely acted as a trigger.

Diagnostic modalities and current literature

MRI is the primary investigation of choice in an event of ascending myelopathy. The Imaging of the cord postoperatively shows signal intensity changes- primarily increased T2 signal intensity, mild heterogenous T1 intensity change, mild expansion of the cord to occupy the canal.[13] Myelography may show the diffuse oedema of the cord.[19] Usually, the level of clinical myelopathy is unrelated to any mechanical instability or syrinx.[5,12] The cord myelomalacia ascends 4 or more levels above the level of the trauma.[14] There can be presence of thrombus or haemorrhage. The changes do not coincide with the clinical presentation. CSF examination may show increased pressure or signs of infection or inflammation, yet the culture will yield no organism.[2,3,20] Any nerve conduction tests done at the affected dermatome/ myotome would be normal, though there could be signs of motor demyelination as detected by motor evoked potential.[11] While Al Ghatany et al demonstrated the pathological changes associated with SPAM in their paper, no conclusive evidence has been found.[21] They showed presence of apoptotic changes in the affected area. Other pathological features may include formation of cysts and adherence of the spinal cord to the dural sac.[13] In our case, there was significant cord oedema that ascended to the level of C3 (Figure 3).

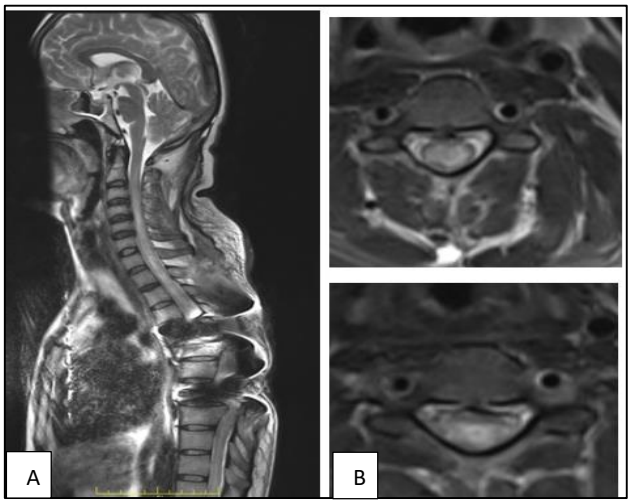

Figure 3 (A and B): Cord myelomalacic changes above the level of fixation seen at 4 weeks post operatively ascending till C3. Sagittal section and axial section of the post operative T2-weighted MRI taken after admission. Postoperative changes can be appreciated at the T3-T6 level of the thoracic cord.

Management of SPAM

Management of the condition has no proposed algorithm yet.[6] Supportive treatment with careful blood pressure monitoring, maintaining mean arterial pressure above 60 mmHg postoperatively to prevent cord ischemia, daily neurological level estimation, close monitoring of the patient, especially after a thoracic level spine fracture.[9,11,22,23] Response to systemic steroid therapy is varied. The administration of high-dose methylprednisolone treatment (30 mg/kg 15-minute bolus followed by 24-hour infusion at 5.4 mg/kg/hour) as per the national acute spinal cord injury study (NASCIS) protocol may have neuroprotective effects.[6] Operative management put forward for the management of SPAM includes detethering of the cord, durotomy, duroplasty, CSF shunts, cordectomy with variable results on a case to case basis.[2,13,24,25] The dilemma of medical management with steroids in a diabetic patient warrants the need for a guideline in such cases. Based on the existing management norms, we would like to put forward a simple algorithm in such cases. Once SPAM is diagnosed, we must rule out the presence of infection, with the help of infection markers in blood and a lumbar puncture. In presence of an infection,

it is optimal to initiate the treatment for infection as per the culture sensitivity report, followed by steroids (Figure 4).

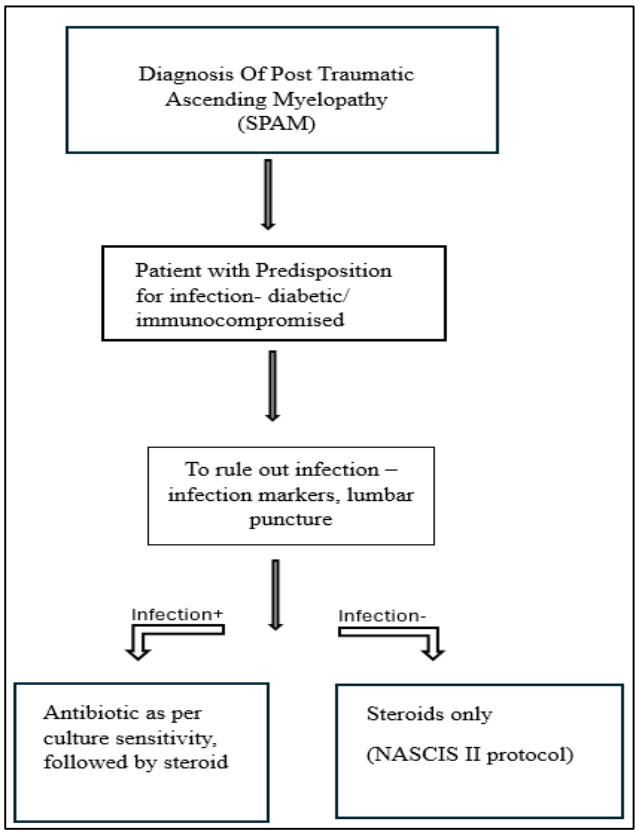

Figure 4: Medical management of a patient with post traumatic ascending myelopathy with special reference to superimposed infection.

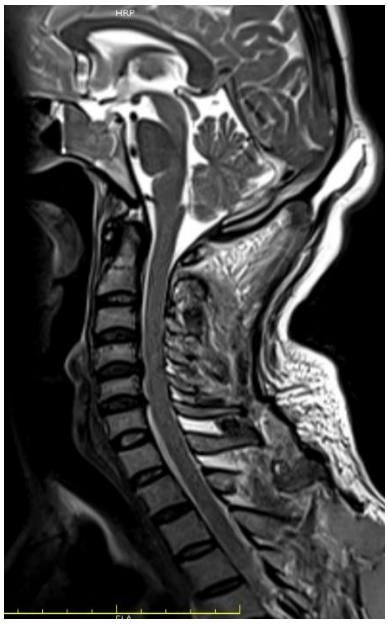

Figure 5: Follow up MRI of recovery of cord oedema-sagittal section of a T2 weighted MRI taken 3 months after diagnosis and initiation of treatment for SPAM in our patient.

Prognosis

Prognosis of SPAM is poor. With early detection and intervention, at least, a descend of myelopathy by one level is predicted. SPAM can be fatal.[6] Even though recovery can be seen on the follow up MRI, the translation into the clinical features, may not be seen.[6,14,26] In our case, the patient made good recovery with supportive management with current clinical condition along with resolution of cord oedema on the follow up MRI by three months (Figure 5) Our patient is currently wheelchair bound, has complete function of upper limbs.

CONCLUSION

SPAM of the spinal cord is a deterioration of the neurology 4 or more levels above the initial pathology. This can even be precipitated by an infection. Though rare, SPAM must be kept as a differential in the diagnosis of a postoperative event with similar clinical picture. A vigilant surgeon and good postoperative follow up would help early detection of the condition and prompt intervention. Earlier detection would be advantageous to the patient in making maximum possible recovery.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Harrop JS, Sharan AD, Vaccaro AR, Przybylski GJ. The Cause of Neurologic Deterioration After Acute Cervical Spinal Cord Injury. Spine. 2001;26(4):340.
2. Zhang J, Wang G. Subacute posttraumatic ascending myelopathy: a literature review. Spinal Cord. 2017;55(7):644-50.
3. Frankel HL. Ascending cord lesion in the early stages following spinal injury. Paraplegia. 1969;7(2):111-8.
4. Coleman-Bock J, Bäcker HC, Johnson MA, Turner P, Cunningham J. Subacute Posttraumatic Ascending Myelopathy: Case Report and Systematic Review of the Literature. Clin Spine Surg. 2023;36(4):157.
5. Eric C. Bélanger, Belanger E, Claude Picard, Picard C, Picard C, Daniel Lacerte, et al. Subacute posttraumatic ascending myelopathy after spinal cord injury. Report of three cases. J Neurosurg. 2000;93(2):294-9.
6. Basu S, Gohil K, Sarangi T. Subacute Posttraumatic Ascending Myelopathy: A Rare Complication After Thoracolumbar Spinal Cord Injury: A Case Report. JBJS Case Connect. 2022;12(1):10.
7. Bock J, Baecker H, Johnson M, Turner P, Cunningham JE. P158. SPAM: subacute posttraumatic ascending myelopathy is a rare but devastating disorder: what do we know? Spine J. 2022;22(9):S203.
8. Govekar S, Anand S, Lakshman LP, Vasanthapuram R, Banda RV. Syndrome Evaluation System for Simultaneous Detection Pathogens Causing Acute

Encephalitic Syndrome in India, Part-1: Development and Standardization of the Assay. Front Med. 2018;5:208.
9. Planner AC, Pretorius PM, Graham A, Meagher TM. Subacute progressive ascending myelopathy following spinal cord injury: MRI appearances and clinical presentation. Spinal Cord. 2008;46(2):140-4.
10. Schmidt BJ. Subacute delayed ascending myelopathy after low spine injury: case report and evidence of a vascular mechanism. Spinal Cord. 2006;44(5):322-5.
11. Aito S, El Masry WS, Gerner HJ, Lorenzo ND, Pellicanò G, D'Andrea M, et al. Ascending myelopathy in the early stage of spinal cord injury. Spinal Cord. 1999;37(9):617-23.
12. Jian Z, Zhang J, Huili W, Wang H, Haiying L, Liu H, et al. Subacute post-traumatic ascending myelopathy after T12 burst fracture in a 32-year-old male: case report and surgical result of cervical durotomy. Spinal Cord Ser Cases. 2016;2(1):16004.
13. Meagher T, Belci M, López de Heredia L. Resolution of SPAM following cordectomy: implications for understanding pathophysiology. Spinal Cord. 2012;50:638-40.
14. Kumar A, Kumar J, Garg M, Farooque K, Gamanagatti S, Sharma V. Posttraumatic subacute ascending myelopathy in a 24-year-old male patient. Emerg Radiol. 2010;17(3):249-52.
15. Katoh S, Ikata T, Tsubo M, Hamada Y, El Masry WS. Possible implication of leukocytes in secondary pathological changes after spinal cord injury. Injury. 1997;28(3):215-7.
16. Fischbein NJ, Dillon WP, Cobbs C, Weinstein PR. The "presyrinx" state: a reversible myelopathic condition that may precede syringomyelia. AJNR Am J Neuroradiol. 1999;20(1):7-20.
17. Aaron C. Tan, Tan AC, Karl Ng, Ng K, Allison Newey, Newey A, et al. Subacute delayed ascending myelopathy: not just a post-traumatic disorder. Spinal Cord. 2014;52(2):1.
18. Zhang J, Wang H, Liu H, Wang G. Subacute post-traumatic ascending myelopathy after T12 burst fracture in a 32-year-old male: case report and surgical result of cervical durotomy. Spinal Cord Ser Cases. 2016;2(1):1-4.
19. Yablon IG, Ordia J, Mortara R, Reed J, Spatz E. Acute Ascending Myelopathy of the Spine. Spine. 1989;14(10):1084.
20. Belanger E, Picard C, Lacerte D, Lavallee P, Levi AD. Subacute posttraumatic ascending myelopathy after spinal cord injury. Report of three cases. J Neurosurg. 2000;93(2):294-9.
21. Al-Ghatany M, Al-Shraim M, Levi ADO, Midha R. Pathological features including apoptosis in subacute posttraumatic ascending myelopathy. Case report and review of the literature. J Neurosurg Spine. 2005;2(5):619-23.
22. Okada S, Saito T, Kawano O, Hayashida M, Matsumoto Y, Harimaya K, et al. Sequential changes of ascending myelopathy after spinal cord injury on magnetic resonance imaging: a case report of neurologic deterioration from paraplegia to tetraplegia. Spine J. 2014;14(12):e9-14.
23. Esmaeilnia M, Asadi M, Sharara H. Posttraumatic ascending myelopathy after spinal cord injury in a young man: A case report. Clin Case Rep. 2022;10(8):e5997.
24. Bracken MB, Shepard MJ, Holford TR, Leo-Summers L, Aldrich EF, Fazl M, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. JAMA. 1997;277(20):1597-604.
25. Lee TT, Arias JM, Andrus HL, Quencer RM, Falcone SF, Green BA. Progressive posttraumatic myelomalacic myelopathy: treatment with untethering and expansive duraplasty. J Neurosurg. 1997;86(4):624-8.
26. Kovanda TJ, Horn EM. Subacute posttraumatic ascending myelopathy in a 15-year-old boy. J Neurosurg Spine. 2014;21(3):454-7.

Cite this article as: Shenoy S, Prasath CSV, Manish R, Bala S. Sub-acute post traumatic ascending myelopathy with superimposed infection in a diabetic: a case report. Int J Res Orthop 2025;11:242-6.