

Subacute post-traumatic ascending myelopathy after cervical spinal cord injury: a rare and fatal complication

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ABSTRACT

Subacute post-traumatic ascending myelopathy (SPAM) is a rare but devastating complication of spinal cord injury (SCI). It is characterized by progressive neurological deterioration extending several segments above the primary lesion within days to weeks after trauma. The underlying pathophysiology remains uncertain, and treatment strategies are not standardized. A 38-year-old man sustained traumatic C6–7 spondylolisthesis with bilateral facet dislocation following a motorcycle accident. Initial magnetic resonance imaging (MRI) demonstrated cord contusion and edema extending from C5 to C7. After traction and reduction, the patient underwent anterior C6 corpectomy with placement of an expandable cage and C5–7 plating, followed by C5–6 total laminectomy and C4–7 posterior instrumentation. Postoperatively, partial neurological recovery was observed. However, on postoperative day 10, the patient developed quadriparesis rapidly progressing to quadriplegia, accompanied by spinal shock and respiratory failure requiring mechanical ventilation. Imaging studies excluded hematoma and implant failure, although postoperative MRI was limited by metallic artifacts. Differential diagnoses, including pulmonary embolism, cardiac dysfunction, and sepsis, were ruled out. Based on the clinical progression and exclusion of alternative causes, a diagnosis of ascending myelopathy was established. Despite intensive supportive care, the patient died on the fourth day of mechanical ventilation. SPAM remains an unpredictable and fatal complication of SCI. Limitations in postoperative imaging, particularly metal-related artifacts, may hinder diagnosis, underscoring the importance of correlating clinical and radiological findings. Vigilant monitoring and continued reporting of cases are essential to improve recognition, refine diagnostic strategies, and guide management of this rare entity.

Keywords: Ascending myelopathy; cervical trauma; complication; spinal cord injury; subacute post-traumatic ascending myelopathy (SPAM).

INTRODUCTION

Spinal cord injury (SCI) is a devastating condition associated with severe neurological impairment and high morbidity.^[1] Although neurological deterioration immediately following trauma is not uncommon, progression of deficits to levels above the initial site of injury represents an unusual and alarming clinical phenomenon.^[2]

Subacute post-traumatic ascending myelopathy (SPAM) is one such complication, characterized by unexpected cranial extension of neurological deficits within days to weeks after the

initial insult.^[3] The condition is poorly understood, with proposed mechanisms including vascular ischemia, cerebrospinal fluid (CSF) flow disturbances, inflammatory processes, and secondary injury cascades.^[4–6]

No standardized treatment has been established, and management remains largely supportive.^[7] We present the case of a patient with traumatic C6–7 spondylolisthesis and bilateral facet dislocation who, despite undergoing anterior and posterior decompression and stabilization, developed fatal ascending myelopathy in the postoperative period.

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CASE REPORT

A 38-year-old man with no significant past medical history was admitted to our emergency department following a motorcycle accident. On arrival, neurological examination revealed bilateral upper extremity weakness (grade 3/5), complete paraplegia of the lower extremities, loss of anal tone, and anesthesia below the C6 level. He was also in spinal shock, presenting with bradycardia and hypotension.

This study was conducted in accordance with the ethical standards of the institutional and national research committees and with the Declaration of Helsinki. Written informed consent was obtained from the patient for publication of this case and any accompanying images.

Initial radiological evaluation demonstrated C6–7 traumatic spondylolisthesis with bilateral facet dislocation and facet interlocking (Figure 1), along with spinal cord contusion and edema extending from C5 to C7 (Figure 2). The patient was placed in cervical traction, achieving reduction. He subsequently underwent an anterior C6 corpectomy with placement of an expandable cage and C5–7 anterior plating, followed by a C5–6 total laminectomy and C4–7 posterior instrumentation and fusion.

High-dose corticosteroid therapy was initiated according to the NASCIS II (National Acute Spinal Cord Injury Study II) protocol, consisting of an intravenous loading dose of 30 mg/kg methylprednisolone administered over one hour, followed by a continuous infusion of 5.4 mg/kg/hour for the subsequent 23 hours. This regimen was continued in the postoperative period as part of anti-edema and neuroprotective management.

In the immediate postoperative period, neurological status improved: upper extremity strength increased to 4/5, and minimal movement (1/5) was noted in the lower extremities. Despite this partial recovery and ongoing high-dose corticosteroid therapy, on postoperative day 10 the patient developed quadriparesis, which rapidly progressed to quadriplegia.

At that time, the level of anesthesia, initially below C6, had ascended into the upper cervical dermatomes to the C3 level, consistent with ascending myelopathy. Features of spinal shock reappeared, accompanied by respiratory depression consistent with diaphragmatic palsy, necessitating intubation and mechanical ventilation.

Repeat cranial and cervical imaging excluded intracranial pathology, implant malposition, or postoperative hematoma (Figure 3). Although magnetic resonance imaging (MRI) was limited by metallic artifacts, no new compressive lesion was identified (Figure 4). Extensive investigations were subsequently performed to exclude other potential causes of deterioration. Thoracic computed tomography (CT) angiography ruled out pulmonary embolism, transthoracic echocardiography demonstrated normal cardiac function, and laboratory analyses, including complete blood count, electrolytes, renal and hepatic profiles, and infection markers, were within normal limits. As no alternative pathology was identified, and given the characteristic ascending neurological progression, a diagnosis of subacute post-traumatic ascending myelopathy was made. Despite intensive supportive treatment, the patient succumbed on the fourth day of mechanical ventilation.

DISCUSSION

Subacute post-traumatic ascending myelopathy is an uncommon but devastating complication of spinal cord injury. It is defined as progressive neurological deterioration extending four or more segments above the initial lesion, usually within days to weeks after trauma.^[8] Although early neurological worsening by one or two levels is frequently observed in SCI,^[2] the distinctive cranial extension seen in SPAM is rare and often associated with respiratory compromise and high mortality.^[9]

Since Frankel's initial description in 1969,^[10] only a limited number of cases and small series have been reported. Yablon et al.^[11] demonstrated that ascending neurological deficits were more frequent among conservatively managed patients

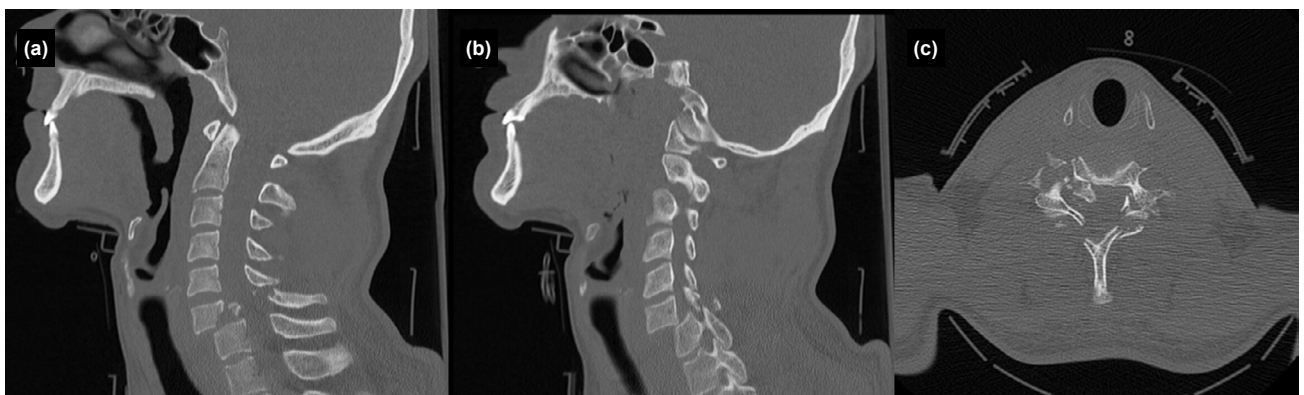


Figure 1. Preoperative cervical computed tomography (CT) images. (a) Sagittal reconstruction showing traumatic C6–7 dislocation. (b) Sagittal view demonstrating bilateral facet interlocking at C6–7. (c) Axial image confirming bilateral facet dislocation with locked facets.



Figure 2. Preoperative sagittal T2-weighted magnetic resonance imaging (MRI) of the cervical spine demonstrating C6–7 dislocation with associated spinal cord contusion and edema extending from C5 to C7.

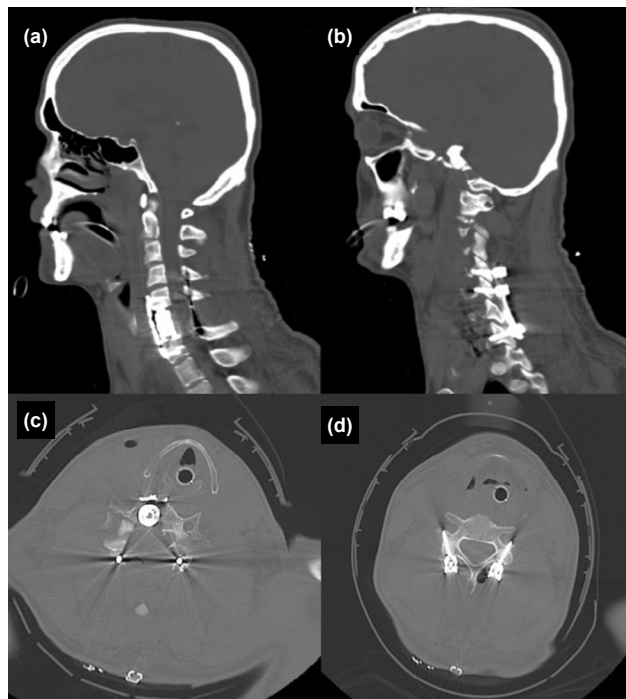


Figure 3. Postoperative cervical computed tomography (CT) images. (a) Sagittal reconstruction showing anterior C6 corpectomy with expandable cage placement and C5–7 anterior plating. (b) Sagittal view demonstrating posterior instrumentation from C4 to C7 with laminectomy at C5–6. (c) Axial CT image at the C6 level showing the anterior cage and plate construct. (d) Axial CT image at the C5 level showing posterior lateral mass screw fixation.

than among those who underwent surgical stabilization, suggesting that stabilization may play a protective role. Belanger et al.^[3] subsequently defined SPAM as a distinct clinical entity, while Planner et al.^[5] described characteristic MRI features, including cranially ascending T2 hyperintensity and cord swelling. More recent reviews, such as Zhang and Wang, confirm its estimated incidence of 0.4–1% and emphasize that no unifying mechanism has been established (Table 1).^[7]

The proposed pathophysiology remains multifactorial, encompassing vascular ischemia or venous hypertension, cerebrospinal fluid flow obstruction, autoimmune inflammation, and reperfusion injury.^[4,7] Pathological studies have identified cord edema, infarction, and apoptotic changes above the index injury, supporting both vascular and secondary injury hypotheses.^[6] Clinically, SPAM often presents with stepwise or rapid deterioration, sometimes preceded by low-grade fever, pain, or autonomic instability.^[5,12]

Therapeutic strategies are not standardized. High-dose corticosteroids, anticoagulation, and surgical approaches such as duraplasty or cord untethering have been attempted, with inconsistent results.^[3,7,13] Supportive management, including hemodynamic stabilization, ventilatory support, and rehabilitation, remains the cornerstone of care.^[7,9] Despite aggressive combined anterior and posterior decompression and stabilization in our case, the patient deteriorated neurologically on postoperative day 10, culminating in quadriplegia and respiratory failure.

A particular limitation of our case was the inability to radiologically demonstrate ascending myelopathy due to signifi-

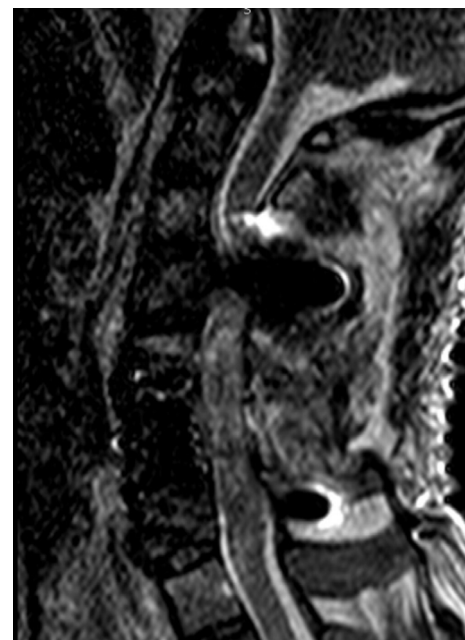


Figure 4. Postoperative sagittal T2-weighted magnetic resonance imaging (MRI) of the cervical spine. Metallic artifact from anterior and posterior instrumentation limits evaluation; however, no definite new compressive lesion is observed.

Table 1. Key reports of subacute post-traumatic ascending myelopathy (SPAM) after spinal cord injury

Author/Year	Study Design	N (Patients)	Pathology/Proposed Mechanism	Location of Pathology	Levels Ascended	Outcome	Follow-up Duration
Frankel, 1969 ^[10]	Case report	1	Early ascending cord lesion	Thoracic to cervical	12 levels 1-4+	Fatal Variable; some fatal	17 days Inpatient/early
Yablon et al., 1989 ^[11]	Cohort study	134 (14 with SPAM)	Acute ascending myelopathy; higher risk with non-operative management	Various (cervical/thoracic)			
Aito et al., 1999 ^[9]	Case series	Several	Ascending myelopathy in early SCI	Cervical and thoracic	3-6	High morbidity	Short-term
Belanger et al., 2000 ^[3]	Case series	3	SPAM defined as a distinct clinical entity	Thoracic and cervical	4-8	Two improved, one deteriorated	Months
Schmidt, 2006 ^[4]	Case report	1	Vascular mechanism (venous hypertension, ischemia)	Lumbar → thoracic	5	Partial recovery	3 months
Planner et al., 2008 ^[5]	Retrospective review	Multiple	MRI: cranially ascending T2 hyperintensity, cord swelling	Cervical/thoracic	≥4	Most stabilized; some residual deficits	Variable
Kumar et al., 2010 ^[12]	Case report	1	Post-traumatic SPAM	Thoracic (T12) → higher levels	6	Survived with deficits	6 months
Al-Ghatany et al., 2005 ^[6]	Case report with pathology	1	Infarction, edema, apoptosis the above lesion	Thoracic cord	Fatal 5-6	Autopsy	
Zhang & Wang, 2017 ^[7]	Literature review	60+ (reviewed)	Multifactorial mechanisms	Poor prognosis;			
Biswas et al., 2022 ^[8]	Case report	1	Ascending paralysis after SCI	Thoracolumbar to upper thoracic	8	Survived with deficits	6 months

The table summarizes study design, number of patients, proposed pathology or mechanism, anatomical location of the pathology, number of spinal levels ascended, clinical outcomes, and follow-up duration. SPAM: Subacute post-traumatic ascending myelopathy; SCI: Spinal cord injury; CSF: Cerebrospinal fluid; MRI: Magnetic resonance imaging.

cant metallic artifacts from instrumentation on postoperative MRI. Although no new compressive lesion or hematoma was identified, the artifact precluded optimal evaluation of intramedullary changes. Thus, the diagnosis of SPAM relied on the characteristic clinical progression and the exclusion of alternative etiologies such as pulmonary embolism, cardiac dysfunction, or sepsis. This limitation underscores the importance of correlating imaging with clinical findings and highlights the potential value of artifact-reducing MRI sequences or CT myelography in similar scenarios.

Overall, our case highlights the unpredictable course of SPAM, its high mortality despite timely surgical stabilization, and the diagnostic challenges posed by postoperative imaging limitations.

CONCLUSION

Subacute post-traumatic ascending myelopathy remains a rare but devastating complication of spinal cord injury, characterized by rapid neurological deterioration and high mortality. Its pathophysiology is still unclear; imaging may be limited in the postoperative setting, and no standardized treatment exists. Supportive care, therefore, remains the cornerstone of management, while vigilant neurological monitoring and continued reporting of cases are essential to improve early recognition, refine diagnostic strategies, and guide future therapeutic approaches.

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OLGU SUNUMU - ÖZ

Servikal omurga travması sonrası gelişen subakut posttravmatik asendan myelopati: Nadir ve ölümcül bir komplikasyon

Subakut posttravmatik asendan myelopati (SPAM), omurilik yaralanmalarının nadir fakat yıkıcı komplikasyonlarından biridir. Travmadan sonraki günler veya haftalar içinde, başlangıç lezyonunun birkaç segment üzerinde nörolojik kötüleşme ile kendini gösterir. Patofizyolojisi tam olarak bilinmemekte olup standart bir tedavi yaklaşımı yoktur. Otuz sekiz yaşındaki erkek hasta motosiklet kazası sonrası acil servise getirildi. Başlangıç radyolojik incelemelerde C6–7 düzeyinde travmatik spondilolistezis, bilateral faset dislokasyonu ve C5–7 düzeyinde medulla spinalis kontüzyonu ile ödem saptandı. Redüksiyon sonrası anterior C6 korpektomi, ekspandibl kafes ve C5–7 plak yerleştirilmesi yapıldı, ardından C5–6 total laminektomi ve C4–7 posterior enstrümantasyon uygulandı. Postoperatif dönemde kısmi nörolojik düzelme izlendi. Ancak 10. günde kuadriparezi hızla kuadriplejiye ilerledi; spinal şok bulguları ve solunum depresyonu gelişti. Kontrol görüntülemelerinde hematoma veya implant malpozisyonu saptanmadı, ancak manyetik artefakt nedeniyle medulla intrensek patolojisi net değerlendirilemedi. Pulmoner emboli, kardiyak disfonksiyon ve sepsis gibi ayırıcı tanıları ekarte edildi. Klinik seyir asendan myelopati ile uyumlu değerlendirildi ve yoğun destek tedavisine rağmen hasta kaybedildi. SPAM, servikal omurga travmalarının nadir fakat ölümcül bir komplikasyonudur. Postoperatif dönemde metalik artefakt nedeniyle radyolojik tanı sınırlı olabilir. Bu nedenle klinik-radyolojik korelasyon, dikkatli nörolojik takip ve yeni vaka bildirimleri bu nadir sendromun daha iyi anlaşılması ve yönetimi açısından kritik öneme sahiptir.

Anahtar sözcükler: Asendan miyelopati; servikal travma; komplikasyon; omurilik yaralanması; SPAM.

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