**Subacute posttraumatic ascending myelopathy in a 15-year-old boy**

**Case report**

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Secondary injury following initial spinal cord trauma is uncommon and frequently attributed to mismanagement of an unprotected cord in the acute time period after injury. Subacute posttraumatic ascending myelopathy (SPAM) is a rare occurrence in the days to weeks following an initial spinal cord injury that is unrelated to manipulation of an unprotected cord and involves 4 or more vertebral levels above the original injury. The authors present a case of SPAM occurring in a 15-year-old boy who sustained a T3–4 fracture-dislocation resulting in a complete spinal cord injury, and they highlight the imaging findings and optimum treatment for this rare event.

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**Key Words**
- subacute posttraumatic ascending myelopathy
- trauma
- secondary spinal cord injury
- traumatic spinal cord injury

**Subacute posttraumatic ascending myelopathy (SPAM)** is a rare disorder occurring days to weeks after traumatic spinal cord injury. It is characterized by ascending myelopathy at least 4 segments above the original injury. SPAM is distinct from acute secondary spinal injuries that are often caused by manipulation of an unprotected spinal cord and chronic secondary injuries that are often caused by syrinx formation or myelomalacia. We present a case of SPAM in a 15-year-old boy who underwent a posterior spinal fusion after sustaining a fracture-dislocation at the T3–4 level resulting in a complete spinal cord injury, and we describe the imaging findings and treatment strategy of this rare phenomenon.

**Case Report**

**History and Examination.** This previously healthy 15-year-old boy presented after a dirt bike accident while traveling approximately 50 mph. He was wearing a helmet and a neck brace at the time of the accident, but he landed upside down in a pile of concrete rubble. He presented to the emergency department with loss of sensation at the T-4 level and paraplegia. Strength and sensation in the upper extremities were completely intact aside from hand intrinsic strength of Grade 4+/5 on the right. On presentation, CT scanning and thoracic MRI demonstrated a T3–4 fracture-dislocation with 50% subluxation of T-3 onto T-4 and an epidural hematoma spanning caudally from the level of the injury (Figs. 1 and 2). MRI on presentation was motion limited but demonstrated diffuse T2 signal hyperintensity within the spinal cord spanning from the T-2 level to the T-5 level. No definitive evidence of hemorrhage within the spinal cord itself was noted on gradient echo imaging. Cervical CT scanning was negative. His only other injuries were bilateral pulmonary contusions with a small right pneumothorax and a minimally displaced broken 4th rib.

**Operation.** The morning after presentation, our patient was brought to the operating room for spinal cord decompression, fracture reduction, and pedicle screw fixation. Intraoperatively, soft-tissue injury was noted throughout the exposure. Laminectomies at T3–5 were performed to decompress the injured spinal cord and allow for adequate fracture reduction. The T-4 pedicles were removed in a piecemeal fashion as they had sustained significant damage. Pedicle removal provided access to the T-4 vertebral body, which was partially removed prior to fracture reduction. A small dural laceration was noted during the decompression, but no CSF was visualized. After adequate decompression was achieved, towel clamps were used to reduce the fracture. The fracture site was then fixed using T1–7 pedicle screws and cobalt chrome rods.

**Postoperative Course.** Postoperatively, the patient remained hemodynamically stable. On postinjury Day 8, he was discharged from the hospital with mild bilateral intrinsic hand weakness (Grade 4+/5). At this time he was playing video games and texting on a cellular phone without difficulty.

On postinjury Day 38, the patient presented with Grade 3/5 bilateral intrinsic hand weakness and Grade 4+/5 handgrip and wrist extension. Sensation remained intact above the original injury level. MRI showed a T2 hyperintensity extending from the T-11 to the C-3 level.
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rostrally with focal cord enlargement (Fig. 3). It was unclear whether these findings represented a posttraumatic syrinx. The patient was placed on high-dose dexamethasone (4 mg every 6 hours) with a long taper of 3 weeks and continued physical and occupational therapy. His strength continued to slowly deteriorate over the next month and then stabilized, at which time physical examination demonstrated Grade 4/5 deltoid, biceps, and triceps strength bilaterally. At 4 months after injury, his strength began to improve and MRI revealed dramatically decreased T2 signal hyperintensity in the cervical spinal cord (Fig. 4). One year after injury, his upper-extremity strength had returned to normal bilaterally.

Discussion

Deterioration occurring after initial traumatic spinal cord injury represents an uncommon disorder that can usually be traced to a specific incident during management involving manipulation of the unprotected cord. In a series of 283 patients presenting to 5 different trauma centers with a spinal cord injury, Marshall et al. identified 14 patients (4.9%) who deteriorated further during the acute period of hospitalization. Factors associated with deterioration during hospitalization included operative intervention, skeletal traction application, halo vest application, rotobed rotation, and Stryker frame rotation. In this series and other similar reports, the initial injuries tended to be at the level of the cervical spine.

Harrop et al. described a temporal association between secondary ascending injuries and their causes of neurological deterioration. Patients with secondary injuries occurring within 24 hours of presentation (early deterioration) resulted from traction and immobilization.

Fig. 1. Left: Sagittal CT scan on presentation demonstrating T3–4 fracture-dislocation with 50% subluxation of T-3 onto T-4 (arrow). Right: Sagittal T2-weighted MR image at presentation again demonstrating subluxation of T-3 onto T-4 (arrow) as well as an associated epidural hematoma (asterisk). Increased intramedullary T2 signal is only present up to the T-1 level.

Fig. 2. A–F: Cranial to caudal progressive axial T2-weighted MR images at presentation through fracture-dislocation site, again demonstrating radiographic cord compression (arrow) as well as an epidural hematoma (asterisk).
Secondary injuries occurring within 1–7 days of presentation correlated with fracture-dislocation patients who had undergone periods of sustained hypotension after injury. Finally, in 1 patient who had deterioration 7 days after initial presentation, evidence was demonstrated of vertebral artery injury leading to cervicomedullary ischemia and eventually death. The injury ascended a maximum of 3 levels in all but 2 of the patients described. Both of these patients had sustained vertebral artery dissections. Similar to the study completed by Harrop et al., Yablon et al. described a series of 14 patients who experienced ascending myelopathy to a maximum of 4 levels above the initial injury site, occurring within 4 weeks of injury.

Late-onset deterioration after spinal cord injury can also occur. Posttraumatic syrinx formation is a well-documented phenomenon, and posttraumatic myelomalacia can also result in late neurological deterioration. These secondary pathological processes generally occur months to years after the initial insult.

Although early- or late-onset deterioration after spinal cord injury is not rare, deterioration days to weeks after an initial injury occurs only in a very small number of patients. This complication, known as subacute posttraumatic ascending myelopathy (SPAM), is defined as neurological deterioration ascending 4 or more vertebral levels above the injury site and occurring within the first few weeks of initial injury that is unrelated to manipulation of an unprotected spinal cord or syrinx formation. Most patients with SPAM present within 3 weeks of injury, unlike our patient who presented 5 weeks postinjury. In cases of SPAM, T2-weighted MRI reveals intramedullary hyperintensity above the original level of injury with cord expansion, similar to what was seen in our patient. Recovery from SPAM varies widely, ranging from death when the brainstem is involved to complete recovery.

Other authors have proposed various mechanisms of injury in SPAM, but the etiology remains a matter of debate. Planner et al. have suggested that alterations in CSF flow could be responsible for SPAM. This theory was supported by the fact that 5 patients in their series developed intramedullary hematomas, which could alter CSF flow. Still, the authors argued that decompression procedures in SPAM patients should normalize this altered CSF flow, making this cause less likely. Our patient developed an epidural hematoma with his injury; this lesion could theoretically change the pressure of the adjacent subarachnoid space and thus alter CSF flow (Figs. 1 and 2). Interestingly, even after a decompressive surgery and resolution of this epidural hematoma, there was possible evidence of altered CSF flow in our patient.

Although it cannot be definitively demonstrated in our case, this remodeling possibly occurred because of alterations in CSF flow secondary to the injury.
and could suggest there was a change in CSF dynamics during this remodeling period. The theory of CSF flow alterations as a cause of SPAM correlates well with the time period of clinical symptoms and imaging findings of bone remodeling in our patient. Still, considering that this remodeling has not been described in any other patients with SPAM, it is unlikely that it explains the whole story.

After an autopsy of a patient with SPAM and initial injury at T-4 revealed apoptotic cells at sites distant from the original injury, Al-Ghatany et al.2 acknowledged that aberrant apoptosis may be involved. This theory is supported by a number of studies demonstrating the role of apoptosis in spinal cord injury and by an animal study by Crowe et al.3 demonstrating the role of apoptosis in the demyelination of fiber tracts at sites distant from the initial injury.2,12 Al-Ghatany et al.2 acknowledged that apoptosis is unlikely to be the lone cause of SPAM. For instance, patients with SPAM frequently improve both clinically and radiographically after subacute deterioration, suggesting apoptosis is not the only process involved.

Belanger et al.3 have proposed a number of different mechanisms of injury in SPAM. Anterior spinal artery thrombosis can cause secondary injury after spinal cord injury.3 This injury tends to present acutely instead of progressively, and unlike SPAM, recovery is not frequently observed. A nucleus pulposus embolism can cause a similar secondary injury following an initial spinal cord insult. These events tend to occur with rapid onset of symptoms in a manner similar to anterior spinal artery thrombosis, which is not consistent with SPAM.3 Venous thrombosis and congestive ischemia may cause a posttraumatic ascending myelopathy.3,10 This theory is supported by a case presented by Schmidt16 in which MRI demonstrated vascular markings consistent with venous congestion. Additionally, the patient appeared to deteriorate after being placed in a sitting position and while wearing a thoracolumbosacral orthotic brace, both of which could increase venous congestion.16 Still, these associations are not found in all cases of SPAM, suggesting that another mechanism may be involved as well.

**Conclusions**

Subacute posttraumatic ascending myelopathy is a rare condition involving 4 or more vertebral levels. Our patient was successfully treated with steroids and physical therapy, and this highlights the importance of recognizing this entity when it occurs so that proper treatment can be implemented. The pathophysiology of SPAM remains unknown, although a number of hypotheses have been suggested. More work is needed before a mechanism of injury in SPAM can be elucidated.

**Disclosure**

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