**Chronic relapsing ascending myelopathy: a treatable progressive neurological syndrome following traumatic spinal cord injury**

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**Abstract**

**Background**

We describe a novel progressive neurological syndrome complicating traumatic spinal cord injury (TSCI). Based on clinical and radiological features, we propose the term ‘Chronic Relapsing Ascending Myelopathy’ (CRAM). We distinguish between the previously described sub-acute progressive ascending myelopathy (SPAM) and post-traumatic syringomyelia (PTS), which may lie on a spectrum with CRAM.

**Case report**

A 60-year-old man sustained a T4 ASIA-A complete TSCI. Four months post-injury, he developed a rapidly progressive ascending sensory level to C4. Clinical and radiological evaluation revealed ascending myelopathy with progressive T2 hyper-intense cord signal change. He underwent cord detethering and expansion duroplasty. Following an initial dramatic resolution of symptoms, the patient sustained two relapses, each 1-month post-discharge characterised by recurrence of disabling ascending sensory changes, each correlating with the radiological recurrence of cord signal change. Symptoms and radiological signal change permanently resolved with more extensive detethering and expansion duroplasty. There is radiological and clinical resolution at 1-year follow-up.

**Conclusion**

Acute neurological deterioration post-TSCI may be due to SPAM or may occur after years due to PTS. We propose CRAM as a previously unrecognised phenomenon. The radiological characteristics overlap with SPAM. However, CRAM presents later and, clinically, behaves like PTS, but without cord cystic change. Cord detethering with expansion duroplasty are an effective treatment.

**Introduction**

Delayed neurological deterioration following traumatic spinal cord injury (TSCI) is well-described. Once biomechanical instability of the spinal column has been ruled out as the cause, there are two main possibilities: The first, ‘sub-acute progressive (or “post-traumatic”) ascending myelopathy’ (SPAM), originally reported by Frankel,[1](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) is a rarely encountered phenomenon (incidence 1.5% in his series). It is an acute ascending cord injury, presenting from day 1 up to 4 weeks post-TSCI.[2](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) MRI confirms cord expansion and T2 hyper-intensity involving at least four segments above the injury site.[3](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) Pyrexia is often found at the onset of neurological deterioration.[1–3](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) The aetiology is unclear and conservative therapy is generally advocated.[1](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true),[4](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) Only one of Frankel’s patients was operated on; however, SPAM progressed despite two decompressive laminectomies (‘swollen and pink cord intra-operatively’). Mortality was 10% when the cord injury extends into the brainstem causing respiratory failure.[3](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) The second, typically chronic complication post-TSCI, is post-traumatic syringomyelia (PTS). Originally, Bastian in 1867 and then Hallopeau in 1871 documented the association between TSCI and syringomyelia.[5](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true),[6](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) Its reported incidence is 0.02 − 4.50%, twice as common in complete than incomplete injuries.[7–10](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) PTS is often under-recognised due its insidious and initially sensory nature, developing on average 9 years post-injury[7](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true),[9](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) (range 6 − 34 years[9](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true),[11](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true)). PTS primarily affects males with average age of 44 years in one UK series.[9](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) PTS presents with progressive segmental neuropathic pain and sensory loss due to a lesion of the spinothalamic tract. A classic dissociated sensory loss is more common than complete sensory loss, with MRI confirming the diagnosis.[11](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) PTS is slowly progressive and up to 17 − 50% patients remain static without treatment over 10 years.[11](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) There is no consensus on the pathophysiology or optimal management; arachnoiditis and scarring causing disturbances in cerebrospinal fluid (CSF) flow may play a role.[11–13](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) Treatment options are shunting the syrinx to the peritoneal/pleural cavity or sub-arachnoid space, detethering ± expansion duroplasty and, ultimately, cordectomy.[7](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) Here, we present an original case where, clinically, the temporal progression and relapsing nature are inconsistent with SPAM and the radiological features are inconsistent with PTS.

**Case description**

A 60-year-old man sustained a TSCI during a cycling expedition, falling 20 metres down a gorge. He was airlifted to the regional spinal unit and found to have a T4–T5 dislocation causing a T4 American Spinal Injury Association (ASIA) grade A TSCI. He had a T3–T7 pedicle screw fixation and was admitted to his local spinal rehabilitation unit. Four months later, he developed left-sided upper limb neuralgia and altered sensation in the T1 and T2 dermatomes. Right-sided sensory symptoms followed with acute ascending sensory dysfunction correlating with ascending T2 hyper-intense cord signal change on MRI. Following initial deterioration of symptoms and new MRI findings, a regional neurosurgical opinion was sought and the plan was to treat conservatively with close supervision. The sensory dysfunction progressed cranially by a dermatome every 10–20 days, accelerating to every 4 days, up to C5 bilaterally. He described the sensation as a ‘moving bush-fire, leaving an area lacking in sensory function’. As the neurological symptoms and MRI findings were progressive, a diagnosis of SPAM was considered and an opinion from the authors who described the clinical features of SPAM was taken.[3](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) Following their advice, a referral was made to the senior author of this report. Against the diagnosis of SPAM was the deterioration occurring late, at 4 months post-TSCI. MRI did not reveal a syrinx and cord tethering was noted at the injury site. On admission to the neurosurgical unit, he had marked neuropathic pain in the upper limbs, worse on the left and progressive lower limb spasms with ASIA A TSCI, bilateral sensory level C5 and motor level T1. Upper limb motor function was intact. Serial MRI mirrored the history with ascending increased T2 cord signal change from C7 to C4 without cyst, between 6 weeks to 1 day pre-operatively ([Figure 1(A–C)](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true#F0001)). MRI showed prominent CSF pulsation artefact above the injury but not below ([Figure 1(D,E)](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true#F0001)). The patient was admitted for cord detethering with arachnolysis and expansion duroplasty to restore CSF dynamics and arrest progressive neurological injury.

Figure 1. MRI spine. T2 mid-sagittal view at (A). Six weeks before first surgery (duroplasty + arachnolysis) (B). Four weeks before first surgery (C). One day before first surgery (D). CSF above (E). CSF below injury site. Arrows: white (CSF pulsation artefact), black (no CSF pulsation artefact), grey (injury site). D-Inset: CSF pressure simultaneously monitored during first surgery above and below the injury site; patient supine before dural opening. T2 mid-sagittal view (F). Two weeks after first surgery (G). One week before second surgery (more extensive duroplasty and arachnolysis) (H). Two months after second surgery.



**First surgery: detethering of spinal cord and expansion duroplasty**

Intra-operatively, there was pulsatile CSF above the injury compared with non-pulsatile CSF below at higher pressure ([Figure 1(D)](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true#F0001) inset). The dura was then opened with microsurgical arachnolysis and dissection done under the operating microscope. An expansion duroplasty was fashioned by suturing an artificial dural patch to the dural edges. The size of the duroplasty was limited below by the cross-connecting metal rod. Cord samples and CSF were obtained to refute the possibility of demyelination or vasculitis.

**Post-operative course**

Immediately, the patient reported restored sensation and total resolution of left arm neuropathic pain. In 2 weeks, his right sensory level descended to the injury site. This corresponded with resolution of the cord signal change on MRI ([Figure 1(F)](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true#F0001)). Intra-operative CSF and serum samples showed no oligoclonal bands, no paraprotein and no demyelination (Serum IgG 5.9 g/L, IgA 0.8 g/L, IgM 0.2 g/L; CSF albumin 0.26 mg/L and CSF IgG 0.03 mg/L). The cord was gliotic and oedematous with prominent small and medium vascular channels with no chronic inflammatory infiltrates. Labelling for CD3 showed a moderate population of small T-cells. Immunohistochemistry for CD68 showed sheets of microglial cells and activated macrophages. Immunostaining for GFAP confirmed brisk reactive gliosis in keeping with the histological findings of PTS.[14](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) Post-operative ASIA testing at 2 weeks demonstrated sensory improvement, T5 sensory level on the right and C6 on the left. He was discharged at 2 weeks following inpatient rehabilitation.

**First relapse and shunt treatment**

One month following discharge, the ascending sensory symptoms recurred with MRI demonstrating ascending T2 hyper-intense cord signal change similar to his original MRI. The patency of the duroplasty could not be ascertained due to metal artefact. We performed a shunt procedure, bridging the injury site (aiming to equalise the CSF pressures above and below) with a lumbar-peritoneal shunt with initial clinical improvement, particularly in the left arm sensory symptoms.

**Second relapse and re-do detethering of spinal cord with expansion duroplasty**

A further symptomatic recurrence at 1 month, prompted further MRI that showed new, marked T2 signal change in the cervical spinal cord up to C1 ([Figure 1(G)](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true#F0001)). Further surgery was undertaken. We removed the metalwork to allow space for a more extensive duroplasty. After durotomy, we found that the injured cord had re-tethered, thus obstructing CSF flow. Extensive arachnolysis and a larger duroplasty were performed. His symptoms improved immediately with dramatic MRI change to normal cervical cord that has persisted to date ([Figure 1(H)](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true#F0001)). On each admission, we noted episodes of fever, with no infective focus identified. At 1-year post-expansion duroplasty, the patient remains asymptomatic, and has returned to baseline function. His quality of life is much improved with marked resolution of previous severe pain and his mobility enhanced with a power add-on to his wheelchair. Serial MRI spine scans reveal persistent resolution of cord signal change, and he has been discharged.

**Discussion**

We described a previously unreported phenotype of delayed neurological deterioration in TSCI, for which we propose the term chronic relapsing ascending myelopathy (CRAM). CRAM is treatable by cord detethering and expansion duroplasty that results in resolution of symptoms.

What causes CRAM? The higher CSF pressure and lack of CSF pulsation artefact below the injury and the adhesions obstructing CSF flow around the injury site, evident intra-operatively, favour a mechanical aetiology. Demyelination and vasculitis are unlikely given the lack of CSF oligoclonal bands and the cord histological findings primarily showing gliosis. Adhesions at the injury site may result from blood products accumulating in the dependent position[15](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) causing progressive cord tethering to the dura. We propose that SPAM, CRAM and PTS lie on the same spectrum; which one occurs may depend on how long it takes for the adhesions to obliterate the CSF space around the injured cord. It is unclear whether SPAM and CRAM are pre-syrinx states. Fast onset with generally severe, even fatal, symptoms may lead to SPAM, sub-acute with generally less severe symptoms to CRAM and delayed onset often sub-clinical to PTS.

We think it is appropriate to label this CRAM because the name accurately describes the condition. It is Chronic, because it does not satisfy the criteria for sub-acute as described for SPAM (1–4 weeks). It is Relapsing, the cause of which may be either inadequate initial surgery or progressive scarring or another reason. It is Ascending because the MRI signal change spreads cranially from the injury site. And it is obviously a Myelopathy. Once the adhesions have occluded the CSF space around the cord, two non-communicating CSF compartments form (above + below injury site) with CSF pulsation above but not below, evident on MRI. The differential CSF pressures may drive CSF entry into the cord causing SPAM, CRAM or PTS. Flexion-extension of the neck[8](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) and cardiac pulsations[15](https://www.tandfonline.com/doi/full/10.1080/02688697.2022.2102146?needAccess=true) normally cause the cord to move cranio-caudally. After cord tethering, such movements may become restricted causing stress forces in the cord that lead to progressive neurological damage. Interestingly, our patient also had non-infective fever, as reported in SPAM, which is likely neurogenic.

The unprecedented situation we faced of relapsing and progressive symptoms necessitated different surgical strategies, initially with only partial success. The fact that symptoms recurred raises the possibility of whether the first operation was inadequate. Both operations involved a duroplasty, the second more extensive with removal of the cross-linking rod. Ultimately, the patient responded well clinically and radiologically to extensive cord de-tethering and a large expansion duroplasty. We thus advise that a generous duroplasty from the outset might avoid recurrence. There are two take home messages: First, careful monitoring and an urgent MRI scan are essential if a person with acute TSCI develops new neurological symptoms. Second, rehabilitation and neurosurgical teams need to be aware of the spectrum of SPAM, CRAM and PTS and the benefits of surgery in managing these. Early expansion duroplasty should be considered as opposed to a ‘watch and wait’ strategy, which may cause significant neurological injury and even mortality in some of these conditions, as already described in SPAM.3

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**Disclosure statement**

No potential conflict of interest was reported by the author(s).

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