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Response to: Very rare incidence of ascending paralysis in a patient of traumatic spinal cord injury: a case report

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TO THE EDITOR:

We read with interest the paper by Biswas et al. [1] reporting on a "Very rare incidence of ascending paralysis in a patient of traumatic spinal cord injury: a case report". They comprehensively describe a case of delayed neurological deterioration following traumatic spinal cord injury (TSCI). Two weeks post-operatively, their patient developed progressive sensory loss and weakness of the abdominal musculature. There was ascension of the level of injury from T12 to T2 at 25 days post-op, with compatible hyperintense T2 signal change on MRI. The authors attribute the clinical presentation to Subacute Progressive Ascending Myelopathy (SPAM).

Delayed neurological deterioration following TSCI is well-documented in the literature, and not "Very rare" as Biswas et al report, including SPAM and post-traumatic syringomyelia (PTS). We recently published our experience of a related condition, which we term Chronic Relapsing Ascending Myelopathy (CRAM) [2]. Our patient experienced delayed ascending neurological deterioration at four months post-TSCI. In contrast, the neurological level of injury ascended from T4 to C5, responded to surgery with a relapsing course, and overall improved to their neurological baseline. We propose that SPAM, CRAM, and PTS, lie on a spectrum, each with differing clinical, radiological, and temporal patterns.

Importantly, CRAM is treatable. Our patient's symptoms resolved with surgical detethering and expansion duroplasty of the spinal cord. Biswas et al postulate several theories for the neurological decline. Similar to their case, the biochemical, CSF and histological markers in our CRAM case were not consistent with underlying inflammatory, vasculitic or demyelinating pathology. However, intra-operative spinal cord monitoring in our case favours a mechanical aetiology. We demonstrated pulsatile CSF above the injury compared with non-pulsatile CSF below, at higher pressure. The pressure differential was corroborated by post-traumatic adhesions visualised intra-operatively, which may act as an obstructive focus of tethering.

Biswas and colleagues treated the patient with methylprednisolone infusion. They acknowledge that arrested neurological decline cannot be ascribed with certainty to the drug. We propose that progressive symptoms or adverse neuroradiological change in either SPAM, CRAM or PTS should lead to consideration of neurosurgical detethering, through adhesiolysis +/− expansion duroplasty.

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AUTHOR CONTRIBUTIONS

RV was responsible for writing the manuscript. MCP was responsible for review and feedback on the manuscript.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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