SHORT REPORT

Delayed resolution of extensive T2-weighted intramedullary signal changes after oblique corpectomy for cervical spondylotic myelopathy

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Abstract
We report two cases of cervical spondylotic myelopathy (CSM) with extensive T2-weighted intramedullary changes noted on preoperative imaging extending far beyond the level of compression. A delayed resolution 2 years after cervical oblique corpectomy was noted in both cases. This short report cautions against diagnosing this unusual magnetic resonance imaging (MRI) finding as an intramedullary tumour, demyelination or an inflammatory process.

Key words: Cervical spondylotic myelopathy, T2-weighted signal changes, oblique corpectomy.

Introduction
This report discusses two cases of CSM with extensive T2-weighted intramedullary changes noted on preoperative imaging with their delayed resolution 2 years after cervical oblique corpectomy. In our series of 167 consecutive patients with CSM who underwent an oblique corpectomy, 125 showed T2-weighted intramedullary hyperintensity at the level of compression. This report highlights the occurrence of extensive T2-weighted changes extending far beyond the levels of compression in two patients and their late but ultimate resolution with good clinical outcomes after surgery.

Case report

Case 1
A 53-year-old male presented with progressive spastic quadripareis over 18 months and paresthesias in all four limbs for 1 year. His functional status was graded as Nurick Grade 3 and a JOA score of 12/18. The cervical spine MRI (Fig. 1a) showed cord compression due to C5, 6 and C6,7 disc prolapses and calcification of the posterior longitudinal ligament behind the C6 vertebral body. The most notable feature was a long T2-weighted intramedullary hyperintensity from C4-T1 without any T1-weighted changes. After he underwent C5 and C6 oblique corpectomies, the cord was well decompressed with restoration of the ventral subarachnoid space as seen on the intraoperative ultrasound. Postoperatively, there was an improvement in spasticity.

His cervical MRI (Fig. 1b) at 6 months showed bulging of the ventral subarachnoid space into the corpectomy defect along with a significant expansion of the cervical cord from C4-T1. The gadolinium T1 sequence showed patchy enhancement at C5-6 (Fig. 1c), while the dynamic cervical X-rays showed no instability albeit with a mild kyphosis. Since he had improved to Nurick Grade 2 with a JOA score of 16/18, he was advised close follow-up. Two years later, the cervical MRI showed complete reduction of the cord expansion with a small residual T2-weighted hyperintense signal at C5. He reported further improvement in spasticity and paresthesia and at 3 years (Fig. 1d), the MR changes had resolved with residual cord atrophy.

Case 2
A 35-year-old man with neck pain, paresthesias and spasticity in all limbs for 4 months was Nurick Grade 2 and had a JOA score of 14/18. The cervical MRI showed a kyphotic spine with ventral compression of the cord at C4 and C5. There was calcification of the posterior longitudinal ligament. The striking feature was an extensive T2-weighted intramedullary hyperintensity from C1-7 without T1W changes.
He underwent C4 and C5 oblique corpectomies and reported a significant improvement in the lower limb spasticity. At 1 year follow-up, his JOA score had improved to 16/18 and the MRI showed a significant reduction in the T2-weighted changes without expansion of the cord. There was no change in the kyphosis.

**Discussion**

Intramedullary T2-weighted hyperintensity at the level of compression is seen in the majority of patients with CSM on MRI and is presumed to be due to myelomalacia, gliosis, demyelination or edema. The persistence of these changes along with contrast enhancement following decompressive surgery has been recognised however, most studies indicate that T2-weighted hyperintensities do not correlate with outcome. There is a suggestion that hypotense areas on T1-weighted images predict a poor prognosis. Occasionally, diagnostic uncertainty arises when spinal canal stenosis co-exists with extensive intramedullary hyperintensities raising the possibility of an intramedullary tumor. Thus an intramedullary glioma should be considered in cases where T2-weighted hyperintensities and contrast enhancement persist after surgical decompression of cervical spondylotic myelopathy particularly in the setting of clinical worsening on follow-up.

The usual craniocaudal extent of intramedullary signal intensity is limited to the level of compression, however, in both our cases the length of T2-weighted changes extended far beyond the level of compression. Although the post-operative regression of signal intensity appears to correlate with clinical improvement the time course of this resolution is not clear. Mastronardi et al. report an immediate complete resolution of T2-weighted signal changes at the end of surgery using intraoperative MRI in 17.4% of patients. Another 52% regressed at 6 months follow-up while the remaining showed no change. The evolution of signal intensity correlated with clinical outcome after surgical decompression. However, their rate of resolution at a later follow-up was not mentioned. Yagi et al. observed postoperative expansion of the T2-weighted intramedullary hyperintensity on T2-weighted MR images in 17 (34%) of 50 patients and instability of the cervical spine and severe compression along the ventral side of the cord were implicated as significant risk factors for these changes. However, information regarding the subsequent management of these patients and the radiological outcome of these cases is not available. In both our cases, there was no instability noted on dynamic X-rays taken at follow-up. Lee et al. reported six cases of CSM presenting with post-operative aggravation of spinal cord edema at the compression level that was present at least 3 months after surgery. Five out of their six patients exhibited clinical improvement over varying periods of time (mean follow-up 21 months) and one patient’s neurological status remained unchanged.

In conclusion, extensive intramedullary T2 hyper-intensity beyond the level of compression occurs rarely in CSM, however, when present it does not indicate a poorer prognosis. In such patients, surgical decompression may result in rapid expansion of the cord without clinical deterioration but is usually followed by a delayed resolution of the edema.

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**References**


**Fig. 1.** (A) Pre-operative sagittal T2-weighted MR images showing extensive T2W intramedullary signal intensities from C4-T1 exceeding the level of compression. (B) Six months post-operative T2-weighted sagittal images showing bulging of the ventral subarachnoid space into the corpectomy defect with T2-weighted intramedullary hyperintensities and expansion of the cervical cord from C4-T1 with enhancement of the cord at C5-6 corresponding to the level of compression seen on the T1W sequence after Gd administration. (C) and (D) T1- and T2-weighted sagittal images showing resolution of the changes with atrophy of the cord at the level of compression 3 years after oblique corpectomy. (E) and (F) Plain cervical dynamic radiographs showing no instability.