

7T MRI of the pathological spinal cord

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Target audience. Researchers/clinicians using spinal cord MRI.

Purpose. Patients with neurodegenerative diseases and traumas affecting the spinal cord could benefit from better structural imaging to improve accuracy of diagnosis, prognosis and monitoring treatment responses. Improved understanding of disease mechanisms and progression might also emerge as happened with brain MRI. More specifically: ability to quantify gray matter atrophy might provide a biomarker of motor neuron diseases including amyotrophic lateral sclerosis; ability to assess demyelination in white matter pathways could inform on the overall damage after spinal cord injury, and characterizing lesion load, size and location in multiple sclerosis and neuromyelitis optica could help characterize new disease phenotypes. MRI is currently the modality of choice for imaging the spinal cord. Despite recent advances in multi-parametric MRI for detecting demyelination [1] or the use of high resolution for quantifying gray matter atrophy [2], current standards in RF coils and field strength limit for the maximum spatial resolution that can be achieved at reasonable acquisition time. Recently, ultra-high field MRI (7T and beyond) has yielded dramatic improvements in spatial resolution and contrast [3]. In this paper we translate recent developments in 7T MRI and Rx coil technologies to two clinical areas: spinal cord injury (SCI) and amyotrophic lateral sclerosis (ALS).

Methods. *Case #1: SCI.* A man with 25 years of mild left neck, arm, and leg paresthesias had initial MRI in 1996 identifying left C3-4 dorsal-horn cavernous hemangioma. In 1997, hemorrhage (C3-T3) and resection induced left arm>leg proprioceptive loss and clumsiness. Three-month post-resection left upper-body pain recurred; two years later disabling colocalizing itch recurred. *Case #2: ALS.* Subject was a 52 year-old man who developed progressive weakness and spasticity in the left leg 23 months before scanning. He had signs of both upper and lower motor neuron dysfunction in the bulbar, cervical, and lumbar segments. The revised ALS functional rating scale (ALSFRS-R) score was 38/48. The healthy control was 48 years old and had no medical history of neurological disorders. *Data acquisition* was done on a 7T whole-body scanner (Siemens Healthcare). Transmission/reception was performed with a custom-made 4-channel transmit coil (used in single-transmit) and a 19-channel receive coil [4]. Multiple automatic B₀-shimming was first performed with a gradient echo sequence. High-resolution images were then acquired with a multi-echo T₂*-weighted fast low angle shot (FLASH) sequence: 13 axial slices of 3 mm thickness covering C2-C7, TR = 514 ms, TE = (7.83,15,22.21,29.43) ms, 2 averaging, matrix = 534 × 480, in-plane resolution = 0.37 × 0.37 mm² (no interpolation), R=2 acceleration, bandwidth = 195 Hz/pix, acquisition time = 4:24 min. The sequence included a phase-correction scheme based on a reference echo (non phase-encoded).

Results. *Case #1* (see Figure 1). 7T MRI localized hemosiderin (iron-storage complex, seen as hypointensity) at specific dorsal-horn laminae and detected rostral (C1-C3) hypersignal that was previously unseen on conventional MRIs. No abnormality was detected below C7. *Case #2.* T₂*-weighted hyperintensity was visible in both lateral segments, from C2 to C6 (Figure 2). The location of the hyperintensity corresponds to the lateral corticospinal tract and possibly includes the rubrospinal and lateral reticulospinal tracts. The most lateral segments of the cord corresponding to the ascending spinothalamic and spinocerebellar tracts do not exhibit hyperintensity. When comparing T₂*-weighted signal in the lateral segment normalized by signal in the dorsal segment, ANOVA showed a significant difference between the control subject and the ALS patients ($F=65.29, p=3.55 \times 10^{-15}$), and there was no significant difference between right and left segments ($F=1.02, p=0.31$).

Discussion. In the SCI patient (Case #1), hyperintensity was detected above—but not to below—the hemosiderin-induced hypointensity in the dorsal left column (ascending pathways), suggesting Wallerian degeneration. This new finding generated the hypothesis that late-onset central itch observed in this patient might be related to delayed white-matter degeneration. The ALS patient (Case #2) presented clear T₂*-weighted hyperintensities in both lateral segments, suggesting degeneration of the CST. While previous studies reported similar results at lower fields [5, 6], the unprecedented spatial resolution (0.37 micron) at high contrast-to-noise enabled delineation of abnormalities in the CST area with minimum partial volume effect with neighboring ascending tracts, presumably not affected by ALS.

Conclusion. 7T MRI combined with advanced coil technologies has the potential to bring new and relevant diagnosis information in the pathological spinal cord. More details can be found in [7, 8].

References. [1] Cohen-Adad J et al., *NeuroImage*, 2011. **55**(3): p. 1024-33. [2] Yiannakas MC et al., *NeuroImage*, 2012. **63**(3): p. 1054-1059. [3] Sigmund EE et al., *NMR Biomed.*, 2011. [4] Zhao W et al., *ISMRM, Melbourne*, 2012. **310**. [5] Mascalchi M et al., *AJNR*, 1995. **16**: p. 878-80. [6] Terao S et al., *J Neurol Sci*, 1995. **133**: p. 66-72. [7] Cohen-Adad J et al., *Muscle Nerve*, (in press). [8] Cohen-Adad J et al., *Neurology*, (in press).

Acknowledgments. Study funded by the NIH NCRR P41 RR14075, NIBIB R01EB006847, NINDS K24NS059892 and National MS Society [FG1892A1/1].

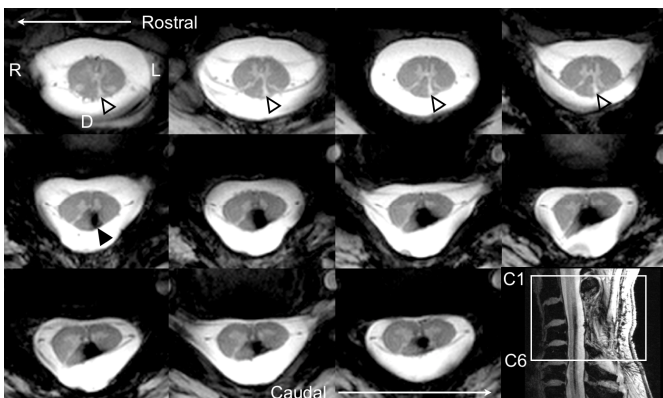


Figure 1. 7T-MRI of spinal cord injury. At-level hyposignal (plain arrow) indicates hemosiderin while above-level hypersignal (empty arrows) suggests dorsal-column Wallerian degeneration. Ultra-high-resolution enabled exquisite details of spinal-cord anatomy.

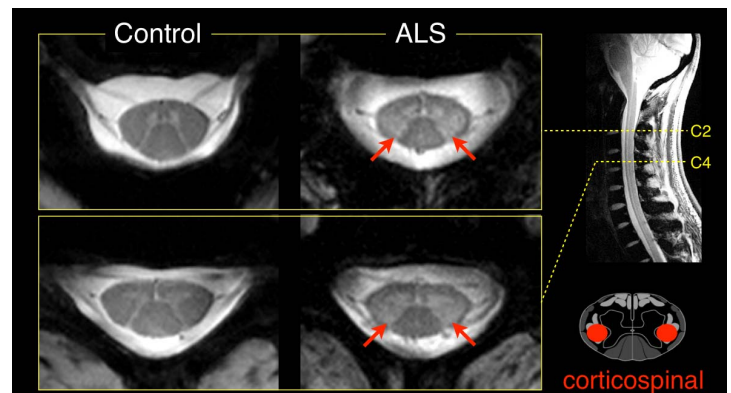


Figure 2. Axial view of T₂*-weighted images in the control subject and in the ALS patient. Slices are centered at the C2 (top row) and C4 (bottom row) vertebral levels. Hyperintensity is clearly visible in both lateral segments of the spinal cord in the ALS patient, likely corresponding to the lateral corticospinal tracts (CST).