MAGNETIC RESONANCE SPECTROSCOPY IN THE MOTOR AND SENSORY CORTICES FOLLOWING SURGERY FOR CERVICAL SPONDYLOTIC MYELOPATHY

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Target Audience: Clinicians and scientists interested in the metabolite level changes in the brain caused by spinal cord compression.

Purpose: Cervical spondylotic myelopathy (CSM) is the most common type of spinal cord impairment in adults over the age of 55 and leads to metabolic and functional changes in the brain.[1] It is commonly treated by spinal decompression surgery.[2] The purpose of this study was to compare the time course of changes in *N*-acetylaspartate (NAA) in the motor and sensory cortices following decompression surgery with the recovery of motor and sensory function.

Methods: We studied 24 patients (mean age = 53, 20 male) with CSM and 8 control subjects (mean age = 51, 5 male) using long echo-time (PRESS, TE=135, TR=2000, nt=192) ^{1}H MR spectroscopy (MRS) acquired on a 3.0T Siemens MRI. Absolute levels of *N*-acetylaspartate (NAA) were measured from two 8 cm^3 voxels positioned over the motor and sensory cortices in each subject on the side of greater motor deficit or on both sides in control subjects. Absolute metabolite levels were measured by referencing to the total unsuppressed voxel water signal (nt=8) and adjusting for gray matter/white matter / cerebral spinal fluid partial volume measured from T_1-weighted images (1mm isotropic resolution) as previously described.[3] Measurements were made at baseline and repeated in 17 patients at 6 weeks and 6 months following spinal decompression surgery. The American Spinal Injury Association (ASIA) physician-objective neurological outcome measure was also performed at each visit to grade motor and sensory symptom changes. Baseline NAA levels were compared between groups using a ttest. Repeated measures ttests was used to compare metabolite levels as a function of time in each brain region (in all comparisons $p<0.05$ was considered statistically significant).

Results: Patients had lower levels of NAA ($p<0.05$) in both motor and sensory cortices compared to healthy controls (Figure 1). In patients with follow-up data (N=17), NAA was significantly decreased in the motor cortex at 6-weeks ($p<0.05$) and at 6-months post surgery ($p=0.05$) compared to baseline (Figure 2). Decreased NAA was also found in the sensory cortex at 6 months post-surgery ($p<0.05$) compared to baseline, as well as between 6-weeks and 6-months post-surgery ($p<0.05$) (Figure 3). Neurological function measured with the (ASIA) neurological classification demonstrated significant motor function improvements from baseline to 6 weeks ($p<0.05$, Figure 2) and significant sensory function improvements from 6-weeks to 6-months ($p<0.05$, Figure 3).

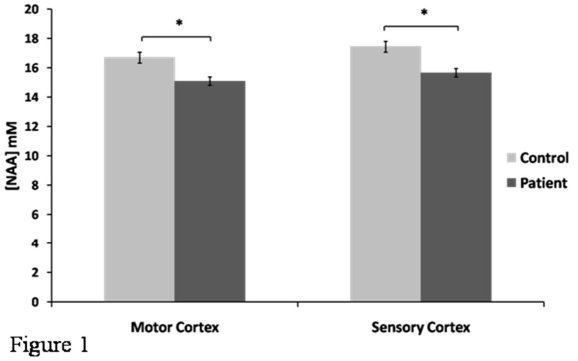

Figure 1

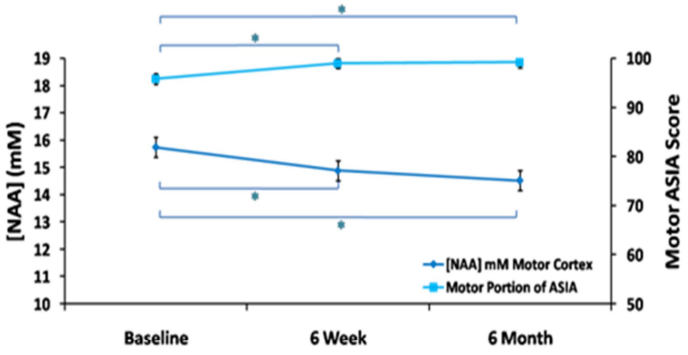

Figure 2

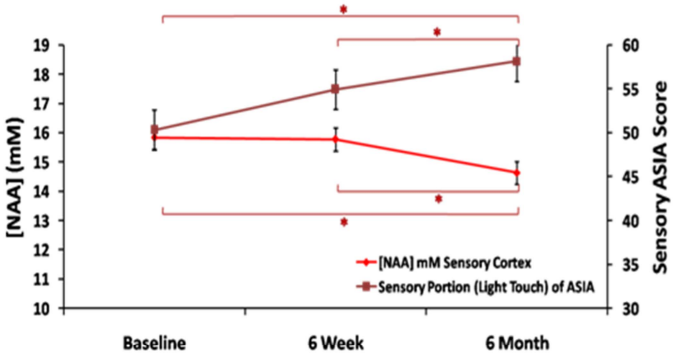

Figure 3

Discussion and Conclusion: Lower baseline NAA levels in CSM patients compared to healthy controls indicates metabolic impairment in both the motor and sensory cortex consistent with a previous study that found reduced NAA/Cr in the motor cortex in CSM patients.[1] After spinal decompression surgery, a further decline in NAA levels was found in both motor and sensory cortices that mimic the time course of functional recovery - although in the opposite direction. Future studies should investigate the neurological effects caused by declining NAA levels in the presence of functional recovery.

References: **1**.Kowalczyk I, et.al., Brain, 2012. 135 (Pt 2): p. 461-8. **2**.Fehlings and Arvin, J Neurosurg Spine, 2009. 11 (2): p.97-100. **3**.Bartha, R., et.al., NMR Biomed, 1999. 12(4): p. 205-16.