

DIAGNOSTICS

Do Quantitative Magnetic Resonance Imaging Parameters Correlate With the Clinical Presentation and Functional Outcomes After Surgery in Cervical Spondylotic Myelopathy?

A Prospective Multicenter Study

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Study Design. A prospective multicenter cohort study.

Objective. To establish the relationship between preoperative quantitative magnetic resonance imaging (MRI) parameters and clinical presentation and postoperative outcomes in patients with cervical spondylotic myelopathy.

Summary of Background Data. Correlation of magnetic resonance imaging with clinical presentation and outcomes in cervical spondylotic myelopathy is poorly understood.

Methods. A total of 134 magnetic resonance imaging scans were reviewed from 12 sites across North America. The transverse area (TA) of the spinal cord at the site of maximal compression was computed, and spinal cord signal intensity (SI) changes on T1-/T2-weighted imaging (WI) were evaluated. Detailed clinical assessments—neurological signs, symptoms, Nurick grade, modified Japanese Orthopaedic Association, segmental-tract score, and long-tract score of modified Japanese Orthopaedic Association, 30-m walk

test, Short-Form 36 questionnaire, and neck disability index were performed at admission, 6 months, and 12 months postoperatively.

Results. The total number of neurological signs in a patient correlated with TA ($P = 0.01$) and SI changes on T1-/T2WI ($P = 0.05$). Pre- and postoperative Nurick grade ($P = 0.03$, $P = 0.02$), modified Japanese Orthopaedic Association score ($P = 0.005$, $P = 0.001$), segmental-tract score ($P = 0.05$, $P = 0.006$), and long-tract score ($P = 0.006$, $P = 0.002$), 30-m walk test ($P = 0.002$, $P = 0.01$) correlated with TA. There was no significant difference in pre- and postoperative clinical scores in patients with/without SI changes. Patients with severe cord compression showed SI changes on T1-/T2WI more frequently ($r = -0.27$, $r = -0.38$). Pyramidal signs—plantar response, Hoffmann reflex and hyper-reflexia correlated with TA ($P = 0.003$, $P = 0.0004$, $P = 0.024$, respectively) and SI changes on T1/T2WI ($P = 0.02$).

Conclusion. TA closely mirrors the clinical presentation of cervical spondylotic myelopathy and may be used in predicting surgical outcomes. Pyramidal signs correlated with TA and/or SI changes on T1-/T2WI. The total number of neurological signs in a patient correlated with TA. There was no significant relationship between TA, age and duration of symptoms.

Key words: cervical spondylotic myelopathy, quantitative MRI, clinical outcomes.

Level of Evidence: 3

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Cervical spondylotic myelopathy (CSM) is the most common underlying cause of spinal cord injury in individuals older than 55 years.¹ This condition is caused by the degeneration of various components of the vertebral column leading to narrowing of the spinal canal and subsequent compression in many cases.²

Magnetic resonance imaging (MRI) is commonly used in the diagnosis and treatment of CSM. It also determines the timing of surgical intervention, influences the type of

surgery, and is useful in the prognosis of the outcome. Previous studies have not shown a consistent and definitive relationship between MRI parameters and the functional/neurological measures used to define the severity of CSM.³⁻⁸ In the existing CSM literature, clinical presentation is assessed using self-reported symptoms⁹⁻¹¹ and objective neurological signs,^{10,12-14} Nurick grade,^{6,15,16} modified Japanese Orthopaedic Association (mJOA),¹⁷⁻²⁰ neck disability index (NDI),^{21,22} and the Short-Form 36 questionnaire (SF-36).^{22,23} It has been suggested that MRI can predict the degree of functional recovery after surgery for those with CSM. However, definitive information is not available because MRI-based measures in previous studies have been either qualitative in nature,²⁴⁻²⁷ or, when quantitative, have not been prospective cohort studies.^{3,4,28} Given these shortcomings in the existing literature, we sought to conduct a prospective study to define the association between frequently used quantitative MRI parameters—transverse area (TA) and signal intensity (SI) changes—with different neurological signs and symptoms along with various clinical scores. We also sought to assess the role of quantitative MRI in predicting surgical outcomes in patients with CSM.

MATERIALS AND METHODS

Ethical approval was attained at all sites involved in the study (12 sites across North America). A total of 277 patients with CSM (164 males and 113 females of whom 220 patients were younger than 65 yr) were consecutively and prospectively enrolled from academic institutions between February 2006 and November 2007. Of these, adequate MRI sequences were available for 134 patients (Table 1). Fifty-six patients were excluded because of medical contraindications for MRI (*e.g.*, pacemaker). Another 87 patients who met these criteria were excluded because of poor-quality imaging and motion artifacts that made it difficult to perform accurate quantitative MRI measurements. All patients underwent preoperative and postoperative imaging (computed tomography/MRI). All patients had adequate cord decompression as confirmed by MRI scans at 6 months postoperatively, as per study protocol. None of them required revision surgery for inadequate cord decompression. One hundred fourteen patients had cervical spondylosis, 13 had herniated disc, 1 had ossification of posterior longitudinal ligament, and 6 had a combination of more than 1 cause mentioned in the earlier text. Posterior decompression was performed in 46 patients, anterior decompression in 84 patients and a combination of both in 4 patients. Clinical diagnosis of CSM was confirmed by MRI or computed tomographic findings. Inclusion and exclusion criteria are described in Table 1.

Procedures and Outcome Measures

Data collection was monitored externally to ensure integrity and completeness. Primary outcome measures included: Nurick grade,¹⁵ mJOA,²⁹ each subscore of the mJOA, segmental-tract score (STS), and long-tract score (LTS) of

TABLE 1. Demographics, Clinical Findings, and MRI Descriptive of Patients With Cervical Myelopathy in Our Cohort

Characteristics	(N = 134)
Duration of symptoms, mean ± SEM (mo)	25.0 ± 2.77
Age, mean ± SEM (yr)	55.6 ± 1.00
Transverse area of spinal cord ± SEM (mm ²)	45.6 ± 1.21
Signal intensity changes, no. (%)	
Normal T1/normal T2	44 (33)
Normal T1/high T2	65 (49)
Low T1/high T2	21 (16)
Missing	4 (2)
Sex, no. (%)	
Female	49 (37)
Male	85 (63)
Severity of cervical myelopathy, no. (%)	
Mild (mJOA ≥15)	45 (34)
Moderate (mJOA, 12–14)	51 (38)
Severe (mJOA <12)	38 (28)
Cause of myelopathy, no. (%)	
OPLL	1 (1)
Spondylosis	114 (85)
Disk	13 (9)
More than 1 cause	6 (5)
Cervical alignment, no. (%)	
Lordosis	50 (37)
Neutral	52 (52)
Kyphosis	14 (11)
Type of surgery, no. (%)	
Anterior	84 (63)
Posterior	46 (34)
Both anterior and posterior	4 (3)
Improvement in 1 year, no. (%)	
mJOA	2.7 ± 0.24
Nurick	1.4 ± 0.11
Segmental*	1.5 ± 0.15
Long tract*	1.2 ± 0.14
Walking test	6.1 ± 1.76 s (time)/ 5.3 ± 1.44 (cadence)

(Continued)

TABLE 1. (Continued)

Characteristics	(N = 134)
NDI†	11.7 ± 1.75
SF-36‡	0.08 ± 0.01

*Segmental and long-tract segmental score of mJOA (sum of motor dysfunction of the upper extremities and hand sensations) and long-tract score of mJOA (the sum of motor dysfunction of the lower extremities and sphincter dysfunction) (Hirabayashi et al³⁰).

†NDI.²¹

‡Short Form 36 (SF-36).²³

The scale assesses upper extremity function (5 points), lower extremity function (7 points), sensory function (3 points), and urinary bladder function (3 points). Scores range from 0–18 with higher scores indicating better function (Benzel et al²⁹).

Inclusion and exclusion criteria: clinical diagnosis of CSM was confirmed by MRI or computed tomographical findings. CSM was defined as a constellation of symptoms and signs supported by appropriate radiological findings, including at least one neurological symptom (numb clumsy hands, impairment of gait, bilateral arm paresthesia, Lhermitte phenomenon) and at least 1 neurological sign (corticospinal distribution motor deficits, atrophy of hand intrinsic muscles, hyper-reflexia, positive Hoffman sign, up going plantar responses, lower limb spasticity, broad-based unstable gait). Patients with asymptomatic cervical cord compression, previous surgery for CSM, active infection, neoplastic disease, rheumatoid arthritis, ankylosing spondylitis, trauma, and concomitant symptomatic lumbar spinal stenosis were excluded from the study.

CSM indicates cervical spondylotic myelopathy; OPLL, ossification of the posterior longitudinal ligament; SEM, standard error of the mean; mJOA, modified version of Japanese Orthopaedic Association Scale; MRI, magnetic resonance imaging; NDI, neck disability index.

the mJOA separately,³⁰ time (Wt) and cadence (Wc) of the 30-m walk test,³¹ and self-reported measures (SF-36²³ and NDI²¹). The clinical scores were recorded at admission, 6 months, and 12 months of follow-up.

MRI Interpretation

Radiologists were blinded to the patient's clinical presentation. They analyzed all MRI scans using 2 parameters: TA of the spinal cord at the level of maximal compression (Figure 1), and changes in spinal cord SI on T1- and T2-weighted sequences (T1W and T2W) (Figure 2B, A, respectively). TA was measured on T2-weighted axial MR images with a digitizer linked to a computer (Issa Network station 3.1).^{27,31,32}

TA of the spinal cord was classified into 2 groups—TA_{critical} (<0.45 cm²) and TA_{noncritical} (>0.45 cm²). The cutoff point is the critical point beyond which functional impairments of the spinal cord become irreversible.³² The appearance of SI changes on T1-/T2 (weighted imaging) WI sequences were classified into 3 patterns: type 0, normal T1WI/normal T2WI; type 1, normal T1WI/high T2WI; and type 2, low T1WI/high T2WI.^{4,6,33} Increased or decreased SI was defined on the T2W sagittal images and T1W axial images, respectively, as a high-intensity area in relation to the signal of the normal medulla at the unaffected level. Earlier studies have reported this method to be reliable and the concordance correlation coefficient between 2 observers on a single occasion was 0.62 ($k = 0.37$; $P = 0.0063$).⁴

Statistical Analysis

Univariate analyses were carried out for all neurological symptoms, signs, and outcome measures. Where it was applicable, recovery ratio or change in scores was calculated and correlated with the quantitative preoperative MRI parameters. Statistical analysis included the Fisher protected least significant difference, Spearman rank correlation, and Mann-Whitney tests. A P value of less than 0.05 was selected as significant. All analyses were performed using constructed data sets in SAS version 9.3 (SAS Institute Inc., Cary, NC).

RESULTS

There were 85 males and 49 females in this cohort with their mean age being 56 years (range: 29–82 yr). Table 1 presents the demographics and diagnostic characteristics of the sample. Six-month and 12-month follow-up assessments were performed in more than 80% of the cases.

Overall there were improvements at 6 months and 12 months postoperatively for the mJOA score (2.18 ± 0.22 and 2.68 ± 0.24), STS of mJOA score (1.18 ± 0.13 and 1.46 ± 0.14), LTS of mJOA score (1 ± 0.14 and 1.22 ± 0.14), Nurick grade (1.18 ± 0.12 and 1.41 ± 0.11), change in Nurick grade (0.44 ± 0.06), and NDI ($10.50\% \pm 1.74\%$ and $11.71\% \pm 1.75\%$) ($P < 0.05$). There were also improvements in the SF-36 Index (0.06 ± 0.01 and 0.08 ± 0.01),

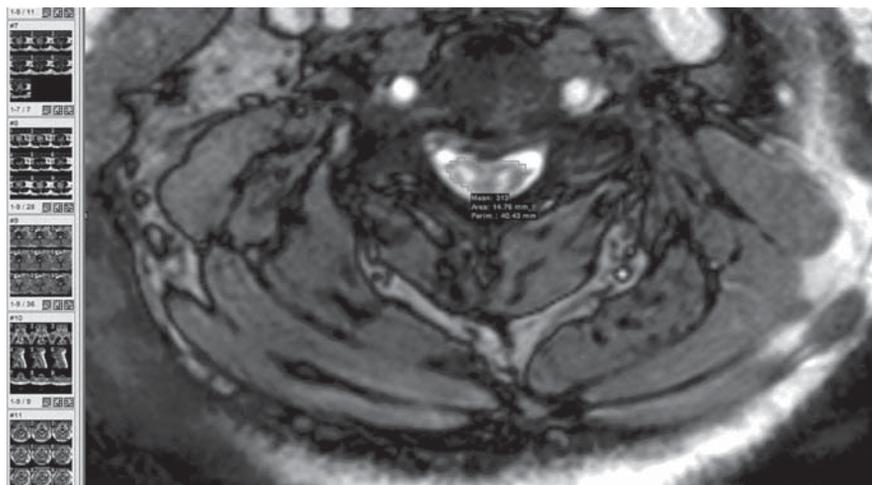


Figure 1. Measurements for the TA of the spinal cord using T2-weighted axial MR image with a digitizer linked to a computer (Issa Network station 3.1). TA indicates transverse area; MR, magnetic resonance.

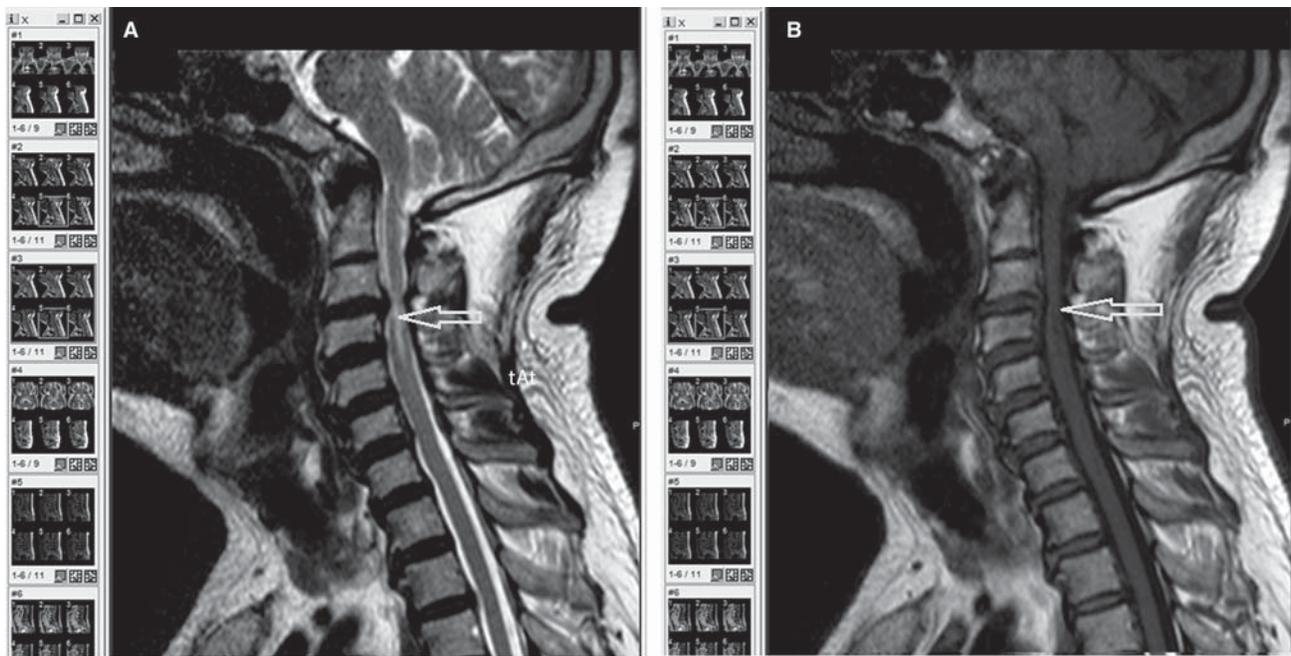


Figure 2. T2-weighted sagittal MRI (A) of the cervical spine with SI changes in the spinal cord as indicated by the white arrow head and T1-weighted sagittal MRI (B) of the cervical with SI changes in the spinal cord as indicated by the arrow head. The appearance of spinal cord SI changes on T1-/T2-weighted sequences was classified into 3 patterns: type 0, normal T1WI/normal T2WI; type 1, normal T1WI/high T2WI; and type 2, low T1WI/high T2WI. Increased or decreased SI was defined on the T2WIs and T1WIs, respectively, as a high-intensity area in relation to the signal of the normal medulla at the unaffected level. MRI indicates magnetic resonance imaging; SI, signal intensity; WI, weighted imaging.

Wt (18.06 ± 4.54 s and 18.06 ± 4.54), and Wc (16.35 ± 3.33 and 16.35 ± 3.33 steps). All scores were significantly lower, indicating improvement, postoperatively ($P < 0.05$). There was also a significant difference between these scores at 6 months compared with those at 12 months postoperatively ($P < 0.05$) except for the self-reported scores NDI and SF-36 Index scores ($P = 0.26$, $P = 0.19$, respectively).

Association Between Quantitative MRI Parameters and Clinical Features

Total number of neurological signs recorded in a patient correlated with TA but not with SI changes ($r = 0.34$, $P = 0.013$ and $r = 0.14$, $P = 0.054$). TA and SI changes correlated with upgoing plantar reflex and numbness of hands. TA individually correlated with Hoffman and hyper-reflexia and SI changes with Lhermitte phenomenon (Table 2). Interestingly, 26 of the 43 patients with MR images showing no SI changes and 48 of 62 with T2 SI changes had Lhermitte phenomenon, whereas 16 of 19 patients with both T1 and T2 SI changes exhibited this phenomenon ($P = 0.021$). The overall prevalence of myelopathic signs and symptoms were higher among people who exhibited T2SI changes, as shown in Figure 3.

Association Between TA and Patient Demographics/Outcome Scores

There was no significant relationship between TA, age, and duration of symptoms ($P > 0.05$). Consistently, an association between TA and baseline mJOA scores ($r = 0.24$, $P = 0.005$), Nurick grade ($r = -0.3$, $P = 0.03$) and Wt ($r = -0.3$, $P = 0.0015$) was observed. At 6-month follow-up, patients

with a lesser degree of spinal cord compression or high TA had lower postoperative Nurick grade ($r = -0.23$, $P = 0.02$), Wc ($r = -0.26$, $P = 0.01$), and a higher mJOA score ($r = 0.3$, $p = 0.001$). Similar results were seen at 12 months; patients with high TA had lower postoperative Nurick grade ($r = -0.18$, $P = 0.047$), decreased Wt ($r = -0.22$, $P = 0.04$), decreased Wc ($r = -0.27$, $P = 0.01$), and higher mJOA score ($r = 0.22$, $P = 0.02$) (Tables 2, 3).

Association Between SI and Patient Demographics/Outcome Scores

There was no significant relationship between SI, age, and duration of symptoms ($P > 0.05$). More specifically, the mean duration of symptoms was 25.5 ± 8.74 , 24.3 ± 7.65 , and 25.1 ± 9.2 months in groups with SI changes on T2WI only, T1-/T2WI and normal, respectively. For analysis of SI, the sample was divided into 3 groups: 44 patients had no SI changes, 65 patients had SI changes on T2WI only, and 21 patients had SI changes on both T1 and T2WI. The data showed no significant association between SI changes on either T1 or T2WI, or both, and clinical outcomes as measured by mJOA, Nurick grade, STS and LTS functions of mJOA score, Wt, Wc, NDI, and SF-36 Index (Table 2). There was a trend toward a better functional status, measured by mJOA, Nurick grade, SF-36, NDI, and Wt, associated with hypointensity on T1WI and hyperintensity on T2WI at 6-month follow-up. Improved functional status, measured by Nurick grade, mJOA, SF-36, and the NDI scales, was associated with high T2SI at 12-month follow-up (Table 4). However, the trend was insignificant.

TABLE 2. Correlation of Preoperative MRI-Based Measures of the Degree of Severity of CSM and Preoperative Clinical Presentation in the Cohort (*r* and *P*, Significance is 0.05)

Clinical Measure	Transverse Area of Spinal Cord (mm ²)	Normal T1/Normal T2 vs. Normal T1/High T2 vs. Low T1/High T2
Signs count	$r = 0.34, P = 0.013$	$r = 0.14, P = 0.0542$
Corticospinal distribution motor deficits	NS	NS
Atrophied hand	NS	NS
Hyper-reflexia	$r = 0.19, P = 0.0242$	NS
Positive Hoffman sign	$r = 0.30, P = 0.0004$	NS
Up going plantar responses	$r = 0.26, P = 0.0028$	$r = -0.2, P = 0.0188$
Lower limb spasticity	NS	NS
Broad-based, unstable gait	NS	NS
Symptoms count	NS	NS
Numbness of hands	$r = 0.18, P = 0.0359$	$r = -0.2, P = 0.0173$
Clumsy hands	NS	NS
Impaired gait	NS	NS
Bilateral arm paresthesia	NS	NS
Lhermitte phenomenon	NS	$r = 0.2, P = 0.0182$
Weakness	NS	NS
mJOA total	$r = 0.24, P = 0.005$	NS
Motor dysfunction of the upper extremities	$r = 0.22, P = 0.008$	NS
Motor dysfunction of the lower extremities	$r = 0.19, P = 0.03$	NS
Sensation	NS	NS
Sphincter dysfunction	$r = 0.22, P = 0.0414$	NS
Upper limbs	$r = 0.17, P = 0.05$	NS
Long tract	$r = 0.23, P = 0.01$	NS
Nurick	$r = -0.3, P = 0.03$	NS
SF-36	NS	NS
NDI	NS	NS
Walking time	$r = -0.3, P = 0.0015$	NS
No. of steps	NS	NS

MRI indicates magnetic resonance imaging; CSM, cervical spondylotic myelopathy; mJOA, modified Japanese Orthopaedic Association; SF-36, Short-Form 36 questionnaire; NDI, neck disability index; NS, not significant.

Association Between SI and TA (or TA_{critical} and TA_{noncritical})

We correlated TA with SI changes in patients with any form of SI changes and a significant correlation was found ($r = -0.27, P < 0.0001$). Interestingly, when TA was compared across 2 groups (presence *vs.* absence of SI changes on T2WI only), an even stronger correlation was found ($r = -0.38, P < 0.0001$). Fifty-eight patients were found to have TA_{critical} of which 47 (81%) had SI changes. Of these, 37 had SI changes on T2WI only (63.8%). In patients with TA greater than the

critical value, 34 patients had SI changes (50%) and 25 of these had changes only on T2WI (37%). Hence, patients with TA less than critical have a higher chance of developing SI changes, especially so on T2WI ($P = 0.0023$).

DISCUSSION

The relationship between the degree of radiologically observed cord compression with baseline clinical severity scores and their predictive value is still controversial in the setting of CSM.^{5,16,19,20,28,34-37} In our study, there was an

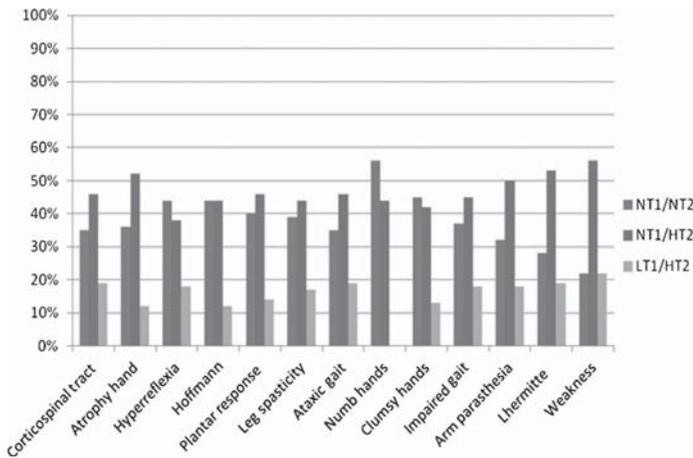


Figure 3. Bar graph shows the frequency of each sign and symptom in patients with CSM with no signal intensity changes on T1- or T2WI, patients with high T2 signal change, and patients with both high T2 and low T1 signal changes. CSM indicates cervical spondylotic myelopathy; WI, weighted imaging.

overall improvement in clinical outcomes at 6 months compared with the preoperative scores. When objective scales were used, patients continued to improve up to 12 months postoperatively, however, self-reported scores failed to reflect this. This is perhaps a reflection of the fact that patients with CSM undergoing surgery noticed a maximal improvement in their quality of life in the first 6 months and/or the symptomatology of pain and well-being were not improved with the procedure.

Quantitative MRI Parameters and Clinical Features

Controversy remains regarding the neurological signs and symptoms of CSM. The total number of neurological signs recorded in a patient had a strong relationship with TA. We suggest that this could be due to progression in the mechanical and vascular compromise to the cord, resulting in a greater number of deficits. It has been shown in animals that one of the potential molecular mechanisms of progressive loss of neurons and demyelination in the cord is progressive cord compression.³⁸

Both TA and SI correlated with upgoing plantar reflexes and numbness of hands: TA individually correlated with Hoffman Sign and hyper-reflexia and SI with Lhermitte phenomenon. Few studies have attempted to establish an association between MRI findings and clinical signs/symptoms.^{12,39-46} Hyper-reflexia was associated with the preoperative SI changes on both T1-/T2WI and was found in patients with poorer outcomes after surgery.¹² In our study, although SI changes on T1 and T2 did not correlate with hyper-reflexia, the degree of spinal cord compression (TA) did. It was also found that sphincter dysfunction correlated to the degree of spinal cord compression observed on MRI and, therefore, the clinical severity of CSM. However, sphincter dysfunction is inconsistently reported as a clinical symptom of CSM.^{9-11,20} Moreover, unlike previous studies,^{9,12-14,47} our study found no association between MRI-based measures of spinal cord

compression and clinical features such as leg spasticity, hand muscle atrophy or gait impairment. The clinical definition of CSM continues to remain ambiguous.^{12,15,46,48,49} The presence of pyramidal signs such as hyper-reflexia, Hoffmann Sign, clonus, and Babinski Sign, are considered to be the key features of myelopathy.⁵⁰ It is recommended that the clinician focuses on these signs in the preoperative assessment because they indirectly indicate the degree of cord compression and hence dictate the urgency for surgical decompression. Also, the fact that these quantitative MRI parameters correlated with both clinical signs and baseline mJOA, make them a more sensitive measurement tool.

Association Between TA, SI, and Patient Demographics/Outcome Scores

Our study demonstrated that TA consistently correlated with the severity of CSM on presentation and after surgery. Other studies have shown associations between MRI findings and pre- and postoperative JOA and mJOA scores.^{4,51} In this study, there was no statistically significant correlation between SI changes and clinical outcomes at 6 months and 12 months in our study. This trend was consistent across “multiple clinical scales.” Similar trends with lack of statistical significance were found in prior clinicoimaging studies.^{19,20,28,34,35,37} Other studies showed that the presence of SI changes on T2WI seemed to be a promising predictor of higher functional recovery.^{5,11,36,52} In addition, in our study we used a T1-weighted sequence to help differentiate the phases of neural damage. On the assumption that a hypointense signal in the cord on T1WI reflects more severe damage to neural tissue, perhaps irreversible ischemia or frank infarction, a poorer clinical outcome would be expected. Morio *et al*⁴ reported a significant role of SI on T1WI in patients’ with worse outcomes after surgery. However, our study found no differences in preoperative and postoperative clinical observations regardless of SI changes on T1WI. Although no relationship was detected between SI and postoperative outcome in this study, findings from others and a larger study from our own team⁵³ are not in keeping with this. It may be that this study was underpowered to detect the relationship or those other factors such as variability of MRI technique or lack of validated assessments to quantitate SI changes affected the current findings.

The Separate Functions of the Long Tracts and Involved Segments

Splitting the mJOA into its separate components enables researchers to understand if any specific functions are affected by cord compression to a greater extent than the global mJOA score. Our study found that both STS and LTS functions are affected equally (with similar correlation coefficients, both being statistically significant) when the spinal cord is compressed. We could not identify a significant relationship between SI changes and any specific function of the global mJOA score. However, other studies have reported that SI changes on axial T2WI were significantly more frequent among patients with CSM with STS

TABLE 3. Correlation of Preoperative MRI-Based Measures of the Degree of Severity of CSM and Postoperative Outcomes in the Cohort (*r* and *P*, Significance is 0.05)

Clinical Measure	Transverse Area of Spinal Cord (TA) and Postoperative Score at 6-mo Follow-up	Transverse Area of Spinal Cord (TA) and Postoperative Score at 12-mo Follow-up
Nurick (postoperative score)	$r = -0.23, P = 0.02$	$r = -0.18, P = 0.047$
Change in Nurick	NS	NS
Recovery ratio (%)	$r = -0.19, P = 0.05$	NS
mJOA (postoperative score)	$r = 0.3, P = 0.001$	$r = 0.22, P = 0.02$
Change in mJOA (postoperative score)	NS	NS
Recovery ratio, mJOA score (%)	NS	NS
Motor dysfunction of the upper extremities	NS	NS
Motor dysfunction of the lower extremities	$r = 0.34, P = 0.0204$	NS
Sensation	$r = 0.28, P = 0.0265$	$r = 0.27, P = 0.0148$
Sphincter dysfunction	NS	NS
Upper limbs	$r = 0.26, P = 0.0061$	$r = 0.23, P = 0.0136$
Long tract	$r = 0.29, P = 0.0015$	$r = 0.19, P = 0.0415$
SF-36	NS	NS
NDI	NS	NS
Walking time	NS	$r = -0.22, P = 0.04$
Walking (no. of steps)	$r = -0.26, P = 0.01$	$r = -0.27, P = 0.01$

MRI indicates magnetic resonance imaging; CSM, cervical spondylotic myelopathy; mJOA, modified Japanese Orthopaedic Association; SF-36, Short-Form 36 questionnaire; NDI, neck disability index; NS, not significant.

problems rather than LTS problems assessed at 3 months postoperatively.³⁰ All our patients were followed up postoperatively at 2 time points (6 and 12 mo), considered to be the optimal points in recovery after decompressive surgery. The differences between our results and the study of Hirabayashi *et al*³⁰ 2000 could be attributed to the timing of follow-up assessment.

Association Between SI and TA_{critical}

Our results found a significant correlation between TA and pathological changes in the spinal cord such as SI changes on T1 and T2WI. In contrast, several studies reported no significant relationship between the presence of SI changes and TA of the spinal cord found on MRI in patients with cervical myelopathy.^{39,54} Duration of symptoms tends to be longer in patients with SI changes on T1-/T2WI. Our findings highlight that there may be a critical value (TA_{critical}) at which the spinal cord loses its resilience and fails to accommodate compressive forces.³² TA correlated with SI changes in our study, as anticipated. However, contrary to expectation, TA was found to be lowest in patients with SI changes on T2-weighted MR images only and not in the group with changes in both T2

and T1WI. There is a neuropathological correlation with T2SI changes in the form of cystic necrosis resulting from mechanical compression and venous infarction.²⁶ Others have found an association between T2SI and gliosis, myelomalacia, cystic necrosis, and syrinx formation.^{3,4,27,28} It is possible that the data from our study indirectly provide support for the fact that T2SI changes are more detrimental to the clinical outcome than T1SI changes. Recently, the volumetric analysis has been suggested as a more sensitive measure for cord compression but this still needs to be confirmed by further studies.⁵⁵

LIMITATIONS

Although a prospective study holds advantages in overcoming some of the limitations of previous retrospective studies in CSM. There are still a few shortcomings in our study. Ideally, the presence of a control group and randomization would allow MRI characteristics to be adequately correlated with the postoperative prognosis. However, because these patients were surgical candidates, randomization was not possible. Therefore, we conducted a prospective cohort study with stringent criteria of MRI interpretation. Furthermore, a

TABLE 4. Associated Trends in Functional Improvements in Groups at 6- and 12-Month Follow-ups

Clinical Measure	Normal T1/Normal T2	Normal T1/High T2	Low T1/High T2
Nurick (preoperative score)	3.9 ± 0.26	4.2 ± 0.24	4.0 ± 0.21
Nurick (postoperative score) at 6 mo	2.8 ± 0.43	3.0 ± 0.41	2.4 ± 0.36
Nurick (postoperative score) at 12 mo	2.6 ± 0.4	2.5 ± 0.38	2.7 ± 0.33
mJOA (preoperative score)	13.5 ± 0.73	12.7 ± 0.69	13.1 ± 0.60
mJOA (postoperative score) at 6 mo	15.5 ± 0.77	15.0 ± 0.74	16.0 ± 0.65
mJOA (postoperative score) at 12 mo	15.9 ± 0.68	16.1 ± 0.35	15.7 ± 0.56
Recovery ratio at 6 mo (%)	11.40 ± 4.14	12.50 ± 4.01	12.30 ± 3.56
Recovery ratio at 12 mo (%)	13.06 ± 4.37	17.90 ± 4.16	12.50 ± 3.65
Change in mJOA (postoperative score) at 6 mo	1.99 ± 0.74	2.3 ± 0.71	2.3 ± 0.63
Change in mJOA (postoperative score) at 12 mo	2.3 ± 0.76	3.3 ± 0.72	2.3 ± 0.63
Recovery at 6 mo (%)	-0.87 ± 0.35	-0.79 ± 0.34	-1.07 ± 0.07
Recovery at 12 mo (%)	-0.35 ± 0.08	-0.43 ± 0.08	-0.29 ± 0.07
SF-36 index	0.55 ± 0.04	0.60 ± 0.04	0.62 ± 0.03
SF-36 index at 6 mo	0.63 ± 0.05	0.66 ± 0.04	0.69 ± 0.04
SF-36 index at 12 mo	0.66 ± 0.05	0.69 ± 0.04	0.65 ± 0.04
NDI	41.7 ± 5.94	39.7 ± 5.61	34.0 ± 4.85
NDI at 6 mo	31.7 ± 6.31	27.9 ± 6.04	23.4 ± 5.30
NDI at 12 mo	30.6 ± 5.43	24.9 ± 6.18	28.6 ± 5.43
Walking time	26.1 ± 5.44	30.8 ± 5.30	28.7 ± 4.50
Walking time at 6 mo	27.1 ± 3.95	24.6 ± 3.90	21.9 ± 3.42
Walking time at 12 mo	23.5 ± 3.13	27.0 ± 3.61	23.2 ± 3.13
Walking (cadence)	42.9 ± 4.36	47.2 ± 4.26	42.7 ± 3.60
Walking (cadence) at 6 mo	39.9 ± 4.10	43.5 ± 4.04	41.1 ± 3.55
Walking (cadence) at 12 mo	39.8 ± 3.80	41.7 ± 3.63	39.8 ± 3.80

Values denote mean ± SD.

JOA indicates modified Japanese Orthopaedic Association; SF-36, Short-Form 36 questionnaire; NDI, neck disability index.

12-month period of follow-up is sufficient to detect optimal recovery; a longer period of follow-up would be desirable in future studies to understand the long-term effects of decompressive surgery and ongoing neurological change.

We also acknowledge that there may be statistical limitations in our article that could explain why some of our findings, such as those showing that SI was not correlated with clinical presentation or clinical outcome scores, are not consistent with the literature. Work from our group⁵³ has presented more valid, quantitative approaches to assessing MRIs in future work in this area. Despite these limitations, we think that the study makes a useful contribution to a controversial area of study.

CONCLUSION

TA of the spinal cord seems to be a reliable MRI parameter to define the clinical severity of CSM and also to be used as a predictor of postoperative outcomes. We did not find a correlation between clinical presentation or postoperative outcomes and SI changes on T1-/T2WI but cannot rule out that this relationship exists. The total number of neurological signs recorded in patients correlated with the degree of spinal cord compression (TA) for those patients. Upgoing plantar reflexes, numbness of hands, Hoffman Sign, hyper-reflexia, and Lhermitte phenomenon seem to be the more important clinical signs of CSM as they directly correlated with the degree of cord compression. In particular,

the pyramidal-tract signs such as plantar response, Hoffmann reflex and hyper-reflexia correlated with TA and/or SI changes on T1-/T2WI.

➤ Key Points

- ❑ A prospective multicenter trial of patients with CSM attempting radiological correlation with clinical features and postoperative outcomes in a large sample was studied.
- ❑ TA of the spinal cord is a reliable MRI parameter to define clinical severity of the total number of neurological signs present in a patient correlated with the degree of spinal cord compression.
- ❑ Upgoing plantar reflexes, numbness of hands, Hoffman reflex, hyper-reflexia, and Lhermitte phenomenon seem to be the clinically significant signs of CSM.

References

1. Montgomery DM, Brower RS. Cervical spondylotic myelopathy. Clinical syndrome and natural history. *Orthop Clin North Am* 1992;23:487-93.
2. Baptiste DC, Fehlings MG. Pathophysiology of cervical myelopathy. *Spine J* 2006;6(suppl 6):190S-7S.
3. Kasai Y, Uchida A. New evaluation method using preoperative magnetic resonance imaging for cervical spondylotic myelopathy. *Arch Orthop Trauma Surg* 2001;121:508-10.
4. Morio Y, Teshima R, Nagashima H, et al. Correlation between operative outcomes of cervical compression myelopathy and MRI of the spinal cord. *Spine* 2001;26:1238-45.
5. Yukawa Y, Kato F, Yoshihara H, et al. MR T2 image classification in cervical compression myelopathy. *Spine* 2007;32:1675-8.
6. Avadhani A, Rajasekaran S, Shetty AP. Comparison of prognostic value of different MRI classifications of signal intensity change in cervical spondylotic myelopathy. *Spine* 2010;10:510-1.
7. Shen HX, Li L, Yang ZG, et al. Position of increased signal intensity in the spinal cord on MR images: does it predict the outcome of cervical spondylotic myelopathy? *Chin Med J* 2009;122:1418-22.
8. Shin JJ, Jin BH, Kim KS, et al. Intramedullary high-signal intensity and neurological status as prognostic factors in cervical spondylotic myelopathy. *Acta Neurochir (Wien)* 2010;10:1687-94.
9. Gregorius FK, Estrin T, Crandall PH. Cervical spondylotic radiculopathy and myelopathy. A long-term follow-up study. *Arch Neurol* 1976;33:618-25.
10. Lee TT, Manzano GR, Green BA. Modified open-door cervical expansive laminoplasty for spondylotic myelopathy: operative technique, outcome, and predictors for gait improvement. *J Neurosurg* 1997;86:64-8.
11. Sinha S, Jagetia A. Bilateral open-door expansive laminoplasty using unilateral posterior midline approach with preservation of posterior supporting elements for management of cervical myelopathy and radiculomyelopathy—analysis of clinical and radiological outcome and surgical technique. *Acta Neurochir (Wien)* 2011;153:975-84.
12. Alafifi T, Kern R, Fehlings M. Clinical and MRI predictors of outcome after surgical intervention for cervical spondylotic myelopathy. *J Neuroimaging* 2006;17:315-22.
13. Bertalanffy H, Eggert H. Clinical long-term results of anterior discectomy without fusion for treatment of cervical radiculopathy and myelopathy. A follow-up of 164 cases. *Acta Neurochir (Wien)* 1988;90:127-35.
14. Chiles BW III, Leonard MA, Choudhri HF, et al. Cervical spondylotic myelopathy: patterns of neurological deficit and recovery after anterior cervical decompression. *Neurosurgery* 1999;44:762-9.
15. Nurick S. The pathogenesis of the spinal cord disorder associated with cervical spondylosis. *Brain* 1972;95:87-100.
16. Singh A, Crockard HA, Platts A, et al. Clinical and radiological correlates of severity and surgery-related outcome in cervical spondylosis. *J Neurosurg* 2001;94(suppl 2):189-98.
17. Chen CJ, Lyu RK, Lee ST, et al. Intramedullary high-signal intensity on T2-weighted MR images in cervical spondylotic myelopathy: prediction of prognosis with type of intensity. *Radiology* 2001;221:789-94.
18. Mastronardi L, Elsawaf A, Roperto R, et al. Prognostic relevance of the postoperative evolution of intramedullary spinal cord changes in signal intensity on magnetic resonance imaging after anterior decompression for cervical spondylotic myelopathy. *J Neurosurg Spine* 2007;7:615-22.
19. Fernandez de Rota JJ, Meschian S, Fernandez de Rota A, et al. Cervical spondylotic myelopathy due to chronic compression: the role of signal intensity changes in magnetic resonance images. *J Neurosurg Spine* 2007;6:17-22.
20. Houten JK, Cooper PR. Laminectomy and posterior cervical plating for multilevel cervical spondylotic myelopathy and ossification of the posterior longitudinal ligament: effects on cervical alignment, spinal cord compression, and neurological outcome. *Neurosurgery* 2003;52:1081-7.
21. Vernon H, Mior S. The neck disability index: a study of reliability and validity. *J Manipulative Physiol Ther* 1991;14:409-15.
22. Arvin B, Kalsi-Ryan S, Karpova A, et al. Postoperative magnetic resonance imaging can predict neurological recovery after surgery for cervical spondylotic myelopathy: a prospective study with blinded assessments. *Neurosurgery* 2011;69:362-8.
23. Ware JE Jr, Sherbourne CD. The MOS 36-item Short-Form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992;30:473-83.
24. Nagata K, Kiyonaga K, Ohashi MS, et al. Clinical value of magnetic resonance imaging for cervical myelopathy. *Spine* 1990;15:1089-96.
25. Nagata K, Ohashi T, Abe J, et al. Cervical myelopathy in elderly patients: clinical results and MRI findings before and after decompression surgery. *Spinal Cord* 1996;34:220-6.
26. Uchida K, Nakajima H, Sato R, et al. Multivariate analysis of the neurological outcome of surgery for cervical compressive myelopathy. *J Orthop Sci* 2005;10:564-73.
27. Matsuyama Y, Kawakami N, Mimatsu K. Spinal cord expansion after decompression in cervical myelopathy. Investigation by computed tomography myelography and ultrasonography. *Spine* 1995;20:1657-63.
28. Chung S, Chung KH. Factors affecting the surgical results of expansive laminoplasty for cervical spondylotic myelopathy. *Int Orthop* 2002;26:334-8.
29. Benzel EC, Lancon J, Kesterson L, et al. Cervical laminectomy and dentate ligament section for cervical spondylotic myelopathy. *J Spinal Dis* 1991;4:286-95.
30. Hirabayashi S, Tsuzuki N, Abe R, et al. The value of a new method for assessing the separate functions of the long tracts and involved segments in patients with cervical myelopathy. *Int Orthop* 2000;24:75-9.
31. Singh A, Crockard H. Quantitative assessment of cervical spondylotic myelopathy by a simple walking test. *Lancet* 1999;354:370-3.
32. Fukushima T, Ikata T, Taoka Y, et al. Magnetic resonance imaging study on spinal cord plasticity in patients with cervical compression myelopathy. *Spine* 1991;16(suppl 10):S534-8.
33. Yagi M, Ninomiya K, Kihara M, et al. Long-term surgical outcome and risk factors in patients with cervical myelopathy and a change in signal intensity of intramedullary spinal cord on magnetic resonance imaging. *J Neurosurg Spine* 2010;12:59-65.
34. Yamazaki T, Yanaka K, Sato H, et al. Cervical spondylotic myelopathy: surgical results and factors affecting outcome with special reference to age differences. *Neurosurgery* 2003;52:122-6.
35. Yone K, Sakou T, Yanase M, et al. Preoperative and postoperative magnetic resonance image evaluations of the spinal cord in cervical myelopathy. *Spine* 1992;17(suppl 10):S388-92.

36. Matsuda Y, Shibata T, Oki S, et al. Outcomes of surgical treatment for cervical myelopathy in patients more than 75 years of age. *Spine* 1999;24:529–34.
37. Wada E, Ohmura M, Yonenobu K. Intramedullary changes of the spinal cord in cervical spondylotic myelopathy. *Spine* 1995;20:2226–32.
38. Lee J, Kalsi-Ryan S, Fehlings MG. Development and characterization of a novel rat model of cervical spondylotic myelopathy: the impact of chronic cord compression on clinical, neuroanatomical, and neurophysiological outcomes. *J Neurotrauma* 2012;29:1012–27.
39. Coronado R, Hudson B, Sheets C, et al. Correlation of magnetic resonance imaging findings and reported symptoms in patients with chronic cervical dysfunction. *J Man Manip Ther* 2009;17:148–53.
40. Okada E, Matsumoto M, Ichihara D, et al. Aging of the cervical spine in healthy volunteers: a 10-year longitudinal magnetic resonance imaging study. *Spine* 2009;34:706–12.
41. Cook C, Roman M, Stewart KM, et al. Reliability and diagnostic accuracy of clinical special tests for myelopathy in patients seen for cervical dysfunction. *J Orthop Sports Phys Ther* 2009;39:172–8.
42. Houten JK, Noce LA. Clinical correlations of cervical myelopathy and the Hoffmann sign. *J Neurosurg Spine* 2008;9:237–42.
43. Wong TM, Leung H, Wong WC. Correlation between magnetic resonance imaging and radiographic measurement of cervical spine in cervical myelopathic patients. *J Orthop Surg (Hong Kong)* 2004;12:239–42.
44. Cook CE, Hegedus E, Pietrobon R, et al. A pragmatic neurological screen for patients with suspected cord compressive myelopathy. *Phys Ther* 2007;87:1233–42.
45. McCormick WE, Steinmetz MP, Benzel EC. Cervical spondylotic myelopathy: Make the difficult diagnosis, then refer for surgery. *Cleve Clin J Med* 2003;70:899–904.
46. Emery S. Cervical spondylotic myelopathy: diagnosis and treatment. *J Am Acad Orthop Surg* 2001;9:376–88.
47. Wang MY, Green BA. Laminoplasty for the treatment of failed anterior cervical spine surgery. *Neurosurg Focus* 2003;15:E7.
48. Cleland J. *Orthopaedic Clinical Examination: An Evidence-Based Approach for Physical Therapists*. 1st ed. Bridgewater, NJ: Elsevier Health Sciences; 2005.
49. Magee DJ. *Orthopedic Physical Assessment*. 4th ed. Philadelphia, PA: Saunders; 2002.
50. Chikuda H, Seichi A, Takeshita K, et al. Correlation between pyramidal signs and the severity of cervical myelopathy. *Eur Spine J* 2010;19:1684–9.
51. Okada Y, Ikata T, Yamada H, et al. Magnetic resonance imaging study on the results of surgery for cervical compression myelopathy. *Spine* 1993;18:2024–9.
52. Mizuno J, Nakagawa H, Inoue T, et al. Clinicopathological study of “snake-eye appearance” in compressive myelopathy of the cervical spinal cord. *J Neurosurg Spine* 2003;2:162–8.
53. Arvin B, Kalsi-Ryan S, Mercier D, et al. Preoperative magnetic resonance imaging is associated with baseline neurological status and can predict postoperative recovery in patients with cervical spondylotic myelopathy. *Spine* 2013;38:1170–6.
54. Kadanka Z, Kerkovsky M, Bednarik J, et al. Cross-sectional transverse area and hyperintensities on magnetic resonance imaging in relation to the clinical picture in cervical spondylotic myelopathy. *Spine* 2007;32:2573–7.
55. Yanase M, Matsuyama Y, Hirose K, et al. Measurement of the cervical spinal cord volume on MRI. *J Spinal Disord Tech* 2006;19:125–9.