# Cortical anatomy plasticity in cases of cervical spondylotic myelopathy associated with decompression surgery
## A strobe-compliant study of structural magnetic resonance imaging

Meng Liu, MD[a], Yongming Tan, MD[b], Chenlei Zhang, MD[b], Laichang He, MD[b,*] ID

**Abstract**

Using voxel-based morphometry (VBM), we studied cortical gray matter volume changes in patients with cervical spondylotic myelopathy (CSM) before and after cervical cord surgical decompression. We then discussed the structural damage mechanisms and the neural plasticity mechanisms involved in postsurgical CSM.

Forty-five presurgical CSM patients, 41 of the same group followed-up 6 months after decompression surgery and 45 normal controls (NC) matched for age, sex and level of education underwent high-resolution 3-dimensional T1-weighted scans by 3.0 T MR. Then, VBM measurements were compared and cortical gray matter volume alterations were assessed among pre- or postsurgical CSM patients and NC, as well as correlations with clinical indexes by Pearson correlation.

Compared with NC, presurgical CSM patients showed reduced gray matter volume in the left caudate nucleus and the right thalamus. After 6 months, postsurgical CSM patients had lower gray matter volume in the bilateral cerebellar posterior lobes but had higher gray matter volume in the brain-stem than did presurgical CSM patients. Postsurgical CSM patients had significantly lower gray matter volume in the left caudate nucleus but greater regional gray matter volume in the right inferior temporal gyrus, the right middle orbitofrontal cortex (OFC) and the bilateral lingual gyrus / precuneus /posterior cingulate cortex than did NC. Abnormal areas gray volume in presurgical CSM and postsurgical CSM patients showed no significant correlation with clinical data ($P > .05$).

Myelopathy in the cervical cord may cause chronic cerebral structural damage before and after the decompression stage, markedly in outlier brain regions involving motor execution/control, vision processing and the default mode network and in areas associated with brain compensatory plasticity to reverse downstream spinal cord compression and respond to spinal cord surgical decompression.

**Abbreviations:** CSM = cervical spondylotic myelopathy, DMN = default mode network, JOA = Japanese Orthopedic Association, MRI = magnetic resonance imaging, NC = normal controls, NDI = Neck Disability Index, OFC = orbitofrontal cortex, PCC = posterior cingulate cortex, PCu = precuneus, SCI = spinal cord injury, VBM = voxel-based morphometry.

**Keywords:** brain plasticity, cervical spondylotic myelopathy, MRI, surgical decompression, voxel-based morphometry

## 1. Introduction

Cervical spondylotic myelopathy (CSM) is characterized by chronic incomplete spinal cord injury (SCI) associated with substantial motor and sensory loss below the injury site due to interruption of ascending and descending tracts. Because of the profound impact of CSM, numerous restorative therapies focused on improved behavioral recovery are being studied. Nevertheless, most current studies of CSM focus on either local spinal cord damage or promoting axon regeneration.[1,2]

Neuronal plasticity allows neurons in the brain and spinal cord to compensate for injury by adjusting their activities or structures in response to new situations. Rat model studies have demonstrated that apoptotic cell death occurs in cortical motor neurons due to SCI.[3] Presurgical or postsurgical CSM patients have been reported to have functional or metabolic abnormalities of the central nervous system, including altered intrinsic regional homogeneity in primary sensory cortex/primary motor cortexes and increased right superior parietal lobules,[4] and postsurgical brain functional adaptations or recruitments in addition to improvement of clinical functional recovery has been observed in CSM patients,[5,6] as well as metabolic alterations of the N-acetyl aspartate /creatine metabolite ratio in primary motor cortex in pre-[7,8] and postsurgical CSM patients.[8,9] However, there have not applied effective noninvasive methods to evaluate structural damage in cerebral cortex neurons in patients with CSM. Voxel-based morphometry (VBM) is an available tool in neuroscience used for statistical quantification of neuroanatomic structure alterations. Moreover, the central structural adaptation mechanisms underlying the recovery of motor and sensory functions after decompression surgery are poorly understood. Given that the cerebral cortex is involved in structural injury or plasticity, the clinical relevance of pre- or postsurgical CSM patients is not doomed to a one-to-one correspondence. Abnormal gray matter volume may be related to cortical and subcortical compensatory mechanisms after spinal cord injury in monkey.[10] Although the capability of extensive reorganization after decompression in CSM patients has been demonstrated in the adult human brain,[4,11] structural plasticity has not been evaluated longitudinally. VBM is a classical, fully automated quantitative magnetic resonance imaging (MRI) technique extensively employed to reveal in vivo neuropathological changes in brains, and this technique has been transformed by recent advances in neuroimaging and by increasingly sophisticated analytical tools. VBM reveals cortical atrophy in various neurologic conditions and has been widely used in neuropsychiatric and neurological disorders, serving as a basic tool for understanding cortical atrophy in CSM patients.

The goal of this study was to investigate cerebral structural alterations in CSM using VBM to determine whether cortex anatomical changes occur in CSM patients with chronic incomplete cervical injury before and after surgery. VBM was measured voxel-wise via volumetric analysis in whole brains, which tested unbiased measurement and comprehensive analysis of every voxel in the volumetric data instead of the segmented region of interest, and reflected gray matter volume. Gray matter volume was then compared across pre- or postsurgical CSM and NC. Then, these alterations were correlated with disease severity and disease duration to assess their clinical relevance. We hypothesize that presurgical CSM will be associated with significant decreases in regional gray matter volume and increased gray matter volume following decompression, and

we hoped to shed light on the possible underlying cerebral structural injuries and plasticity pathogenesis of pre- or postsurgical CSM.

## 2. Materials and methods

### 2.1. Subjects and clinical evaluation

With the approval of the medical ethics committee (the First Affiliated Hospital of Nanchang University ethics committee), 45 right-handed patients (28 females and 17males; mean age 51.24 ± 7years) presenting with clinical features of CSM were recruited, informed consent was obtained from these subjects. Exclusion criteria:

(1) patients with a history of brain surgery, traumatic brain or other neurological disorders.
(2) patients who could not undergo an MRI scan.

Forty-five normal controls (matched for handedness, education, age and sex) (28 females and 17males; mean age 50.47 ± 5years) were recruited from the Medical Examination Center, except for patients with a history of cardiovascular, neurological or psychiatric diseases. Conventional MRI scans of the head and cervical spine were obtained for all normal controls and CSM patients. All CSM patients were diagnosed with CSM by clinical evidence and cervical MRI. No physiotherapy sessions were scheduled for these patients after surgery. The disease duration of presurgical patients was66.5 ± 8 months (from6 month to 5 years). Almost all of the presurgical CSM patients underwent an MR scan again6 months after surgery; however,4 patients failed to undergo the scheduled scan. The Japanese Orthopedic Association (JOA) and Neck Disability Index (NDI) values were 12.45 ± 1.20 and 45.10% ± 12% in presurgical CSM patients, respectively, and 16.55 ± 2.52 and 64% ± 10% inpostsurgical CSM patients, respectively.

### 2.2. High-resolutionT1 data acquisition

All participants were scanned in a 3.0T MR scanner (Trio Tim, Siemens, Erlangen, Germany) with a 16 channels head coil. High-resolution, 3-dimensional, T1-weighted anatomy data parameters: including entire brain, MP-RAGE sequence, repetition time = 1900 ms, echo time = 2.26 ms, flip angle = 9°, field of view = 256 × 256 mm, resolution = 256 × 256 matrix, slices = 176, thickness = 1 mm, voxel size = $1.0 \times 1.0 \times 1.0$ mm$^3$ and interslice gap = 0.5 mm. One hundred and seventy-six sagittal slices images were acquired.

### 2.3. VBM data processing

We adopted the Data Preprocessing & Analysis for Brain Imaging,[12] running on MATLAB R2009a (Version 7.8.0Math-Works, Natick, MA), to preprocess original MR DICOM data. The general pipeline of VBM was as follows: First, the original MR 3D-T1 DICOM images were transformed into HDR/img format. For the nonuniformity correction, individual T1 images were spatially standardized to T1 template brain (Montreal Neurological Institute space), subsequently extracting gray matter, white matter, and cerebrospinal fluid. Second, the gray matter images were normalized using diffeomorphic anatomical registration through exponentiated lie algebra [13] and modulated with the linear transformation parameters as computed during

the normalization procedures. The mean image was calculated from the realigned images for each participant, and this image was used as a reference image for the subsequent spatial alignment. Third, the realigned images were corrected for field inhomogeneity in relation to the reference mean image. Fourth, tissue segmentation was performed in the bias-corrected mean reference image and the bias-corrected realigned images using the default Montreal Neurological Institute template. Fifth, diffeomorphic anatomical registration through exponentiated lie algebra spatial normalization parameters were estimated using tissue segments (gray and white matter) of the bias-corrected mean reference image. Sixth, normalization parameters were applied to tissue segments of the bias-corrected realigned images. Finally, the resulting normalized tissue segments of each participant were smoothed with an 8-mm Gaussian kernel.

### 2.4. Statistical analysis

We first reported within-group statistic map of gray matter volume for pre- or postsurgical CSM groups and NC group using one-sample t test ($P < .05$, FDR corrected). Between-group comparison was performed by using one-way analysis of covariance (ANCOVA) with age, sex, and years of education as covariates. A Bonferroni correction was further performed for pair-wise group comparisons in post-hoc analyses (AlphaSim correction, voxel $P$ value $< .01$, cluster $P$ value $< .001$). Significant differences in gray matter volume were overlaid onto an individual's T1-weighted anatomical image for visualization. The JOA scores, the NDI scores and other clinical measurements were statistically analyzed by SPSS 17.0 (SPSS Inc., Chicago, IL) ($P < .05$ denoted statistical significance). Pearson correlation coefficient was employed to examine the correlations between the gray matter volume in anomaly regions and the clinical measurements, including the JOA scores, the NDI scores and the disease duration.

## 3. Results

### 3.1. Group difference of gray matter volume

Fig. 1 shows the group-level gray matter volume difference between presurgical CSM and NC. Compared with NC, presurgical CSM exhibited a significantly lower gray matter volume (blue spots in Fig. 1) in the left caudate nucleus and the right thalamus.

Six months after spinal cord surgical decompression, the postsurgical CSM patients had lower gray matter volume in the bilateral cerebellar posterior lobe and higher gray matter volume in the brain-stem (Fig. 2) than did presurgical CSM patients. Compared with NC, postsurgical CSM patients had significantly less gray matter volume in the left caudate nucleus (Fig. 3). However, postsurgical CSM patients had significantly higher regional gray matter volume (red spots in Fig. 3) in the right inferior temporal gyrus, right middle orbitofrontal cortex (OFC) and bilateral lingual gyrus/precuneus/posterior cingulate cortex.

### 3.2. Correlations between VBM value and clinical variables

No brain regions demonstrated significant correlations with the clinical data (NDI scores, JOA scores, disease duration) in the presurgical (Table 1) or postsurgical CSM group ($P > .05$) (Table 2).
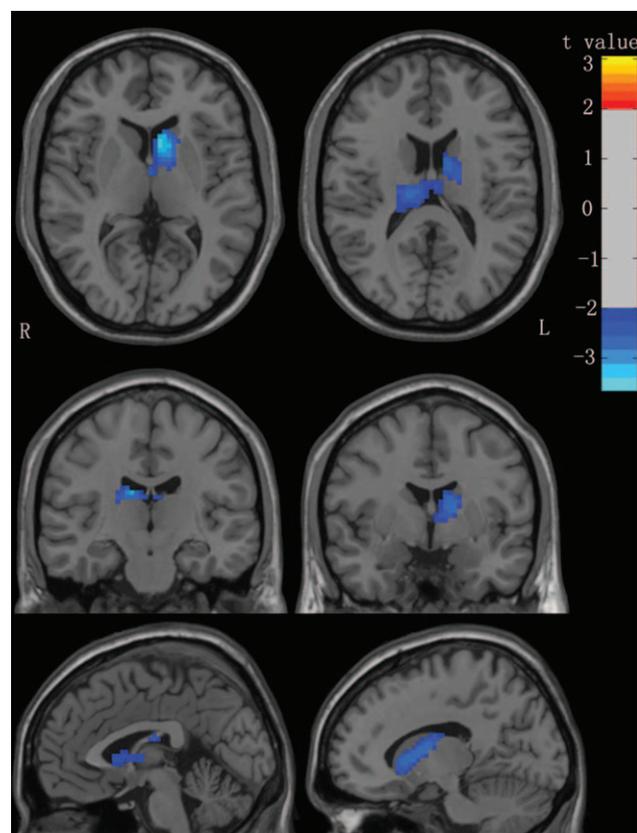


**Figure 1.** Gray matter volume alterations in presurgical CSM patients and NC ($P < 0.01$, AlphaSim correction). The yellow and blue colors denote increased and decreased gray matter volume, respectively. The color bars indicate the t-values.

## 4. Discussion

CSM is the most common cause of incomplete reversible spinal cord dysfunction in middle-aged and elderly people. The pathophysiologic consequences of CSM have been studied extensively within the cord[1,14] and the periphery; however, these consequences remain relatively unexplored in the brain. Although multilevel changes are shown both below and above the site of injury, the change in the supraspinal level of the injury is considered an important factor for predicting the degree of recovery. Therefore, we used advanced MRI technologies to understand how brain structure in CSM patients changes before and after cervical cord surgical decompression. The current study demonstrated that presurgical CSM patients showed significantly more gray matter atrophy in the left caudate nucleus and the right thalamus than did NC. After 6 months, postsurgical CSM had significant gray atrophy in the bilateral cerebellar posterior lobe but more gray matter volume in the brain-stem than presurgical CSM patients; Compared with NC, postsurgical CSM patients had significantly less gray matter volume in the left caudate nucleus. However, postsurgical CSM patients had significantly higher regional gray matter volume in the right inferior temporal gyrus, right middle OFC and bilateral lingual gyrus/precuneus /posterior cingulate cortex.

VBM alterations may represent cerebral neuron structural damage or plasticity that can strongly affect cerebral cortex
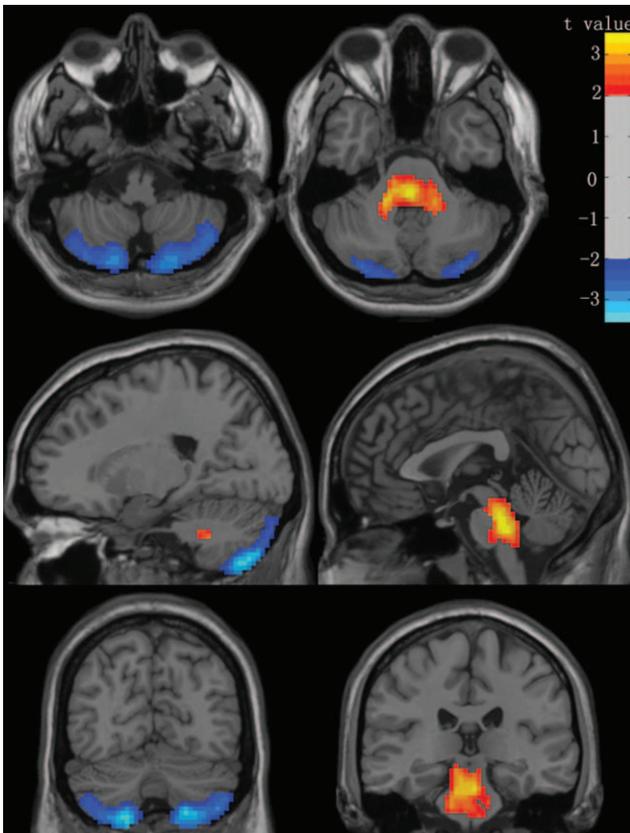
**Figure 2.** Postsurgical versus presurgical CSM patient gray matter volume differences (paired 2-tailed student's t test, $P < 0.01$, AlphaSim correction).

neuronal excitability involved in information processing. Any gray or white matter reduction in the cerebral cortex might be responsible for deficits in sensory, motor and executive functions, as revealed in parkinsonism,[15] Huntington disease[16] and amyotrophic lateralizing sclerosis.[14] We showed that compared with NC, pre- or postsurgical CSM patients had diffusely abnormal gray matter volume. These results suggested that neuronal structures were notably altered at the brain level in CSM patients. Our findings may provide evidence to suggest that CSM can affect neuronal structure of the human brain in several regions.

Gray matter volume significantly decreased in the left caudate nucleus and the right thalamus in presurgical CSM patients. These regions accord with previous fMRI study results in monkeys and mice with incomplete SCI.[6,17] It is well known that the caudate nucleus makes an important contribution to maintain the posture of body and limbs, controlling the speed and accuracy of directed motions,[18] whereas the thalamus is a relay station associated with the transmission of neural information with the sensory/motor cortex and cognitive signal decisions by the prefrontal/parietal/temporal cortex onto the thalamus.[19] As an important part of cortex-thalamus-striatum reflex, thalamic round-trip fibers contacting the cortex and the extrapyramidal system play an important role in the process of sensory/motor control. The decreased gray matter volume in the caudate nucleus and the thalamus areas are possibly induced by a deficit of sensory input from damaged ipsilateral spinothalamic tracts. Our

results also demonstrated significantly lower gray matter volume in the left caudate nucleus in postsurgical CSM patients than in NC. Postsurgical CSM patients had sensory deficits, possibly explained by caudate nucleus atrophy. The observed caudate nucleus atrophy might reflect the motor execution deficits, including speed and accuracy of directed motions that may be irreversible cortical structure injuries in these patients. However, studies have demonstrated that postinjury brain reorganization may follow a dynamic time course.[20–22] Thus, we conducted a longitudinal study of the more chronic stages of post-decompression CSM.

Through a rich afferent and efferent fiber feedback system, the cerebellar posterior lobe communicates with the brain, the brainstem and the spinal cord, participating in body balance and muscle tension regulation, as well as in control of voluntary movement. Clinical studies suggested that cerebellar lobes V and VI are principally engaged in motor control and somatosensory function.[23] An explanation for the observed increased gray matter volume in the brain stem may be increased or reconnected efferent motor and afferent sensory pathways in the brain stem, showing that new movement patterns occur after the initial motor pattern is destroyed. The lower gray matter volume in the bilateral cerebellar posterior lobe of postsurgical CSM patients suggests that the "cortex–pons–cerebellum" system compensates for sensorimotor loss, possibly with disinhibition of cortical and subcortical structural plasticity.

The gray matter volume of postsurgical CSM patients was significantly increased in widespread brain regions, including in the right inferior temporal gyrus, the right middle OFC and the bilateral lingual gyrus/precuneus (PCu)/posterior cingulate cortex (PCC). These brain regions can be divided into two categories: the region involved with vision association information processing or temporal-occipital association cortex of the vision processing network (inferior temporal gyrus, lingual gyrus) and the component of the "default mode network" (DMN) (PCC, PCu, and OFC). A recent study showed that SCI can change the regional synchronism of brain activity in the sensorimotor system and the default mode network,[24] confirming the abnormal structural state of the "default mode network." PCC/ PCu is considered to be the core node of DMN that receives signals from the frontal, parietal[25] and temporal lobes,[26] playing a pivotal role in monitoring the surrounding environment[24] and maintaining self-consciousness and cognitive-affective interaction.[27] The inferior temporal cortex receives information from the ventral stream, as it is known to be the essential ventral stream region of visual processing, including recognizing patterns, faces, and objects.[28] Ventral occipitotemporal cortex, including lingual gyrus, is related to visual information processing[29] and working memory.[30,31] Choe et al[32] reported that SCI enhanced the functional connection between the sensorimotor cortex and the visual cortex. Nonhuman primate scan change the regional synchronism of brain activity in the sensorimotor system and the default mode network after spinal cord injury.[24,33] Noor et al[34] demonstrated SCI-induced changes of the default network (DMN) and the fronto-lateral network in SCI patients with varying degrees of sensory deficiency. Our findings, combined with those of previous studies, may reflect postsurgical CSM patients modulating increased brain gray matter volume of the DMN to integrate the surrounding environment and to control information caused by neural deafferentation within the sensorimotor cortex. This potentially attempts to orchestrate dysfunctional
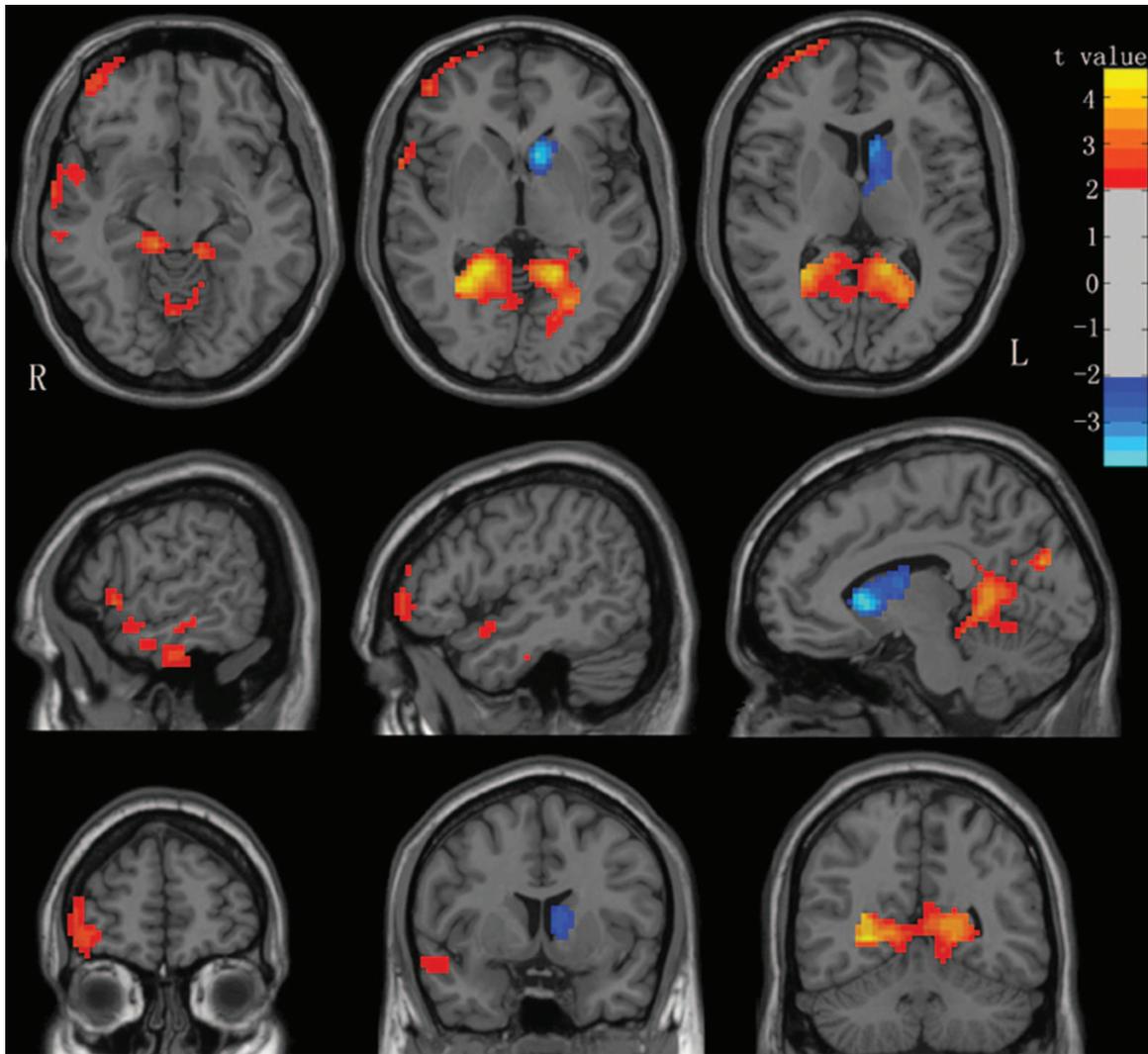
**Figure 3.** Postsurgical CSM versus NC gray matter volume differences ($P < .01$, AlphaSim correction).

environment information efferent responses and generation of appropriate afferent feedback. This result may show the modulation of the DMN and vision association cortex to rebuild coordination after decompression, guided by lack of sensory input and motor output.

Unexpectedly, there was no clear relationship between the altered degree of brain gray matter volume and the clinical characteristics (JOA scores, NDI scores, disease duration). Possibly, following incomplete injury of upstream and downstream spinal fibers, spinal neuron modify the synaptic connections of new neural circuits together with brain plasticity

to jointly maintain compensatory function, especially in the early and middle period dysfunction after decompression.

There were some shortcomings in this study. First, NC subjects did not provide follow-up scans again, which may confused be the results of typical age-related changes, not derived from surgical outcomes. Additionally, varying degrees of compression give rise to varying degrees of injury in the spinal cord and may have direct correlation with cortical injury and plasticity compensation. Diffusion tensor imaging (DTI) and high-resolution MRI were used to detect the microstructural injury of cervical spinal cord and to elucidate its correlation with brain plasticity in the future. Last but not the least, the sample of follow-up time was short. Therefore, we are continuing to collect CSM patients and prolong the follow-up time. The last, because of the decompress surgery, cerebral blood flow or slight brain shift may slight brain shift might lead to comparisons between gray matter and white matter on preoperative or postoperative VBM,[35] so these factors should be considered.

In conclusion, our findings showed cerebral cortex structural deficits and reorganization attributes in CSM patients. VBM data analysis showed that CSM patients with chronic compression

### Table 1

**Correlations between gray matter volume in anomaly regions and clinical status indexes in presurgical CSM patients.**

|  | Correlation coefficient (*P* value) | | |
| --- | --- | --- | --- |
|  | JOA scores | NDI scores | Disease duration |
| Left caudate nucleus | .075 (.664) | .077 (.626) | .165 (.560) |
| Right thalamus | .085 (.729) | .079 (.605) | .244 (.286) |

**Table 2**

Correlations between gray matter volume of anomaly regions and clinical status indexes in postsurgical CSM patients.

| | Correlation coefficient (*P* value) | | |
| --- | --- | --- | --- |
| | JOA scores | NDI scores | Disease duration |
| Cerebellar posterior lobe | −.268 (.267) | .626 (.147) | −.687 (.290) |
| Brain-stem | .060 (.715) | .156 (.561) | .125 (.664) |
| Left caudate nucleus | −.069 (.875) | −.272 (.260) | −.069 (.875) |
| Right inferior temporal gyrus | .046 (.860) | .166 (.496) | .065 (.911) |
| Right middle OFC | −.211 (.687) | .086 (.727) | −.624 (.425) |
| Lingualgyrus/precuneus/posterior cingulate cortex | .014 (.955) | .109 (.656) | .095 (.827) |

spinal cord injury may have cerebral structural damage undergoing dynamic neural plasticity before and after the decompression stage, markedly in outlier brain regions involving motor execution/control, vision processing and the default mode network, all of which are associated with brain compensatory plasticity to reverse downstream spinal cord compression and responses to spinal cord surgical decompression.

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## Author contributions

**Conceptualization:** Laichang He.
**Data curation:** Meng Liu, Yongming Tan.
**Formal analysis:** Yongming Tan.
**Funding acquisition:** Laichang He.
**Investigation:** Meng Liu.
**Project administration:** Laichang He.
**Resources:** Yongming Tan.
**Software:** Yongming Tan, Chenlei Zhang.
**Supervision:** Laichang He.
**Visualization:** Chenlei Zhang.
**Writing – original draft:** Meng Liu.
**Writing – review & editing:** Laichang He.

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