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ORIGINAL RESEARCH

Altered Brain Structure in Hemifacial Spasm Patients: A Multimodal Brain Structure Study

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Objective: Hemifacial spasm (HFS) is a clinical neurosurgical disease, which brain structural alterations caused by HFS remain a topic of debate. We evaluated changes in brain microstructure associated with HFS and observed their relevance to clinical characteristics.

Methods: We enrolled 72 participants. T1-weighted structural and diffusion tensor images were collected from all participants using 3.0T magnetic resonance equipment. Voxel-based morphometry (VBM) and tract-based spatial statistics (TBSS) were used to identify changes in gray matter volume (GMV) and disruptions in white matter (WM) integrity. The severity of the spasms was graded using the Cohn scale.

Results: VBM analysis revealed that the GMV was significantly reduced in the left Thalamus and increased GMV in the right Cerebellum IV-V of the HFS group. TBSS analysis showed that FA in the left superior longitudinal fasciculus (SLF) of the HFS group was significantly increased. GMV in the thalamus showed a negative correlation with disease duration and Cohn grade, while FA in the left SLF had a positive correlation with both the disease duration and Cohn grade.

Conclusion: We identified regions with altered GMV in HFS patients. Additionally, we determined that FA in the left SLF might serve as a significant neural indicator of HFS.

Keywords: hemifacial spasm, voxel-based morphometry, diffusion tensor imaging, tract-based spatial statistics, Cohn grading

Introduction

Hemifacial spasm (HFS) is a common clinical cerebral nerve disease, manifested as unilateral or bilateral involuntary, recurrent, and paroxysmal contractions.[1] The primary pathogenesis of HFS is believed to be due to the compression of the nerve by opposing blood vessels, leading to retrograde transmission in the facial nerve and increased excitability of the facial nerve nucleus.[2–4] However, the comprehensive structural changes in the brain due to HFS are still debated.

Voxel-based morphometry (VBM) is a method that detects the gray matter volume (GMV) of the entire brain without pre-identifying regions of interest. It is commonly used to identify neurological and psychiatric alterations in GMV.[5,6] Previously studies reported decreased GMV in the right parietal lobe and increased GMV in the right cerebellum VIII in HFS patients using VBM.[7] Another study detected reduced GMV in the thalamus, putamen, globus pallidus, dorsolateral prefrontal cortex, amygdala, and parahippocampal gyrus in HFS patients.[8] The GMV changes identified in these studies vary, and it remains unclear if these GMV changes are secondary pathological adaptations or part of the HFS pathogenesis.

Diffusion tensor imaging (DTI) can analyze the diffusion characteristics of water molecules in a three-dimensional space in vivo, representing the anatomical structure and degenerative degree of nerve fibers through the movement of water molecules between cells.[9,10] Tract-based spatial statistics (TBSS) analysis has been employed to evaluate fractional anisotropy (FA), radial diffusivity (RD), axial diffusivity (AD), and mean diffusivity (MD) for white matter (WM) fiber bundle integrity.[11] Through TBSS, previous studies have reported abnormal changes in the WM fibers of the bilateral superior longitudinal fasciculus (SLF) in patients with left HFS, with increased FA in this region.[12] In contrast, another study showed that HFS patients exhibited decreased FA in WM tracts of the right inferior longitudinal fasciculus (ILF)

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and inferior frontal-occipital fasciculus (IFOF).[13] Investigations into the abnormal alterations of WM fiber tracts in HFS patients have not only identified various affected regions but have also shown discrepancies in FA value changes.

Recent studies have not extensively explored the brain structure of HFS patients. In this study, we gathered data from patients with right HFS to corroborate earlier findings. Our objective was to further identify alterations associated with HFS in brain microstructure using VBM and TBSS, and to examine their relationships with disease duration and spasm severity.

Methods

Participants

36 right-sided HFS patients, confirmed by the department of neurosurgery and scheduled for undergo neurovascular decompression, were selected as the HFS group. 36 healthy volunteers, matched in age and gender, were recruited as the control group. The diagnosis of HFS was made by two experienced neurologists based on clinical symptoms and cranial nerve MRI, and was confirmed by microvascular decompression. None of the participants had other neurological conditions. Secondary HFS, such as that caused by tumor compression or multiple sclerosis, was excluded through relevant examinations and past medical history. The HFS severity was evaluated using the Cohen grading scale (1: slight spasm, 2: mild spasm without functional impairment, 3: moderate spasm with mild functional impairment, 4: severe functional impairment and spasm).[14,15]

The study adhered to the "Declaration of Helsinki". All tests were conducted with the informed consent of the participants. The research received approval from our hospital's ethics committee.

MRI Data Acquisition

MRI data were obtained using a 3.0-T MR scanner (Verio system; Siemens, Erlangen, Germany) with a 12-channel head coil. 3D high-resolution T1-weighted structural data were acquired using a magnetization prepared rapid acquisition gradient echo (MP-RAGE) sequence. The sequence parameters included: repetition time = 1900 ms, echo time = 2.52 ms, field of view = 256 mm×256 mm, voxel size = 1 mm×1 mm×1 mm, flip angle = 9°, 192 sagittal slices, slice thickness/gap = 1.0/0 mm. DTI data were obtained using a single-shot echo-planar imaging (SE-EPI) sequence. The sequence parameters included: repetition time = 8000 ms, echo time = 95 ms, field of view = 256 mm×256 mm, voxel size = 2 mm×2 mm×3 mm, 48 axial slices, slice thickness/gap = 3.0/0 mm, 64 non-linear diffusion directions (b = 1000 s/mm^2) along with an acquisition without diffusion-weighted image (b = 0 s/mm^2).

VBM Analysis

VBM analysis was performed using the Computational Anatomy Toolbox 12 (CAT 12, http://dbm.neuro.uni-jena.de/wordpress/) in MATLAB (version 2013b, https://www.mathworks.com/products/matlab.html). Preprocessing steps included: (1) T1-weighted structural images were converted to 3D NIFTI format with the Dcm2nii tool. (2) Non-brain tissue from the converted data was removed using the brain extracting tool (BET). (3) The extracted data were segmented into GM, WM, and cerebrospinal fluid (CSF). (4) Total intracranial volume (TIV), GM, WM, and CSF volumes were calculated from the segmented images.

VBM steps included: (1) The GM data were normalized using a high-dimensional DARTEL protocol in the Montreal Neurological Institute (MNI) space.[16,17] (2) The normalized GM data were modulated non-linearly using Jacobian determinants from volumetric changes associated with individual brain volumes. (3) The modulated GM data were smoothed with a 10-mm full width at half-maximum (FWHM) Gaussian kernel. (4) A two-sample *t*-test was conducted to identify brain regions with significant GMV differences between the two data groups, considering age, sex, and TIV as covariates. (5) The statistical maps were corrected for multiple comparisons (FWE, $p < 0.05$) at the voxel level. (6) The location of GMV changes was determined using Xjview on the Hopkins brain template (ICBM-DTI-81 White-Matter Labels)

TBSS Analysis

DTI preprocessing and analysis were executed using the FDT toolkit of the Oxford University image processing software FSL v.5.0.11 (http://www.filion.ucl.ac.uk/spm/software/spm8/). Preprocessing methods were: (1) DTI data was converted to 4D NIFTI format using the Dcm2nii tool. (2) The eddy current and head motion effects were corrected with the Eddy current tool. (3) Non-brain tissue like the scalp and skull were removed by BET. (4) The diffusion parameters (FA, RD, AD, and MD) for each voxel in the brain for all subjects were calculated using the DTIFIT tool.[18,19]

TBSS steps included: (1) FA images were aligned with the FMRIB58-FA template in MNI space utilizing a non-linear registration calculation and average FA skeleton images (threshold = 0.2) were established. (2) Normalized FA images were projected onto the mean FA skeleton images. (3) RD, AD, and MD were extracted from the identified differentiated brain regions for each subject to gather various DTI index data. (4) A two-sample *t*-test was set up with the Glm tool, with age and gender as covariates, and run using the Randomize tool to investigate the whole-brain non-parametric statistical threshold values (permutations = 5000; threshold value = 0.2). (5) Results were corrected by threshold-free cluster enhancement (TFCE, $P < 0.05$) and multiple comparison analysis (FWE, $p < 0.05$). (6) The locations of WM fiber tracts alterations were detected by the Atlas query tool on the Hopkins brain template (ICBM-DTI-81 White-Matter Labels).[11]

Statistical Analysis

The gender distribution between the HFS and HC groups was assessed using the Chi-square test with SPSS 25.0 software, and an independent-sample *t*-test was conducted to compare the age distribution between the two groups. Clusters of interest with differences in GMV, FA, RD, AD, and MD between the groups were identified and extracted for the HFS group using the cluster toolbox. A partial correlation analysis was conducted to determine the relationship between the mean values of each DTI parameter and clinical features (disease duration, spasm severity), accounting for age and gender as covariates.

Results

Demographic and Clinical Features

The demographic and clinical features of all subjects were summarized in Table 1. No significant differences were found between the two groups in age and gender. The average HFS duration was 7.44 ± 4.21 years (range: 1–20 years), and Cohen grading was 2.78 ± 0.93 points (range: 0–4 points) for HFS patients.

Gray Matter Abnormalities

GM alterations in patients with right HFS are depicted in Figure 1. Compared to the HC group, the HFS group showed significantly decreased GMV in the left thalamus and increased GMV in the right cerebellum IV-V($P < 0.05$, FWE corrected). No significant differences were found between the two groups for whole brain volumes of TIV, GM, WM, and CSF (Table 2).

Table 1 Demographic and Clinical Data of HFS Patients and Healthy Controls

Characteristics	HFS group	HC group	*P*-value
Number	n=36	n=36	
Age (mean ±SD)	52.69±8.68	54.14±8.80	0.49*
Gender (males/females)	6/30	6/30	1**
Duration (mean ±SD)	7.44±4.21	NA	NA
Cohen grading (mean ±SD)	2.78±0.93	NA	NA

Notes: *P-value from *t*-test. **P-value from Chi-square test.
Abbreviations: HFS, hemifacial spasm; HC, healthy controls; NA, not available; SD, standard deviation.

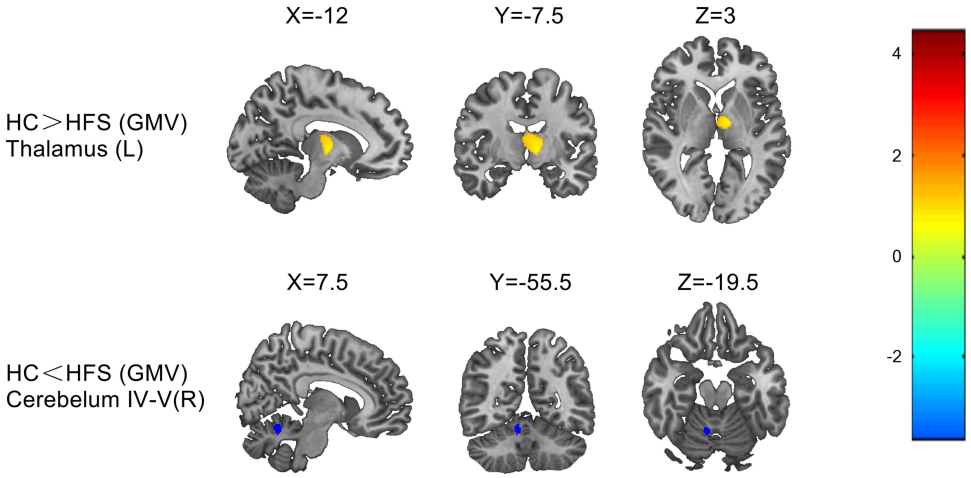

Figure 1 Voxel-based morphometry. VBM analysis demonstrated decreased GMV in the left thalamus and increased GMV in the right cerebellum IV-V ($P < 0.05$, FWE corrected) in the HFS group. Hot and cold colors indicate GMV increases and decreases, respectively.
Abbreviations: FEW, family-wise error; GMV, gray matter volume; HC, healthy control; HFS, hemifacial spasm.

White Matter Abnormalities

WM alterations in patients with right HFS are illustrated in Figure 2. Compared ro the HC group, the HFS group had significantly increased FA in the left SLF ($P < 0.05$, FWE corrected). No significant differences in RD, MD, and AD between the two groups.

Brain-Behavior Association

Partial correlation analysis showed that GMV was negatively correlated with HFS duration ($r = -0.384$, $P = 0.025$) and spasm severity ($r = -0.445$, $P = 0.008$). FA in the left SLF was positively associated with HFS duration ($r = 0.435$, $P = 0.010$) and spasm severity ($r = 0.527$, $P = 0.001$). However, GMV in the right Cerebellum IV-V was not significantly correlated with clinical variables (Figure 3 and Table 3).

Discussion

VBM offers insights into detailed anatomical changes in the brain and relates these findings to neurological symptoms. The results present objective, unbiased information about brain structure. Lately, voxel-based brain structure studies have gained prominence in cranial nerve disorders, including trigeminal neuralgia and HFS.[7,12,13,20,21]

In previous studies, the etiology of HFS was attributed to chronic vascular compression at the root entry zoom (REZ) of the facial nerve.[22,23] In our study, we considered GMV alterations as a secondary outcome of the central nervous system. Bao et al[8] identified GM changes in HFS patients, noting significant decreases in GMV in areas such as the thalamus, putamen, globus pallidus, dorsolateral prefrontal cortex, amygdala, and parahippocampal gyrus. By adjusting for HAMA and HAMD scores, they inferred that GMV changes in the amygdala might be tied to emotional factors.

Table 2 Global Tissue Volumes of Patients with HFS and Healthy Controls

Global brain volumes	HFS group		HC group		*P*-value
	Mean	SD	Mean	SD	
Grey matter volume	649.8	63.4	636.3	56.5	0.34
White matter volume	494.1	56.2	490.2	50.6	0.76
Cerebrospinal fluid volume	310.8	31.4	306.9	45.0	0.67
Total intracranial volume	1454.6	127.5	1433.3	118.9	0.47

Note: *P*-value from *t*-test.
Abbreviations: HFS, hemifacial spasm; HC, healthy controls; SD, standard deviation.

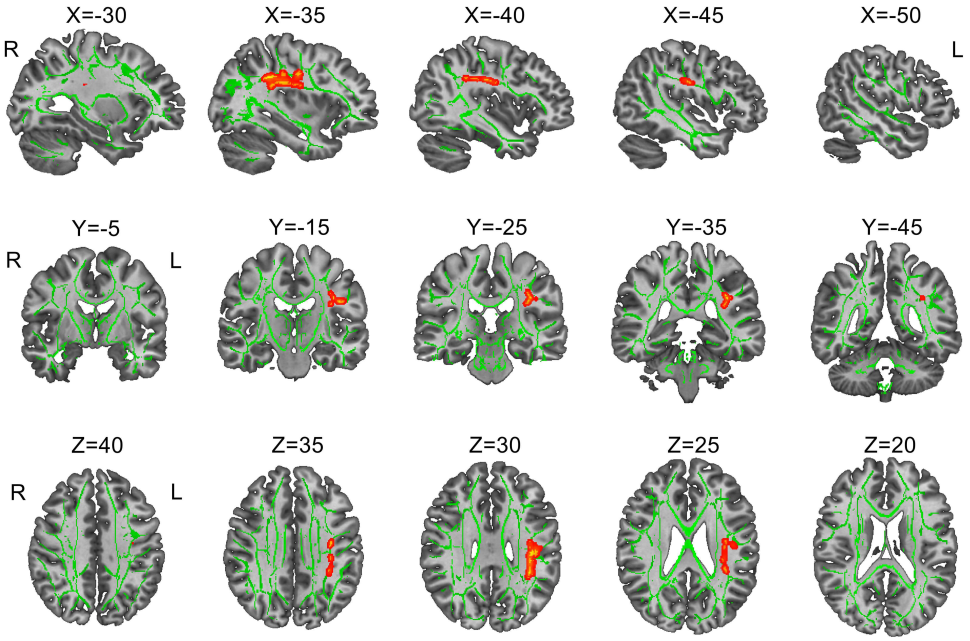

Figure 2 Tract-based spatial statistics. TBSS analysis demonstrated increased FA in the left superior longitudinal fasciculus ($P < 0.05$, FWE corrected) in the HFS group. The hot color indicated increased FA, and the green represents the mean FA skeleton of all subjects.
Abbreviations: FA, fractional anisotropy; FWE, family-wise error; HFS, hemifacial spasm.

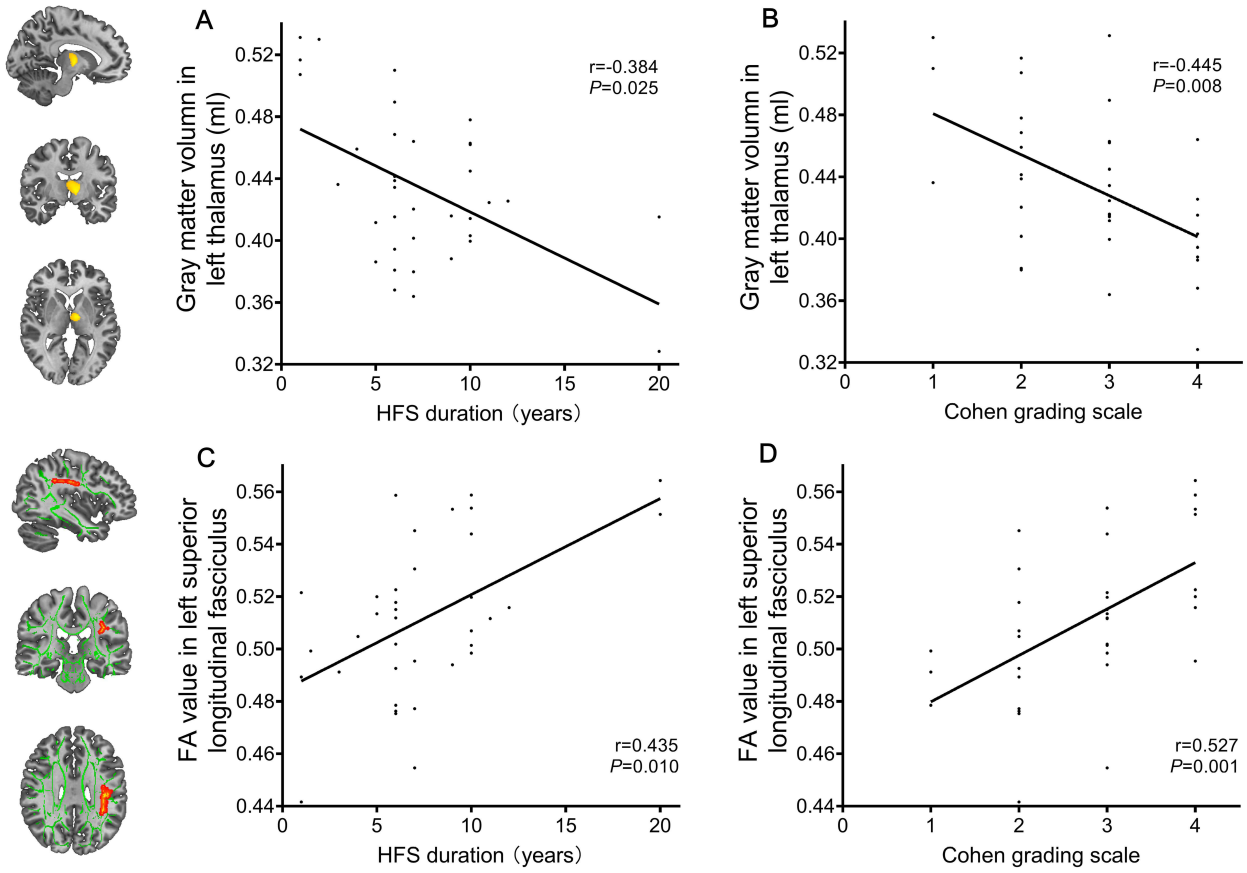

Figure 3 Partial correlation analysis. Partial correlation analysis demonstrated correlations between clinical variables and structure parameters. (**A**) The GMV in left thalamus is negatively correlated with HFS duration ($r = -0.384$, $P = 0.025$) in the HFS group. (**B**) The GMV in the left thalamus is negatively correlated with Cohen grading ($r = -0.445$, $P = 0.008$) in the HFS group. (**C**) The FA value in left superior longitudinal fasciculus is positively correlated with HFS duration ($r = 0.435$, $P = 0.010$) in the HFS group. (**D**) The FA value in left superior longitudinal fasciculus is positively correlated with Cohen grading ($r = 0.527$, $P = 0.001$) in the HFS group.
Abbreviations: FA, fractional anisotropy; HFS, hemifacial spasm.

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Table 3 Correlations Analysis Between Structure Parameters and Clinical Features

Clusters	Voxels	MNI Coordinate			Duration		Cohn	
		X	Y	Z	r	P	r	P
HC>HFS (GMV) Cerebellum IV-V(R)	53	7.5	−55.5	−19.5	0.148	0.405	0.017	0.924
HC<HFS (GMV) Thalamus (L)	527	−12	−7.5	3	−0.384	0.025	−0.445	0.008
HC<HFS (FA) SLF (L)	508	−36	−37	26	0.435	0.010	0.527	0.001

Note: *P*-value and r value from partial correlation.
Abbreviations: HFS, hemifacial spasm; HC, healthy controls; SLF, superior longitudinal fasciculus; MNI, Montreal Neurological Institute.

Notably, their findings align with our study, which also observed reduced GMV in the thalamus of HFS patients. Earlier neuroimaging studies linked the basal ganglia-thalamus-cortical motor circuit with the pathophysiology of blepharospasm.[24] Meanwhile, the somatosensory input induced by skin spasms in HFS patients may activate the somatosensory afferent pathway and the basal ganglia - thalamus - cortical motor circuit.[25] Thus, we can speculate that the pathophysiology of HFS is also related to the basal ganglia-thalamus-cortical motor circuit. This may help explain the reduced GMV in HFS patients observed in this study.

Tu et al[7] assessed the decreased GMV in the right parietal lobe of HFS patients using VBM and found that the GMV in this brain area was negatively correlated with disease duration. Meanwhile, the GMV in the right cerebellum VIII was significantly increased. They speculate that these changes may be related to abnormalities in brain regions involved in motor control for HFS patients. Our study found that the GMV reduction in HFS patients was located in the right cerebellum IV-V, also known as the parietal cerebellum, which is part of the sensorimotor cerebellum.[26] This suggests a compensatory neuroadaptation of the cerebellum to motor control in HFS patients. Therefore, our findings on GM in HFS patients are well-aligned with previous studies, confirming the quality of our sample and further pinpointing the areas of GM change in HFS patients.

DTI can effectively display the diffusion characteristics of water molecules in living tissue and can enhance the localization of information to WM lesions. It can also directly reflect damage to WM fiber bundles.[27,28] WM microstructures are involved in regulating neuronal activity and connecting brain regions to behaviors.[29,30] TBSS has been widely used in the study of the pathogenesis of various neuropsychiatric diseases and is noted for its advantages in compensating for the limitations of artificially drawn regions of interest (ROI) with low repeatability and subjective perceptions.[31,32] Since most HFS patients have abnormal blood vessels compressing the facial nerve at the REZ, this may result in a series of pathophysiological changes such as myelin demyelination, myelin formation disorder, and excessive collagen deposition, leading to abnormal water diffusion in nerve fibers.[33,34] Therefore, we need to analyze the WM indices in HFS patients to understand these changes. FA reflects the integrity of white matter fiber demyelination and axon cell membrane, RD is a sensitive indicator for evaluating demyelination or myelination disorders, AD reflects the severity of axon damage or degeneration, and MD reflects the severity of cell degeneration.[35]

Our previous study, based on TBSS, found that left HFS patients had increased FA and decreased RD in the bilateral SLF and FA in the left SLF was positively associated with HFS duration and spasm severity.[12] Interestingly, in our study of patients with right-sided HFS, FA was found to increase only in the left SLF. However, FA values in this region were also positively correlated with HFS duration and spasm severity. The identification of the same WM-altered regions in patients on different sides prompted us to reconsider the previous statistical experiment in which the images of patients on different sides were reversed along the central axis. We compared TBSS studies with conflicting results in patients with HFS. Tu et al[7] found no significant changes in DTI index regarding WM microstructure of HFS patients. Notably, in their study, both left and right HFS cases were included, and the images of the right cases were reversed along the central axis. This might explain why the experiment failed to identify WM changes. Guo et al[13] found that the integrity of WM was extensively compromised, especially regarding RD changes in the right ILF and IFOF. In their study, the duration of

HFS in patients was shorter, and there were significant differences in HAMA and HAMD scores between the HFS and HC groups. This indicates that the results of this study were less influenced by HFS and more by depression.

The SLF is a tract in the white matter that connects the parietal, frontal, temporal, and ipsilateral frontal cortices, facilitating intralobular and interlobular transmission of sensory information. The SLF is relatively long, and its membrane quality is high, potentially making the bundle fiber more sensitive to injury.[36] Previously, we proposed three hypotheses for the increase of FA in the superior longitudinal bundles. First, it might be related to sleep quality defects in HFS patients.[37] Secondly, the specific correlation of visual-spatial construction disorder in HFS patients was analyzed.[38] Third, WM remodeling due to involuntary facial muscle contraction might result in "enhanced or excessive performance".[39] We now lean towards the third hypothesis, given past studies that found increased FA in the SLF correlates with behavioral changes due to alterations in white matter connectivity in the SLF caused by myelination.[40] We hypothesize that this highly active area triggers the symptoms of HFS and believe that increased FA in the left SLF may offer significant implications for assessing HFS severity and duration.

However, our study has limitations. Firstly, it does not consider the effects of drugs on GM and WM in the brain. Second, this study is a single-center cross-sectional study with a limited sample size, potentially influencing the results of the statistical analysis. Third, further cognitive and affective studies are required to clarify the relationship between WM changes and HFS. Fourth, to maintain sample consistency, this study only analyzed right-sided HFS patients, leaving the inclusion of bilateral cases in the brain structure study an open question.

Conclusion

In conclusion, we identified altered GMV regions in HFS patients, characterized by reduced volume in the thalamus and compensatory volume increase in the cerebellum. Additionally, we verified that the FA in the left SLF might serve as a crucial neural marker for HFS, offering valuable insights for diagnosis and prognosis. Future studies will investigate changes in brain structure in HFS patients post-microvascular decompression.

Data Sharing Statement

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

The Changzheng Hospital's institutional review board (Shanghai, China) granted ethical permission for this study before the start of data collection. All tests were conducted with the informed consent of the participants themselves.

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Disclosure

The authors declare that they have no competing interests in this work.

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