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Role of Cortico-ponto-cerebellar Tract from Supplementary Motor Area in Ataxic Hemiparesis of Supratentorial Stroke Patients

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HIGHLIGHT

- Subcortical lesions of supplementary motor area (SMA) have a role in post-stroke ataxia.
- Diffusion tensor imaging of cortico-ponto-cerebellar (CPC) tract could reflect ataxia severity.
- Subcortical lesions of the CPC tract from the SMA are important in ataxic hemiparesis.

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Role of Cortico-ponto-cerebellar Tract from Supplementary Motor Area in Ataxic Hemiparesis of Supratentorial Stroke Patients

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ABSTRACT

Cortical lesions of the supplementary motor area (SMA) are important in balance control and postural recovery in stroke patients, while the role of subcortical lesions of the SMA has not been studied. This study aimed to investigate the subcortical projections of the SMA and its relationship with ataxia in supratentorial stroke patients. Thirty-three patients with hemiparesis were divided into 3 groups (severe ataxia, n = 9; mild to moderate ataxia, n = 13; no ataxia, n = 11). Ataxia severity was assessed using the Scale for Ataxia Rating Assessment. Diffusion tensor imaging analysis used the fractional anisotropy (FA) values and tract volume as parameters of white matter tract degeneration. The FA values of regions related to ataxia were analyzed, that is the SMA, posterior limb of the internal capsule, basal ganglia, superior cerebellar peduncle, middle cerebellar peduncle, inferior cerebellar peduncle, and cerebellum. Tract volumes of the corticostriatal tract and cortico-ponto-cerebellar (CPC) tract originating from the SMA were evaluated. There were significant differences among the 3 groups in FA values of the subcortical regions of the CPC tract. Furthermore, the volume of the CPC tract originating from the SMA showed significant negative correlation with ataxia severity. There was no correlation between ataxia and corticostriatal tract volume. Therefore, we found that subcortical lesions of the CPC tract originating from the SMA could contribute to ataxia severity in stroke patients with ataxic hemiparesis.

Keywords: Ataxia; Stroke; Diffusion Tensor Imaging; White Matter; Supplementary Motor Area

INTRODUCTION

The role of cortical lesions in the supplementary motor area (SMA) in ataxia has been emphasized recently. Studies in stroke patients demonstrated that the SMA is crucial for postural recovery and regulates postural muscle tone through cortico-reticular spinal networks [1]. Previous studies on healthy subjects showed that cortical lesions of the SMA affect balance control [1-3]. Moreover, other studies have suggested that the cortical area of the SMA can be a potential target for neuromodulative treatments such as transcranial

Conflict of Interest
The authors have no potential conflicts of interest to disclose.

magnetic stimulation, electric stimulation, and neurofeedback to augment balance recovery after stroke [4-6]. However, subcortical white matter projections from the SMA and its relationship with ataxia have not been investigated in stroke patients.

The white matter tracts starting from the SMA were examined in both cerebellar ataxia patients and normal controls, to study the connectivity of the cortico-striatal tract and cortico-cerebellar networks using neuroimaging techniques [7]. White matter tracts originating from the SMA in humans consist of association fibers, projection fibers, and commissural fibers [8-10]. Among these white matter tracts emerging from the SMA, the corticostriatal tract reaches the striatum, and the cortico-ponto-cerebellar (CPC) tract extends to the cerebellum [7,11]. Although both the corticostriatal tract and the CPC tract are expected to contribute to ataxia and balance control, their role in ataxic hemiparesis (AH) remains elusive.

The core symptom of AH is ipsilateral ataxia with pyramidal signs on one side [12]. A large-scale study of ischemic stroke patients showed that 1.5% of patients were diagnosed with AH [13,14]. AH could lead to poor balance control with gait disturbance. Patients with AH could have an increased dependency in activities of daily living, not because of motor weakness, but because of ataxia [15]. Moreover, ataxia severity is considered a prognostic factor in patients with mild stroke [15].

Previously known lesions related to ataxia are infratentorial lesions, such as those in the cerebellum and brainstem. However, recent studies have reported supratentorial lesions contributing to ataxia, such as those in the basal ganglia, corona radiata, internal capsule, and fronto-ponto-cerebellar (FPC) pathway [16-18]. The specific supratentorial lesion that leads to AH remains controversial. We hypothesized that lesions in the subcortical projections from the SMA are associated with AH.

The objective of this study was to investigate the subcortical projections of the SMA and its relationship with ataxia in supratentorial stroke patients using diffusion tensor imaging (DTI).

MATERIALS AND METHODS

Subject selection and study population

We performed a retrospective medical record review of 454 stroke patients admitted to the rehabilitation department of a university hospital between January 2010 and June 2021. Patients with unilateral supratentorial stroke and hemiparesis who underwent DTI analysis were enrolled. The exclusion criteria were (i) stroke patients with cortical lesions in the SMA; (ii) motor weakness with Medical Research Council muscle scale test grade 3 or lower; (iii) severe cognitive impairment (Korean-Mini Mental Status Exam [K-MMSE] < 10); (iv) other etiologies inducing ataxia and Parkinson's disease; and (v) infratentorial infarction. **Fig. 1** shows a flowchart of the subject selection process. The Scale for Ataxia Rating Assessment (SARA) was used to evaluate ataxia severity [19].

A total of 33 patients were included and divided into 3 groups according to the ataxia severity (severe ataxia: SARA > 11.5; mild to moderate ataxia: SARA ≤ 11.5; no ataxia: SARA = 0) [20].

The institutional review board approved the waiver of informed consent due to the retrospective design of the study (KUH1180037).

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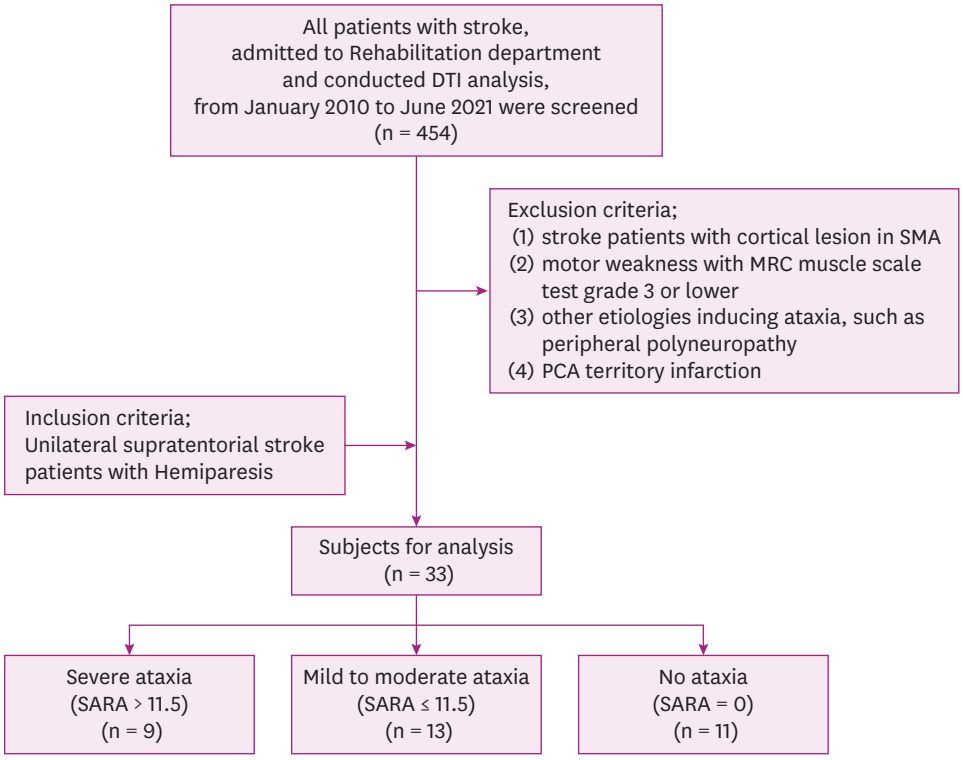

Fig. 1. Flow chart of subject selection.
DTI, diffusion tensor imaging; SMA, supplementary motor area; MRC, Medical Research Council; PCA, principal component analysis; SARA, Scale for Ataxia Rating Assessment.

Clinical assessment

The SARA score is a reliable and valid method to evaluate ataxia [19,20], with a total score of 0 (no ataxia) to 40 (most severe ataxia), and consists of 8 items: gait, stance, sitting, speech disturbance, finger chase, nose-finger test, fast alternating hand movements, and heel-shin slide [19,21]. The subjects were assessed using the following scales at the time of admission to the rehabilitation department: National Institutes of Health Stroke Scale (NIHSS), Motricity Index (MI) of the impaired side, Fugl-Meyer Assessment (FMA) of the impaired side, and K-MMSE.

DTI acquisition

DTI scans were acquired at 4 weeks after onset using a 3-T MRI scanner (Discovery MR750; GE Healthcare, Chicago, IL, USA) with an 8-channel head coil. DTI was performed with a single shot echo-planar imaging sequence with the following parameters: matrix = 120 × 120 matrix, field of view = 240 × 240 mm^2, echo time = 84 ms, repetition time = 16,000 ms, flip angle = 90, b-value = 1,000 mm^2s^{-1}, slice thickness = 2 mm, diffusion gradient direction number = 15. Fractional anisotropy (FA) values of each lesion and tract volume were investigated.

DTI analysis of FA values

DTI Studio software (Johns Hopkins Medical Institute, Johns Hopkins University, Baltimore, MD, USA) was used to calculate the FA values. We investigated regions of interest (ROIs) in the corticostriatal tract and CPC tract pathways [22,23]. The 7 ROIs were determined using reproducible anatomical landmarks on the color map of axial and coronal images used in

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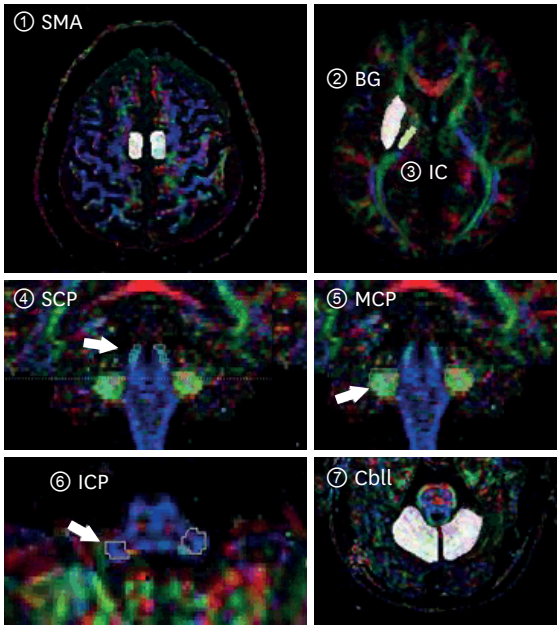

Fig. 2. Localization of the ROIs. ROIs were determined using reproducible anatomical landmarks on color map images. ROIs were superimposed on color map axial views of one subject.
SMA, supplementary motor area; BG, basal ganglia; IC, posterior limb of internal capsule; SCP, superior cerebellar peduncle; MCP, middle cerebellar peduncle; ICP, inferior cerebellar peduncle; Cbll, cerebellum; ROI, region of interest.

previous studies [23,24]. The FA values of the lesional and contralesional sides were also investigated [25,26]. Seven ROIs were analyzed: the SMA, basal ganglia, posterior limb of the internal capsule [23], superior cerebellar peduncle, middle cerebellar peduncle (MCP), inferior cerebellar peduncle (ICP), and cerebellum on both lesional and contralesional sides [27]. ROIs were hand-drawn by one experimenter based on DTI images in order to prevent misregistration effects between DTI and T1-weighted images [23]. **Fig. 2** shows the axial view of ROIs superimposed on the color map.

DTI analysis for measuring tract volume

The tract volumes of the corticostriatal tract and the CPC tract emerging from the SMA were analyzed using the DTI Studio software. **Fig. 3** shows the trajectory of the corticostriatal tract

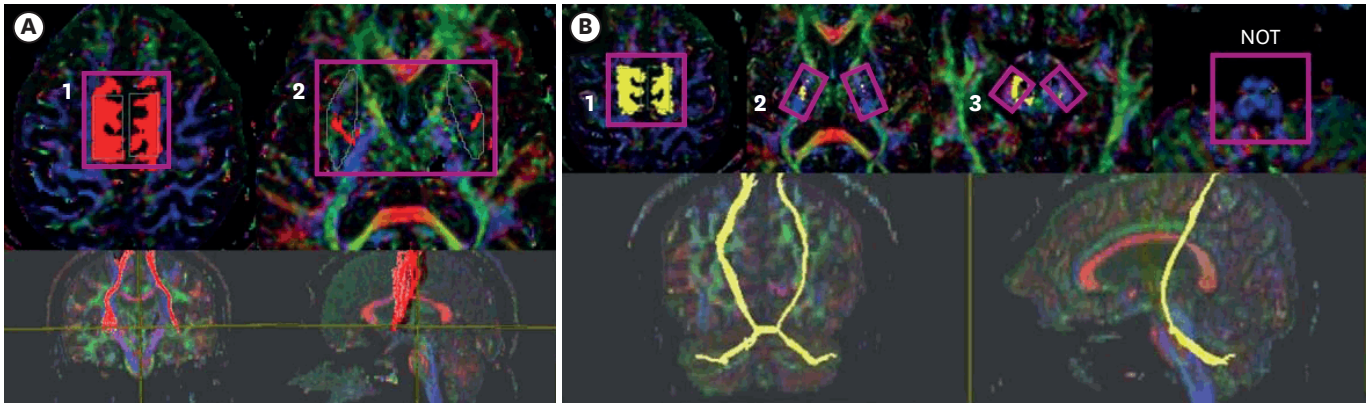

Fig. 3. Diffusion tensor tractography of subjects (red tract: corticostriatal tract, yellow tract: CPC tract). (A) ROI 1: SMA, ROI 2: striatum. (B) ROI 1: SMA, ROI 2: fibers generated in the lateral portion of the crus cerebri. ROI 3: Not operation was done to remove part of the CST that may have been included with the CPC tract.
CPC, cortico-ponto-cerebellar; ROI, region of interest; SMA, supplementary motor area; CST, corticospinal tract.

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and the CPC tract. ROI locations (ROI 1 and ROI 2) were used for reconstruction of the tracts in color maps at various slice locations and orientations. **Fig. 3A** shows the 2 ROIs needed for the demonstration of the corticostriatal tract: ROI 1 in the SMA and ROI 2 in the striatum [23]. **Fig. 3B** shows the 3 ROIs used in the reconstruction of the CPC tract: the location of ROI 1 in the SMA and ROI 2 in the fibers generated in the lateral portion of the crus cerebri. A non-operation was also performed to remove part of the corticospinal tract (CST) [28].

We investigated CST volume on the lesional side, considering that ataxia symptoms are independent of motor weakness. The CST was reconstructed using 2 ROIs on the axial view of the color-coded map, ROI 1 on the posterior limb of the internal capsule, and ROI 2 on the anterior pons [29].

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA). A Kruskal-Wallis test was performed to compare the FA values and tract volumes of the groups. Spearman's correlation analysis was used to test the correlation between ataxia severity and the FA value of each ROI. The relationship between ataxia severity and tract volume of the corticostriatal tract, CPC tract, and CST was studied using Spearman's correlation analysis in the subgroup of patients with AH (SARA > 0).

RESULTS

Patient characteristics

Thirty-three patients were divided into 3 groups (severe ataxia, n = 9; mild to moderate ataxia, n = 13; no ataxia, n = 11). Patient demographics and clinical characteristics were compared among the 3 groups (**Table 1**). There were no significant differences between the groups in sex, age, NIHSS, MI of upper extremity, MI of lower extremity, FMA of upper extremity, FMA of lower extremity, and K-MMSE ($p > 0.05$). The mean SARA score of the severe ataxia group (SARA > 11.5) was 15.83 ± 2.97, and that of the mild to moderate ataxia group (SARA ≤ 11.5) was 6.03 ± 2.89. The SARA score of the no ataxia group was 0.

Table 1. Patient demographic and clinical characteristics

Characteristics	Severe ataxia group (n = 9)	Mild to moderate ataxia group (n = 13)	No ataxia group (n = 11)	p value
Sex (M/F)	7/4 (63.6/36.4)	10/3 (76.9/23.1)	8/1 (88.9/11.1)	0.431
Age (yrs)	64.89 ± 15.82	57.85 ± 10.99	61.00 ± 15.86	0.739
Lesion side (right/left)	5/6 (45.5/54.5)	6/7 (46.2/53.8)	5/4 (55.6/44.4)	0.886
Type of stroke (infarction/hemorrhage)	6/5 (54.5/45.5)	8/5 (61.5/38.5)	8/1 (88.9/11.1)	0.247
Ataxia severity (SARA)	15.83 ± 2.97	6.03 ± 2.89	0.000	0.000
NIHSS	4.11 ± 3.10	4.38 ± 2.63	4.00 ± 1.61	0.913
MI_UE	72.89 ± 8.76	82.85 ± 14.54	75.45 ± 18.58	0.294
MI_LE	71.94 ± 7.54	84.61 ± 14.52	77.18 ± 17.66	0.202
FMA_UE	49.67 ± 7.83	60.00 ± 6.88	45.91 ± 14.70	0.010
FMA_LE	29.89 ± 2.76	29.69 ± 3.95	29.91 ± 10.61	0.688
MMSE	19.22 ± 6.02	22.77 ± 8.31	22.18 ± 7.18	0.258

Data are presented as ratio or mean ± standard deviation. The p value was derived from the Kruskall-Wallis test.
SARA, Scale for Ataxia Rating Assessment; NIHSS, National Institutes of Health Stroke Scale; MI_UE, Motricity Index of affected upper extremity; MI_LE, Motricity Index of affected lower extremity; FMA_UE, Fugl-Meyer Assessment of affected upper extremity; FMA_LE, Fugl-Meyer Assessment of affected lower extremity; K-MMSE, Korean-Mini Mental Status Exam.

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Table 2. Comparison of the FA values of ROIs among the groups

ROIs	Severe ataxia group (n = 9)	Mild to moderate ataxia group (n = 13)	No ataxia group (n = 11)	p value
SMA				
Lesion side	0.25 ± 0.03	0.25 ± 0.05	0.24 ± 0.05	0.863
Contralesional side	0.25 ± 0.03	0.27 ± 0.02	0.26 ± 0.03	0.260
BG				
Lesion side	0.22 ± 0.07	0.26 ± 0.07	0.28 ± 0.10	0.235
Contralesional side	0.26 ± 0.09	0.32 ± 0.13	0.32 ± 0.10	0.206
IC				
Lesion side	0.44 ± 0.11	0.60 ± 0.11	0.55 ± 0.14	0.010*
Contralesional side	0.61 ± 0.12	0.72 ± 0.05	0.70 ± 0.12	0.096
SCP				
Lesion side	0.56 ± 0.09	0.57 ± 0.07	0.55 ± 0.07	0.529
Contralesional side	0.57 ± 0.05	0.59 ± 0.07	0.61 ± 0.08	0.277
MCP				
Lesion side	0.62 ± 0.13	0.71 ± 0.05	0.71 ± 0.07	0.184
Contralesional side	0.56 ± 0.06	0.70 ± 0.07	0.75 ± 0.05	0.001**
ICP				
Lesion side	0.54 ± 0.07	0.61 ± 0.06	0.58 ± 0.11	0.079
Contralesional side	0.56 ± 0.07	0.64 ± 0.06	0.63 ± 0.09	0.015*
Cbll				
Lesion side	0.22 ± 0.04	0.24 ± 0.02	0.22 ± 0.03	0.094
Contralesional side	0.21 ± 0.10	0.22 ± 0.03	0.24 ± 0.04	0.001**

Data are presented as mean ± standard deviation. The p value was derived from the Kruskall-Wallis test.
FA, fractional anisotropy; ROI, region of interest; SMA, supplementary motor area; BG, basal ganglia; IC, posterior limb of internal capsule; SCP, superior cerebellar peduncle; MCP, middle cerebellar peduncle; ICP, inferior cerebellar peduncle; Cbll, cerebellum.
*p value < 0.05; **p value < 0.01.

FA values analysis of the ROIs

The FA values of the posterior limb of the internal capsule on the lesion side (p = 0.01), MCP on the contralesional side (p = 0.001), ICP on the contralesional side (p = 0.015), and cerebellum on the contralesional side (p = 0.001) were significantly different among the groups. Additionally, the FA value of the MCP on the contralesional side and the FA value of the ICP on the contralesional side were significantly increased with ataxia severity in each group (**Table 2**). The FA value of the MCP on the contralesional side showed a significant negative correlation in Spearman's correlation analysis (r = −0.673, p = 0.001) (**Table 3**).

Table 3. Correlation between the SARA score and FA values of each ROI

ROIs	Side	Correlation coefficient	p value
SMA	Lesion	−0.135	0.550
	Contra-lesion	0.157	0.485
BG	Lesion	0.180	0.423
	Contra-lesion	0.013	0.955
IC	Lesion	−0.284	0.200
	Contra-lesion	−0.042	0.853
SCP	Lesion	−0.208	0.354
	Contra-lesion	0.098	0.665
MCP	Lesion	−0.003	0.991
	Contra-lesion	−0.673	0.001**
ICP	Lesion	−0.243	0.275
	Contra-lesion	−0.374	0.087
Cerebellum	Lesion	−0.233	0.297
	Contra-lesion	−0.367	0.093

The p value was derived from the Spearman's correlation analysis.
SARA, Scale for Ataxia Rating Assessment; FA, fractional anisotropy; ROI, region of interest; SMA, supplementary motor area; BG, basal ganglia; IC, posterior limb of internal capsule; SCP, superior cerebellar peduncle; MCP, middle cerebellar peduncle; ICP, inferior cerebellar peduncle.
**p value < 0.01.

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Table 4. Comparison of the tract volumes between the groups

Tract	Severe ataxia group (n = 9)	Mild to moderate ataxia group (n = 13)	No ataxia group (n = 11)	p value
CPC tract volume	1,256.11 ± 927.72	2,472.62 ± 1,670.34	5,240.64 ± 3,073.93	0.003**
Corticostriatal tract volume	533.00 ± 406.41	769.23 ± 592.29	693.82 ± 278.90	0.365
CST tract volume	952.67 ± 394.81	1,167.08 ± 398.70	760.09 ± 636.65	0.215

Data are presented as mean ± standard deviation. The p value was derived from the Kruskall-Wallis test.
CPC, cortico-ponto-cerebellar; CST, corticospinal tract.
**p value < 0.01.

Table 5. Correlation between SARA score and tract volume

Tract	Correlation coefficient	p value
CPC tract volume	−0.447	0.037*
Corticostriatal tract volume	−0.228	0.307
CST tract volume	−0.223	0.319

The p value was derived from the Spearman's correlation analysis.
SARA, Scale for Ataxia Rating Assessment; CPC, cortico-ponto-cerebellar; CST, corticospinal tract.
*p value < 0.05.

Although the FA values of the posterior limb of the internal capsule on the lesion side, ICP on the contralesional side, and cerebellum on the contralateral side were significantly different, they did not have significant correlation with the SARA score (**Table 3**). The cortical area of the SMA (p > 0.05) and the basal ganglia (p > 0.05) on lesion side did not show significant differences among the groups.

Tract volume analysis

The tract volume of the CPC tract showed a significant difference among the groups (p = 0.003) (**Table 4**). Spearman's correlation analysis showed a statistically significant correlation between

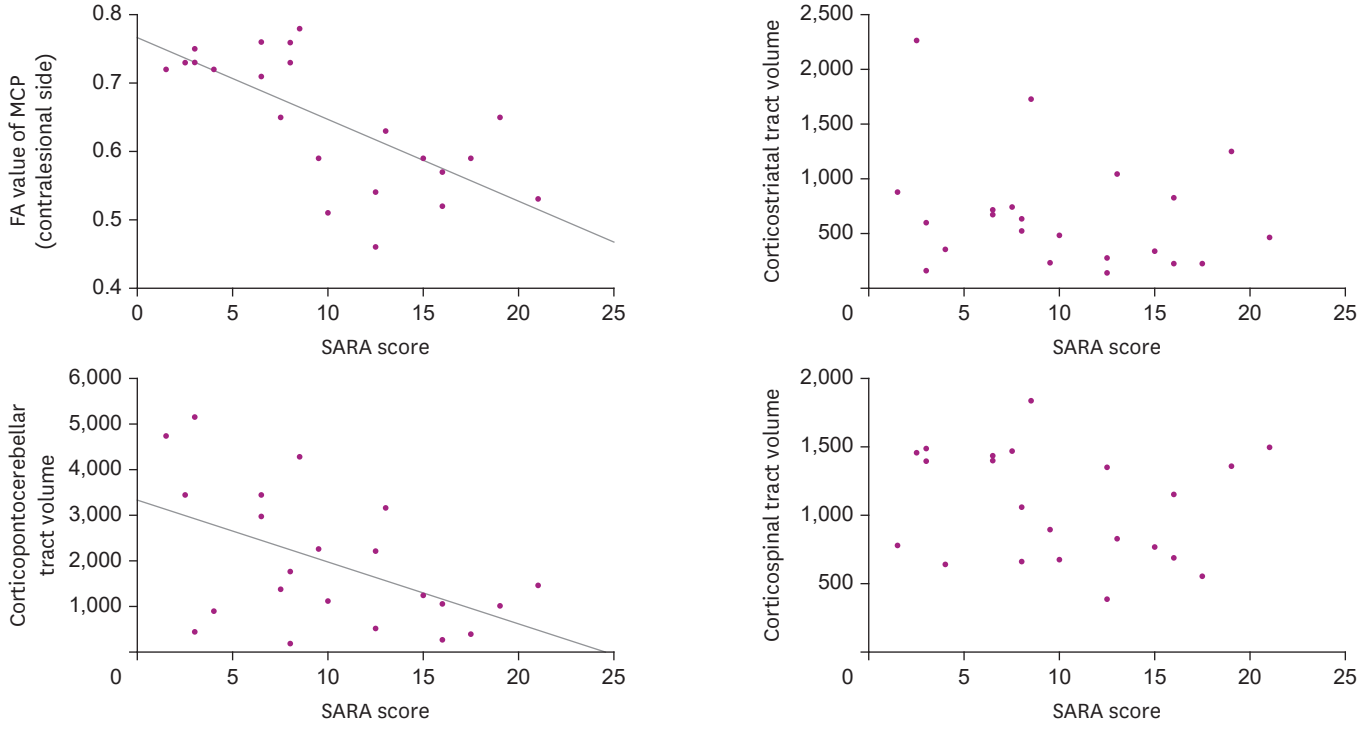

Fig. 4. Spearman's correlation between tract volume and SARA score. There was a moderate negative correlation between the CPC tract volume and SARA score. Both corticostriatal tract volume and corticospinal tract volume had no correlation with SARA score. In addition, the FA value of MCP on the contralesional side and the SARA score showed a negative correlation.
SARA, Scale for Ataxia Rating Assessment; CPC, cortico-ponto-cerebellar; MCP, middle cerebellar peduncle; FA, fractional anisotropy.

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the SARA score and CPC tract volume (**Table 5**). CPC tract volume was negatively correlated with the SARA score ($r = -0.447$, $p < 0.05$) (**Fig. 4**). However, coticostriatal tract volume and CST tract volume showed no differences among the groups. The corticostriatal tract volume ($r = -0.228$, $p > 0.05$) and CST tract volume ($r = -0.223$, $p > 0.05$) showed no correlation with the SARA score (**Fig. 4**).

DISCUSSION

This study suggests that the CPC tract starting from the SMA could be a supratentorial lesion causing ataxia in stroke patients. This study employed DTI to investigate the association between subcortical lesions and AH in supratentorial stroke patients, all of whom had MCA territory stroke lesions. The DTI parameters, such as the FA value and tract volume of the CPC tract, showed significant differences among the groups according to ataxia severity. The tract volume of the CPC tract originating from the SMA had a negative correlation with ataxia severity. The FA value reflects the integrity of the white matter tract, and the tract volume shows the remaining number of white matter tracts after stroke. A decrease in the FA value and tract volume suggests injury in the white matter tracts. Therefore, the CPC tract originating from the SMA could be a significant region for the subcortical lesions leading to AH in supratentorial stroke patients without cortical lesions in the SMA.

The FA values of the internal capsule on the lesional side, MCP on the contralesional side, and cerebellum on the contralesional side showed differences among the 3 groups. This result suggests that the white matter lesions of the CPC tract, as it passes through the internal capsule and decussates to pass the MCP on the contralesional side and cerebellum, according to a previous study [22]. The CPC pathway was reported in a previous study to pass through the following anatomical regions: starting from the cerebral cortex and passing through the ipsilateral internal capsule, the contralateral MCP, and the contralateral cerebellum [22]. The FA value of the MCP on the contralesional side was significantly correlated with ataxia severity, suggesting a relationship between ataxia and the white matter pathways passing through the MCP.

In addition, the FA value of the ICP on the contralesional side was significantly different among the groups. This is consistent with a previous study, which also suggested the ICP as an important lesion site related to ataxia in ischemic stroke patients, and the number of white matter fibers passing the ICP is decreased in stroke patients compared to controls [25]. Another previous study demonstrated the function of the ICP as integrating proprioception sensory input and contributing to balance control and posture [30]. Although there were significant differences among the groups, the FA values of ICP did not correlate with the severity of ataxia. This might be due to the small size of the study population in each group, and a study with a larger population is required.

This study demonstrated that the FA value of the SMA did not show a relationship with ataxia severity, whereas previous studies emphasized that the cortical lesion of the SMA contributes to ataxia and balance control [1,31]. They also insisted that the cortical lesion of the SMA is crucial for postural recovery after stroke. This discrepancy might be due to the heterogeneous assessment techniques. Previous studies used assessment techniques for cortical activities while the subjects conducted a task, while this study used DTI.

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In particular, the tract volume of the CPC tract originating from the SMA was significantly correlated with ataxia severity, which is consistent with the results of previous studies. A clinical case report confirmed injury in the CPC tract with DTI in a patient with mild traumatic brain injury presenting mild truncal ataxia [32]. Another study reconstructed CPC tracts with tractography and proposed that the integrity of the CPC tract could be used as an indicator of dystonia and ataxia [22]. However, there are few studies regarding the tract volume of the CPC tract starting from the SMA in patients with stroke. The CPC tract starts from various cerebral cortices, including the FPC tract, parieto-ponto-cerebellar (PPC) tract, occipito-ponto-cerebellar (OPC) tract, and temporo-ponto-cerebellar (TPC) tract [28]. The PPC tract is involved in the convergence of vision and proprioception, the OPC tract starts from visual cortices and is involved in visual recognition of movement patterns, and the TPC tract conducts high-level visual processing and recognition. Particularly, the FPC tract starts from the premotor cortex, such as the SMA, and its function is to plan coordinated movements [33]. The CPC tract volume could be used as a biomarker of ataxia severity, especially in supratentorial stroke patients.

Moreover, this study demonstrated that the CPC tract is related to ataxia in patients without cerebellar lesions. Ataxia due to supratentorial stroke should be considered in rehabilitation, as severe ataxia in patients with mild stroke could result in a lower functional level at the time of discharge [15]. Damage to the CPC tract can cause severe ataxia, leading to difficulties in activities of daily living and poor prognosis. A previous study reported that patients with supratentorial lesions show a relatively short recovery time, contrary to patients with cerebellar lesions [34].

Other studies have demonstrated that the corticostriatal tract contributes to postural muscle tone and ataxia [35,36]. In contrast, our study showed that the FA value of the basal ganglia on the lesion side and the tract volume of the corticostriatal tract originating from the SMA did not correlate with ataxia severity. This might be due to the dominant effect of the cortical area of the SMA on the corticostriatal tract rather than the subcortical area of the SMA, as we excluded patients with cortical lesions of the SMA in this study. Further studies are needed to evaluate the relationship between the cortical area of the SMA and corticostriatal tract.

CST tract volume analyzed by DTI reflects motor deficits in stroke patients [37]. We investigated CST tract volume and found no significant correlation between CST tract volume and ataxia severity. This could increase the sensitivity of this study by overcoming the limitation of the ROI approach of DTI analysis with operator-dependent validity [38].

The limitation of this study is the small sample size, which was due to difficulty in patient enrollment, as the incidence of stroke with AH is 1.25%–2% [13,14]. To overcome this limitation, we conducted a retrospective medical record review of over 10 years to include as many patients as possible. Further studies with a larger sample size are needed to validate our observations. Another limitation of this study is that we did not investigate other cerebral cortex lesions at sites from where CPC tract starts. The CPC tract starts from the cerebral cortex, including the FPC tract, PPC tract, OPC tract, and TPC tract [28,33]. The FPC tract starts from the premotor cortex, including the SMA. Other tracts are the PPC tract starting from the somatosensory cortex, the OPC tract starting from the visual cortex involved in visual processing, and the TPC tract from the inferior temporal gyrus involved in visual processing and recognition [33]. In this study, we mainly focused on the SMA as we tried to investigate lesions related to ataxia symptoms. Future studies should consider other tracts.

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In conclusion, we found that ataxia severity was related to the CPC tract originating from the SMA in stroke patients with AH. Subcortical lesions of the SMA, especially the CPC tract, should be investigated thoroughly for intervention regarding the prognostic value of ataxia. The volume of the CPC tract originating from the SMA could be suggested as a biomarker for ataxia and prognosis in supratentorial stroke patients with AH.

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