Evidence of Corticospinal Tract Injury at Midbrain in Patients With Subarachnoid Hemorrhage

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Background and Purpose—Clear elucidation of the exact pathophysiological mechanisms of motor weakness in patients with subarachnoid hemorrhage has not yet been achieved. We attempted to investigate injury to the corticospinal tract in patients with subarachnoid hemorrhage using diffusion tensor imaging.

Methods—Twenty-two patients with subarachnoid hemorrhage and 24 control subjects were recruited for this study.

DTI-Studio software was used for reconstruction of the corticospinal tract. We measured fractional anisotropy and apparent diffusion coefficient values at 5 regions of interest along the corticospinal tract pathway including: the corona radiata, the posterior limb of the internal capsule, the upper midbrain, the midpons, and the upper medulla.

Results—Fractional anisotropy value for the midbrain region of interest was lower in the patient group compared with the control group without change of apparent diffusion coefficient value (P<0.05). By contrast, fractional anisotropy and apparent diffusion coefficient values of the other 4 regions of interest were not different between the patient and control groups.

Conclusions—Injury of the corticospinal tract at the midbrain was observed in patients with subarachnoid hemorrhage.

Injury of the corticospinal tract at the midbrain appears to be one of the various pathophysiological mechanisms for motor weakness after subarachnoid hemorrhage. (Stroke. 2012;43:00-00.)

Key Words: corticospinal tract ■ diffusion tensor imaging ■ subarachnoid hemorrhage
fiber tracking. Voxel-based ROIs were drawn in the middle corona radiata, the posterior limb of the internal capsule, the midbrain at the level of superior colliculus, the midpons, and the medulla along the CST pathway (Figure). Mean FA value and mean apparent diffusion coefficient value were measured for each ROI.

**Statistical Analysis**

An independent t test was performed for determination of the statistically significant differences in particular variables between the patient and control groups. Correlation between DTI parameters for each ROI and MI score for the contralateral limbs were determined using the Spearman correlation test. Statistical analyses were performed using SPSS software (Version 15.0; SPSS, Chicago, IL), and statistical significance was set at $P<0.05$.

**Results**

Twenty-two patients (8 males; mean age, 54.0 years; range, 40–70 years) and 24 age- and sex-matched control subjects (10 males; mean age, 52.8 years; range, 41–72 years) with no history of neurological or psychiatric disease were recruited for this study. Seven of 22 patients had undergone shunt surgery for treatment of hydrocephalus and 10 patients were accompanied with intracerebral hemorrhage or intraventricular hemorrhage. Average Fisher CT grade for the patient group was 3 (3–4) and average MI score for the patient group was 74.2 (69.5–100); quadripleasis in 19 patients and hemiparesis in 3 patients.

FA value of the midbrain ROI was lower in the patient group compared with the control group ($P<0.05$); in contrast, no differences in FA values of the other 4 ROIs for the CST were observed between the patient and control groups ($P>0.05$; Table). We could not observe any difference in apparent diffusion coefficient value between the patient and control groups ($P>0.05$). On the other hand, in the patient group, no correlation was observed between MI scores and DTI parameters for each ROI for the CST ($P>0.05$).

**Discussion**

In the current study, we investigated injury of the CST in patients with SAH using DTI and found FA value of the midbrain ROI for the CST was lower in the patient group, compared with the control group, without change of apparent diffusion coefficient value. FA value represents the degree of directionality of microstructures and reduced FA value appears to be related to disintegration of those fibers. Results showing that, among the ROIs for the CST, a decreased value for FA was observed only at the midbrain level appears to indicate injury of the CST at the midbrain level.
No study of the pathophysiological mechanism of CST injury at the midbrain as a result of SAH has been conducted. However, based on the pathophysiological mechanism of periventricular white matter injury by intraventricular hemorrhage, we can make the assumption that the CST at the midbrain in patients with SAH can be exposed to hemorrhage such as injury to periventricular white matter by intraventricular hemorrhage.7 Findings from previous studies have suggested that injury to periventricular white matter could occur through mechanical (increased intracranial pressure or direct mass) or chemical mechanisms (a blood clot itself can cause extensive damage).8 In this study, considering the location of the CST from the cerebral cortex to the medulla, due to its close proximity to the cistern, the CST at the midbrain can easily be affected by hemorrhage.7 In addition, frequent occurrence of SAH into perimesencephalic cisterns could be ascribed to injury of the CST at the midbrain.1 These mechanisms of injury might be associated with mild weakness (mean MI, 74.2) of subjects in the patient group, which was similar to 4/5 on the Medical Research Council. This mild weakness might have contributed to the result showing no correlation between FA value at the midbrain and motor function in the patient group. In addition, weakness of the patient group might be ascribed in part to injury observed in other nonpyramidal motor tracts such as the corticoreticulo-spinal tract.

In conclusion, we observed injury of the CST at the midbrain in patients with SAH. Injury of the CST at the midbrain appears to be one of the various pathophysiological mechanisms for motor weakness after SAH. To the best of our knowledge, this is the first study to demonstrate injury of the CST at the subcortical area in patients with SAH. The major limitation of this study was the small number of subjects. In addition, we recruited the patients among the patients with SAH who had been admitted for rehabilitation. Therefore, it is possible that we recruited patients with severe clinical manifestations among all patients with SAH.

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Disclosures
None.

References
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