Classification of Cause of Motor Weakness in Traumatic Brain Injury Using Diffusion Tensor Imaging

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Background: Many studies have attempted to elucidate the causes of motor weakness in patients with traumatic brain injury (TBI). Most of these studies have focused on the specific cause of motor weakness. However, little is known about the classification and elucidation of the causes of motor weakness in consecutive patients with TBI.

Objective: To attempt to classify with diffusion tensor imaging the causes of motor weakness in patients with TBI by conducting an analysis of the injury mechanism of the corticospinal tract (CST).

Design: Retrospective study.

Setting: Rehabilitation department of a university hospital.

Patients: We recruited 41 consecutive patients who showed motor weakness among patients with TBI admitted for rehabilitation.

Main Outcome Measures: We classified the causes of weakness according to the injury mechanism of the CST on diffusion tensor imaging.

Results: Injury mechanisms of the CST were classified as follows, in order: diffuse axonal injury, 24 patients (58.5%); traumatic intracerebral hemorrhage, 9 patients (21.9%); transtentorial herniation, 6 patients (14.6%); and focal cortical contusion, 4 patients (9.8%). In patients with diffuse axonal injury, the mean number of lesions composing CST injury was 3.6 (range, 2-6) and CST injury locations were as follows: the pons (61%), the cerebral peduncle (50%), the medulla (40%), the posterior limb of the internal capsule (17%), and the corona radiata (13%).

Conclusion: We found that diffusion tensor imaging was useful in elucidation and classification of the causes of motor weakness resulting from CST injury in patients with TBI.
of motor weakness in consecutive patients with TBI has been conducted. In the current study, using DTI, we attempted to classify the causes of motor weakness by conducting an analysis of the CST in patients who showed motor weakness following TBI.

METHODS

SUBJECTS

We reviewed retrospectively medical records of 246 patients with TBI who had been admitted to the rehabilitation department of a university hospital. Among 246 consecutive patients with TBI, 41 patients (29 male; 12 female; mean age, 52.7 years; range, 23-74 years) were recruited according to the following inclusion criteria: (1) first-ever TBI, (2) age 20 to 75 years, (3) DTI scanning between 2 weeks and 3 months after TBI onset, (4) definite motor weakness: any motor weakness (score <4 of 5 on manual muscle testing) to rule out mild general weakness that was caused by deconditioning or any detectable motor asymmetry (score <5 of 5 on manual muscle testing);7 and (5) no peripheral nerve injury on electrodiagnostic test findings. Causes of TBI were as follows: motor vehicle collisions (28 patients), falls (9 patients), and other (blunt trauma: 2 patients, slip down: 2 patients). The Motricity Index was used for measurement of motor function of the affected extremities, with a maximum score of 100. Reliability and validity of the Motricity Index is well established.21 This study was approved by our institutional review board.

Injury mechanisms for motor weakness were classified according to the following criteria2-26-31: (1) diffuse axonal injury (DAI); injury to the CST was defined as petechial microhemorrhages on T2-weighted gradient recall echo images in the absence of visible lesions in T1-weighted, T2-weighted, and fluid-attenuated inversion recovery images,19,32,33 (2) traumatic sense of visible lesions in T1-weighted, T2-weighted, and fluid-attenuated inversion recovery images in the absense of visible lesions in T1-weighted, T2-weighted, and fluid-attenuated inversion recovery images;19,32,33 (2) traumatic intracerebral hemorrhage (TICH): any evidence showing that the CST was injured by hematoma, (3) transtentorial herniation (TH): TH on brain computed tomography at TBI onset, the CST was injured by hematoma, (4) transtentorial herniation (TH): TH on brain computed tomography at TBI onset, the CST was injured by hematoma, (5) no peripheral nerve injury on electrodiagnostic test findings.

Patient demographic data according to the injury mechanism of the CST on DTI is shown in the Table. No significant differences were observed in sex (P = .08), age (P = .16), duration from onset to DTI scanning (P = .23), and motor function (P = .12) among injury mechanisms of the CST. Injury mechanisms of the CST were classified as follows, in order: DAI, 24 patients (58.5%); TICH, 9 patients (21.9%) (all hemorrhages were located mainly at the basal ganglia); TH, 6 patients (14.6%); and FCC, 4 patients (9.8%). Two patients were detected as having combined injuries on diffusion tensor tractography (1 patient: DAI and FCC, 1 patient: DAI and TICH). The mean Motricity Index scores of the affected extremities were 43.4 in patients with DAI, 25.2 in patients with TICH, 30.0 in patients with FCC. On diffusion tensor tractography of patients with DAI, disruption of the CST was observed in 10 patients of 24 patients. All 24 patients had more than 1 lesion composing CST injury; the mean number of lesions composing CST injury was 3.6 (range, 2-6). Corticospinal tract injury was present at the following locations: the pons (61%), the cerebral peduncle (30%), the medulla (40%), the posterior limb of the internal capsule (17%), and the corona radiata (13%). In patients with TICH, disruption (5 patients) at the lesion or wallerian degeneration (4 patients) of the CST was observed in all 9 patients. All 6 patients with TH showed disruption of the CST at the cerebral peduncle or pons level. In patients with FCC,
disruption of the CST was observed below the lesion of the primary motor cortex in all 4 patients (Figure).

**COMMENT**

In the current study, using DTI, we attempted to classify the causes of motor weakness in patients with TBI by conducting an analysis of the CST. Four injury mechanisms for the CST were found to cause motor weakness in TBI: DAI (58.5%), TICH (21.9%), TH (14.6%), and FCC (9.8%). Some of the patients with TICH (11.1%) and FCC (25%) had DAI simultaneously. To the best of our knowledge, only 1 study that classified the causes of motor weakness in consecutive patients with TBI, like our study, has been reported. In 1998, on the basis of brain computed tomography or magnetic resonance imaging findings, Katz et al found that injury mechanisms of patients with upper extremity weakness following TBI were as follows: DAI in 72.7%; FCC, including ICH, in 40.5%; herniation in 15.9%; and hypoxic-ischemic injury in 6.8%. This incidence is similar to that of our results. As for DAI, the mean number of lesions composing CST injury was 3.6 (range, 2-6) and CST injury locations were as follows: the pons (61%), the cerebral peduncle (50%), the medulla (40%), the posterior limb of the internal capsule (17%), and the corona radiata (13%). In 2009, Jang et al found that the mean number of CST injuries was 3.6 (range, 2-7) per patient in 14 patients with DAI and that the CST was involved at the following locations: the pons (61%), the cerebral peduncle (39%), the corona radiata (21%), the medulla (14%), and the posterior limb of the internal capsule (11%). These results are also consistent with those of our study. As for TICH, several studies have reported that the incidence of traumatic basal ganglia hemorrhage is approximately 3% (range, 2.4%-3.4%) among patients with TBI. By contrast, the incidence of DAI was 21.9% in our study. The reason that the incidences of TICH were different between previous studies and our study seemed to be caused by difference of inclusion criteria. The previous studies estimated the incidence of TICH from patients with TBI irrespective of motor weakness; in contrast, our study estimated the incidence of TICH only from the patients with TBI who showed motor weakness.

Since the introduction of DTI, it has been used in analysis of the CST in patients with TBI for elucidation of the causes of motor weakness, mainly DAI. In 2006, Lee et al used DTI to demonstrate DAI in 2 patients with CST injury by DAI. During the same year, Ahn et al reported on 2 patients who showed DAI lesions at the brainstem level that were demonstrated by DTI. In 2007, Yasokawa et al found that motor dysfunction revealed by motor-evoked potential showed significant correlation with DTI in patients with DAI. During the same year, Han et al demonstrated recovery of motor function of a patient that occurred as a result of recovery of a CST injured by DAI. In 2009, Jang et al reported on the incidence and distribution of CST injury in DTI for 14 patients with DAI, as mentioned earlier. Jang et al recently demonstrated the usefulness of diffusion tensor tractography in elucidation of the causes of motor weakness in patients with TBI. Therefore, as far as we are aware, this is the first DTI study to classify the causes of motor weakness in patients with TBI.

In conclusion, in the current study, we recruited 41 consecutive patients with TBI and classified the causes of weakness by analysis of the injury mechanism of the CST on DTI. We found that DTI was useful in elucidation and classification of the causes of motor weakness resulting from CST injury in patients with TBI. On the other hand, the extrapyramidal pathways such as the reticulospinal tract, vestibulospinal tract, and rubrospinal tract can be involved in motor function in the human brain although the CST is an important motor tract for voluntary skilled movements. Injury of these extrapyramidal pathways might cause motor weakness in patients with TBI. However, we could not estimate these extrapyramidal pathways in this

**Table. Patients’ Demographic Data According to the Injury Mechanism of the CST on Diffusion Tensor Imaging**

<table>
<thead>
<tr>
<th>Involvement</th>
<th>Type of CST Injury Mechanism</th>
<th>Traumatic ICH</th>
<th>Transientorial Herniation</th>
<th>Focal Cortical Contusion</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, No. *</td>
<td>No. (%)</td>
<td>24 (58.5)</td>
<td>9 (21.9)</td>
<td>6 (14.6)</td>
<td>4 (9.8)</td>
</tr>
<tr>
<td>M</td>
<td>15</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>9</td>
<td>2</td>
<td>6</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Age, y, mean (SD) *</td>
<td>54.9 (15.7)</td>
<td>50.0 (11.6)</td>
<td>45.7 (16.8)</td>
<td>66.5 (11.9)</td>
<td>53.7 (15.2)</td>
</tr>
<tr>
<td>Duration, d, mean (SD) *</td>
<td>51.1 (22.9)</td>
<td>40.4 (21.1)</td>
<td>33.0 (9.8)</td>
<td>43.0 (14.5)</td>
<td>45.6 (21.1)</td>
</tr>
<tr>
<td>MI score, mean (SD) *</td>
<td>43.4 (17.6)</td>
<td>25.2 (23.4)</td>
<td>36.5 (17.6)</td>
<td>30.0 (27.5)</td>
<td>37.4 (20.6)</td>
</tr>
<tr>
<td>Vector</td>
<td>Hemiplegia</td>
<td>4</td>
<td>8</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Quadruplegia</td>
<td>20</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>MVC</td>
<td>18</td>
<td>6</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>FD</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviations: CST, corticospinal tract; DAI, diffuse axonal injury; Duration, the duration from onset to diffusion tensor imaging scanning; FD, fall down; ICH, intracerebral hemorrhage; MI, Motricity Index; MVC, motor vehicle collision.

*No significant between-group difference (P > .05).
Figure. Patient 1: Brain computed tomography (CT) images at onset show no focal lesion (A). Brain T2-weighted images at 4 weeks after onset show no focal lesion (B). Diffusion tensor tractography images for the corticospinal tract show disruption at the right midpons (green arrow) and left cerebral peduncle (blue arrow) (C). A indicates anterior; P, posterior; and R, right. Patient 2: Brain CT images at onset show hematoma in the right corona radiata and basal ganglia (A). Brain T2-weighted images at 11 weeks after onset show leukomalatic changes in the right corona radiata and basal ganglia (B). Diffusion tensor tractography images for the corticospinal tract show disruption at the lesion (green arrow) (C). Patient 3: Brain CT images at onset show left transtentorial herniation (blue arrow) (A). Brain T2-weighted images at 5 weeks after onset show leukomalatic changes in the left cerebral peduncle (B). Diffusion tensor tractography images for the corticospinal tract show disruption below the cerebral peduncle (blue arrow) (C). Patient 4: Brain CT images at onset show hematoma in the right frontal area (A). Brain T2-weighted images at 6 weeks after onset show leukomalatic change in the right frontal lobe, including the primary motor cortex (B). Diffusion tensor tractography images for the corticospinal tract show disruption below the focal cortical contusion (green arrow) (C).
study because DTI techniques for analysis of the extrapyramidal pathways have not been developed so far. Therefore, we think that DTI studies on the extrapyramidal pathways would be necessary for thorough elucidation of motor weakness in TBI in the near future. Clinical correlation studies comparing the degree of the CST injury with motor weakness are also necessary.

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