

ONLINE FIRST

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.

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TRAUMATIC BRAIN INJURY (TBI) is one of the most common neurologic disorders causing disability, and motor weakness is one of the main sequelae, along with cognitive dysfunction and behavior problems.¹⁻³ Previous studies have reported the incidence of motor weakness as 9% to 56% following TBI.^{1,2,4-7} Elucidation of the cause of motor weakness is necessary for successful rehabilitation in TBI; this information enables establishment of scientific rehabilitative strategies, estimation of the rehabilitative period, and prediction of final outcome for patients with TBI.^{1,6,8-17}

Many studies have attempted to elucidate the causes of motor weakness in patients with TBI; various methods have been used, including clinical manifestation, brain computed tomography, conventional brain magnetic resonance imaging, or transcranial magnetic stimulation.^{1,8-11,18-20} Most of these studies have fo-

cused on the specific cause of motor weakness.^{8-11,19,20} However, little is known about the classification and elucidation of the causes of motor weakness in consecutive patients with TBI.¹ In addition, many difficulties have been encountered in the attempt to elucidate the exact causes of motor weakness because tools for use in evaluation are limited in that they do not allow for estimation and visualization of neural tracts 3-dimensionally.

Recent advances in diffusion tensor imaging (DTI) allow for both morphologic and quantitative estimation of the state of neural tracts indirectly at the subcortical level.²¹⁻²³ Therefore, several studies have demonstrated the usefulness of DTI in elucidation of the cause of weakness by indirect estimation of the state of the corticospinal tract (CST), which is the most important motor tract in the human brain.^{12-17,24-26} However, to our knowledge, no DTI study for classification of the injury mechanisms of the CST as the causes

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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),² 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.²⁷ This study was approved by our institutional review board.

Injury mechanisms for motor weakness were classified according to the following criteria^{1,28-31}: (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 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, and brain magnetic resonance imaging showed no other specific lesion to explain the weakness, except for TH, and (4) focal cortical contusion (FCC): cortical contusion on brain magnetic resonance imaging, which could explain the patient's weakness.^{1,28,34} We defined wallerian degeneration as the discontinued upper end of the injured CST being detected far from below the lesion on diffusion tensor tractography.

DTI ACQUISITION

The DTI data were acquired at an average time of 45 days (range, 14-90 days) after TBI onset using a 1.5-T Philips Gyroscan Intera system equipped with a Synergy-L Sensitivity Encoding head coil with a single-shot spin echo planar imaging sequence. For each of the 32 noncollinear and noncoplanar diffusion-sensitizing gradients, we acquired 60 contiguous slices parallel to the anterior commissure-posterior commissure line. Imaging parameters were as follows: matrix=128×128; field of view=221×221 mm²; echo time=76 milliseconds; repetition time=10 726 milliseconds; Synergy-L Sensitivity Encoding factor=2; echo planar imaging factor=67; b=1000 s/mm²; number of signals acquired=1; and slice thickness=2.3 mm.

DTI ANALYSIS

Oxford Centre for Functional Magnetic Resonance Imaging of the Brain software (FSL; www.fmrib.ox.ac.uk/fsl) was used in preprocessing of DTI data sets. Affine multiscale 2-dimen-

sional registration was used for removal of eddy current-induced image distortions and motion artifacts. DTI Studio software (Center of Magnetic Resonance Microimaging, Johns Hopkins Medical Institute) was used for evaluation of the CST; fiber tracking was performed using the Fiber Assignment by Continuous Tracking algorithm, a brute-force reconstruction approach, and a multiple regions of interest (ROIs) approach. The seed ROI was drawn in the CST portion of the anterior midpons on a number of 2-dimensional fractional anisotropy color maps. The target ROI was drawn in the CST portion of the anterior lower pons. Fiber tracts passing through both ROIs were designated as the final tracts of interest. Termination criteria used for fiber tracking included fractional anisotropy less than 0.2 and an angle change more than 60.³⁵ In patients with DAI, we evaluated DTI using fractional anisotropy in ROIs along the CST. Circular ROIs were drawn in the middle corona radiata, the posterior limb of the internal capsule, the midportion of the cerebral peduncle, the pons, and the medulla along the CST. To include only the region of the CST, the typical ROI size was set between 5 and 10 voxels. We defined a CST injury as a finding of a fractional anisotropy value 2 SDs below that of the normal control value of the previous study.¹²

STATISTICAL ANALYSIS

Data were analyzed using SPSS 17.0 software (IBM SPSS). All the data used were proved to be normally distributed by the Kolmogorov-Smirnov normality test. Then we used a parametric 1-way analysis of variance test for comparison of age, duration from onset to DTI scanning, and motor function among injury mechanisms of the CST. The Fisher exact test was used to compare the differences of sex among groups. The significance level was set at $P=.05$.

RESULTS

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, 36.5 in patients with TH, and 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 (50%), 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,

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

	Type of CST Injury Mechanism				Total
	DAI	Traumatic ICH	Transtentorial Herniation	Focal Cortical Contusion	
No. (%)	24 (58.5)	9 (21.9)	6 (14.6)	4 (9.8)	41 (100)
Sex, No. ^a					
M	15	7	6	1	29
F	9	2	0	3	12
Age, y, mean (SD) ^a	54.9 (15.7)	50.0 (11.6)	45.7 (16.8)	66.5 (11.9)	53.7 (15.2)
Duration, d, mean (SD) ^a	51.1 (22.9)	40.4 (21.1)	33.0 (9.8)	43.0 (14.5)	45.6 (21.1)
MI score, mean (SD) ^a	43.4 (17.6)	25.2 (23.4)	36.5 (17.6)	30.0 (27.5)	37.4 (20.6)
Involvement					
Hemiplegia	4	8	4	3	19
Quadriplegia	20	1	2	1	22
Vector					
MVC	18	6	3	3	28
FD	3	3	3	0	9
Other	3	0	0	1	4

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.

^aNo significant between-group difference ($P > .05$).

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 al¹ found that injury mechanisms in 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 al¹² 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.^{10,36-38} By contrast, the incidence of TICH 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.¹²⁻¹⁷ In 2006, Lee et al¹⁵ used DTI to demonstrate DAI in 2 patients with CST injury by DAI. During the same year, Ahn et al¹³ reported on 2 patients who showed DAI lesions at the brainstem level that were demonstrated by DTI. In 2007, Yasokawa et al¹⁶ found that motor dysfunction revealed by motor-evoked potential showed significant correlation with DTI in patients with DAI. During the same year, Han et al¹⁴ 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 al¹² reported on the incidence and distribution of CST injury in DTI for 14 patients with DAI, as mentioned earlier. Jang et al¹⁷ recently demonstrated the usefulness of diffusion tensor tractography in elucidation of the causes of motor weakness in 5 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.³⁹⁻⁴³ Injury of these extrapyramidal pathways might cause motor weakness in patients with TBI. However, we could not estimate these extrapyramidal pathways in this

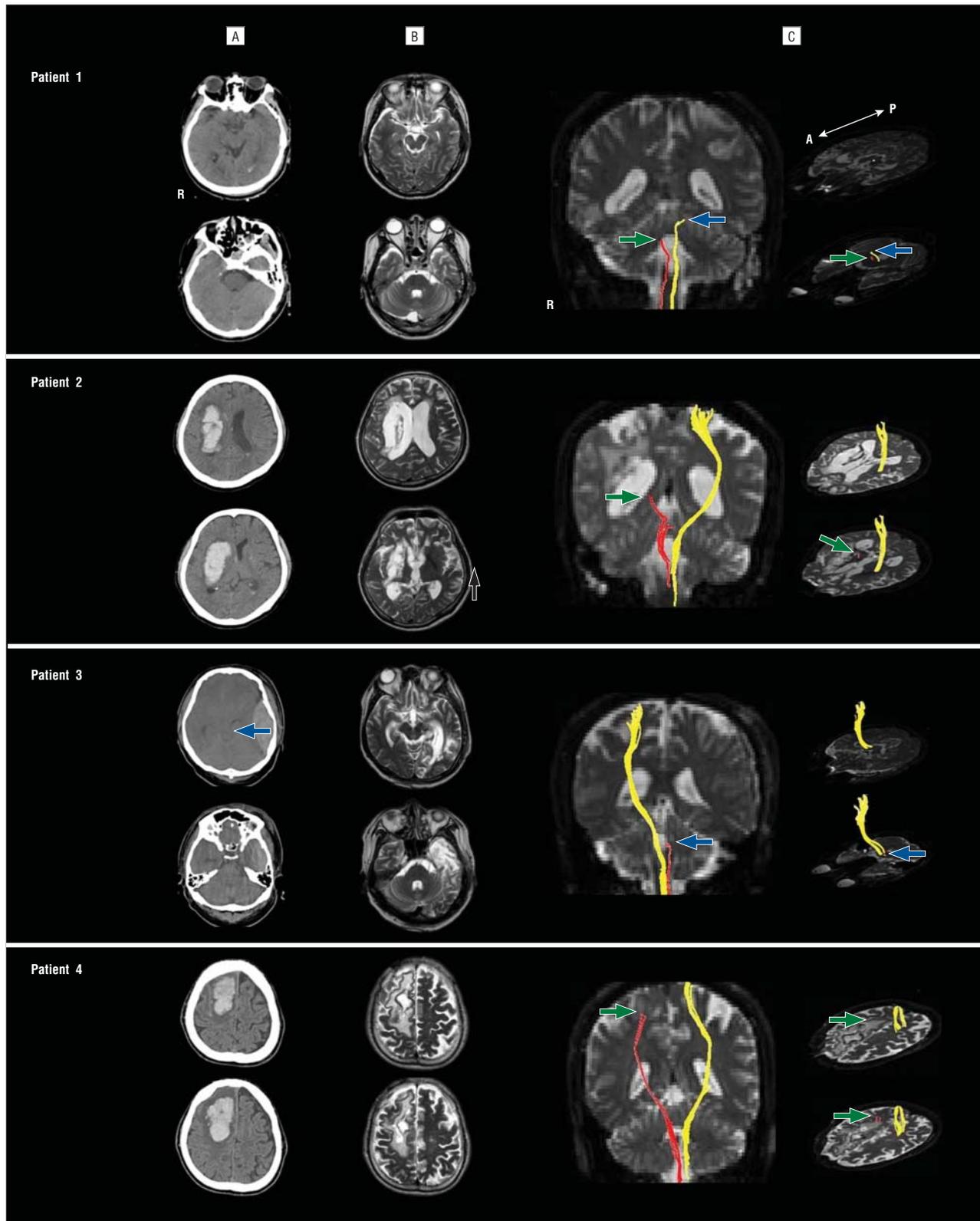


Figure. Patient 1: Brain computed tomography (CT) images at onset show no focal lesion (A). Brain T2-weighted images at 4 weeks after onset show no focal lesion (B). Diffusion tensor tractography images for the corticospinal tract show disruption at the right midpons (green arrow) and left cerebral peduncle (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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