

Extensive traumatic axonal injury of brain due to violence

A case report

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Abstract

Rationale: Many studies using diffusion tensor imaging (DTI) have demonstrated traumatic axonal injury (TAI) in patients whose conventional brain magnetic resonance imaging (MRI) results are negative following head trauma. Injury mechanism for TAI in these patients has been mainly associated with motor vehicle accident, whereas very little is known about TAI by violence.

Patient concerns: A 42-year-old male patient presented after experiencing head trauma due to violence. His face was hit several times by 2 men, and 1 of the men kicked the right side of the patient's head, after which the patient's left parietal area hit the ground while falling. After the head trauma, he felt mild motor weakness of the left upper and lower extremities and had mild articulation difficulty, cognitive dysfunction including memory impairment, and excessive daytime sleepiness.

Diagnoses: The patient was diagnosed as TAI.

Interventions: Clinical assessments and DTI were performed at 10 days after the head trauma.

Outcomes: He showed mild left hemiparesis (5/4), mild dysarthria, mild cognitive abnormality (Clinical Dementia Rating: 0.5) and mild abnormality on the Epworth Sleepiness Scale (score: 12; cut-off score: 10, maximum score: 24). DTI showed the following configurational abnormalities: right corticospinal tract narrowing, left corticobulbar tract narrowing, discontinuations in the anterior portion of both cingula, discontinuation of the left fornical crus, non-reconstruction of the right dorsolateral prefronto-thalamic tract, and narrowing in both lower ventral ascending reticular activating systems.

Lessons: Extensive TAI of various neural tracts was demonstrated by performing DTI of a patient with head trauma due to violence. Analysis of the neural tracts via DTI can be useful in detection of TAI in patients who show various neurological features following head trauma due to violence.

Abbreviations: ARAS = ascending reticular activating system, CBT = corticobulbar tract, CST = corticospinal tract, DTI = diffusion tensor imaging, fMRI = functional magnetic resonance imaging of the brain, MRI = magnetic resonance imaging, ROI = region of interest, TAI = traumatic axonal injury, TBI = traumatic brain injury.

Keywords: diffusion tensor imaging, traumatic axonal injury, traumatic brain injury, violence

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1. Introduction

Traumatic brain injury (TBI) is a common neurological disorder in the present era. Neural axons in the white matter are known to be vulnerable to head trauma due to mechanical loading of the brain during TBI.^[1–3] Traumatic axonal injury (TAI) is the tearing of axons due to indirect shearing forces during acceleration, deceleration, and rotation of the brain, or the result of direct head trauma.^[3,4–7] TAI has been demonstrated in many pathological studies by examining animal brain or post-mortem human brain.^[4,5,8] However, diagnosis of TAI in living patients has been limited because of the insufficient sensitivity of conventional brain magnetic resonance imaging (MRI) for detection of TAI in subjects with mild TBI.^[3,7,9]

Since the development of diffusion tensor imaging (DTI), which provides invaluable information about subcortical white matter that cannot be obtained via conventional MRI, TAI has been demonstrated in many DTI-based studies of TBI patients whose conventional brain MRI results are negative.^[9–16] Injury mechanisms for TAI in these patients have been associated with motor vehicle accidents (predominant mechanism), sport accidents, falling objects, falls, and so on.^[9,12–16] However, very little has been reported on the detection of TAI resulting from violence, even though it is important medically and legally.

In this study, we report on a patient who, following head trauma due to violence showed extensive TAI of various neural tracts on DTI.

2. Case report

A 42-year-old male patient presented after experiencing head trauma due to violence. His face was hit several times by 2 men. Subsequently, 1 of the men kicked the right side of the patient's head, after which the patient's left parietal area hit to the ground while falling. The patient experienced loss of consciousness and post-traumatic amnesia for 5 hours. The patient's Glasgow Coma Scale score was 15 when he arrived at the emergency room of a university hospital. He did not have any medical history of neurological or psychiatric disease, and no previous incidents of head trauma. After the head trauma, he experienced mild motor weakness of the left upper and lower extremities, mild articulation difficulties, cognitive dysfunction including memory impairment, and excessive daytime sleepiness. Ten days after the head trauma, he was admitted to the rehabilitation department of a university hospital. He showed mild left hemiparesis (5/4), mild dysarthria, mild cognitive abnormality (Clinical Dementia Rating = 0.5, range 0~3; higher score indicates more abnormality), and mild sleeping abnormality (Epworth Sleepiness Scale = 12, range 10~24; higher score indicates more sleepiness).^[17,18] No specific lesion was observed on brain MRI (T1-weighted, T2-weighted, and fluid-attenuated inversion recovery images) and electromyography did not detect any abnormality. Ethical approval for this study was provided by Institutional Review Board of Yeungnam university hospital (YUMC-2015-07-064) and the written informed consent for publication of the case details was obtained from the patient.

2.1. DTI

The DTI data were acquired 10 days after TBI onset by using a 6-channel head coil on a 1.5 T Philips Gyroscan Intera (Philips, Best, Netherlands) with single-shot echo-planar imaging. For each of the 32 non-collinear diffusion sensitizing gradients, 67 contiguous slices were acquired parallel to the anterior commissure-posterior commissure line. Imaging parameters were as follows: acquisition matrix = 96×96 , reconstructed to matrix = 192×192 , field of view = $240 \text{ mm} \times 240 \text{ mm}$, TR = 10,398 ms, TE = 72 ms, parallel imaging reduction factor (SENSE factor) = 2, echo-planar imaging factor = 59 and $b = 1000 \text{ s/mm}^2$, NEX = 1, and slice thickness = 2.5 mm.

2.1.1. Reconstruction of the corticospinal tract (CST), cingulum, and fornix. Fiber tracking was performed by using the fiber assignment continuous tracking (FACT) algorithm implemented within the DTI task card software (Philips Extended MR WorkSpace 2.6.3, Philips, Amsterdam, Netherlands). Each DTI replication was intra-registered to the baseline "b0" images to correct for residual eddy-current image distortions and head motion effects by using a diffusion registration package (Philips Medical Systems, Amsterdam, Netherlands) (threshold fractional anisotropy = 0.15, angle = 27). For reconstruction of the CST, the seed region of interest (ROI) was placed on the anterior blue portion of the upper pons on the axial image of the color map, and the second ROI was placed on the anterior blue portion of the lower pons on the axial image of the color map.^[14] For analysis of the cingulum, the seed and target ROI were placed on the middle and posterior portions, respectively, of the cingulum on the color

map of coronal images.^[19] Fiber tracking for the CST and cingulum was performed by using a fractional anisotropy of <0.15 and a trajectory angle threshold of $>27^\circ$. For reconstruction of the fornix, the seed ROI was placed at the junction between the body and column of the fornix, and the target ROIs were placed on each side of the crus of the fornix.^[19] Fornix fiber tracking was performed by using a fractional anisotropy threshold of >0.2 and a direction threshold $<45^\circ$.

2.1.2. Reconstruction of the corticobulbar tract (CBT), prefronto-thalamic tract, and lower ventral ascending reticular activating system (ARAS). The Oxford Centre for Functional Magnetic Resonance Imaging of the Brain (FMRIB) Software Library (www.fmrib.ox.ac.uk/fsl) was used to analyze diffusion-weighted imaging data. Affine multi-scale 2-dimensional registration was used to correct for the head motion effects and image distortion due to eddy currents. FMRIB Diffusion Software with the routine option (0.5 mm step lengths, 5000 streamline samples, curvature thresholds = 0.2) was used to reconstruct fiber tracking. For analysis of the CBT, the seed ROI was placed on the lower pons, and the target ROI was placed on the lower portion of the precentral gyrus and in the section at the top of the lateral ventricles.^[20] To reconstruct the prefronto-thalamic tract, the seed ROI was placed on the mediodorsal nucleus (MD), and target ROIs were on the dorsolateral prefrontal cortex (DLPFC), the ventrolateral prefrontal cortex (VLPFC), and the orbitofrontal cortex (OFC), respectively.^[21] To analyze the lower ventral ARAS, the seed ROI was placed on the pontine reticular formation and the target ROI on the hypothalamus.^[22] A threshold of 2 streamlines was applied to the fiber tracking results.

DTI showed the following configurational abnormalities: narrowing of the right CST and the left CBT, narrowing of both cingula, discontinuations in the anterior portion of the fornix and in left fornical crus, narrowing in both lower ventral ARAS, and non-reconstruction of the right dorsolateral prefronto-thalamic tract (Fig. 1B).

3. Discussion

In this study, abnormal findings (narrowing, discontinuation, and non-reconstruction) indicative of neural injuries were observed in a patient who suffered head trauma due to violence.^[3] The CST and CBT are reported to be mainly involved in motor control of extremities and articulation muscles, respectively.^[23,24] The fornix transfers episodic memory, whereas the cingulum and the dorsolateral prefronto-thalamic tract are involved in short-term memory.^[25-27] Recently, injury of the lower ventral ARAS, which is connected to the hypothalamus, was reported to result in excessive daytime hypersomnia.^[22] The clinical features recorded for the patient in this study appear to be associated with injury of each of those neural tracts as follows: mild left hemiparesis associated with injury of the right CST; mild dysarthria associated with injury of the left CBT; memory impairment associated with injuries of the fornix, both anterior cingula, and the dorsolateral thalamocortical tract; and excessive daytime sleepiness associated with injuries of both lower ventral ARAS. Because no specific lesion was observed on conventional brain MRI of this patient, it appears that the injuries of those neural tracts (i.e., CST, CBT, fornix, cingulum, and dorsolateral thalamocortical tract and lower ventral ARAS) indicate the presence of TAI.^[3,4,7,9]

In conclusion, by using DTI, extensive TAI was demonstrated in several neural tracts of a patient with head trauma due to

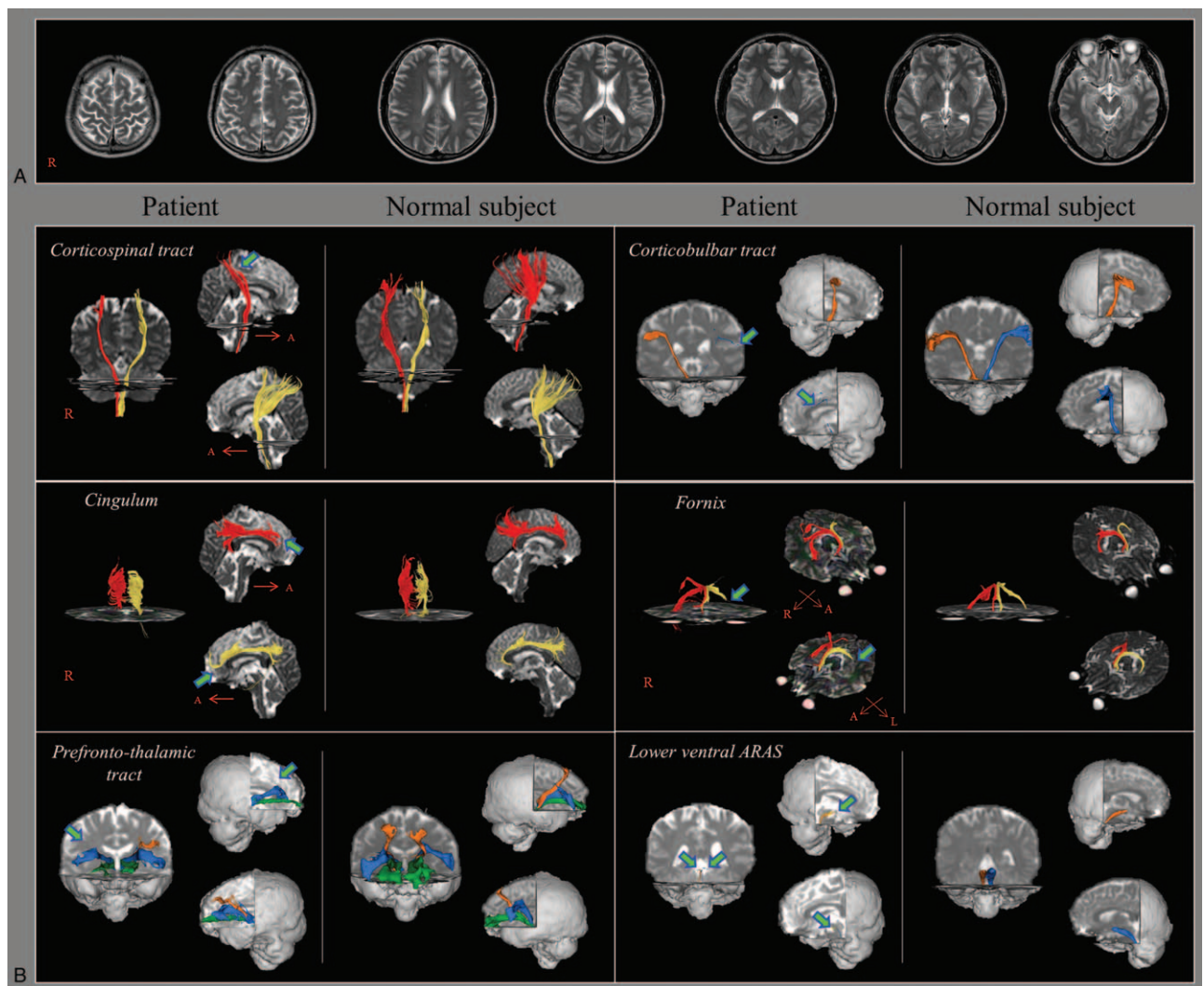


Figure 1. (A) T2-weighted brain magnetic resonance images at 10 days after traumatic brain injury onset show no abnormality. (B) Results of diffusion tensor tractography show the following configurational abnormalities: narrowing of the right corticospinal tract and left corticobulbar tract; discontinuities in the anterior portions of bilateral cingula; discontinuation in the left fornical crus; non-reconstruction of the right dorsolateral prefronto-thalamic tract; and narrowing in both lower ventral ascending reticular activating systems (abnormality locations indicated by arrows).

violence. The results suggest that analysis of the neural tracts via DTI can be useful in detection of TAI in patients who show various neurological features following head trauma due to violence. However, DTI has a significant limitation in reconstruction of some neural tracts due to the partial volume effect and crossing fibers, which can prevent full reconstruction of fiber and tract architecture.^[28] The individual patients who have been demonstrated TAI using DTI following head trauma were injured by motor vehicle accidents, sport accidents, falling objects, falls, and so on.^[9,12–16] By contrast, this is the first study to report TAI following head trauma due to violence as far as we are aware.

Author contributions

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