Clinical Case Report

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Limb-kinetic apraxia in a patient with mild traumatic brain injury

A case report

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Abstract

Rationale: We report on a patient who developed limb-kinetic apraxia (LKA) due to an injured corticofugal tract (CFT) from the secondary motor area following mild traumatic brain injury (TBI), demonstrated on diffusion tensor tractography (DTT).

Patient concerns: She was struck in the right leg by a sedan at a crosswalk and fell to the ground. She lost consciousness and experienced post-traumatic amnesia for approximately ten minutes. She was obliged to wear a cast for a left humerus fracture for two months, and she found she could not move her left hand quickly with intention after removal of the cast; consequently her left hand was almost non-functional. When she visited the rehabilitation department of a university hospital two years after the crash, she had mild weakness of the left upper extremity (manual muscle test: 4/5). However, the movements of the left hand were slow, clumsy, and mutilated when executing grasp-release movements of her left hand.

Diagnoses: A 44-year-old female suffered head trauma resulting from a pedestrian car accident.

Interventions: When she extended all her left fingers, it took approximately eight seconds at her fastest speed to perform the pattern extending from the thumb to little finger sequentially.

Outcomes: On two-year DTT, narrowing and partial tearing was observed in the right supplementary motor area (SMA)-CFT.

Lessons: Injury of the right SMA-CFT was demonstrated in a patient with LKA in a hand following mild TBI. Our results stress the need to evaluate the CFTs from the secondary motor area for patients with unexplained motor execution problems following mild TBI.

Abbreviations: CFT = corticofugal tracts, CST = corticospinal tract, DTI = diffusion tensor imaging, DTT = diffusion tensor tractography, LKA = limb-kinetic apraxia, PMC = premotor cortex, SMA = supplementary motor area, TBI = traumatic brain injury.

Keywords: corticofugal tract, diffusion tensor tractography, limb-kinetic apraxia, mild traumatic brain injury

1. Introduction

Injury of the corticofugal tracts (CFTs) from the secondary motor area including the premotor cortex (PMC) and supplementary motor area (SMA) is accompanied by limb-kinetic apraxia (LKA).[1] Precise diagnosis of LKA is important in neurorehabilitation because motor symptoms by LKA can be improved by intensive rehabilitation including dopaminergic drugs.[2–5] However, diagnosis of LKA depends mainly on clinical assessment, because assessment of the CFTs from the secondary motor area is limited with conventional brain MRI.

Diffusion tensor tractography (DTT), which is reconstructed from diffusion tensor imaging (DTI) data, allows 3-dimensional estimation of the CFTs from the secondary motor area.[6] Many studies using DTT demonstrated injury of the CFTs from the secondary motor area following brain injury and most of these studies focused on the stroke patients.[6–12] However, little is known about traumatic brain injury (TBI).[12]

In this study, we report on a patient with LKA due to an injured CFT from the secondary motor area following mild TBI, demonstrated on DTT.

2. Case report

A 44-year-old female with no history of neurological, physical, or psychiatric illness suffered head trauma resulting from a pedestrian car accident. She was struck in the right leg by a sedan at a crosswalk and fell to the ground. She lost consciousness and experienced post-traumatic amnesia for approximately 10 minutes. The patient's Glasgow Coma Scale score was 15. She was obliged to wear a cast for a left humerus fracture for 2 months, and she found she could not move her left hand quickly with intention after removal of the cast; consequently her left hand was almost nonfunctional. When she visited the rehabilitation department of a university hospital 2 years after the crash, she had mild weakness of the left upper extremity (manual muscle test: 4/5). However, the movements of the left hand were slow, clumsy, and mutilated when executing grasp-release movements of her left hand. When she extended all her left fingers, it took

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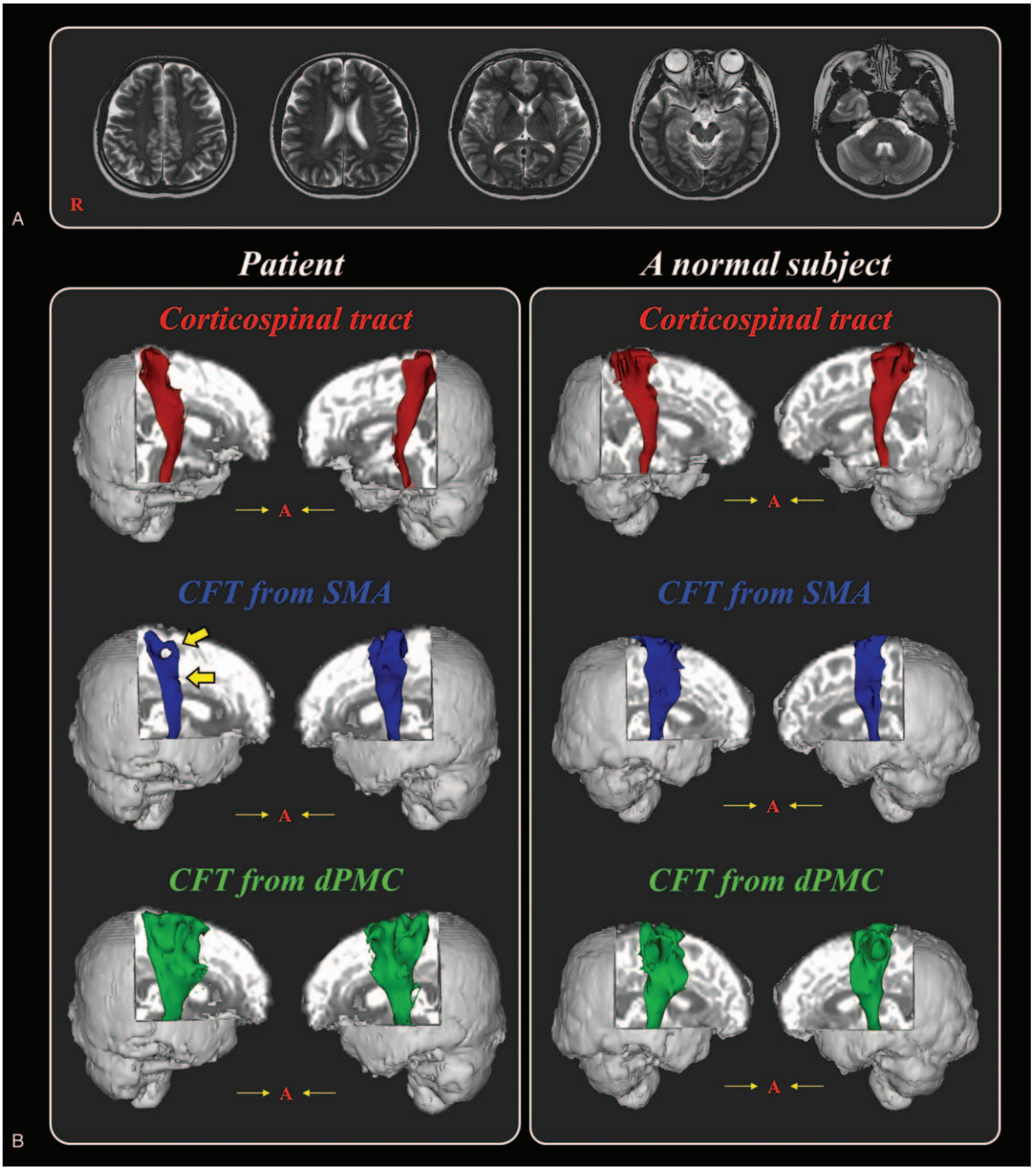

Figure 1. (A) T2-weighted brain MR images at 2 years after onset show no abnormality. (B) On 2-year diffusion tensor tractography, narrowing and partial tearing (arrows) is observed in the right supplementary motor areas-corticofugal tract. MR = magnetic resonance.

approximately 8 seconds at the fastest speed to perform a pattern extending from the thumb to little finger sequentially. No specific lesion was observed on brain MRI (T1-weighted, T2-weighted, and fluid attenuated inversion recovery [FLAIR] images) (Fig. 1A). Electromyography and nerve conduction studies for her left upper extremity were normal. Motor evoked potentials (MEPs) obtained from the abductor pollicis brevis muscle (APB) and tibialis anterior muscle (TA) were also normal (right APB-latency: 18.6 ms; amplitude: 2.7 mV, left APB-latency: 18.7 ms; amplitude: 2.3 mV).[13] Her scores on the Mini-Mental State Exam and the ideomotor apraxia test were 30 (cut-off score < 25) and 34 (cut-off score < 32), respectively.[14,15] After 3 month's administration of dopaminergic drugs for the improvement of apraxia (ropinorole, 3 mg; bromocriptine, 10 mg; levodopa, 375 mg), her left hand slowness recovered rapidly to the point that she was able to extend all fingers within 2 seconds.[2–4] In addition, the clumsiness and mutilated movement almost vanished. The patient provided signed, informed consent and our institutional review board approved the study protocol.

3. Diffusion tensor imaging

DTI data were acquired 2 years after the TBI using a 16-channel head coil on a 3.0 T MR scanner (Achieva TX, Philips, Ltd, Best, The Netherlands) with single-shot echo-planar imaging. For each of the 32 noncollinear diffusion sensitizing gradients, 70

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 matrix; field of view = 240 mm × 240 mm; TR = 10,398 ms; TE = 72 ms; parallel imaging reduction factor (SENSE factor) = 2; EPI factor = 59; $b = 1000\,s/mm^2$; NEX = 1; and a slice thickness of 2.5 mm (acquired isotropic voxel size 2.5 mm × 2.5 mm × 2.5 mm).

Fiber tracking was performed using the Oxford Centre for Functional Magnetic Resonance Imaging of the Brain (FMRIB) Software Library (FSL; www.fmrib.ox.ac.uk/fsl). Affine multi-scale 2-dimensional registration was used to correct head motion effect and image distortion due to eddy current. Fiber tracking was performed using a probabilistic tractography method based on a multifiber model, and applied in the current study utilizing tractography routines implemented in FMRIB Diffusion (5000 streamline samples, 0.5 mm step lengths, curvature thresholds = 0.2).[16] To analyze the corticospinal tract (CST), the seed region of interest (ROI) was placed on the middle pons, and the target ROI was placed on primary motor cortex. For the CFTs, the seed ROI was placed on the cerebral peduncle of the midbrain. The target ROI was defined as the dPMC (dPMC-CFT) and SMA (SMA-CFT) area at the cortex level.[11] Out of 5000 samples generated from each seed voxel, results for each contact were visualized threshold and weightings of tract probability at a minimum of 3 streamlines through each voxel for analysis.

On 2-year DTT, the integrity of both the CSTs, both the dPMC-CFT and the left SMA-CFT were well preserved. However, narrowing and partial tearing was observed in the right SMA-CFT.

4. Discussion

In this case report, we describe a patient with motor execution problems of the left hand following mild TBI. We think that these problems in this patient are mainly ascribed to LKA, for the following reasons. First, diagnosis of LKA is dependent mainly on clinical observation of movements.[1,7,17–19] The clinical characteristics of the left-hand movements (slow, clumsy, and mutilated) were compatible with those of LKA (awkward, clumsy, coarse, mutilated pattern of execution of simple movements that are confined mainly to movements of the affected hand). However, because the patient showed normal cognition for motor performance and a normal result on the ideomotor apraxia test, we were able to rule out ideational and ideomotor apraxia. Second, we confirmed that the right CST findings were normal in terms of MEP parameters (latency and amplitude) on transcranial magnetic stimulation and configuration on DTT.[20] Because the electromyography study and nerve conduction study of the left upper extremity revealed no abnormality, we could rule out peripheral nerve or CST pathology as causes of the motor problem. However, we found injury of the left SMA-CFT (narrowing and partial tearing). Therefore, we thought that the motor problem of the left hand was attributable mainly to the injury of the SMA-CFT. Third, dopaminergic drugs, effective for treatment of apraxia, appeared to be effective to improve of LKA.[2,3,5,21] The patient was prescribed dopaminergic drugs starting 2 years after the TBI without any other rehabilitative management, and her LKA symptoms almost disappeared. We think that this improvement with dopaminergic drugs is additional evidence of LKA.

In conclusion, injury of the right SMA-CFT was demonstrated in a patient with LKA in a hand following mild TBI. Since the introduction of DTI, several studies using DTT have reported on injury of the CFTs from the secondary motor area,[6–12] although the limitations of DTT should be considered, as regions of fiber complexity and fiber crossing in the subcortical white matter might cause false results.[22] To the best of our knowledge, only one study has reported on LKA in TBI.[12] In 2017, Jang and Seo[12] reported on a patient with mild motor execution problems (while driving his car, when he tried to turn to the right, he felt he tried to turn to the left and when he tried to brake, he could not do this action as smoothly as he intended) following mild TBI due to partial injury of both SMA-CFTs on DTT. Our results stress the need to evaluate CFTs from the secondary motor area for patients with unexplained motor execution problem following mild TBI.

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