

Recovery of an injured medial lemniscus with concurrent recovery of pusher syndrome in a stroke patient: a case report

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Abstract

Rationale: A 67-year-old, right-handed male patient underwent craniotomy and drainage for hematoma removal related to an intracerebral hemorrhage (ICH) in the right thalamus and basal ganglia at the neurosurgery department of a university hospital.

Patient concerns: He presented with severe motor weakness of left extremities, impairment of proprioception, and severe pusher syndrome at the start of rehabilitation.

Diagnoses: He was diagnosed as ICH in the right thalamus and basal ganglia.

Interventions: The patient received comprehensive rehabilitative therapy, movement therapy, and somatosensory stimulation.

Outcomes: Four months after onset, left leg motor function (Motricity Index [MI] = 51) did not show significant recovery from that at two months after onset (MI = 41); however, in the same period, Nottingham Sensory Assessment and scale for contraversive pushing significantly improved. At four months, the patient was able to stand independently but required manual contact of one person during independent walking on an even floor. At seven months after onset, he was able to walk independently on an even floor.

Lessons: Recovery of a severely injured medial lemniscus with concurrent recovery of impaired proprioception and pusher syndrome.

Abbreviations: DTI = diffusion tensor imaging, DTT = diffusion tensor tractography, FAC = functional ambulatory scale, ICH = intracerebral hemorrhage, ML = medial lemniscus, NSA = Nottingham Sensory Assessment, ROI = region of interest, SCP = scale for contraversive pushing, STT = spinothalamic tract.

Keywords: diffusion tensor tractography, intracerebral hemorrhage, medial lemniscus, pusher syndrome

1. Introduction

Pusher syndrome is defined as pushing away from the non-hemiparetic side with a shift of body weight toward the affected side.^[1,2] It is one of the postural imbalances that can manifest after a stroke.^[2] Previous studies have reported pusher syndrome incidence to be as high as 10.5% in acute stroke patients with hemiparesis.^[3] Compared with patients without pusher syndrome, patients with pusher syndrome need more time for recovery of physical function.^[1,4] Therefore, elucidation of the pathogenetic mechanism associated with pusher syndrome after stroke would be important for rehabilitation.

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Perception of body position can be achieved through somatosensory information.^[5] There are 2 main somatosensory tracts in human brain: the medial lemniscus (ML) and the spinothalamic tract (STT). The ML is responsible for proprioception, which is involved in the conscious awareness of body orientation in space, whereas the STT is the neural tract responsible for sensing pain and body temperature.^[6–8] Thus, among the somatosensory neural tracts, the ML might be most closely related to the pusher syndrome. Introduction of diffusion tensor tractography (DTT), which is derived from diffusion tensor imaging (DTI) data, has enabled three-dimensional reconstruction of the ML.^[6–9] Recent studies using DTT have demonstrated recovery of ML in patients who exhibit recovery of proprioception.^[8,9] However, very little has been reported on the association of the ML with pusher syndrome.

In this study, we report on a patient with an intracerebral hemorrhage (ICH) who showed recovery of an injured ML, demonstrated by DTT, and concurrent recovery of pusher syndrome.

2. Case report

A 67-year-old, right-handed male patient underwent craniotomy and drainage for hematoma removal related to an ICH in the right thalamus and basal ganglia at the neurosurgery department of a university hospital (Fig. 1A). At 1 week after onset, he was transferred to the rehabilitation department of the same university hospital to undergo rehabilitation. The patient provided signed informed consent; the study protocol was approved by our institutional review board.

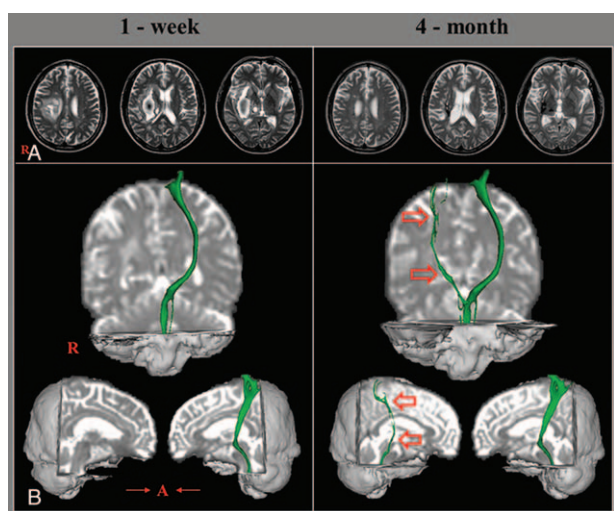


Figure 1. A, T2-weighted magnetic resonance images at 1 week and 4 months after stroke onset showing intracerebral hemorrhage on thalamus and basal ganglia in the right hemisphere. B, Diffusion tensor tractography (DTT) results. The right medial lemniscus (ML) was not reconstructed from 1-week postonset DTT results. In contrast, on 4-month postonset DTT, a thin ML was reconstructed in the right hemisphere (red arrow).

At onset, he presented with severe motor weakness of left extremities (motricity index [MI] for the left leg=0; full score=100), impairment of proprioception (Nottingham Sensory Assessment [NSA] proprioception score=8; full score=24), and severity of pusher syndrome (scale for contraversive pushing [SCP] score=5.5; full score=6; higher score indicates more severe pusher syndrome) at the start of rehabilitation (Table 1).^[2,10] The patient received comprehensive rehabilitative therapy, movement therapy, and somatosensory stimulation. Movement therapy and somatosensory stimulation focused on improvement of sensory-motor function and pusher syndrome of the left upper and lower extremities and was performed during physical and occupational therapy sessions 5 times per week for 4 months. Two months after onset, left leg weakness had recovered to MI=47; however, the patient did not show significant recovery of proprioception (NSA = from 8 to 10 points) or pusher syndrome (SCP = from 5.5 to 5.0 points). At 4 months after onset, motor function of the left leg did not show significant improvement (MI=51) from that at 2 months after onset (MI=47); however, over that period there were significant improvements in proprioception (NSA = from 10 to 20 points) and pusher syndrome (SCP = 5.0–2.3 points). As a result, at

Table 1
Changes in clinical data for intracerebral hemorrhage patient with pusher syndrome.

Clinical data	1 wk	2 mo	4 mo
MI (1–100)	9	41	49
NSA (1–24)	8	10	20
SCP (0–6)			
Total	5.5	5	2.25
Post	2	2	0.25
Ext	1.5	1.5	1
Resis	2	1.5	1

Ext=use of arm or leg to extend (max, 2), MI=motricity index, NSA=subscale for kinesthetic sensation of the Nottingham sensory assessment, Post=symmetry of spontaneous posture (max, 2), Resis=resistance to passive correction of posture (max, 2), SCP=scale for contraversive pushing.

4 months, the patient was able to stand independently but required manual contact with another person during independent walking on an even floor (functional ambulatory scale [FAC]=2). At 7 months after onset, he was able to walk independently on an even floor (FAC=3; required verbal supervision during walking).

3. Diffusion tensor tractography

DTI scans were obtained twice (at 1 week and 4 months after onset) by using a 6-channel head coil on a 1.5 T Philips Gyroscan Intera (Philips, Best, The Netherlands) with single-shot echo-planar imaging. For each of the 32 noncollinear diffusion sensitizing gradients, imaging parameters were as follows: acquisition matrix=96×96, reconstructed to a 192×192 matrix, field of view=240 mm×240 mm, repetition time=10,398 milliseconds, echo time=72 milliseconds, parallel imaging reduction factor (SENSE factor)=2, echo-planar imaging factor=59 and b=1000 s/mm², number of excitations=1, and slice thickness=2.5 mm. Analysis of diffusion-weighted imaging data was performed by using the Oxford Centre for Functional Magnetic Resonance Imaging of the Brain (FMRIB) Software Library (www.fmrib.ox.ac.uk/fsl). Fiber tracking was performed by using a probabilistic tractography method based on a multifiber model and was applied in the current study by utilizing tractography routines implemented in the FMRIB Diffusion Toolbox. For the analysis of the ML, the seed region of interest (ROI) was placed on the medial posterior region of the medullary pyramids. The target ROI was placed on the ventroposterolateral nucleus of the thalamus.^[7] A streamline threshold of was applied to obtain the fiber tracking results.

The DTT data for the right ML obtained at 1 week after onset was not reconstructed, whereas, on the 4-month DTT, a thin ML was reconstructed in the right hemisphere (Fig. 1B).

4. Discussion

In this study, we investigated changes in the ML of an ICH patient who showed concurrent recovery of impaired proprioception and severe pusher syndrome. The right ML appeared to be severely injured at 1 week because the right ML was not reconstructed from a 1-week DTT after onset. However, on the 4-month DTT, the right ML was thinly reconstructed which indicates partial recovery of the severely injured ML. Clinical assessment showed that motor function of the affected (left) leg was partially recovered at 2 months after onset; however, at that time, he could not stand because his proprioception and pusher syndrome had not shown sufficient recovery. At 4 months after onset, he could stand independently and walk at 7 months after onset following further recovery of proprioception and the pusher syndrome. His regaining of gait ability was closely related to the recovery of proprioception and pusher syndrome, indicating that recovery of impaired proprioception might be associated with the improvement of the pusher syndrome.

Many studies have tried to elucidate the pathogenetic mechanisms associated with pusher syndrome including disruption of somatosensory information, impaired somesthetic perception of vertical and visual problems, as well as motor and proprioception impairments.^[4,11–15] Among the pathogenetic mechanisms suggested for the pusher syndrome, a few studies have reported a subjective sense of body position and orientation. In 2000, Karnath et al^[11] investigated the angle of postural tilt in acute stroke patients with pusher syndrome. They

reported that patients with pusher syndrome indicated their body was upright when they were actually tilted by nearly 20° to the side.^[11] In 2006, Johannsen et al^[13] investigated leg to trunk orientation in 35 patients with pusher syndrome. The patients were tilted about 15° into the ipsiversive direction, and the nonparetic leg of the pusher syndrome patients showed a constant ipsiversive tilt across the whole tilt range for an amount which was observed in the nonpusher subject.^[13] The results of these previous studies indicate that the pusher syndrome is associated with a severe misperception of body orientation. Body orientation is related to bodily proprioception, which, in turn, is important for a sense of body position.^[15,16] Our results support those reported in the above studies.

In conclusion, recovery of a severely injured ML with concurrent recovery of impaired proprioception and pusher syndrome was demonstrated in a patient with ICH by using DTT and clinical assessments. Although 2 previous studies have demonstrated recovery of ML by using DTT, this is the first study to suggest that recovery of an injured ML may contribute to recovery of pusher syndrome in a stroke patient.^[8,9] However, this study has limitations as it is a single case report. Additional complementary studies with larger numbers of patients are needed.

Author contributions

Conceptualization: SungHo Jang.

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