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A change in injured corticospinal tract originating from the premotor cortex to the primary motor cortex in a patient with intracerebral hemorrhage★

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Abstract

Many studies have attempted to elucidate the motor recovery mechanism of stroke, but the majority of these studies focus on cerebral infarct and relatively little is known about the motor recovery mechanism of intracerebral hemorrhage. In this study, we report on a patient with intracerebral hemorrhage who displayed a change in injured corticospinal tract originating from the premotor cortex to the primary motor cortex on diffusion tensor imaging. An 86-year-old woman presented with complete paralysis of the right extremities following spontaneous intracerebral hemorrhage in the left frontoparietal cortex. The patient showed motor recovery, to the extent of being able to extend affected fingers against gravity and to walk independently on even ground at 5 months after onset. Diffusion tensor imaging showed that the left corticospinal tract originated from the premotor cortex at 1 month after intracerebral hemorrhage and from the left primary motor cortex and premotor cortex at 5 months after intracerebral hemorrhage. The change of injured corticospinal tract originating from the premotor cortex to the primary motor cortex suggests motor recovery of intracerebral hemorrhage.

Key Words: diffusion tensor imaging; corticospinal tract; motor recovery; reorganization

Abbreviations: ICH, intracerebral hemorrhage; CST, corticospinal tract; PMC, premotor cortex; DTI, diffusion tensor imaging; MEPs, motor-evoked potentials; ET, excitatory threshold

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INTRODUCTION

Because motor weakness is one of the most serious disabling sequelae of stroke, so it is important to elucidate the motor recovery mechanism for successful rehabilitation^[1-2]. Many studies have attempted to elucidate the motor recovery mechanism of stroke and several motor recovery mechanisms have been suggested^[3-13]. However, the majority of these studies focus on cerebral infarct and relatively little is known about the motor recovery mechanism of intracerebral hemorrhage (ICH)^[7, 14-18]. Diffusion tensor imaging (DTI) can help to investigate the motor recovery mechanism of ICH by enabling the direct visualization and estimation of the corticospinal tract (CST)^[7, 14-20]. Some recovery mechanisms have been suggested^[7, 14-20]. In this study, we report on a patient with ICH who showed a change in the origin of an injured CST from the premotor cortex (PMC) to the primary motor cortex (M1).

CASE REPORT

An 86-year-old woman presented with complete paralysis of the right upper and lower extremities, which occurred at the

onset of spontaneous ICH (Motricity Index (MI): 0 (full mark: 100)) (Table 1)^[21].

Table 1 Longitudinal changes in motor function

Duration from onset	Onset	After onset (month)		
		1	2	5
MRC				
Shoulder abductor	0	0	1	2
Elbow flexor	0	0	1	2
Finger flexor	0	0	1	3
Finger extensor	0	0	1	3
Hip flexor	0	0	1	3
Knee extensor	0	0	2	4
Ankle dorsiflexor	0	0	0	1
MI				
Upper extremity	0	0	30	50
Lower extremity	0	0	23	53
Total	0	0	27	52

MRC: Medical Research Council. 0: No contraction; 1: palpable contraction, but no visible movement; 2: movement without gravity; 3: movement against gravity; 4: movement against a resistance lower than the resistance overcome by the healthy side; 5: movement against a resistance equal to the maximum resistance overcome by the healthy side. MI: Motricity Index (range 0-100). The high score represented the mild motor impairment.

T2-weighted MR images showed a hematoma in the left fronto-parietal lobe, including M1, at 1 month after onset, that resolved at 5 months after onset (Figure 1A). From 1 month to 5 months after onset, she received comprehensive rehabilitative management, which included the administration of neurotrophic drugs (ropinirole, bromocriptine, levodopa, and amantadine), movement therapy, and neuromuscular electrical stimulation of the affected finger extensors and ankle dorsiflexors^[22-23]. Movement therapy focused on improving the motor functions of the right upper and lower extremities and included physical and occupational therapy sessions five times per week. Weakness of her left extremities improved from an MI score of 0 point at 1 month to 27 points at 2 months and to 52 points (5 months after ICH onset) after 4 months of rehabilitation. As a result, she was able to extend the affected fingers against gravity and to walk independently on even ground. The patient provided informed consent.

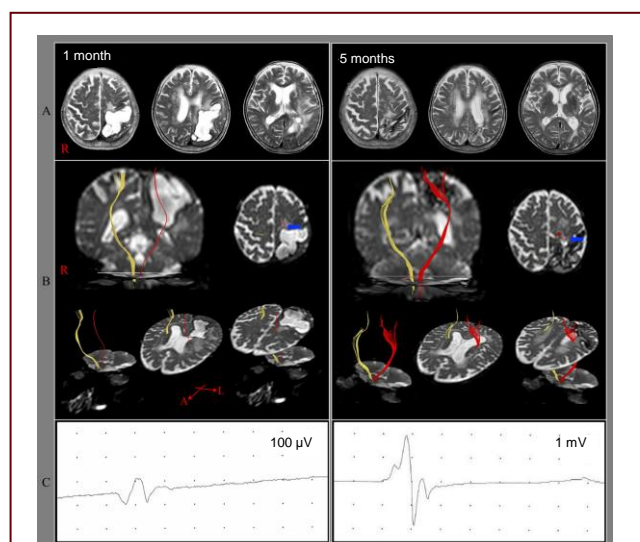


Figure 1 Brain magnetic resonance images, diffusion tensor imaging (DTI) and transcranial magnetic stimulation results of an 86-year-old female patient with a hematoma in the left fronto-parietal lobe.

(A) T2-weighted magnetic resonance images showed a hematoma in the left fronto-parietal lobe including the primary motor cortex at 1 month after onset, which was resolved at 5 months after onset.

(B) DTI findings of the corticospinal tract (CST). DTI results showed that at 1 month and 5 months after intracerebral hemorrhage, the right CSTs originated from the cerebral cortex, including the primary motor cortex, and then passed through the known CST pathway. In the left (affected) hemisphere, the CSTs originated from the left premotor cortex at 1 month after onset, and from the left primary motor cortex and premotor cortex at 5 months after onset.

(C) Transcranial magnetic stimulation results. A motor evoked potential of low amplitude (100 μ V) was obtained from right abductor pollicis brevis muscle by the affected (left) hemisphere stimulation at 1 month after onset. However, motor evoked potential amplitude was increased to the normal range (3.8 mV) at 5 months after onset.

Clinical evaluation

ICH volume was measured on CT image and T2-weighted MRI images through Picture Archived Communication System (PACS, Marotech, Korea). The maximum width (X), length (Y) and height (Z) of lesion at the level where a hematoma was clearly seen were measured^[24]. The ICH volume was calculated according to the formula:

$$\text{ICH volume (mV)} = 4/3 \times \pi \times X (\text{cm}) \times Y (\text{cm}) \times Z (\text{cm}) \times 1/16$$

The volume of ICH was 37.19 mV on the CT images at the ICH onset and 26.71 mV on the MR images at 1 month after onset.

DTI examination

Diffusion tensor images were acquired using a synergy-L sensitivity encoding (SENSE) head coil on a 1.5-T Philips Gyroscan Intera system (Hoffman-LaRoche, Mijdrecht, the Netherlands) using a single-shot echo planar imaging with a navigator echo. For each of the 32 noncollinear diffusion-sensitizing gradients, 60 contiguous slices (matrix = 192 \times 192, field of vision = 240 mm, repetition time/echo time = 10 726/76 ms, b = 600 mm^2/s , number of excitations = 1, thickness = 2.5 mm) were acquired. Three-dimensional reconstructions of fiber tracts were obtained using the DTI task card software (Philips Extended MR Work Space 2.6.3) (threshold fractional anisotropy (FA) = 0.15, angle = 45 $^\circ$)^[25]. Fiber tracts passing through both region of interests (CST portion of the anterior mid-pons and low-pons on the color map) were designated final tracts of interest.

At 1 month and 5 months after ICH onset, DTI results showed that in the right (non-affected) hemisphere, the right CST originated from the cerebral cortex (including M1) and passed through the known CST pathway. However, in the left (affected) hemisphere, the CST originated from the left PMC at 1 month and from the left M1 and PMC at 5 months (Figure 1B).

Transcranial magnetic stimulation (TMS)

TMS was performed using a Magstim Novamatrix 200 magnetic stimulator equipped with a 9-cm mean diameter circular coil (Novamatrix Medical Systems Inc, Wallingford, CT, USA). Cortical stimulation was then performed with the coil held tangentially over the vertex. The left hemisphere was stimulated by a counterclockwise current and the right hemisphere was stimulated by a clockwise current. Motor-evoked potentials (MEPs) were obtained from both abductor pollicis brevis muscles in a relaxed state. Excitatory threshold (ET) was defined as the minimum stimulus required to elicit an MEP with a peak-to-peak amplitude of 50 μ V or greater during two of four attempts. Stimulation intensity was set at the ET plus 20% of the maximum stimulator output. Each site was stimulated three times with inter-stimulus intervals of \geq 10 seconds, and shortest latency and average peak to peak

amplitude were estimated.

MEP (latency: 23.5 ms; amplitude: 100 μ V; ET: 100%) was evoked from the affected (left) hemisphere during the TMS study conducted at 1 month after ICH onset, but the amplitude increased to the normal range at 5 months after ICH onset (latency: 19.7 ms; amplitude: 3.8 mV; ET: 70%) (Figure 1C).

DISCUSSION

In this patient, we investigated changes of the injured left CST and found that the injured CST seemed to recover as detected by DTI, TMS and clinical observation. At 1 month after ICH onset, the left CST originated from the PMC by DTI, and the MEP obtained at the right hand showed low amplitude and a latency compatible with that of the CST^[26-27]. By contrast, 5-month results revealed that the left CST originated from the left M1 by DTI and that MEP amplitude had improved to the normal range by TMS^[26-27]. The M1 is the major origin of the CST, although the CST is known to originate from the extensive cerebral cortex, including the PMC, the primary somatosensory cortex, and the prefrontal cortex. On the other hand, the amplitude of the MEP reflects fiber numbers in the CST. Consequently, the DTI and TMS results of this patient suggest that the severely injured left CST had recovered over 4 months in terms of its origin and fiber number^[9, 28-29]. Furthermore, clinically, the finding that the patient was able to flex and extend against gravity at 5 months after ICH onset provides additional evidence of left CST recovery^[30-31]. As a result, right extremity motor functions seemed to have been recovered due to reorganization of the injured left CST originating from the left PMC to left M1. It is also possible that compression of the CST into the left PMC recovered in concert with hematoma resolution.

For the motor recovery mechanism in ICH, several DTI based studies have described recovery of an injured CST or the contribution made to recovery by the contralateral unaffected CST^[7, 14-18]. During the last 5 years, a number of serial DTI studies have described the recovery of injured CST after ICH^[14, 17-18]. Jang *et al*^[15] demonstrated the recovery process of an injured CST in a patient with a subcortical ICH by serial DTI. The patient presented with complete paralysis of right extremities at onset, but over 16 months, motor functions of affected extremities slowly recovered to nearly normal. Furthermore, DTT showed that the origin of the CST had changed from the posterior parietal cortex at 1 month to the primary somatosensory cortex at 4 months and M1 at 16 months. The authors suggested that recovery of the origin of the damaged CST was due to a process of normalization from the parietal cortex to M1. By contrast, our patient showed a change in the origin of the injured CST from the PMC to M1.

This study described changes of an injured CST that occurred in concert with motor recovery in a patient with ICH. To the best of our knowledge, this is the first

longitudinal study to demonstrate a change in the origin of an injured CST from the PMC to M1 in ICH. Results from this study suggest a motor recovery mechanism of ICH and the important implications regarding brain plasticity and brain rehabilitation after ICH. However, this study is obviously limited by case numbers, and further complementary studies involving larger case numbers are warranted.

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Author contributions: Sang Seok Yeo contributed to data acquisition, manuscript development and oversight, technical support, data acquisition, and manuscript writing. Sung Ho Jang was responsible for conceiving and designing the study, manuscript development, funding, and manuscript authorization.

Conflicts of interest: None declared.

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