

Peri-infarct reorganization of an injured corticospinal tract in a patient with cerebral infarction

Min Kyeong Cho, Sung Ho Jang*

Corticospinal tract (CST), a major neural tract in the human brain for motor function, is involved mainly in the movement of the distal extremities (Jang and Lee, 2019). Recovery of an injured CST is essential for good recovery of impaired motor function in stroke patients (Jang and Lee, 2019). Peri-infarct reorganization of an injured CST is an important mechanism underlying recovery of motor function in stroke patients (Jang, 2007). In this study, we reported on a patient with cerebral infarction who showed recovery of an injured CST by peri-infarct reorganization using diffusion tensor tractography (DTT) and transcranial magnetic stimulation (TMS).

A 57-year-old, right-handed male patient who was admitted to the Rehabilitation Department of Yeungnam University Hospital presented with right hemiplegia due to an infarct in the left corona radiata (CR; **Figure 1A**). He had histories of cerebral infarction in the left pontine tegmentum and anterior CR at 15 years and 3 months earlier, respectively; however, he had recovered almost completely without sequelae from those previous infarcts. When he started rehabilitation at 2 weeks after the most recent infarction, he had severe weakness of the right extremities (Motricity Index [MI]: 28 points; full score: 100 points) and complete weakness of the right hand (finger flexors and extensors) (Medical Research Council [MRC]: 0 point; full score: 5 points) (Demeurisse et al., 1980; Gregson et al., 2000). At 2–6 weeks after cerebral infarction onset, he underwent comprehensive rehabilitative therapy, including movement therapies provided by physical and occupational therapists (motor strengthening of the right upper and lower extremities, and exercises for trunk stability and control, static and dynamic balance training on sitting and standing positions, twice a day, 40 minutes once, 5 days per week), took neurotrophic drugs (Pramipexole, Ropinirole, Amantadine sulfate, Levodopa, Bromocriptine), neuromuscular electrical stimulation for the right finger extensors and ankle dorsiflexors, repetitive TMS therapy using a MAGPRO stimulator (Medtronic Functional Diagnostics, Skovlunde, Denmark)

with the device's left precentral knob set at a frequency of 10 Hz, intensity of 80% motor threshold, and 160 pulses for 8 minutes. At 4 weeks after cerebral infarction onset, his right hemiparesis recovered to an MI score of 41 points (right finger flexors and extensors: 2/5) with further recovery to an MI score of 64 points (right finger flexors and extensors: 3/5) at 8 weeks after onset. At that time, he was able to perform grasp-release movements using his right hand and to walk independently.

A 6-channel head coil on a 1.5T Philips Gyroscan Intera (Philips, Ltd., Best, the Netherlands) with 32 gradients and single-shot echo-planar imaging was used to acquire diffusion tensor imaging data. On 2-week post-onset DTT, the left CST (fiber number: 1299) was visualized as almost discontinuous with only a small fiber connection to the cerebral cortex passing through the posterior portion of the infarct lesion in the CR. The severely limited continuity of the left CST was shown as restored on the 4-week post-onset DTT (fiber number: 1387). The tract was notably thicker on the 8-week post-onset DTT (fiber number: 1774) (**Figure 1B**). Signed informed consent was obtained from the patient. The study protocol was approved by the Institutional Review Board of Yeungnam University Hospital, Republic of Korea (YUMC 2019-06-032) on June 21, 2019.

TMS was performed using a Magstim Novamatrix 200 magnetic stimulator with a circular coil with the mean diameter of 9 cm (Novamatrix Medical Systems Inc, Wallingford, CT, USA). On 2-week post-onset TMS, motor evoked potentials (MEPs) were not detected for the right abductor pollicis brevis muscle. By contrast, MEPs were obtained on 4-week TMS (mean latency 25.3 ms and amplitude 100 μ V) and the amplitude was further increased on 8-week post-onset TMS (mean latency 25.2 ms and amplitude 300 μ V) (**Figure 1C**).

In this patient, the hand-associated somatotopic fibers of the severely injured left CST had recovered via peri-infarct reorganization which indicates the transfer of motor function into adjacent areas of an

infarct through the posterior portion of the infarct lesion. We concluded the following: First, the infarct location of the left CR corresponded to the hand somatotopic area for the left CST. Second, complete weakness of the right hand at 2 weeks after onset was recovered sufficiently to allow grasp-release movements at 8 weeks after onset. Third, restoration of the almost discontinued left CST was indicated by increased left CST fiber numbers on serial DTTs performed during the 8 weeks after onset. Fourth, MEP recovery of the affected hand was based on the visualization of an MEP at 4 weeks after onset and the presence of increased MEP amplitude at 8 weeks after onset. Fifth, this mode of progression indicates that this motor recovery could be ascribed to brain plasticity, and not to the resolution of local factors such as edema which usually occurs within 1–2 weeks after onset (Furlan et al., 1996; Witte, 1998). Taken together, the recovery indicated by DTT and MEP results for the left CST appears to be consistent with the concurrent motor function recovery of his right hand (Rossini et al., 1998; Cramer et al., 2000; Jaillard et al., 2005; Grefkes and Ward, 2014; Jang et al., 2015; Tennant et al., 2015; Zhang et al., 2015; Jang and Jang, 2016; Jang and Seo, 2018). Although a few studies have reported on peri-infarct reorganization to the posterior area of CR infarct, this study has unique characteristics to demonstrate the recovery process using serial DTT and TMS from the early stage to chronic stage of CR infarct (Jang et al., 2015; Kwon et al., 2007). Our findings about the motor recovery mechanism in stroke from the current study can provide useful information for planning specific rehabilitation strategies, estimating the duration of rehabilitation, and predicting the prognosis.

Min Kyeong Cho, Sung Ho Jang*

Department of Physical Medicine and Rehabilitation, College of Medicine, Yeungnam University, Namku, Daegu, Republic of Korea

*Correspondence to: Sung Ho Jang, MD, strokerehab@hanmail.net.

<https://orcid.org/0000-0001-6383-5505> (Sung Ho Jang)

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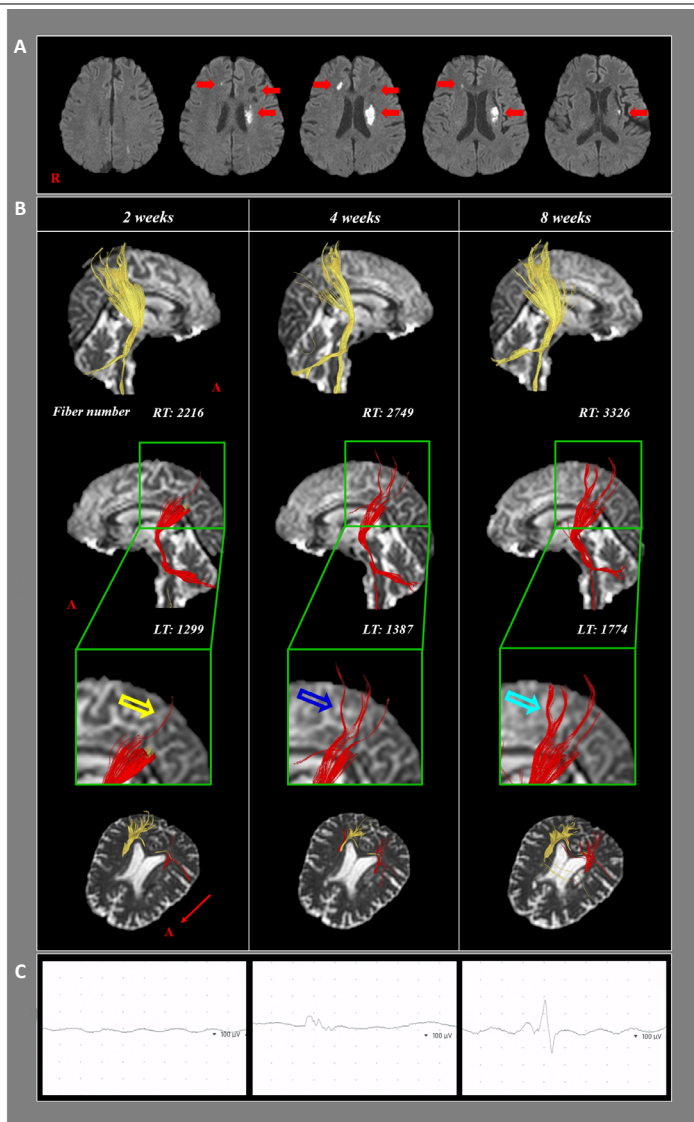


Figure 1 | Results of diffusion tensor tractography for peri-infarct reorganization of an injured corticospinal tract in a 57-year-old, right-handed male patient with cerebral infarction.

(A) T2-weighted magnetic resonance images obtained 2 days after cerebral infarction onset show infarctions in the left corona radiata and the right prefrontal lobe and a leukomalactic lesion (red arrows) in the left anterior corona radiata. (B) Results of diffusion tensor tractography (DTT). On 2-week DTT, the left corticospinal tract (CST) is almost discontinuous with the presence of only a small fiber connection to the cerebral cortex (yellow arrow) passing through the posterior portion of the infarcted lesion in the corona radiata. This severely limited continuity of the left CST is shown as partially restored on the 4-week DTT (blue arrow) and notably thickened on the 8-week DTT (sky-blue arrow). (C) Results of transcranial magnetic stimulation (TMS). On 2-week post-onset TMS, a motor evoked potential for the right abductor pollicis brevis muscle is not present. By contrast, a motor evoked potential is present on the 4-week TMS (latency 25.3 ms and amplitude 100 μ V) and its amplitude was increased on the 8-week TMS (latency 25.2 ms and amplitude 300 μ V). LT: Left; RT: right.

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Declaration of patient consent: Both authors certify that they have obtained the appropriate

patient consent form. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that his name and initial will not be published and due efforts will be made to conceal his identity.

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References

- Cramer SC, Moore CI, Finklestein SP, Rosen BR (2000) A pilot study of somatotopic mapping after cortical infarct. *Stroke* 31:668-671.
- Demeurisse G, Demol O, Robaye E (1980) Motor evaluation in vascular hemiplegia. *Eur Neurol* 19:382-389.
- Furlan M, Marchal G, Viader F, Derlon JM, Baron JC (1996) Spontaneous neurological recovery after stroke and the fate of the ischemic penumbra. *Ann Neurol* 40:216-226.
- Grefkes C, Ward NS (2014) Cortical reorganization after stroke: how much and how functional? *Neuroscientist* 20:56-70.
- Gregson JM, Leathley MJ, Moore AP, Smith TL, Sharma AK, Watkins CL (2000) Watkins, Reliability of measurements of muscle tone and muscle power in stroke patients. *Age Ageing* 29:223-228.
- Jaillard A, Martin CD, Garambois K, Lebas JF, Hommel M (2005) Vicarious function within the human primary motor cortex? A longitudinal fMRI stroke study. *Brain* 128:1122-1138.
- Jang SH (2007) A review of motor recovery mechanisms in patients with stroke. *NeuroRehabilitation* 22:253-259.
- Jang SH, Jang WH (2016) Recovery of an injured corticospinal tract by subcortical peri-lesional reorganization in a patient with intracerebral hemorrhage. *Neural Regen Res* 11:1191-1192.
- Jang SH, Lee J, Seo JP (2015) Reorganization of the corticospinal tract to anterior area of corona radiata infarct. *Int J Stroke* 10:e76-77.
- Jang SH, Lee SJ (2019) Corticoreticular tract in the human brain: A mini review. *Front Neurol* 10:1188.
- Jang SH, Seo JP (2018) Perilesional reorganization in a patient with brain tumor. *Am J Phys Med Rehabil* 97:e31-32.
- Kwon YH, Lee CH, Ahn SH, Lee MY, Yang DS, Byun WM, Park JW, Jang SH (2007) Motor recovery via the peri-infarct area in patients with corona radiata infarct. *NeuroRehabilitation* 22:105-108
- Rossini PM, Caltagirone C, Castriota-Scanderbeg A, Cicinelli P, Del Gratta C, Demartin M, Pizzella V, Traversa R, Romani GL (1998) Hand motor cortical area reorganization in stroke: a study with fMRI, MEG and TCS maps. *Neuroreport* 9:2141-2146
- Tennant KA, Kerr AL, Adkins DL, Donlan N, Thomas N, Kleim JA, Jones TA (2015) Age-dependent reorganization of peri-infarct "premotor" cortex with task-specific rehabilitative training in mice. *Neurorehabil Neural Repair* 29:193-202
- Witte OW (1998) Lesion-induced plasticity as a potential mechanism for recovery and rehabilitative training. *Curr Opin Neurol* 11:655-662
- Zhang M, Lin Q, Lu J, Rong D, Zhao Z, Ma Q, Liu H, Shu N, He Y, Li K (2015) Pontine infarction: diffusion-tensor imaging of motor pathways-a longitudinal study. *Radiology* 274:841-850.

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