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Gait recovery by recovery of an injured corticospinal tract in a patient with a cerebral infarct

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Case Report

A 81-year-old, right-handed female presented with complete weakness of the left upper and lower extremities due to infarcts in the right anterior and middle cerebral artery territories (Figure 1). She was transferred to the rehabilitation department of a university hospital at 3 weeks after onset. She showed complete weakness of the left upper and lower extremities (The Manual Muscle Test [MMT]: upper; 0/5, lower; 0/5). Brain MRI at 3 weeks after onset showed extensive infarct lesions in the right primary motor cortex except for a small portion of leg somatotopy, premotor cortex, and supplementary motor area (Figure 1A).

During a period of 9 weeks, from 3 to 12 weeks after onset, she underwent comprehensive rehabilitative therapy, including administration of neurotrophic drugs (venlafaxine, pramipexole, ropinirole, levodopa, and amantadine), neuromuscular electrical stimulation for the right hip extensors and ankle dorsiflexors, repetitive transcranial magnetic stimulation therapy ([MAGPRO, Medtronic Functional Diagnostics, Skovlunde, Denmark]: the leg somatotopy of the right primary motor cortex, frequency of 10 Hz, intensity of 80% motor threshold, and a total of 160 pulses for 8 minutes, and seven sessions per week), and movement therapy performed during the physical and occupational therapy sessions five times per week (70 minutes/day) [1-4]. At 12 weeks after onset, the proximal muscles of her left leg were recovered to movement against gravity (MMT: hip flexor; 3/5, knee extensor; 3/5, ankle; 0/5). As a result, she was able to walk independently on an even floor with fitting ankle foot orthosis. The patient provided signed, informed consent and our institutional review board approved the study protocol.



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DTI data were acquired two times (3 weeks and 12 weeks after onset) using a sensitivity-encoding head coil on a 1.5-T Philips Gyroscan Intera (Hoffman-LaRoche Ltd, Best, The Netherlands) with single-shot echo-planar imaging and navigator echo. Imaging parameters were as follows: acquisition matrix=96×96; reconstructed to matrix=192×192matrix; field of view=240×240mm²; TR= 10,398ms; TE= 72ms; parallel imaging reduction factor=2; EPI facto=59; b=1000s/mm²; and a slice thickness of 2.5mm. Fiber tracking was performed using the Fiber Assignment Continuous Tracking (FACT) algorithm implemented within the DTI task card software (Philips Extended MR Work Space 2.6.3). Each DTI replication was intra-registered to the baseline "b0" images to correct for residual eddy-current image distortions and head motion effect, using a diffusion registration package (Philips Medical Systems). For reconstruction of the Corticospinal Tract (CST), the first Region Of Interest (ROI) was placed on the upper pons (portion of anterior blue color) on the color map with an axial image. The second ROI was placed on the mid pons (portion of anterior blue color) on the color map with an axial image. The termination criteria used for fiber tracking were FA<0.1, angle<27° [5]. For analysis, the Corticoreticulospinal Tract (CRT) was reconstructed using fibers passing through two Regions Of Interest (ROIs) on the color map. The first Region Of Interest (ROI) was placed on the reticular formation of the medulla, and the second ROI was placed on the tegmentum of the midbrain. The termination criteria used for fiber tracking were FA<0.1, angle<27° [6].

On 3-week Diffusion Tensor Tractography (DTT), the right CST was discontinued at the subcortical white matter below the cerebral cortex and a small portion of the CRT fibers was connected to the cerebral cortex. By contrast, on 12-week DTT, the discontinued right CST was thickened and extended to the cerebral cortex, however, the right CRT was discontinued at the subcortical white matter without significant change of the left CST and CRT (Figure 1B).

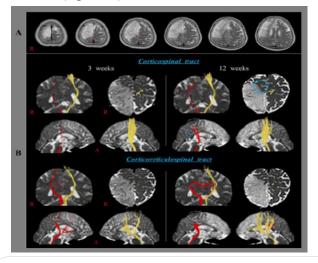


Figure 1: (A) T2-weighted images at three weeks after onset show an infarct in the right anterior and middle cerebral artery territories (extensive infarct lesions in the right primary motor cortex except for a small portion of leg somatotopy [arrows], premotor cortex, and supplementary motor area).

(B) Results of diffusion tensor tractography (DTT). On 3-week DTT, the right corticospinal tract (CST) is discontinued at the subcortical white matter below the cerebral cortex and a small portion of the corticoreticulospinal tract (CRT) fibers is connected to the cerebral cortex. By contrast, on 12-week DTT, the discontinued right CST is thickened (arrows) and extends to the cerebral cortex (circle), however, the right CRT is discontinued at the subcortical white matter without significant change of the left CST and CRT.

At 3 weeks after onset when the patient started rehabilitation, she showed complete weakness of her left upper and lower extremities. We found that the right primary motor cortex except for a small portion of the leg somatotopy, premotor cortex, and supplementary motor area were completely injured and the right CST was discontinued below the cerebral cortex. We assumed that the leg somatotopy of the left CST was partially injured and accompanying severe limb-kinetic apraxia was observed in this patient [7-9]. Considering these findings, we set the goal for this patient as independent walking even though the patient was too old and had extensive brain lesion. After 9 week's intensive and comprehensive rehabilitation for recovery of the partially injured right CST and limb-kinetic apraxia including neurotrophic drugs and repetitive transcranial magnetic stimulation therapy, her left leg showed significant motor recovery, so that she was able to walk independently with ankle foot orthosis. On the changes of DTTs, the left CST showed recovery (thickening and extension to the cerebral cortex) while the left CRT showed degeneration without change of the right CST and CRT. As a result, the motor recovery of her left leg appeared to be mainly attributed to the recovery of the injured right CST. In addition, the improvement of limb-kinetic apraxia due to injury of the right premotor cortex and supplementary motor area by dopaminergic drugs might also be attributed in part to recovery of motor weakness in this patient [8-11].

Conclusion

In conclusion, recovery of an injured CST concurrent with gait recovery was demonstrated in a patient with a cerebral infarct using DTT. Our results suggest the importance of precise goal setting at the start of rehabilitation, and intensive and comprehensive rehabilitation during the recovery phase even if the patient is too old and has an extensive lesion [12-14]. However, limitation of DTT should be considered: DTT may underestimate the neural tracts due to regions of fiber complexity and crossing [15].

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