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CASE REPORT

DELAYED RECOVERY OF GAIT FUNCTION IN A PATIENT WITH INTRACEREBRAL HAEMORRHAGE

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Objective: **We report here on a patient with intracerebral haemorrhage (ICH), who showed unusually delayed motor recovery of the leg, which started at 6 months after ICH onset.**

Case report: **A 53-year-old man underwent craniotomy and drainage for a right putaminal ICH. The patient presented with complete paralysis of the left extremities at ICH onset. Despite having undergone rehabilitation starting at 7 weeks after onset, the patient did not show significant motor recovery of the left extremities until 6 months after onset, when the affected left leg began to show motor recovery to the point that he was able to extend his hip and knee without gravity 7 months after onset. As a result, he was able to walk independently on an even floor 9 months after onset.**

Results: **On 6-month diffusion tensor tractography, the right corticospinal tract showed disruption with Wallerian degeneration to the pontomedullary junction.**

Conclusion: **This case study highlights the extensive potential for the human brain to aid in the recovery of walking after injury. In conclusion, clinicians should attempt to determine the causes of difficulty walking when examining patients with ICH and then perform intensive rehabilitation for the recovery of walking.**

Key words: diffusion tensor imaging; gait; motor recovery; corticospinal tract.

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INTRODUCTION

The brain has the characteristic of plasticity, which is defined as the ability of neuronal reorganization after brain injury. Brain plasticity can occur actively for a limited period of time following brain injury. In general, most motor recovery following stroke is known to occur within 3–6 months after onset; therefore, this period has been regarded as critical for motor recovery in stroke patients (1–3). Some studies have demonstrated long-term motor recovery over a period which started within the critical period (3–6 months) after onset of stroke (4, 5). However, little is known about motor recovery that begins after the critical period in stroke patients except for intervention-induced motor recovery during short-term periods.

We report here on a patient with an intracerebral haemorrhage (ICH), who showed unusually delayed motor recovery of the leg, which started 6 months after ICH onset.

CASE REPORT

A 53-year-old, right-handed man underwent craniotomy and drainage for right putaminal ICH at a hospital department of neurosurgery. The patient presented with complete paralysis of the left arm and leg at onset of ICH (Table I). The patient was diagnosed with heterotopic ossification in the left anterior upper thigh and inguinal area 2 weeks after ICH onset. The patient received rehabilitative management for his left hemiplegia at the rehabilitation department of Yeungnam University Hospital (for 3 weeks) and at a local rehabilitation hospital (for approximately 3 months) from 7 weeks to 6 months after onset. However, no significant motor recovery of the left leg was observed. Seven weeks after onset, spasticity of the left elbow and knee on the modified Ashworth scale were $G1^{+}$ and G1, respectively (6). Tactile sensation scores for the Semmes-Weinstein monofilaments of the right and left hands were 3.22 and 6.65, respectively (7). The patient wished to be readmitted to the rehabilitation department of Yeungnam University hospital in order to undergo repeat rehabilitation. He underwent com-

Table I. *Medical Research Council data for the patient*

	Duration from onset			
	2 months	6 months	7 months	9 months
Shoulder abductor	1	2	2	2
Elbow flexor	1	1	2	2
Finger flexor	0	0	0	0
Finger extensor	0	0	0	0
Hip extensor	0	0	2	3
Knee extensor	0	0	2	3
Ankle dorsiflexor	0	0	0	0

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.

prehensive rehabilitative management, including administration of neurotrophic drugs (pramipexole, 1.5 mg; bromocriptine, 20 mg; amantadine, 200 mg), movement therapy and neuromuscular electrical stimulation of the affected finger extensors and ankle dorsiflexors. Movement therapy focused on improvement in the motor function of the left hemiplegia and was performed 5 times per week. Motor weakness of the left leg showed improvement to the point that the patient was able to extend his hip and knee without gravity (Medical Research Council; MRC 2) after one month of rehabilitation (7 months after onset) and with gravity (MRC 3) after 3 months of rehabilitation (9 months after onset), respectively. As a result, the patient gained the ability to walk independently with an ankle-foot orthosis on an even floor 9 months after onset. T2-weighted brain magnetic resonance images, which were taken 6 months after onset, showed a leukomalactic lesion in the right corona radiata and basal ganglia (Fig. 1). Scores for the Mini-Mental State Examination and the ideomotor apraxia test were 27 (cut-off score < 25) and 35 (cut-off score < 32), respectively, at 6 months after onset (8, 9). The patient provided signed, informed consent and our institutional review board approved the study protocol.

Diffusion tensor tractography

Diffusion tensor imaging (DTI) were obtained 6 months after ICH 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 with a navigator echo. For each of the 32 non-collinear diffusion-sensitizing gradients, we acquired 67 contiguous slices parallel to the anterior commissure-posterior commissure line. Imaging parameters used were as follows: matrix = 128 × 128, field of view = 221 × 221 mm^2, repetition time/echo time = 10,726/76 ms, SENSitivity Encoding factor = 2; echo-planar imaging factor = 67 and b = 1000 s/mm^2, number of excinations = 1, and thickness=2.3 mm. The Oxford Centre for Functional Magnetic Resonance Imaging of the Brain Software Library (www.fmrib.ox.ac.uk/fsl) was used for analysis of diffusion-weighted imaging data. Affine multi-scale two-dimensional registration was used for correction of head motion effect and image distortion due to eddy current. DTI-Studio software (CMRM, Johns Hopkins Medical Institute, USA) was used for evaluation of the corticospinal tract (CST). Fibre tracking was based on the fibre assignment continuous tracking (FACT) algorithm (10, 11). Fibre tracking was initiated at the centre of the seed voxel with a fractional anisotropy > 0.2 and a tract-turning angle < 60 degrees. The seed region of interest (ROI) was given on the CST area of the anterior pontomedullary junction on the axial slice and the target ROI was drawn in the CST area of the anterior medulla.

The CST of the left hemisphere originated from the primary sensory-motor cortex and descended through the corona radiata, cerebral peduncle, anterior pons and the anterior medulla, which were the known CST pathways. By contrast, the right CST showed a disruption with Wallerian degeneration to the pontomedullary junction.

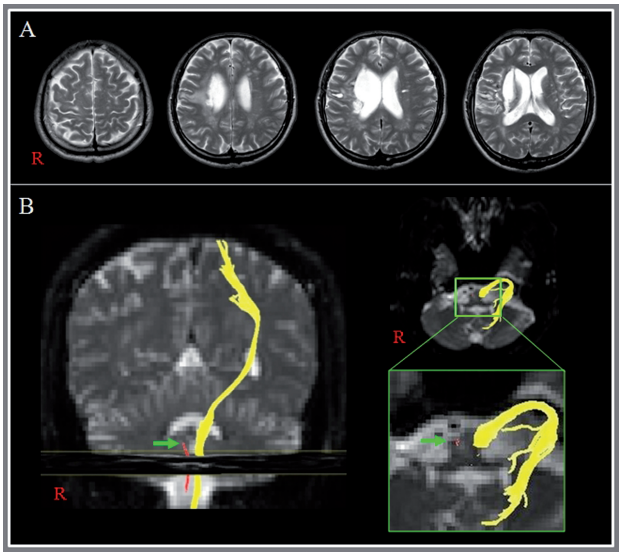

Fig. 1. (A) T2-weighted brain magnetic resonance images show a leukomalactic lesion in the right corona radiata and basal ganglia level. (B) Diffusion tensor tractography for the corticospinal tract (CST). The CST of the left hemisphere originated from the primary sensori-motor cortex and descended through the known CST pathway. However, the CST of the right hemisphere showed a disruption with Wallerian degeneration to the pontomedullary junction (*arrow*).

DISCUSSION

In this patient, we confirmed complete injury of the lateral CST in the affected hemisphere based on the DTT findings. Motor control of the hand and leg is known to differ. The lateral CST is the major motor pathway in the human brain and is responsible mainly for the dexterous motor skills of the hand (12). Therefore, stroke patients are unable to carry out fine motor activities of the hands after complete injury of the lateral CST (12–15); in contrast, studies have demonstrated that stroke patients can walk even after complete injury of the lateral CST, suggesting that the lateral CST is not essential for walking (13, 14). Descending motor pathways that could potentially play a role in walking include non-CSTs, such as the reticulospinal tract, the vestibulospinal tract, and the anterior CST (12, 16). Therefore, we thought that the motor weakness of the left leg in this patient might have recovered, to some extent at least, prior to 6 months after ICH onset, even though the right lateral CST was completely injured. However, the patient only began to show motor recovery of the affected leg 6 months after onset and was able to walk independently 9 months after ICH onset.

We hypothesize that the main reason that the patient did not show motor recovery of the affected leg until 6 months after ICH onset might be ascribed to a type of apraxia; therefore, the delayed motor recovery of the affected leg appeared to be attributed to the resolution of apraxia for the following reasons (17–19). This patient showed relatively rapid recovery over a period of 1 month, starting from 6 months after onset, to the point that he was able to extend the affected leg without

gravity (MRC 2). If this motor recovery was the usual motor recovery by neuronal reorganization during the critical period, it should have begun before 6 months after ICH onset. In addition, motor recovery of the affected leg appeared to be facilitated by dopaminergic drugs, which are known to be effective for the treatment of apraxia (20–22). There are 3 types of limb apraxia: ideational, ideomotor and limb-kinetic apraxia. Because the patient showed normal findings on the Mini-Mental State Examination and the ideomotor apraxia test, we were able to rule out ideational and ideomotor apraxia. Limb-kinetic apraxia is defined as an execution disorder of movements, which is mainly the result of injury to the premotor area with preservation of CST integrity (18, 20). This patient was not completely compatible with the definition of limb-kinetic apraxia; however, among three types of limb apraxia, the apraxia of this patient was closest to limb-kinetic apraxia because the neural pathway from the right premotor area was injured at the corona radiata, even though the right CST was completely injured. We assume that non-CSTs such as the reticulospinal tract, the vestibulospinal tract or the anterior CST, might contribute to motor recovery of the left leg (12, 16). On the other hand, delayed early rehabilitation due to heterotopic ossification might affect the delayed recovery of gait function in this patient. In addition, attention or visuospatial function can also affect functional recovery in stroke.

In conclusion, we report here on a patient who showed unusual delayed motor recovery of the affected leg, which might be through motor tracts apart from the completely injured lateral CST. This case study highlights the extensive potential of the human brain to aid in the recovery of walking after injury. In conclusion, clinicians should attempt to determine the causes of difficulty walking when examining patients with ICH and then perform intensive rehabilitation for the recovery of walking. This study is limited to a case report. Further complementary studies involving larger case numbers are warranted.

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