



Recovery process of bilaterally injured corticoreticulospinal tracts in a patient with subarachnoid hemorrhage

Case report

Sung Ho Jang, MD^a, Chul Hoon Chang, MD^b, Young Jin Jung, MD^b, You Sung Seo, MS^{c,*}

Abstract

Rationale: A few studies using diffusion tensor tractography (DTT) have demonstrated recovery of injured corticoreticulospinal tract (CRT) in patients with intracerebral hemorrhage and infarct. However, no study reported on a patient who showed peri-infarct reorganization of an injured CRT following a middle cerebral artery territory infarct.

Patient concerns: A 56-year-old right-handed male patient was diagnosed as spontaneous subarachnoid hemorrhage (SAH) and intraventricular hemorrhage (IVH) and underwent clipping for a ruptured anterior communicating artery aneurysm and right frontal extraventricular drainage for IVH at the department of neurosurgery of a university hospital. After onset, he presented with complete weakness of both legs.

Diagnoses: The patient was diagnosed as spontaneous SAH and IVH.

Interventions: Clinical assessment and DTT were performed at 1, 3, 6, and 20 months after onset.

Outcomes: The weakness of both legs showed slow recovery for 10 months until 11 months after onset (medical research council: 6 months; 3/3 and 11 months; 4/4). As a result, he was able to walk independently on an even floor at 6 months and on stairs at 11 months after onset. The discontinued both CRTs on 1-month DTT were restored to the cerebral cortex on 3-month DTT, and then thickened consecutively on 6-month and 20-month DTTs.

Lessons: The recovery process of injured CRTs concurrent with recovery of leg weakness was demonstrated in a patient with SAH using DTT. This study has important implications in terms of regaining gait function by the recovery of bilaterally injured CRTs which was facilitated by the long-term rehabilitation.

Abbreviations: CRT = corticoreticulospinal tract, DTI = diffusion tensor imaging, DTT = diffusion tensor tractography, IVH = intraventricular hemorrhage, MRC = medical research council, ROI = regions of interest, SAH = subarachnoid hemorrhage.

Keywords: brain plasticity, corticoreticulospinal tract, diffusion tensor tractography, subarachnoid hemorrhage

1. Introduction

The corticoreticulospinal tract (CRT) is mainly involved in gait function by controlling proximal and axial muscles.^[1] The

Editor: N/A.

This research was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education (NRF-2018R1A6A3A11050913).

^a Department of Physical Medicine and Rehabilitation, ^b Department of Neurosurgery, ^c Department of Physical Medicine and Rehabilitation, College of Medicine, Yeungnam University, Daegu, Republic of Korea.

* Correspondence: You Sung Seo, Department of Physical Medicine and Rehabilitation, College of Medicine, Yeungnam University, 317-1 Daemyung dong, Namku, Daegu 705-717, Republic of Korea (e-mail: yousung1008@daum.net).

Copyright © 2018 the Author(s). Published by Wolters Kluwer Health, Inc. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

Medicine (2018) 97:50(e13401)

Received: 19 July 2018 / Accepted: 1 November 2018 http://dx.doi.org/10.1097/MD.000000000013401 introduction of diffusion tensor tractography (DTT), which is derived from diffusion tensor imaging (DTI), has enabled threedimensional visualization and estimation of the CRT in the live human brain.^[2] Recovery of an injured CRT in patients with intracerebral hemorrhage and cerebral infarct has been reported in a few studies^[3–5]; however, it has not reported in patients with subarachnoid hemorrhage (SAH) so far.

In this study, we attempted to demonstrate the recovery process of injured CRTs in a patient with SAH, which was demonstrated on follow-up DTTs.

2. Case report

A 56-year-old right-handed male patient was diagnosed as spontaneous SAH and intraventricular hemorrhage (IVH) and underwent clipping for a ruptured anterior communicating artery aneurysm and right frontal extraventricular drainage for IVH at the department of neurosurgery of a university hospital. The CT images at onset show SAH and IVH (Fig. 1A). After 1 month from onset, he was transferred to the rehabilitation department of the same university hospital for rehabilitation. Brain MR images at 1 month showed leukomalactic lesions in both prefrontal areas (Fig. 1B). He presented with complete weakness of both legs (Medical Research Council [MRC]: 0/0) (Table 1).^[6] Comprehensive rehabilitative therapy was administered until 3 months

The authors have no conflicts of interest to disclose.



Figure 1. (A) Brain CT images at onset show subarachnoid hemorrhage and intraventricular hemorrhage. (B) T2-weighted MR images at 1 month show leukomalactic lesions in both prefrontal areas. (C) On 1-month DTT for the CRT, both CRTs show discontinuation at the corona radiata level (green arrows) with partial tearing at the midbrain level (blue arrows). However, both discontinued CRTs were restored to the cerebral cortex on 3-month DTT, and then thickened consecutively on 6-month and 20-month DTTs. CRT = corticoreticulospinal tract, DTT = diffusion tensor tractography.

after onset, which included neurotropic drugs (dopaminergic drugs [ropinirole, amantadine, and levodopa] and methylphenidate), movement therapy, and neuromuscular electrical stimulation of both knee extensors and ankle dorsiflexors. Movement therapy focusing on improvement of function of both legs and trunk was conducted five times per week in our physical and occupational therapy department. His rehabilitation was continued at a local rehabilitation hospital until 11 months after onset. The weakness of both legs showed slow recovery for 10 months until 11 months after onset (MRC: 6 months; 3/3 and 11 months; 4/4) (Table 1). As a result, he was able to walk independently on an even floor at 6 months and on stairs at 11 months after onset.^[7] The patient provided informed consent, and the study protocol was approved by our institutional review board.

2.1. Diffusion tensor tractography

DTI data were acquired four times (1, 3, 6, and 20 months 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 = 192×192 matrix; field of view = 240×240 mm²; repetition time = 10,398 ms; echo time = 72 ms;

Table 1						
Longitudinal	changes	of motor	function	in	the	pa

Longitudinal changes of motor function in the patient.									
		Onset	1 month	3 months	6 months	11 months			
	Shoulder abductor	2-/2-	3+/3+	5/5	5/5	5/5			
	Elbow flexor	2-/2-	4/4	5/5	5/5	5/5			
	Finger extensor	2-/2-	4/4	5/5	5/5	5/5			
MRC	Hip flexor	0/0	0/0	3-/3-	3/3	4/4			
	Knee extensor	0/0	0/0	3-/3-	3/3	4/4			
	Ankle dorsiflexor	0/0	0/0	3/3	3/3	4/4			
	Functional Ambulation Category	0	0	1.5	3	4			

MRC = Medical Research Council scale. 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. Functional Ambulation Category: 0 = nonambulatory, 1 = a need for continuous support from one person, 2 = a need for intermittent support from one person, 3 = a requirement for verbal supervision only, 4 = help required on stairs and uneven surfaces, 5 = ability to walk independently anywhere.

parallel imaging reduction factor=2; echo-planar imaging facto=59; b=1000 s/mm²; and a slice thickness of 2.5 mm. Fiber tracking was performed using the fiber assignment continuous tracking algorithm implemented within the DTI Task Card software (Philips Extended MR WorkSpace 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 analysis of the CRP, it was reconstructed using fibers passing through two regions of interest (ROIs) on the color map. The first 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 fractional anisotrophy < 0.1, angle < 27°.

On 1-month DTT for the CRT, both CRTs showed discontinuation at the corona radiata level with partial tearing at the midbrain level. However, both discontinued CRTs were restored to the cerebral cortex on 3-month DTT, and then thickened consecutively on 6-month and 20-month DTTs (Fig. 1C).

3. Discussion

In this study, change of the injured CRTs on DTT with the recovery of leg weakness was evaluated in a patient with SAH. At the start of rehabilitation, we found that both CRTs were partially torn at the midbrain level and discontinued at the corona radiata level. Considering the results of a recent study showing that the CRT injury following SAH was related to weakness in the contralateral proximal and leg muscles, this patient's severe leg weakness was ascribed to bilateral injuries of the CRTs.^[8] In addition, because both CRTs were partially torn at the midbrain level, it appeared that both CRTs were injured mainly by SAH. We think that this result is also consistent with the result of a recent study which demonstrated that the midbrain is the most vulnerable area for the CRT following SAH.^[8]

We believe that the motor function of both legs was recovered by the brain plasticity, for the following reasons. At the start of rehabilitation at 1 month after onset, both legs showed complete weakness and both CRTs were discontinued at the corona radiata level. The motor weakness of both legs recovered slowly during 10 months' rehabilitation from 1 to 11 months after onset to the extent that he was able to move against gravity (MRC: 4/4). As a result, the patient had recovered his ability to walk independently on an even floor at 6 months after onset and on stairs at 11 months after onset. Both discontinued CRTs were connected to the cerebral cortex at 3 months after onset and thickened consecutively with the passage of time. This mode of recovery indicates that the motor recovery of the affected legs could be ascribed to plastic change of the injured CRTs, and not to the resolution of local factors such as edema.^[9,10]

Since the introduction of DTI, the recovery of an injured CRT has been demonstrated in a few studies using DTT.^[3–5] In 2013, Yeo and Jang^[3] reported on a patient with intracerebral hemorrhage who presented with recovery of a discontinued CRT through the normally existing pathway of the CRT to the cerebral cortex. Subsequently, a patient with intracerebral hemorrhage showed recovery of an injured CRT connected to the unaffected hemisphere via transcallosal fibers.^[4] A recent study reported on a patient who showed peri-infarct reorganization of an injured CRT following a middle cerebral artery territory infarct.^[5] As a result, to the best of our knowledge, this is the first study to demonstrate the recovery process of bilaterally injured CRTs following SAH.

In conclusion, the recovery process of injured CRTs concurrent with recovery of leg weakness was demonstrated in a patient with SAH. This study has important implications in terms of regaining gait function by the recovery of bilaterally injured CRTs which was facilitated by the long-term rehabilitation. However, this study is limited because it is a single case study. Conduct of further studies including larger case numbers is warranted. In addition, conduct of studies on rehabilitation strategies to induce or facilitate this recovery mechanism should be encouraged, and further studies on other recovery mechanisms following injury of the CRTs are also needed.

Author contributions

Sung Ho Jang: Manuscript development and writing, Chul Hoon Chang, Young Jin Jung: Study concept and design, You Sung Seo: Study concept and design, Acquisition and analysis of data, and Manuscript authorization.

Conceptualization: You Sung Seo.

Data curation: Chul Hoon Chang, Young Jin Jung.

Resources: Sung Ho Jang.

References

- [1] Mendoza JE, Foundas AL. Clinical neuroanatomy: a neurobehavioral approach. Springer, New York, London:2007.
- [2] Yeo SS, Chang MC, Kwon YH, et al. Corticoreticular pathway in the human brain: diffusion tensor tractography study. Neurosci Lett 2012;508:9–12.
- [3] Yeo SS, Jang SH. Recovery of an injured corticospinal tract and an injured corticoreticular pathway in a patient with intracerebral hemorrhage. NeuroRehabilitation 2013;32:305–9.
- [4] Jang SH, Yeo SS. Recovery of an injured corticoreticular pathway via transcallosal fibers in a patient with intracerebral hemorrhage. BMC Neurol 2014;14:108.

- [5] Jang SH, Lee J, Lee HD. Peri-infarct reorganization of an injured corticoreticulospinal tract in a patient with cerebral infarct. Int J Stroke 2015;10:E62–3.
- [6] Oliver B, Maik K. Aids to the examination of the peripheral nervous system. Medical Research Council, Great Britain:1976.
- [7] Holden MK, Gill KM, Magliozzi MR, et al. Clinical gait assessment in the neurologically impaired. Reliability and meaningfulness. Phys Ther 1984;64:35–40.
- [8] Jang SH, Choi BY, Kim SH, et al. Injury of the corticoreticular pathway in subarachnoid haemorrhage after rupture of a cerebral artery aneurysm. J Rehabil Med 2015;47:133–7.
- [9] Furlan M, Marchal G, Viader F, et al. Spontaneous neurological recovery after stroke and the fate of the ischemic penumbra. Ann Neurol 1996;40:216–26.
- [10] Witte OW. Lesion-induced plasticity as a potential mechanism for recovery and rehabilitative training. Curr Opin Neurol 1998;11:655–62.