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Case Report

Two cases of hemiplegia at 5 years post-stroke onset showing changes in corticospinal and cortico-reticular pathways after 1 month of intensive rehabilitation including repetitive facilitative exercise

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ABSTRACT

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Case reports have described the findings of diffusion tensor tractography (DTT) during the recovery phase of stroke hemiplegia but not during the chronic phase. We report here the cases of two patients with hemiplegia due to stroke, at 5 years post-onset, who underwent 1 month of intensive rehabilitation therapy including repetitive facilitative exercises and a phenol block, whose spasticity and paralysis improved. In both cases, significant changes were observed post-versus pre-DTT; the corticospinal tract (CST) was not visualized on the lesion side before treatment, but was visualized originating in M1 and extending downward after treatment. In the first case, over 5 years, transcallosal fibers (TCF) and transpontine fibers (TPF) extended from the contralateral side to the affected hemisphere, and the cortico-reticular pathway (CRP) of the DP descending motor pathways (DP) increased on both the affected and contralateral sides (particularly the former). However, after treatment, the CRP decreased on both sides. In the second case, the affected and contralateral sides. In the second case, the TCF became denser and the CRP on the contralateral side increased significantly. Previous case reports tracking changes in DTT were all within 1 year of hemiplegia onset. The two cases discussed here were 5 years post-stroke, and intensive rehabilitation not only improved upper limb and hand motor function in a short period but also produced imaging changes consistent with functional improvements on DTT. This is the first study to track changes in DTT before versus after intensive rehabilitation in old cases of stroke-induced hemiplegia. Most of the changes, such as the emergence of new CST patterns, increased CRP, and appearance of the TCF, were consistent with the working hypothesis derived from the accumulation of previous cases within 1 year of onset. However, in one case, the CRP decreased after treatment. This finding represents new insights not included in the working hypothesis and is discussed herein.

Key words: stroke hemiplegia, chronic phase, functional recovery, DTT, repetitive facilitative exercises

Introduction

Diffusion tensor tractography (DTT), which can visualize post- injury brain axonal changes, is attracting much attention as a tool for investigating plasticity [1]. In the recovery of post- stroke hemiplegia following stroke, two of the six descending motor pathways (DP) originating from the motor cortex are considered crucial [2]. The cortico-spinal tract (CST), which originates from M1 (Brodmann area 4), controls voluntary movements of the distal joints (fingers, wrist, toes, and ankle) of the contralateral limb. The

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cortico-reticular pathway (CRP), which originates from the premotor cortex and supplementary motor area (Brodmann area 6), connects directly to the trans-corpus callosum fibers (TCF) and connects to the trans-pontine fibers (TPF), and controls voluntary movements of the proximal joints of the contralateral limbs (shoulder, elbow, trunk, hip, and knee). The CRP further activates the reticulospinal pathway and disinhibits the basal ganglia, thereby regulating postural control by adjusting axial alignment and muscle tone bilaterally in the trunk and upper and lower limbs. Unlike the corticospinal tract, the CRP is a motor descending pathway that bilaterally controls movement, making it an important compensatory pathway for recovering voluntary movements in the proximal hemiplegic regions [2].

The TCF and TPF are also important compensatory pathways.

The corpus callosum transmits information between the bilateral motor cortices, inhibits signal transmission to the contralateral hemisphere, and shares motor experience and planning between them. The TCF forms a compensatory pathway that supports motor function on the damaged side [2]. The TPF crosses the pons, transmitting information between the cerebral cortex and cerebellum. In cases in which the CST is severely damaged, the TPF creates a compensatory pathway for the CST [3].

Case reports describing multiple rounds of DTT imaging during the recovery phase of stroke hemiplegia and observing changes were first reported by Yang et al. [4], followed by Seo et al. [5] and others, for a total of seven reports. However, all were single case reports and did not discuss the dynamic patterns of DTT in recovery from hemiplegia.

Therefore, in the present study we collected cases of recovery-phase stroke hemiplegia in which multiple DTT scans were performed and set the following working hypothesis to identify any patterns in the dynamics of DTT. Specifically, we speculated that the recovery of hemiplegia involves the following: 1) CST reconstruction and proliferation occurs on the lesion side; 2) compensatory CST contralateral control occurs via the TCF or TPF; 3) CRP reconstruction and proliferation occurs on the lesion side; 4) contralateral CRP proliferation occurs; and 5) the first four steps are achieved through fiber regeneration and proliferation.

While testing this hypothesis, we conducted a 1-month intensive rehabilitation treatment including repetitive facilitative exercise [6] and phenol block (PB) in two cases of stroke-induced hemiplegia that had progressed for 5 years post-onset and performed DTT imaging before and after treatment.

Through this experience, we identified two new findings: (1) the need to revise the working hypothesis 5) (i.e., fiber proliferation may lead to reduction and reorganization); and (2) the absence of reports describing short-term changes in DTT following rehabilitation in cases in which more than 1 year had passed post-onset (5 years here in). We report these findings as new insights.

Case 1: Right-handed man in his 70s

Five years earlier, the patient had developed left hemiplegia due to a cerebral infarction on the right side of the brain. Nineteen days post-onset, he was admitted to our hospital's rehabilitation ward with hemiplegic function test (HFT) [7] scores of 2 for the upper limbs and fingers and 3 for the lower limbs, as well as a total Function Independence Measure (FIM) score of 91 (58 for motor function, 33 for cognitive function). Four months later, the HFT scores improved to 6,3 and 7 for the upper limbs, and fingers, and 7 for the lower limbs, as well as a total FIM score of 122 (87 for motor function, 35 for cognitive function), allowing for discharge to home. Post discharge, the patient attended outpatient rehabilitation weekly and walked independently outdoors with a left short leg brace and four-point cane. At 5 years post-onset, spasticity of the upper-limbs and hands had worsened, and so the patient was readmitted to our hospital for a 1-month intensive rehabilitation program including PB.

At the start of the rehabilitation treatment, the Fugl-Meyer Assessment (FMA) score was 20 points for the upper limbs. The HFT scores were 6 for the upper limbs and 0 for the fingers. Significant movement restrictions were noted in the left upper limb and fingers due to paralysis and spasticity. The passive and active joint range of motion (ROM) were restricted (Table 1).

Similar restrictions were observed in shoulder flexion, elbow extension, and thumb palmar abduction, and the patient was unable to pinch the area between the index finger and. On the Modified Ashworth Scale (MAS), left upper-limb and finger spasticity was 1−2 during shoulder abduction, elbow extension, forearm supination, wrist dorsiflexion, and II-V finger metacarpophalangeal joint extension. The total FIM score was 108 (73 for motor function and 35 for cognitive function).

1. Treatment

Intensive rehabilitation consisted of a total of 1-hour treatment time, including repetitive facilitative exercises, orthotic therapy (elastic C-bar for I–II finger pinch movements, elastic taping for proximal interphalangeal extension of each finger), and voluntary movement facilitation using integrated volitional control electrical stimulation (IVES).

Vibration therapy was used to alleviate spasticity during movement along with transcranial direct current stimulation (tDCS) to the right M1 at 2 mA for 20−40 min three times a week, followed by conventional therapy.

The PB was administered as described previously [8], with muscle contractions confirmed via electrical

Table 1. Treatment outcomes of two cases.

		Case 1		Case 2	
		Before treatment	One month after treatment	Before treatment	One month after treatment
paralysis	FMA upper limb (points)	20	39	35	40
	HFT (grade)				
	Upper limb	6	7	7	8
	Fingers	0	2	6	8
	SIAS (points)				
	Proximal upper limb	3	3	3	4
	Distal upper limb	0	1	0	1
Muscle tone	MAS	1~2	0~1	1~2	0~1
ROM	Should abduction (passive, active)	80° 50°	145° 90°	80° 60°	95° 90°
	Forearm supination	90° 40°	90° 45°	−20° −20°	0° 20°
	Wrist dorsiflexion	20° −5°	45° 10°	45° 0°	55° 25°
	Extension of index finger MP	0° −20°	20° 0°	0° −10°	0° −5°
	Extension of index finger PIP	0° −90°	0° −80°	0° −10°	0° 0°
	Flexion of index finger MP	90° 90°	90° 90°	80° 30°	80° 60°
	Flexion of Index finger PIP	90° 80°	90° 90°	90° 35°	90° 70°
STEF (points)		0	0	0	5
Hemiplegia upper limb ability		unsused hands	Auxiliary hand B	Assistant B	Auxiliary hand B
Motor Activity Log	Frequency of use	0.12	0.25	0.125	0.125
	Quality of movement	0.12	0.37	0.125	0.125

FMA: Fugle-Meyer Assessment
HFT: Hemiplegia Function Test
SIAS: Stroke Impairment Assessment Set
MAS: Modified Ashworth Scale
ROM: Range of motion
STEF: Simple Test for Evaluating hand Function
MP: Metacarpophalangeal joint
PIP: Proximal Interphalangeal joint

stimulation, followed by a 5% phenol injection. Treatment targets included the left upper limb's elbow flexors, flexor carpi muscles, and finger flexors.

Treatment effects were evaluated using the HFT, MAS, FMA, Simple test for Evaluating Hand Function (STEF) [9], and Motor Activity Log.

DTT explored whether changes in motor function following intensive rehabilitation could be captured as changes in DP (CST and CRP) reconstruction (fiber quantity or trajectory changes) or TCF and TPF. DTT imaging was performed using an Ingenia 1.5T streams HeadNeckSpine coil (Philips) to visualize the motor descending pathways, Image processing was conducted using the included Fiber Tracking Application software. The regions of interest (ROIs) for DTT were defined as described previously [2], with the CST corresponding to M1, posterior limb of the internal capsule (PLIC), and cerebral peduncle and CRP corresponding to the premotor cortex, PLIC, midbrain tegmentum, and pontomedullary reticular formation. For the TCF and TPF, when CST and CRP are defined as their respective ROIs, both are automatically detected if compensatory pathways, so there was no need to set separate ROIs.

The normal DTT criteria were based on the normal allotype of descending motor pathways (NDP) in healthy individuals reported by Jang et al. [10] and Yoo et al. [11]. According to Jang et al. [12], the average number of DP fibers in healthy individuals was 994 for CST and 1177 for CRP. In our institution, no software was available to automatically calculate the number of DP fibers. When the DP appears fan-shaped on DTT images, we magnify the DTT image fourfold and manually count the fibers, then estimate the count using the formula by Kisa et al. [2] ($\pi\times$ [fiber count^2]/4). When the DP fibers are bundled, the diameter is measured in millimeters using the attached measuring tool and entered into the formula ($\pi\times$ [bundle diameter^2]/4) to calculate the estimated number of fibers. The changes in the estimated number of fibers before versus after treatment were compared to determine whether the fiber number increased or decreased.

The validity of this calculation method was verified as follows. Specifically, since the DP form bundles at the thalamic level, the CST and CRP diameters at the same location in the NDP diagram by Yoo et al. [11] were measured visually and substituted into Kisa et al.'s formula ($\pi\times$ [bundle diameter ^{2}]/4). The estimated right and left CST/CRP fiber ratios were 0.879 and 0.826, respectively, similar to those reported by Jang et al. [12] (CST, 994 fibers; CRP, 1177 fibers). Therefore, we concluded that Kisa et al.'s formula could be applied in this study.

The ROIs settings were determined by the radiographer and confirmed by the main author. The DTT analysis was performed by the main author (rehabilitation medicine specialist), who proposed findings (described in the figure captions below). The final decision was made through consultation with a neurologist, neurosurgeon, and radiologist who were

unfamiliar with the patient's symptoms.

2. Follow-up

After 1 month of intensive rehabilitation (Table 1), the paralysis improved from grade 6 to 7 for the upper limbs and from 0 to 2 for the fingers on the HFT, as well as from 20 to 39 points on the FMA for the upper limbs. The spasticity improved from 1−2 to 0−1 on the MAS scale, hemiplegia upper-limb function improved from unused hand to assisted use (B), and the frequency of use of the paralyzed limb improved from 0.12 to 0.25.

Upper-limb and finger spasticity decreased from 0 to 1 on the MAS scale immediately after PB, making voluntary movements easier. Under tDCS, thumb extension and abduction occurred and repetitive movements were easily performed.

Two weeks after treatment, the patient developed shoulder-hand syndrome in the left upper limb, thus the repetitive facilitative exercises were discontinued. The addition of oral administration of vaccinia virus-infected rabbit inflammatory skin extract and near-infrared irradiation to the left stellate ganglion, led to symptom improvement, allowing discharge 2 weeks later. The shoulder-hand syndrome improved at 2 months post-discharge.

3. Imaging findings

DTT was performed three times (Figure 1): 3 months post-onset (Figures 1a, 1b), 5 years post-onset but before treatment (Figures 2a; Figures 3a-c), and after treatment (Figures 2b, 2c; Figure 3d-f). Post-onset, the CST was not detected in the DP, while the CRP was barely visible. At the pretreatment stage at 5 years post-onset, the TCF [2, 4] and TPF [3] extended from the contralateral to the affected side in the corpus callosum. The DP increased significantly by 196-fold on the lesion side and slightly increased on the contralateral side (Figure 2a). A single CST was newly detected, but it did not originate in M1 (Figure 3b). The CRP increased on the lesion and contralateral sides (Figure 2a; Figures 3b, 3c). At 1month post-treatment, DTT revealed that the DP had become sparse (Figures 3e, 3f), and the origin of the CST extended from M1 (Figure 3e). Meanwhile, at 1 month after treatment, the bilateral CRP had decreased significantly (to 13% and 16% of the pretreatment level on the lesion and contralateral sides, respectively).

Case 2: Right-handed man in his 60s

Five years earlier, the patient had suffered a right thalamic hemorrhage causing left hemiplegia. Four weeks later, he was transferred to a rehabilitation facility. At that time, his HFT scores were 2 and 3 for the upper and lower limbs, respectively, along with severe superficial and deep sensory impairments. However, the higher brain function test results were normal, and his total FIM score was 93 (58 for motor function, 35 for cognitive function). At discharge, the HFT identified motor paralysis (scores: upper limbs. 7; fingers. 8; lower limbs. 9) and sensory deficits (superficial and deep sensations), while the total FIM improved to 121 (87 for motor function and 34 for

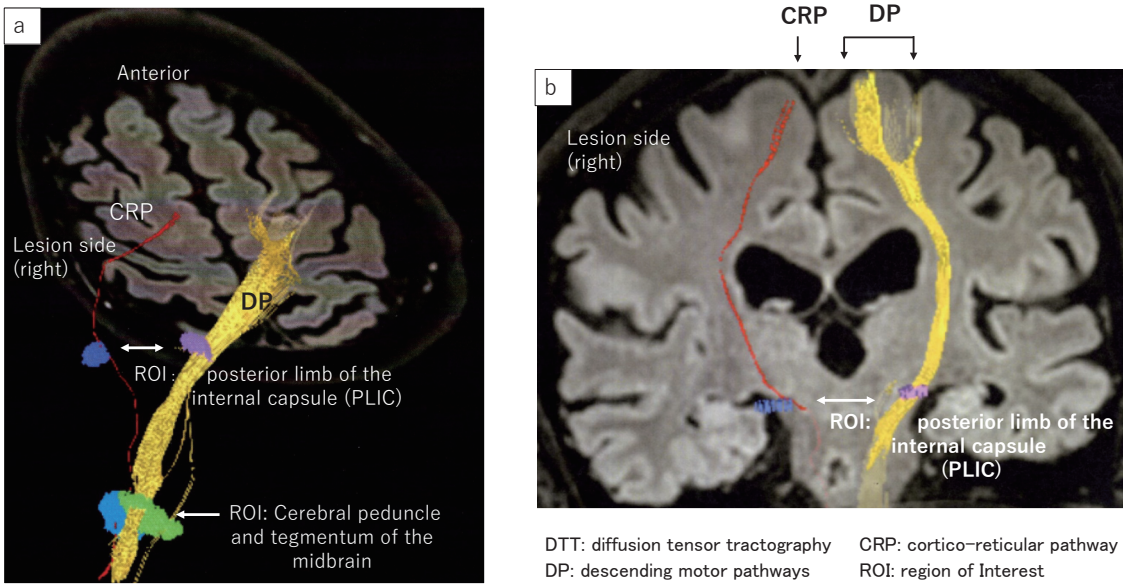

Figure 1. DTT images taken 3 months post-onset in case 1.
a) DTT image (horizontal section) taken at 3 months post-onset in Case 1. No CST was observed on the lesion side, while one CRP was present in the motor cortex area and the DP was observed on the contralateral side.
b) DTT image (frontal plane) taken at 3 months post-onset. No CST was observed on the lesion side. One DP corresponding to the CRP was present in the medial cortical area and observed on the contralateral side.

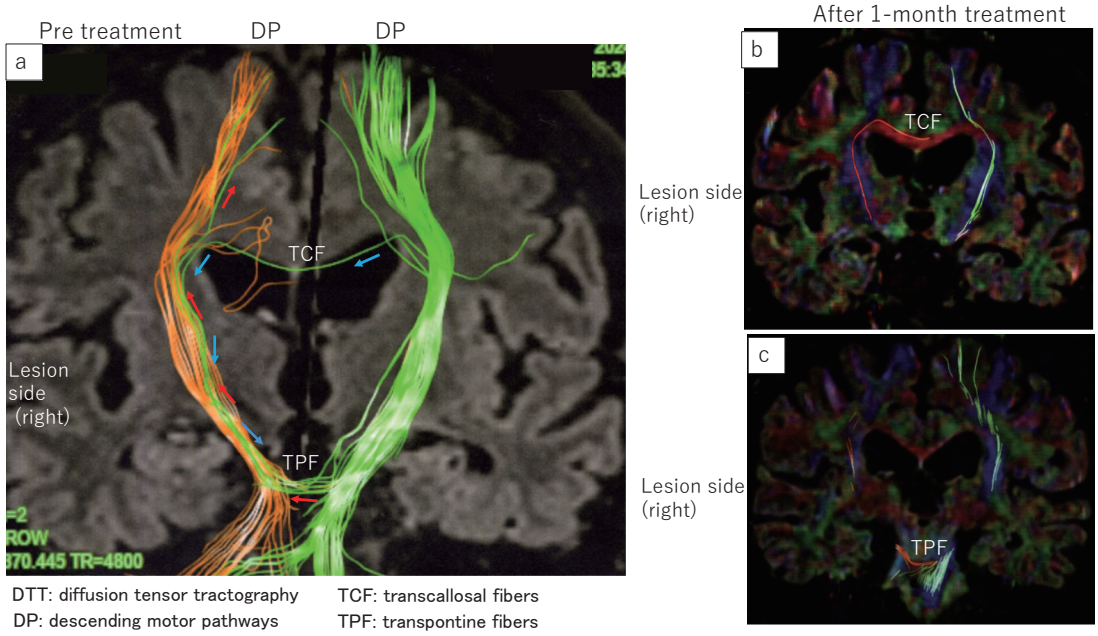

Figure 2. DTT image (frontal plane) taken at 5 years post-onset (before vs. after treatment) in case 1. Compared to 3 months post-onset, the DP increased on the lesion and contralateral sides, the TCF was extended through the corpus callosum to the lesion side and then descended (blue arrow), and the TPF extended through the pons to the lesion side and then ascended (red arrow). After treatment, the ROI settings for DP visualization remained the same, while DP visualization significantly decreased bilaterally and the TCF and TPF remained thinly preserved.

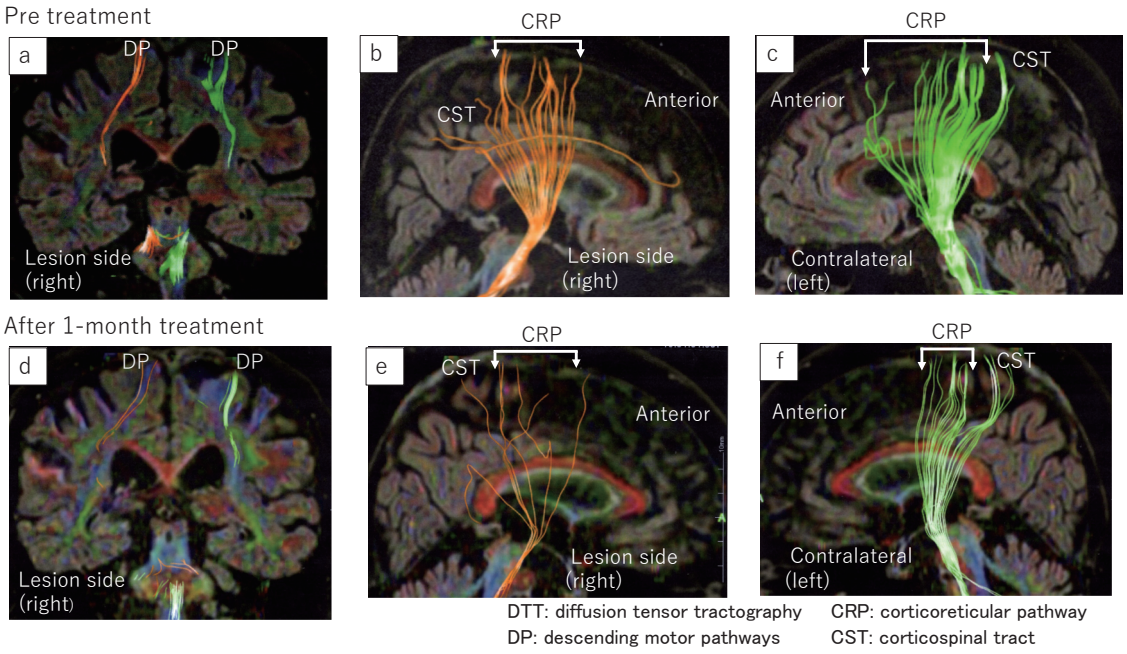

Figure 3. DTT images taken at 5 years post-onset before vs. after treatment in case 1.
a) DTT image (frontal plane) before vs. after treatment. DP was present bilaterally before treatment but decreased after treatment.
b, e) DTT image (lesion side, sagittal section) taken before treatment (b) and after treatment (e). Before treatment, a single CST originating from outside the M1 for the upper limb and hand was observed on the lesion side with an increased CRP. After treatment, this CST originated from the M1 for the upper limb and hand and descended with the CRP becoming sparse.
c, f) DTT image (contralateral side, sagittal plane) before treatment (c) vs. after treatment (f). CRP increased before treatment but decreased after treatment. Meanwhile, the number of CST fibers in the contralateral side remained unchanged.

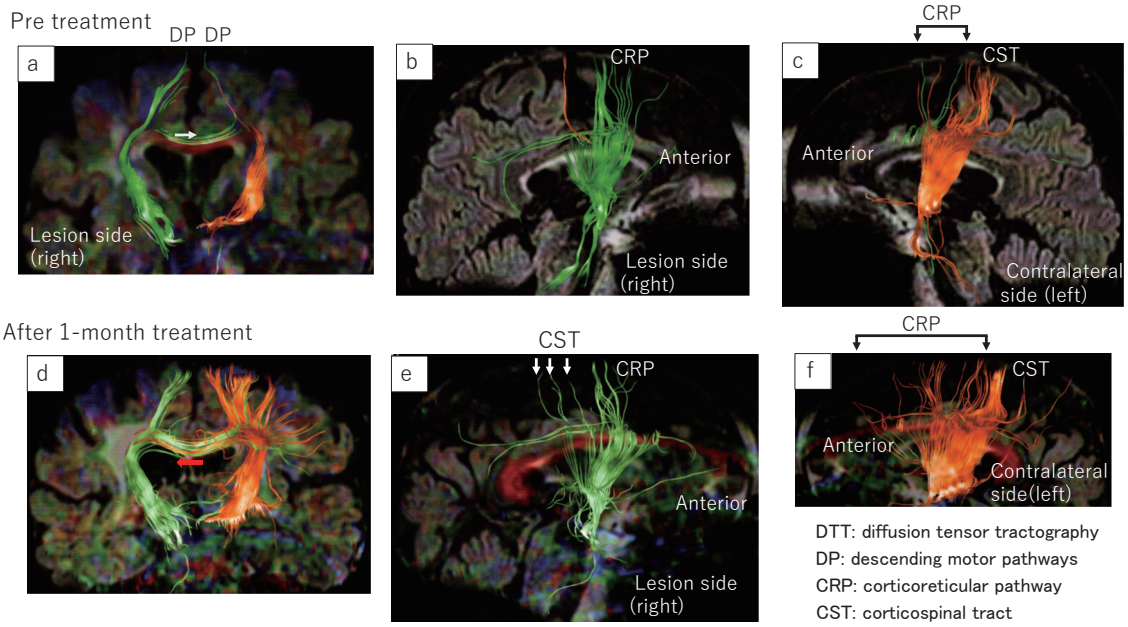

Figure 4. DTT images taken at 5 years post-onset before vs. after treatment in case 2.
a, d) DTT images (frontal plane) taken before (a) vs. after (d) treatment. Prior to treatment, extension of the TCF was observed from the lesion side (white arrow). After treatment, TCF expansion was detected on the contralateral side (red arrow), while the DP was significantly increased.
b, e) DTT images (lesion side, sagittal section) before (b) and after (e) treatment. Prior to treatment, CST was not observed in the area where it should have been present on the lesion side. After treatment, three CSTs were identified from M1 (white arrows).
c, f) DTT images (contralateral sagittal section) taken before (c) and after (f) treatment. CRP on the contralateral side was rare before treatment but significantly increased after treatment.

cognitive function). He was discharged home with outdoor walking without any assistive devices and returned to work.

Post-discharge, the patient continued outpatient rehabilitation once weekly but was admitted for a 1-month intensive rehabilitation program including PB due to worsening upper-limb and hand spasticity.

At the start of treatment, the patient's range of motion of the left upper limb and finger ROM (Table 1) differed widely between passive and active ROM, and he was unable to pinch the area between the II–V fingers and the thumb due to restricted automatic flexion. Left upper limb and fingers spasticity was 1–2. The total FIM was 108 (73 for motor function, 35 for cognitive function).

Intensive rehabilitation was initiated on the day PB was administered, as the left upper-limb and finger spasticity had improved from 0 to 1 on the MAS scale. The treatment focused on repetitive facilitative exercises, supplemented by orthotic therapy (cock-up splints, C-bars), tapings for fingers flexion and extension, and IVES use in some aspects of the repetitive facilitative exercise techniques.

1. Symptom improvements

The 1-month treatment results are shown in Table 1. Significant physical function improvements were observed on the FMA, including increases from 7 to 8 for upper limbs. 6 to 8 for fingers, and 35 to 40 for FMA. Range of motion improved in many joints, spasticity decreased from 1–2 to 0–1 in PB treated joints, while upper limb function improved from 0 to 5 on the STEF scale.

2. Imaging findings

The DTT images are shown in Figure 4. Although the TCF extended from the lesion side (Figure 4a) prior to treatment, no CST was visible on the lesion side (Figure 4b), and that on the contralateral side was sparse (Figure 4c). (Figure 4e). At 1 -month post-treatment, the TCF extending from the contralateral to lesion side was also visualized, and its total thickness increased (Figure 4d), while the contralateral DP significantly increased (Figure 4d). On the lesion side, three new CST originating from M1 were visualized (Figure 4e), while the contralateral CRP had increased to 22.5 times the pre-treatment level (Figure 4f).

In both cases 1 and 2, no intensive intervention targeted the lower limbs, while no changes were noted in lower-limb paralysis such as in muscle strength or walking ability.

Discussion

In these two cases of hemiplegia at 5 years post-stroke onset, a 1-month intensive rehabilitation including

repetitive facilitative exercises for the upper limbs and fingers and a phenol block, resulted in functional improvement and significant DTT imaging changes. In both cases, the CST originating from M1 of the upper limbs and fingers was not detected at the start of treatment, whereas images taken post-treatment showed the CST originating from the same area extending downward.

In the first case, the TCF and TPF extended from the contralateral to affected side prior to treatment. In the second case, the TCF extended sparsely from the affected and contralateral sides prior to treatment but increased in density after treatment. The CRP levels in the lesion and contralateral side decreased and became sparse after the intensive rehabilitation in case 1 but increased and became increasingly dense in the second case.

Kisa et al. [2] investigated the relationship between motor descending pathways (CST, CRP) in 49 patients admitted to a rehabilitation ward during the recovery phase and the association between TCF and motor function prognosis. They demonstrated that the CST on the ipsilesional side of the lesion and the CRP on the contralateral side contribute to upper-limb paralysis improvements and that the CST on the ipsilesional side contributes to hand paralysis. They also noted that cases of TCF tended to show more advanced separation synergic movements of the upper limbs and hands. The findings in the two DTT cases in this study differed from those in the chronic versus recovery phase but were consistent with those reported by Kisa et al. [2] as follows. First, the CST, which was not observed at the origin in either case, was depicted on the lesion side after treatment. Second, the TCF was observed in both cases before treatment. Third the CRP on the contralateral side was increased in size in both cases; however, in case 1, it was already increased before treatment, while in case 2, it increased after treatment.

According to Kisa et al. [2], there have been seven case reports to date describing the recovery course of stroke-induced hemiplegia. Four of these used manual muscle testing to evaluate hemiplegia, while the other three based their evaluations on this method, but failed to adequately assess central paralysis influenced by synergic movements. As a result, the association between DTT findings and motor function recovery in the proximal and distal parts of the upper and lower limbs is commonly unclear.

The two cases presented in this study are chronic and had progressed for 5 years but demonstrated improvements in upper limb and finger motor function through short-term intensive rehabilitation therapy and structural changes on DTT images that support the functional changes. To our knowledge, this is the first report globally to demonstrate clear changes on DTT images in the chronic phase within a short (1-month) period.

In case 1, the motor descending pathways, which had densely increased prior to intensive rehabilitation therapy including PB and tDCS, became less dense and the number of fibers decreased in the affected and contralateral sides after treatment.

The background to these rapid DTT changes, including axonal changes, is influenced by the intensive rehabilitation, particularly PB, which expands ROM; tDCS, which to increases motor cortex excitability; and repetitive facilitative exercises, which repeatedly practice target movements.

The improvements in shoulder, elbow, forearm, and hand movements suggest that synapses previously "mobilized" and affected by these movements underwent long-term potentiation through repeated intensive treatment, leading to synaptic pruning.

The improvements in shoulder, elbow, forearm, and hand movements are due to the conversion and consolidation of the roles of existing axons. This point is illustrated by a report by Jang et al. [13] in which a 57-year-old man suffered a hemorrhage in the left basal ganglia of the brain, that resulting in complete right-sided paralysis. At 1 month post-onset, DTT revealed that the CST in the injured hemisphere was interrupted at the midbrain level. However, 9 months later, DTT showed new branches connecting the posterior parietal cortex of the injured hemisphere to the main trunk of the CST at the thalamic level. By 11 months, these branches were thickened, and significantly improved motor paralysis was noted.

Jang et al. [13] reported that the cortical origin of the damaged CST may shift from other areas to M1. We also consider this a "role conversion" of existing axons in which areas originally involved in sensory integration rather than the primary motor pathway may transform into alternative pathways for motor output. This branch thickening represents the convergence of diffuse compensatory routes into the final selected route.

The significant chronic-phase plasticity observed on DTT not only highlights the importance of the rehabilitation program during the acute and recovery phases but it underscores the importance of the rehabilitation program aimed at reconstructing and strengthening damaged neural pathways in the chronic phase, presenting a significant challenge for future development.

The large-scale spine imaging method developed by Ikegaya et al. [14, 15] in 2005 enables wide-range, high-resolution dynamic observation of dendritic spines and is considered a groundbreaking technique for visualizing experience-dependent neural plasticity.

The synaptic dynamics (axon sprouting, selective spinal stabilization) obtained with this technique underlie the mechanisms behind axonal re-extension, compensatory pathway reconstruction, and convergence phenomena observed in the two cases reported here or in previous reports [13].

It is difficult to speculate on the reasons for the

conflicting DTT results in these cases. In the first case, the fiber sizes had already maximized, and the intensive rehabilitation may have led to the pruning of inefficient pathways (fibers). In the second case, fiber size had not yet reached its maximum and was able to increase further.

While measurement variability cannot be completely ruled out, the possibility is small since the same method was used each time.

The unexpected phenomenon of "synaptic pruning" encountered in this study, which was not anticipated in the initial working hypothesis, remains an unresolved research question for future investigations.

This study's limitations include the fact that, as a case report, changes in paralysis or spasticity may not necessarily correlate with changes evident on imaging. Moreover, changes in treatment interventions may not necessarily correlate with changes noted on imaging. Thus, further quantitative analyses with larger sample sizes, comparative studies, and functional evaluations using transcranial magnetic stimulation in conjunction with DTT are necessary to validate our findings.

Ethics declaration:

After obtaining approval from the Ethics Committee of Matsue-Seikyo Hospital (approval number:202304), the purpose of the study, method of implementation, and management and use of the obtained data were explained to the participants, and their written consent to participate in the study was obtained.

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