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A Review of Studies on the Role of Diffusion Tensor Magnetic Resonance Imaging Tractography in the Evaluation of the Fronto-Subcortical Circuit in Patients with Akinetic Mutism

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Akinetic mutism (AM) is characterized by the complete absence of spontaneous behavior (akinesia) and speech (mutism) with the preservation of executive functions for movements and speaking. Elucidation of the pathophysiological mechanisms or neural correlates for AM is clinically important because patients can recover from AM after medication and neuromodulation. The fronto-subcortical circuit is a critically important neural structure in the pathophysiology of AM. Using diffusion tensor tractography, a few neural tracts in the fronto-subcortical circuit can be reconstructed. This mini-review article evaluated 6 DTT-based studies on the fronto-subcortical circuit injury in patients with AM. According to these results, the neural tracts among the fronto-subcortical circuit, which are related to AM, were as follows (in decreasing order of importance): 1) the prefronto-caudate tract, 2) the prefronto-thalamic tract, and 3) the cingulum. In particular, the medial prefrontal cortex is an important brain area related to recovery from AM. However, only 6 studies on this topic have been published, and most were case reports. In addition, these studies analyzed only a few neural tracts in the fronto-subcortical circuit. Because AM is a rare disorder, studies involving a large number of subjects might be impossible. Nevertheless, an analysis of various neural tracts in the fronto-subcortical circuit is necessary. For this, reconstruction of the other neural tracts in the fronto-subcortical circuit should be performed first. This review aims to present the findings from recent studies on the role of DTT in evaluation of fronto-subcortical circuit injury in patients with AK.

Keywords: Akinetic Mutism • Brain Injuries • Brain Injuries, Traumatic • Diffusion Tensor Imaging • Stroke

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Background

Akinetic mutism (AM) is the most severe disorder among the 3 disorders of diminished motivation (AM, abulia, and apathy) [1]. AM is characterized by the complete absence of spontaneous behavior (akinesia) and speech (mutism) with the preservation of executive functions for movements and speaking [1-3]. AM is clinically important because it causes in ability to perform activities of daily living and imposes a heavy burden on caregivers [1,4,5]. Elucidation of pathophysiological mechanism or neural correlates for AM is clinically important because patients can recover from AM after receiving medication, such as dopamine agonists and brain stimulants [1,3]. In addition, recently developed neuro-stimulation techniques, such as repetitive transcranial magnetic stimulation and transcranial direct current stimulation, can be applied to the neural correlates for AM recovery [1,3,6]. However, the pathophysiological mechanism of AM is not completely understood.

The fronto-subcortical circuit (frontal lobe and its subcortical connections) is a critically important neural structure in the pathophysiology of AM [1,3,7-9]. Specifically, injury to the cortico-striatal-pallidal-thalamic circuit has been suggested to be the most plausible pathophysiological mechanism of AM [1,3,7]. The reconstruction of various neural tracts in the fronto-subcortical circuit is essential for elucidating the pathophysiological mechanism of AM. However, precise reconstruction of the fronto-subcortical circuit in a live human brain has been impossible. Since Cairns et al reported the first case of AM in 1941, many studies have reported the neural correlates for AM as the following specific neuropathological brain regions, which were commonly associated with bilateral hemispheric pathologies, using conventional brain imaging or nuclear imaging: (1) the frontal lobe (the medial frontal cortex and anterior cingulate cortex), (2) basal ganglia (striatum and pallidum), (3) thalamus (anterior and medial nuclei), and (4) midbrain (ventral tegmental area) [3,10-16]. AM can be classified into 2 subtypes according to the anatomical location of the brain lesions: (1) the frontal AM (hyperpathic), lesion in the frontal region; and (2) mesencephalic AM (somnolescent or hypopathic), lesions in the thalamus and midbrain [16].

The diffusion tensor imaging (DTI), which generates images based on estimations of the diffusion of water molecules in various microstructures, has enabled evaluation of the entire microstructural features of the white matter [17,18]. In particular, using diffusion tensor tractography (DTT) reconstructed based on DTI data, the neural tracts in a live human brain can be reconstructed three-dimensionally [19,20]. The main advantage of DTT is that the entire neural tract can be evaluated in terms of the DTT parameters (fractional anisotropy, mean diffusivity, and tract volume) and configurational analysis (integrity and configuration) [2,21-23]. Some neural tracts of the fronto-subcortical circuit, including the prefronto-caudate tract, prefronto-putaminal tract, prefronto-thalamic (mediodorsal nucleus) tracts, and cingulum, were reconstructed using DTT [2,24-27]. As a result, DTT has transformed research into AM from a brain lesion study to a fronto-subcortical circuit study. Based on the above studies, several papers have reported that AM is related to injuries of the above neural tracts [2,21,24,28-30].

In this study, we reviewed 6 DTT studies that demonstrated neural tract injury in patients with AM after a brain injury [2,21,24,28-30] (**Table 1**). This review aims to present the findings from recent studies on the role of DTT in evaluation of fronto-subcortical circuit injury in patients with AK.

Table 1. Diffusion tensor tractography studies on the fronto-subcortical circuit injury in patients with akinetic mutism.

Authors	Publication year	Number of patients	Pathology of brain injury	Analysised neural tracts	DTT analysis method
Jang & Kwon [2]	2017	1	Mild TBI	PCT, PTT	Configuration
Burks et al [24]	2017	40	Anterior butterfly glioma	PCT, PTT	Configuration
Jang et al [21]	2017	1	TBI	PCT	Change of congfiguration
Jang et al [28]	2017	1	Aneurysmal SAH	PCT	Change of congfiguration
Jang et al [29]	2018	1	TBI	PCT	Configuration
Byun & Jang [30]	2022	1	SAH, ICH, IVH	PCT, PTT, ARAS	Configuration

DTT – diffusion tensor tractography; TBI – traumatic brain injury; PCT – prefronto-caudate tract; PTT – prefronto-thalamic tract; SAH – subarachnoid hemorrhage; ICH – intracerebral hemorrhage; IVH – intraventricular hemorrhage; ARAS – ascending reticular activating system.

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DTT Studies on Fronto-Subcortical Circuit Injury in Patients with Akinetic Mutism

In 2017, Jang and Kwon reported the case of a patient who showed AM with injuries and subsequent degeneration of the prefronto-caudate and the prefronto-thalamic tracts after a mild traumatic brain injury (TBI) [2]. A 20-year-old man had a mild TBI caused by a pedestrian-car accident. Since the onset of mild TBI, he revealed the clinical features of abulia (decreased activity and speech). His abulia aggravated to AM approximately 1 year after onset. He presented the typical clinical features of AM as follows. He remained lying down all day with no spontaneous movement or speech. Even during mealtime, he was fed by a caregiver and chewed according to the caregiver's order because he could not consume a meal or chew by himself [2]. On the 1-month DTT, the prefronto-caudate tract (the neural connectivity of the caudate nucleus to the medial prefrontal cortex) showed partial discontinuation in both hemispheres. These findings were aggravated on the 2-year DTT, indicating neural degeneration of injured prefronto-caudate tracts. However, the orbitofrontal-thalamic tract was thin in the left hemisphere on 1-month-DTT. In contrast, this tract became thinner in both hemispheres on the 2-year DTT, suggesting neural degeneration of the injured left orbitofrontal-thalamic tract. The authors concluded that follow-up DTTs demonstrated injuries to the prefronto-caudate tract and the orbitofrontal-thalamic tract and subsequent degeneration of these injured neural tracts concurrent with an aggravation of abulia to AM in this patient. This study showed that the aggravation of abulia to AM was concurrent with the degenerations of the injured prefronto-caudate and the orbitofrontal-thalamic tracts. However, the authors could not discern which neural tract – the injured prefronto-caudate tract or the orbitofrontal-thalamic tract – was responsible for his AM. Furthermore, the authors estimated the neural tracts only by configurational analysis without DTT parameter data. Another limitation of this study was that only 2 neural tracts (the prefronto-caudate and prefronto-thalamic tracts) were estimated among the many neural tracts in the fronto-subcortical tract.

During the same year, Burks et al [2007] compared the incidence of abulia or AM after the standard surgical method (non-anterior cingulum-sparing technique) or the anterior cingulum-sparing technique to prevent injury to the anterior cingulate gyrus while resecting an anterior butterfly glioma in 40 patients [24]. Eleven out of the 25 patients (44%) had significant abulia or AM 1 day after surgery using the standard surgical technique, whereas only 1 out of 15 patients (7%) presented abulia or AM with the anterior cingulum-sparing technique. Six weeks after surgery, 7 out of 25 (28%) showed abulia or AM with the standard surgical technique, but no patients presented with abulia or AM with the anterior cingulum-sparing technique. The authors concluded that the anterior butterfly gliomas could be removed safely by preserving the anatomical connectivity of the anterior cingulum and its relevant cingulate gyrus. As a result, the authors demonstrated that the anterior cingulum and its relevant cingulate gyrus are important neural correlates for abulia or AM. That study had some limitations. First, the authors did not provide the precise incidence of abulia or AM. Precise discrimination of abulia or AM based on the medical record might have been difficult because it was a retrospective study. Second, the authors did not differentiate the cingulum state between the 2 surgical techniques in terms of configuration and DTT parameters.

Subsequently, in 2017 Jang et al reported a patient who showed an improvement of AM concurrent with recovery of an injured prefronto-caudate tract following TBI, using follow-up DTTs [21]. A 72-year-old woman was diagnosed with a subdural hematoma on the right frontal lobe and subarachnoid hemorrhage after falling down a set of stairs and underwent conservative management. She presented with the typical clinical features of AM (no spontaneous movement or speech) at 5 weeks after onset when starting rehabilitation. She underwent comprehensive rehabilitative management, including dopaminergic drugs to improve AM (pramipexole: 1.5 mg, amantadine: 300 mg, ropinirole: 2.75 mg, and levodopa: 750 mg) [1,3]. During rehabilitation for 5 weeks, her AM improved gradually, and she could perform some activities of daily living (eg, eating, dressing, and walking). On 5-week DTT, the prefrontal-caudate tract revealed injury findings in both hemispheres (the neural connectivity of the caudate nucleus to the medial prefrontal cortex) (Broadmann area: 10 and 12), and the orbitofrontal cortex (Broadmann area 11 and 13) was decreased in both hemispheres [31]. On the 10-week DTT, however, partial recovery of the injured left prefronto-caudate tract was observed (the neural connectivity of the left caudate nucleus to the left medial prefrontal cortex was increased). The authors concluded that the recovery of the injured prefronto-caudate tract concurrent with the improvement of AK was demonstrated in this patient, using follow-up DTTs. The strength of this study is that it demonstrates the effects of dopaminergic drugs on AM and recovery of the injured prefronto-caudate tract [1,3]. However, it suggested that the medical prefrontal cortex appears to be related more to AM than the orbitofrontal cortex because the neural connectivity to the medial prefrontal cortex was increased with the recovery of AM in this patient. On the other hand, this study analyzed only the prefronto-caudate tract among various neural tracts in the fronto-subcortical circuit. Moreover, this study provided only configurational data without DTT parameter data.

In 2017, Jang et al reported the case of a stroke patient who showed recoveries of AM and injured prefronto-caudate tract, which was related to relief of the hydrocephalus, using follow-up DTTs [28]. A 76-year-old female patient was diagnosed

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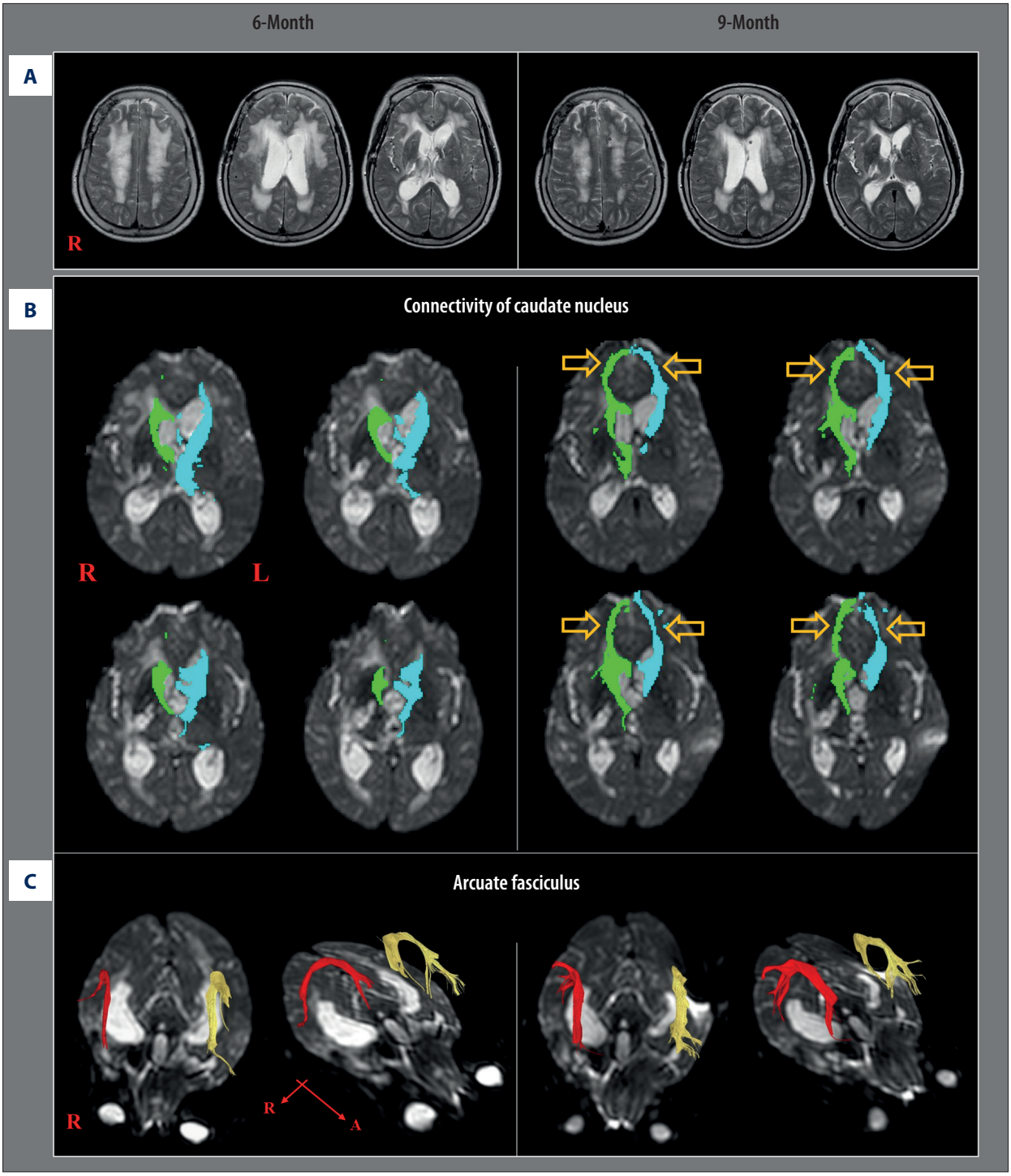

Figure 1. A patient who shows recovery from akinetic mutism and injured prefronto-caudate tract following shunt operation for hydrocephalus and rehabilitation. (**A**) T2-weighted brain magnetic resonance images at 6 months after onset, showing leukomalactic lesions in both fronto-parieto-occipital areas, right thalamus, and hydrocephalus, and relief of hydrocephalus at 9 months after onset. (**B**) On 6-month diffusion tensor tractography (DTT), the neural connectivity of the caudate nucleus to the medial prefrontal cortex (Broadmann area: 10 and 12) and orbitofrontal cortex (Broadmann area 11 and 13) is decreased in both hemispheres. However, the neural connectivity of the caudate nucleus to the medial prefrontal cortex is increased on both sides (arrows) on 9-month DTT. (**C**) The integrity of arcuate fasciculus is preserved in both hemispheres on both 6- and 9-month DTTs. (*Reprinted with permission from Medicine, Medicine [Baltimore]: 2017; 96: e9117*).

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with an aneurysmal subarachnoid hemorrhage caused by rupture of a right posterior communicating artery aneurysm. She underwent craniotomy and clipping of the aneurysmal neck. At 6 months after onset, when she started rehabilitation at another university hospital, she revealed the typical clinical features of AM (she did not move or speak spontaneously). Hydrocephalus was detected on the brain magnetic resonance images (**Figure 1**). The patient received comprehensive rehabilitative management, including dopaminergic drugs to improve AM (pramipexole, amantadine, ropinirole, and levodopa) [1,3]. Despite 2 months of rehabilitation, her AM did not improve significantly. Consequently, she received a ventriculoperitoneal shunt operation for hydrocephalus 8 months after onset. After the shunt operation, she remained in the AM state. Before her shunt operation, she underwent similar comprehensive rehabilitative management. During 1-month rehabilitation, her AM improved gradually; she could perform some daily living activities herself, including washing, eating, and dressing, 9 months after onset. In addition, she could speak with some fluency. The prefronto-caudate tract showed injury findings in both hemispheres on the 6-month DTT (the neural connectivity of the caudate nucleus to the medial prefrontal and orbitofrontal cortices was decreased in both hemispheres) (**Figure 1**). By contrast, these injured prefronto-caudate tracts showed recovery on the 9-month DTT (the neural connectivity of the caudate nucleus to the medial prefrontal cortex was increased on both sides). The integrity of the arcuate fasciculus was preserved in both hemispheres on both 6- and 9-month DTTs. The authors concluded that this patient had recovered from AM and injured prefronto-caudate tracts. This study provides some take-home messages. First, the medial prefrontal cortex is more important for AM than the orbitofrontal cortex because this patient's AM recovered along with the increased neural connectivity to the medial prefrontal cortex**.** Second, although dopaminergic agonists are effective in the recovery of AM, relief of the hydrocephalus is more important when combined with AM. Third, this study presented the intact findings of AF in both hemispheres on both 6- and 10-month DTT, suggesting that the mutism in this patient occurred irrespective of the language ability. However, the study was limited because the prefrontal tract was analyzed in terms of configuration without DTT parameter data.

In 2018, Jang et al reported a patient who showed AM concurrent with severe injuries of both prefronto-caudate tracts due to an accident [29]. A 72-year-old man had head trauma resulting from an injury to his frontal area caused by an electric grinder while working at his home. He was diagnosed with a traumatic intracerebral hemorrhage in both frontal lobes, intraventricular hemorrhage, and subarachnoid hemorrhage and underwent hematoma removal and decompressive craniectomy. At 2 months after onset, when starting rehabilitation, he had the typical clinical features of AM; he remained lying down all day, with no spontaneous movement and speech. At 2 months after onset, brain magnetic resonance images showed leukomalactic lesions in both prefrontal areas, including both cingulate gyri. The 2-month DTT revealed a severe injury of the prefronto-caudate tract in both sides (decreased neural connectivity of the caudate nucleus to the medial prefrontal and orbitofrontal cortices). Hence, injuries of both prefronto-caudate tracts were observed in this patient who developed AM following TBI. The authors suggested that injury of the prefronto-caudate tract might be a pathophysiological mechanism of AM in patients with brain injury. However, only the prefronto-caudate tract was analyzed: even the anterior cingulum, which appeared to be injured severely in both sides on brain magnetic resonance images, was not analyzed.

Recently (2022), Byun and Jang reported the case of a patient in whom a differential diagnosis of AM with a disorder of consciousness was made using DTT [30]. A 69-year-old female patient was diagnosed with a subarachnoid hemorrhage, intraventricular hemorrhage, and an intracerebral hemorrhage produced by the subarachnoid hemorrhage. She exhibited impaired consciousness with a Coma Recovery Scale-Revised score of 13 until 1 month after onset. Her impaired consciousness slowly recovered to a normal state according to the Coma Recovery Scale-Revised (23 points: full score) at 7 weeks after onset, but she showed the typical clinical features of AM (no spontaneous movement or speech). On the DTT performed at 1 month, the upper and lower dorsal ascending reticular activating systems, which are related to a disorder of consciousness, showed an almost normal state. In contrast, the prefronto-caudate and prefronto-thalamic tracts, which are related to AM, showed severe injuries. These DTT results suggested that the patient's main clinical features were not a disorder of consciousness, but AM. The authors concluded that DTT for the ascending reticular activating system and the prefronto-caudate and prefronto-thalamic tracts could provide additional evidence for a differential diagnosis of disorders of consciousness and AM at the early stages of stroke.

Conclusions

This mini-review article evaluated 6 DTT-based studies on fronto-subcortical circuit injury in patients with AM and the pathophysiological mechanisms involved [2,21,24,28-30]. The information provided in these reviewed studies suggests that DTT could be helpful for elucidating the pathophysiological mechanism of AM. According to the results of these studies, the neural tracts among the fronto-subcortical circuit, which were related to AM, are as follows in decreasing order: (1) prefronto-caudate tract, (2) prefronto-thalamic tract, and (3) cingulum [2,21,24,28-30]. In particular, the medial prefrontal cortex was suggested as an important brain area for the recovery

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of AM [21,28]. These studies could facilitate the neurorehabilitation of patients with AM [6]. Only 6 studies on this topic have been published; most were case reports. In addition, these studies analyzed only a few neural tracts in the fronto-subcortical circuit. Studies involving a large number of subjects might be impossible because AM is a rare disorder, but analysis of various neural tracts in the fronto-subcortical circuit is needed. For this, use of the reconstruction methods for the other neural tracts in the fronto-subcortical circuit should be encouraged. In addition, a precise three-dimensional reconstruction method for the prefronto-caudate tract and the prefronto-striatal tract, which were reported the neural connectivity from 1 region of interest, is needed [21,25,28,29]. On the other hand, all 6 reviewed studies corresponded to the frontal AM. Further studies on mesencephalic AM based on DTI or DTT are needed.

This review presented the findings from the only 6 identified main studies on the role of DTT in evaluation of fronto-subcortical circuit injury in patients with AK. Although this in vivo imaging method shows promise for evaluating the fronto-subcortical circuit in stroke, TBI, and brain tumors, further studies are required to evaluate the role of DTT in other brain pathologies with AK.

Delaration of Figures' Authenticity

Figure 1, is reproduced here with permission.

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