

A Case Report of Three-Dimensional Fiber Tractography of Anterior Thalamic Radiation in a Capsular Genu Infarction

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Interruption of the anterior thalamic radiation (ATR) might explain cognitive impairment in an infarction of the genu of the internal capsule. However, a defect of the ATR in the genu infarction has received little attention. We report on a patient who developed cognitive impairment following the genu infarction. Diffusion tensor imaging revealed an interruption of the white matter fibers in the ATR. We suggested that cognitive impairments in the genu infarction were caused by a defect in the ATR.

Key Words: *Genu of the internal capsule, Anterior thalamic radiation, Diffusion tensor imaging*

INTRODUCTION

Cognitive impairments had been traditionally thought to reflect grey matter damage related to the Papez circuit [1] or the basolateral limbic circuit proposed by Yakovlev [2]. Kooistra and Heilman have since suggested that a capsular genu infarction involving the white matter tracts can lead to the development of cognitive impairment [3]. Several of the following reports replicated that white matter tract damage could evoke the cognitive impairments; [4-12]. They tried to investigate the mechanism of cognitive impairment in patients who had a capsular genu infarction, but the results were unclear.

The recent advent of diffusion tensor imaging (DTI) has allowed the white matter connectivity among arbitrary regions of the brain to be delineated [13]. DTI fiber tractography could supply information regarding the microstructures in white matter, such as the directionality of water diffusion (MD) and the spatial orientations of the fibers (FA). For these reasons, DTI fiber tractography is a more useful imaging method to localize lesions within functionally specific white matter tracts [13].

We report on a patient who had a capsular genu infarction and presented with amnesia and behavioral changes. The results of detailed neuropsychological tests and DTI provided us a better understanding of the mechanisms of

the neuropsychological deficits that were seen in this patient.

CASE REPORT

A 79 yr-old right-handed hypertensive man with sixteen years of education presented with memory disturbances and behavioral changes. These symptoms developed seven days prior to his visit to our memory clinic. Just prior to his initial presentation, he was not able to memorize his appointment time or to recall a telephone conversation. He benefited from cues, and could recall that he had called on the telephone. However, he could not remember what he said in more detail and could not remember the date of the telephone call. He had difficulty finding the right words, and he often said that he had experienced difficulty expressing his thoughts verbally, despite knowing what he wanted to say. His wife noticed that he had lost emotional responsiveness at that time. According to his wife, he did not start anything until prompted. His interest in friends and daily

activities diminished. He had suffered from depression since his daughter became bed-ridden several years ago due to trauma. He seemed to be physically comfortable. He had no complaints other than the cognitive impairments. Upon examination seven days post-onset, he showed mild dysarthria and decreased swinging in the left arm. A brain magnetic resonance imaging (MRI) (3.0 tesla device, Philips) performed on the same day showed a small lesion, which was confined to the genu of the internal capsule and the globus pallidum (Fig. 1).

He underwent a detailed neuropsychological test on the same day, at which time his score on the MMSE was 27/30 (memory recall, 2/3; attention and calculation, 4/5; and repetition, 0/1). He showed word-finding difficulty with mild

Table 1. Neuropsychological test scores for the patient

Attention	Digit span (for/back)	5/3
Language & related functions		
Spontaneous speech		NL
Comprehension		ABNL
Repetition		NL
K-BNT (60)		24*
Reading		NL
Writing		NL
Praxis		NL
Right-left orientation		NL
Calculation		NL
Body part identification		NL
Visuospatial functions		
Interlocking pentagon		NL
Copy of rey-osterrieth complex figure		33
Memory		
Orientation (time/place)		5/5
3 word recall		2
SVLT (1st/2nd/3rd)	Immediate recall	10* (2/4/4)
	Delayed recall	3
Recognition discriminability index (True positive-false positive)		8-2
Rey-osterrieth complex figure immediate recall		19.5
Delayed recall		15.5
Recognition discriminability index (True positive-false positive)		9-0
Frontal executive functions		
Contrasting program		NL
Square & triangle		NL
Luria loop		NL
COWAT (Animal/supermarket/phonemic)		6*/4*/12*
Stroop test (color naming)		37*
MMSE		27

*, <16%tile.

AB, abnormal; NL, normal; SVLT, Seoul Verbal Learning Test; K-BNT, the Korean version of Boston Naming Test.

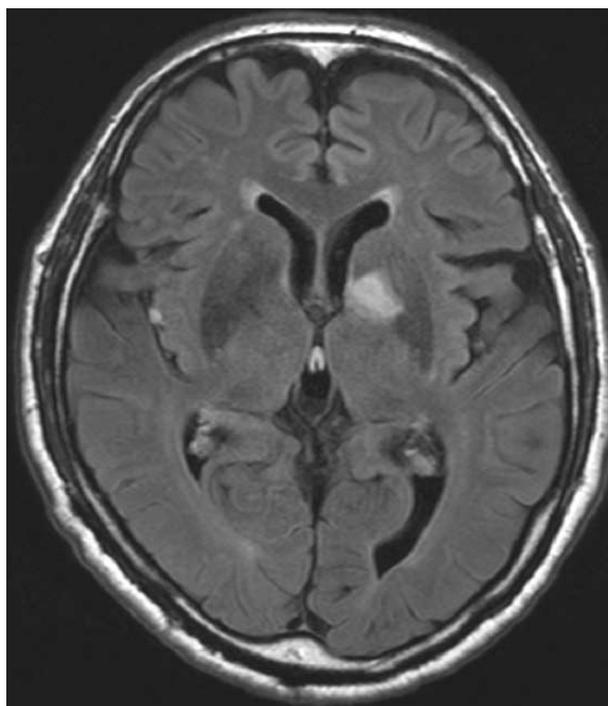


Fig. 1. The brain MRI of the patient. The Fluid attenuated inversion recovery axial view shows an infarct involving the left genu of the internal capsule and part of the globus pallidum.

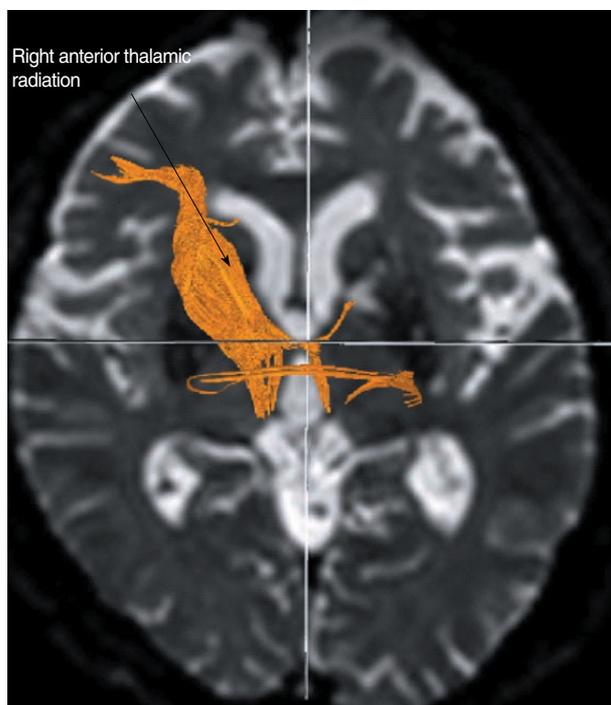


Fig. 2. The diffusion tensor image of the patient. It shows an interruption of the white matter fibers in the left anterior thalamic radiation.

hesitancy. His score on the Korean-Boston Naming Test (K-BNT) was 24/60 (0.01%ile of age, sex, and duration of education, compared to matched control subjects). His errors were characterized primarily by the substitution of semantically similar words. On the Seoul Verbal Learning Test (SVLT), the score for free recall was 10/36 (4.65%ile). The score for 20 min delayed recall was 3/12 (16.85%ile). The discrimination index of recognition was 6 (8-2) (22.96%ile). The Animal score on the Controlled Oral Word Association Test (COWAT) was six (1.32%ile). The supermarket score was 4 (0.21%ile). The phonemic total score was 12 (9.34%ile), and the score for color reading on the Stroop test was 37 (2.07%ile). Detailed neuropsychological results are given in Table 1.

DTI fiber tractography conducted three weeks after symptom onset revealed an interruption of the white matter fibers in the left anterior and inferior thalamic radiations (Fig. 2). The tracts were terminated if the fractional anisotropy in the voxels was below 0.3 or if the angle between adjacent voxels along the tract exceeded 85 degrees.

DISCUSSION

He had a profound amnesia and behavioral changes such as apathy, abulia, and asponaneity. His MRI demonstrated that lesions were restricted to genu portion of internal capsule and globus pallidum in left hemisphere. Some reports showed that patients with bilateral globus pallidum lesions had frontal dysfunctions such as abulia, apathy, withdrawal and loss of interest [14]. However, unilateral lesions of the globus pallidum cause more frequently movement disorders such as dystonia than neuropsychiatric deficits such as amnesia, abulia or disinhibition [15], thus we considered that our patient's amnesia might be more related to capsular infarction rather than pallidal infarction. Moreover, other memory-related structures such as anterior thalamus, fornix, and mammillothalamic fasciculus seemed to be intact on MRI.

The cognitive impairment in this patient was consistent with that in previous reports, characterized by fluctuating alertness, inattention, memory loss, apathy, abulia, and psychomotor retardation [4-12]. These features are similar to the clinical manifestations found in infarctions of the paramedian thalamus [16], the origin of the anterior thalamic radiation.

DTI fiber tractography showed a prominent defect in the ATR that connects the orbitofrontal lobe and the thalamus. The ATR is one part of the frontal subcortical circuit and is associated with frontal dysfunctions, such as disinhibition, apathy, and frontal executive dysfunction. The ATR is one part of the basolateral limbic circuit. This circuit consists of the orbital cortex, anterior temporal cortex, amygdala, the dorsomedial thalamus via the uncinate fasciculus, the ventral amygdalofugal pathway, and the ATR [2]. This circuit is known to play an important role in memory function [2]. Lesions of the anterior thalamic radiation could result in amnesia and behavioral changes. To our knowledge, there have been few reports showing a defect in the anterior thalamic radiation in patients who have an infarction of the internal capsule genu. Previous functional imaging studies suggested that an interruption of the ATR might explain the cognitive impairment that is seen after a capsular genu infarction. These imaging studies also showed that patients with

a capsular genu infarction had hypometabolism or hypoperfusion in the frontal region [4, 8, 12, 17]. However, the patients could not directly visualize the defect involved in these white matter tracts.

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