Clinical Case Report

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Restoration of the ascending reticular activating system compressed by hematoma in a stroke patient

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Abstract

Rationale: We report on restoration of the ascending reticular activating system (ARAS), compressed by an intracerebral hematoma and perihematomal edema following a stroke. The restoration of the ARAS was demonstrated by diffusion tensor tractography (DTT).

Patient concerns: In a 60-year-old male, a brain MRI taken at 2 weeks after the surgery showed a hematoma and perihematomal edema in the left posterolateral pons and cerebellum, which were markedly resolved on a brain MRI after 5 weeks.

Diagnoses: Intraventricular hemorrhage.

Interventions: Navigation-guided stereotactic drainage of a hematoma in the left cerebellum, comprehensive rehabilitative therapy, including hypersomnia medication (modafinil), physical therapy, and occupational therapy.

Outcomes: His hypersomnia improved significantly with rehabilitation, with no daytime hypersomnia beginning 3 weeks after the surgery. On 2-week DTT, neither the neural tract of the left lower dorsal or ventral ARAS were reconstructed, but these neural tracts were wellreconstructed on 5-week DTT.

Lessons: In conclusion, restoration of nonreconstructed neural tracts of the lower ARAS with the resolution of the hematoma and perihematomal edema was demonstrated in a stroke patient, using DTT.

Abbreviations: ARAS = ascending reticular activating system, DTT = diffusion tensor tractography, DTI = diffusion tensor imaging, TBI = traumatic brain injury.

Keywords: ascending reticular activation system, consciousness, diffusion tensor tractography, stroke

1. Introduction

The ascending reticular activating system (ARAS) is complex and not easily discriminated from adjacent neural structures. Consequently, precise delineation of the ARAS is problematic. However, diffusion tensor tractography (DTT), which is derived from diffusion tensor imaging (DTI), allows 3-dimensional reconstruction and estimation of the ARAS in the human brain.[1–3] Injury of the ARAS or recovery of an injured ARAS in patients with brain injury has been demonstrated in many studies.[4–8] However, no study on restoration of an ARAS compressed by intracerebral hematoma has been reported.

In the present study, we report on a stroke patient in whom the ARAS, compressed by an intracerebral hematoma and perihematomal edema, was restored, and demonstrated by DTT.

2. Case report

A 60-year-old male underwent navigation-guided stereotactic drainage of hematoma in the left cerebellum and received conservative management for intraventricular hemorrhage at the department of neurosurgery of a university hospital (Fig. 1A). At 2 weeks after the surgery, he was transferred to the rehabilitation department of the same university hospital. T2-weighted brain MR images taken at 2 weeks after the surgery showed a hematoma and perihematomal edema in the left posterolateral pons and cerebellum, which markedly resolved on 5-week brain MRI (Fig. 1B). At 2 weeks after the surgery, the patient exhibited intact consciousness, with full marks on the Glasgow Coma Scale score: 15 and Coma Recovery Scale-Revised score: 23.[9,10] However, he suffered severe hypersomnia following the surgery; he constantly fell asleep without external stimulation. He underwent comprehensive rehabilitation therapy, including hypersomnia medication (modafinil), physical therapy, and occupational therapy. His hypersomnia improved significantly with rehabilitation, and his daytime hypersomnia stopped 3 weeks after the surgery. The patient provided signed, informed consent, and the study protocol was approved by our institutional review board.

2.1. DTI

DTI data were acquired twice (2 and 5 weeks after onset) using a sensitivity-encoding head coil on a 1.5-T Philips Gyroscan Intera

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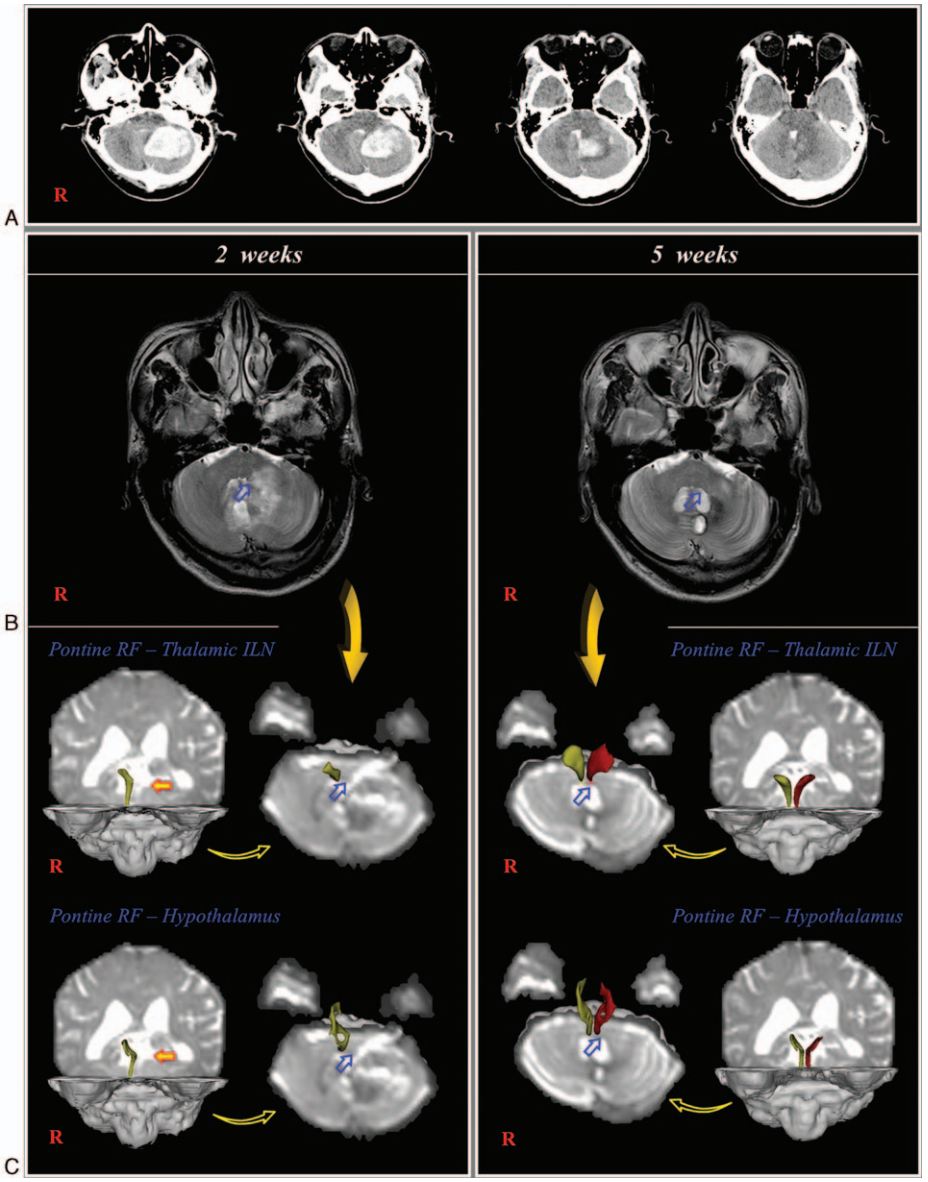

Figure 1. (A) Brain CT images at onset show an intracerebral hemorrhage in the left cerebellum and an intraventricular hemorrhage in the fourth ventricle. (B) Brain MRI taken at 2 weeks after onset shows a hematoma and peri-hematomal edema (blue arrow) in the left posterolateral pons and cerebellum, which are markedly resolved (blue arrow) on 5-week brain MRI. (C) Results of diffusion tensor tractography (DTT) of the patient. On 2-week DTT, neither neural tract of the left lower dorsal and ventral ascending reticular activating system is reconstructed (yellow arrows); however, these neural tracts were well-reconstructed on 5-week DTT.

(Hoffman-LaRoche Ltd, Best, The Netherlands). For each of the 32 noncollinear diffusion sensitizing gradients, 67 contiguous slices (reconstructed matrix: 192×192, acquisition matrix: 96×96, field of view: $240 \times 240\,mm^2$, echo time: 76 ms, repetition time: 10,726 ms, number of excitations: 1, echo-planar imaging factor: 49, b: $1000\,s/mm^2$, parallel imaging reduction factor [SENSE factor]: 2, and a slice thickness of 2.5 mm with no gap) were acquired parallel to the anterior commissure-posterior commissure line.

2.2. Probabilistic fiber tracking

Using the Oxford Centre for Functional Magnetic Resonance Imaging of the Brain Software Library (FSL; www.fmrib.ox.ac.uk/fsl), diffusion-weighted imaging data were analyzed. For eddy current, Affine multiscale 2-dimensional registration was used to correct head motion effect and image distortion. Fiber tracking was performed using a probabilistic tractography method based on a multifiber model. FMRIB Diffusion Software with routines option (0.5-mm step lengths, 5000 streamline samples, curvature thresholds = 0.2) was used for fiber tracking.[11–13] Two portions of the ARAS were reconstructed by selection of fibers passing through regions of interest (ROIs). For analysis of the lower dorsal ARAS, the seed ROI was placed on the pontine reticular formation, and the target ROI with option of termination was placed on the intralaminar thalamic nucleus.[2] For reconstruction of the lower ventral ARAS, the seed ROI was placed on the pontine reticular formation and the target ROI with option of termination was placed on the hypothalamus.[3] Out of 5000 samples generated from a seed voxel, results were visualized at the threshold of 2 streamlines through each voxel for analysis.

On 2-week DTT, neither neural tract of the left lower dorsal and ventral ARAS was reconstructed. However, these neural tracts were well-reconstructed on 5-week DTT (Fig. 1C).

3. Discussion

In the present study, the change of the lower ARAS was evaluated with DTT in a stroke patient. The neural tracts of the left lower dorsal and ventral ARAS, which were not reconstructed on 2-week DTT, were well reconstructed on 5-week DTT. We believe that nonreconstruction of these neural tracts observed on 2-week DTT was attributable to compression by the cerebellar hematoma and perihematomal edema in the posterior-lateral area of the pons, the origin of the lower ARAS (the pontine reticular formation). Therefore, the reconstruction of these neural tracts observed on 5-week DTT apparently resulted from the resolution of hematoma and perihematomal edema, which was observed on follow-up brain MRI. In addition, the rapid recovery of these neural tracts over 3 weeks, from 2 to 5 weeks post-surgery, suggests that this recovery resulted from resolution of local factors (hematoma and perihematomal edema), and not to brain plasticity.[14,15] On the contrary, the patient did not show consciousness impairment except for hypersomnia. Even the hypersomnia, possibly caused by the brainstem lesion, was quickly resolved with modafinil treatment, although the left lower ARAS was not reconstructed on 2-week DTT. This could be additional evidence that the injury of the left lower ARAS was not severe, but rather simple compression of the left lower ARAS.[2,16]

In conclusion, restoration of nonreconstructed neural tracts of the lower ARAS with resolution of the hematoma and perihematomal edema was demonstrated in a stroke patient, using DTT. To the best of our knowledge, this is the first study to demonstrate restoration of the ARAS by decompression, although a few studies have reported on the recovery of injured ARAS.[4,6]

However, limitations of DTT should be considered: regions of fiber complexity and crossing can prevent full reflection of the underlying fiber architecture by DTT. Therefore, DTT may underestimate the fibers of neural tracts.[17]

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