

Hypersomnia due to injury of the ventral ascending reticular activating system following cerebellar herniation

A case report

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Abstract

Rationale: We report on a patient with hypersomnia who showed injury of the lower ascending reticular activating system (ARAS) following cerebellar herniation due to a cerebellar infarct, detected on diffusion tensor tractography (DTT).

Patient concerns: A 53-year-old male patient was diagnosed as a left cerebellar infarct, and underwent decompressive suboccipital craniectomy due to brain edema at 2 days after the onset of a cerebellar infarct. Three weeks after onset when the patient started rehabilitation, he showed hypersomnia without impairment of consciousness; he fell asleep most of daytime without external stimulation and showed an abnormal score on the Epworth Sleepiness Scale: 15 (full score: 24, cut off for hypersomnia: 10).

Diagnoses and Outcomes: On 3-week DTT, narrowing of the upper portion of the lower ventral ARAS between the pontine reticular formation and the hypothalamus was observed on both sides. In addition, partial tearing was observed in the middle portion of the right lower ventral ARAS.

Lessons: In conclusion, we found injury of the lower ventral ARAS in a patient with hypersomnia following cerebellar herniation due to a cerebellar infarct.

Abbreviations: ARAS = ascending reticular activating system, DTI = diffusion tensor imaging, DTT = diffusion tensor tractography, RF = reticular formation, ROI = region of interest.

Keywords: ascending reticular activating system, cerebellar herniation, diffusion tensor imaging, hypersomnia, stroke

1. Introduction

Hypersomnia is a common sequela in stroke patients: 1 study reported that 5.6% of stroke patients showed persistent hypersomnia.^[1] Hypersomnia has been mainly reported in patients with lesions in the thalamus, hypothalamus, or pons.^[2–9] Several recent studies using diffusion tensor tractography (DTT) for the ascending reticular activating system (ARAS) have demonstrated the association of hypersomnia with injury of the ARAS in patients with brain injury.^[10–13] However, the pathogenesis of hypersomnia has not been clearly elucidated.

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In the present study, using DTT, we report on a patient with hypersomnia who showed injury of the lower ventral ARAS following cerebellar herniation due to a cerebellar infarct.

2. Methods

2.1. Case report

A 53-year-old male patient was diagnosed with a left cerebellar infarct, and underwent decompressive suboccipital craniectomy due to brain edema at 2 days after onset of a cerebellar infarct at the neurosurgery department of a university hospital (Fig. 1A). A leukomalactic lesion was observed in the left cerebellum on brain magnetic resonance imaging taken at 3 weeks after onset, however, no abnormal lesion was observed in the midbrain (Fig. 1A). Three weeks after onset, he was transferred to the rehabilitation department of a university hospital in order to undergo rehabilitation. He showed hypersomnia without impairment of consciousness; he fell asleep most of daytime without external stimulation. At the time of diffusion tensor imaging (DTI) scanning (3 weeks after onset), he showed an abnormal score on the Epworth Sleepiness Scale: 15 (full score: 24, cut off for hypersomnia: 10).^[14] Epworth Sleepiness Scale as a questionnaire was made once by the patient's own. The patient signed an informed consent statement, and the study protocol was approved by the Yeungnam University hospital Institutional Review Board of a university hospital (Table 1).

2.2. Diffusion tensor tractography

DTI data were acquired at 3 weeks after onset of the cerebellar infarct using a 1.5T Philips Gyroscan Intera (Philips, Ltd, Best,

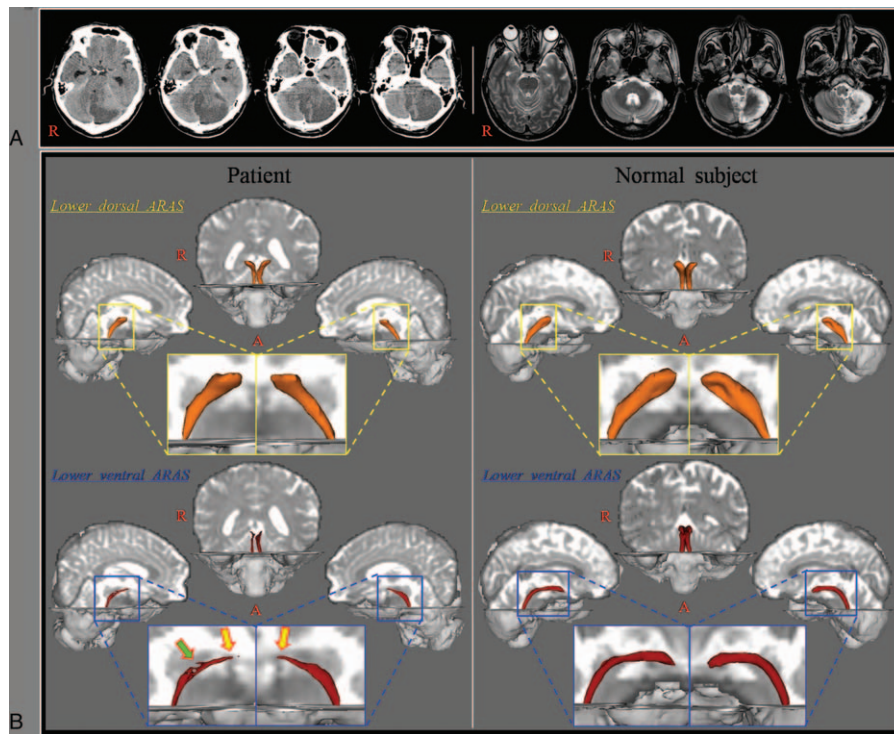


Figure 1. (A) Brain CT images at onset show the left cerebellar infarct and cerebellar herniation and T2-weighted brain MR images at 3 weeks after onset show a leukomalactic lesion in the left cerebellum. (B) Results of diffusion tensor tractography: narrowing of the upper portion (yellow arrows) of the lower ventral ascending reticular activating system is observed on both sides. In addition, partial tearing (green arrow) is observed in the middle portion of the right lower ventral ascending reticular activating system.

The Netherlands) with 32 gradients. Imaging parameters were as follows: acquisition matrix = 96×96 ; reconstructed to matrix = 192×192 ; field of view = $240 \times 240 \text{ mm}^2$; repetition time = 10,398 milliseconds; echo time = 72 milliseconds; parallel imaging reduction factor = 2; echo-planar imaging factor = 59; $b = 1000 \text{ seconds/mm}^2$; and a slice thickness of 2.5 mm. Before the fiber tracking, eddy current correction was applied for correction of the head motion effect and image distortion using the Oxford Centre for Functional Magnetic Resonance Imaging of the Brain (FMRIB) Diffusion Software. Fiber tracking was performed using probabilistic tractography with default tractography option in the FMRIB Diffusion Software (5000 streamline samples, 0.5 mm step lengths, curvature thresholds = 0.2).^[15] Two portions of the

ARAS were reconstructed by selection of fibers passing through regions of interest (ROI) as follows^[16,17]: the lower dorsal ARAS, between the pontine reticular formation (RF, ROI 1) and the intralaminar thalamic nucleus (ROI 2),^[16] the lower ventral ARAS, between the pontine reticular formation (ROI 1) and the hypothalamus (ROI 2).^[17] The threshold of 2 streamlines was applied for the results of fiber tracking.

3. Results

Narrowing of the upper portion of the lower ventral ARAS between the pontine RF and the hypothalamus was observed on both sides and partial tearing was observed in the middle portion of the right lower ventral ARAS (Fig. 1B).

4. Discussion

In the present study, the 2 lower portions of the ARAS were evaluated using DTT: the lower dorsal ARAS between the pontine RF and the intralaminar thalamic nucleus, and the lower ventral ARAS between the pontine RF and the hypothalamus. Narrowing of the upper portion of both the lower ventral ARAS and partial tearing of the middle portion of the right lower ventral ARAS were observed. These findings indicated injury of the lower ventral ARAS on both sides. It appeared that hypersomnia of this patient was ascribed to the injury of the lower ventral ARAS. Our results appeared to coincide with the results of previous studies reporting close association of the hypothalamus with hypersomnia.^[7-9]

Several recent studies using DTT have reported on the association of hypersomnia with injury of the lower ARAS.^[10-13]

No.	Situation	Score (0-3)
1	Sitting and reading	2
2	Watching TV	1
3	Sitting, inactive in a public place (e.g., a theater or a meeting)	1
4	As a passenger in a car for an hour without a break	3
5	Lying down to rest in the afternoon when circumstances permit	3
6	Sitting and talking to someone	1
7	Sitting quietly after lunch without alcohol	2
8	In a car, while stopped for a few minutes in traffic	2
	Total	15

Meaning of score: 0—would never doze or sleep, 1—slight chance of dozing or sleeping, 2—moderate chance of dozing or sleeping, and 3—high chance of dozing or sleeping.

In 2006, Jang et al^[10] reported on a patient who showed hypersomnia following spontaneous pontine hemorrhage. Although this patient showed injury of both the lower dorsal and ventral ARAS, the injury of the lower ventral ARAS was more severe. Subsequently, Jang et al^[11] demonstrated injury of the lower ventral ARAS in a patient who showed hypersomnia and narcolepsy following mild traumatic brain injury. During the same year, Jang et al^[12] reported on a patient who showed recovery of hypersomnia concurrent with the recovery of an injured lower dorsal and ventral ARAS following spontaneous subarachnoid hemorrhage. This patient also showed more severe injury and more recovery of the lower ventral ARAS than the lower dorsal ARAS. Jang and Kwon^[13] recently reported on 2 patients who showed hypersomnia and fatigue with injury of the lower dorsal and ventral ARAS following mild traumatic brain injury. Considering the results of the above-mentioned studies, it appeared that the lower ventral ARAS was more responsible for hypersomnia and it is also consistent with our results.^[10–13]

In conclusion, we found injury of the lower ventral ARAS in a patient with hypersomnia following cerebellar herniation due to a cerebellar infarct and we think that injury of the lower ventral ARAS could be a probable cause for hypersomnia in this patient. Since the introduction of DTT for the ARAS, a few studies have reported on injury of the ARAS due to brain herniation including transtentorial herniation, subfalcine herniation, and Kernohan phenomenon.^[18–20] However, in terms of cerebellar herniation, to the best of our knowledge, this is the first study to demonstrate injury of the ARAS. Nevertheless, limitations of this study should be considered. First, because it is a case report, this study is limited; therefore, conduct of further studies comprising a large number of patients would be necessary. Second, despite being a powerful anatomic imaging tool, DTI may underestimate or overestimate the fiber tracts because regions of fiber complexity and crossing can prevent full reflection of the underlying fiber architecture by DTI.^[21]

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