Clinical Case Report

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Absent-mindedness and injury of the ascending reticular activating system in a patient with mild traumatic brain injury

A case report

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Abstract

Rationale: No study on the association of absent-mindedness and injury of the ascending reticular activating system (ARAS) has been reported. We report on a patient who showed absent-mindedness and injury of the ARAS following mild traumatic brain injury.

Patient concerns: The patient complained of absent-mindedness since the head trauma, which was mainly observed while dining for several (5–10) minutes approximately 3 to 4 times a day: according to the patient's family, he usually stopped eating while holding the spoon in the air for approximately 5∼10 minutes.

Diagnoses: A 19-year-old man suffered from head trauma resulting from being hit on his head by a falling glass from a large window (1.5 × 2 m, approximately 100 kg) at a cafe.

Interventions: His absent-mindedness showed slow improvement with the passage of time and had almost disappeared at seven months after onset.

Outcomes: The lower portion of both lower dorsal ARAS and the upper portion of the left lower ventral ARAS of the patient were thinner, and partial tearing was observed in the right lower ventral ARAS. Decreased neural connectivity of the intralaminar thalamic nucleus to the prefrontal cortex, basal forebrain, parietal cortex, and occipital cortex was detected in both hemispheres.

Lessons: Injury of the ARAS and injury of the cerebral cortex was demonstrated in a patient with absent-mindedness following mild traumatic brain injury. The absent-mindedness in this patient might be related to the injury of the ARAS.

Abbreviations: ARAS = ascending reticular activating system, DTT = diffusion tensor tractography, ROI = regions of interest.

Keywords: absent-mindedness, ascending reticular activating system, mild traumatic brain injury

1. Introduction

Absent-mindedness, characterized by lapses of conscious awareness, could impair goal-directed behavior and could be a serious consequence of trauma such as a traffic accident.[1,2] Elucidation of the pathogenetic mechanism of absent-mindedness would be clinically important in development of a guide for management of absent-mindedness. The pathogenetic mechanism of absent-mindedness has not been elucidated, however absent-mindedness following brain injury has been regarded as an attentional problem.[2–4] Therefore, relation of the brain areas relevant to attention including the prefrontal cortex, anterior cingulate cortex, and fronto-parietal cortex with absent-mindedness has been suggested.[2–4] However, no study on the association of absent-mindedness and injury of the ascending reticular activating system (ARAS) has been reported so far.

In the current study, we report on a patient who developed absent-mindedness, and injury of the ARAS following mild traumatic brain injury.

2. Case report

A 19-year-old man patient, who had no history of a neurological or psychiatric disorder, suffered from head trauma resulting from being hit on his head by a falling glass from a large window (1.5 × 2 m, approximately 100 kg) at a cafe, however, the glass was not broken. He experienced post-traumatic amnesia for approximately 3 min without loss of consciousness. The patient's Glasgow Coma Scale score was 15. No specific lesion was observed on the conventional brain MRI performed at 2 months after onset. The patient complained of absent-mindedness since the head trauma, which was mainly observed while dining for several (5–10) minutes approximately 3 to 4 times a day himself: according to the patient's family, he usually stopped eating while holding the spoon in the air for approximately 5 to 10 minutes. Sometimes, he could stop his absent-minded symptom with other people's order when it extended too much over 10 to 15 minutes.

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Although he was an extrovertive and active person before the head trauma without attention problem, he changed as an introvertive person. In addition, he also suffered from dizziness, insomnia, and demotivation. However, he did not show excessive daytime-sleepness, depression, or seizure-like symptoms. Methylphenidate was prescribed since 3 months after onset. His absent-mindedness showed slow improvement with the passage of time and had almost disappeared at seven months after onset. The patient provided an informed consent, and the study protocol was approved by our institutional review board.

Diffusion tensor imaging data were acquired at 2 months after onset of head trauma using a 1.5T Philips Gyroscan Intera (Philips, Ltd Best, The Netherlands) with 32 noncollinear diffusion sensitizing gradients by single-shot echo-planar imaging. Imaging parameters were as follows: acquisition matrix = 96×96; reconstructed to matrix = 192×192; field of view = $240 \times 240\,mm^2$; TR = 10,398 ms; TE = 72 ms; parallel imaging reduction factor (SENSE factor) = 2; EPI factor = 59; $b = 1000\,s/mm^2$; slice gap = 0 mm and a slice thickness = 2.5 mm. The Oxford Centre for Functional Magnetic Resonance Imaging of the Brain (FMRIB) Software Library was used for analysis of diffusion tensor imaging data. A probabilistic tractography method based on a multifiber model, and FMRIB Diffusion Software with routines option (0.5 mm step lengths, 5000 streamline samples, curvature thresholds = 0.2) were used for fiber tracking.[5] We evaluated 3 portions of the ARAS: the lower dorsal ARAS, the lower ventral ARAS, and the upper ARAS. Three portions of the ARAS were reconstructed by selection of fibers passing through regions of interest (ROIs) as follows[6–8]: the dorsal lower ARAS (seed ROI: the pontine reticular formation, target ROI: the thalamic intralaminar nucleus),[6] the ventral lower ARAS (seed

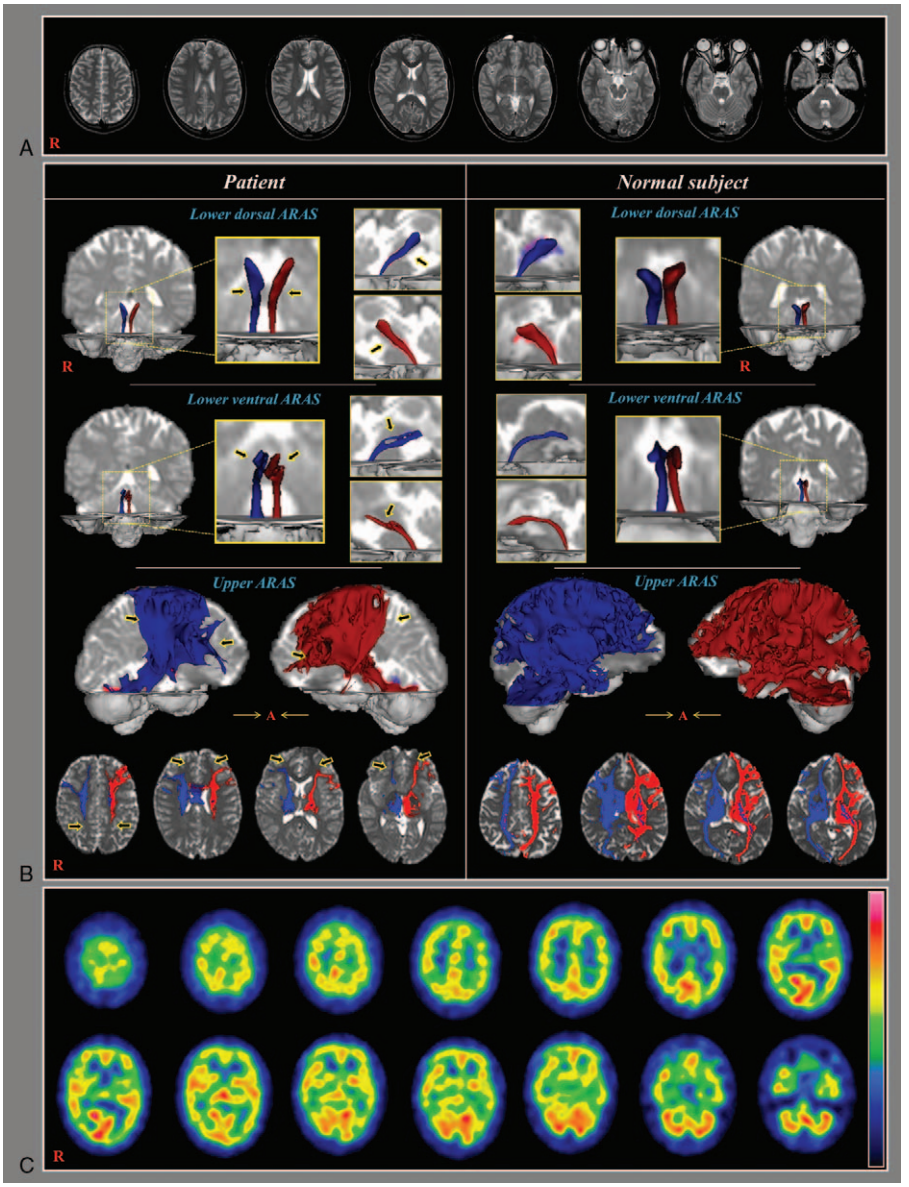

Figure 1. (A) Brain MR images taken at 2 months after onset show no abnormal lesion. (B) Results of diffusion tensor tractography at 2 months after onset. The lower portion of both lower dorsal ascending reticular activating systems (ARAS) and the upper portion of the left lower ventral ARAS of the patient are thinner compared with a normal control subject (20-year old male), and partial tearing is observed in the right lower ventral ARAS. Decreased neural connectivity of the thalamic intralaminar nucleus to the prefrontal cortex, basal forebrain, parietal cortex, and occipital cortex was deyected in both hemispheres. RF: reticular formation, ILN: intralaminar thalamic nucleus. (C) Brain SPECT (Single Photon Emission Computed Tomography; Tc-99m ECD) at 3 months after onset shows mild reduction of tracer uptake in the brain stem and both fronto-parieto-temporal lobes. ARAS = ascending reticular activating systems, RF = reticular formation, ILN = intralaminar thalamic nucleus.

ROI: the hypothalamus, target ROI: the pontine reticular formation),[8] and the upper ARAS (the neural connectivity of the thalamic intralaminar nucleus (seed ROI) to the cerebral cortex was analyzed).[7]

The lower portion of both lower dorsal ARAS and the upper portion of the left lower ventral ARAS of the patient were thinner compared with a normal subject, and partial tearing was observed in the right lower ventral ARAS. Decreased neural connectivity from the intralaminar thalamic nucleus to the prefrontal cortex, basal forebrain, parietal cortex, and occipital cortex was detected in both hemispheres. Brain SPECT (Tc-99m ECD), which performed at 3 months after onset, showed a mild reduction of tracer uptake in the brain stem and both fronto-parieto-temporal lobes (Fig. 1).

3. Discussion

In this study, using diffusion tensor tractography, 3 portions of the ARAS were evaluated in a patient with absent-mindedness following mild traumatic brain injury. According to our findings, injuries were observed in all 3 portions of the ARAS: thinning of both the lower dorsal and the left lower ventral ARAS, tearing of the right lower ventral ARAS, decreased neural connectivity to the basal forebrain, prefrontal cortex, and parietal lobe, which are important areas of the brain for consciousness.[9–11] The findings of the ARAS appear to be compatible with the findings of brain SPECT showing reduction of tracer uptake in the brain stem and both fronto-parieto-temporal lobes. Our results suggest that the injury of the ARAS might be a pathogenetic mechanism of absent-mindedness as well as injury of the cerebral cortex.

A few neuroimaging studies have been reported so far.[2,4] In 2006, using functional MRI, Weissman et al[2] reported that attentional lapses begin with reduced prestimulus activity in the anterior cingulate and right prefrontal regions involved in controlling attention and less efficient stimulus processing during attentional lapses was associated with increased activity in widespread regions of the frontal and parietal cortex. They also found that the mechanism for recovery from attentional lapses was related to increased stimulus-evoked activity in the right inferior frontal gyrus and the right temporal-parietal junction.[2] Chee et al reported that sleep deprivation-related lapses differ from lapses of equivalent duration after a normal night's sleep by reduced ability of frontal and parietal control regions to raise activation in response to lapses, dramatically reduced visual sensory cortex activation, and reduced thalamic activation during lapses that contrasted with elevated thalamic activation during nonlapse periods, using functional MRI.[4] Therefore, to the best of our knowledge, this is the first study to demonstrate injury of the ARAS in a patient with absent-mindedness following brain injury. However, several limitations of this study should be considered. First, the diffusion tensor tractography (DTT) technique is operator-dependent. Second, DTT may underestimate the fiber tracts due to crossing in regions of fiber complexity.[12–14] Third, We did not performed electroencephalography to rule out the partial seizure and DTT for the other nerual tracts which are related with attention and prefrontal function.

In conclusion, injury of the ARAS and injury of the cerebral cortex was demonstrated in a patient with absent-mindedness following mild traumatic brain injury. The absent-mindedness in this patient might be related to the injury of the ARAS. Further studies on this topic should be encouraged.

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