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【 CASE REPORT 】

Rotatory Vertigo Caused by a Small Hemorrhage in the Superior Temporal Gyrus

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Abstract:
Rotatory vertigo is known to have not only peripheral causes, e.g., Meniere's disease, vestibular neuritis, and benign paroxysmal positional vertigo, but also central causes, e.g., stroke, hemorrhage, and tumor. In most cases, central rotatory vertigo is caused by a lesion in the brainstem or cerebellum, but rare cases with a cerebral lesion have also been reported. We herin describe a unique case with acute rotatory vertigo following a small hemorrhage in the left superior temporal gyrus, which probably led to a dysfunction of the visual-vestibular system.

Key words: rotatory vertigo, superior temporal gyrus, vestibular cortex

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Introduction

Rotatory vertigo is a vestibular symptom that can be divided into peripheral causes, e.g., Meniere's disease, vestibular neuritis, and benign paroxysmal positional vertigo, and central causes, e.g., stroke, hemorrhage, and tumor (1, 2). Such vestibular symptoms are associated with the vestibular cortical system, involving the parietoinsular vestibular cortex in monkeys (3) and the temporo-peri-Sylvian vestibular cortex, posterior insular cortex, and inferior parietal lobe in humans (3-5). In the vestibular cortical system, the superior temporal gyrus (STG) is a cortical association area in which visual, somatosensory, and auditory information as well as high-order vestibular perception are processed (4-8). The present report describes a rare case with susceptibility to rotatory vertigo caused by a focal lesion in the left STG.

Case Report

A 60-year-old man was referred to our department because of sudden vertigo, similar to feeling drunk, which he experienced upon standing. He felt unsteady and had weakness of his right leg. When he got in his car, he found it difficult to push on the accelerator pedal with his right leg.

His medical history was notable for a 6-year history of hypertension without treatment. On examination at 5 hours after the onset of symptoms, he could stand unaided, but tended to fall without a directional preponderance. He had no auditory symptoms including hearing loss, tinnitus, and vomiting. The vertigo occurred continuously and worsened upon standing. His vital signs were calculated to be: systolic blood pressure 172 mmHg, diastolic blood pressure 110 mmHg, heart rate 60 beats per minute, respiratory rate 16 breaths per minute, and body temperature 36.2℃. Examination of the cranial nerves showed no abnormalities; there was no nystagmus, and his eye movements were normal. Neurological examinations of the limbs (sensory function, coordination, and reflexes) were normal. He had no weakness of the limbs, other than the right leg. Romberg testing showed abnormally increased body sway (especially with closed eyes). The right leg was positive for the Mingazzini test, while the bilateral arms were negative for Barré's sign.

Head computed tomography imaging showed a small hyper-dense area in the left STG (Figure A). Magnetic resonance imaging on transverse susceptibility-weighted images (Figure B) and coronal and sagittal fluid-attenuated inversion recovery images (Figure C, D) also showed a small (1.1×1.3 cm) hemorrhage in the left STG. There were no lesions of the brain stem or cerebellum on head magnetic resonance imaging. He was admitted with a diagnosis of an acute small hemorrhage, with a suspected cavernous heman-

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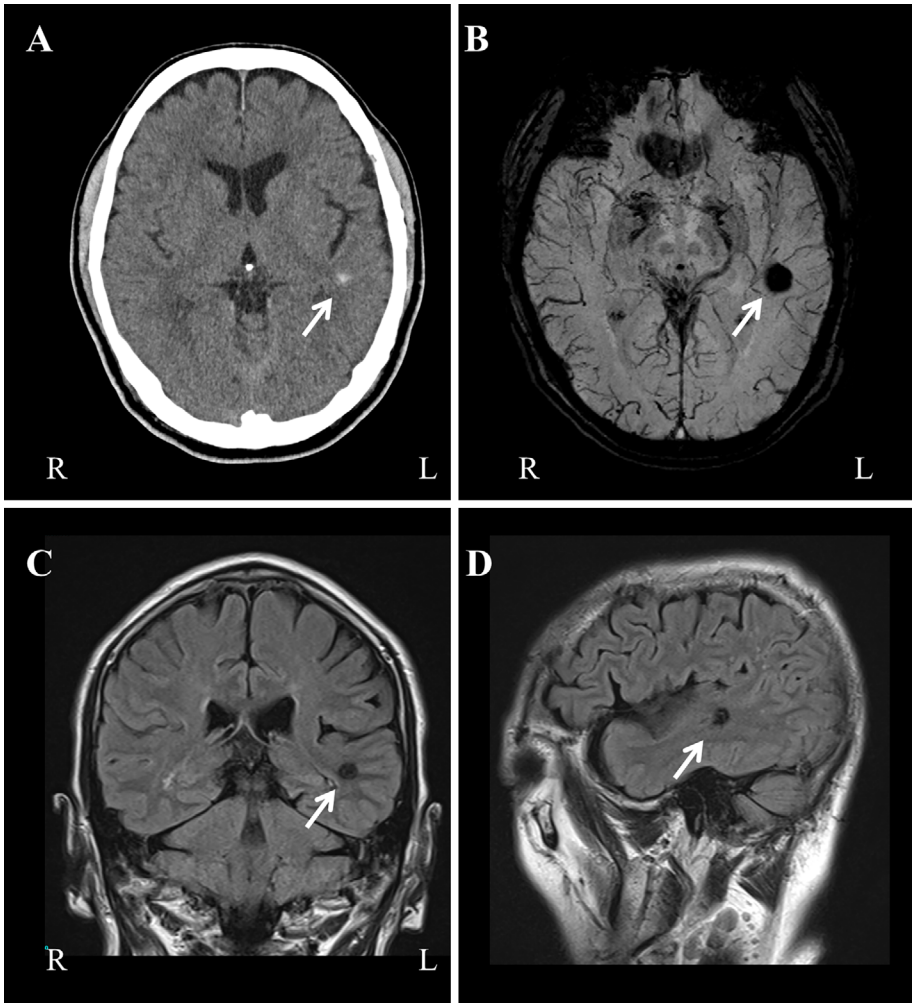

Figure. **A: Head computed tomography image showing a hyper-dense area in the left superior temporal gyrus (STG). B: Transverse susceptibility-weighted image. C: Coronal fluid-attenuated inversion recovery (FLAIR) image. D: Sagittal FLAIR image. Each white arrow shows a hyper-dense area located in the left STG. The area is consistent with a cavernous hemangioma.**

gioma.

At 2 days after admission, optokinetic nystagmus appeared and his bilateral arms deviated to the left on an arm deviation test. At 8 days after admission, he felt his body axis rotating to the right. At 9 days after admission, optokinetic nystagmus disappeared with his rotatory vertigo improvement and he was discharged home. After discharge, the patient is now 71 years of age. He has experienced rotatory vertigo without nystagmus every 2 years to date which lasts a few days and is induced by head motion to the left.

Discussion

The present case developed rotatory vertigo after suffering a small cortical hemorrhage. His presentation is unique due to the presence of a focal lesion in the left posterior STG, a part of the vestibular cortical system.

Imaging studies have shown that the vestibular cortical system in humans, as in primates, includes parietal areas (areas 2v and 7) as well as the premotor regions of the frontal cortex, temporal areas (visual posterior Sylvian area and medial superior temporal area), and a central core region corresponding to the parietoinsular vestibular cortex, which consists of part of the granular insular and retro insular regions close to the acoustic cortex (7, 9-11). In previous case reports, sixteen patients experienced central rotatory vertigo, which was induced by a cerebral lesion in the temporo-peri-Sylvian vestibular cortex, posterior insular cortex, or inferior parietal lobe (2, 8, 10, 12-18). Thirteen out of 16 patients were over 50 years old (age 26-82 years, mean ± standard deviation 56±16 years), and there was right lateralization of the lesions responsible for vestibular cortical system (12 right and 4 left lesion sides) (2, 8, 10, 12-18). However, patients do not necessarily exhibit rotatory vertigo when these areas are affected (14, 19). Most vestibular functions can rely on visual and somatosensory input (5, 20), so that visual-vestibular interactions might be important for rotatory vertigo, based on motion perception and orientation (19). To our knowledge, the present case is a rare description of susceptibility to rotatory vertigo caused by a small lesion in the left, dominant, posterior STG, which might be a very important component of visual-vestibular interactions.

The authors state that they have no Conflict of Interest (COI).

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