

Case Report

## 전정피질경색과 연관된 회전성 현훈과 불안정보행

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### Rotational Vertigo and Unsteady Gait Associated with Vestibular Cortical Infarction

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A 77-year-old man developed acute vertigo and unsteady gait. Neurological examination revealed spontaneous left-beating nystagmus in the primary position. He fell to the left when walking without support. Magnetic resonance imaging showed an acute infarction involving the right parieto-temporal lobe. Although the vertigo and unsteady gait are most often associated with vestibular disorders involving the infratentorial structures, those may occur in cerebral infarction of the parieto-temporal lobe. (Korean J Clin Neurophysiol 2014;16:32-34)

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Vertigo is most often associated with vestibular disorders involving the infratentorial structures such as the semi-circular canals, vestibular nerve, brainstem, or cerebellum.<sup>1,2</sup> However, since vestibular signal reach the vestibular cortex through the thalamus, any supratentorial lesion that disturbs this transmission can also produce vestibular dysfunction.<sup>2</sup>

Here, we report a rare case of parieto-temporal infarction that presented with rotational vertigo and unsteady gait.

### Case Report

A right-handed 77-year-old man developed acute rotational vertigo and unsteady gait 5 days prior to admission. He also complained of nausea and vomiting. The vertigo was not posture-related and was present throughout the day. He denied tinnitus, sensory disturbances, or muscle weakness. He was taking antihypertensive medication. His electrocardiogram showed atrial fibrillation. On admission, neurological examination revealed spontaneous left-beating nystagmus in the primary and every eccentric position. The nystagmus was not suppressed by visual fixation. A head impulse test was normal in both horizontal directions. The pupils were symmetric and reactive to light. His range of eye movements was full. He was able to stand unassisted, but could not stand with his feet together. He also fell to the left

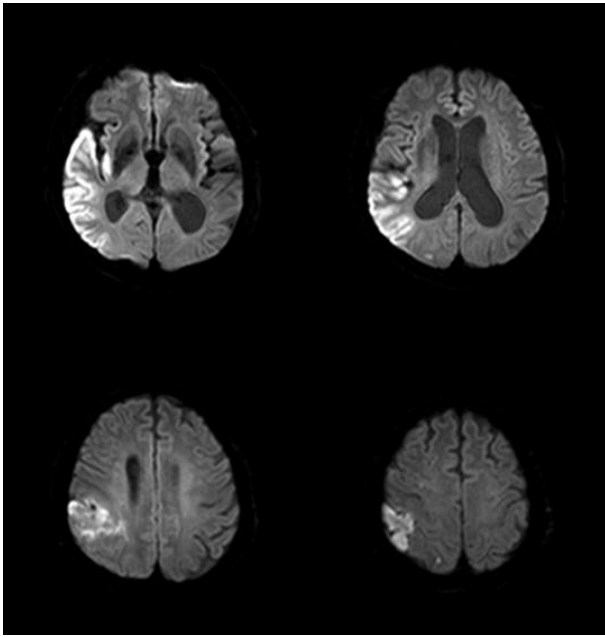
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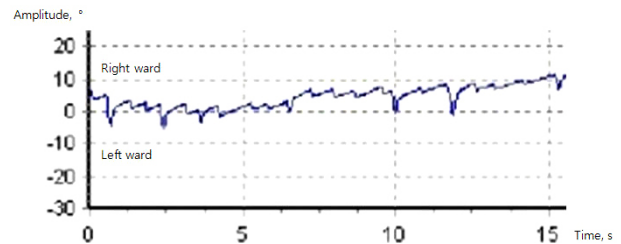
**Figure 1.** Diffusion-weighted magnetic resonance images indicate an acute infarction of the right parieto-temporal lobe.

when walking without support. He did not show any problems with appendicular coordination such as dysmetria or dysidiadochokinesia. Findings of motor and sensory examinations were normal with normoactive deep tendon reflexes. The remainder of the neurologic examinations was unremarkable.

Magnetic resonance imaging (MRI) performed 5 days after the onset of symptoms showed an acute infarction involving the right parieto-temporal lobe on diffusion-weighted images (Fig. 1). Computed tomographic angiography showed a moderate focal stenosis in the left carotid bulb. Video-based oculography, 7 days after the onset of symptoms, documented spontaneous left-beating nystagmus (Fig. 2) (Supplementary material 1). Bithermal caloric tests were normal. Electroencephalography (EEG) showed no epileptiform discharges. He began to take warfarin to prevent a secondary vascular event. His rotational vertigo and unsteady gait improved gradually, and he was discharged 10 days later.

## Discussion

This patient complained of acute rotational vertigo and unsteady gait, and neurological examination revealed spontaneous left-beating nystagmus and axial lateropulsion



**Figure 2.** Video-based oculography shows a left-beating horizontal nystagmus in the primary position with a 1-2 Hz frequency, a mean slow-phase velocity of 3°/s, and an amplitude 3-5° (horizontal recording).

to the left side. MRIs revealed an acute infarction in the parieto-temporal lobe without any lesions of the cerebellum or brainstem. MRI was performed on this patient 5 days after the onset of symptoms, which allowed sufficient time to discover the lesion on diffusion-weighted images. Thus, the possibility that another lesion at the infratentorial level was missed is low. His spontaneous left-beating nystagmus not suppressed during visual fixation, axial lateropulsion, and normal head impulse tests and calorics suggested central vertigo. Seizure is an unlikely cause of vertigo because the symptoms lasted throughout the day, and epileptiform discharges were not found on EEG. Thus, the parieto-temporal infarction appears to be the cause of his vertigo and unsteady gait.

The vestibular cortex receives vestibular afferents via thalamus, and projects down to the vestibular nuclei in the brainstem. In animal studies, area 2v, area 3av, area 7, the parieto-insular cortex, the temporal sylvian area, and the medial superior temporal area have been identified as the vestibular cortices.<sup>3</sup> However, in humans, the precise localization and functions of the vestibular cortex are unknown. Recently, studies using cerebral mapping or functional neuroimaging have suggested that the parieto-temporal cortex is likely a part of the vestibular cortex.<sup>4,5</sup> Specifically, the posterior insular cortex and surrounding regions have been identified as essential for vestibular otolith perception and are considered to be the core regions of the vestibular cortex.<sup>4,5</sup> Thus, ischemic infarction involving the posterior insular cortex and surrounding regions could cause rotational vertigo and unsteady gait. Our patient had the lesions involving the posterior insular cortex and other areas of the parieto-temporal lobe. Indeed, there have been a few other

reports on cerebral infarction involving the insular cortex and presenting with rotational vertigo or unsteady gait.<sup>1,3,4</sup> Although our patient did not permit a precise localization of the vestibular cortex due to the relatively large size of infarction, this case demonstrated that ischemic infarction of the portion of the parieto-temporal lobe, which may be the part of the vestibular cortex, can produce nystagmus.

Vestibular dysfunction may be the most reasonable explanation for the nystagmus in cerebral infarctions involving the insular cortex. A previous report suggested that the insular cortex shares significant overlap in vestibular connectivity with portions of the frontal cortex that are responsible for generation of saccadic eye movements and possibly the fast phase of nystagmus.<sup>5</sup> Thus, cerebral infarction involving the insular cortex, as in our patient, may produce nystagmus. Insular cortical infarction may also explain our patient's unsteady gait. The insular cortex is related to the parietal cortex, which is connected to the spinocerebellum.<sup>2</sup> Thus, insular cortical infarction could produce spinocerebellar dysfunction that would result in axial lateropulsion. Another possible explanation is that the lesions to the insular cortex may have induced axial lateropulsion by involving the dentatorubrothalamic tract. The insula also has connections with the thalamus that participates in the dentatorubrothalamic tract.<sup>2</sup> Thus, an insular cortical lesion may also induce cerebellar dysfunction and resultant axial lateropulsion.

Cerebral hemispheric lesions involving the human homologues of medial temporal and middle superior temporal areas in monkeys can induce pursuit-paretic nystagmus due

to impairment of controlling horizontal smooth pursuit.<sup>6,7</sup> However, pursuit-paretic nystagmus usually beats toward the side of the lesion, whereas the nystagmus in our patient beats away from the side of the lesion.<sup>6,7</sup> Thus the possibility of pursuit-paretic nystagmus was low in our patient.

We conclude that the rotational vertigo and unsteady gait in our patient resulted from a cerebral infarction of the parieto-temporal lobe.

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