

Rotational Vertigo Associated with Putaminal Infarction

Makoto Nakajima, MD,* Yuichiro Inatomi, MD,* Toshiro Yonehara, MD,*
Teruyuki Hirano, MD,† and Makoto Uchino, MD‡

A 57-year-old man was admitted because of left hemiparesis. A magnetic resonance imaging scan of the brain revealed a recent infarct from the dorsal side of the right putamen to the corona radiata. Three hours after onset, he complained of rotational vertigo. Truncal ataxia was also found on standing a few days later, which persisted until the chronic phase. Repeat magnetic resonance imaging scans on days 2 and 11 revealed no additional lesions. The present case indicates that rotational vertigo might result from a small, supratentorial, subcortical lesion. **Key Words:** Nystagmus—stroke—supratentorial—truncal ataxia—vertigo—vestibular pathway.

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Rotational vertigo is rarely seen as a symptom of acute stroke patients with supratentorial infarction,^{1,2} even in a large prospective study.³ A patient with a small putaminal infarction who developed rotational vertigo in the acute phase is described.

Case Report

A 57-year-old man with sudden left-sided weakness was transferred to our hospital. He had been prescribed an antihypertensive agent and aspirin because of hypertension and asymptomatic multiple lacunar infarctions. On admission, the patient had mild left-sided palsy. A magnetic resonance imaging (MRI) scan of the brain revealed a vague hyperintensity lesion from the dorsal side of the right putamen to the corona radiata (Figs 1A-1E). Multiple old ischemic lesions were observed in the bilateral subcortex, thalamus, and pons. Three

hours after onset, the patient suddenly complained of rotational vertigo and vomited. He displayed spontaneous nystagmus to the left side in all eye positions. An MRI scan of the brain on day 2 revealed no additional lesions. An MRI on day 11 (Figs 1F-1O) also revealed no other recent lesions. An electroencephalogram revealed no abnormal findings. Although his vertigo, nystagmus, and the mild left-sided hemiparesis decreased rapidly, truncal ataxia was found when the patient stood up. Even after 2 months of rehabilitation, truncal ataxia was persistent.

Discussion

A rare case with rotational vertigo presumably caused by a small subcortical infarction was described. An early seizure^{3,4} was an unlikely cause of the vertigo because no epileptic discharges were confirmed on electroencephalographic examination. Peripheral vertigo was also unlikely, because truncal ataxia was sustained for several months. Old ischemic lesions—in particular, lesions in the dorsal pons—may also have affected the vestibular afferent pathways in our patient. Another possible mechanism was that truncal ataxia and/or rotatory vertigo might be related to the new brainstem or cerebellar ischemic lesions, which could not be detected on the repeated MRI.

The human vestibular cortex is considered to localize in the anteroparietal cortex near the parietoinsular

From the *Departments of Neurology at the Stroke Center, Saiseikai Kumamoto Hospital; †Faculty of Life Science, Kumamoto University; and ‡Jonan Hospital, Kumamoto, Japan.

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Address correspondence to Makoto Nakajima, MD, Department of Neurology, Stroke Center, Saiseikai Kumamoto Hospital, Chikami 5-3-1, Kumamoto 861-4193, Japan. E-mail: nakajima04@gmail.com.

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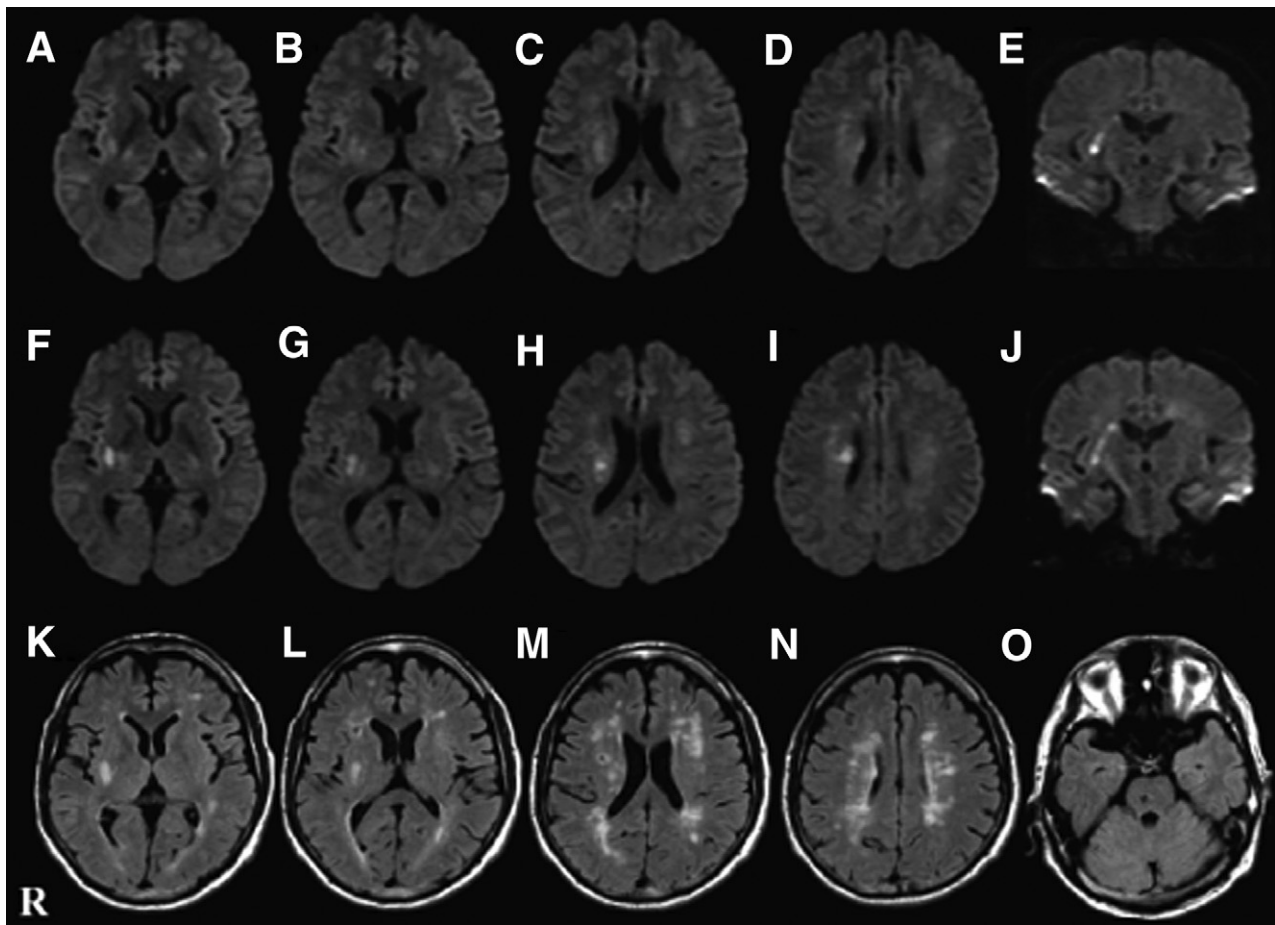


Figure 1. Diffusion-weighted imaging (DWI; repetition time [TR] 6000 ms; echo delay time [TE] 100 ms; $b = 1000$) on day 1 (A-D, axial view; E, coronal view). A vague hyperintense lesion is shown from the posterior putamen to the corona radiata. The hyperintense lesion becomes obvious and slightly enlarged on DWI on day 11 (F-K, axial view; J, coronal view). Fluid-attenuated inversion recovery (FLAIR; TR 6100 ms; TE 105 ms) imaging on the same day reveals an old ischemic lesion in the middle pons and bilateral white matter change in the cerebrum, including the index infarction (K-O).

vestibular cortex (PIVC). Brandt et al⁵ reported that an overlapping area of ischemic lesions was identified in stroke patients with vertigo. The small putaminal infarction in our patient would impair the afferent pathway from the ipsilateral posterior parts of the ventroposterior complex and the medial pulvinar to the PIVC.⁶

Some authors have reported single cases of rotational vertigo with supratentorial cerebrovascular lesions^{1,2,6,7} or cerebral venous thrombosis.⁸ They speculated that the supratentorial lesions affected a part of the vestibular cortex. In other reports, predominance of the right hemisphere in spatial perception has been described.^{5,9,10}

While stroke patients commonly have putaminal lesions, they rarely complain of rotational vertigo.^{3,6} Possible hypotheses are that many afferent pathways might exist in the supratentorium, or that vestibular function of the cortex exists bilaterally.⁵

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