Isolated Vestibular Nucleus Infarction Mimicking Acute Peripheral Vestibulopathy

Hyun-Ah Kim, MD; Hyung Lee, MD

Background and Purpose—Although several articles have been published on central vestibular syndrome mimicking acute peripheral vestibulopathy (ie, pseudo–acute peripheral vestibulopathy), there are no reports of a brainstem infarct that selectively involves the vestibular nucleus and causes isolated vertigo.

Summary of Case—We report a patient with an isolated vestibular nucleus infarction who presented with isolated prolonged vertigo, spontaneous horizontal nystagmus with a torsional component, a positive head impulse test result, and unilateral canal paresis to caloric stimulation.

Conclusions—This is the first report of pseudo–acute peripheral vestibulopathy associated with isolated vestibular nucleus infarction. Isolated vestibular nucleus infarction should be considered in the differential diagnosis of central vascular vertigo syndrome, especially when the patient has unilateral canal paresis but without other neurologic symptoms or signs. **(*Stroke*. 2010;41:1558-1560.)**

Key Words: infarction ■ isolated vertigo ■ pseudo-acute peripheral vestibulopathy ■ vestibular nucleus

Acute peripheral vestibulopathy (APV) is characterized by acute prolonged vertigo (lasting several days), spontaneous horizontal nystagmus with a torsional component beating toward the unaffected side, postural imbalance, unilateral canal paresis (CP), and a positive head-impulse test result without other accompanying neurologic or audiologic symptoms or signs.[1] Although several articles[2–7] have been published on central vestibular syndrome mimicking APV (ie, pseudo-APV), most have focused on lesions in the caudal cerebellum[2–5] or the root entry zone of the eighth nerve.[6,7] To the best of our knowledge, we know of only 1 prior report of pseudo-APV associated with vestibular nucleus (VN) infarction.[8] However, in this report, the lesion was not localized to the VN but extended to the proximal portion of the vestibular fascicle, which is also known to be associated with pseudo-APV. Furthermore, the patient in this report had no CP, a prerequisite for the diagnosis of APV. Therefore, in such cases, clinicians may conclude that vertigo is caused by damage to the central vestibular structure. We report a patient with a tiny infarct selectively involving the VN who also had isolated vertigo and unilateral CP.

Clinical Summary

A 53-year-old woman with a history of hypertension and diabetes developed sudden onset of prolonged vertigo with perceived counterclockwise rotation of the environment (ie, from the patient's point of view) and imbalance on the day before her admission. She also had nausea and vomiting without auditory symptoms. On admission, she showed spontaneous left-beating nystagmus with a torsional component, which was attenuated by visual fixation (Figure 1). Horizontal gaze-evoked nystagmus was also evident, which had greater amplitude when she looked in the direction of the spontaneous nystagmus. Horizontal saccade was normal, and smooth pursuit eye movement toward the left side was interrupted by the spontaneous nystagmus. A head-impulse test was positive on the right side. The patient did not have dysarthria, diplopia, ophthalmoparesis, limb weakness, dysmetria, or sensory loss. She could stand without support but veered to the right when she walked. There were no other abnormal findings on neurologic examination. Tests of the subjective visual vertical showed conjugate rightward deviation of the settings (ie, clockwise from the patient's point of view): 9.4° with the right eye, 11.5° with the left eye, and 8.9° with both eyes (normal range, <2.0° for each eye in Kim et al[9]). Fundus photography showed a 14° extorsion of the right eye (normal range, 0.5° to 11.5° in Kim et al[9]) and 0° intorsion of the left eye (normal range, 0.5° to 12° in Kim et al[9]; Figure 2A). She had a skew deviation with a left hypertropia of 5 prism diopters in the primary gaze. A vestibular-evoked myogenic potential test disclosed decreased amplitudes on the right side (normal=asymmetry of amplitudes <15%[9]), but the latencies were normal on both sides (Figure 2B). Video-oculographic recording (SMI, Teltow, Germany; resolution of 0.1°, sampling rate of 60 Hz) of quantitative, bithermal caloric tests revealed a CP of 54% on the right side (Figure 2C). Diffusion-weighted

Received February 25, 2010; accepted March 12, 2010.

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Stroke **is available at http://stroke.ahajournals.org** **DOI: 10.1161/STROKEAHA.110.582783**

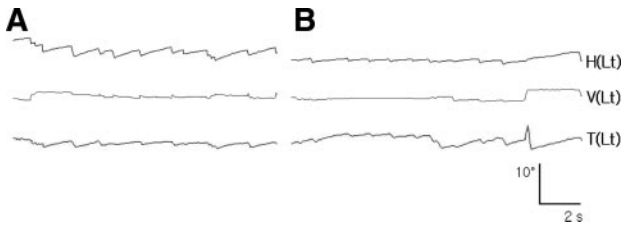

Figure 1. Video-oculographic recording showing spontaneous left-beating nystagmus with counterclockwise torsional and upbeat components (A), which was attenuated by visual fixation (B). H indicates horizontal recording; V, vertical recording; T, torsional recording.

images revealed an acute, tiny infarct selectively involving the right VN, and magnetic resonance angiography disclosed no abnormalities (Figure 3). She was treated with an antiplatelet agent. During a course of several days, vertigo, nystagmus, and unsteadiness subsided. On discharge, she reported only mild dizziness when walking.

Discussion

The summarized symptoms and signs in this case were acute onset of severe, prolonged vertigo, spontaneous nystagmus with horizontal torsional components, attenuation of nystagmus by visual fixation, mild to moderate imbalance, no other accompanying neurologic symptoms or signs, a positive head-impulse test result, unilateral CP, ipsilesional conjugate ocular torsion, and asymmetry in amplitude with no difference in latency on either side by the vestibular-evoked myogenic potential test, all of which are characteristic features of APV. However, our patient had an acute infarct selectively involving the right VN.

Although CP usually indicates a lesion of a peripheral vestibular structure such as the inner ear and the eighth nerve, lesions affecting central vestibular structures such as the root entry zone of the eighth nerve, the proximal portion of the vestibular fascicle, and the VN could also cause CP. In the case of VN, lesions that predominantly affect the medial subnucleus appear to be the most important causes of central CP.[10] Similarly, although a positive head-impulse test result and asymmetry of amplitude with no difference in the latencies of either side by the vestibular-evoked myogenic potential test are usually signs of impairment of unilateral peripheral vestibular function, on rare occasions, central lesions also cause these abnormalities.[11,12] In our case, all of these uncommon signs may be explained by the fact that the VN is involved in the relaying and central processing of peripheral vestibular signals.

In our case, the infarcted area on magnetic resonance imaging is usually supplied by a penetrating branch from the medial branch of the posterior inferior cerebellar artery. Presumably, the mechanism for infarction was small-vessel arteriosclerosis, because there was no obvious source of emboli and the large vessels were normal on magnetic resonance angiography.

We have previously reported pseudo-VN due to cerebellar infarction.[2–4] Together, these reports highlight the importance of isolated, central vertigo syndrome caused by infarction in

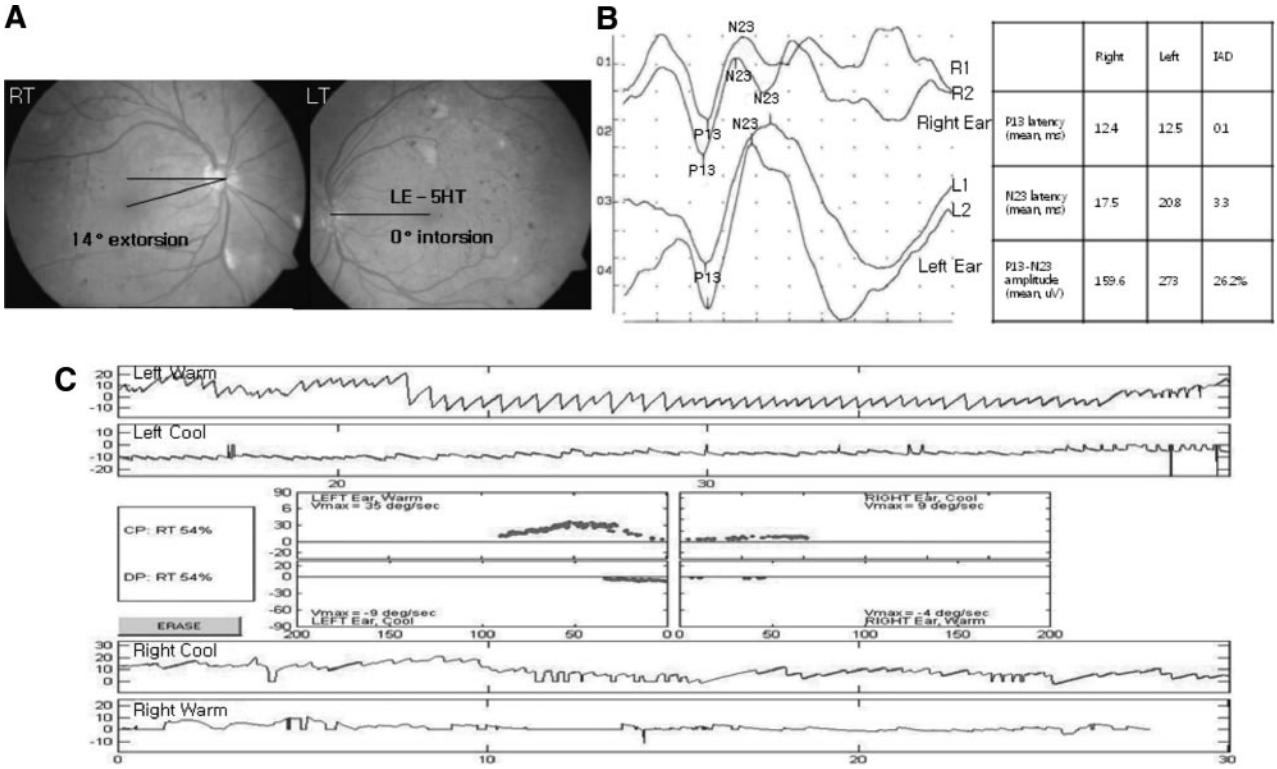

Figure 2. A, Conjugate rightward torsion of the eyes (ie, clockwise from the patient's point of view): 14° extorsion of the right eye and 0° intorsion of the left eye. RT indicates right; LT, left; LE, left eye; HT, hypertropia. B, Vestibular-evoked myogenic potential test demonstrating decreased amplitudes on the right side (26.2%) but normal latencies on both sides. C, Video-oculographic recording of bithermal caloric tests showing right CP (54%). IAD indicates interaural difference.

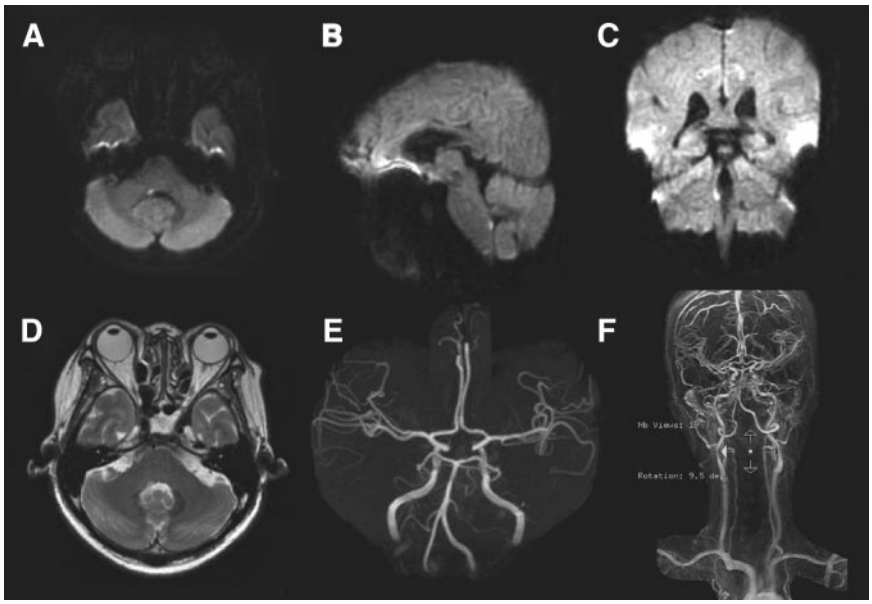

Figure 3. Diffusion-weighted axial (A), sagittal (B), coronal (C), and T2-weighted axial (D) magnetic resonance imaging images of the brain, showing a very small infarct that selectively involved the right VN at the floor of the fourth ventricle at the pontomedullary junction level. Magnetic resonance angiography (E and F) showed no vascular abnormalities.

either the VN or the nodulus.[2–4] Clinicians should be aware of the possibility of isolated VN infarction in patients with acute onset of prolonged vertigo, especially when the patient has unilateral CP, but when other neurologic symptoms or signs are absent.

Disclosures

None.

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Stroke. 2010;41:1558-1560; originally published online May 20, 2010;
doi: 10.1161/STROKEAHA.110.582783
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0039-2499. Online ISSN: 1524-4628