



Video head impulse findings in the ictal period of vestibular migraine

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Dear Sirs,

Vestibular migraine (VM) manifests as central or peripheral vestibular dysfunction during vertigo attacks [1]. However, no studies have reported the findings of video head impulse tests (vHIT) during the ictal period of VM. Herein, we report unique findings of vHIT in a patient with VM during a vertigo attack.

A 40-year-old man presented with positional vertigo without auditory symptoms. Sitting up from the supine position, but not vice versa, aggravated his vertigo. Over a 4-year period, he had suffered from several episodes of vertigo lasting from 48 to 72 h that was followed by a migrainous headache with photophobia, phonophobia, and nausea for 1–2 days. He reported that the clinical features of this vertigo were quite similar to previous recurrent attacks. His symptoms met the Barany Society's diagnostic criteria for vestibular migraine [2]. His father and older sister also had recurrent episodes of headache without vertigo.

On examination, the patient veered slightly to the left and revealed spontaneous nystagmus beating rightward and torsional, with the upper poles of the eyes beating toward the right ear (clockwise) (Fig. 1a). Bedside HIT was unremarkable. Positional maneuvers augmented the spontaneous nystagmus and induced a down-beating component during seated head bending. Adopting a chin-up position while

sitting or lying down reversed the patient's nystagmus into beating leftward, upward, and torsional, with the upper poles of the eyes beating toward the left ear (counterclockwise). Horizontal head shaking augmented the spontaneous nystagmus. Vertigo and positional nystagmus did not improve after repeated canalith-repositioning maneuvers.

Caloric testing revealed 37.5% left side canal paresis (Fig. 1b). Sinusoidal harmonic acceleration tests revealed decreased vestibulo-ocular reflex (VOR) gain, deviation to the left side, and phase lead (Fig. 1c). Step-velocity test showed decreased time constant of the VOR on the left side than on the contralateral side (8 vs. 14 s). VOR cancellation with visual fixation was normal. vHIT revealed covert saccades and early acceleration and premature deceleration (EAPD) during stimulation of the left horizontal (HC) and left posterior semicircular canal (PC), respectively [3] (Fig. 1d). Fundus photography showed counterclockwise ocular torsion (Fig. 1e, 12.8° in the left eye, -11.1° in the right eye; normal range: 0°–12.6°; positive values indicate extorsion). Vestibular-evoked myogenic potentials, pure tone audiometry, and brain magnetic resonance imaging were unremarkable.

Two days after vertigo onset, the patient developed a pulsatile headache that lasted for 24 h. The patient's symptoms disappeared 4 days after vertigo onset. His left side canal paresis improved (21.0%). Follow-up vHITs were performed 4 days, 2 months, and 1 year later. The EAPD patterns gradually disappeared (Fig. 2 and video). There was no recurrence of headache or vertigo over the 1-year follow-up period.

In our patient, the origin of EAPD pattern during vHIT for PC stimulation is difficult to establish. Considering that no similar artifacts have been reported [4] and that the same findings were observed in repeated tests, our findings are unlikely to be artifactual. A previous study reported this pattern in patients with nucleus prepositus hypoglossi lesions during stimulation of the anterior semicircular canal (AC) [3] and concluded it to be due to increased VOR gain resulting from the loss of ipsilateral AC inhibition from the flocculus, which preferentially inhibits AC pathways. However,

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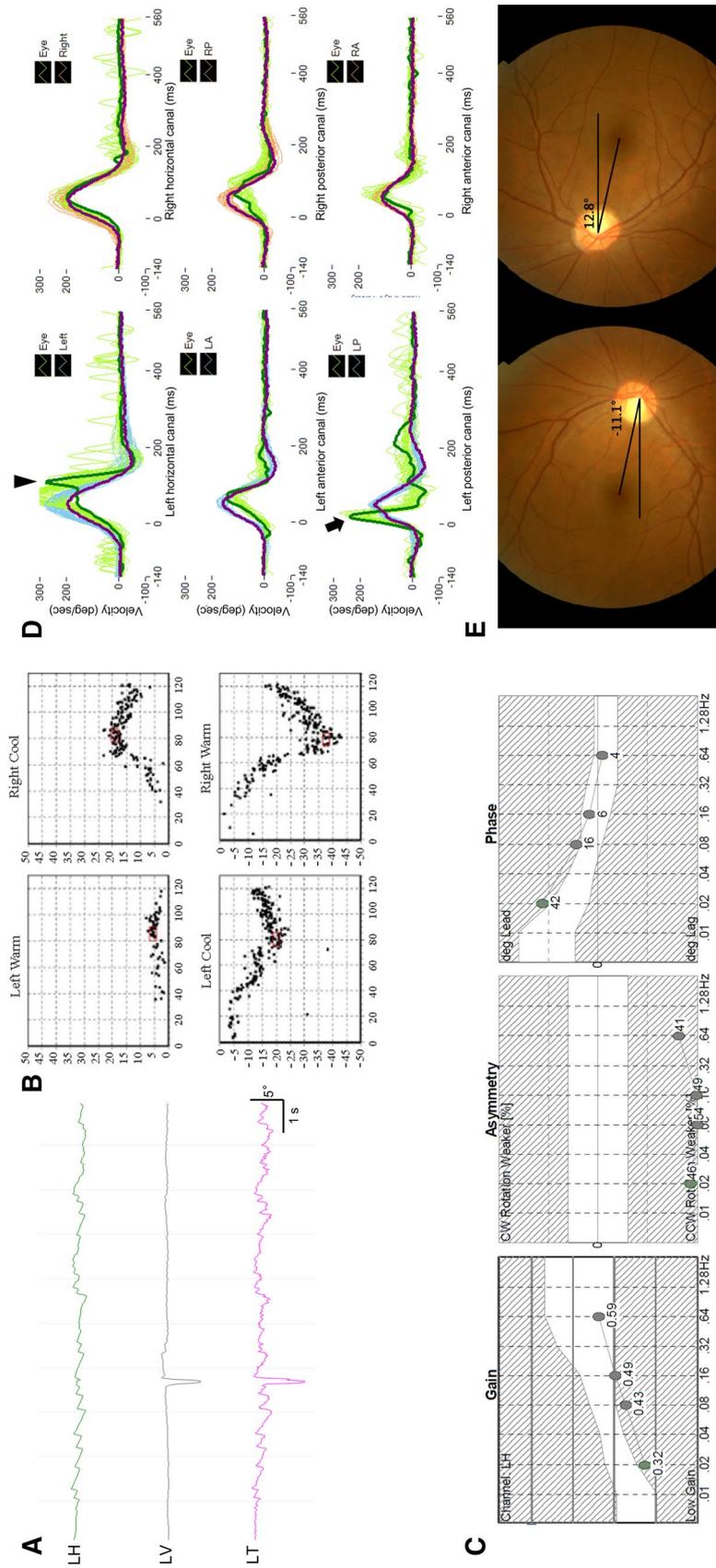


Fig. 1 **a** Video-oculography (3D VNG, SLMED, Seoul, Korea) during an attack showed spontaneous nystagmus beating rightward and torsional, with the upper poles of the eyes beating toward the right ear (clockwise); **b** caloric testing showed 37.53% canal paresis on the left side. **c** Rotatory chair tests revealed a decreased VOR gain, deviation to the left side, phase lead on sinusoidal rotation, and a decreased time constant of the VOR in the step-velocity test. **d** Video head impulse tests revealed covert saccades and early acceleration and premature deceleration during stimulation of the left horizontal and left posterior semicircular canal, respectively. **e** Fundus photography showed counterclockwise ocular torsion

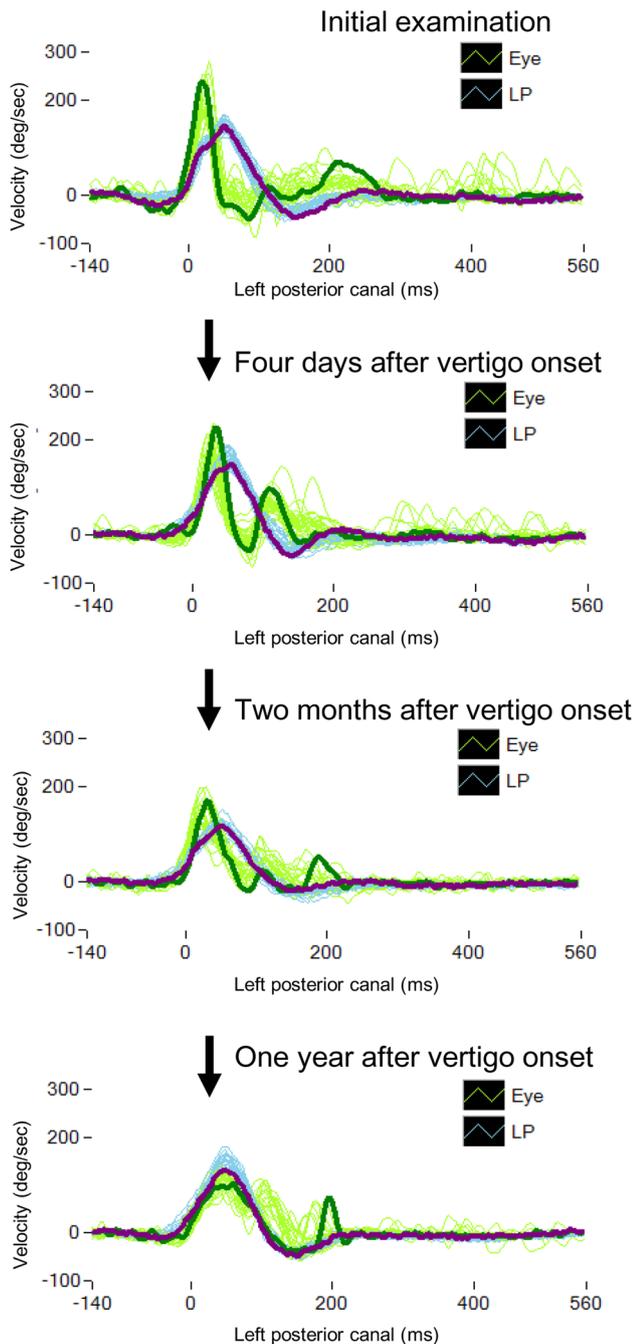


Fig. 2 The video head impulse test findings from the initial test and 4 days, 2 months, and 1 year after vertigo onset. The early acceleration and premature deceleration patterns gradually disappeared throughout the follow-up period

the gain calculated by the area under the curve in our patient was significantly low, and the corrective saccades appeared in the direction of the VOR, suggesting that our finding was due to PC hypofunction rather than hyperfunction. Our findings, which were indicative of HC hypofunction on the same

side as the caloric paresis, covert saccades on vHIT, and decreased VOR gain with phase lead on the rotary chair test, further support this hypothesis. The HC hypofunction seemed to produce peripheral vestibulopathy features; however, these could be caused by central vestibular syndrome such as the involvement of medial vestibular nuclei, flocculonodular lobes, or other neural integrators [5, 6]. The ocular torsion on the fundus suggested the involvement of the graviceptive pathway such as brainstem or otolithic dysfunction [7, 8]. Additionally, the spontaneous nystagmus and its modulation pattern in our patient could not be explained by peripheral vestibular disorders.

In conclusion, the neurologic findings observed during the ictal period in our patient suggest that coexisting central and peripheral vestibulopathy features can be observed and most of the findings were explained only by central vestibular syndrome during the ictal period of VM. The pattern of EAPD is also an indication of vHIT abnormality.

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Conflicts of interest The authors declare that they have no competing interest.

Ethical standards The authors declare that this single case report has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

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