ORIGINAL COMMUNICATION

Isolated vestibular nuclear infarction: report of two cases and review of the literature

Hyo-Jung Kim · Seung-Han Lee · Jae Han Park · Jung-Yoon Choi · Ji-Soo Kim

Abstract Cerebral infarction presenting with isolated vertigo remains a diagnostic challenge. To define the clinical characteristics of unilateral infarctions restricted to the vestibular nuclei, two patients with isolated unilateral vestibular nuclear infarction had bedside and laboratory evaluation of the ocular motor and vestibular function, including video-oculography, bithermal caloric irrigation, the head impulse test (HIT) using magnetic scleral coils, and cervical and ocular vestibular-evoked myogenic potentials (VEMPs). We also reviewed the literature on isolated vertigo from lesions restricted to the vestibular nuclei, and analyzed the clinical features of seven additional patients. Both patients showed spontaneous torsional-horizontal nystagmus that beat away from the lesion side, and direction-changing gaze-evoked nystagmus. Recording of HIT using a magnetic search coil system documented decreased gains of the vestibular-ocular reflex for the horizontal and posterior semicircular canals on both sides, but more for the ipsilesional canals. Bithermal caloric tests showed ipsilesional canal paresis in both patients. Cervical and ocular VEMPs showed decreased or absent responses during stimulation of the ipsilesional ear. Initial MRIs including diffusion-weighted images were normal or equivocal, but follow-up imaging disclosed a circumscribed acute infarction in the area of the vestibular nuclei. Infarctions restricted to the vestibular nuclei may present with isolated vertigo with features of both peripheral and central vestibulopathies. Central signs should be sought even in patients with spontaneous horizontal–torsional nystagmus and positive HIT. In patients with combined peripheral and central vestibulopathy, a vestibular nuclear lesion should be considered especially when hearing is preserved.

Keywords Vestibulopathy · Vestibular nucleus · Lateral medullary infarction · Vertigo

H.-J. Kim and S.-H. Lee equally contributed to this study.

Electronic supplementary material The online version of this article (doi:10.1007/s00415-013-7139-0) contains supplementary material, which is available to authorized users.

H.-J. Kim
Biomedical Research Institute, Seoul National University
Bundang Hospital, Seongnam, Korea

H.-J. Kim · J. H. Park · J.-S. Kim (✉)
Department of Neurology, Seoul National University College
Medicine, Seoul National University Bundang Hospital, 300
Gumi-dong, Bundang-gu, Seongnam, Gyeonggi 463-707, Korea
e-mail: jisookim@snu.ac.kr

S.-H. Lee
Department of Neurology, Chonnam National University
Medical School, Gwangju, Korea

J.-Y. Choi
Department of Neurology, Korea University College of
Medicine, Korea University Ansan Hospital, Ansan, Korea

Introduction

Combine peripheral and central vestibulopathy poses a diagnostic challenge since the clinical features of peripheral vestibulopathy may overshadow the central one, or vice versa in this disorder [1, 2]. The combined peripheral and central vestibulopathy of insidious onset usually indicates a mass lesion involving the cerebellopontine angle [3]. In contrast, an infarction in the territory of anterior inferior cerebellar artery should be a prime suspicion when acute since this artery supplies both the central and peripheral vestibular structures [1, 4, 5]. However, a lesion restricted to the brainstem may also show typical features

of peripheral vestibulopathy when the lesions are located in the vestibular root entry zone or the vestibular nuclei [6]. Isolated vertigo from lesions restricted to the vestibular nuclei has rarely been reported, but mostly without full neurotological evaluation [7–9]. We report two patients who presented with isolated acute spontaneous vertigo with features of both peripheral and central vestibulopathies from a unilateral infarction restricted to the vestibular nuclei. We also reviewed the literature on isolated vertigo from lesions restricted to the vestibular nuclei, and analyzed the clinical features in seven additional patients.

Materials and methods

Case reports

Patient 1

A 34-year-old man with hypertension for 2 years developed acute spontaneous vertigo with vomiting and imbalance. He had no other neurological or auditory symptoms. Examination about 7 h after symptom onset showed
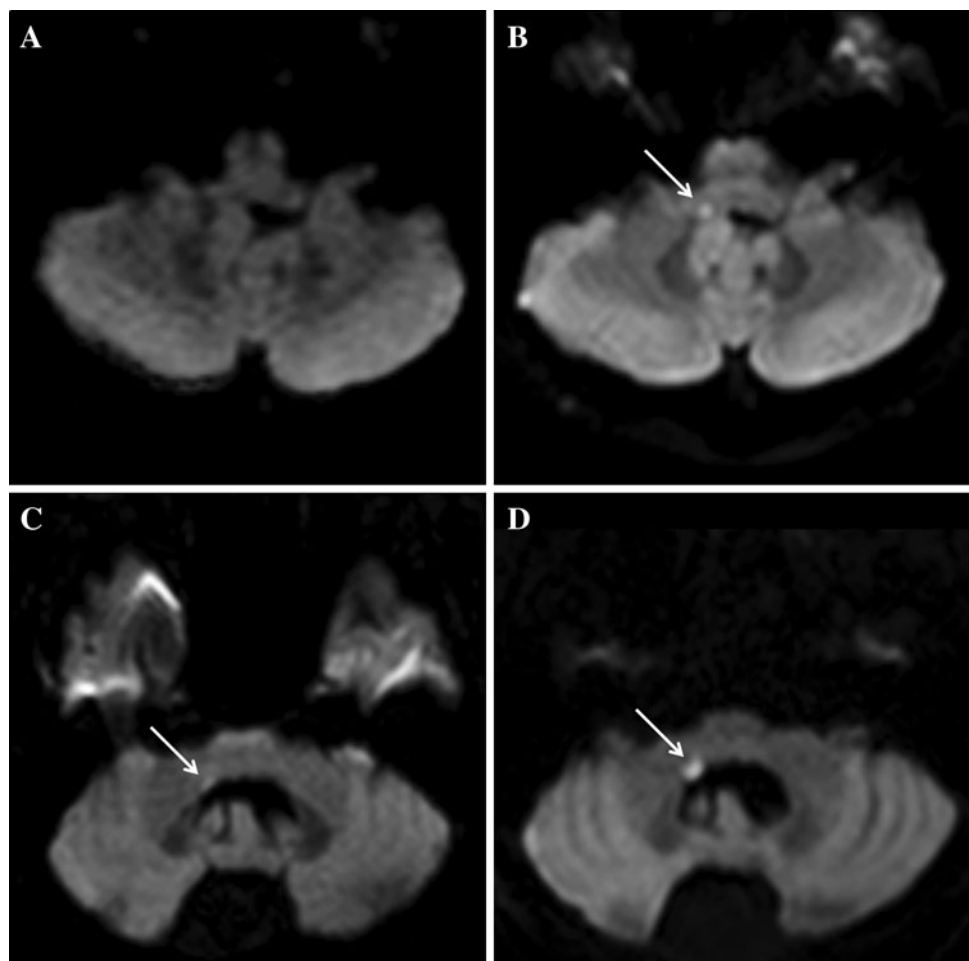
spontaneous left-beating horizontal-torsional nystagmus that increased during left eccentric gaze and with removal of visual fixation, and changed into right beating during rightward gaze (video 1). He had no head tilt or skew deviation. Bedside head impulse tests (HIT) were positive for the right horizontal semicircular canal (HC) (video 2). Initial MRIs including diffusion-weighted images 4 h after symptom onset were normal (Fig. 1a), but follow-up diffusion-weighted MRIs 4 days later disclosed a small acute infarction restricted to the right medial vestibular nucleus (Fig. 1b).

The patient was treated with antiplatelet and antihypertensive medication. The vertigo and imbalance resolved within 5 days of symptom onset, and follow-up evaluation 1 month later showed resolution of all the abnormal findings observed during the acute phase.

Patient 2

A 67-year-old man with diabetes mellitus developed acute spontaneous vertigo with vomiting and imbalance in the absence of other neurological or auditory symptoms. Examination 2 days after symptom onset showed

Fig. 1 MRIs of the patients. In patient 1, the initial diffusion-weighted MRIs 4 h after symptom onset were normal (a), but follow-up imaging 4 days later disclosed acute tiny infarction (arrow) in the area of right medial vestibular nucleus (b). In patient 2, the initial diffusion-weighted MRIs 2 days after the symptom onset showed a suspicious lesion in the middle cerebellar peduncle (c, arrow), but follow-up imaging 5 days later disclosed a definite acute infarction (arrow) in the area of right medial and inferior vestibular nuclei (d)

spontaneous left-beating horizontal-torsional nystagmus that increased with removal of visual fixation and during left eccentric gaze, and changed into right beating during rightward gaze (video 3). He had no head tilt or skew deviation. Bedside HITs were positive for the HCs and the posterior semicircular canals (PCs) on both sides (video 4). Initial MRIs including diffusion-weighted images 2 days after symptom onset showed an indistinct lesion in the right middle cerebellar peduncle (Fig. 1c), but follow-up diffusion-weighted MRIs 5 days later disclosed a definite acute infarction restricted to the right medial and inferior vestibular nuclei (Fig. 1d).

The patient was discharged with mild dizziness and imbalance seven days after symptom onset.

Bedside neurotologic examinations

Both patients had evaluation of spontaneous and gaze-evoked nystagmus (GEN) with fixation. Spontaneous, vibration-induced (VIN) and head-shaking nystagmus (HSN) were also observed on a video monitor without fixation using video Frenzel goggles (SLMED, Seoul, Korea). Bedside HITs were performed manually with a rapid rotation of the head of $\sim 20°$ amplitude in the planes of the HC and the vertical canals. HIT was considered abnormal if a corrective saccade supplemented the inadequate slow phase in the plane of the semicircular canal stimulated [10].

Oculography

Nystagmus was recorded binocularly at a sampling rate of 60 Hz using a video-oculography (SensoMotoric Instruments, Teltow, Germany) [11]. While wearing the video-oculography goggles in a seated position, spontaneous nystagmus was recorded both with and without fixation in the straight-ahead gaze. GEN in the horizontal ($\pm 30°$) and vertical ($\pm 20°$) planes were recorded. VIN was recorded by applying a hand-held vibration stimulator (VVIB 100, Synapsis, Marseille, France) that had a frequency of 100 Hz (± 5 %) and the contact area at 0.9 cm^2. The stimuli were given for 10 s at both mastoids with an interval of 5 s. HSN was induced by passive head-shaking. The examiner pitched the patient's head forward by 30° to bring the HCs into the plane of stimulation. The patients' heads were then grasped firmly with both hands and shaken horizontally in a sinusoidal fashion at a rate of 2.8 Hz with an approximate amplitude of 10° for 15 s [11]. To induce positional nystagmus, the patients lay supine from sitting and turned their heads to either side while supine. Then the patients were moved from a supine to a sitting position and the head was bent forward [12]. The patients also had the right and left Dix-Hallpike maneuvers and the straight head-hanging test [13].

For recording of the HIT, head and eye movements were measured using a magnetic search coil technique in a 70-cm cubic search coil frame (Skalar, Delft, The Netherlands) [14]. A scleral annulus ring (CHRONOS VISION, Berlin, Germany) was placed on the subject's left eye after anesthetizing the conjunctiva with 0.5 % proparacaine hydrochloride (Alcon, Seoul, Korea). A second coil was fixed at the center of the forehead. Calibration of the eye and head positions was conducted by a gimbal that could be rotated independently around three different axes. In addition to in vitro calibration using the gimbal system, we also used horizontal ($\pm 10°$) and vertical ($\pm 10°$) fixation spots for in vivo eye calibration. The patients were instructed to fixate on a red target placed 1.2 m in front of them. The head impulses were a passive, unpredictable, low-amplitude, and high-acceleration head rotation in the planes of the HC, left anterior semicircular canal (AC) and right PC, and right AC and left PC while subjects sat upright [15]. A minimum of five impulses were applied in each direction. The gain of the vestibular-ocular reflex (VOR) was calculated for each trial as the ratio of the peak velocity of the eye over the peak velocity of the head. Eye and head position signals were digitized at 200 Hz with an analog to digital converter (EZAD, Seoul, Korea) and were displayed on a computer screen to allow eye-motion monitoring during the tests. Digitized data were analyzed with MATLAB software (version R2011b; The MathWorks Inc., Natick, MA, USA) [14]. Ten healthy subjects (seven men and three women, age 29–70 years, mean $\pm$ SD = 52 $\pm$ 14 years) with no history of vestibular or neurological disorders served as controls. We defined reduced responses of the eye velocity during HIT when the mean VOR gains were less than a mean-2SD of the control data (<0.70 for the HC, <0.72 for the AC, and <0.76 for the PC) [14].

Bithermal caloric tests

The caloric stimuli comprised alternate periods of irrigation for 25 s with 50 ml of cold (30 °C) and hot (44 °C) water. Nystagmus was recorded binocularly with video-oculography (ICS Medical, Schaumburg, IL, USA). Asymmetry of the vestibular function was calculated using Jongkees' formula, and caloric paresis was defined as a response difference of at least 25 % between the ears [11].

Ocular torsion

Ocular torsion was evaluated by taking photographs of the fundus with a scanning laser ophthalmoscope (CF-60 UVI Fundus Camera, Canon, Tokyo, Japan). The degree of ocular torsion was quantified by measuring the angle formed by a horizontal meridian running through the center

of the disc and a straight line passing through the center of the disc and the fovea. Abnormal ocular torsion was defined as being present when the eye showed any intorsion or extorsion of more than 12.6° [16].

Subjective visual vertical (SVV)

The SVV tilt was measured by seating the subject upright in a dark room and asking him or her to align a rod (8.4 cm long and 0.4 cm wide) vertically using a mouse in the preferred hand. The rod was presented at various angles from the vertical randomly on an LCD monitor positioned 70 cm from the subject's eyes, and the subject viewed the rod through a hole (15 cm in diameter) in a black panel in front of the monitor. The SVV tilt was considered abnormal when it exceeded normal values ($-3.8 \sim 3.1°$ in the left eye, $-3.1 \sim 3.0°$ in the right eye, and $-2.4 \sim 2.6°$ in both eyes; a negative value indicates a counterclockwise rotation) [17].

Cervical and ocular vestibular-evoked myogenic potentials (VEMPs)

Cervical VEMPs were recorded with the subject supine on a bed with the head raised approximately 30° from the horizontal and rotated contralaterally in order to activate the sternocleidomastoid muscles (SCM). The surface electromyographic (EMG) activity was measured from an active electrode placed over the belly of the contracted SCM and from a reference electrode located on the medial clavicle. A ground electrode was attached to the forehead. A short burst of alternating tone (110 dB nHL, 123.5 dB SPL, 500 Hz, rise time = 2 ms, plateau = 3 ms, and fall time = 2 ms) was applied at 2.1 Hz monaurally via a headphone. The analysis time for each stimulus was 50 ms and responses elicited by up to 80 stimuli were averaged for each test. The signal was bandpass filtered at $30 \sim 1,500$ Hz, and the mean values of at least two trials were obtained from each ear for all participants. During each recording the amplified EMG activities of the SCM were also monitored and digitized at 1 kHz using an analog-to-digital converter (NI PCI-4461, National Instruments, Austin, TX, USA). The LabVIEW program (National Instruments) was used to analyze the peak-to-peak amplitudes and calculate the mean tonic activation during the recording. The absolute cervical VEMP amplitude was then normalized against the mean tonic activation of the SCM during the recording. To compare the normalized p13–n23 amplitudes of the cervical VEMP on the affected side with that on the intact side, the interaural difference ratio of the normalized amplitudes (IADamp, %) was also calculated as $[(Ai-Ac)/(Ai + Ac) \times 100]$, where Ai and AC are the normalized p13–n23 amplitude on the

ipsilesional and contralesional sides, respectively. Both the p13 and n23 peak latencies were also calculated.

Ocular VEMPs were recorded with the subject sitting while looking at a target that was displaced more than 2 m from the eyes and at an angle of more than 20° upward. EMG activities were recorded using surface electrodes. An active electrode was placed 1 cm below the center of the lower eyelid and the reference electrode was attached to the cheek 2 cm below the active electrode. The ground electrode was located on the forehead at Fpz. Ocular VEMPs were elicited by vibration stimuli at AFz using a hand-held minishaker (Model 4810, Bruel and Kjaer P/L, Denmark; 500 Hz). Ocular VEMPs in response up to 60 stimuli were averaged for each test, and the average latency of the initial negative peak (n1) and the n1–p1 amplitude were analyzed. The interaural difference ratio of the amplitude of the ocular VEMPs was calculated as IADamp (%) $[(Ai-Ac)/(Ai + Ac) \times 100]$, where Ai and Ac are the n1–p1 amplitude on the ipsilesional and contralesional sides, respectively. Cervical and ocular VEMPs were recorded using a Nicolet Viking Select unit (NicoletBiomedical, Madison, WI, USA).

MRI

MRI was performed with 3.0 T or 1.5 T unit (Intera, Philips Medical Systems, Best, The Netherlands) using our standard imaging protocol (axial turbo spin-echo T2-weighted imaging, axial spin-echo T1-weighted imaging, and axial gradient-echo imaging) [18].

Standard protocol approvals and patient consents

All experiments followed the tenets of the Declaration of Helsinki and this study was approved by Institutional Review Board of Seoul National University Bundang Hospital.

Results

Oculography

Both patients showed torsional-horizontal nystagmus beating to the intact side during straight-ahead gaze. The nystagmus increased with removal of visual fixation, and when looking to the intact side. On the contrary, the nystagmus changed directions when looking to the lesion side, which is consistent with GEN (Fig. 2). The spontaneous nystagmus was augmented by application of vibratory stimuli to the mastoid on either side. However, horizontal head shaking and positional changes did not affect the spontaneous nystagmus significantly. Recording of HIT using a magnetic search coil system documented decreased

Fig. 2 Spontaneous and gaze-evoked nystagmus in patient 1. Patient 1 shows spontaneous nystagmus beating leftward, upward, and counter-clockwise (from the patient's perspective), which increases with removal of visual fixation (**a**). The spontaneous left beating nystagmus increases during leftward gaze and changes into right beating during rightward gaze (**b**). In each recording, the upward deflection indicates rightward, upward and clockwise torsional eye motion. *H* horizontal eye position, *T* torsional eye position, *V* vertical eye position
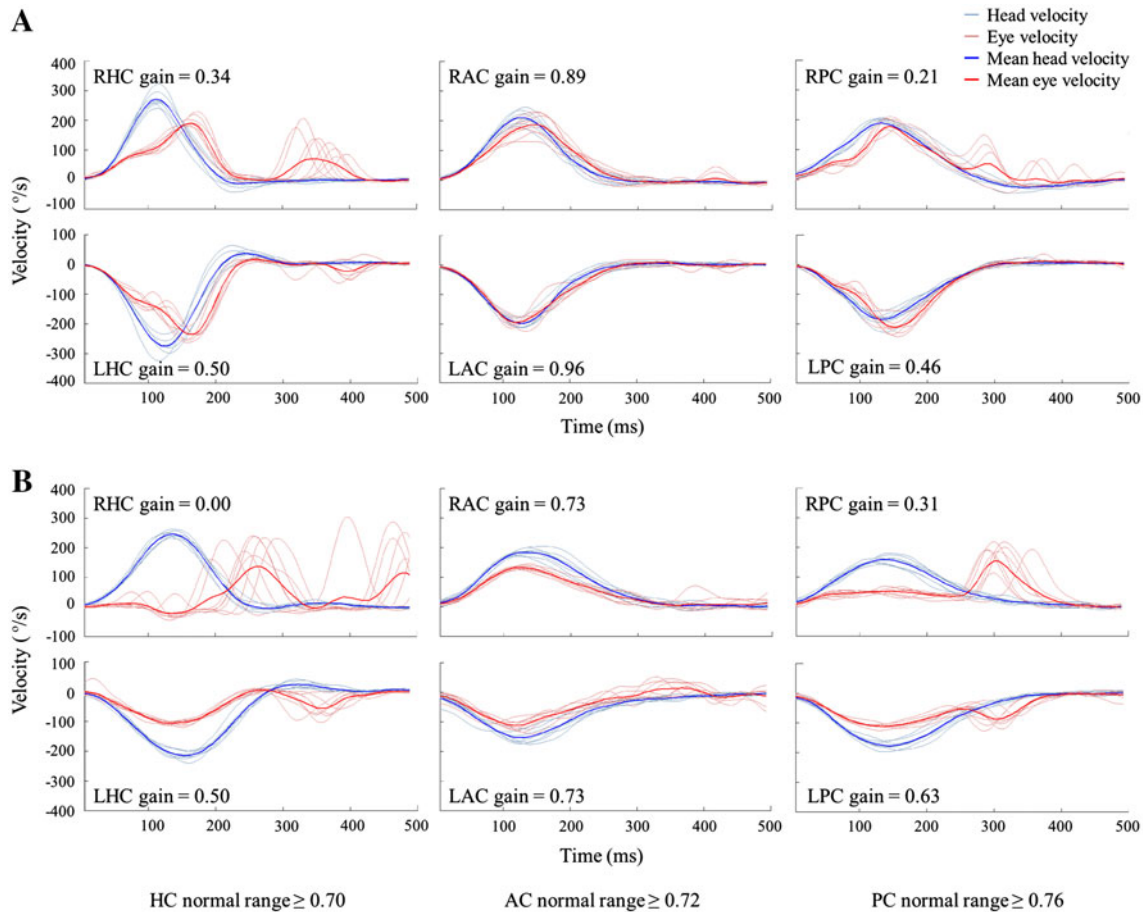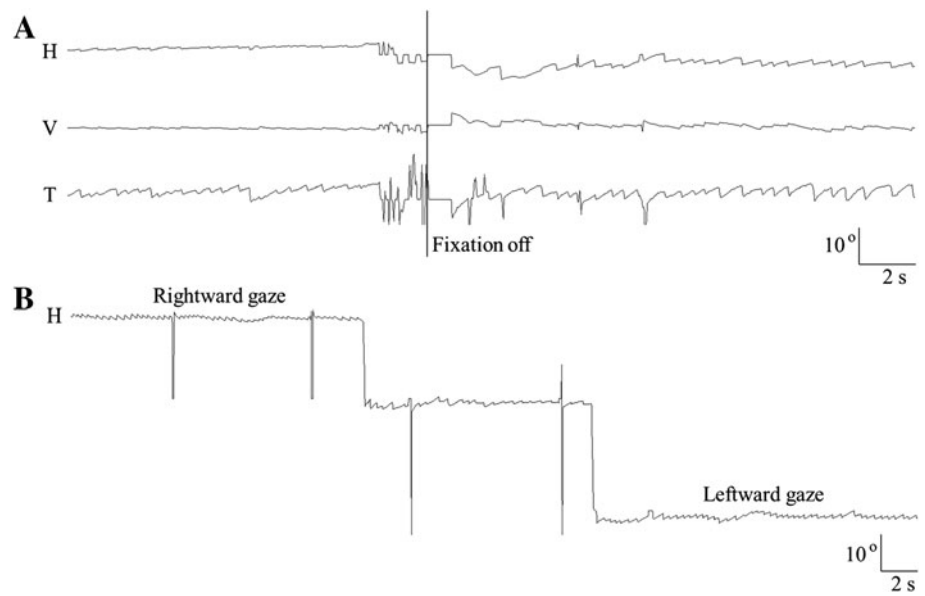
Fig. 3 Head impulse tests (HIT) using the magnetic scleral coil technique. In both patients (**a** patient 1, **b** patient 2), the gains of the vestibulo-ocular reflex (VOR) were reduced during HITs for the horizontal (HCs) and posterior semicircular canals (PCs) on both sides, but more marked for ipsilesional ones. In patient 1, both covert and overt saccades were evident during stimulation of both HCs and right PC, but only covert saccades were seen during stimulation of left PC. In patient 2, only overt saccades were observed during stimulation of HCs and PCs on both sides. In both patients, the reduction of the VOR gains was more marked for ipsilesional HC and PC. *AC* anterior canal, *L* left, *R* right

VOR gains for the HCs and PCs on both sides, but more for the ipsilesional canals (Fig. 3). Bithermal caloric irrigation showed ipsilesional canal paresis (57 % for patient 1 and 81 % for patient 2) in both patients (Fig. 4a).

Other neurotological tests

The patients showed no abnormal ocular torsion on fundus photography or abnormal tilt of the subjective visual vertical. Pure tone and speech audiometry were normal (Fig. 4b). Cervical VEMPs elicited by sound stimuli and ocular VEMPs in response to vibratory stimuli were decreased or absent during stimulation of the ipsilesional ear (Fig. 4c, d). Initial MRIs including diffusion-weighted images were normal or equivocal for an acute infarction, but follow-up imaging a few days later disclosed a circumscribed acute infarction in the area of the vestibular nuclei in both patients.
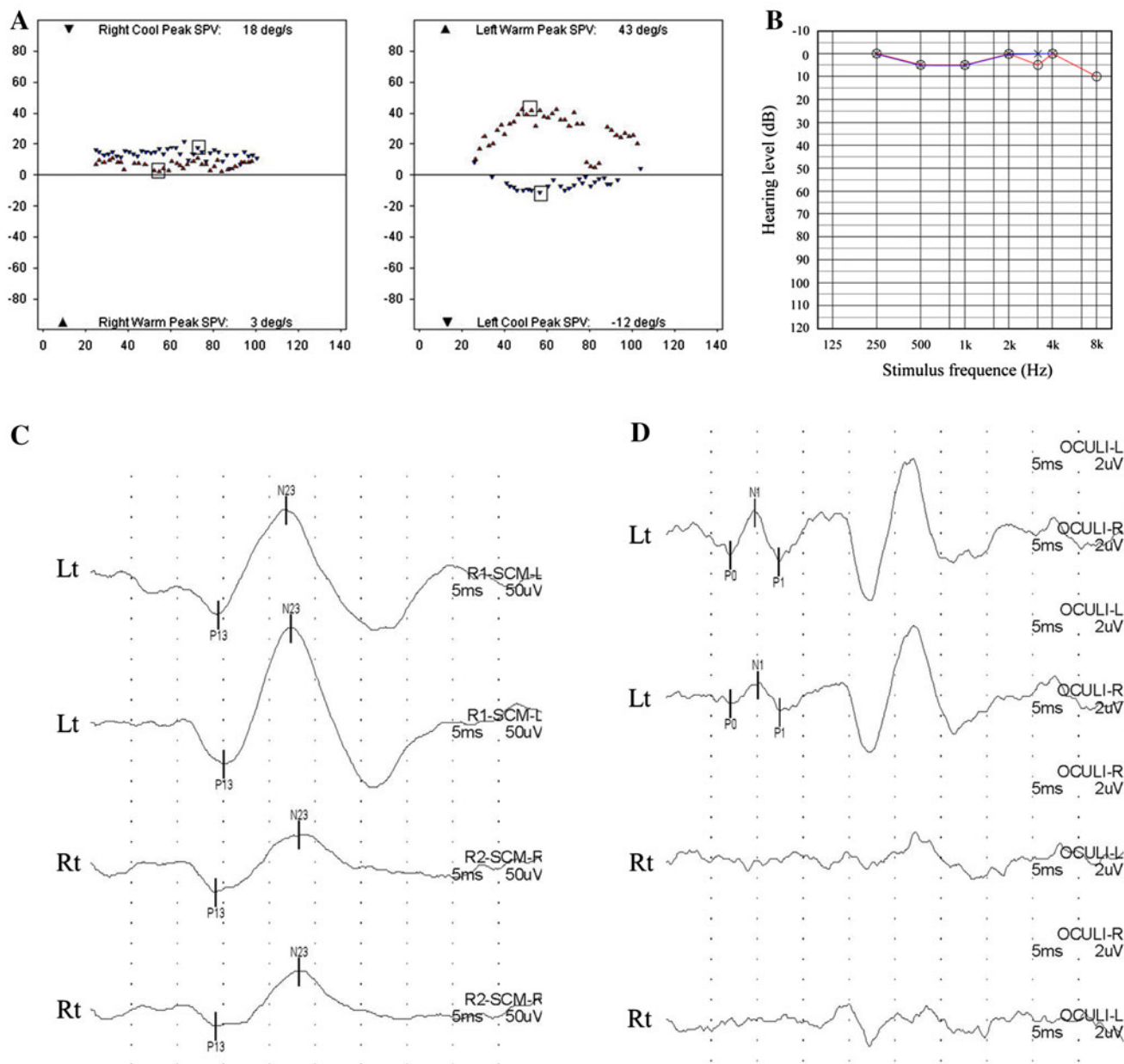

Fig. 4 Other neurotological findings in patient 1. Bithermal caloric irrigation showed right canal paresis of 57 % (a). Pure tone audiometry was normal (b). Cervical vestibular-evoked myogenic potentials (VEMP) in response to air-conducted tone burst sounds showed decreased amplitudes (39.7 %, normal range ≤21.5 %) during right ear stimulation (c), and ocular VEMPs elicited by vibratory stimuli showed no wave formation when recorded below the left eye (right ear stimulation, d)

Review of the literature

Through the literature search, we were able to find seven more patients with isolated vertigo from vestibular nuclear infarctions (Table 1; Fig. 5) [7, 9, 19–21]. Most of them had acute spontaneous vertigo and nystagmus. Although comprehensive evaluation of the vestibular function was not available for most of them, abnormal neurotological findings included contralesional (patients 3, 6, and 8–9) or ipsilesional (patient 7) spontaneous nystagmus, positive bedside HIT during stimulation of the ipsilesional HC (patients 6 and 8), ipsilesional caloric paresis (patients 6), and abnormal VEMPs during ipsilesional ear stimulation (patient 6). Furthermore, four of them showed positive HINTS, including GEN in three (patients 4, 6–7) and skew deviation (patients 3 and 6). Accordingly, isolated

Table 1 Clinical features of the patients with isolated vestibular nuclear infarction

Patient	Age/sex	Lesion side	Territory	Sx	SN	GEN	HIT (HC)	Caloric paresis	Skew
1[a]	34/M	Rt	PICA	V/Vo	C (H, T)	+	B[b]	I (57 %)	–
2[a]	67/M	Rt	AICA	V/Vo	C (H, T)	+	B[b]	I (81 %)	–
3 [7]	64/M	Rt	PICA	V/Vo/Di	C (H, T)	?	?	?	I
4 [7]	56/F	Lt	PICA	V/Vo	–	+	?	?	–
5 [7]	68/M	Lt	PICA	V/U	?	?	?	?	?
6 [9]	53/F	Rt	PICA	V/Vo	C (H, T)	+	I	I (54 %)	C
7 [19]	50/M	Lt	AICA	V/Vo/U	I (H, T)	+	–	NL	?
8 [20]	69/F	Rt	AICA	V/Vo	C(H)	?	I	?	–
9 [21]	78/F	Lt	AICA	V/Vo	C (H, T)	–	–	NL	–

A absent, *AICA* anterior inferior cerebellar artery, *B* both sides, *CP* canal paresis, *C* contralesional, *Di* diplopia, *GEN* gaze-evoked nystagmus, *H* horizontal component, *HC* horizontal semicircular canals, *I* ipsilateral, *Lt* left, *N* nystagmus, *NL* normal, *PICA* posterior inferior cerebellar artery, *Rt* right, *SN* spontaneous nystagmus, *T* torsional component, *U* unsteadiness, *V* vertigo, *Vo* vomiting

[a] Patients reported in this study

[b] The results were obtained with head impulse test (HIT) using a magnetic search coil technique
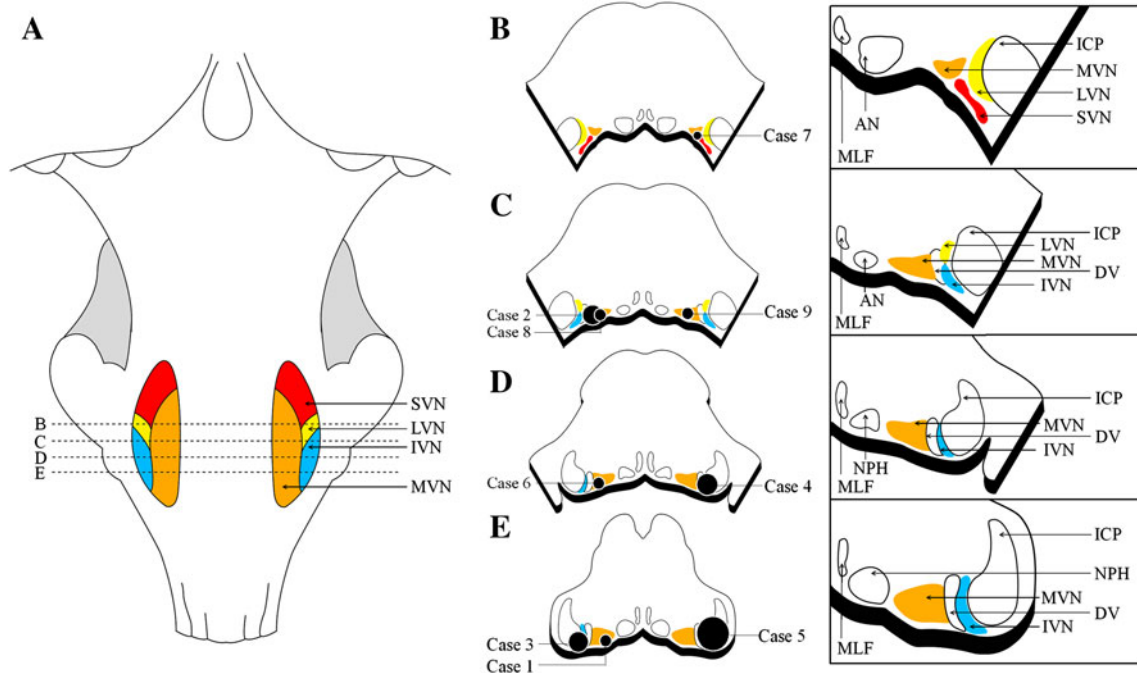

Fig. 5 Lesion location and involved structures in our and previously reported patients. All patients show an infarction restricted to the vestibular nuclei and inferior cerebellar peduncle. The case numbers correspond to those in the table. *AN* abducens nucleus, *DV* descending vestibular root, *ICP* inferior cerebellar peduncle, *IVN* inferior vestibular nucleus, *LVN* lateral vestibular nucleus, *MLF* medial longitudinal fasciculus, *MVN* medial vestibular nucleus, *NPH* nucleus prepositus hypoglossi, *SVN* superior vestibular nucleus (adapted from Duvernoy's Atlas of the Human Brain Stem and Cerebellum, Springer, New York)

vestibular nuclear lesions appear to mostly show the features of both peripheral and central vestibulopathies.

Discussion

Our patients with isolated vestibular nucleus infarction presented with isolated spontaneous vertigo with features of both peripheral and central vestibulopathies. The horizontal-torsional spontaneous nystagmus increasing with removal of visual fixation, positive HIT, unilateral caloric paresis, and decreased or absent VEMP responses were all consistent with unilateral peripheral vestibulopathy [22, 23]. However, our patient also showed direction-changing GEN that is a central oculomotor sign.

Since the neural integration for horizontal eye motion is accomplished by the medial vestibular nucleus/nucleus prepositus hypoglossi complex [24] and the flocculus [25], direction-changing GEN commonly occurs in lesions involving the brainstem or cerebellum. In a previous study, three ocular motor signs, normal horizontal HIT, direction changing nystagmus, skew deviation ("HINTS"), were more sensitive for detecting acute central vestibular syndrome than early MRIs [26]. Indeed, our patients with isolated vestibular nucleus infarction also showed direction changing GEN during lateral eccentric gazes in the presence of normal or equivocal MRIs initially during the acute phase.

One (patient 1) of our patients showed positive bedside HIT for the right HC, and recording of HIT using magnetic scleral coils additionally revealed a decreased VOR gain for the contralesional left HC and both PCs even though decrease of the VOR gains were more marked for the ipsilesional HC and PC. In the other patient (patient 2), recording of HIT using a magnetic scleral coil technique also documented decreased VOR gains for both HCs and both PCs, more marked for the ipsilesional canals, even though bedside HIT was less obvious for the contralesional HC and PC. Bedside HIT is less sensitive than quantitative HIT, especially when the vestibular deficits are partial [17], or the covert saccades mask the corrective catch-up saccades, preventing accurate identification of the vestibular impairments [16]. Furthermore, bedside HITs in the planes of the vertical canals are more difficult for interpretation than the horizontal HIT. Positive HIT has generally been considered a peripheral vestibular sign, even though it may be rarely observed in brainstem or cerebellar lesions [27, 28]. Our patients also showed that lesions involving the vestibular nuclei may cause positive HITs along with other features of peripheral vestibulopathy. Sparing of the VOR pathway from the ACs is consistent with the known anatomy of the vestibular pathways. The signals from the HCs and PCs are mainly mediated by the medial vestibular nucleus while the inputs from the ACs are processed via the superior as well as the medial vestibular nucleus [29]. The decreased VOR gains for the contralesional HC and PC may be an adaptive mechanism, probably through the inhibitory interneurons within the vestibular nuclei [28].

The vestibular nuclei are located in the dorsolateral portion of the rostral medulla and caudal pons. The medial and inferior vestibular nuclei are supplied by the posterior inferior cerebellar artery in the rostral medulla [30]. In contrast, the anterior inferior cerebellar artery irrigates all four vestibular nuclei in the caudal pons [30]. In both patients, the initial MRIs including diffusion-weighted images were normal or only suspicious for an infarction during the acute phase. Occasionally, MRI including diffuse weighted imaging fails to detect a cerebral infarction, especially during the acute stage [31, 32]. Indeed, diffusion-weighted MRIs were falsely negative during the acute phase in 12–18 % of the patients with acute vestibular syndrome due to a stroke [26, 33]. The reason for this initial negativity of MRI remains speculative, but explanations may include functional impairments without a completed stroke, limited resolution of MRI, increasing infarction size during the follow-up, and progression from isolated inner ear infarction into infarction involving the whole territory of the anterior inferior cerebellar artery [2, 32]. Isolated vestibular syndrome has generally been ascribed to inflammatory disorders involving the labyrinth, i.e. vestibular neuritis or labyrinthitis [34]. Recognition of infarction as an occasional cause of isolated vestibular syndrome is important since it requires an evaluation for the cerebral vasculature and stroke risk factors [35]. Furthermore, patients with an infarction should receive proper treatments including acute management and lifetime prophylaxis for stroke [35]. Accordingly, central signs including the HINTS should be carefully sought even in patients with features of peripheral vestibulopathy and negative MRIs [4, 26].

References

1. Huh YE, Koo JW, Lee H, Kim JS (2013) Head-shaking aids in the diagnosis of acute audiovestibular loss due to anterior inferior

cerebellar artery infarction. Audiol Neurootol 18(2):114–124. doi:10.1159/000345643

2. Choi KD, Lee H, Kim JS (2013) Vertigo in brainstem and cerebellar strokes. Curr Opin Neurol 26(1):90–95. doi:10.1097/WCO.0b013e32835c5edd

3. Bergenius J, Magnusson M (1988) The relationship between caloric response, oculomotor dysfunction and size of cerebellopontine angle tumours. Acta Otolaryngol 106(5–6):361–367

4. Kim JS, Lee H (2009) Inner ear dysfunction due to vertebrobasilar ischemic stroke. Semin Neurol 29(5):534–540. doi:10.1055/s-0029-1241037

5. Lee H, Kim JS, Chung EJ, Yi HA, Chung IS, Lee SR, Shin JY (2009) Infarction in the territory of anterior inferior cerebellar artery: spectrum of audiovestibular loss. Stroke 40(12):3745–3751. doi:10.1161/STROKEAHA.109.564682

6. Francis DA, Bronstein AM, Rudge P, du Boulay EP (1992) The site of brainstem lesions causing semicircular canal paresis: an MRI study. J Neurol Neurosurg Psychiatry 55(6):446–449

7. Kim JS (2000) Vertigo and gait ataxia without usual signs of lateral medullary infarction: a clinical variant related to rostraldorsolateral lesions. Cerebrovasc Dis 10(6):471–474. (doi 16110)

8. Moon IS, Kim JS, Choi KD, Kim MJ, Oh SY, Lee H, Lee HS, Park SH (2009) Isolated nodular infarction. Stroke 40(2):487–491. doi:10.1161/STROKEAHA.108.527762

9. Kim HA, Lee H (2010) Isolated vestibular nucleus infarction mimicking acute peripheral vestibulopathy. Stroke 41(7):1558–1560. doi:10.1161/STROKEAHA.110.582783

10. Halmagyi GM, Curthoys IS (1988) A clinical sign of canal paresis. Arch Neurol 45(7):737–739

11. Choi KD, Oh SY, Kim HJ, Koo JW, Cho BM, Kim JS (2007) Recovery of vestibular imbalances after vestibular neuritis. Laryngoscope 117(7):1307–1312. doi:10.1097/MLG.0b013e31805c08ac

12. Han BI, Oh HJ, Kim JS (2006) Nystagmus while recumbent in horizontal canal benign paroxysmal positional vertigo. Neurology 66(5):706–710. doi:10.1212/01.wnl.0000201184.69134.23

13. Lee SH, Kim JS (2010) Benign paroxysmal positional vertigo. J Clin Neurol 6(2):51–63. doi:10.3988/jcn.2010.6.2.51

14. Choi KD, Oh SY, Kim HJ, Kim JS (2007) The vestibulo-ocular reflexes during head impulse in Wernicke's encephalopathy. J Neurol Neurosurg Psychiatry 78(10):1161–1162. doi:10.1136/jnnp.2007.121061

15. Halmagyi GM, Aw ST, Cremer PD, Curthoys IS, Todd MJ (2001) Impulsive testing of individual semicircular canal function. Ann N Y Acad Sci 942:192–200

16. Weber KP, Aw ST, Todd MJ, McGarvie LA, Curthoys IS, Halmagyi GM (2008) Head impulse test in unilateral vestibular loss: vestibulo-ocular reflex and catch-up saccades. Neurology 70(6):454–463. doi:10.1212/01.wnl.0000299117.48935.2e

17. Walker MF, Zee DS (1999) Directional abnormalities of vestibular and optokinetic responses in cerebellar disease. Ann N Y Acad Sci 871:205–220

18. Oh YM, Oh DH, Jeong SH, Koo JW, Kim JS (2008) Sequential bilateral hearing loss in multiple sclerosis. Ann Otol Rhinol Laryngol 117(3):186–191

19. Chang TP, Wu YC (2010) A tiny infarct on the dorsolateral pons mimicking vestibular neuritis. Laryngoscope 120(11):2336–2338. doi:10.1002/lary.20935

20. Yi MJ, Yang HD, Han SJ (2012) Pseudo-vestibular neuronitis caused by medial vestibular nucleus infarction. J Korean Neurol Assoc 30(2):156–157

21. Choi JH, Hwang SJ, Seo JD, Choi KD (2013) Pseudo-vestibular neuritis associated with isolated vestibular nucleus infarction. Clin Neuroophthalmol 3(1):55–57

22. Baloh RW (2003) Clinical practice. Vestibular neuritis. N Engl J Med 348(11):1027–1032. doi:10.1056/NEJMcp021154

23. Oh SY, Kim JS, Yang TH, Shin BS, Jeong SK (2013) Cervical and ocular vestibular-evoked myogenic potentials in vestibular neuritis: comparison between air- and bone-conducted stimulation. J Neurol 260(8):2102–2109. doi:10.1007/s00415-013-6953-8

24. Cannon SC, Robinson DA (1987) Loss of the neural integrator of the oculomotor system from brain stem lesions in monkey. J Neurophysiol 57(5):1383–1409

25. Baier B, Dieterich M (2011) Incidence and anatomy of gaze-evoked nystagmus in patients with cerebellar lesions. Neurology 76(4):361–365. doi:10.1212/WNL.0b013e318208f4c3

26. Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE (2009) HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke 40(11):3504–3510. doi:10.1161/STROKEAHA.109.551234

27. Newman-Toker DE, Kattah JC, Alvernia JE, Wang DZ (2008) Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. Neurology 70(24 Pt 2):2378–2385. doi:10.1212/01.wnl.0000314685.01433.0d

28. Park HK, Kim JS, Strupp M, Zee DS (2013) Isolated floccular infarction: impaired vestibular responses to horizontal head impulse. J Neurol 260(6):1576–1582. doi:10.1007/s00415-013-6837-y

29. Leigh RJ, Zee DS (2006) The neurology of eye movements. Oxford University Press, USA

30. Tatu L, Moulin T, Bogousslavsky J, Duvernoy H (1996) Arterial territories of human brain: brainstem and cerebellum. Neurology 47(5):1125–1135

31. Kim JS, Cho KH, Lee H (2009) Isolated labyrinthine infarction as a harbinger of anterior inferior cerebellar artery territory infarction with normal diffusion-weighted brain MRI. J Neurol Sci 278(1–2):82–84. doi:10.1016/j.jns.2008.12.002

32. Lee H, Kim HJ, Koo JW, Kim JS (2009) Progression of acute cochleovestibulopathy into anterior inferior cerebellar artery infarction. J Neurol Sci 278(1–2):119–122. doi:10.1016/j.jns.2008.11.019

33. Kim HW, Choi JH, Choi KD, Cho HJ, Sung SM, Kim HJ, Kim JS, Jung DS (2013) Isolated vestibular syndrome in posterior circulation infarction: Frequency and involved structures. Neurol Clin Pract (in press)

34. Jeong SH, Kim HJ, Kim JS (2013) Vestibular neuritis. Semin Neurol 33(3):185–194. doi:10.1055/s-0033-1354598

35. Kim JS, Lee H (2013) Vertigo due to posterior circulation stroke. Semin Neurol 33(3):179–184. doi:10.1055/s-0033-1354600