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Isolated Nodular Infarction

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Background and Purpose—Isolated nodular infarction has rarely been described in human. The purpose of this study is to report clinical and laboratory findings of isolated nodular infarction.

Methods—Eight patients with isolated nodular infarction were recruited from 6 hospitals in Korea. All patients underwent a complete and standardized neurotological evaluation including ocular torsion, bithermal caloric tests, and rotatory chair test in addition to MRI and MR angiography.

Results—All patients presented with isolated vertigo and moderate to severe imbalance. The most common manifestation was unilateral nystagmus and falling in the opposite direction, which mimicked peripheral vestibulopathy. Six patients had unilateral lesion, and 2 showed bilateral lesions. The direction of the spontaneous nystagmus was all ipsilesional in the unilateral lesion. However, head impulse and bithermal caloric tests were normal. Other findings include periodic alternating nystagmus, perverted head shaking nystagmus, paroxysmal positional nystagmus, and impaired tilt suppression of the postrotatory nystagmus. Hypoplasia of the ipsilesional vertebral artery was the only abnormal finding on MR angiography in 3 patients. The prognosis was excellent.

Conclusions—Isolated nodular infarction mostly presents with isolated vertigo mimicking acute peripheral vestibulopathy. However, severe imbalance and a negative head impulse test are important clinical discriminants between nodular infarcts and peripheral vestibular dysfunction. The findings of isolated nodular infarctions are consistent with impaired gravito-inertial processing of the vestibular signals and disrupted nodular inhibition on the vestibular secondary neurons and the velocity storage mechanism. (*Stroke*. 2009;40:487-491.)

Key Words: nodular infarction ■ cerebellum ■ vertigo ■ imbalance ■ head impulse test

The nodulus lies in the midline cerebellum between the inferior medullary velum and uvula, and constitutes the vestibulocerebellum along with the flocculus, paraflocculus, and ventral uvula.¹ The nodulus receives afferent inputs from the vestibular system and is involved in controlling eye movements and in postural adjustments to gravity.² Lesions involving the nodulus increase the duration of vestibular responses and produce periodic alternating nystagmus (PAN),³⁻⁵ positional downbeat⁶ and perverted head-shaking nystagmus (HSN),^{4,7,8} inability to suppress postrotatory nystagmus by head-tilt,^{3,4,9} and loss of vestibular habituation.¹⁰ In addition, “pseudolabyrinthine” syndrome^{11,12} and contraversive ocular tilt reaction¹³ have been reported in the caudal cerebellar lesions involving the nodulus.

The nodulus is irrigated by the medial branch of the posterior inferior cerebellar artery (mPICA).¹⁴ Nodular infarction is usually associated with ischemic lesions in other areas supplied by mPICA, and isolated nodular infarction is extremely rare. To the best of our knowledge, only 2 patients

have been reported by the authors until now.^{4,12} Herein, we report clinical and laboratory findings of 8 patients with isolated nodular infarction.

Materials and Methods

Subjects

Eight patients with isolated nodular infarction had been recruited from 2002 to 2007 at 6 hospitals in Korea. All the patients received a complete and standardized neurological evaluation by the authors (Table). Two patients (patients 6 and 7) included in this study were previously reported as a case report by the authors (J.S.K., S.Y.O., H.L.).^{4,12}

Neurotological Evaluation

Spontaneous, head shaking, and positional nystagmus were observed without fixation using Frenzel goggles. HSN were assessed using a passive head-shaking maneuver.¹⁵ Imbalance was graded from 0 to III as follows: Grade 0 (normal), able to stand on tandem Romberg with the eyes open for 3 seconds; grade I (mild), unable to stand on tandem Romberg with the eyes open at least for 3 seconds; grade II (moderate), unable to stand on Romberg with the eyes open at least

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Table. Clinical and Laboratory Findings of the Patients

Patient/Sex/Age	1/M/52	2/M/67	3/F/66	4/M/51	5/F/64	6/M/69	7/M/37	8/F/75
Risk factors	Dyslipidemia smoking	Smoking	Hypertension	Hypertension	Hypertension	...
Lesion side	R	Both	R	L	L	Both	L	R
Spontaneous nystagmus	R	R/D	R	L	...	PAN	L/CCW	R
Head-shaking nystagmus	R	R/D	R/D	L	L	D	NA	...
Positioning nystagmus	DCPN (apogeotropic)
Head tilt	L	L
Ocular torsion	L
Head impulse test	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Falling (grade*)	L (III)	L (II)	L (III)	R (II)	R (II)	NA (III)	R (III)	Both (II)
Rotatory chair test	Normal	No tilt suppression	NA	NA	NA	No tilt suppression	Normal	NA
MR angiography	HVA (R)	Normal	Normal	HVA (L)	Normal	Normal	Normal	HVA (R)

CCW indicates counterclockwise; D, downbeat; DCPN, direction-changing positional nystagmus; HVA, hypoplastic vertebral artery; L, left; NA, not applicable; PAN, periodic alternating nystagmus; R, right.

*For grading of the imbalance, refer to the text.

for 3 seconds; grade III (severe), unable to stand or sit without support.

Laboratory examination included measurements of ocular torsion, bithermal caloric tests, and rotatory chair test. Ocular torsion was determined 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 using fundus photographs.¹⁶

The caloric stimuli comprised alternate irrigation for 25 seconds with 50 mL of cold and hot water (30°C and 44°C). Nystagmus was recorded binocularly by video-oculography. Asymmetry of vestibular function was calculated using Jongkees' formula, and caloric paresis was defined by the response difference of 25% or more between the ears.¹⁶

Four patients also underwent measurements of horizontal vestibulo-ocular reflex (VOR) using CHARTR rotary vestibular test system (ICS Medical). For evaluation of the VOR, the participants underwent sinusoidal oscillation about a vertical axis at harmonic frequencies ranging from 0.01 to 0.32 Hz with the peak angular velocity of 50°/s at each frequency. The head was pitched forward 30° during the VOR testing. The peak velocity of the eye movement elicited was compared with the peak velocity of the stimulus (assumed to be the same as head velocity). For velocity-step test, the participants were subjected to a series of velocity steps, first to the right and after then to the left. For a rightward velocity step, the subjects underwent an angular acceleration of 100°/s² for 1 second. At the end of the acceleration, the subjects continued to rotate at a constant velocity of 100°/s to the right. The velocity was maintained for another 60 seconds, after which the subjects were decelerated to 0°/s within 1-second time period. The above steps were repeated for a leftward rotation. In response to the stimuli, time constants (TC) of the pre- and postrotatory nystagmus were calculated. The eye motion was detected with electrodes placed using the standard methods and was digitized at 160 Hz with a frequency response of 0 to 30 Hz and a notch filter at 60(50) Hz. To evaluate tilt suppression of the VOR, the participants were asked to pitch their head forward further at the end of the step rotation stimuli in each direction. TC was calculated again as described above. How effectively did the forward head tilt suppress the postrotatory nystagmus was expressed by tilt suppression index (TSI) that was calculated by the equation, TSI (%)=[(TC without head tilt-TC with head tilt)/TC without head tilt×100].

Normative data were obtained from 50 healthy volunteers (24 men and 26 women) with ages ranging from 24 to 70 years (mean±SD=43.0±15.4). All experiments followed the tenets of the Declaration of Helsinki, and informed consents were obtained after

the nature and possible consequences of this study had been explained to the participants.

Brain Imaging

All patients underwent MRI and MR angiography. Involved side was assessed on the diffusion- and T2-weighted images by a neurologist and neuroradiologist without knowing the clinical and laboratory features of the patients. We defined hypoplastic vertebral artery as a lumen diameter of less than 2 mm at the midportion of V2 (the portion within the vertebral columns) on MR angiography.¹⁷

Representative Case Descriptions

Patient 1

A 52-year-old man with a history of dyslipidemia and smoking (50-pack-year) developed sudden vertigo and imbalance 4 days before the admission. The vertigo was initially recurrent, lasting several minutes, and became persistent 3 days later. He also suffered from nausea and vomiting without auditory symptoms. On admission, he showed leftward head tilt and spontaneous right beating nystagmus without fixation, which was augmented by horizontal head shaking. Positional maneuver did not change the spontaneous nystagmus. There was no gaze-evoked nystagmus or PAN. Extraocular movements were full with normal pupils and fundi. Horizontal and vertical saccades were normal. Smooth pursuit was intermixed with the nystagmus. Head impulse tests¹⁸ were normal. He showed no dysarthria or appendicular ataxia. However, he fell to the left on standing if unsupported. Other findings of the neurological examinations were normal. Fundus photography showed abnormal intorsion of the right eye (-7.2°, normal range: 0 to 12.6°). The responses to bithermal caloric stimulation were symmetrical. He showed normal gains and phases of the VOR during sinusoidal harmonic acceleration and normal TCs of the pre- and postrotatory nystagmus during step velocity rotations. Tilt suppression of the postrotatory nystagmus was normal. Brain MRI revealed an acute nodular infarction mainly involving the right side (Figure), and MR angiography disclosed hypoplasia of the right vertebral artery. No abnormal finding was detected in other parts of the brain including the brain stem and cerebellum. He was treated with antiplatelet and lipid-lowering drugs and the symptoms and signs resolved over the following week.

Patient 5

A 64-year-old woman experienced sudden paroxysmal vertigo on turning her head to the right while supine. The vertigo resolved within 1 minute even though the provoking position was maintained.

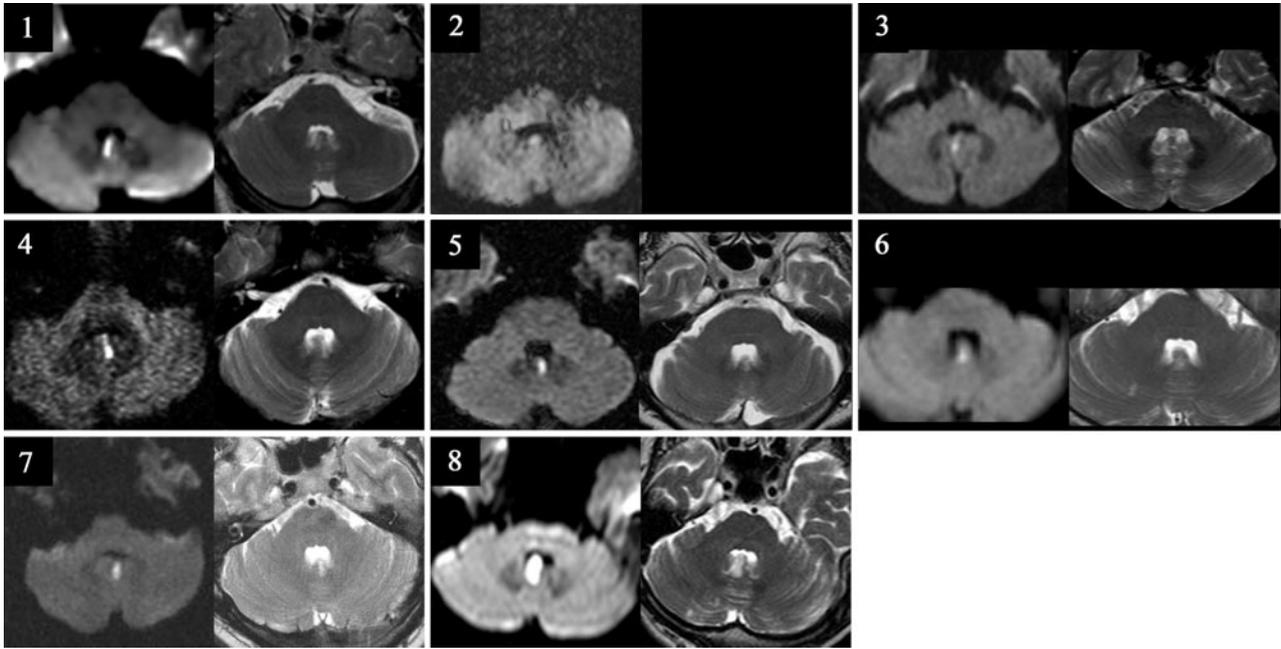


Figure. Diffusion- and T2-weighted MR images of the patients. Six patients have lateralized lesions (3 left- and 3 right-sided lesions), whereas 2 (patients 2 and 6) show lesions involving both sides of the nodulus.

She had undergone a surgery for breast cancer 2 years before. She fell to the right on standing with the feet together. She had no spontaneous or gaze-evoked nystagmus, but horizontal head shaking induced left beating nystagmus. Head turning to either side while lying down induced apogeotropic nystagmus, which lasted approximately 1 minute without a discernible latency. The nystagmus was more intense when turning the head rightward. Saccades, smooth pursuit, and horizontal head impulse were normal. Other findings of the neurological examination were normal. Bithermal caloric tests were also normal. MRI disclosed an acute infarction mainly involving the left side of the nodulus (Figure). MR angiography was normal.

Results

Patients included 5 men and 3 women with the age ranging from 37 to 75 years (mean=60.1). MRI documented lateralized lesions in 6 patients (3 left- and 3 right-sided lesions) whereas 2 (patients 2 and 6) had lesions involving both sides of the nodulus (Figure). MR angiography showed ipsilesional vertebral artery hypoplasia in 3 patients (patients 1, 4, and 8). Otherwise, findings of MR angiography were normal. Risk factors for stroke include hypertension ($n=3$), smoking ($n=2$), and dyslipidemia ($n=1$, Table). Three patients had no discernible risk factor.

All patients presented with sudden vertigo and imbalance, mimicking peripheral vestibular disorders (Table). However, the imbalance was moderate to severe that the patients were unable to maintain Romberg posture (with the eyes open) for more than 3 seconds and 4 of them even could not stand without support. Five of the 6 patients with unilateral lesion fell to the contralesional side, and the remaining 1 patient (patient 8) fell in any direction (Table).

Six patients showed spontaneous nystagmus which was all ipsilesional in 5 of them with unilateral lesion, and was right beating in the remaining patient (patient 2) with bilateral lesion. In 2 patients (patients 2 and 7), downbeat or torsional component was associated with the horizontal nystagmus.

One patient (patient 6) showed PAN. Horizontal head oscillation augmented the spontaneous nystagmus in 4 patients (patients 1 to 4) and induced horizontal nystagmus in 1 patient (patient 5). In 3 patients (patients 2, 3, and 6), horizontal head shaking induced downbeat nystagmus (perverted HSN). Nystagmus did not change with horizontal head shaking in 1 patient (patient 8). In 1 patient (patient 5), apogeotropic type of positional nystagmus was observed during head-turning in the supine position. The nystagmus was more prominent during contralesional head turning. Contralesional head tilt and ocular torsion were observed in 1 patient (patient 1) with unilateral lesion, and 1 (patient 2) with bilateral lesion showed leftward head tilt. Head impulse and bithermal caloric tests were normal in all the patients.

Four patients (patients 1, 2, 6, and 7) underwent rotatory chair tests, and all showed normal gains and phases of the horizontal VOR during sinusoidal harmonic acceleration and normal TCs of the pre- and postrotatory nystagmus during step velocity rotations. However, forward head tilt did not suppress the postrotatory nystagmus in 2 (patients 2 and 6) with bilateral lesions.

The vertigo, imbalance, and spontaneous nystagmus resolved within a few days from the symptom onset and the patients usually became symptom free over the following week.

Discussion

The cerebellum may be divided anatomically and functionally into 3 major regions.¹ (1) The cerebellar hemispheres and a small part of the posterior vermis form the pontocerebellum, which receives input from the cerebral cortex via the pontine nuclei. (2) The anterior lobe and most of the posterior vermis make up the spinocerebellum, which receives afferents from the spinal cord. (3) The nodulus and flocculus are connected with the vestibular nuclei and constitute the vestibulocerebel-

lum. Most cases of cerebellar pathology affect more than one region and different pathways, and focal syndromes after restricted cerebellar lesions are rare. All our patients presented with “vertigo and imbalance without other neurological deficits.”

Nodular lesions may show diverse manifestations which may be related to reciprocal connections with numerous structures in the peripheral and central vestibular networks.^{1,2,19,20} The nodulus and adjacent ventral uvula receive afferents mostly from the vestibular nuclei, nucleus prepositus hypoglossi, inferior olivary nucleus, and vestibular nerve, whereas their main efferents project to the vestibular nucleus.¹ The nodulus and uvula can be divided into 4 para-sagittal zones comprising strips of Purkinje cells innervated by subnuclei of the contralateral inferior olive.²⁰ TCs of the horizontal and vertical/torsional VOR are separately controlled by the lateral and central portions of the nodulus and uvula.²¹ The nodulus, as a coordination center for eye, head, and body motion in space,¹ participates in inertial processing of vestibular signals,^{22,23} modulating the velocity-storage mechanism,^{3,24} and tuning of the spatial orientation of the angular VOR.^{21,25}

All our patients with isolated nodular infarction presented with vertigo and imbalance without other neurological deficits, which mimicked vertigo of peripheral origin. Furthermore, 5 of the 6 patients with unilateral nodular lesion showed ipsilesional spontaneous nystagmus, and 4 of them fell to the contralesional side, which resembles damage to the contralesional labyrinth. Previously, one of the authors described symptoms and signs of vestibular neuritis, eg, body tilting in the opposite direction of spontaneous nystagmus without other neurological deficits, in a patient (patient 7) with isolated nodular infarction.¹² In this study, we confirm that this kind of manifestation is rather common in isolated nodular infarction. Disruption of the nodular inhibition on ipsilateral vestibular nucleus has been invoked to explain the ipsilesional spontaneous nystagmus.^{11,12} The disinhibition would augment tonic resting activity of the vestibular neurons in the same side, and induce ipsilesional spontaneous nystagmus.

However, head impulse and caloric tests were all normal in our patients with isolated nodular infarction, even in the patients with spontaneous horizontal nystagmus. These are in contradiction to the findings of peripheral vestibulopathy. The head impulse test can reliably detect unilateral peripheral vestibulopathy,^{26,27} and highly sensitive during the acute stage.¹⁶ Accordingly, negative head impulse test during the acute phase of vestibulopathy accompanying spontaneous nystagmus strongly suggests central lesions.^{28,29}

Most of our patients fell on standing. Because most patients with peripheral vestibulopathy are able to maintain their balance using intact vision and proprioception, the moderate to severe imbalance in our patients also indicates a central pathology.^{30,31} Central vestibular lesions not only cause abnormal eye motion attributable to impaired VOR but also generate direction-specific disorientation and postural imbalance attributable to deranged vestibulospinal reflexes.³² Nodular Purkinje cells receive heavy otolithic inputs and participate in the postural control of the head and body in the gravity.^{19,33}

Five patients showed augmentation of spontaneous nystagmus (patients 1 to 4) or induction of nystagmus (patient 5) by horizontal head shaking. In peripheral vestibulopathy, HSN may occur from the velocity storage mechanism and Ewald's second law which states that excitatory vestibular inputs are more effective than inhibitory ones.^{34,35} The nodulus and ventral uvula project to the vestibular nuclei and inhibit the velocity-storage mechanism of the VOR.^{3,24} Because the effect of the nodulus on velocity-storage is unilateral,²⁴ unilateral or asymmetrical nodular lesion would generate asymmetry in the velocity storage, which would increase with horizontal head-shaking and induce the nystagmus or augment the spontaneous nystagmus. In 3 patients (patients 2, 3, and 6), horizontal head-shaking induced downbeat nystagmus (perverted HSN). Perverted HSN may be explained by inappropriate storage of asymmetrical vertical vestibular signal by activating the horizontal VOR pathway (cross-coupling).⁷ Nodular lesions may generate perverted HSN because the nodulus controls spatial orientation of the angular VOR and participates in cross-coupling of the velocity storage during head tilt.^{21,25}

One patient presented with positional vertigo, and examination revealed apogeotropic positional nystagmus during head turning in supine, which can be observed in certain cerebellar or brain stem lesions.²⁵ Because change in graviceptive (otolithic) input induced by positional change is the precipitating factor for central paroxysmal positioning nystagmus, nodular lesion may generate positional nystagmus attributable to impaired transduction of the gravity-related otolithic signal.

Head tilts and ocular torsion in the same direction were observed in 2 patients, contraversive in one (patient 1) with unilateral lesion and leftward in the other (patient 2) with bilateral lesion. Ocular tilt reaction (OTR) is a pathological synkinetic triad of head tilt, ocular torsion, and skew deviation.¹ OTR may be explained by asymmetrical otolithic neuronal input owing to interruption of peripheral or central otolithic pathway. OTR seems to receive cerebellar modulation because unilateral cerebellar lesions induce partial OTR,¹³ or skew deviation.^{36,37} Furthermore, isolated nodular or combined uvulonodular lesions may impair the otolith-ocular reflexes and otolithic modulation of the semicircular canal VOR.^{23,33} The OTR in our patients may be ascribed to asymmetry in the tonic resting activity of secondary otolithic neurons attributable to asymmetrical nodulo-vestibular inhibition. The leftward OTR, leftward falling, and right beating nystagmus observed in patient 2 with bilateral lesion suggest asymmetrical damage to the nodulus, more to the right than to the left side.

Forward head pitching at the onset of postrotatory nystagmus diminishes TC of the horizontal VOR.³ This tilt suppressive response of horizontal VOR was lost in 2 patients (patients 2 and 6) with bilateral lesions. The otolithic inputs are believed to rapidly discharge the activities from the velocity storage during tilt suppression.³ In animals and humans, tilt suppression of the VOR disappears after selective lesions of the nodulus and uvula.^{4,9,21,38}

Three patients exhibited ipsilesional vertebral artery hypoplasia, whereas 5 patients showed normal findings on MR

angiography. Recently, vertebral artery hypoplasia is considered a risk factor of posterior circulation ischemia.^{17,39} Even though the mechanism of infarction remains undetermined in most patients, large artery atherosclerosis related to hypoplastic ipsilesional vertebral artery may have caused isolated nodular infarction.^{17,40}

Summary

Isolated nodular infarction mostly presents with isolated vertigo mimicking acute peripheral vestibulopathy. The findings include moderate to severe imbalance, spontaneous nystagmus, periodic alternating nystagmus, perverted head shaking nystagmus, paroxysmal positional nystagmus, and impaired tilt suppression of the postrotatory nystagmus. Those findings are consistent with impaired gravito-inertial processing of the vestibular signals and disrupted nodular inhibition on the vestibular secondary neurons and the velocity storage mechanism. Severe imbalance and a negative head impulse test are important clinical discriminants between nodular infarcts and peripheral vestibular dysfunction.

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Disclosures

None.

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