Which Vestibulopathy is Vertebral Artery Hypoplasia Related with in Vestibular Migraine?

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Abstract-

Purpose: A possible relationship between vertebral artery hypoplasia (VAH) and vestibular migraine (VM) has been suggested at some medical conferences, few studies of this condition have elucidated which vestibulopathy is VAH associated with during the vestibular episodes of VM.

Methods: We performed a retrospective case-series control study to elucidate the above issue. From 2008 January to May 2010, 18 VM patients received magnetic resonance imaging. Of them, 44.4% (n=8) were the VAH subgroup and 55.6% (n=10) were the non-VAH subgroup. We reviewed the ictal electronystagmogram battery of the two subgroups. A Fisher's exact test was used with alpha of 0.01.

Results: VAH was not more significantly prevalent in the VM patients than the non-VM ones. In the VM group, there was a significant difference in the 4 sub-divisions of vestibulopathy between the VAH and non-VAH subgroups (p=0.0096).

Conclusion: In this small neurotological study, VAH was closely related with central vestibulopathy rather than peripheral or mixed vestibulopathy so the topographic factor of VAH little influenced the ipsilateral peripheral vestibular labyrinth in the vestibular episodes of VM.

Key Words: vertebral artery hypoplasia, vestibular migraine, migraine, vestibulopathy, vertigo

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INTRODUCTION

Background

Vertebral artery hypoplasia (VAH) is defined when the vertebral artery (VA) is less than 2.0 mm in diameter on three-dimensional time-of-flight magnetic resonance angiography (MRA), and is different from a steno-occlusive VA, in that the former shows decreased blood flow in the whole vessel and the latter shows decreased blood flow over a short vessel segment. It has

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been reported in 2-6% of autopsy findings and digital subtraction cerebral angiograms, but there is a higher incidence of VAH in Asian groups (15.2-20.2%), studied by computed tomographic angiogram and MRA[1–3]. Although the clinical relevance and hemodynamic impact are as yet inconclusive [4–6], VAH has been reported as possibly contributing to acute ischemic stroke and migraine with aura[4,7]; in addition, its topographic domain was considered a regional hemodynamic negative contributory factor to the ipsilateral vestibular labyrinth by Fahraeus-Lindqvist effect, contributing to the development of vestibular neuropathy [8].

Objective

Although a possible relationship between VAH and vestibular migraine (VM) has been suggested at some medical reports, few of them have elucidated which vestibulopathy is VAH associated with during the vestibular episodes of VM. We hypothesize VAH would be more associated with peripheral or mixed vestibulopathy than simply central vestibulopathy during the vestibular episodes of VM by the mechanism of topographic hemodynamic negative contributory factor to the ipsilateral vestibular labyrinth [8].

MATERIALS AND METHODS

Study design

In order to avoid the issues of ethics, this research was done only by chart reviewing from a specific neurotological clinic of a regional hospital in north Taiwan, and a retrospective case-series control study was managed to elucidate the above issue.

Study setting

From January 1st, 2008 to May 31st, 2010, the first author led a specific neurotological clinic at the department of otorhinolaryngology of a regional hospital in north Taiwan. At the clinic, a junior residency physician, who took the illness history and performed physical examination, routinely interviews the vertigo/dizziness patients. Then the first author interviewed the patient again and checked the medical record, and anyhow, the two physicians identified all subjective symptoms and objective signs. If there were any doubtful neurologic abnormal symptom or sign, a neurologist would be consulted. Afterwards, the patients were investigated with electronystagmogram (ENG) battery. The final diagnosis included benign paroxysmal positional vertigo, Ménière's disease, vestibular neuritis, VM, traumatic otolithic syndrome, perilymph fistula and others. With several reasons including limitation of refund from the Taiwan National Health Insurance, not all patients would receive neuroimagings.

Study participants and size

Only 96 patients who had the chief complaints of vertigo/dizziness and received magnetic resonance imaging (MRI) (1.5 Tesla system, Picker Edge Eclipse, Picker 98 International, USA) were collected. They were all clear, intelligent and cooperative, and were confirmed not to have any diplopia, convergence disorder, visual defect, visual apraxia, dysmetria, frontal release signs, dementia or parkinsonism by history takings, chart reviews, neurologic consultations and physical examinations. There was no acute infarction, meningitis, intracranial hypo- or hypertension, intracranial hemorrhage, cerebral neoplasm or multiple sclerosis demonstrated in these 96 selected patients. In addition, they were VAH was defined as a VA diameter less than 2.0 mm in the cervical MRA studies (TR/TE/excitation: 29/6/1). Of them, 18.8% (n=18)(9 women and 9 men) were diagnosed with VM were enrolled as the VM group. Their age was 45.3±12.8 (average±SD) years.

Diagnostic criteria of VM

By reviewing the their charts, VM was confirmed with the following four criteria [9]: (i) at least 5 episodes of severe or moderate vestibular symptoms, including spontaneous internal or external vertigo, positional vertigo, visually-induced vertigo, head motion-induced vertigo, head motion-induced dizziness with nausea, lasting from 5 minutes to 72 hours, (ii) current or previous history of migraine with or without aura according to criteria of the International Classification of Headache Disorders, (ICHD) 3rd edition (β version) [10] including: (a) headache (with at least two of the following characteristics: one-sided location, pulsating quality, moderate or severe pain in intensity, and aggravation by routine physical activity), (b) photophobia and phonophobia, and (c) visual aura before

the headache, (iii) more than 50% of migraine episodes featuring the vestibular attacks, and (iv) unable to be explained by any other diagnosis of vestibular disorder or headache according the ICHD [11].

Data measurements

Ictal ENG battery (NY-41, Rion, Japan)

During onsets of migraine of all these 96 patients, ENG battery, including (i) horizontal visual-ocular study, consisting of horizontal pursuit, horizontal saccade, and optokinetic nystagmus tests, and (ii) vestibulo-ocular reflex study, consisting of bithermal caloric tests, and (iii) visual-vestibular reflex study, consisting of post-caloric visual fixation suppression tests, were done.

The negative sensor electrode was placed on the right inferior mid-eyelid, and the positive sensor electrode on the right mid-temple region juxtaposed to the right lateral canthus. During the horizontal eyeball tracing movement study, the examinee was asked to look at the screen about 3.0 m in front, and watch the red point, which was projected by an automatic laser pointer.

From the recording 0 to 10 seconds, the red point smoothly moved to and fro horizontally at amplitude of 20º every 5 seconds so led the examinee's eyeball to smoothly rotate rightward and then leftward, and so on, producing a horizontal pursuit report. From the recording 10 to 20 seconds, the red point abruptly jumped right and left horizontally at amplitude of 20º every 2 seconds so led the examinee's eyeball to abruptly rotate rightward and then leftward, and so on, producing a horizontal saccade report.

From the recording 20 to 100 seconds, the red point abruptly jumped rightward and then smoothly moved leftward at amplitude of 20º every 1 second so produced a rightward optokinetic nystagmus report, which is composed of rightward saccade, leftward pursuit, rightward saccade, leftward pursuit…and so on. From the 100 to 180 seconds, producing a leftward optokinetic nystagmus report, which is composed of leftward saccade, rightward pursuit, leftward saccade, rightward pursuit… and so on.

During vestibulo-ocular reflex study, the examinee lied in supine position with neck flexing in 30º and closed the eyes. The right external auditory canal was irrigated with 100 mL 44°C water, and the spontaneous warm caloric nystagmus was recorded for 20 seconds by ENG. Then, the patient was asked to open eyes and look at in front, and the remitting post-caloric nystagmus was recorded for the following 20 seconds. The same method was performed for left external auditory canal. In addition, the both ears also received cool caloric tests with 33°C water. The unilateral-weakness percentage was defined as the percentage of difference of the maximum slow-phase velocity between both ears divided by the sum of the maximum slow-phase velocity in both ears according to Jongkee's formula [12].

Unilateral-weakness percentage: ([RW + RC]-[LW + LC])/[RW + RC + LW + LC]) × 100%

RW and LW: the maximum slow-phase velocity of warm caloric nystagmus in right (RW) or left (LW) ear.

RC and LC: the maximum slow-phase velocity of cool caloric nystagmus in right (RC) or left (LC) ear.

Table 1 shows the order of the ENG tests and the definitions for pathological test results.

Table 1. Electronystagmogram battery and the definitions for pathological test results

Electronystagmogram	Definition for pathological test result
1. Horizontal pursuit test	The wavy route of eyeball tracing movement is irregular or ragged in the eyeball tracing movement phase so sharp spikes of corrective (catchup or backup) saccade appears in the wavy route of eyeball velocity in the eyeball velocity phase
2. Horizontal saccade test	At least 2 saccades had undershoots or overshoots in the eyeball tracing movement phase so small extra-spikes inlay among the regular sharp spikes in the eyeball velocity phase
3. Optokinetic nystagmus test	The demonstration of the clustered slow-phase (pursuit) spikes is poor in the eyeball velocity phase, or is not symmetrical in rightward and leftward direction
4. Bithermal caloric test	The unilateral-weakness percentage is over 25%
5.Visual fixation suppression test	The slow-phase waves of eyeball velocity could not be inhibited by eye opening immediately in the eyeball velocity phase

Classification of vestibulopathy

In order to define the nature of the vestibulopathy, we categorized our findings into 4 groups: (i) central vestibulopathy: less than 25% of unilateral-weakness ratio and presence of one or more of the following pathologic results: horizontal pursuit, horizontal saccade, optokinetic nystagmus, and post-caloric-test visual fixation suppression test; (ii) peripheral vestibulopathy: over 25% of unilateral-weakness ratio, and unremarkable results in all of the following tests: horizontal pursuit, horizontal saccade, optokinetic nystagmus, and post-caloric visual fixation suppression test; (iii) mixed vestibulopathy: over 25% of the unilateral-weakness ratio, and presence of one or more of the following pathologic results: horizontal pursuit, horizontal saccade, optokinetic nystagmus, and post-caloric visual fixation suppression test; (iv) no vestibulopathy: unremarkable findings in the all ENG battery.

Study size, data sources and quantitative variables

The VM group (n=18) was separated into two subgroups: (i) VAH subgroup: 8 patients with VAH (4 men and 4 women) (patients 1-8 in Table 2 and Figure A), and (ii) non-VAH subgroup: 10 patients without VAH (5 men and 5 women) (patients 9-18 in Table 2). The other 78 of these 96 patients, who did not have VM, were defined as the non-VM group and among this group, cervical MRA showed that 26.9% (n=21) had VAH. Because the VM group always had severe or moderate vestibular symptoms, the ENG studies were all hospitalization examinations. In order to prevent nausea and vomiting and make the examinee more submissive to the following electrophysiologic study, one vial of prochlorperazine was routinely intravenously administered before the ENG battery.

By reviewing the medical chart, details records of these 18 VM patients, including age, gender, systemic disease (hypertension and/or diabetic mellitus), precipitating factor, vestibular symptoms (pattern and duration), migraine (with or without aura), headache features (location, duration and pain intensity) and medication were collected.

Table 2. Participant data and the result of electronystagmogram battery

Patient	Age/Gender	Systemic disease	Migraine	Headache location	VAH side	Vestibulopathic condition
A. with VAH (N=8)						
1	49/M	No	with typical aura	Holocephalic	Left	Central vestibulopathy
2	47/M	No	with brainstem aura	Frontal	Right	No
3	49/M	No	without aura	Holocephalic	Left	Central vestibulopathy
4	66/M	No	without aura	Holocephalic	Left	Central vestibulopathy
5	40/F	HTN	with brainstem aura	Hemicephalic	Right	Central vestibulopathy
6	36/F	No	without aura	Holocephalic	Right	Central vestibulopathy
7	33/F	No	without aura	Holocephalic	Right	Central vestibulopathy
8	67/F	No	without aura	Hemicephalic	Right	No
B. without VAH (N=10)						
9	40/M	No	with typical aura	Hemicephalic		Central vestibulopathy
10	47/M	DM	without aura	Vertex		Mixed vestibulopathy
11	39/M	No	without aura	Hemicephalic		No
12	28/M	No	without aura	Hemicephalic		Mixed vestibulopathy
13	50/F	No	with typical aura	Holocephalic		Central vestibulopathy
14	52/F	No	with brainstem aura	Hemicephalic		No
15	13/F	No	with brainstem aura	Holocephalic		Mixed vestibulopathy
16	60/F	HTN, DM	with brainstem aura	Occipital		Central vestibulopathy
17	45/F	No	without aura	Holocephalic		No
18	54/M	No	without aura	Vertex		Central vestibulopathy

HTN= hypertension, DM= diabetic mellitus, VAH=vertebral artery hypoplasia

Statistical methods

An χ^2 test was used to compare the difference in the prevalence of VAH between the VM and non-VM groups. A student-t test was used to compare with the difference in the age between the VAH and non-VAH subgroups in the VM group. In the VM group, a Fisher's exact test was used to compare (i) the differences of gender, systemic diseases and migraine with aura between the VAH and non-VAH subgroups, (ii) the differences of the characteristics of the vestibular symptoms and the migraine features between the VAH and non-VAH subgroups, and (iii) the differences of the 4 sub-divisions of vestibulopathy between the VAH and non-VAH subgroups. In order to avoid any arbitrary statistical analysis, the α values of the above statistical methods were all 0.01.

Interpretation Bias

The reports of the ictal ENG battery of the 18 patients were not only interpreted by the first author but also the third author, a senior neurotologist, in order to prevent the inter-observer interpretation bias. All the vestibulopathic conditions were confirmed by the same two neurotologists.

RESULTS

Participants

There was not a significant difference in the prevalence of VAH between the VM and non-VM groups (44.4% vs 26.9%) (p=0.144, χ^2 test). The characteristics of participants were described in Table 2. In the VM group, none of the patients had an eardrum perforation or chronic otitis media. Therefore, the bi-thermal caloric test could be performed for all of them. None of gender, age and systemic disease age was different between the VAH and non-VAH subgroups (p=0.363 in gender; p=0.386 in age, p=0.441 in systemic disease) (Table 2).

Outcome Data

The ENG findings of the two subgroups are described as below.

VAH subgroup (n=8)

The male-to-female ratio was 1.0, and they were aged 48.4±11.8 (average±SD). One of the 8 patients had systemic disease. Five patients had right-sided VAH, and

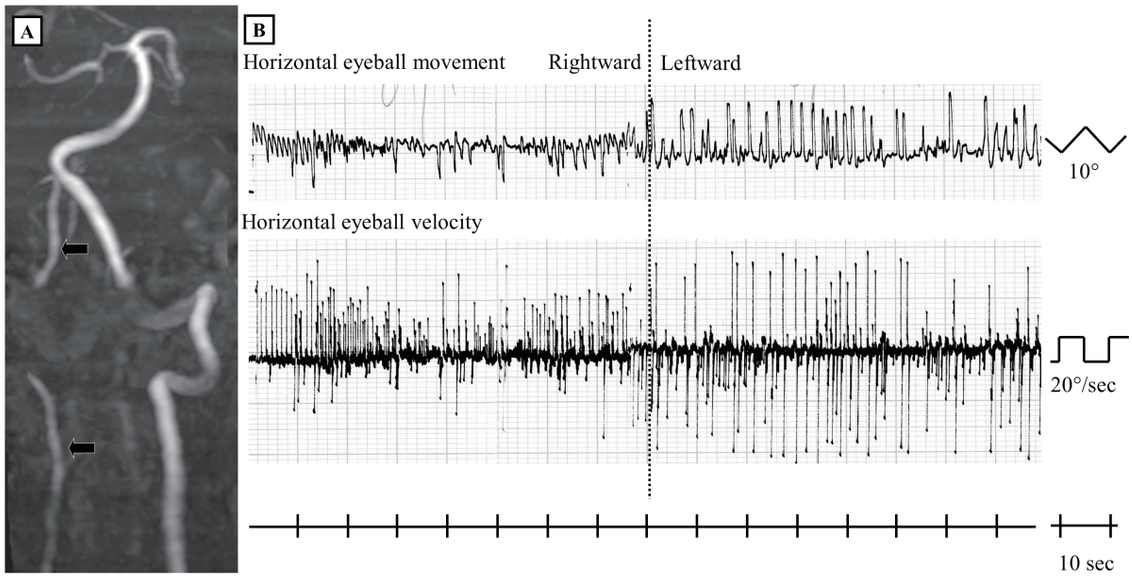

Figure 1. (A) Time-of-flight magnetic resonance angiogram of Patient 5 shows a hypoplastic right vertebral artery (diameter 1.8 mm)(filled arrow). (B) An electronystagmogram shows the demonstrations of clustered slow-phase spikes (pursuit) are poor in both rightward or leftward optokinetic nystagmus when this patient presented with vertigo. A central vestibulopathy is impressed.

the others had left-sided VAH (Table 2) (Figure A). During the vestibular episodes in VM attacks, one patient had a pathological horizontal pursuit test (patient 6), none of them had a pathologic horizontal saccade test, 5 patients had pathologic optokinetic nystagmus tests (patients 1, 3-5 and 7) (Figure B), and no patients had a pathological bithermal caloric test or pathological visual suppression test. Two patients (patients 2 and 8) with unremarkable ENG findings were thought to have no vestibulopathy, and 6 patients (patients 1, 3-7) were thought to have central vestibulopathy (Figure)(Table 2).

Non-VAH subgroup (n=10)

The male-to-female ratio was 1.0, and they were aged 42.8±7.0. Two of the 10 patients had systemic disease. During the vestibular episodes in VM attacks, 5 had pathological horizontal pursuit tests (patients 13-16 and 18), 5 patients had pathological saccade test (patients 10, 12, 13, 15 and 18), 5 patients had pathological optokinetic nystagmus test (patients 10, 12, 13, 16 and 18), 3 patients had pathological bi-thermal caloric tests (patients 10, 12 and 15), and none of the 10 patients had a pathological visual suppression test. Thus, 3 patients (patients 11, 14 and 17) with unremarkable ENG findings were thought to have no vestibulopathy; 4 patients (patients 9, 13, 16 and 18) had central vestibulopathy, and 3 patients (patients 10, 12 and 15) were thought to have mixed vestibulopathy (Table 2).

Descriptive data and other analyses

The characteristics of vestibular symptom were described in Table 3. There was a significant difference in duration of vestibular symptoms (p=0.002) between the two subgroups. Fifty percent of the VAH subgroup (4 in 8) had vestibular symptoms for 1-6 hours during headache, but 50% of the non-VAH subgroup (5 in 10) had these symptoms for only 5-10 minutes. There were not any significant difference in precipitating factor (p=0.029) and vestibular pattern (p=0.118).

The characteristics of the migraine were described in Table 4. There were not any significant difference in the ratio of migraine with and without aura (p=0.323), headache location (p=0.019), headache duration (p=0.101), pain intensity (p=0.343) and medication (p=0.170-0.183). The medication included (i) β-antagnonist (propanolol), benzodiazepam (alprozolam, oxazolam or brotizolam) and SSRI (trazodone) for migraine prophylaxis, (ii) anti-vertigo agents (diphenidol, betahistine or nicametate),

Table 3. Description of the vestibular symptoms

Characteristics (n [%])	A: With VAH (N=8)	B: Without VAH (N=10)	p value
Precipitating factor			0.029
Anger	1 (12.5%)		
Sleep deprivation	1 (12.5%)	5 (50%)	
Alcohol drinking		1 (10%)	
None	6 (75.0%)	4 (40%)	
Pattern			0.118
Persistent vertigo	7 (87.5%)	6 (60%)	
Persistent dizziness		2 (20%)	
Persistent dizziness with head motion-induced paroxysmal vertigo	1 (12.5%)	2 (20%)	
Duration			0.002
≥ 5 minute, < 10 minutes		5 (50%)	
≥ 10 minutes, < 1 hour	3 (37.5%)	2 (20%)	
≥ 1 hour, < 6 hours	4 (50.0%)	1 (10%)	
≥ 6 hours, < 1 day	1 (12.5%)	1 (10%)	
≥ 1 day, < 3 days		1 (10%)	

VAH=vertebral artery hypoplasia

Table 4. Description of the migraine features

Characteristics (n [%])	A: With VAH (N=8)	B: Without VAH (N=10)	p value
Migraine			0.323
With aura	3 (37.5%)	5 (50%)	
Without aura	5 (62.5%)	5 (50%)	
Headache location			0.019
Frontal	1 (12.5%)		
Hemicephalic	2 (25.0%)	4 (40%)	
Holocephalic	5 (62.5%)	3 (30%)	
Occipital		1 (10%)	
Vertex		2 (20%)	
Duration			0.101
< 4 hours		1 (10%)	
≥ 4 hours, < 1 day	2 (25.0%)	5 (50%)	
≥ 1 day, < 3 days	6 (75.0%)	4 (40%)	
Pain intensity			0.343
Moderate	6 (75.0%)	9 (90%)	
Severe	2 (25.0%)	1 (10%)	
Medication			
Prophylaxis	6 (75.0%)	10 (100%)	0.183
Symptomatic control	6 (75.0%)	8 (80%)	0.412
Both	4 (50.0%)	8 (80%)	0.170

VAH=vertebral artery hypoplasia

antiemetics (prochlorperazine), dihydroergotamine, NSAID (aspirin or diclofenac), and analgesics (acetaminophen) for symptomatic control, and (iii) agents for both prophylaxis and symptomatic control.

Main result

Totally (Table 2), there was a significant difference (p=0.0096) in the 4 sub-divisions of vestibulopathy between the VAH and non-VAH subgroups: 75% (6 in 8) of those with VAH had central vestibulopathy, and 25% (2 in 8) had no vestibulopathy; 40% (4 in 10) of those without VAH had central vestibulopathy, and 30% (3 in 10) had mixed vestibulopathy, and 30% (3 in 10) had no vestibulopathy.

DISCUSSION

Subjects and methods

Although digital subtraction cerebral angiography is the standard study for searching cerebral vascular diseases, it is an invasive procedure and about 0.4% of the patients remained to have persistent neurologic complications after this checkup [13]. In recent days, cerebral MRA has been thought to be able to replace traditional cerebral digital subtraction angiography because of the convenience and safety to detect intracranial vascular abnormalities, and it has a sensitivity of 94.7% and specificity of 97.3% in detecting steno-occlusive disease [14]. However, such a neuroimaging study is not recommended for patients with nonacute and recurrent migraine featuring unchanged headache pattern in a short time and without any neurologic focal sign [15], and a neuroimaging study was thought to play little role in the diagnosis of VM [9,11].

With several reasons including limitation of refund from the Taiwan National Health Insurance, migraine patients with vestibular complaints did not routinely accepted imaging study of the brain unless a central vertigo was highly suspected. The patients were recruited from 2008 to 2010 and at that time, the diagnostic criteria of VM we have used in this paper have not yet been published [9]; however, delicate chart records of the disease pattern, characteristic, frequency, duration, interval,

accompanied symptoms and others could provide us with enough information to make the diagnosis of VM. In order to elucidate the important issue that vestibulopathy in VM was a primary migraine-associated insult rather than an event secondary to the intracranial lesions including acute infarction, meningitis, intracranial hypo- or hypertension, hemorrhage, neoplasm or multiple sclerosis, neuroimaging studies were done in these VM patients.

The ENG battery was the basic electrophysiologic study at the neurotological clinic. In the horizontal pursuit test, the rightward or leftward visual-ocular circuit, which was modulated by the cortico-ponto-cerebellar circuit, passing through ipsilateral occipital lobe (area 19), ipsilateral pontine gaze center, and ipsilateral abducens nucleus/nerve, was investigated. In the horizontal saccade test, the rightward or leftward visual-ocular circuit, passing through contralateral frontal lobe (area 8), ipsilateral pontine gaze center, and ipsilateral abducens nucleus/nerve, was investigated. In the optokinetic nystagmus test, the fast phase of horizontal saccade and the slow phase of contralateral horizontal pursuit, passing through contralateral occipital (area 19), parietal lobe, frontal lobe (area 8), bilateral pontine gaze centers, bilateral abducens nucleus/nerves, were investigated. In the bithermal caloric test, the vestibulo-ocular reflex, which was modulated by the ipsilateral cerebellar flocculonodular lobe, passing through the ipsilateral vestibular nerve, ipsilateral vestibular nucleus, abducens nuclei/nerves, and oculomotor nuclei/nerves, was checked up [16]. In the visual suppression test, the visual-vestibular reflex, modulated by the cerebellar flocculonodular lobe, was checked up [17]. It was a limitation in this study that the ENG battery could only investigate the pathophysiologic condition of the central nervous system above the ponto-medullary junction rather than that below the level.

However, before the ENG study, the patients were confirmed not to have any neurologic disease except migraine, and not to have any cerebral organic lesion by neuroimagings. Theoretically, all of saccade, pursuit and optokinetic nystagmus should be normal in a normal examinee, and a pathologic smooth pursuit, saccade and optokinetic nystagmus indicated impairment of central vestibulopathy. A pathologic vestibular-ocular reflex indicated peripheral vestibulopathy, but a pathologic visual fixation suppression test indicated central vestibulopathy.

Interpretation and implication

Some mechanisms of VM have been assumed. The pain reflex from the trigeminal nerve system might either activate the sensory C-nerves of the trigeminal vascular system in the inner ear and induce peripheral impairment directly, or might activate the afferent fibers of the olivocochlear system via the interconnection between the brainstem and vestibular nucleus and induce central vestibulopathy indirectly [18,19]. The neuropeptides, such as substance P, neuropeptide A, calcitonin gene related peptide, etc., released from the trigeminal nerve system might influence the local hemodynamics of the brainstem or vestibular labyrinth and might further induce the central or peripheral vestibulopathy [20]. In the total VM group of our study, VAH was closely related with central vestibulopathy rather than peripheral or mixed vestibulopathy in the vestibular episodes of VM. Hence, the topographic factor of VAH little influenced the ipsilateral peripheral vestibular labyrinth in the vestibular episodes of VM although we could not explain the close correlation between VAH and central vestibulopathy.

In one study, the risk of VAH in patients who had migraine with aura was 14 times higher than in the normal controls [7]. However, in our VM group, there was no statistical difference in the migraine with aura and migraine without aura between the VAH and non-VAH subgroups. Besides, the VAH subgroup is prone to have vestibular symptom with duration of 1-6 hours, and however, the non-VAH subgroup is prone to have vestibular symptom with duration of 1-10 minutes.

Limitations

Although 18.8% (18 of 96) had VM in this study, in fact there were more patients diagnosed with VM, but without neuroimaging, at the specific neurotological clinic. Those with only migraine without vertigo/dizziness usually directly visited the neurologic clinics rather than the neurotological clinic in the department of otorhinolaryngology, let alone those with neurological focal signs, conscious change, dementia or other morbid systemic disease; therefore, it was not available for the difference in the VM incidence between migraine with aura and without aura from the neurotological clinic, and it was not unexpected that acute infarction, meningitis, intracranial hypo- or hypertension, hemorrhage, neoplasm

or multiple sclerosis was not found in the neuroimaging of these 96 patients' patients. Therefore, these above-mentioned conditions should be considered as results restricted to those with VM in our study, and further studies are needed to address these important issues in VM and a further generalizability of the study result.

CONCLUSION

Although VAH happened more often to the right VA, there were equal numbers of VAH at both sides (right/left: 4/4) in our study, and there was no significant difference in the prevalence of VAH between the VM and non-VM groups in the vertigo/dizziness patients. In addition, VAH was not more significantly prevalent in the VM patients than the non-VM ones. The topographically selective damage of peripheral vestibular labyrinth was small by VAH in the vestibular episodes of VM.

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