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Case Report

Definite Meniere's Disease with Definite Migrainous Vertigo: Two Case Reports

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ABSTRACT

Although the patho-physiological mechanism is currently considered to be different between Meniere's disease (MD) and migraine, the clinical overlap of vestibular-cochlear symptoms in patients meeting criteria for migrainous vertigo (MV) and MD remains an important unsolved issue. In the past three years, 53 patients (33 women and 20 men) were diagnosed with definite MD. Among them, two patients' (one man and one woman) vertigo also fulfilled the diagnostic criteria of definite MV. The first patient, MD developing from MV, which was associated with migraine without aura; and the second patient, MD as auras of basilar-type migraine. Both disease courses contained both definite MD and definite MV, and neither could be excluded. We hope to address whether definite MD with definite MV can be ascribed to "migrainous MD"

on account of the diagnostic particularity, which is not attributable to another type of central vertigo. (Rawal Med J 2011;36:322-325).

Key words

Meniere's disease, migrainous vertigo, migraine without aura, basilar-type migraine, central vertigo.

INTRODUCTION

Although certain Meniere's disease (MD) is diagnosed by histopathological evidence of endolymphatic hydrops, definite MD is confirmed by four clinical conditions adopted by American Academy of Otolaryngology-Head and Neck Surgery guidelines of 1995 (Table 1A). [1] Migraine may influence the central and/or peripheral vestibular pathway and thus induce migrainous vertigo (MV). [2] Definite MV was confirmed using the following four conditions suggested by Neuhauser et al (Table 1B). [2] Although the patho-physiological mechanism between MD and migraine is currently considered to be different, [3] there is a possible relationship because migraine has a higher prevalence in the MD group (56%) than in a control group (25%); [4] in addition, a study with air-conducted vibration-cervical vestibular evoked myogenic potential (ACV-cVEMP) also demonstrated the scenario. [5]

In the past three years, 53 Taiwanese (33 women and 20 men) aged 45.7±11.1 (average±SD) years were diagnosed at our subspecialty clinic with definite MD. Among them, two patients' disease course also fulfilled the diagnostic criteria of definite MV. Because the clinical overlap of vestibulo-cochlear symptoms in patients meeting criteria for MV and MD remains an important unsolved issue in vestibular research, herein we report them.

CASE PRESENTATION 1

A 49-year-old male motorcycle repairman was weekly bothered by moderate throbbing holo-cephalalgia with right tinnitus, vertigo, nausea, vomiting, phonophobia, photophobia, intolerance of head movement and head motion intolerance between 1998 and 2002. The symptomatic duration was between four hours and a day. From 2002, the vertiginous headaches did not recur any more but vertigo with right tinnitus, nausea, vomiting, phonophobia, photophobia and intolerance of head movement still recurred at intervals of between four and six months. He was conservatively treated with diazepam, diphenidol and nicametate for each vertiginous attack.

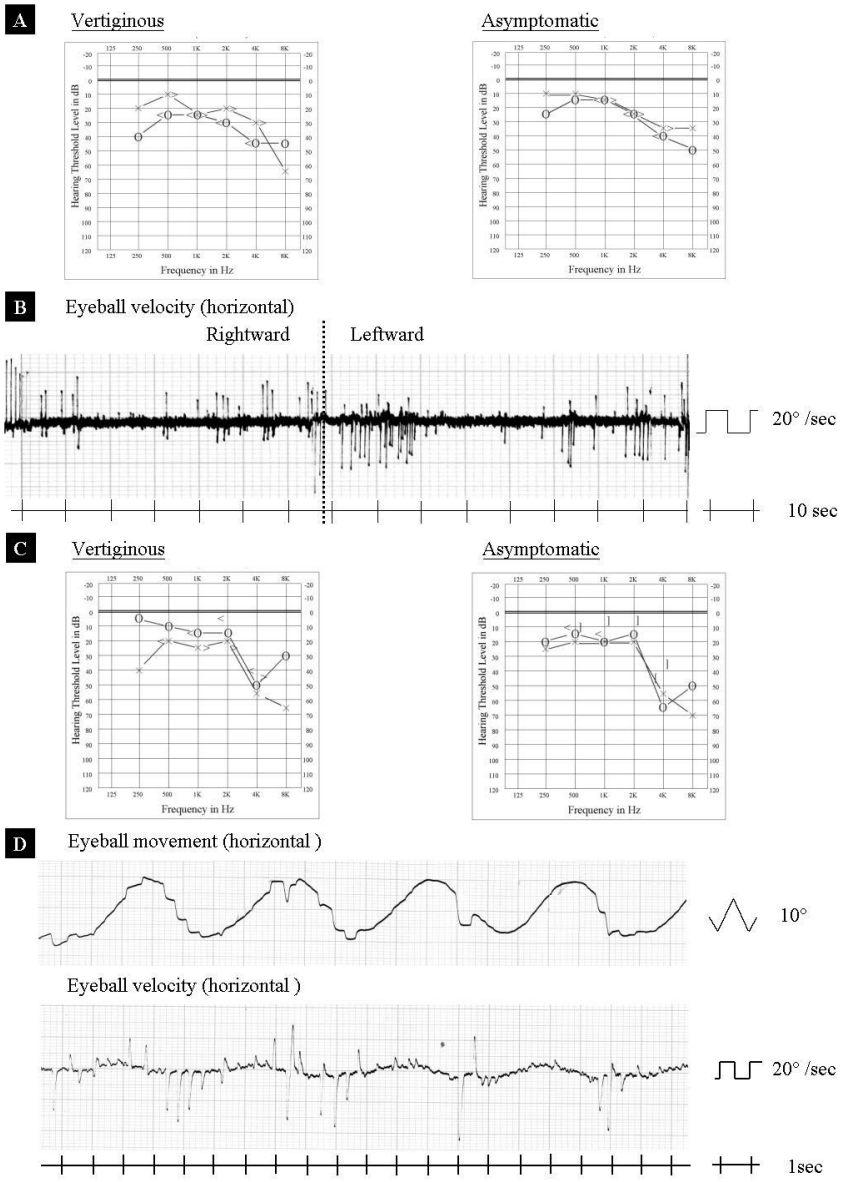
Table 1. Diagnostic criteria.

A: Definite Meniere's disease [1]	**B: Definite migrainous vertigo** [2]
(a) at least two definitive spontaneous episodes of vertigo persisting at least 20 minutes (b) pure tone audiometry documentation of hearing loss on at least one occasion (c) tinnitus or aural fullness occurs in the affected ear (d) when other causes are excluded	(a) severe or moderate episodic vestibular symptoms, including rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance and sensations of imbalance, or illusory self/object motion that is provoked by head motion (b) migraine (ICHD-II) [12] (c) migrainous headache, photophobia, phonophobia, visual or other auras during at least two vertiginous attacks (d) when other causes are excluded by appropriate investigations

During one vertiginous attack, rightward gaze nystagmus with direction preponderance was noted. Pure tone audiometry (AC 40, Interacoustics, Denmark) documented the average hearing threshold between 500 and 2,000 Hz was 26 dB hearing level (dBHL) in the right ear and 13 dBHL in the left ear (Fig. 1A). The bilateral tympanogram was A-type. Optokinetic electronystagmogram (NY-41, Rion, Japan) showed poor manifestation bilaterally (Fig. 1B).

The caloric tests (20°C water, 20 seconds) was normal. The latency (amplitude) of ACV-cVEMP (580-NAVPRO, Bio-Logic, USA) p13 and n23 was 20.54 msec (-12.36μV) and 23.77 msec (21.21μV) in the right ear, and 19.71 msec (-12.28μV) and 29.39 msec (41.35μV) in the left ear, respectively. The interaural amplitude difference ratio was –0.23.

Fig. 1. Pure tone audiometries (A) and optokinetic electronystagmogram (B) of case 1. Pure tone audiometries (C) and gaze pursuit electronystagmogram (D) of case 2. keys of audiogram present as follows: ○=right-ear air conduction; <= right-ear bone conduction; [=right-ear masking bone conduction; ╳ =left-ear air conduction; >=left-ear bone conduction;]=left-ear masking bone conduction.



Color-coded duplex ultrasonography (EnVisor, Philips, Andover, MA, USA) for total average cerebral blood flow, which was the sum of average blood flows measured from the two internal carotid arteries and the two vertebral arteries, was 931 mL/min. Magnetic resonance imaging (1.5 Tesla system, Picker Edge Eclipse, Picker 98 International, USA) did not show any remarkable abnormality.

When he was asymptomatic, the average hearing threshold was 18 dBHL in the right ear and 17 dBHL in the left ear (Fig. 1A). A follow-up optokinetic electronystagmogram was normal. The

total average cerebral blood flow was 880 mL/min. In conclusion, he was diagnosed with (a) right definite MD and (b) definite MV associated with migraine without aura (ICHD-II).

CASE PRESENTATION 2

A 53-year-old housewife with type II diabetes mellitus had been bothered with left severe throbbing hemi-cephalalgia, nausea, vomiting, intolerance of head movement, and head motion intolerance for nine times in the past 20 years. Left tinnitus, vertigo and photophobia always were present for 30 minutes before each headachy attack, and then remitted on the following headachy day. She was conservatively treated with diphenidol, nicametate, betahistine and oxazolam for each symptomatic attack, and by aspirin for the following headache

During one vertiginous attack before one headache attack, leftward gaze nystagmus with directional preponderance was noted. The average hearing threshold was 22 dBHL in the right ear and 13 dBHL in the left ear (Fig. 1C). The bilateral tympanogram was A-type. A gaze pursuit electronystagmogram showed unsmooth waves in the movement phase and spiking waves in the velocity phase (Fig. 1D). The caloric tests were normal. The latency (amplitude) of ACV-cVEMP p13 and n23 was 18.67 msec (-13.97μV) and 26.68 msec (11.21μV) in the right ear, and 16.48 msec (-17.32μV) and 25.64 msec (13.51μV) in the left ear, respectively. The interaural amplitude difference ratio was –0.10. The total average cerebral blood flow was 775 mL/min. During the following headachy attack, a squat-to-stand test and a hyperventilation test were able to induce the same left tinnitus, vertigo and photophobia as the vertiginous attack. When she was headachy, magnetic resonance imaging did not show any remarkable abnormality.

When she was asymptomatic, the average hearing threshold was 23 dBHL in the right ear and

17 dBHL in the left ear; in addition, the hearing threshold of 250 Hz had progressed from a vertiginous 40 dBHL to an asymptomatic 25 dBHL, as had the 1,000 Hz threshold from a vertiginous 25 dBHL to an asymptomatic 20 dBHL (Fig. 1C). A follow-up electronystagmogram was normal. The total average cerebral blood flow was 744 mL/min. In conclusion, she was diagnosed with (a) left definite MD and (b) definite MV associated with basilar-type migraine (ICHD-II).

DISCUSSION

The electronystagmogram and caloric tests investigate the vestibulo-ocular reflex passing through the upper brainstem, whereas ACV-cVEMP investigates the sacculo-collic reflex passing through the lower brainstem. In healthy Taiwanese adults, the latencies of ACV-cVEMP p13 and n23 are respectively 16.3±3.2 and 24.4±5.0 (average±2SD) msec, [6] and the normal absolute value of the interaural amplitude difference ratio should be below 0.33 (average+2SD). [7] When vertiginous, the two patients' caloric tests were normal, and the interaural ACV-cVEMP amplitude difference ratios were both in the normal range; therefore their vertigos were not purely attributable to MD. [7] Furthermore, with the first case patient, the abnormal bilateral ACV-cVEMP p13 latencies and abnormal optokinetic electronystagmogram indicated he was suffering from sacculo-affecting peripheral vertigo [8] and central vertigo. [9] With the second case patient, the abnormal unsmooth pursuit electronystagmogram indicated she was suffering from central vertigo. [9]

There were no focal neurological signs associated with disease courses of the two patients and therefore vertebral-basilar insufficiency (the presence of at least two focal signs) was unlikely by definition. [10] In addition, their total average cerebral blood flows were greater in the

vertiginous period than in the asymptomatic period and therefore a transient ischemic attack was ruled out by the color-coded duplex ultrasonography. [11] By the rules of Taiwan's National Health System, magnetic resonance imaging is not yet routinely recommended unless a central lesion is suspected; besides, the imaging is time-consuming (about 30 minutes) so could not be immediately available within the vertiginous 30 minutes' attack of the second case patient. Both of our patients' magnetic resonance imaging had been used to rule out cerebral ischemia or another organic lesion. Based on the above, definite MV was the preferred central nervous disease according to their clinical histories. Usually, either MD or MV is confirmed when the other has been excluded; however, it is difficult to make a definite diagnosis with these two patients because their disease courses fulfill both the criteria of definite MD and the criteria of definite MV, and neither could be excluded.

MV of the first case patient resulted from migraine without aura and that of the second case patient from basilar-type migraine. The mechanisms of the clinical overlap of vestibulo-cochlear symptoms in the two patients meets criteria for MV and MD. With the first patient, MD just came into being after the migraine without aura had recurred many times; therefore, the disease course is likely to be MD developing from MV. This is because the migrainous headache becomes less and less obvious as the migraineurs become older,[12] but MV remains and thus this is coincident with MD; alternatively, the migraine cerebral vasospasm has injured the endolymphatic sac, and the following attacks lead to delayed endolymphatic hydrops. [13] With the second patient, the MD always occurred before each attack of basilar-type migraine and the disease course would seem to be MD as auras of basilar-type migraine. In such a patient, the electrophysiological change originates from the brainstem and induces auditory

and vestibular dysfunctions by interfering with the brainstem or cerebellar pathways before a migrainous attack.

Although MV only occurs in a migraineur by the current diagnostic criteria, [2] a migraineur's vertigo is not always definite MV, and could be another vertiginous disease, such as definite MD; [14] similarly, a migraineur's definite MD may be MV or alternatively it may not be MV. Anyway, the two cases need longer follow up to see the value of differentiating MV or MD and the outcome.

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