Vertigo as a Symptom of Migraine

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Migraine and vertigo are common disorders, affecting about 14% and 10%, respectively, of the general population. If migraine and vertigo were unrelated, the expected comorbidity would be 1%, whereas recent epidemiological studies indicate that 3.2% of the population have both migraine and vertigo. The excess comorbidity may be attributed to two factors: 1) vertigo syndromes (including Menière’s disease, benign paroxysmal positional vertigo, and anxiety-related dizziness) that are more common in migraineurs than in controls and 2) vestibular migraine (VM) (vertigo as a symptom of migraine.) VM presents with attacks of spontaneous or positional vertigo lasting seconds to days. Headaches are often absent during acute attacks, but other migrainous features such as photophobia or auras, may be present. Like migraine headaches, VM triggers include stress, sleep deprivation, and hormonal changes. During acute attacks, there may be central spontaneous or positional nystagmus and, less commonly, unilateral vestibular hypofunction. In the symptom-free interval, vestibular testing shows mostly minor and nonspecific findings. The pathogenesis of VM is uncertain, but migraine mechanisms may interfere with the vestibular system at the labyrinth, brainstem, and cerebral cortex. Treatment includes vestibular suppressants for acute attacks and migraine prophylaxis for patients with frequent recurrences. However, treatment efficacy has not been validated by properly controlled clinical trials. VM does not fit into the 2004 International Headache Society Classification, in which “basilar-type migraine” must have at least two posterior circulation manifestations; isolated vertigo would not satisfy this criterion.

Key words: migraine; vestibular; vertigo; dizziness

Introduction

That migraine may commonly cause vertigo is recently achieving greater acceptance.1 A wider recognition of vestibular migraine began with Kayan and Hood’s 1984 paper,2 and the clinical features have since been delineated in several case series.3–8 Unfortunately, the terminology has not been uniform, with various terms (migraine-associated vertigo, migraine-related vestibulopathy, migrainous vertigo, and basilar migraine) applied to roughly the same patient population. We prefer “vestibular migraine” (VM), a term that avoids confusion with nonvestibular dizziness or motion sickness associated with migraine.9

Epidemiological Links between Migraine and Vertigo

Epidemiological observations indicate a greater than chance association of migraine...
with vertigo and dizziness. The frequency of migraine is increased in patients with dizziness and, particularly, those with unclassifiable vertigo. Conversely, significantly more patients with migraine also have vertigo compared with patients with tension headaches and headache-free controls. In the general population, migraine headaches and vestibular vertigo concur about three times more frequently than would be expected by chance. The lifetime prevalence of migraine is 14% and of vestibular vertigo is 7%; chance concurrence of the two would be 1%, but a large population study showed it to be 3.2%. Clinicians must attempt to determine whether individual patients have VM (i.e., vestibular vertigo caused by migraine), dizziness/vertigo of an unrelated cause (by chance occurring in a migraineur), or one of several vestibular and nonvestibular dizziness syndromes with increased prevalence in migraineurs. The latter group includes Menière’s disease, benign paroxysmal positional vertigo, motion sickness, and orthostatic hypotension. We will now describe the core syndrome of VM, followed by a discussion of other dizziness syndromes that are statistically associated with migraine.

**Diagnostic Criteria for Vestibular Migraine**

The current International Classification of Headache Disorders (ICHD) of the International Headache Society (IHS) does not include vertigo as a migrainous symptom in adults, except in the framework of basilar-type migraine. Although more than 60% of basilar-type migraine patients have vertigo, for a diagnosis of basilar-type migraine, the ICHD requires at least two posterior circulation manifestations lasting between 5 and 60 min, followed by a migraine headache. Less than 10% of patients with VM fulfill these criteria. Indeed, isolated vertigo is not recognized as a migraine aura in the ICHD, meaning that most adult patients with VM cannot be classified with the current criteria.

Like migraine itself (except for rare forms such as familial hemiplegic migraine), VM is not diagnosable by specific biological markers. A preliminary classification, using operational clinical criteria modeled on the ICHD, proposed two separate diagnostic categories: definite, and the more sensitive but less specific, probable vestibular migraine (Table 1). In accordance with most published reports, the proposed criteria conceptualize VM as an episodic

<table>
<thead>
<tr>
<th>TABLE 1. Diagnostic Criteria for Vestibular Migraine</th>
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<tr>
<td><strong>Definite vestibular migraine</strong></td>
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<tr>
<td>A. Episodic vestibular symptoms of at least moderate severity</td>
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<tr>
<td>B. Current or previous history of migraine according to the 2004 criteria of the IHS</td>
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<tr>
<td>C. One of the following migrainous symptoms during ≥2 attacks of vertigo: migrainous headache, photophobia, phonophobia, visual or other auras</td>
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<tr>
<td>D. Other causes ruled out by appropriate investigations</td>
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**Comment:** Vestibular symptoms are rotational vertigo or another illusory self or object motion. They may be spontaneous or positional. Vestibular symptoms are “moderate” if they interfere with but do not prohibit daily activities and “severe” if patients cannot continue daily activities.

<table>
<thead>
<tr>
<th><strong>Probable vestibular migraine</strong></th>
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<tbody>
<tr>
<td>A. Episodic vestibular symptoms of at least moderate severity</td>
</tr>
<tr>
<td>B. One of the following:</td>
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<td>(a) Current or previous history of migraine according to the 2004 criteria of the IHS</td>
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<td>(b) Migrainous symptoms during vestibular symptoms</td>
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<td>(c) Migraine-precipitants of vertigo in more than 50% of attacks: food triggers, sleep irregularities, hormonal change</td>
</tr>
<tr>
<td>(d) Response to migraine medications in more than 50% of attacks</td>
</tr>
<tr>
<td>C. Other causes ruled out by appropriate investigations</td>
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...
vestibular disorder. Although several reports included patients with nonspecific dizziness\textsuperscript{3–5} or with permanent symptoms, for the sake of simplicity, the core syndrome was defined first, with exceptions and variations planned to be included at later date. Furman and colleagues developed a diagnostic interview applying these criteria.\textsuperscript{18}

**Prevalence and Demographic Aspects of Vestibular Migraine**

According to the above diagnostic criteria, the frequency of definite VM was 7\% in a group of 200 consecutive dizziness clinic patients, and 9\% in a group of 200 migraine patients.\textsuperscript{7} Using the criteria, a two-stage population-based study ($N = 4869$ adults) with screening interviews followed by expert telephone interviews, estimated the lifetime prevalence of VM to be 0.98\% (95\% CI 0.7–1.37).\textsuperscript{15} Of note, definite VM accounted for only a third of migraineurs previously diagnosed with “vestibular vertigo,” which emphasizes the necessity of a thorough neuro-otological evaluation to exclude other diagnoses.\textsuperscript{15}

VM may occur at any age,\textsuperscript{3,4,6} with a reported female-to-male ratio between 1.5- and 5-to-1.\textsuperscript{4–7} Familial occurrence is not uncommon, probably based on an autosomal dominant pattern of inheritance with decreased penetrance in men.\textsuperscript{20} In most patients, migraine headaches begin earlier in life than VM.\textsuperscript{6,7} Some patients were headache-free for years before VM first manifested.\textsuperscript{6} VM seems to occur more often in patients with migraine without aura than in those with aura,\textsuperscript{2,5,6} and the headaches are often replaced by isolated vertigo attacks in menopausal women.

**Clinical Features**

Since migraineurs can present with dizziness or vertigo due to multiple causes, the first diagnostic step is distinguishing between vertigo (a vestibular symptom) and nonvestibular dizziness. This distinction can usually be made by a careful history: a sense of rotation or other illusory sensations of motion indicate vertigo; whereas sensations of lightheadedness, dizziness, or impending faint imply nonvestibular dizziness. Nonspinning dizziness occurring only during standing or walking usually indicates a cause other than VM. A residual area of uncertainty often remains, either as a semantic problem or because mild vestibular dysfunction may present with dizziness rather than vertigo.

Patients with VM typically report spontaneous or positional vertigo. Some experience a sequence of spontaneous vertigo transforming into positional vertigo after several hours or days.\textsuperscript{21,22} This positional vertigo is distinct from benign paroxysmal positional vertigo (BPPV) with regard to duration of individual attacks (often as long as the head position is maintained in VM versus only seconds in BPPV), duration of individual episodes (minutes to days in VM versus weeks to months in BPPV), and nystagmus findings (heterogeneous in direction and persistent in VM versus coplanar to the affected canal and transient in BPPV). Forty to 70\% of VM patients experience positional vertigo, but not necessarily with every attack.\textsuperscript{2,20,23} Head motion intolerance, quite similar to motion sickness (i.e., imbalance, illusory motion, and nausea) is a frequent additional symptom,\textsuperscript{4,11} as is visual vertigo (i.e., vertigo provoked by moving visual scenes).\textsuperscript{4,19,24} Both attack duration and frequency can vary between patients and in individual patients. The duration of vertigo ranges from seconds (about 10\%) and minutes (30\%) to hours (30\%) and several days (30\%),\textsuperscript{2,3,5,6,25} Some patients require weeks to recover from an attack. The attacks may occur days, months, or even years apart in an irregular fashion. Only 10\% to 30\% of VM patients have vertigo with the typical duration of a migraine aura—5 to 60 minutes.\textsuperscript{6,7}

VM, in addition to not often conforming to the ICHD duration criteria for an aura,
TABLE 2. Clinical Features of Definite Vestibular Migraine in 33 Patients

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>%</th>
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<tr>
<td>Vestibular symptoms</td>
<td></td>
</tr>
<tr>
<td>Rotational vertigo</td>
<td>70</td>
</tr>
<tr>
<td>Other illusory self or object motion</td>
<td>18</td>
</tr>
<tr>
<td>Positional vertigo</td>
<td>42</td>
</tr>
<tr>
<td>Head motion intolerance</td>
<td>48</td>
</tr>
<tr>
<td>Duration of vestibular symptoms</td>
<td></td>
</tr>
<tr>
<td>Seconds to 5 min</td>
<td>18</td>
</tr>
<tr>
<td>5 to 60 min</td>
<td>33</td>
</tr>
<tr>
<td>1 hr to 1 day</td>
<td>21</td>
</tr>
<tr>
<td>&gt; 1 day</td>
<td>28</td>
</tr>
<tr>
<td>Migrainous symptoms during vertigo</td>
<td></td>
</tr>
<tr>
<td>Migrainous headache</td>
<td>94</td>
</tr>
<tr>
<td>Always</td>
<td>46</td>
</tr>
<tr>
<td>Sometimes</td>
<td>48</td>
</tr>
<tr>
<td>No headache</td>
<td>6</td>
</tr>
<tr>
<td>Photophobia</td>
<td>70</td>
</tr>
<tr>
<td>Phonophobia</td>
<td>64</td>
</tr>
<tr>
<td>Visual or other auras</td>
<td>36</td>
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</tbody>
</table>

aSeveral patients had more than one type of vestibular symptoms.
bNone of the patients had only head-motion intolerance.

also differs in its relationship to the migraine headaches. Vertigo may precede headache, as would be typical for an aura, may begin with the headache, or may appear late in the headache phase. Many patients experience attacks both with, and without, headache.3,5,7 Quite frequently, patients have an attenuated headache with their vertigo as compared to their usual migraine.5 In some, vertigo and headache never occur together,3,5,7 Here the diagnosis must be based on migrainous symptoms during the attack, rather than a headache. These symptoms include photophobia, phonophobia, osmophobia, and visual or other auras. Such accompaniments are of diagnostic importance, since they may represent the only apparent connection between vertigo and migraine (Table 2). Patients must be asked specifically about these migrainous symptoms, as they often fail to mention them. The renowned migraine authority, Neil Raskin (personal communication to Robert Daroff), commented that most VM patients neglect to mention accompanying scintillations and photopsias because these symptoms are not disturbing or for fear they would be considered as “crazy.” A dizziness diary is useful for prospective recording of these associated features.

Hearing loss and tinnitus are not prominent symptoms of VM.2,4,5,26 The hearing loss is usually mild, transient, and not progressive,5 although some patients do have fluctuating hearing loss, suggestive of Menière’s disease, as well as migrainous features during attacks of presumed VM.2,5,27

Migraine-specific precipitants of vertigo attacks may provide valuable diagnostic information. These include menstruation, deficient or irregular sleep, excessive stress, specific foods (including sharp cheese, red wine, and glutamate) and sensory stimuli such as bright or scintillating lights, intense odors, or noise.

When migrainous accompaniments and typical precipitants are absent, VM may still be considered diagnostically after other potential causes have been eliminated. In such instances, a favorable response to antimigraine drugs may support the suspicion of an underlying migraine mechanism. However, the apparent efficacy of a drug cannot be a definite confirmation of the diagnosis, since spontaneous improvement, placebo response, and additional drug effects (e.g., anxiolytic or antidepressant) may be operative.

In summary, the clinical presentation of VM is quite variable, and its connection to migraine can be subtle. Keys to the diagnosis are repeated concurrence of migraine symptoms and vertigo, migraine specific precipitants, and the possible response to antimigraine drugs.

Neuro-otologic Findings

In most VM patients, the general neuro-logical and otologic examinations are normal in the symptom-free period,3 but about 20% have unilateral hypo-excitability to caloric
stimulation, and about 10% have directional preponderance of caloric-induced nystagmus. These findings are not specific for VM and may also occur in other vestibular syndromes, and even in migraine patients without vestibular symptoms. Neuro-ophthalmological evaluation may reveal mild ocular motor abnormalities in the absence of other brainstem or cerebellar signs. Vitkovic and coworkers found that patients with definite VM became nauseated after caloric testing four times more often than patients with other vestibular disorders. Von Brevern and colleagues, studying 20 patients during the acute phase of VM, noted that 19 had imbalance with increased sway on tandem Romberg or tandem walking, or both. Fourteen had pathological nystagmus: three had spontaneous peripheral nystagmus, another three had spontaneous central nystagmus, five had central positional nystagmus, and three had both central spontaneous and positional nystagmus. Unlike BPPV, VM positional nystagmus always persisted as long as the provoking position was maintained, and usually did not beat in the plane of positioning. A unilateral deficit of the horizontal vestibulo-ocular reflex was present in the three patients with peripheral spontaneous nystagmus, one of whom did not recover peripheral function when asymptomatic. These authors concluded that during acute VM episodes, 10 patients (50%) had central vestibular dysfunction, three (15%) peripheral vestibular dysfunction, and seven (35%) could not be classified.

In clinical practice, history will usually provide more diagnostic clues than vestibular testing, as there are no specific abnormalities for VM. In patients with clear-cut histories, no additional vestibular tests are required, but testing in symptom-free intervals can reassure patients and physicians as to the absence of an alternative diagnosis. This is particularly relevant in patients whose studies during, or shortly after, an attack demonstrated distinct peripheral or central abnormalities, which would be expected to have improved within a few weeks.

**Pathophysiology**

The pathophysiological mechanisms of VM are uncertain, but investigators have proffered several hypotheses. Cortical spreading depression, the presumed mechanism of migraine auras, may be operative in patients with short attacks. Spreading depression could produce vestibular symptoms by involving the multisensory cortical areas that process vestibular signals, which are mainly located in the posterior insula and at the temporo-parietal junctions. However, canal paresis and complex positional nystagmus during the acute stage of VM cannot be explained by cortical dysfunction.

Transmitters, putatively involved in migraine (calcitonin-gene-related peptide, serotonin, norepinephrine, and dopamine), modulate the activities of central and peripheral vestibular neurons and could contribute to the pathogenesis of VM. Unilateral release of these substances—analogous to the usual unilateral location of migraine headaches—could cause a static vestibular imbalance leading to rotational vertigo, whereas bilateral release would cause a state of altered vestibular excitability leading to a motion sickness–type of dizziness.

Genetic defects of ion channels cause various paroxysmal neurological disorders. The abnormal voltage-gated calcium-channel gene CACNA1A in familial hemiplegic migraine (FHM) and episodic ataxia type 2 (EA-2)—both of which may have vertigo and migraine headache as prominent symptoms—has prompted the search for a susceptibility gene for VM in the same region, but no such genetic defect could be identified.

The only hypothesis that is based on an experimental model of VM relates to the reciprocal connections between the trigeminal and vestibular nuclei. Trigeminal activation by painful electrical stimulation of the forehead produces spontaneous nystagmus in migraine patients but not in controls, indicating that migraineurs have a lowered threshold for
crosstalk between these neighboring brainstem structures.37

**Treatment**

When attacks of VM are severe and frequent, acute or prophylactic treatment is warranted but, apart from one small and inconclusive study on the use of zolmitriptan for acute VM,38 current treatment recommendations are based on expert opinion rather than randomized placebo-controlled trials.

A few case reports suggest that medication used for migraine prophylaxis may be effective. These include propranolol,39 metoprolol,6 tricyclic antidepressants,8 pizotifen,8,19 and flunarizine.6 The carbonic anhydrase inhibitors, acetazolamide40 and dichlorphenamid,41 which are not ordinarily used for migraine prophylaxis, have also been used successfully, but these studies were not randomized or controlled.

Treatment of acute VM with acute migraine medication can be attempted with triptans42,38 and vestibular suppressants such as promethazine, dimenhydrinate, and meclizine.42 A retrospective study found that the effect of triptans on vertigo correlated with its effect on headache.43 Nonpharmaceutical approaches may be more efficacious than drugs in individual patients. A thorough explanation of the migrainous origin of the attacks may relieve unnecessary fears, and avoidance of identified triggers and vestibular rehabilitation may also be helpful.44

**Migraine, Motion Sickness, and Sensitivity to Visual Motion**

Discomfort thresholds to sensory stimulation are decreased in migraineurs, both during attacks and in the intervals. Examples include sensitivity to light, sound, and smells. The vestibular system is no exception. Motion sickness occurs more frequently (30% to 70%) in patients with migraine than in headache-free controls or patients with tension-type headaches (20% to 40%).2,11,24 The association is more pronounced in children15 and in migraine with aura.11 Migraineurs also have more “visual vertigo” induced by optokinetic stimuli,11,24 which can be conceptualized as a decreased threshold for visual–vestibular interaction. In addition, headache, scalp tenderness, and photophobia can be more easily provoked by optokinetic stimulation in migraine patients than in controls.46

Differentiating between episodic motion sickness and attacks of VM induced by motion stimuli may be difficult, but the distinction can be made based on the type and duration of symptoms. Nausea and dizziness improving after cessation of the motion stimulus suggests motion sickness, whereas the persistence of rotational or positional vertigo suggests VM triggered by passive motion. Chronic VM19 may be explained by a constantly lowered threshold to motion stimuli. Of note is that motion sickness could be prevented by rizatriptan in VM, but not in patients with migraine alone.47

**Cerebellar Dysfunction**

Cerebellar dysfunction causes imbalance that patients may describe as “dizziness.” Some families with FHM develop progressive cerebellar ataxia and nystagmus.48 Mutations in the CACNA1A gene coding for the $\alpha_{1A}$ subunit of a neuronal Ca$^{2+}$ channel, which is heavily expressed in the cerebellum, occurs in FHM as well as in EA-2 and spinocerebellar ataxia type 6.50 EA-2 is characterized by short bouts of cerebellar ataxia, often with vertigo, interictal nystagmus, and, in approximately 50%, migraine.51 Both FHM and EA-2 have the typical symptoms of basilar-type migraine.52,51 Cerebellar signs are usually not present in the common types of migraine, but some investigators found subclinical hypermetria and other subtle cerebellar signs in patients with migraine, with or without aura.53,30 May and colleagues
suggested dysfunctional Ca\textsuperscript{2+} channels as a possible cause, based on involvement of the CACNA1A gene region in some families with nonhemiplegic migraine aura.\textsuperscript{54} Another link between migraine and cerebellar dysfunction is the recent observation that migraineurs, particularly when they have migraine with aura, have a high frequency of subclinical cerebellar infarctions.\textsuperscript{55}

**Menière’s Disease**

An increased frequency of migraine in patients with Menière’s disease (MD) is well documented.\textsuperscript{56,27} Migraine was twice as high in a group of 78 patients with unilateral or bilateral MD (based on the American Academy of Otolaryngology criteria\textsuperscript{57}), than in an age- and sex-matched control group (56\% versus 25\%, \(P < 0.001\)).\textsuperscript{27} Migraine leads to a greater susceptibility of developing MD, as suggested by a study in which MD patients had an earlier onset of symptoms and a greater susceptibility to bilateral hearing loss when they also had migraine.\textsuperscript{58} Vestibular function tests only modestly assist in differentiating between VM and MD,\textsuperscript{59} but the distinction can usually be made based on hearing loss being only occasional, mild, and nonprogressive in VM,\textsuperscript{5} while it is a regular aggressive accompaniment of MD. Nevertheless, in some patients, the distinction is not possible.\textsuperscript{27} A possible explanation might be that MD and VM are different manifestations of a shared genetic susceptibility that leads to a spectrum of migrainous, vertiginous, and cochlear symptoms.\textsuperscript{60}

**Benign Paroxysmal Positional Vertigo**

BPPV is the most common cause of recurrent vestibular symptoms presenting to a dizziness clinic, both in unselected patients\textsuperscript{6} and in migraineurs.\textsuperscript{7} Although BPPV and migraine are separate entities, there is evidence of a linkage. Migraine is three times more common in patients with idiopathic BPPV than in those with BPPV secondary to trauma or surgical procedures,\textsuperscript{61} and is also twice as common in patients with idiopathic BPPV than in age- and sex-matched controls.\textsuperscript{62}

**Psychiatric Causes of Dizziness**

The interrelations of migraine, dizziness, and certain psychiatric disorders are complex. There are bidirectional associations of migraine with both major depression and panic disorder, with migraine being a risk factor for first-onset major depression and panic disorder, and vice versa.\textsuperscript{63,64} After palpitations, dizziness is the second most common symptom of panic attacks\textsuperscript{65} and can also be a symptom of major depression. Patients with panic and anxiety have an increased rate of vestibular test abnormalities,\textsuperscript{66} which may reflect an elevated risk of patients with vestibular disorders developing an anxiety disorder.\textsuperscript{67} Compared to other vertigo syndromes, VM patients show the highest rate of concurrent anxiety or depressive disorders.\textsuperscript{68} Because of the frequent association of dizziness, migraine, and anxiety, Furman and colleagues proposed a new syndrome designated “migraine–anxiety related dizziness” (MARD).\textsuperscript{69}

**Orthostatic Hypotension**

Patients with migraine report not only more vertigo than controls, but also significantly more “dizzy spells,” which can usually be attributed to nonvestibular causes, (32\% versus 13\%).\textsuperscript{11} However, mild vestibular dysfunction may also present with dizziness rather than vertigo. Syncope during migraine attacks occurred in 5\% of 500 unselected migraineurs.\textsuperscript{70} A large population study found an elevated frequency of syncope (46\% versus 31\%) and orthostatic intolerance (32\% versus 12\%) in migraineurs compared with controls.\textsuperscript{71} Of interest is that
orthostatic hypotension can be induced by small doses of dopamine agonists, and counteracted by dopamine antagonists, in migraineurs but not in controls, suggesting hypersensitivity to dopaminergic stimulation as the underlying mechanism.72

**Dizziness Due to Antimigraine Medication**

Dizziness occurs as a side effect of many drugs, some of which are used in the treatment of migraine. Therefore, it is useful to elicit a detailed drug history and ascertain the onset of dizziness in relation to any changes in medication, such as beta blockers and tricyclic antidepressants.

**Closing Comment**

Like migraine itself, VM is usually diagnosed by a careful history, with laboratory tests having only minor roles. However, a history of both migraine and vertigo is insufficient evidence for VM, as they may represent a concurrence by chance alone or specific vertigo syndromes that are epidemiologically associated with migraine.

**Conflicts of Interest**

The authors declare no conflicts of interest.

**References**