



## Review Article

## Vestibular Migraine: from Differential Diagnosis and Pathophysiology to Treatment Options

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### ABSTRACT

**Background:** The occurrence of migraine and vertigo is common in the general population. Migraine vertigo is challenging and has several common symptoms with inner ear pathologies like Endolymphatic Hydrops. This paper presents a review of recent findings about symptoms, test results, pathophysiology and differential diagnosis.

**Methods:** The present study is a review of 35 papers in the field of vestibular migraine. They were selected by searching the keywords vertigo, dizziness, migraine, treatment and rehabilitation in Pubmed, ScienceDirect, Scopus and Google Scholar search engines. Only human studies were included.

**Results:** Recent findings have proposed several common pathophysiologies between vestibular system and migraine including spreading depression in the basilar artery, vasospasm in the internal auditory artery, involvement of the connection with locus coeruleus, dorsal raphe nucleus, disorder of multisensory integration and channelopathy.

**Conclusion:** For the differential diagnosis of vestibular migraine, it appears that comprehensive case history as well as clinical testing and patient follow-up, are the best combination. In addition, it has been proven that vestibular rehabilitation is beneficial to patients with vestibular migraine.

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### Introduction

Both migraine and vertigo are common in the general population with prevalence of about 16% for migraine and 7% for vertigo [1, 2]. Therefore, a concurrence of both conditions can be 1.1% of the general population by chance. However, the actual comorbidity is higher, about 3.2%. Vestibular Migraine (MV) or migraine associated vertigo (MAV) affects more than 1% of the general population, about 10% of patients in dizziness clinics and at least 9% of patients in migraine clinics [1]. Generally, 30 to 50% of patients with migraine complain

about vertigo and dizziness [3]. Regarding age, MV can occur at any age although the mean age of dizziness beginning is about 40 years. Female preponderance has also been observed (prevalence ratio female to male is 5:1) [2, 4, 5]. As a result of its high prevalence, this review article will discuss important issues and findings in this field.

### Methods

The present study is a review of 35 papers in the field of vestibular migraine. They were selected by searching the keywords vertigo, dizziness, migraine, treatment and rehabilitation in Pubmed, ScienceDirect, Scopus and Google Scholar search engines. Only human studies were included.

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## 1 Overview for vestibular migraine and its differential diagnosis

### 1-1 Symptoms

Clinically, MV experience attacks of spontaneous or positional vertigo lasting between seconds to days. Other symptoms like headache, phonophobia, photophobia or auras are common [1].

In one study, patients with definite MV (dMV) were selected according to accepted diagnostic criteria after 9 years follow-up. The majority of patients (87%) had recurrent vertigo at follow-up. An increase in vertigo was recorded in 29% [6]. In another study, 42.8% of patients suffered from motion sickness [3].

It appears that spontaneous vertigo, positional vertigo and motion sickness are prevalent in MV. Spontaneous vertigo is the first symptom; other symptoms (vertigo and finally head motion intolerance) appear later. The occurrence of spontaneous vertigo was seen in 21–83% of patients, positional vertigo and dizziness in 17–65% and head motion intolerance in 31 and 77%. A high proportion of the patients show attacks of unsteadiness of stance and gait, mostly lateral or backward pulsion [7].

Some of symptoms of MV can be similar to those seen in BPPV patients including sensitivity to head motion, rolling over in bed, and generalized imbalance, particularly among the elderly [8].

Cochlear symptoms may be associated with MV [1, 6]. There is usually an increase in cochlear symptoms from 15% initially to 49% in 9 years follow up [6]. Some studies could not show any hearing loss during acute MV. However, some patients reported short-lasting aural pressure during the episode [7]. Cochlear symptoms such as hearing loss, tinnitus and aural fullness, sometimes present like Meniere's disease [3, 7].

ICHD-3 (International Classification of Headache Disorders-third edition) included four migraine equivalents in infancy and childhood. This migraine is defined as having the following symptoms: cyclic vomiting, abdominal pain, benign paroxysmal vertigo, and benign paroxysmal torticollis. Some of them may have a vestibular system involvement [3, 9].

### 1-2 Vestibular test results in vestibular migraine

Some common balance tests including caloric testing, rotary chair testing, Dix-Hallpike evaluation, oculomotor assessment, posturography, and vestibular evoked myogenic potential (VEMP) have not shown any characteristic findings in patients with MV [8].

In a study, it was shown that most patients experience internal vertigo (feeling self-rotation) (73%) more common than external vertigo (feeling environment spinning) (25%), whereas in another research, internal and external vertigo recorded similar prevalence [3].

During acute attacks, central spontaneous or positional nystagmus (PN) is common. In the symptom-free interval, vestibular testing is usually normal [1]. The most frequent finding can be PN [6].

In MV, there is a low probability for unilateral vestibular hypofunction [1]. It has been shown that patients with MV might be over-sensitive to vestibular

testing and caloric irrigation. Some MV patients vomit after a caloric stimulus, refuse to complete the caloric testing before irrigations completion, or develop an attack or exacerbation of vestibular migraine for hours or days after balance testing. Some patients with vestibular migraine seem to have significantly low total eye speeds/ TES in caloric test (below 79 deg/s) [8].

Typically, body sway is abnormal with eyes open and further increased with eyes closed, indicating vestibulospinal dysfunction [7]. Ocular motor signs, such as saccadic pursuit and gaze-evoked nystagmus are rare, indicating that the vestibulo-cerebellum is not involved in MV [7]. A 9-year follow-up study on definite MV patients showed that there was an increase in ocular motor abnormalities from 16% initially to 41% [6].

### 1-3 Migraine vs. Meniere's disease

Prosper Meniere suggested the existence of a possible link between migraine and Meniere's disease. The prevalence of migraine is significantly higher in patients with Meniere's disease [7, 10]. In addition, 45% of patients with Meniere's disease always experience migrainous symptoms during vertigo attacks. These findings may be due to a common pathophysiological mechanism between MV and Meniere's disease [3, 7].

The prevalence of Meniere's disease is 5 to 10 times lower than that of MV [11-13]. For both conditions, early and accurate diagnosis (or its exclusion) enables the correct management of patients. The long-term management of migraine requires lifestyle changes like avoiding triggers of migraine and/or prophylactic drugs, if attacks are too frequent. The long-term management of Meniere's disease involves lifestyle changes (low salt diet), medications (betahistine, steroids), and ablative therapy (e.g., intratympanic gentamicin) [12].

There are several challenges in differentiating MV from MD. MD patients typically have aural pressure that may sound like headache and patients with migraine may also experience tinnitus and hearing loss. Phonophobia is a symptom in MV but can also be seen in MD. MV can cause vertigo, dizziness, or other imbalances which last from 5 min to 72 h, which is similar to MD patients. Patients with definite MD must have low-frequency to middle-frequency hearing loss. However, hearing loss is not part of the probable MD. Moreover, hearing loss (fluctuating or progressive) can occur in MV (up to 25% of patients with migraine) [11].

A study revealed that cervical and ocular VEMP might help differentiating MV and MD patients but not in all cases. VEMP is more commonly involved in MD [14, 15]. It appears that caloric testing and motion-sensitivity questionnaires are unsuccessful in the differential diagnosis of MV and MD. This may be due to the common origin of these two disorders. One common origin for MV and MD might be chronic low blood supply of the end-organ [16].

In MV, hearing loss may be present but is typically bilateral. In MD, however, bilateral hearing loss is rare at its onset. Over an average period of 9 years, cochlear symptoms of tinnitus, aural fullness, and hearing loss

become more prevalent in MV patients. However, hearing levels in MD patients decrease to a mean of 50–60 dB (decibels) in 5–10 years. After several years, the majority of MV patients still suffer from vertigo, while MD patients experience fewer vertigo episodes [11].

## 2 Vestibular migraine pathophysiology and treatment

### 2-1 Pathophysiology

The pathophysiology of MV is still unknown. It is not even known whether the origin is in the central or peripheral vestibular system [7].

Various hypotheses have been proposed for MV. The migraine aura is likely to be the clinical equivalent of a spreading depression. Vertigo is the most common manifestation of an aura in basilar artery migraine [7, 17]. The spreading of depression in the brainstem might be responsible for the short-lasting episodes of MV. Vasospasm of the internal auditory artery could explain peripheral vestibular and auditory symptoms in migraine [7, 18].

During acute migraine attacks, functional imaging studies have shown activation of brainstem regions in projection to the locus coeruleus and the dorsal raphe nucleus. It is proposed that these neural structures are involved in the migraine attacks [7]. As the vestibular nuclei receives noradrenergic projections from the locus coeruleus and serotonergic input from the dorsal raphe nucleus, it is suggested that activation of these structures in migraine also affects central vestibular processing [19, 20]. It has been proposed that there are ion channels defects in migraine [21, 22]. A channelopathy could result to central and peripheral vestibular dysfunction [7, 23].

There are reciprocal connections between vestibular nuclei and trigeminal nuclei which can result in vestibular symptoms in migraine [11, 24]. The sensitization of thalamic pathways may be the cause of motion-sickness susceptibility in MV patients [11, 23, 25]. Studies have shown that rotational and caloric testing trigger migraines more in those with a history of migraine than in those without [26]. Caloric stimulation has been reported to trigger migraine even in those without migraine history. This shows that the peripheral vestibular system may have the ability to modulate migraines [11].

In conclusion, MV is a heterogeneous vestibular disorder and various pathophysiological mechanisms may be involved [7].

### 2-2 Definite and probable vestibular migraine

The diagnosis of definite MV is based on the following criteria: (i) episodic vestibular symptoms with at least moderate severity (rotational vertigo, other illusionary self or object motion, positional vertigo, head motion intolerance); (ii) migraine according to the IHS (International Headache Society, 2004); (iii) one of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras; and (iv) other causes ruled out by appropriate investigations [2, 7].

The criteria for probable MV are: (i) episodic vestibular symptoms of at least moderate severity; (ii) at least one

of the following: migraine according to the criteria of the IHS; migrainous symptoms during vertigo; migraine-specific precipitants of vertigo, e.g. specific foods, sleep irregularities and hormonal changes; response to anti-migraine drugs; and (iii) other causes ruled out by appropriate investigations [2, 7].

Recently, a joint committee between the IHS and the Bárány Society (International Society for Neuro-Otology) established the following diagnostic criteria for both the definite and probable vestibular migraine (MV) [1, 27]. Definite MV: 1- At least five episodes with vestibular symptoms of a moderate or severe intensity, lasting between 5 min to 72 h. 2- Current or previous history of migraine with or without aura according to the ICHD. 3-One or more migraine features in at least 50% of the vestibular episodes: Headache with at least two of the following characteristics: one-sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity, photophobia and phonophobia, visual aura; 3- Not better accounted for by another vestibular or ICHD diagnosis. In order to diagnose probable MV, only one of criteria 2 or 3 must be observed [3, 28, 29].

### 2-3 Treatment and rehabilitation

The prophylactic medication for migraine improves dizziness, motion sickness and headaches [30]. However, vestibular rehabilitation for patients with MV might be beneficial. The vestibular migraine group generally shows poorer subjective performance (e.g. in dizziness handicap index (DHI), activities specific balance confidence (ABC)), at the onset of therapy. It appears that patients with MV show the same degree of subjective performance improvement as non-migrainous patients regardless of medication treatment [31].

In addition to subjective performance improvement, studies have shown that vestibular rehabilitation is effective for objective balance improvements like gait index as well as “timed up and go” [32]. In fact, MV patients show improvements in physical performance measures (e.g. dynamic gait index (DGI) and computerized dynamic posturography (CDP)) and self-perceived abilities after vestibular rehabilitation [33].

Reports have shown that 92% of patients who experienced motion sensitivity or postural instability reported subjective improvements when treated with a combination of vestibular rehabilitation and pharmacological therapy [34]. It appears that the medication allows the subjects to be more tolerant of the motions in the vestibular rehabilitation program [34, 35].

## Conclusion

It appears that comprehensive case history in addition to clinical testing and patient follow-up are the best combination for vestibular migraine differential diagnosis. In addition, vestibular rehabilitation has been proven to be beneficial in patients with vestibular migraine.

**Conflict of interest:** None declared.

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