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REVIEW

# Vestibular migraine: diagnosis challenges and need for targeted treatment

## Migrânea vestibular: desafios diagnósticos e a necessidade de tratamentos específicos

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**ABSTRACT**

Approximately 1% of the general population suffers from vestibular migraine. Despite the recently published diagnostic criteria, it is still underdiagnosed condition. The exact neural mechanisms of vestibular migraine are still unclear, but the variability of symptoms and clinical findings both during and between attacks suggests an important interaction between trigeminal and vestibular systems. Vestibular migraine often begins several years after typical migraine and has a variable clinical presentation. In vestibular migraine patients, the neurological and neurotological examination is mostly normal and the diagnosis will be based in the patient clinical history. Treatment trials that specialize on vestibular migraine are scarce and therapeutic recommendations are based on migraine guidelines. Controlled studies on the efficacy of pharmacologic interventions in the treatment of vestibular migraine should be performed.

**Keywords:** migraine; vestibular migraine; dizziness; vertigo.

**RESUMO**

Cerca de 1% da população apresentam o diagnóstico de migrânea vestibular. Apesar dos critérios diagnósticos terem sido publicados recentemente, ainda é uma condição subdiagnosticada. Os mecanismos neurais exatos da migrânea vestibular ainda não estão claros, mas a variabilidade dos sintomas e achados clínicos durante e entre os ataques sugere uma interação importante entre os sistemas trigeminal e vestibular. A migrânea vestibular geralmente começa alguns anos após a migrânea típica e tem apresentação clínica variável. Em pacientes com migrânea vestibular, o exame neurológico e otoneurológico são geralmente normais e o diagnóstico é baseado na história clínica do paciente. Estudos sobre tratamento da migrânea vestibular são escassos e recomendações terapêuticas são baseadas em diretrizes do tratamento da migrânea. Estudos controlados sobre a eficácia das intervenções farmacológicas para o tratamento da migrânea vestibular devem ser realizados.

**Palavras-chave:** migrânea; migrânea vestibular; tontura; vertigem.

Dizziness is one of the most common complaints in clinical practice, affecting 20-30% of the general population[1] and is often reported by patients with migraine[2]. The prevalence of migraine is higher in patients with dizziness[2], and benign recurrent vertigo is the most prevalent type of dizziness in this population[3,4]. The relationship between dizziness and migraine was first described by the ancient Greek physician Aretaeus of Cappadocia in 131 BC[5]. In 1873, Edward Liveing observed an association between migraine and dizziness in some of his patients[6]. However, detailed observation of this association was initiated only in the last 30 years. Since the first studies of Kayan et al. about the vestibular manifestations of migraine[7] the number of articles addressing the relationship between vertigo and migraine has grown exponentially in the last 25 years[8,9]. Different terms have been used to designate the relationship of vertigo and migraine including migraine-associated vertigo, migraine-associated dizziness, migraine-related vestibulopathy, migrainous vertigo, benign recurrent vertigo. More recently the term vestibular migraine (VM) was defended as a condition that covers the vestibular manifestations that may occur in migraine, avoiding confusion with nonvestibular dizziness that may also be associated with migraine[10].

## EPIDEMIOLOGY

Despite vestibular migraine is one of the most common diagnoses in dizziness units (the second most common cause of recurrent vertigo after benign paroxysmal positional vertigo (BPPV)) accounting for 6-9% of all diagnoses, it is still underdiagnosed[2,9]. In a study conducted by a specialized clinic in Switzerland, dizziness was diagnosed as vestibular

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migraine in 20.2% of patients, although VM was previously suspected by the requesting physicians in only 1.8%. The diagnosis of "uncertain dizziness" accounted for almost 60% of patients[11]. In another study in Germany, with 33 patients, two-thirds of patients diagnosed with VM had consulted a doctor because of vertigo, but only 20% were diagnosed with VM. The remaining patients were diagnosed with other diseases such as anemia, diabetes, hypovolemia[12]. Vestibular migraine has a year prevalence of 0.89% and represents about 10% of patients treated for either migraine and dizziness[13]. Hsu and colleagues reported a year prevalence of vestibular migraine in women aged 40-54 years of 5%[14]. Forty percent of patients with vestibular migraine reported missing work because of their symptoms, showing the impact of the disease on daily life[12]. Vestibular migraine can occur at any age, but the average age of onset of dizziness in migraine is about 40, and a first attack late age in 72 years has been reported. It has a female predominance, the female and male ratio of 5: 1[2,12,15]. In older patients, particularly post-menopausal women, typical migraine attacks are sometimes replaced by isolated episodes of vertigo, dizziness or transient feeling of imbalance[16]. In a population-based study, the prevalence of recurrent vertigo probably related to migraine was estimated at 2.8% in children with 6-12 years[17]. Vestibular migraine is diagnosed more often in children than adults (35% vs 6%)[8]. The most common cause of vertigo in children is benign paroxysmal vertigo, which has a strong association with a family history of migraine and may predict the development of typical migraine[18].

## Pathophysiology

The exact neural mechanisms of vestibular migraine are still unclear. The variability of symptoms and clinical findings both during and between attacks suggests that migraine interacts with the vestibular system at various levels[19].

The vestibular nuclei receive noradrenergic inputs from the locus ceruleus and serotoninergic inputs from the dorsal raphe nucleus[20,21]. Therefore, activation of these nuclei during migraine attacks may give rise to vestibular symptoms. Since the caudalis trigeminal nucleus also has reciprocal connections with the vestibular nuclei, and neurogenic inflammation of the trigeminal system is believed to be a mechanism of migraine, trigeminal activation may provoke vestibular symptoms during migraine attacks. Trigeminal activation by painful electrical stimulation of the forehead produced spontaneous nystagmus in migraine patients, but not in controls, indicating that those with migraine have a lowered threshold for crosstalk between these neighboring brainstem structures[22]. Shin and colleagues studied 2 patients with vestibular migraine who underwent FDG-PET images. During attacks of vestibular migraine, the increased metabolism of the temporo-parieto-insular areas and bilateral thalami indicated activation of the vestibulothalamo-vestibulocortical pathway, and the decreased metabolism in the occipital cortex may represent reciprocal inhibition between the visual and vestibular systems[23]. Another image study comparing vestibular migraine patients with migraine without aura (MwoA) and healthy controls (HC) showed that patients with VM showed a significantly increased left mediodorsal thalamic activation in response to an ipsilateral vestibular stimulation, relative to both HC and patients with MwoA. Furthermore, the magnitude of left thalamic activation was uniquely correlated with frequency of migraine attacks in patients with VM[24]. The role of mediodorsal thalamus in VM pathophysiology could reflect the involvement of a dysfunctional vestibulo-thalamocortical network, which overlaps with the migraine circuit[19].

Cortical spreading depression (CSD) in both multisensory vestibular areas of the cortex and brainstem was used as an explanation for 'aura'-like dizziness/vertigo attacks with consecutive headache. However, an isolated CSD, which is limited to the brain stem without causing any other symptoms, is not very likely[25].

Some authors suggested a genetic inheritance. A linkage analysis in a four-generation family with 10 affected individuals mapped the locus for vestibular migraine to chromosome 5q35[26]. In a larger study, familial vestibular migraine was found to be genetically heterogeneous with a subgroup linking to chromosome 22q12[27].

The most current pathophysiologic model of vestibular migraine is summarized in Figure.

## Diagnostic criteria

The International Headache Society (IHS) and the Bárány-Society (International Society for Neuro-Otology) created a consensus document with diagnostic criteria for vestibular migraine, which was added in the appendix of the new ICHD-3 beta version of the International Headache Classification[28] (Table 1).

## Clinical presentation

Vestibular migraine often begins several years after typical migraine[2,15]. In a previous study, migraine manifested before VM in 74% of participants and in more than half of these (52%) migraine was preceded by VM by more than 5 years, and in 26% even by more than 10 years. The vast majority of patients (85%) had experienced both VM and migraine during the last 12 months[12]. Vestibular migraine is more common in patients without aura than in patients with aura[29]. The most frequent vestibular symptoms associated with migraine are spontaneous vertigo in 67% followed by positional vertigo in 24% of these patients[12]. Other commonly described symptoms are: imbalance, head motion intolerance, visual vertigo and non-vertiginous dizziness such as lightheadness or "boat like" rocking[13,30].

The duration of attacks can vary from a few seconds (10% of patients) to some minutes (30% of patients), some

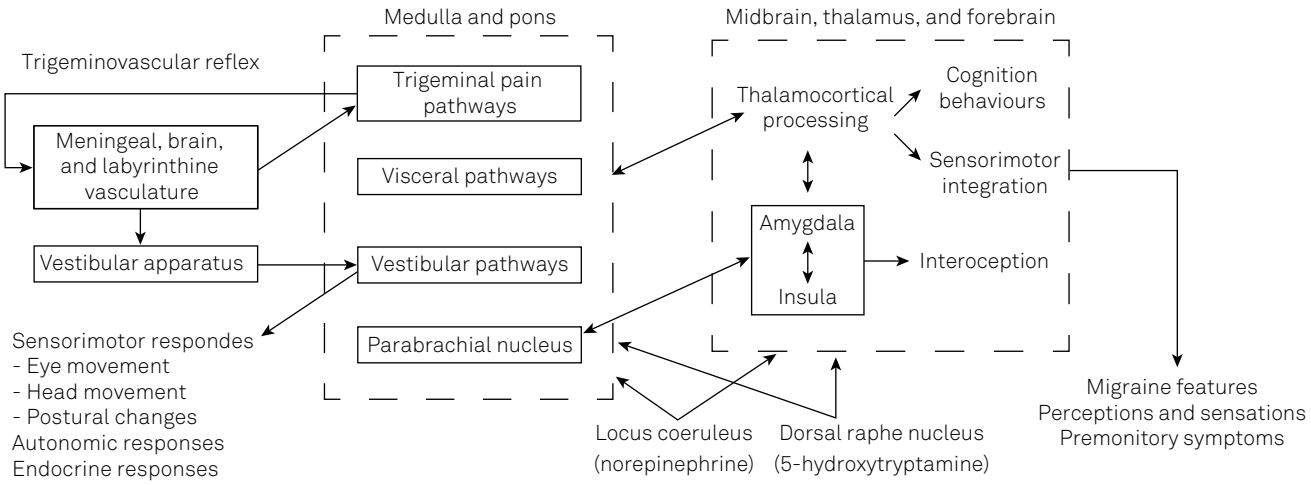


**Figure.** Summary of patophysiologic model of vestibular migraine.

hours (30% of patients) and even up to a few days (30% of patients)[25]. Only 10-30% of patients described a typical vestibular aura. Vestibular symptoms can occur before, during or after the migraine attack and in 30% of the patients the two symptoms never occurred together[2,31]. Some patients reported vertigo as the most disabling symptom and only report a slight felling of pressure in the head replacing a typical migraine headache[32,33].

Auditory symptoms, including hearing loss, tinnitus, and aural pressure have been reported in up to 38% patients with vestibular migraine. Hearing loss is usually mild and transient, without or with only minor progression in the course of the disease[2,34,35].

Radtke et al. reassessed 61 patients with definitive VM according to validated diagnostic criteria after a follow-up time of 9 years and their findings are shown on Table 2[36].

Episodes of vestibular migraine can be brought about by the same triggers as those for migraine headache, including menstruation, irregular sleep, stress, physical exertion, dehydration, food and drinks, and intense sensory stimulation, mostly movement [29].

In most patients, the neurologic and otologic examination is normal during the interictal phase[32]. About 10 to 30% of patients with vestibular migraine have unilateral hypoexcitability to caloric stimulation and 10% have directional preponderance of nystagmus responses. Such findings, however, are not specific for vestibular migraine, because they can be found also in migraine patients without vestibular symptoms and in many other vestibular syndromes[37]. In one study, patients with vestibular migraine became nauseous after caloric testing four times more often than migraine patients and patients with other vestibular disorders[38]. A neuro-otologic study of 20 patients during the acute phase of vestibular migraine showed pathological nystagmus in 14 patients, mostly central spontaneous or positional nystagmus. Three patients had a peripheral spontaneous nystagmus and a unilateral deficit of the horizontal

**Table 1.** Vestibular migraine: diagnostic criteria.

| |
|---|
| A. At least five episodes fulfilling criteria C and D. |
| B. A current or past history of 1.1 Migraine without aura or 1.2 Migraine with aura. |
| C. Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours. |
| D. At least 50% of episodes are associated with at least one of the following three migrainous features: |
| 1. headache with at least two of the following four characteristics: |
| a) unilateral location; |
| b) pulsating quality; |
| c) moderate or severe intensity; |
| d) aggravation by routine physical activity. |
| 2. photophobia and phonophobia; |
| 3. visual aura. |
| E. Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder. |

ICHD-3: International Classification of Headach Disorders 3rd edition (beta version).

Table 2. Clinical characteristics of vertigo and concomitant symptoms in 61 patients with definite vestibular migraine.[36]

| | Initial presentation (%) | Follow up (%) |
|---|---|---|
| Type of vertigo | | |
| Spontaneous | 85 | 95 |
| Spinning | 75 | 82 |
| Positional | 39 | 80 |
| Head-Motion-Induced | 61 | 84 |
| Unsteadiness | 66 | 90 |
| Duration of vertigo attacks | | |
| < 1 min | 31 | 75 |
| 1-5 min | 30 | 56 |
| 5-60 min | 34 | 64 |
| < 24h | 49 | 74 |
| > 24h | 52 | 69 |
| Cochlear symptoms during vertigo spells | | |
| Tinnitus | 10 | 33 |
| Aural fullness | 13 | 26 |
| Hearing loss | 12 | 26 |
| Migraine symptoms during vertigo attacks | | |
| Headache | 75 | 90 |
| Photophobia | 59 | 80 |
| Phonophobia | 54 | 77 |
| Aura | 18 | 44 |

vestibulo-ocular reflex. Imbalance was observed in all patients except one[39]. Since there are no specific abnormalities in vestibular migraine, in general practice the diagnosis will be based in the patient clinical history.

## Differential diagnosis

The main differential diagnosis of VM is Ménière's disease (MD). An association between MD and migraine was already suggested by Prosper Ménière himself in 1861[40]. Some studies have ratified a higher prevalence of migraine in patients with MD[41,42], almost 30% of patients with Ménière syndrome may also have VM[41]. The overlap or coexistence of both diseases may make the patient's diagnosis difficult and the most reliable distinguishing feature is the low-frequency hearing loss in MD[42]. In general, MD distinguished from vestibular migraine by symptoms of ear fullness or pain preceding the attack or at attack onset, and accompanying tinnitus and/or hearing loss during the episode. These symptoms are not very prominent in vestibular migraine[41,42].

Endolymphatic hydrops is the primary pathological entity seen in Ménière's disease[43]. Recent progress has made it possible to reliably visualize endolymphatic hydrops in living humans by employing a Locally Enhanced Inner ear MRI technique. Gurkov et al. investigated the occurrence of endolymphatic hydrops (EH) in patients with VM and auditory symptoms. Nineteen patients with definite or probable VM and auditory symptoms were examined by locally enhanced inner ear MR imaging. Of the 19 included patients, four patients (21 %) demonstrated evidence of cochlear and vestibular endolymphatic hydrops[44]. In contrast, Nakada et al. investigated endolymphatic hydrops in seven patients with definitive VM and 7 patients with MD. EH was not found in VM patients but all patients with MD showed significant EH[45].

BPPV is the most common cause of recurrent vertigo[46]. Episodic vertigo related to migraine occurs several times per year or month with a duration of some hours up to few days, whereas BPPV leads to episodes of short lasting vertigo typically lasting weeks to months without therapy[47]. During acute attacks of vertigo, the analysis of the positional nystagmus usually permits differentiation of positional VM from BPPV[47].

Transient ischemia within the vertebrobasilar system is a common cause of episodic vertigo in older people. It is abrupt in onset, usually lasts several minutes, and is frequently associated with nausea

and vomiting[48]. Baloh studied 42 patients with vertigo in a neurotology service with the diagnosis of vertebrobasilar insufficiency and found that 62% had at least one episode of isolated vertigo and in 19% the transient ischemic attack began with an isolated episode of vertigo[49]. Therefore, it is reasonable to investigate older patients with sudden onset of unilateral deafness and vertigo, particularly if there is a prior history of TIA, stroke, or known atherosclerotic vascular disease.

Basilar migraine requires at least two aura symptoms, which are assignable to the vertebrobasilar territory, lasting between 5 and 60 minutes and followed by a typical migraine headache[25]. Less than 10% of the patients with VM meet the criteria for basilar type migraine[2].

## Treatment

Current VM treatment recommendations are most based on expert opinions rather than on solid data from randomized controlled trials. Most therapeutic approaches are based on case reports, retrospective cohort studies and open labels trials. In general, the scientific literature suggests that drugs efficacious for prophylaxis of migraine are also appropriate for prophylaxis of vestibular migraine[49].

Neuhauser and colleagues suggested a benefit of zolmitriptan 2.5 mg in 38% of patients with vestibular migraine whereas in the placebo group a positive effect was seen in 22%. Unfortunately, the study had some limitations such as the large interval of confidence and the small number of patients recruited with only 17 reported attacks[50].

In another study, 48 patients with VM diagnose received flunarizine 10 mg daily along with betahistine and paracetamol during the migraine episodes and compared to the control group who received only the symptomatic treatment during the episodes. The frequency of vertiginous episodes showed a significant difference between the two groups, although the headache frequency and intensity did not improve in a significant degree[51].

One retrospective single center open label investigation compared the effect of cinnarizine on VM and migraine with brainstem aura patients. Cinnarizine reduced the headache variables ( frequency, duration and intensity) in both types of migraine with greater outcomes in the vestibular migraine group. Vertigo was also decreased in both groups with significant reduction in the VM group in the first month[52].

One retrospective, open-label study investigated the efficacy of 100 mg lamotrigine in 19 patients (6 male, 13 female) with vestibular migraine over 3–4months. The average vertigo frequency per month was reduced from 18.1 to 5.4, headache frequency dropped from 8.7 to 4.4, but did not reach statistical significance. Therefore, lamotrigine appears to mainly act on vestibular symptoms and only to a lesser extent on headaches[53].

A large retrospective cohort compared 100 VM patients with and without prophylactic migraine treatment. All patients on prophylactic treatment showed a decrease of duration, intensity, and frequency of episodic vertigo as well as its associated features ($p < 0.01$). Medications used were beta-blocker, 49 patients, metoprolol (median dose 150 mg) or propranolol (median dose 160 mg), valproic acid, 6 patients (median dose 600 mg), topiramate, 6 patients (median dose 50 mg), butterbur extract, 4 patients (median dose 50 mg), lamotrigine, 3 patients (median dose 75 mg), amitriptyline 2 patients (75 to 100 mg), flunarizine, 1 patient (5 mg), and magnesium 3 patients (median dose 400 mg). The group without prophylactic therapy showed a reduction of vertigo intensity only[54]. An ongoing trial will test the efficacy of metoprolol in vestibular migraine (Prophylactic treatment of vestibular migraine with metoprolol - PROVEMIG trial)[55].

Behavioral modifications can be tried. One retrospective study showed that 14% of 38 patients enrolled reported an improvement in symptoms after caffeine cessation[56].

Vestibular rehabilitation exercises were described to be beneficial in patients with vestibular migraine in addition to medical treatment or as stand-alone treatment option[57]. Improvement in physical performance measures and self-perceived abilities after a 4-month vestibular physical therapy was observed in 14 patients with the diagnosis of migraine related vertigo[58]. In another cohort study, patients with vestibular disorders with or without a history of migraine demonstrated improvements after nine weeks in both subjective and objective measures of balance after 15 minutes daily physical therapy[59].

## Clinical implications

Vestibular migraine is quite prevalent but still underdiagnosed, and the impact on daily life activities and well-being is considerable. Treatment is not well established and therapeutic recommendations are based on migraine guidelin. The need of specific treatment is urgent. The migraine patient should be asked about vestibular symptoms by routine and controlled studies on the efficacy of pharmacologic interventions in the treatment of vestibular migraine should be performed.

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