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MINI REVIEW

Vestibular Migraine, an Underdiagnosed Cause of Vertigo: Practical Review of the Literature

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Abstract

Vestibular migraine is the most common cause of central episodic vertigo. It has been postulated that it shares pathophysiological mechanisms with classic migraine. The diagnosis of vestibular migraine is clinical and is based on the criteria established by the Bárány Society and the International Headache Society. A complete history is essential to guide pharmacological and non-pharmacological treatment in the therapeutic approach to patients with vestibular migraine.

This article provides a practical review of the current literature on the diagnostic and therapeutic approach to vestibular migraine, illustrating the available scientific evidence covering epidemiology, pathophysiology, diagnosis and treatment.

Keywords: Vestibular Migraine, Migraine, Vertigo, Dizziness.

1. Introduction

Migraine and vertigo are prevalent conditions in general population. Migraine affects 14-15% of the global population and vertigo 15-20% of adults annually. There is a link between them: vertigo is three times more frequent in patients with migraine, especially in migraine with aura (present in 30-50% of patients). Furthermore, vertigo coexists with other migraine symptoms in almost 50% of acute classical migraine attacks and may be part of the aura in migraine with brainstem aura, previously known as basilar migraine. Finally, the main disorder that connects vertigo with classical migraine is vestibular migraine (VM), previously known as migrainous vertigo or migraine-associated vertigo (1).

VM is one of the most common neurological disorders causing vertigo and dizziness. Despite its high prevalence, it remains an underdiagnosed disease (1,2). This article provides a practical review of the current literature on the diagnostic and therapeutic approach to VM, illustrating the available scientific evidence about epidemiology, pathophysiology, diagnosis and treatment.

2. Epidemiology

VM affects approximately 1 to 3% of the general population and up to 30% of patients treated in specialized vertigo or headache centers (1). Chia et al. evaluated 208 patients with benign recurrent vertigo in a neuro-otology clinic and they found that 87% met ICHD-3 criteria for migraine (62% with aura) (3). In fact, vestibular symptoms are twice as likely to occur in patients with migraine with aura compared to individuals without aura. Importantly, the presence of migraine is extremely high in patients with unclassified recurrent vertigo (60 to 80%) (1,3).

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Furthermore, according to Martinez et al. almost 40% of patients with VM meet the criteria for chronic migraine and 25% have excessive use of analgesics (1,4).

VM is more prevalent in women, with a ratio of 1.5 to 5.1. The average age of onset is between 38 and 50 years. Migraine usually precedes vestibular symptoms. The average delay between headache and vertigo is 8 to 14 years (5). In postmenopausal women, typical acute migraine attacks may be replaced by vestibular symptoms (1). Most patients with VM (50 to 70%) have a family history of migraine (6).

Benign positional vertigo of childhood is a migraine equivalent and the majority will develop migraine in adulthood. Age younger than 40 years, female sex, mood disorders, and previous head trauma were associated with significantly higher odds of developing VM (1).

Regarding the lack of timely diagnosis, one study showed that only 20% of VM patients were correctly diagnosed after visiting a physician (8). One study showed that 19% of otolaryngologists and 14.5% of neurologists had never treated a VM patient before (9).

3. Pathophysiology

Understanding the pathophysiology of VM remains a challenge. The wide range of clinical and laboratory findings that overlap between migraine and vestibular disorders suggests a diverse pathophysiological basis involving multiple mechanisms (10).

Currently, the trigeminovascular system, neurogenic inflammation, and altered vestibular-nociceptive processing are known to be the main protagonists (10). Traditionally, activation of the trigeminovascular system has been associated with the development of acute migraine attacks. However, excitation of the trigeminal nucleus and the release of vasoactive neuropeptides (substance P, calcitonin gene-related peptide (CGRP), among others) also contribute to the appearance of vestibular symptoms because the vestibular nuclei of the brain stem overexpress CGRP receptors, which produces alterations in the processing of vestibular-nociceptive information at relay points (insula, thalamus and somatosensory cortex) (11).

Furthermore, it has been suggested that the cortical depression wave described by Leão may occur outside the cerebral cortex, for example in the vestibular nuclei. Hence the hypothesis that VM is a "brain stem aura". However, VM and migraine with brain stem aura (MBSA) are different entities (12).

On the other hand, it is postulated that vestibular symptoms can be attributed to a hereditary channelopathy in some patients with migraine (13). For example, one of the cardinal symptoms in the diagnosis of episodic ataxia type 2, a rare autosomal dominant disorder caused by malfunction of calcium channel expressed in purkinje cell, is the presence of episodic vertigo and up to 50% of patients with this diagnosis have concomitant migraine (14) sharing genes with familial hemiplegic migraine.

These mechanisms reveal a complex interaction between genetics, neurochemistry and sensory processes underlying VM, influencing multisensory integration at cortical and subcortical levels.

4. Diagnosis

The diagnosis of this entity is clinical, for which the Bárány Society and the International Headache Society, in their ICHD-3, have jointly developed clinical criteria (15,16) (Table 1).

Table 1. *VM criteria*

A. At least five episodes of vestibular symptoms* of moderate or severe intensity**, and a duration between 5 minutes and 72 hours
B. One or more migraine characteristics with at least 50% of vestibular episodes, including: a) Headache with at least two of the following characteristics (unilateral, pulsating, moderate or severe intensity, worsening with usual physical activity) b) Photophobia and phonophobia c) Visual aura
C. Current or past history of migraine (with or without aura)
D. Symptoms are not better explained with another diagnosis

**Spontaneous vertigo, positional vertigo, visually induced vertigo, head motion-induced vertigo, or non-vertigo-induced dizziness induced by head motion with nausea.*

***Vestibular symptoms are considered moderate to severe if they interfere with daily activities.*

The Bárány Society proposes the diagnosis of probable VM when patient has migraine-like features during vestibular episodes or a current or past history of migraine, but not both (16) (Table 2). In these cases, diagnostic aids are required to evaluate alternative causes of the symptoms.

Table 2. *Probable VM criteria*

A. At least five episodes of moderate or severe vestibular symptoms, duration between 5 minutes and 72 hours
B. Only one of criteria B and C for vestibular migraine is met (migraine history or migraine characteristics during the episode)
C. Symptoms are not better explained with another diagnosis

5. Differential Diagnoses

The differential diagnosis of VM includes other causes of episodic vestibular syndrome such as Ménière's disease (MD) and benign paroxysmal positional vertigo (BPPV), as well as MBSA, vertebrobasilar ischemia, and less common causes such as episodic ataxia, superior semicircular canal dehiscence syndrome, persistent perceptual postural dizziness, vestibular paroxysmia, and recurrent vestibulopathy.

In BPPV, recurrent episodes of vertigo last less than 1 minute and are triggered by head movements. There is a reciprocal association and notable overlap between VM and MD. In the first year after symptom onset, differentiation between the two entities can be challenging, as MD may present with vestibular symptoms only (3,7) and many patients with MD present with BPPV as a complication of inner ear injury.

VM is distinct from MBSA, which usually presents with at least two symptoms, including vertigo, dysarthria, tinnitus, hearing loss, diplopia, ataxia, and/or decreased level of consciousness. Vertigo is a common aura symptom, but not the only symptom. In addition, most patients with MBSA develop headache within 5 to 60 minutes after the onset of the aura (17).

In vertebrobasilar ischemia, vertigo is a common presenting symptom, presenting in approximately 50% of cases. However, isolated vertigo is uncommon in a transient ischemic attack (TIA) or stroke (18,19). Depending on the anatomical location, it is usually accompanied by manifestations such as dysarthria, diplopia, dysphagia, weakness, ataxia, Horner syndrome, among others (20,21).

6. Diagnostic Evaluation

There are no specific diagnostic tools for VM. Studies are indicated to exclude alternative causes of episodic vertigo in patients with recent onset symptoms who do not meet the diagnostic criteria for VM.

These tests would be justified in patients with atypical presentation: brief (seconds or a few minutes) or prolonged (more than 72 hours) vestibular symptoms, altered consciousness during the episode, auditory symptoms (sudden, fluctuating or progressive hearing loss), altered neurological examination or systemic manifestations (fever, skin lesions). Its use is also recommended in those who do not respond adequately to treatment for VM.

6.1 Neuroimaging

Neuroimaging is suggested in cases of recent acute vestibular episodes to evaluate the presence of a stroke, particularly those that are accompanied by alterations in the neurological examination or in patients with cardiovascular risk factors. Although widely available computed tomography (CT) of the brain has bone artifact. Magnetic resonance imaging (MRI) of the brain is beter for evaluating the posterior fossa. Vascular imaging with CT angiography or cranial or cervical MRI is performed to identify vascular stenosis or occlusion as a cause of symptoms. In patients with persistent or chronic acute vestibular symptoms, contrast-enhanced brain MRI that includes high-resolution 3D protocols with submillimeter slices through the internal auditory canal is recommended to evaluate possible structural causes such as posterior fossa tumors.

6.2 Vestibular Function Tests

Vestibular laboratory tests are generally not necessary to diagnose VM and often show only nonspecific abnormalities (22). However, they may be useful in selected cases to detect significant central or peripheral vestibular dysfunction in the presence of an alternative diagnosis.

Various vestibular function tests are used to evaluate patients with VM, including video head impulse testing (vHIT), caloric testing, cervical or ocular vestibular evoked myogenic potentials (VEMP), and stabilometric studies including sensory organization testing (SOT) (23). Studies have indicated that abnormal vHIT and caloric testing results, indicating semicircular canal dysfunction, are associated with prolonged medication requirements and poor prognosis in patients with VM (23). Although peripheral vestibular abnormalities are common in patients with VM, the majority have normal audiovestibular testing results, including symmetric VEMPs and age-consistent audiometry (22). Audiometry is recommended in patients who experience prominent or monaural auditory symptoms during episodes, as well as in those whose symptoms

persist after resolution of episodes, in order to evaluate the presence of MS or superior semicircular canal dehiscence syndrome (24).

7. Treatment

Treatment options are primarily based on strategies for migraine management due to the limited number of high-quality randomized controlled trials specific to VM (25,26). Management typically includes a three-step approach: lifestyle modifications, treatment of acute attacks, and long-term prophylaxis (27).

7.1 Lifestyle Modifications

While evidence is limited, there may be a role for lifestyle modifications on the outcome of VM, including regular exercise, quality sleep, avoidance of fasting and potential dietary triggers (28,29). One study found significant improvement in vertigo and headache episodes after implementing lifestyle changes, and better sleep was particularly associated with symptom reduction (30).

7.2 Acute Treatment

Acute treatment is usually reserved for those patients with prolonged (more than 30 minutes) or severe symptoms. Medications are generally not helpful for episodes that resolve quickly. The choice of treatment is based on the patient's individual symptoms, comorbidities, and risk of adverse effects. There is insufficient evidence to recommend any specific pharmacological therapy for the termination of acute attacks of vertigo in VM (28). The evidence for interventions used to treat acute attacks of VM is very sparse (31).

Traditionally, and extrapolating from the evidence for the management of acute vertigo, treatment is offered with drugs such as antihistamines, antiemetics or benzodiazepines. A meta-analysis of 17 clinical trials with 1586 patients evaluated the efficacy of antihistamines and benzodiazepines for acute vertigo, finding that single-dose antihistamines provided greater relief of vertigo at two hours than single-dose benzodiazepines, but neither benzodiazepines nor antihistamines used daily were superior to placebo at one week or one month (32). Although there is no specific evidence for these agents in VM, it is important to avoid taking these medications more than 10 days per month, because regular use may cause sensitization and withdrawal (28).

Although there is little evidence, triptans could be considered in patients who do not respond to initial acute treatment options and those who experience headache during vertigo attacks. Only two randomized controlled trials provide evidence for the use of rizatriptan and zolmitriptan in acute attacks of VM. Both studies suggest that triptans may not make a significant difference to the proportion of people whose vertigo improves up to two hours after taking the medication. However, the certainty of this evidence was very low. Only one of the studies assessed serious adverse events, and no events were observed in either group. Further research is required to draw definitive conclusions (31).

7.3 Prophylactic Treatment

Indications for preventive therapy should be similar to those for other forms of migraine, considering the frequency, duration, and disabling nature of attacks. The American Headache Society suggests preventive medication in patients with at least three to six migraine episodes per month, depending on severity. Although one may choose to start prevention at a lower threshold based on patient need. Titration of medication is generally recommended, and in case of lack of response to therapeutic doses, some experts suggest polytherapy (33).

Main preventive treatment options include flunarizine, propranolol, tricyclic antidepressants, sodium valproate, low-dose topiramate, and venlafaxine (28). A meta-analysis of preventive treatment for VM was published in 2021, but the authors were unable to establish a defined treatment strategy due to low quality of evidence and heterogeneity of study design and outcome reporting (34).

7.3.1 Beta-Blockers

Propranolol has been shown to significantly improve VM symptoms, reducing attack frequency, severity, and disability scores while improving quality of life (35). A meta-analysis determined that propranolol was the most effective first-line treatment, with 60% of patients achieving complete symptom control (36).

The PROVEMIG trial (Prophylactic Treatment of Vestibular Migraine with Metoprolol), the first multicenter, double-blind RCT in the treatment of VM, sought to demonstrate that metoprolol succinate is superior to placebo in preventing VM, but had to be stopped due to low accrual of participants (37).

Beta-blockers were generally well tolerated, although in two studies approximately 12% of patients discontinued the medication (37,38). They should be considered in patients who present with hypertension in addition to VM and caution should be used in patients with asthma.

7.3.2 Anti-Seizure Medications

Sodium valproate has only been examined in multi-arm studies in VM. One study found that low-dose sodium valproate decreased headache frequency and vestibular symptoms in patients with migraine, although it did not significantly affect electronystagmography findings (39). A comparative trial demonstrated that sodium valproate was effective in reducing the frequency of vertigo attacks and improving the Dizziness Handicap Inventory (DHI) score, although it was less effective than venlafaxine in decreasing vertigo severity (40).

Studies have shown that topiramate reduces the intensity, duration, and frequency of vertigo in patients with VM, reporting improvement rates of 68% (41) and superiority when compared to other preventatives such as flunarizine in reducing vestibular symptoms and improving quality of life (42). Topiramate is associated with a high rate of dose-dependent adverse effects (28). The advantage is that it does not cause changes in the patient's weight as other preventives often do.

In previous pathophysiological models, vestibular symptoms in VM were believed to be a manifestation of migraine aura, which generated interest in the use of lamotrigine for the prevention of VM (28). A small retrospective study showed that lamotrigine significantly reduced the frequency of vertigo from 18.1 to 5.4 episodes per month (43). Participants reported no side effects.

7.3.3 Antidepressants

Despite being one of the most widely used preventative treatments for migraine, there have been no specific studies on antidepressant medications in VM, and they have only been examined in small multi-treatment arm studies (28).

Venlafaxine has shown promising results in several trials, demonstrating improvement in vertigo intensity and emotional well-being (40). The response of venlafaxine is greater when compared to other preventatives such as flunarizine and sodium valproate (40) and a meta-analysis suggests it as a first-line treatment (36). Venlafaxine is likely to have additional benefits in patients with VM who present with concurrent low mood (28).

In a retrospective study, 13 patients treated with amitriptyline showed significant improvement in vestibular symptoms and headache after 3 months of use (44). In another study of 24 patients treated with amitriptyline for 5 weeks, reductions in the average monthly frequency of vertigo (17.5 to 5.4) were observed. Side effects were common, including xerostomia in 67% and daytime sleepiness in 61% (45).

7.3.4 Calcium Channel Blockers

A recent meta-analysis found that flunarizine significantly improved DHI scores compared to placebo, although it had greater adverse effects (46). Lepcha and colleagues conducted an open-label randomized trial with 52 patients with VM, 88% of the group receiving flunarizine reported a "low frequency of vertigo" (2-3 attacks or less every 3 months) compared to 52% of the control group (47).

There is no evidence to support the use of verapamil in VM. Three patients were treated with verapamil in a retrospective multi-arm study, in which the duration of treatment was not specified. On average, the DHI was slightly reduced (9.3 points) from before to after treatment (48).

Calcium channel blockers had a higher rate of adverse effects 24% vs 9% in the control group (47).

7.3.5 Botulinum Toxin

Botulinum toxin type A (BTX-A) has shown good results in the treatment of VM, particularly in patients resistant to conventional therapies. Studies have shown significant reductions in the frequency of vertigo and migraine episodes, as well as improvements in disability scores, quality of life, and neuropsychiatric inventories (49,50).

7.3.6 CGPR Antagonists

Monoclonal antibodies (mAbs) directed against CGRP-related peptide or its receptor are widely used in migraine but have rarely been considered in the treatment of VM. One study investigated the efficacy of erenumab, fremanezumab, and galcanezumab in the treatment of 50 patients with VM who had failed or had a contraindication to at least three traditional preventive treatments. A significant reduction in the frequency of vestibular symptoms (from 10.3 to 0.8 days) as well as in the frequency of headache and associated disability was observed. No differences in efficacy were found among the three monoclonal antibodies (51).

7.4 Other Therapies

Neuromodulation can be used for both acute and prophylactic treatment of VM, two have shown initial promise (noninvasive vagus nerve stimulation and external trigeminal nerve stimulation), improving vertigo within minutes of use, but long-term benefits are unknown and devices are not readily available (28).

Vestibular rehabilitation is safe and can improve vertigo, headache, anxiety and depression symptoms in some patients with VM, especially when symptoms have become chronic with incomplete resolution, or there is associated BPPV. In general, patients who are most likely to benefit are those who can tolerate some degree of movement (28). Exercises should be tailored based on vestibular testing, usually including balance exercises, gaze stability training and habituation exercises (52).

8. Prognosis

The prognosis for VM appears to be less favorable compared to other types of migraine (25). In a long-term follow-up (median of nine years) of 61 patients with VM, 87% of patients continued to have recurrent vertigo, although the frequency of episodes was reduced in more than half of the cases (53).

9. Conclusion

VM is one of the most common causes of vertigo and dizziness, but it remains underdiagnosed due to the wide spectrum of symptoms and the absence of headache in many cases. The diagnosis of VM is based on clinical criteria, and complementary studies play a secondary role given the absence of typical findings. Early recognition is crucial for proper management, which focuses on strategies similar to those for other forms of migraine. Although there is a lack of high-quality evidence, preventive treatment should be instituted if required to improve the quality of life of patients. Future studies using the relatively recent diagnostic criteria will clarify the best therapeutic approach and minimize the morbidity generated by this pathology.

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