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Review

Vestibular Migraine Revisited: A Narrative Review of Diagnostic Challenges and Treatment Strategies

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Vestibular migraine (VM) is a common yet frequently underdiagnosed neurological condition, marked by recurrent episodes of vertigo and other vestibular symptoms in association with migraine features. It predominantly affects women aged 30-50 years and has an estimated prevalence of 1%-5% in the general population. This narrative review explores current knowledge surrounding VM, including its epidemiology, proposed mechanisms, diagnostic complexities, and treatment approaches. The pathophysiology remains incompletely understood but may involve dysfunction in vestibule-cerebellar pathways, ion channel abnormalities, and trigeminal system activation. Diagnosing VM is clinically driven, requiring careful evaluation of vestibular complaints alongside migraine-associated symptoms. Patients commonly report vertigo and headaches, while clinical assessment may uncover ocular motor disturbances, canal paresis, and balance issues. Supplementary tests such as ocular and cervical vestibular evoked myogenic potentials can aid in diagnosis, though they are not definitive. Differential diagnosis is essential due to symptom overlap with other vestibular disorders like Ménière's disease, episodic ataxia type 2, and benign paroxysmal positional vertigo. Treatment includes acute interventions with vestibular suppressants and triptans, vestibular rehabilitation programs, and preventive pharmacotherapy such as β-blockers, calcium channel blockers, and certain antidepressants. Despite these options, clinical evidence remains scarce, primarily relying on small-scale trials and expert consensus. No universally effective regimen has yet been identified. Overall, VM poses significant diagnostic and therapeutic challenges, underscoring the need for further research to clarify its mechanisms, improve diagnostic precision, and develop evidence-based treatment strategies that could lessen its burden and improve patient outcomes.

KEYWORDS: Migraine-associated vertigo, pathophysiology, prophylaxis, treatment, vestibular migraine

INTRODUCTION

Migraine is frequently associated with vertigo, and vestibular migraine (VM) is a syndrome characterized by recurrent episodes of vertigo or other vestibular symptoms attributed to migraine. Over the years, various terms have been used to describe this condition, including migraine-associated vertigo, migraine-associated dizziness, migraine-related vestibulopathy, and migrainous vertigo. Additionally, the overlap between VM and other conditions, such as Ménière's disease (MD) and migraine with brainstem aura (formerly known as basilar migraine), has added to the challenges in reaching a consensus on its terminology and diagnostic criteria.[1,2]

To address these challenges, diagnostic criteria for VM were developed and subsequently validated to help distinguish VM from other similar conditions, although controversies regarding the sensitivity and specificity of these criteria still exist.[1,3] These criteria assess the probability of VM based on the intensity of vestibular symptoms, the presence of migraine-related features in the patient's medical history, and appropriate laboratory tests.[2] The manuscript refers to both the diagnostic criteria of the International Classification of Headache Disorders, 3rd edition (ICHD-3), and those of the Bárány Society (International Classification of Vestibular Disorders—ICVD). However, primary emphasis is placed on the ICHD-3 criteria, which serve as the main reference standard for

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Table 1. Symptoms Indicative of Vestibular Migraine, According to the International Classification of Headache Disorders-3rd edition (ICHD-3), and the International Classification of Vestibular Disorders (ICVD)*

Vestibular Migraine	
Diagnostic criteria	Remarks
A. At least 5 episodes fulfilling criteria C and D	
B. A current or past history of migraine without aura or migraine with aura	
C. Vestibular symptoms** of moderate or severe intensity***, lasting between 5 minutes and 72 hours****	**Qualifying symptoms include: a) Spontaneous vertigo b) Positional vertigo c) Visually induced vertigo d) Head motion–induced vertigo e) Head motion–induced dizziness with nausea
	***Moderate symptoms interfere with but do not prevent daily activities, while severe symptoms do not allow daily activities to be continued
D. At least 50% of episodes are associated with at least 1 of the following 3 migrainous features: 1. Headache with at least 2 of the following 4 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	****For bouts lasting seconds only, episode duration is defined as the total period during which short attacks recur
E. Not better accounted for by another ICHD-3 diagnosis, or by another vestibular disorder	

*The Bárány Society classification also includes a probable vestibular migraine category.

diagnosing VM, while the Bárány Society criteria are used to complement and contextualize symptom presentation (Table 1). Ensuring diagnostic accuracy is critical, given the high prevalence of VM and its substantial health burden, which necessitates proper patient management. However, some authors continue to debate whether VM constitutes a distinct disease, as it currently lacks definitive clinical or biological markers.[4-8]

The aim of this study is to review the epidemiology, etiology, pathophysiology, and diagnosis of VM and to critically evaluate the evidence supporting its clinical management.

MAIN POINTS

- Vestibular migraine (VM) is a common but underdiagnosed condition marked by recurrent vertigo linked to migraine, affecting mainly women aged 30-50 years.
- The pathophysiology of VM involves dysfunction in vestibulocerebellar pathways, trigeminal activation, neurochemical imbalances, and possibly genetic channelopathies.
- Diagnosis of VM is clinical and complicated by symptom overlap with disorders like Ménière's disease, benign paroxysmal positional vertigo, and psychiatric conditions.
- Treatment options include vestibular suppressants, triptans, vestibular rehabilitation, and various prophylactic drugs, though high-quality evidence is limited.
- There is an urgent need for standardized diagnostic criteria and robust clinical trials to guide effective management and improve patient outcomes.

METHODS

Study Design

The study aimed to evaluate the epidemiology, pathophysiology, diagnostic challenges, and treatment strategies for VM by synthesizing relevant literature.

Inclusion and Exclusion Criteria

Studies were eligible for inclusion if they met the following conditions: a) focused on VM diagnosis, pathophysiology, or treatment, b) published in English, French, or German, and c) presented original clinical data, systematic reviews, or meta-analyses.

Studies were excluded if they met any of the following conditions: a) full-text articles were not available in English, French, or German when no translation was accessible, b) studies focused on non-relevant conditions or lacked sufficient data to contribute meaningfully to the review.

Search Strategy

A literature review was conducted to explore existing research on the topic. Relevant studies published up to February 2024 were identified through searches in MEDLINE, Scopus, CINAHL, and Google Scholar, using Boolean operators (AND, OR, NOT) to refine results. The search terms included ("vestibular migraine" OR "migrainous vertigo" OR "migraine-associated dizziness") for identifying relevant conditions, ("pathophysiology" OR "mechanisms" OR "neurovascular" OR "ion channels") for studies on underlying mechanisms, ("diagnosis" OR "clinical criteria" OR "differential diagnosis" OR "diagnostic accuracy") for diagnostic-related research, and ("treatment" OR

"management" OR "vestibular rehabilitation" OR "pharmacotherapy") for intervention-focused studies. To prioritize high-quality evidence, the search was further refined using ("systematic review" OR "meta-analysis" OR "randomized controlled trial" OR "RCT"), while excluding irrelevant studies with NOT ("case report" OR "editorial" OR "conference abstract"). Additional filters were applied to restrict results to studies published in English, French, or German, and to focus on guidelines, RCTs, and systematic reviews. Grey literature, including recommendations from neurological and neurotological societies across Europe, North America, and Asia, was also reviewed to ensure comprehensive coverage of the topic.

Study Selection

Initially, 3.489 records were identified through database searches (MEDLINE, Scopus, CINAHL, and Google Scholar), with 2.793 duplicate records removed before screening, leaving 696 records for further evaluation. During screening, 252 records were excluded based on their title and 81 based on their abstract, resulting in 363 reports sought for retrieval. Among these, 41 reports were excluded due to full-text unavailability or being in a non-English language. Subsequently, 322 reports were assessed for eligibility, out of which 272 were excluded for various reasons, including lack of specificity to VM, insufficient data, irrelevant full text, or association with other pathologies. Ultimately, 50 studies were included in the final review.

Data Extraction

Data extraction was performed independently by 2 reviewers, focusing on study design (e.g., RCT, observational, case series), sample characteristics (population size, demographics), and key outcomes related to VM diagnosis, pathophysiology, and treatment. The included studies were critically appraised using established evidence-based guidelines (Table 2). Levels of evidence ranged from level I (high-quality RCTs) to level V (expert opinion). Recommendations were graded accordingly, and quantitative data, where available, were tabulated for clarity. Strength of recommendation grades (A-D) were assigned based on the quality of evidence according to the framework in Table 2. In cases where RCT evidence was limited by small sample size, lack of blinding, or methodological constraints, the strength of recommendation was downgraded accordingly, and limitations are explicitly discussed.

Risk of Bias Assessment

The risk of bias was evaluated using the Cochrane Risk of Bias (RoB 2) tool for RCTs and the Newcastle–Ottawa scale for observational studies. Studies were assessed based on selection bias, performance bias, detection bias, attrition bias, and reporting bias. Discrepancies between reviewers were resolved through consensus discussion.

Statistical Analysis

Due to the heterogeneity of included studies, a qualitative synthesis was performed rather than a meta-analysis. The results were synthesized qualitatively, with descriptive summaries provided for diagnostic criteria, treatment strategies, and pathophysiological mechanisms. Quantitative findings, where available, were tabulated for clarity.

RESULTS AND DISCUSSION

A review of the literature reveals that most studies on VM focus on characterizing its clinical presentation, proposing diagnostic criteria, and evaluating pharmacologic interventions such as beta-blockers, calcium channel blockers, antidepressants, and antiepileptic agents. While some small-scale trials and observational studies suggest potential benefits, robust evidence from large, RCTs remains limited. Non-pharmacological treatments, including vestibular rehabilitation (VR) and lifestyle modifications, have been explored, but conclusions about their efficacy are inconclusive due to methodological limitations and underutilization in practice. An overview of the key characteristics of the studies included in this analysis is available in Appendix Table 1.

Epidemiology

Vestibular migraine is a common yet frequently underdiagnosed condition among patients with postural impairment, with a lifetime prevalence estimated between 1% and 5% in the general population.[3,9] Additionally, VM accounts for up to 10% of cases in dizziness clinics.[2,9] Epidemiological studies indicate a marked female predominance, with a female-to-male ratio ranging from 1.5 : 1 to 5 : 1.[4,10]

Table 2. Levels of Evidence Regarding the Primary Research Question in Studies that Investigate the Results of a Treatment and Strength of Recommendation by Category of Evidence for Guideline Development

Category of Evidence	Study Design	Strength of Recommendation
Level I	High quality randomized trial with statistically significant difference, or no statistically significant difference but narrow confidence intervals Systematic review of level I randomized control trials (and study results were homogenous)	A: Directly based on category I evidence
Level II	Lesser quality randomized control trial (e.g. <80% follow up, no blinding, or improper randomization) Prospective comparative study Systematic review of level II studies or level I studies with inconsistent results	B: Directly based on category II evidence or extrapolated recommendation from category I evidence
Level III	Case-control study Retrospective comparative study Systematic review of level III studies	C: Directly based on category III evidence or extrapolated recommendation from category I or II evidence
Level IV	Case series	D: Directly based on category IV evidence or extrapolated recommendation from category I, II, or III evidence
Level V	Expert opinion	

VM primarily affects individuals in their fourth to fifth decades of life.[4,10] Interestingly, migraine and vertigo do not always occur concurrently; headaches often precede vertigo by years. For instance, 1 study reported that 65.6% of patients experienced migraines longer than vertigo, with an average gap of 7.3 years between the onset of headaches and dizziness.[11] The mean onset age for migraines is approximately 23 ± 9 years, compared to 38 ± 13 years for vertigo.[4] Vestibular migraine is more prevalent among individuals without aura than those with aura.[12,13] Moreover, a positive family history of migraine and vertigo is common, reported in up to 70.2% of migraine cases and 66.3% of vertigo cases in different studies.[4,14]

Pathophysiology

The pathophysiology of VM remains incompletely understood, though several mechanisms have been proposed, potentially acting in combination. Processes underlying migraines, including dysfunction in vestibulocerebellar pathways, trigeminal ganglion excitation, cortical spreading depression, neuronal sensitization, and channelopathies, are implicated in VM.[15,16] Novel neurochemical theories suggest that VM may differ from basilar artery migraine, though vascular involvement, such as internal auditory artery vasospasm, might contribute to peripheral vestibular and auditory symptoms. Inner ear ischemia could further explain the association between VM and MD. Neuropeptides like glutamate, acetylcholine, and calcitonin gene-related peptide, which are released during migraines, modulate both the peripheral and central vestibular systems, influencing symptoms.[15,16]

Nociceptive and vestibular signals, sharing neurochemical pathways, converge at brainstem structures such as the parabrachial nucleus, raphe nuclei, and locus coeruleus, suggesting a parallel activation of vestibular and cranial nociceptive pathways during migraine attacks.[16] Marano et al[17] demonstrated that trigeminal nerve activation, via painful skin stimulation, induced or altered nystagmus exclusively in VM patients, underscoring the interaction between the trigeminal and vestibular systems.

Ion channel dysfunction in the vestibular system also plays a role. Von Brevern et al[18] identified mutations in candidate genes such as *CACNA1A* (neuronal calcium channel), *ATP1A2* (Na+/K+-ATPase), and *SCN1A* and *CACNB4* (calcium channel subunits), which may cause changes in endolymph composition, hair cell depolarization, and neurotransmitter release, leading to VM symptoms. However, these genetic mutations appear to have a limited role in VM pathogenesis.

Table 3. Clinical Characteristics in Vestibular Migraine

Clinical Characteristics		
Most common		Vertigo
		Headache
Less common		Motion intolerance
		Oscillopsia
		Nausea
		Photophobia
		Phonophobia
		Aura
	Cochlear symptoms (i.e. aural fullness, tinnitus, hearing loss)	

Despite the lack of a definitive mechanism, evidence points to a combined genetic and environmental contribution to VM. Variability in prevalence based on ethnicity and geographic location, as well as familial clustering, supports this multifactorial model.[19]

Clinical Characteristics

Clinical characteristics of VM are presented in Table 3.

Vertigo

The types of vertigo experienced by patients with VM vary widely and may include spontaneous vertigo, positional vertigo, visually induced vertigo, head-motion-induced vertigo, or head-motion-induced dizziness.[1] In a collaborative study involving neurologists and ENT (Ear Nose Throat) specialists, which included 252 patients with confirmed VM, the most common presentations were internal vertigo, positional dizziness, and unsteadiness.[10] The duration of vertigo attacks also varies significantly; a meta-analysis by Furman et al[15] found that attacks lasted for hours in 28% of patients, days in 25.8%, minutes in 20.2%, and seconds in 12.6%. While approximately 50% of patients experience multiple attacks weekly, others may go a month without an episode.[13]

Headache

Headache is often the most common migrainous symptom during vertigo episodes, though it does not necessarily accompany vertigo in all cases.[12,14] Headaches were identified as the chief complaint in a 9-year follow-up study by Radtke et al.[20] Similarly, in a study by Neuhauser et al,[21] 91% of patients reported experiencing headaches, but only 24% had headaches during every vertigo episode.

Other Symptoms

Vestibular migraine patients frequently present with additional symptoms that can aid in diagnosis, especially in cases of vertigo without concurrent headache. These include motion intolerance, oscillopsia, nausea, photophobia, phonophobia, and aura.[4,12,20] Cochlear symptoms such as aural fullness, tinnitus, and mild hearing loss are also observed, though their prevalence varies across studies.[7,10-14,20]

Physical Examination–Laboratory and Imaging Tests

While neither physical examination nor laboratory testing alone is sufficient for diagnosing VM, they are essential in ruling out alternative causes of episodic vertigo, especially in cases with atypical features or new-onset symptoms that do not meet diagnostic criteria. Diagnostic testing may not be necessary for patients with a well-documented history of episodic vertigo consistent with VM, no atypical features, and a normal interictal examination.

Assessment of static balance and gait is particularly valuable in patients with dizziness or vertigo. Abnormal balance is observed in approximately half of VM patients. Specifically, 19% may exhibit impaired stance and gait, 40% show pathologic tandem walk, 65% have difficulty walking with eyes open, 20%-70% demonstrate a positive Romberg's test, and 54% have a positive tandem Romberg's test.[8,20]

Central ocular abnormalities are present in 60%-70% of VM patients during the interictal period. These may include vertical or horizontal saccadic pursuit, gaze-evoked nystagmus, moderate positional nystagmus (horizontal, pure up- or downbeating, or alternate), spontaneous nystagmus, and head-shaking nystagmus.[20] Oculographic

findings during attacks often suggest a central origin in 50% of cases, a peripheral origin in 15%, and indeterminate findings in 35%.[20]

Vestibular laboratory tests, such as the video head impulse test, caloric testing, and vestibular evoked myogenic potentials (VEMPs), are not typically required for diagnosing VM but may reveal nonspecific abnormalities.[20] Pure-tone audiometry may show mild sensorineural hearing loss, though it is generally age-appropriate and symmetric.[8,20]

Differential Diagnosis

A variety of disorders can present with symptoms similar to those of VM, primarily including MD, episodic ataxia type 2, benign paroxysmal positional vertigo (BPPV), and psychiatric syndromes. Accurate differentiation is essential for appropriate management.

Ménière's Disease

Differentiating VM from MD is challenging due to overlapping symptoms and reliance on clinical markers. Basal cytokine levels, such as interleukin-1β and CCL_3, may serve as biomarkers.[22] The VM predominantly affects women (4 : 1 to 8 : 1 ratio) with a mean onset age of 41 years, compared to 51 years for MD. Ménière's Disease is characterized by unilateral auditory symptoms like tinnitus, aural fullness, and hearing loss, while VM symptoms are milder and often bilateral. Moreover, regarding hearing loss in VM, it does not progress to profound deafness, as it may do in MD, therefore being an important differentiating factor between these 2 clinical entities.[23] The VM is more likely to present with migrainous symptoms such as headaches with or without aura. While only 40% of MD patients report headaches during vertigo attacks, fewer than 9% meet the diagnostic criteria for migraine. However, MD patients exhibit a higher lifetime prevalence of migraine (56%) compared to healthy controls (25%).[20] Advanced diagnostics, including click-evoked cervical VEMP (c-VEMP) and ocular VEMP (o-VEMP) testing and magnetic resonance imaging with gadolinium, help differentiate the conditions, with endolymphatic hydrops present in most MD but fewer VM cases.[24]

Benign Paroxysmal Positional Vertigo

The BPPV should be considered in the differential diagnosis of VM, particularly in cases involving positional vertigo. While VM-associated positional vertigo typically lasts for hours to days, BPPV attacks are shorter, lasting only minutes and correlating with a specific semicircular canal during clinical examination. Direct observation of nystagmus during an attack is critical for differentiation.[18,23]

Episodic Ataxia Type 2

This rare autosomal-dominant paroxysmal disorder can mimic VM. Episodic ataxia type 2 is characterized by truncal ataxia, restricted movement coordination lasting hours, and triggers such as physical or emotional stress. Associated symptoms include vertigo, nausea, vomiting, baseline ataxia exacerbation, and migraine. Gaze-evoked, spontaneous, or positional nystagmus is common during interictal periods but less frequent during VM attacks. Treatment with acetazolamide often alleviates symptoms, and genetic testing for *CACNA1A* mutations confirms the diagnosis.[25]

Psychiatric Syndromes

Vertigo, dizziness, and migraines often overlap with psychiatric conditions such as anxiety, panic disorders, and depression. These symptoms are typically accompanied by circumstantial exacerbation, acute autonomic responses, negative thought patterns, and avoidance behaviors. Although VM and psychiatric syndromes may coexist, differentiating the 2 is challenging. A study reported that 57% of patients with acute vertigo felt that their anxiety symptoms did not match the severity of their vertigo, underscoring the complexity of diagnosis.[26]

Vertebrobasilar Insufficiency

Transient ischemic attacks (TIAs) in the vertebrobasilar arterial system can mimic VM. Vertigo, described as swaying or swimming, is often the sole symptom in one-third of vertebrobasilar insufficiency (VBI) patients. Other accompanying features may include diffuse, stabbing headaches, nausea, vomiting, facial numbness, or visual field deficits. The presence of stroke risk factors should raise suspicion for VBI, emphasizing the importance of a thorough neurological assessment.[27]

Migraine with Brainstem Aura

More than 60% of patients with migraine with brainstem aura (previously basilar migraine) report vertigo. The ICHD-3 criteria require at least 2 reversible brainstem symptoms lasting 5-60 minutes for diagnosis. However, fewer than 10% of VM patients meet these criteria, highlighting the potential for overlap in diagnostic categories.[2]

Vestibular Paroxysmia

This condition presents as brief, recurrent vertigo attacks lasting seconds and occurring multiple times daily. Successful prevention with carbamazepine supports the diagnosis.[2]

Diagnostic Controversies

Despite the significance of VM in cases of episodic vertigo, there is ongoing debate about whether it should be considered a distinct disease or a diagnosis of convenience, applied when no better or more specific explanation is available. The association between migraine and vertigo is primarily supported by epidemiological evidence, and both conditions are known to be linked to anxiety disorders.[5]

Cortical spreading depression, a hypothesized pathophysiological mechanism of VM, is unlikely to selectively induce brainstem depression limited to the vestibular nuclei without also causing concurrent audiological or neurological symptoms.[6,8] Similarly, cortical dysfunction does not adequately explain findings such as canal paresis and complex positional nystagmus observed during migraine episodes in VM patients. Furthermore, pathological findings from diagnostic tools like video-nystagmography, rotational chair testing, and postural control assessments often overlap with those seen in peripheral labyrinthine disorders like MD, complicating the differential diagnosis.[7]

The diagnosis of VM based on symptom regression following the administration of migraine prophylactic medication remains controversial, as the improvement could also be attributed to the anxiolytic effects of these treatments. Additionally, the inclusion of patients without clear migraine or vertigo symptoms within the VM diagnostic criteria further challenges its acceptance as a distinct clinical entity.[4]

Treatment

The treatment of VM involves reducing triggering factors, pharmacotherapy, VR, exercise, and dietary modifications. Acute VM symptoms are commonly managed with vestibular suppressants, including

antihistamines, antiemetics, and benzodiazepines. Triptans are an alternative option for patients who do not respond to initial treatment or experience concurrent headache symptoms. However, the efficacy of pharmacological treatments for VM remains inconclusive due to limited high-quality evidence, with only 2 RCTs focusing on triptans for acute VM symptoms.

In a randomized, double-blinded, placebo-controlled trial by Neuhauser et al,[28] patients with moderate or severe VM were treated with a single dose of 2.5 mg zolmitriptan or placebo.[8] Secondary doses of zolmitriptan, 150 mg dimenhydrinate (for vertigo), or 500 mg paracetamol (for headache) were permitted 2 hours after the initial dose. Improvement in vertigo symptoms was reported in 38% of patients treated with zolmitriptan, compared to 22% in the placebo group, though the small sample size (11 patients and 17 seizures) limited the study's conclusiveness. Similarly, headache relief was achieved in 20% of zolmitriptan-treated patients versus 40% of those receiving placebo.

Another double-blinded RCT by Furman et al[29] evaluated rizatriptan in 25 migraine sufferers with or without migraine-related dizziness. The study found that 87% of patients with vestibular-induced motion sickness experienced symptom improvement, suggesting that rizatriptan may raise the threshold for vestibular motion sickness. However, these results may not generalize to more intense vestibular stimuli. Additionally, almotriptan (12.5 mg orally) was reported to alleviate both vertigo and headache in VM patients in another study.[30] Intravenous methylprednisolone has also shown promise in treating migrainous vertigo, as demonstrated in a small case series involving 4 patients.[31]

Apart from triptans, vestibular suppressants remain a mainstay for acute VM treatment. The selection of specific agents depends on patient symptoms, comorbidities, and risk of adverse effects. A meta-analysis of 17 clinical trials involving 1586 patients found that single-dose antihistamines provided greater acute symptom relief within 2 hours than benzodiazepines.[32]

A 2023 Cochrane Database Systematic Review, which included RCTs and quasi-RCTs comparing various pharmacological agents such as triptans, ergot alkaloids, antihistamines, and NSAIDs to placebo or no treatment, concluded that the evidence for effective medications to treat acute VM attacks is insufficient. This is largely due to the lack of placebo-controlled trials beyond the 2 RCTs focusing on triptans.[33]

Noninvasive treatments, including vagal nerve stimulation and external trigeminal nerve stimulation, have shown potential in reducing vertigo intensity or shortening attack duration in single-center retrospective case series. Further studies are needed to confirm these findings in order to be widely applied to other countries, including India, where vagal nerve stimulation is not yet available.[34]

Vestibular Rehabilitation and Exercise

Vestibular rehabilitation has been proposed as a non-pharmacological approach for managing VM, especially in patients experiencing persistent imbalance or motion intolerance. While VR aims to improve vestibulo-ocular reflexes and facilitate functional adaptation—particularly in patients with altered vestibular responses—its efficacy in VM remains uncertain. A review by Alghadir et al[35] analyzing 6 studies suggested potential benefits in reducing headache-related disability, but emphasized the lack of conclusive evidence for improving vestibular symptoms specifically in VM. Similarly, a retrospective study by Power et al[14] reported that VR was underutilized, with only 14 of 90 patients receiving such intervention, and found no consistent clinical benefit. These limitations underscore the need for more rigorous, methodologically sound clinical trials.

In addition to rehabilitation, exercise has been suggested to play a role in alleviating VM symptoms. A study by Lee et al[36] demonstrated that exercise might suppress pro-inflammatory processes implicated in VM pathophysiology by inhibiting the cyclooxygenase-2 pathway.

Prophylaxis

Prophylactic medications play a key role in preventing vestibular vertigo and mitigating the severity of VM symptoms. A variety of pharmacological agents have been employed, including β-blockers (e.g., metoprolol, propranolol), antiepileptic drugs (e.g., topiramate, carbamazepine, valproic acid, lamotrigine), antidepressants (e.g., amitriptyline, venlafaxine), calcium channel blockers (e.g., flunarizine, cinnarizine, lomerizine, verapamil), CGRP (Calcitonin Gene-Related Peptide) antagonists, antihistamines (e.g., pizotifen), diuretics (e.g., acetazolamide), and others like botulinum toxin and hormonal treatments.

The PROVEMIG trial conducted by Bayer et al[37] was a double-blind, randomized placebo-controlled study involving 130 patients. It reported an incidence rate ratio of 0.983 (95% CI 0.902-1.071) for metoprolol versus placebo. Both groups exhibited a significant reduction in monthly vertigo attacks (factor of 0.830, 95% CI 0.776-0.887). However, the trial was discontinued due to poor participant accrual, and no significant treatment benefit of metoprolol over placebo could be established.

Liu et al[38] compared venlafaxine, flunarizine, and valproic acid in a single-blind randomized trial. They found venlafaxine more effective in alleviating depression, while valproic acid was inferior in reducing vertigo intensity, and flunarizine showed limited efficacy in reducing vertigo frequency. Similarly, a randomized controlled study by Salviz et al[39] demonstrated that venlafaxine and propranolol were equally effective in managing vertigo symptoms, though venlafaxine also alleviated comorbid depressive symptoms.

In another randomized controlled study, Lepcha et al[40] found that adding flunarizine to a regimen of propranolol and betahistine reduced vertigo intensity without significantly affecting headache severity. A 2020 multicenter prospective study involving 31 VM patients prescribed acetazolamide, amitriptyline, flunarizine, propranolol, or topiramate showed significant reductions in vestibular symptom intensity (45.8 points), headache severity (47.8 points), and

monthly attacks (15.6 fewer crises). There were no significant differences between the drugs used.[41]

Observational studies further underscore the effectiveness of specific prophylactic agents. Teggi et al[42] reported that a combination of cinnarizine and dimenhydrinate reduced vertigo attacks by at least 50% in 68% of patients, with 63% also reporting improvements in headaches. Topiramate was found effective in reducing both the frequency and severity of vertigo and headaches in a prospective study involving 30 patients.[43] Additionally, Salmito et al[44] observed significant improvement in 80.9% of VM patients treated with amitriptyline, flunarizine, propranolol, or topiramate ($P < .001$).

Acetazolamide, a carbonic anhydrase inhibitor, demonstrated reductions in vertigo frequency and migraine intensity in a retrospective cohort study of 39 patients, supporting the theory of ion channel dysfunction in VM.[45] A retrospective analysis by Van Ombergen et al showed improvements in 68% of patients treated with flunarizine and 73% treated with propranolol.[21] Cinnarizine, a calcium channel blocker, was also effective in reducing vertigo frequency and headache persistence over 3 months of treatment in a retrospective study.[46]

Other innovative approaches include a stepwise strategy involving caffeine discontinuation followed by pharmacotherapy (e.g., nortriptyline or topiramate), which improved symptoms in 75% of VM patients in a retrospective review.[47] Baier et al[48] found that 80% of patients treated with β-blockers, valproic acid, topiramate, lamotrigine, or magnesium experienced fewer, milder, and shorter attacks compared to those receiving conservative therapy and lifestyle modifications. Lamotrigine, in particular, has been shown to significantly reduce vertigo episodes in 89.5% of patients, though its effect on headaches was less pronounced.[49]

A systematic review and meta-analysis identified propranolol as the most effective prophylactic agent, achieving complete symptom control in 60% of patients. Venlafaxine was also effective, particularly in patients with comorbid depression, while amitriptyline, flunarizine, and cinnarizine showed trends toward efficacy without achieving statistical significance.[50]

Dietary Modifications

Interestingly, a retrospective study by Reploeg and Goebel reported improvement in vertigo and disequilibrium in all patients with migraine-associated dizziness who were treated solely with dietary modifications. These included avoiding trigger foods such as aged cheese, processed meats, and certain red wines.[13] However, combining dietary changes with prophylactic medications, such as nortriptyline, atenolol, or calcium channel blockers, showed less effectiveness, likely due to the inclusion of more severe cases in the combined treatment group.[13]

Currently, the first-line drugs recommended for VM prophylaxis are β-blockers (e.g., propranolol), calcium channel antagonists (e.g., flunarizine), betahistine, and venlafaxine. Despite the limited number of studies, β-blockers, betahistine, and calcium channel antagonists are considered effective in controlling vertigo, with a strength of recommendation A. Calcium channel antagonists may also reduce the frequency and severity of headaches, though the evidence is insufficient for a strong recommendation. Venlafaxine appears to be particularly beneficial for patients with coexisting severe depression, based on small-scale RCTs and retrospective analyses (level II-III evidence). Accordingly, it is assigned a strength B recommendation, reflecting encouraging but limited data.[39,40]

CONCLUSION

Vestibular migraine remains a challenging and underdiagnosed condition, despite its considerable prevalence and impact on patients' quality of life. This review highlights the complexities surrounding its diagnosis and management, emphasizing the need for refined clinical criteria and diagnostic tools to effectively differentiate VM from overlapping conditions such as MD and BPPV. Although current insights suggest a multifactorial pathophysiology involving genetic, environmental, and neurochemical factors, further research is essential to clarify these mechanisms and their clinical relevance. Despite increasing recognition of VM as a distinct clinical entity, no universally effective treatment regimen has been established. Management strategies remain largely empirical, with most interventions, including acute therapies like triptans and vestibular suppressants and prophylactic treatments such as β-blockers and antidepressants, supported by small-scale studies or expert opinion. Non-pharmacological interventions, including VR, lifestyle modifications, and trigger avoidance, also lack robust evidence of efficacy. Advancing the understanding of VM will require high-quality, large-scale RCTs to validate existing therapies and guide the development of targeted, evidence-based treatment protocols. Multidisciplinary collaboration and a patient-centered approach will be critical to reducing the disease burden and improving clinical outcomes for individuals affected by VM.

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REFERENCES

1. Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria. *J Vestib Res*. 2012;22(4):167-172.[CrossRef]
2. Vincent M, Wang S. Headache Classification Committee of the International Headache Society (IHS). Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia*. 2018;38(1):1-211. [CrossRef]
3. Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria1. *J Vestib Res*. 2022;32(1):1-6. [CrossRef]

4. Teggi R, Colombo B, Albera R, et al. Clinical features, familial history, and migraine precursors in patients with definite vestibular migraine: the VM-phenotypes projects. *Headache*. 2018;58(4):534-544. [CrossRef]
5. Kang WS, Lee SH, Yang CJ, Ahn JH, Chung JW, Park HJ. Vestibular function tests for vestibular migraine: clinical implication of video head impulse and caloric tests. *Front Neurol*. 2016;7(SEP):4-8. [CrossRef]
6. Shin JH, Kim YK, Kim HJ, Kim JS. Altered brain metabolism in vestibular migraine: comparison of interictal and ictal findings. *Cephalalgia*. 2014;34(1):58-67. [CrossRef]
7. Obermann M, Wurthmann S, Steinberg BS, Theysohn N, Diener HC, Naegel S. Central vestibular system modulation in vestibular migraine. *Cephalalgia*. 2014;34(13):1053-1061. [CrossRef]
8. Von Brevern M, Zeise D, Neuhauser H, Clarke AH, Lempert T. Acute migrainous vertigo: clinical and oculographic findings. *Brain*. 2005;128(2):365-374. [CrossRef]
9. Huang T-C, Wang S-J, Kheradmand A. Vestibular migraine: an update on current understanding and future directions. *Cephalalgia*. 2020;40(1):107-121. [CrossRef]
10. Colombo B, Teggi R. Vestibular migraine: who is the patient? *Neurol Sci*. 2017;38(S1):107-110. [CrossRef]
11. Morganti LOG, Salmito MC, Duarte JA, Sumi KC, Simões JC, Ganança FF. Migrânea vestibular: aspectosclínicos e epidemiológicos. *Braz J Otorhinolaryngol*. 2016;82(4):397-402. [CrossRef]
12. Zhang Y, Kong Q, Chen J, Li L, Wang D, Zhou J. International Classification of Headache Disorders 3rd edition beta-based field testing of vestibular migraine in China: Demographic, clinical characteristics, audiometric findings and diagnosis statues. *Cephalalgia*. 2016;36(3):240-248. [CrossRef]
13. Reploeg MD, Goebel JA. Migraine-associated dizziness: patient characteristics and management options. *Otol Neurotol*. 2002;23(3):364-371. [CrossRef]
14. Power L, Shute W, McOwan B, Murray K, Szmulewicz D. Clinical characteristics and treatment choice in vestibular migraine. *J Clin Neurosci*. 2018;52:50-53. [CrossRef]
15. Furman JM, Marcus DA, Balaban CD. Migrainous vertigo: development of a pathogenetic model and structured diagnostic interview. *Curr Opin Neurol*. 2003;16(1):5-13. [CrossRef]
16. Lee H, Lopez I, Ishiyama A, Baloh RW. Can migraine damage the inner ear? *Arch Neurol*. 2000;57(11). [CrossRef]
17. Marano E, Marcelli V, Di Stasio ED, et al. Trigeminal stimulation elicits a peripheral vestibular imbalance in migraine patients. *Headache*. 2005;45(4):325-331. [CrossRef]
18. Von Brevern M, Ta N, Shankar A, et al. Migrainous vertigo: mutation analysis of the candidate genes CACNA1A, ATP1A2, SCN1A, and CACNB4. *Headache*. 2006;46(7):1136-1141. [CrossRef]
19. Paz-Tamayo A, Perez-Carpena P, Lopez-Escamez JA. Systematic review of prevalence studies and familial aggregation in vestibular migraine. *Front Genet*. 2020;11:954. [CrossRef]
20. Radtke A, Von Brevern M, Neuhauser H, Hottenrott T, Lempert T. Vestibular migraine Long-term follow-up of clinical symptoms and vestibulocochlear findings. *Neurology*. 2012;79(15):1607-1614. [CrossRef]
21. Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo, and migrainous vertigo. *Neurology*. 2001;56(4):436-441. [CrossRef]
22. Pereira MF, Frejo L, Gallego-martinez A, et al. Differential proinflammatory signature in Vestibular Migraine and Meniere Disease. *Front Genet*. 2019;10:1-11. [CrossRef]
23. Radtke A, Neuhauser H, Von Brevern M, Hottenrott T, Lempert T. Vestibular migraine-validity of clinical diagnostic criteria. *Cephalalgia*. 2011;31(8):906-913. [CrossRef]
24. Sun W, Guo P, Ren T, Wang W. Magnetic resonance imaging of intratympanic gadolinium helps differentiate vestibular migraine from Ménière disease. *Laryngoscope*. 2017;127(10):2382-2388. [CrossRef]
25. Jen J, Kim GW, Baloh RW. Clinical spectrum of episodic ataxia type 2. *Neurology*. 2004;62(1):17-22. [CrossRef]
26. Pollak L, Klein C, Rafael S, Vera K, Rabey JM. Anxiety in the first attack of vertigo. *Otolaryngol Head Neck Surg*. 2003;128(6):829-834. [CrossRef]
27. Cohen JM, Escasena CA. Headache and dizziness: how to differentiate vestibular migraine from other conditions. *Curr Pain Headache Rep*. 2015;19(7):31. [CrossRef]
28. Neuhauser H, Radtke A, von Brevern M, Lempert T. Zolmitriptan for treatment of migrainous vertigo : a pilot randomized placebo-controlled trial. *Neurology*. 2003;60(5):882-883. [CrossRef]
29. Furman JM, Marcus DA, Balaban CD. Rizatriptan reduces vestibular-induced motion sickness in migraineurs. *J Headache Pain*. 2011;12(1):81-88. [CrossRef]
30. Cassano D, Pizza V, Busillo V. P074. Almotriptan in the acute treatment of Vestibular migraine: a retrospective study. *J Headache Pain*. 2015;16(Suppl 1):A114. [CrossRef]
31. Prakash S, Shah ND. Migrainous vertigo responsive to intravenous methylprednisolone: Case reports. *Headache*. 2009;49(8):1235-1239. [CrossRef]
32. Hunter BR, Wang AZ, Bucca AW, et al. Efficacy of benzodiazepines or antihistamines for patients with acute vertigo: a systematic review and meta-analysis. *JAMA Neurol*. 2022;79(9):846-855. [CrossRef]
33. Webster KE, Dor A, Galbraith K, et al. Pharmacological interventions for acute attacks of vestibular migraine. *Cochrane Database Syst Rev*. 2023;4(4):CD015322. [CrossRef]
34. Beh SC, Friedman DI. Acute vestibular migraine treatment with noninvasive vagus nerve stimulation. *Neurology*. 2019;93(18):e1715-e1719. [CrossRef]
35. Alghadir AH, Anwer S. Effects of vestibular rehabilitation in the management of a vestibular migraine: a review. *Front Neurol*. 2018;9:440. [CrossRef]
36. Lee YY, Yang YP, Huang PI, et al. Exercise suppresses COX-2 pro-inflammatory pathway in vestibular migraine. *Brain Res Bull*. 2015;116:98-105. [CrossRef]
37. Bayer O, Adrion C, Al Tawil A, Mansmann U, Strupp M. Results and lessons learnt from a randomized controlled trial: prophylactic treatment of vestibular migraine with metoprolol (PROVEMIG). *Trials*. 2019;20(1):813. [CrossRef]
38. Liu F, Ma T, Che X, Wang Q, Yu S. The efficacy of venlafaxine, flunarizine, and valproic acid in the prophylaxis of vestibular migraine. *Front Neurol*. 2017;8(OCT):1-5. [CrossRef]
39. Salviz M, Yuce T, Acar H, Karatas A, Acikalin RM. Propranolol and venlafaxine for vestibular migraine prophylaxis: a randomized controlled trial. *Laryngoscope*. 2016;126(1):169-174. [CrossRef]
40. Lepcha A, Amalanathan S, Augustine AM, Tyagi AK, Balraj A. Flunarizine in the prophylaxis of migrainous vertigo: a randomized controlled trial. *Eur Arch Otorhinolaryngol*. 2014;271(11):2931-2936. [CrossRef]
41. Domínguez-Durán E, Montilla-Ibáñez MA, Álvarez-Morujo de Sande MG, et al. Analysis of the effectiveness of the prophylaxis of vestibular migraine depending on the diagnostic category and the prescribed drug. *Eur Arch Otorhinolaryngol*. 2020;277(4):1013-1021. [CrossRef]
42. Teggi R, Colombo B, Gatti O, Comi G, Bussi M. Fixed combination of cinnarizine and dimenhydrinate in the prophylactic therapy of vestibular migraine: an observational study. *Neurol Sci*. 2015;36(10):1869-1873. [CrossRef]
43. Gode S, Celebisoy N, Kirazli T, et al. Clinical assessment of topiramate therapy in patients with migrainous vertigo. *Headache*. 2010;50(1):77-84. [CrossRef]
44. Salmito MC, Morganti LOG, Nakao BH, Simões JC, Duarte JA, Ganança FF. Vestibular migraine: comparative analysis between diagnostic criteria. *Braz J Otorhinolaryngol*. 2015;81(5):485-490. [CrossRef]

45. Çelebisoy N, Gökçay F, Karahan C, et al. Acetazolamide in vestibular migraine prophylaxis: a retrospective study. *Eur Arch Otorhinolaryngol*. 2016;273(10):2947-2951. [CrossRef]
46. Taghdiri F, Togha M, Razeghi Jahromi S, Refaeian F. Cinnarizine for the prophylaxis of migraine associated vertigo: a retrospective study. *SpringerPlus*. 2014;3(1):1-5. [CrossRef]
47. Mikulec AA, Faraji F, Kinsella LJ. Evaluation of the efficacy of caffeine cessation, nortriptyline, and topiramate therapy in vestibular migraine and complex dizziness of unknown etiology. *Am J Otolaryngol Head Neck Med Surg*. 2012;33(1):121-127. [CrossRef]
48. Baier B, Winkenwerder E, Dieterich M. "vestibular migraine": effects of prophylactic therapy with various drugs: a retrospective study. *J Neurol*. 2009;256(3):436-442. [CrossRef]
49. Bisdorff AR. Treatment of migraine related vertigo with lamotrigine an observational study. *Bull Soc Sci Med Grand Duche Luxemb*. 2004;2:103-108.
50. Yiannakis C, Hamilton L, Slim M, Kontorinis G. A systematic review and meta-analysis of prophylactic medication of vestibular migraine. *J Laryngol Otol*. 2023;137(9):953-961. [CrossRef]

Appendix Table 1. Characteristics of included studies

Study	Study Design	Sample Size	Key Findings
Lempert et al[1]	Consensus Document	N/A	Established diagnostic criteria for VM.
Vincent et al[2]	Classification System	N/A	Defined migraine-related vertigo under ICHD-3 classification.
Lempert et al[3]	Consensus Document	N/A	Updated and refined VM diagnostic criteria.
Teggi et al[4]	Cross-sectional Study	252	Identified VM phenotypes, family history influence, and clinical features.
Kang et al[5]	Cohort Study	81	Showed diagnostic value of video head impulse and caloric tests.
Shin et al[6]	Case Series	2	Demonstrated metabolic alterations in interictal and ictal phases of VM.
Obermann et al[7]	Case-Control Study	34	Showed central vestibular system involvement in VM pathophysiology.
Von Brevern et al[8]	Prospective Study	20	Described clinical and oculographic features of acute migrainous vertigo.
Huang et al[9]	Review	N/A	Reviewed pathophysiology and therapeutic advances in VM.
Colombo et al[10]	Prospective Study	252	Characterized VM patient profiles and its overlap with other disorders.
Morganti et al[11]	Cross-sectional Study	85	Epidemiological insights into VM prevalence and clinical aspects.
Zhang et al[12]	Cohort Study	67	Field testing of ICHD-3 criteria for VM, highlighting diagnostic accuracy.
Reploeg et al[13]	Retrospective Study	81	Analyzed patient characteristics and treatment options for migraine-associated dizziness.
Power et al[14]	Retrospective Study	90	Little consensus in choice of initial management and vestibular rehabilitation.
Furman et al[15]	Review	N/A	Developed a pathogenetic model linking migraine mechanisms to vestibular dysfunction.
Lee et al[16]	Case Report	1	Explored inner ear damage potential in migraine patients.
Marano et al[17]	Case-Control Study	20	Demonstrated trigeminal stimulation effects on vestibular function in VM patients.
Von Brevern et al[18]	Genetic Analysis	14	Identified no evidence that genetic mutations causing FHM and EA-2 represent major susceptibility loci for MV.
Paz-Tamayo et al[19]	Systematic Review	N/A	Reviewed prevalence studies and familial aggregation in VM cases.
Radtke et al[20]	Longitudinal Study	61	Long-term follow-up of VM patients, examining symptom progression and vestibulo-cochlear dysfunction.
Neuhauser et al[21]	Case-Control Study, Cohort Study	600	Investigated the correlation between migraine, vertigo, and VM.
Pereira et al[22]	Case-Control Study	277	Identified different proinflammatory markers in VM and Meniere's disease.
Radtke et al[23]	Validation Study	75	Validated diagnostic criteria for VM.
Sun et al[24]	Prospective Cohort Study	60	Demonstrated MRI with intratympanic gadolinium differentiates VM from MD.
Jen et al[25]	Genetic Analysis	27	Described clinical variability in genetically defined patients with episodic ataxia.
Pollak et al[26]	Cohort Study	65	Explored anxiety's role in vertigo.
Cohen et al[27]	Review	N/A	Reviewed differential diagnosis of VM.
Neuhauser et al[28]	RCT	19	Zolmitriptan showed moderate effectiveness in treating VM.
Furman et al[29]	RCT	25	Rizatriptan reduced vestibular-induced motion sickness in migraineurs.
Cassano et al[30]	Retrospective Cohort Study	26	Almotriptan improved vertigo and headache in VM patients.
Prakash et al[31]	Case Series	4	Methylprednisolone showed efficacy in treating VM in cases with prolonged or frequent episodes.
Hunter et al[32]	Systematic Review & Meta-analysis	17 RCT (1586 patients)	Single-dose antihistamines provided greater vertigo relief at 2 hours compared to single-dose benzodiazepines
Webster et al[33]	Cochrane Review	Systematic Review	The evidence is very uncertain about the effectiveness of triptans in improving vertigo symptoms
Beh et al[34]	Retrospective chart review.	18	Noninvasive vagus nerve stimulation reduced vertigo symptoms in VM patients.
Alghadir et al[35]	Review	Review-based	VR may improve vestibular symptoms and reduce headache-related disability
Lee et al[36]	Experimental Study	Lab-based	Exercise reduced COX-2 inflammation associated with VM.

Appendix Table 1. Characteristics of included studies

Study	Study Design	Sample Size	Key Findings
Bayer et al[37]	RCT	130	No significant benefit of metoprolol over placebo in reducing vertiginous attacks.
Liu et al[38]	Retrospective analysis	61	Effectiveness of venlafaxine, flunarizine, and valproic acid in preventing vestibular migraine episodes
Salviz et al[39]	RCT	64	Propranolol and venlafaxine equally effective for vestibular migraine symptoms.
Lepcha et al[40]	RCT	48	Flunarizine reduced frequency and severity of vertigo attacks.
Domínguez-Durán et al[41]	Multicenter prospective study	31	Evaluated VM prophylaxis effectiveness based on diagnostic categories and drug types.
Teggi et al[42]	Observational Study	22	Combination of cinnarizine and dimenhydrinate improved VM symptoms.
Gode et al[43]	RCT	30	Topiramate was effective in reducing vertigo and migraine frequency.
Salmito et al[44]	Comparative Analysis	94	Neuhauser criteria and the Bárány Society criteria are reliable for VM diagnosis.
Çelebisoy et al[45]	Retrospective Study	39	Acetazolamide showed benefits in VM prophylaxis.
Taghdiri et al[46]	Retrospective Study	40	Cinnarizine reduced migraine-associated vertigo.
Mikulec et al[47]	Therapeutic Evaluation	44	Caffeine cessation, nortriptyline, and topiramate improved VM symptoms.
Baier et al[48]	Retrospective Study	100	Benefits of various prophylactic drugs for VM treatment
Bisdorff et al[49]	Observational Study	19	Lamotrigine showed promising effects in migraine-related vertigo.
Yiannakis et al[50]	Systematic Review & Meta-analysis	Meta-analysis	Only propranolol significantly improved Vertigo Symptom Scale scores