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A NARRATIVE REVIEW OF VESTIBULAR MIGRAINE: INTEGRATING NEUROLOGY AND OTOLARYNGOLOGY PERSPECTIVES

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ABSTRAK

Abstract:
Vestibular migraine (VM) is a common but underrecognized cause of recurrent vertigo and dizziness at the interface of neurology and otolaryngology. This structured narrative review integrates both perspectives in VM diagnosis and management. A PubMed search was conducted on 13 May 2026 using keywords related to vestibular migraine, vertigo, dizziness, migraine, diagnostic criteria, otolaryngology, neurology, Ménière's disease, vestibular rehabilitation, CGRP, treatment, and management; reference checking added one key consensus document, yielding 46 articles for synthesis. VM remains a clinical, criterion-based, and longitudinal diagnosis without a pathognomonic biomarker. Neurology emphasizes migraine features and central warning signs, whereas otolaryngology emphasizes vertigo phenomenology, auditory symptoms, audiovestibular testing, and differential diagnosis. Management is individualized and multimodal. Integrated neuro-otological reasoning may improve diagnostic accuracy and patient-centered care, but stronger VM-specific trials with standardized vestibular outcomes are needed.

***Abstrak*:**
Migrain vestibular merupakan penyebab vertigo dan dizziness berulang yang sering tetapi masih kurang dikenali, serta berada pada irisan neurologi dan otolaringologi. Tinjauan naratif terstruktur ini mengintegrasikan kedua perspektif tersebut dalam diagnosis dan penatalaksanaan migrain vestibular. Pencarian PubMed dilakukan pada 13 Mei 2026 menggunakan kata kunci terkait vestibular migraine, vertigo, dizziness, migraine, diagnostic criteria, otolaryngology, neurology, Ménière's disease, vestibular rehabilitation, CGRP, treatment, dan management; penelusuran referensi menambahkan satu dokumen konsensus utama, sehingga 46 artikel disintesis. Migrain vestibular tetap merupakan diagnosis klinis, berbasis kriteria, dan longitudinal tanpa biomarker patognomonik. Neurologi menekankan fitur migrain dan tanda bahaya sentral, sedangkan otolaringologi menekankan fenomenologi vertigo, gejala auditorik, pemeriksaan audiovestibular, dan diagnosis banding. Penatalaksanaan bersifat individual dan multimodal. Integrasi penalaran neuro-otologis dapat meningkatkan akurasi diagnosis dan pelayanan berpusat pada pasien, tetapi uji klinis khusus migrain vestibular dengan luaran vestibular terstandar masih diperlukan.

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INTRODUCTION

Vestibular symptoms such as vertigo, dizziness, and imbalance are common complaints in clinical practice and may arise from a wide range of neurological and otolaryngological disorders. Among these, vestibular migraine (VM) has gained increasing recognition as an important cause of episodic vestibular symptoms, particularly in patients with a history of migraine [1], [2], [3]. Despite its relatively high prevalence, VM remains underdiagnosed due to its variable clinical presentation and the absence of specific diagnostic biomarkers [4], [5].

VM is characterized by recurrent vestibular symptoms temporally associated with migraine features, although headache may be absent during some attacks [3], [5], [6]. The condition frequently overlaps with other vestibular disorders, especially Ménière's disease, leading to diagnostic uncertainty and delayed treatment. The complexity of its presentation and the lack of objective diagnostic tools highlight the need for careful clinical evaluation [3], [6].

Although VM has been increasingly discussed in recent literature, several important gaps remain. First, previous reviews have often described VM either as a migraine-spectrum disorder or as a cause of recurrent vertigo, but the complementary roles of neurology and otolaryngology in diagnosis and management are not always clearly integrated [3], [6], [7]. Second, although international diagnostic criteria are shared across specialties, their clinical application may differ: neurologists tend to emphasize migraine history, sensory hypersensitivity, central mechanisms, and migraine-directed treatment, whereas otolaryngologists and neuro-otologists often focus on vertigo characterization, audiovestibular examination, and differentiation from Ménière's disease, benign paroxysmal positional vertigo (BPPV), and other vestibular disorders [8], [9], [10]. Third, the evidence supporting VM management remains heterogeneous, with many pharmacological recommendations extrapolated from migraine headache studies rather than VM-specific trials [7], [11].

Therefore, this narrative review aims to integrate neurological and otolaryngological perspectives in evaluating current evidence on VM, with particular emphasis on diagnostic criteria, audiovestibular assessment, differential diagnosis, and pharmacological and non-pharmacological management. This review specifically asks how these two complementary perspectives can improve diagnostic accuracy and individualized management, and what limitations remain in the current evidence base.

RESEARCH METHOD

This article was designed as a structured narrative review of vestibular migraine from neurological and otolaryngological perspectives. An updated literature search was conducted in PubMed on 13 May 2026 using the following keywords: "vestibular migraine," "vertigo," "dizziness," "migraine," "diagnosis," "diagnostic criteria," "otolaryngology," "neurology," "Ménière's disease," "vestibular rehabilitation," "CGRP," "treatment," and "management." PubMed was selected as the primary biomedical database relevant to neurology, otolaryngology, and vestibular medicine. Reference checking was also performed to identify key consensus documents not captured by the database search.

Records were excluded if they were not directly related to VM, were general vestibular or vertigo literature with limited relevance to VM, were duplicative or lower-priority compared with more recent or higher-level evidence, focused on less central differential-diagnosis topics, were not suitable for an English-language review, or addressed narrow or special-population topics outside the main clinical focus of this review. When multiple articles addressed substantially overlapping topics, priority was given to studies with higher methodological

relevance, greater clinical applicability, more recent publication, and more direct contribution to the review questions. The records were screened by title, citation details, and relevance to the review themes. Articles were selected based on their relevance to epidemiology, pathophysiology, clinical manifestations, diagnosis, differential diagnosis, pharmacological and non-pharmacological management, and special clinical considerations. Evidence was qualitatively appraised according to study design, directness to vestibular migraine, recency, and clinical applicability, with greater interpretive weight given to consensus documents, clinical guidelines, systematic reviews, and randomized or interventional studies.

RESULT AND ANALYSIS

The literature selection process is summarized in Figure 1.

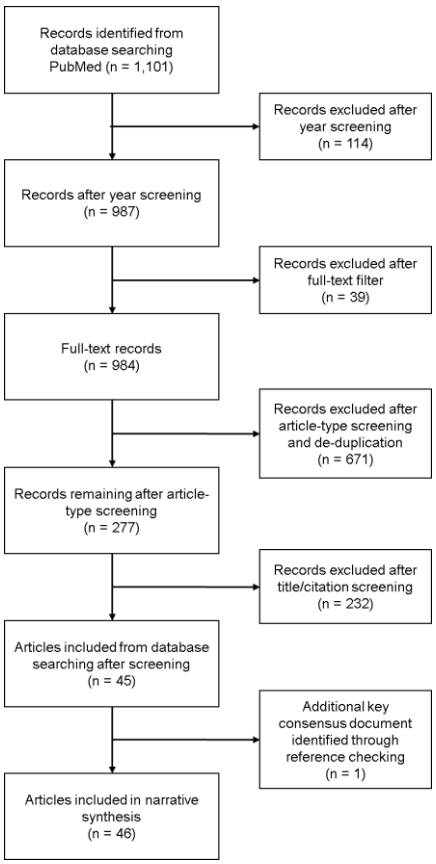

Figure 1. Literature selection flow diagram.

The initial keyword search identified 1,101 records. After applying the publication-year filter from 2014 to 2026, 987 records remained. The full-text filter was then applied, resulting in 948 records. Subsequent article-type screening yielded 277 records. No duplicate records were identified among the PubMed results. After title and citation screening, 45 articles from PubMed were included. One additional key consensus document was retained through reference checking, resulting in 46 articles included in the final narrative synthesis.

DISCUSSION

1. Epidemiology

VM is increasingly recognized as a common cause of recurrent episodic vestibular symptoms, although prevalence estimates vary by setting, diagnostic criteria, and inclusion of probable VM. Population-based and epidemiological studies support an estimated general-population prevalence of approximately 1%–3%, whereas dizziness, headache, and neuro-otology clinics report higher proportions because of referral enrichment [4], [12], [13], [14]. Clinic-based figures should therefore be interpreted as evidence of clinical burden rather than as population prevalence.

Across the included literature, VM consistently shows female predominance and often emerges after the onset of conventional migraine. This temporal dissociation is clinically important because headache may no longer be the dominant complaint when patients present with dizziness or vertigo in otolaryngology, emergency, or primary-care settings [2], [5], [14], [15], [16]. As a result, VM is frequently recognized late or misattributed to nonspecific dizziness, anxiety, BPPV, or Ménière's disease.

The clinical significance of VM extends beyond vertigo frequency. Patients may experience work impairment, activity restriction, visual-motion avoidance, reduced confidence in balance, fear of

falling, fatigue, and anxiety or depressive symptoms [1], [13], [17], [18]. These features may interact with migraine sensory hypersensitivity, vestibular compensation, and persistent postural-perceptual dizziness (PPPD), supporting the view of VM as a disabling neuro-otological disorder requiring coordinated assessment.

Epidemiological evidence remains limited by heterogeneous case definitions, retrospective symptom recall, older diagnostic criteria in some studies, and referral bias in specialty cohorts [13], [14]. Prevalence should therefore be reported as a range linked to the population studied and diagnostic method used, rather than generalized from clinic-based samples to the wider population.

2. Pathophysiology

The pathophysiology of VM is best conceptualized as a multilevel disorder of trigemino-vestibular and multisensory network regulation rather than as an exclusively central or peripheral vestibular disease. Migraine biology provides the principal mechanistic framework: trigeminovascular activation, neurogenic inflammation, serotoninergic modulation, and calcitonin gene-related peptide (CGRP) signaling may influence vestibular nuclei, vestibulo-thalamo-cortical circuits, and inner-ear structures [3], [19], [20], [21].

CGRP is particularly relevant because it links migraine neurobiology with vestibular and cochlear physiology. Its expression in trigeminal and vestibular pathways provides biological plausibility for dizziness, motion sensitivity, and aural symptoms in patients whose headache is absent or secondary [3], [20], [22], [23]. However, CGRP involvement should be interpreted as pathway support rather than as a validated diagnostic biomarker or proof that all VM phenotypes share a single molecular driver.

Thalamocortical and cerebellar network dysfunction offers a complementary explanation for visual-motion sensitivity, abnormal sensory weighting, spatial disorientation, and prolonged motion intolerance. Functional and clinical reviews implicate the parieto-insular vestibular cortex, temporoparietal regions, cerebellar modulation, and brainstem velocity-storage mechanisms [3], [12], [21], [24]. This network model also explains why bedside and laboratory vestibular findings may be variable: abnormalities may reflect attack state, compensation, comorbidity, or nonspecific sensory gain rather than a disease-specific marker.

Peripheral cochleovestibular involvement remains biologically plausible but incompletely resolved. Auditory symptoms, caloric weakness, vestibular evoked myogenic potential (VEMP) abnormalities, and video head impulse test (vHIT) or nystagmus findings have been reported in subsets of patients [25], [26], [27], [28], [29]. The available systematic-review evidence supports possible otolith or vestibular-pathway involvement, but methods and normative values vary substantially, limiting direct diagnostic utility.

The main controversy is therefore not whether VM is central or peripheral, but how migraine-network dysregulation interacts with vestibular end organs, visual dependence, and psychological threat monitoring. Current pathophysiological evidence is largely associative, often based on small samples, narrative synthesis, experimental models, or group-level imaging and physiological differences [3], [19], [20], [28]. No mechanism has yet been validated as a biomarker capable of confirming VM in an individual patient.

3. Clinical Manifestations

VM presents with a broad spectrum of vestibular symptoms, including spontaneous internal or external vertigo,

positional vertigo, visually induced vertigo, head-motion-induced vertigo, and head-motion-induced dizziness with nausea. Patients may also describe rocking, swaying, floating, tilting, spatial disorientation, imbalance, or a sensation of being intoxicated [1], [5], [8], [13], [24]. The diagnostic challenge is that the symptom label used by the patient is often less informative than timing, triggers, associated migraine features, auditory course, and longitudinal recurrence.

Attack duration typically falls within the consensus window of 5 minutes to 72 hours, although brief recurrent symptoms can occur during a longer attack period. Headache is not obligatory during every vestibular episode. Photophobia, phonophobia, visual aura, nausea, motion sensitivity, osmophobia, family history, menstrual association, and behavioral avoidance of light or sound may provide the strongest link to migraine biology [8], [12], [15], [16].

Aural symptoms are frequent but diagnostically nonspecific. Tinnitus, aural fullness, otalgia, hyperacusis, and subjective hearing fluctuation may occur in VM and often drive otolaryngology referral [9], [24], [25]. Nevertheless, progressive unilateral low- or mid-frequency sensorineural hearing loss documented by serial audiometry should redirect the differential toward Meniere's disease or VM-Meniere overlap rather than being attributed automatically to VM.

Interictal symptoms also matter. Many patients experience visual dependence, motion intolerance, imbalance, cognitive fatigue, residual dizziness, anxiety, depressive symptoms, or avoidance between attacks [17], [18], [30]. These features can reflect chronic VM, incomplete vestibular compensation, PPPD, peripheral comorbidity, or maladaptive threat monitoring. The review therefore treats them as clinically meaningful modifiers of disability rather than as reasons to dismiss the vestibular syndrome as psychogenic.

4. Diagnosis

Diagnosis of VM is clinical, criterion based, and longitudinal. The central diagnostic task is not merely to identify the coexistence of migraine and dizziness, but to determine whether recurrent vestibular episodes fulfill accepted VM criteria and are not better explained by another vestibular, otological, neurological, or systemic disorder. This distinction is important because VM has no pathognomonic biomarker; normal interictal vestibular testing does not exclude VM, whereas abnormal caloric, vHIT, VEMP, ocular-motor, or audiometric findings do not independently confirm it [8], [10], [13].

The Bárány Society and International Headache Society criteria remain the diagnostic anchor. These criteria define VM by recurrent vestibular symptoms of moderate or severe intensity lasting 5 minutes to 72 hours, a current or previous history of migraine, migraine features during at least half of vestibular episodes, and exclusion of a better vestibular or headache diagnosis [8]. Probable VM is used when vestibular-episode criteria are met but only one migraine-link element is present. The criteria are summarized in Table 1.

A structured history is therefore the most important diagnostic tool. Clinicians should reconstruct the chronology of vestibular, migraine, auditory, neurological, and contextual features, including attack onset, duration, frequency, recovery, spontaneous versus triggered symptoms, positional or visual provocation, headache timing, photophobia, phonophobia, aura, motion sickness, family history, tinnitus, hearing fluctuation, vascular risk, and persistent interictal dizziness [1], [10], [13], [31]. A history that links recurrent vestibular episodes to migraine biology remains more diagnostically informative than any isolated laboratory abnormality.

Table 1.
Diagnostic criteria of vestibular migraine [8].

Criterion	Vestibular Migraine (ICHD-3 & ICVD)	Probable Vestibular Migraine (ICVD)
A	At least five episodes fulfilling criteria C and D	At least five episodes of vestibular symptoms of moderate or severe intensity lasting 5 minutes to 72 hours
B	Current or past history of migraine with or without aura	Only one of the following criteria is met: history of migraine or migraine features during episodes
C	Vestibular symptoms of moderate or severe intensity lasting 5 minutes to 72 hours	Not better accounted for by another vestibular disorder or ICHD diagnosis
D	At least 50% of episodes associated with one or more migraine features: • Headache with ≥2 of: unilateral location, pulsating quality, moderate/severe intensity, aggravation by routine activity • Photophobia and phonophobia • Visual aura	
E	Not better accounted for by another ICHD-3 diagnosis or vestibular disorder	

Neurological and otolaryngological perspectives are complementary. From the neurological perspective, VM is approached as a migraine-spectrum disorder requiring assessment of migraine history, aura, sensory hypersensitivity, headache chronology, medication exposure, comorbid anxiety or depression, and central warning signs. From the otolaryngological and neuro-otological perspective, VM is approached as a recurrent vestibular syndrome requiring precise characterization of vertigo phenomenology, positional triggers, nystagmus, auditory symptoms, hearing trajectory, and vestibular function [10], [18], [26], [31]. These perspectives use the same diagnostic criteria, but they emphasize different clinical risks and differential diagnoses.

Vestibular and audiological assessment plays an important role in the diagnostic evaluation of VM, not because it confirms the disorder independently, but because it helps characterize the vestibular phenotype, refine the differential diagnosis, and identify alternative or coexisting vestibular and cochlear disorders. This is particularly relevant in distinguishing VM from Ménière's disease, where overlap in vertigo, tinnitus, aural fullness, and migraine-associated symptoms may create substantial diagnostic uncertainty [9]. Bedside examination and selected laboratory testing may reveal supportive but non-pathognomonic findings, especially in ocular-motor and vestibulo-ocular responses [26], [27].

Nystagmus assessment remains clinically useful during symptomatic periods. VM may be associated with spontaneous, positional, or gaze-related nystagmus, but the pattern is variable and should not be interpreted as disease-specific. Positional symptoms are especially important because VM may mimic or coexist with benign paroxysmal positional vertigo, whereas atypical or persistent positional nystagmus should prompt broader neuro-otological interpretation rather than automatic attribution to BPPV [26], [32]. Thus, ocular-motor findings are best regarded as diagnostic clues that must be integrated with attack chronology, migraine features, auditory symptoms, and exclusion of competing diagnoses.

Vestibular function testing can further refine the diagnostic probability. Caloric testing may identify unilateral vestibular weakness, while vHIT evaluates high-frequency vestibulo-ocular reflex gain and corrective saccades; however, neither

test can confirm VM on its own [26], [27]. In the specific differential diagnosis between VM and Ménière's disease, vHIT findings may contribute to interpretation, but available evidence indicates that vHIT gain alone is insufficient as a definitive discriminator [29]. VEMP testing can assess otolith pathway involvement, and abnormalities have been reported in VM, but systematic-review evidence shows substantial heterogeneity in methods, stimulus parameters, normative values, and diagnostic thresholds, limiting its standalone diagnostic utility [28].

Audiological evaluation is essential when Ménière's disease is part of the differential diagnosis. VM may be accompanied by tinnitus, aural fullness, subjective hearing fluctuation, or other auditory symptoms, but these features are nonspecific and do not establish Ménière's disease or VM by themselves [3], [9]. The most clinically useful discriminator is the hearing trajectory: progressive or fluctuating unilateral sensorineural hearing loss favors Ménière's disease, whereas VM more often shows normal hearing or nonprogressive, nonspecific auditory findings [9]. Accordingly, serial audiometry is more informative than a single audiogram when the boundary between VM and Ménière's disease remains uncertain.

Taken together, vestibular and audiological examinations should be used to support differential diagnosis, document comorbidity, and guide longitudinal follow-up rather than to replace consensus diagnostic criteria. A normal vestibular test battery does not exclude VM, and an abnormal vestibular or audiological result does not confirm it independently. The most accurate diagnosis arises from integrating history, migraine features, bedside neuro-otological findings, audiometry, targeted vestibular testing, and follow-up over time [9], [26], [27], [32].

Imaging should be targeted rather than routine. Brain or internal auditory canal MRI and vascular imaging are appropriate when there are red flags such as first or atypical severe attacks, focal neurological deficits, severe gait impairment, new hearing loss, vascular risk, structural signs, or suspected posterior-circulation ischemia [10], [33]. Imaging may exclude mimics, but no MRI, PET, or hydrops-oriented finding is currently sufficient to diagnose VM.

Table 2 summarizes how neurological and otolaryngological perspectives contribute complementary diagnostic tasks in VM without creating separate diagnostic criteria. Finally, treatment response should not be used as a formal diagnostic criterion. Improvement after migraine-directed therapy may support clinical plausibility, but placebo response, spontaneous fluctuation, vestibular compensation, and nonspecific medication effects can influence outcomes. Therefore, response to treatment should be interpreted only as part of longitudinal reassessment, not as a substitute for consensus criteria, differential diagnosis, and targeted audiovestibular evaluation [34], [35].

Table 2.
Complementary neurological and otolaryngological contributions to VM diagnosis.

Diagnostic dimension	Neurology perspective	Otolaryngology / neuro-otology perspective	Integrated diagnostic value
Clinical framing and criteria application	Evaluates migraine history, aura, photophobia, phonophobia, sensory hypersensitivity, headache chronology, and central warning signs.	Characterizes vertigo phenomenology, positional or visual triggers, nystagmus, auditory symptoms, hearing trajectory, and vestibular function.	Applies the same VM criteria while linking vestibular episodes to migraine biology and avoiding premature classification as isolated headache or peripheral

			vertigo.
Differential diagnosis	Prioritizes posterior-circulation ischemia, migraine with brainstem aura, episodic ataxia, demyelinating disease, medication-related dizziness, and systemic neurological causes.	Prioritizes Ménière's disease, BPPV, vestibular neuritis, vestibular hypofunction, third-window syndromes, and cochlear disorders.	Reduces diagnostic error by addressing both dangerous neurological mimics and treatable otological disorders.
Audiovestibular and imaging strategy	Uses focused neurological examination and targeted imaging when red flags or vascular risk are present.	Uses audiometry, positional testing, VNG, caloric testing, vHIT, and VEMP when clinically indicated.	Uses investigations to refine diagnostic probability, document comorbidity, and exclude mimics rather than confirm VM independently.
Longitudinal reassessment	Monitors migraine phenotype, neurological evolution, medication effects, and response to treatment as supportive—not diagnostic—information.	Monitors serial audiometry, recurrent positional vertigo, vestibular compensation, hearing trajectory, and rehabilitation needs.	Revises the diagnosis when the clinical course, hearing pattern, vestibular findings, or functional trajectory becomes inconsistent with the initial impression.

5. Differential Diagnosis

The differential diagnosis of VM is broad because vestibular symptoms are shared across otological, neurological, functional, vascular, and systemic disorders. The safest approach is not to search for a single pathognomonic sign, but to integrate timing, triggers, migraine linkage, auditory trajectory, ocular-motor findings, vascular risk, and longitudinal follow-up [10], [13], [32], [36].

Meniere's disease is the most important otological mimic. Both VM and Meniere's disease may present with recurrent vertigo, nausea, tinnitus, aural fullness, and migraine-like sensory features. The strongest discriminator is audiometrically documented, fluctuating and progressive unilateral low- or mid-frequency sensorineural hearing loss associated with vertigo attacks [9], [37]. Vestibular testing may support the distinction, but vHIT or caloric findings alone are insufficient to settle the diagnosis [29].

BPPV should be considered when vertigo is brief, stereotyped, and position triggered. Canal-specific nystagmus on Dix-Hallpike or supine roll testing supports BPPV, whereas VM-related positional symptoms may be longer, atypical, recurrent in different positions, or associated with photophobia, phonophobia, visual sensitivity, or migraine history [13], [36]. The two conditions can coexist, so persistent symptoms after appropriate canalith repositioning should prompt reassessment for VM, PPPD, or another disorder.

PPPD is characterized by persistent nonspinning dizziness or unsteadiness on most days for at least three months, worsened by upright posture, active or passive movement, and complex visual environments. VM can precipitate or coexist with PPPD; therefore, episodic migraine-linked attacks and persistent perceptual dizziness should be assessed separately [30]. This distinction is clinically useful because migraine prevention, vestibular rehabilitation, and behavioral strategies may all be required.

Posterior-circulation transient ischemic attack or stroke must be prioritized in older patients, those with vascular risk, sudden first attacks, focal neurological deficits, severe gait disturbance, central ocular-motor signs, new hearing loss, or neck pain [10], [33]. A history of migraine should not be allowed to create false reassurance in a

high-risk acute vestibular syndrome. Other mimics include vestibular neuritis or labyrinthitis, migraine with brainstem aura, episodic ataxia, vestibular paroxysmia, post-concussive dizziness, orthostatic disorders, third-window syndromes, medication effects, and systemic disease [13], [32], [36]. Because comorbidity is common, the final diagnosis may be layered rather than singular.

6. Management

Management of VM should be individualized, multimodal, and phenotype driven. The treatment literature is clinically active but methodologically uneven; therefore, direct VM-specific evidence should be separated from recommendations extrapolated from conventional migraine studies [11], [38], [39], [40]. Outcomes should also be tracked separately for vestibular symptoms and headache, because improvement in migraine headache does not necessarily prove improvement in vestibular disability [11], [34].

Education, Lifestyle Measures, and Trigger Management

Education is a core intervention. Patients should be told that VM is a biological migraine-spectrum vestibular disorder, that headache may be absent during attacks, and that normal interictal vestibular testing does not invalidate the diagnosis [1], [15], [18]. Regular sleep, hydration, scheduled meals, graded exercise, stress regulation, and individualized trigger identification are reasonable low-risk foundations.

A prospective lifestyle-modification study reported improvement after a bundled program involving sleep regulation, meals, exercise, and dietary-trigger avoidance, with sleep adherence appearing particularly relevant [41]. Because the study was uncontrolled and several components changed simultaneously, it supports feasibility and clinical plausibility rather than proof of a single effective element. Excessive trigger restriction should be avoided because it may increase hypervigilance and disability.

Vestibular Rehabilitation, Exercise, and Behavioral Strategies

Vestibular rehabilitation therapy (VRT) is most relevant for persistent imbalance, visual-motion sensitivity, reduced balance confidence, incomplete compensation, or PPPD overlap. Programs may include gaze stabilization, habituation, balance and gait training, sensory reweighting, and graded visual exposure [42], [43]. Current evidence supports VRT as part of multimodal management, but heterogeneity in protocols, outcome measures, and patient selection limits certainty.

The randomized trial by Sun et al. reported greater improvement with supervised resistance exercise than progressive muscle relaxation in dizziness handicap, vertigo severity, anxiety, depression, and attack frequency [44]. This trial is clinically promising, but its interpretation should be tempered by single-center design, difficulty of participant blinding, per-protocol analysis, uncertain concomitant prophylactic medication effects, and limited generalizability to broader VM populations. These limitations respond directly to the need for more critical appraisal rather than simple presentation of positive results.

Behavioral strategies, including cognitive-behavioral approaches, are particularly useful when avoidance, catastrophic interpretation, visual dependence, anxiety, depression, or PPPD is present [18], [30]. These interventions should be framed as treatment of disability-maintaining mechanisms and comorbidity, not as evidence that VM is psychogenic.

Acute or Abortive Therapy

Acute or abortive therapy aims to reduce vertigo, nausea, vomiting, headache, motion sensitivity, and functional impairment during VM attacks. Unlike conventional migraine headache, however,

acute VM treatment has limited direct evidence. The Cochrane review on pharmacological interventions for acute VM attacks found very limited and low-certainty evidence, and the 2025 randomized trial of rizatriptan did not demonstrate clinically useful 1-hour relief of vertigo or unsteadiness/dizziness compared with placebo; fatigue and drowsiness were more common with rizatriptan [34], [45]. Therefore, acute treatment should distinguish relief of migraine headache from relief of vestibular symptoms.

Triptans may still be considered when conventional migraine headache accompanies VM attacks and there are no contraindications, but they should not be presented as established rapid vestibular-specific therapy. Earlier treatment reviews discussed triptans such as zolmitriptan, sumatriptan, and rizatriptan for VM attacks, but the newer randomized rizatriptan evidence requires a more cautious interpretation [34], [40], [45]. In practice, response should be evaluated at the attack level, including vertigo severity, nausea, functional recovery, headache relief, rescue-medication use, and adverse effects. Symptom-directed medications remain clinically relevant. Antiemetics and antidopaminergic agents such as metoclopramide, promethazine, and prochlorperazine may be used for nausea and vomiting during acute attacks. Antihistamines such as dimenhydrinate, meclizine, or diphenhydramine may be considered for milder vertigo, motion sickness, or acute vestibular distress, although sedation and anticholinergic effects may limit tolerability [40]. Benzodiazepines may provide short-term vestibular suppression in severe or prolonged attacks, but frequent or prolonged use should be avoided because of sedation, imbalance, falls risk, dependence, and potential interference with vestibular compensation [11], [40].

Intravenous methylprednisolone has been reported for selected severe or persistent migrainous vertigo episodes, but the evidence remains case-level and should not be interpreted as routine acute therapy [40]. Severe, first, atypical, prolonged, or neurologically concerning attacks should prompt reassessment for posterior-circulation ischemia, acute hearing loss, vestibular neuritis, metabolic illness, dehydration, or another competing diagnosis rather than reflexive symptomatic treatment alone [10], [33]. Representative acute symptom-directed pharmacological options for VM are summarized in Table 3.

Table 3.
Drugs used for acute therapy in vestibular migraine

Drug Class	Examples	Potential acute role	Evidence boundary and precautions
Triptans	Zolmitriptan, sumatriptan, rizatriptan	Migraine-headache–dominant VM attacks in selected patients without contraindications	VM-specific vestibular benefit is limited; rizatriptan did not show clinically useful 1-hour vertigo or unsteadiness/dizziness relief in a recent RCT [34]
Antiemetics / antidopaminergics	Metoclopramide, promethazine, prochlorperazine	Nausea and vomiting during acute attacks	Symptom-directed rather than VM-specific therapy; monitor for sedation, hypotension, extrapyramidal effects, and QT prolongation
Antihistamines / vestibular suppressants	Dimenhydrinate, meclizine, diphenhydramine	Short-term relief of milder vertigo, motion sickness, or acute vestibular distress	May cause drowsiness, dry mouth, blurred vision, constipation, and impaired balance; avoid frequent or prolonged use
Benzodiazepines	Diazepam,	Short-term rescue for	Risk of sedation, imbalance, falls,

	lorazepam, clonazepam	severe vertigo or marked acute vestibular distress	dependence, and impaired vestibular compensation; use should be limited
Corticosteroids	Intravenous methylprednisolone	Selected severe or prolonged episodes reported in limited evidence	Case-level evidence only; not routine acute VM therapy; reassess atypical or persistent attacks for alternative diagnoses

Preventive Pharmacotherapy

Preventive therapy is considered for frequent, prolonged, disabling, or functionally disruptive VM attacks. Common options include beta-blockers, calcium-channel agents such as flunarizine where available, tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors, topiramate, lamotrigine, acetazolamide, and sodium valproate [11], [38], [40], [46]. Choice should be driven by comorbidity, contraindications, reproductive safety, adverse-effect profile, and patient preference rather than by unqualified ranking. Commonly used preventive pharmacological options and prescribing considerations are summarized in Table 4.

Table 4.
Commonly used preventive treatment options for vestibular migraine

Drug	Dose	Potential clinical use	Prescribing considerations
Propranolol	40–240 mg/day	Migraine prevention; useful when hypertension or tremor coexists	Fatigue, hypotension, depression, bronchospasm; caution in asthma or bradycardia
Metoprolol	50–200 mg/day	Migraine prevention; useful when hypertension or tachycardia coexists	Fatigue, hypotension, depression; VM-specific RCT evidence is not definitive
Topiramate	50–100 mg/day	Frequent migraine attacks; migraine-dominant phenotype; obesity	Paresthesia, cognitive dysfunction, weight loss; reproductive safety concerns
Amitriptyline	50–100 mg/day	Sleep disturbance, chronic pain, frequent migraine symptoms	Sedation, weight gain, dry mouth, constipation
Nortriptyline	25–75 mg/day	Sleep disturbance or intolerance to amitriptyline	Similar to amitriptyline, often better tolerated
Acetazolamide	250–750 mg/day	Selected aura-dominant or episodic ataxia-like features	Paresthesia, nausea, hypokalemia; VM evidence remains limited
Lamotrigine	25–100 mg/day	Selected vertigo-dominant or aura-associated symptoms	Dizziness, diplopia, headache; rash risk
Valproate	600–900 mg/day	Selected refractory migraine cases	Weight gain, metabolic effects, teratogenicity; avoid in pregnancy potential whenever possible
Venlafaxine / SNRIs	Variable	Comorbid anxiety, depression, PPPD-overlap, chronic dizziness	Nausea, headache, sexual dysfunction; monitor blood pressure
Flunarizine	5–10 mg/day	Frequent vertigo-dominant attacks where available	Weight gain, sedation, depression, reversible parkinsonism
CGRP monoclonal antibodies: erenumab, galcanezumab, fremanezumab, eptinezumab	Variable by agent	Selected refractory or frequent disabling VM, especially when conventional therapy fails or is poorly tolerated	Injection-site reactions, cost/access issues; long-term VM-specific comparative evidence remains limited
Botulinum toxin A	Usually considered in chronic migraine protocols	Selected refractory cases, especially with chronic migraine phenotype	Injection-site pain, cost; VM-specific evidence remains limited

Note: Doses are representative ranges reported in VM or migraine literature and should be individualized according to comorbidities, contraindications, pregnancy potential, local prescribing guidance, availability, and tolerability. Evidence quality differs across agents; several recommendations remain extrapolated from migraine headache studies or supported by small, heterogeneous VM-specific studies.

Several trial findings require calibrated interpretation. In PROVEMIG, metoprolol did not outperform placebo, although premature termination and missing diaries limit certainty [35]. Flunarizine reduced vertigo frequency and severity in a small randomized study but had open-label and short-duration limitations [47]. Propranolol and venlafaxine both improved vestibular outcomes in a randomized comparative study without placebo, and venlafaxine showed greater improvement in depressive symptoms, supporting comorbidity-guided selection [48].

Systematic reviews and meta-analyses support potential benefit across several preventive treatments, but conclusions are constrained by small numbers of VM-specific trials, heterogeneous criteria, indirect comparisons, and inconsistent vestibular outcomes [39], [46], [49], [50]. Therefore, preventive treatment should be described as evidence-informed but still limited by low-to-moderate certainty for many agents.

CGRP-Directed and Emerging Therapies

CGRP-directed therapy is the most important recent mechanism-based advance in VM management. The INVESTMENT placebo-controlled pilot trial found greater improvement with galcanezumab than placebo in VM-PATHI, Dizziness Handicap Inventory (DHI), definite dizzy days, and responder outcomes [51]. A prospective anti-CGRP monoclonal-antibody cohort also reported substantial reductions in vestibular days, headache days, and disability, but its uncontrolled design may overestimate efficacy [23].

Rapid and network evidence syntheses suggest that CGRP blockade is promising, particularly for refractory or frequent VM, but the evidence does not yet establish universal first-line use, class equivalence, long-term safety in VM, cost-effectiveness, or superiority over conventional preventives [22], [50]. Botulinum toxin may be relevant when chronic migraine headache is prominent, but vestibular-specific benefit remains less certain [11], [38]. Gepants and neuromodulation remain mechanistically interesting but require dedicated VM trials before they are presented as established VM-specific therapies [11], [38], [46].

Therapy in Pregnancy and Lactation

Pregnancy and lactation require a separate safety framework because VM-specific pregnancy trials are lacking and many recommendations are extrapolated from migraine and obstetric pharmacology. Non-pharmacological measures, hydration, regular meals, sleep support, vestibular rehabilitation, and careful review of medication necessity should be prioritized [52].

For acute attacks, paracetamol is generally preferred, while nonsteroidal anti-inflammatory drugs require gestational-age restrictions and obstetric review. Sumatriptan has the most pregnancy safety data among triptans and may be considered when clinically necessary. For prevention, propranolol and amitriptyline are commonly considered first-line options when benefits outweigh risks, whereas valproate and topiramate should generally be avoided because of reproductive safety concerns [11], [52].

Calcium-channel blockers and botulinum toxin should not be presented as routine pregnancy treatments for VM. They may be considered only after individualized specialist review, taking gestational timing, maternal disability, fetal or neonatal exposure, available alternatives, and local obstetric guidance into account [11], [52].

7. Clinical Implications and Future Directions

The main clinical implication is that VM should be managed through shared neurological and otolaryngological reasoning rather than through sequential specialty exclusion. A patient presenting with dizziness and migraine features may still require audiometry, positional testing, or stroke safety assessment; conversely, a patient with normal interictal vestibular testing may still meet VM criteria [10], [18], [26].

A practical pathway begins with classification of dizziness by timing and triggers, assessment of red flags, application of definite or probable VM criteria, targeted positional and auditory testing, and follow-up to identify evolving Meniere disease, BPPV, PPPD, vascular disease, or mixed phenotypes [13], [31], [33]. Treatment should then be individualized across education, lifestyle measures, acute symptom control, prevention, rehabilitation, psychological care, and special-population safety. Psychological well-being may also warrant consideration as part of holistic care, particularly when anxiety, depression, or stress is present [53].

The most urgent research priority is high-quality VM-specific trials that use contemporary diagnostic criteria, adequate sample sizes, placebo or active comparators, standardized vestibular outcomes, electronic diaries, and long-term follow-up [18], [45], [46], [50]. Trials should separate dizzy days, vertigo attacks, headache days, DHI, VM-PATHI, quality of life, rescue medication, and adverse effects rather than combining them as if they measured the same construct.

Diagnostic research should move beyond reporting group differences in vestibular tests or imaging. Future studies should evaluate sensitivity, specificity, likelihood ratios, incremental value beyond history, external validation, and clinical utility [27], [28], [29]. Particular attention is needed for VM-Meniere overlap, VM-PPPD transitions, pregnancy and postpartum phenotypes, older adults, men, pediatric populations, and patients from diverse clinical settings

CONCLUSION

Vestibular migraine should be understood as a disabling neuro-otological disorder that requires integration of migraine biology with vestibular and otological assessment. The diagnosis remains clinical, criterion based, and longitudinal. Its central challenge is not simply identifying the coexistence of migraine and dizziness, but determining whether recurrent vestibular episodes fulfill accepted VM criteria while excluding or recognizing alternative and coexisting disorders. No audiovestibular test, imaging finding, or treatment response can independently confirm VM.

Neurological and otolaryngological perspectives are therefore complementary rather than competing. Neurology contributes assessment of migraine history, aura, sensory hypersensitivity, central mechanisms, medication exposure, comorbidity, and vascular or structural warning signs. Otolaryngology and neuro-otology contribute detailed characterization of vertigo phenomenology, nystagmus, auditory symptoms, hearing trajectory, positional triggers, and vestibular function. Their integration is particularly important in distinguishing VM from Ménière's disease, BPPV, PPPD, posterior-circulation ischemia, and mixed vestibular phenotypes.

Management should be individualized, multimodal, and phenotype driven. Education, lifestyle measures, vestibular rehabilitation, behavioral strategies, acute symptom-directed therapy, preventive pharmacotherapy, and selected emerging therapies should be tailored to attack pattern, vestibular disability, headache burden, comorbidity, reproductive safety, and patient preference. Current treatment evidence remains limited by small and heterogeneous VM-specific studies, inconsistent vestibular outcome

measures, and frequent extrapolation from migraine headache trials. Future research should prioritize well-designed VM-specific clinical trials, standardized diagnostic criteria, validated vestibular outcomes, long-term follow-up, and clearer evaluation of overlapping conditions and special populations. This integrated approach may improve diagnostic accuracy, reduce delayed recognition, and support more precise, patient-centered care for vestibular migraine.

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