Chapter

Vestibular Migraine

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Abstract

The consensus diagnostic criteria for vestibular migraine (VM) are used to specifically describe episodic vestibular symptoms associated with migraine. Because of an incomplete understanding of the etiology, a variety of clinical manifestations, and overlap with other vestibular disorders, the precise prevalence of VM is unknown. Clinical examination during vestibular episodes and vestibular laboratory tests interictally are more commonly abnormal in patients with VM than in controls, but none of the findings are specific for the diagnosis. The majority of information about VM treatment originates from case studies and retrospective reviews. In this chapter, the current epidemiology data, pathophysiology, significance of clinical and laboratory findings, and possible therapeutic approaches with existing and new medications or devices will be discussed.

Keywords: vestibular migraine, epidemiology, diagnostic criteria, pathophysiology, treatment

1. Introduction

Migraine and dizziness are frequently reported symptoms in clinical neurology. In a recent meta-analysis [1], it was found that the relative frequency of headache-associated vertigo in patients with migraine was 33.9%; however, there was significant heterogeneity between analyzed studies. Other researchers have reported episodic vertigo as a prodromal symptom in 3.3% of migraine patients [2], whereas headache phase-associated vertigo frequency varied between 6.4% and 44.7% [2–4]. Various names were interchangeably used to define a clinical entity that incorporates vestibular and migraine symptoms, including "migrainous vertigo, migraine-related vestibulopathy, migraine-associated vertigo, and migraine-associated dizziness" [5]. In 1999, Dieterich and Brandt coined the term vestibular migraine (VM), which is now used to describe vertigo or vestibular symptoms during a migraine attack [6]. Due to the heterogeneous clinical presentation, the lack of biological markers, and high comorbidity with other vertigo-causing diseases even in the presence of established diagnostic criteria [5, 7], diagnosis of VM might be challenging. VM is currently either underdiagnosed or misdiagnosed, and disabling vestibular symptoms still lack the approved management [8].

2. Vestibular migraine

2.1 Definition and diagnostic criteria

Currently, there are globally used International Headache Society and Barany Society consensus criteria for VM diagnosis (see **Table 1**) [5]. The diagnostic criteria are solely based on history taking. Only criterion D is based on the absence of physical examination or laboratory findings that might imply an alternative diagnosis.

Even though they are joint criteria of the two societies, in their respective classifications (the third edition of the International Classification of Headache Disorders [9] (ICHD-3) and International Classification of Vestibular Disorders (ICVD)), there are some differences. In ICHD-3, the criteria are more stringent, and VM is classified under the appendix, indicating that further research is needed and VM criteria have not yet been sufficiently validated to include in the main body. In contrast, ICVD includes additional criteria for probable VM in order to minimize the exclusion of patients affected by VM and to facilitate research. Despite the presence of official diagnostic criteria for VM, some researchers argue that they should be even expanded and potential subtypes of VM refined, as has been done for migraine headache [8, 10].

Although VM is considered an episodic vestibular disorder, the available literature indicates that a large proportion of patients experience symptoms between the attacks [5].

International Headache Society criteria for VM and Barany Society consensus criteria for definite VM (ICHD-3, 2018; ICVD, 2012, updated 2022)	**Barany Society consensus criteria for probable VM (ICVD, 2012, updated 2022)**
(A) At least 5 episodes with vestibular symptoms of moderate or severe intensity lasting 5 minutes to 72 hours	(A) At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting 5 minutes to 72 hours
(B) Current or previous history of migraine with or without aura according to the ICHD-3 criteria	(B) Only one of the criteria B and C for vestibular migraine is fulfilled (migraine history or migraine features during the episode)
(C) One or more migraine features with at least 50% of the vestibular episodes: 1. Headache with at least two of the following characteristics: a. One-sided location b. Pulsating quality c. Moderate or severe pain intensity d. Aggravation by routine physical activity 2. Photophobia and phonophobia 3. Visual aura	(C) Not better accounted for by another vestibular or ICHD diagnosis
(D) Not better accounted for by another vestibular or ICHD diagnosis	

ICHD-3—the third edition of the International Classification of Headache Disorders; ICVD—the International Classification of Vestibular Disorders; VM—vestibular migraine.

Table 1.
Criteria for vestibular migraine by the international headache society and for definite and probable vestibular migraine by the Barany society [5, 7, 9].

ICVD discusses chronic VM, which may be associated with interictal visually induced, head motion-induced, or persistent dizziness. Because these symptoms are reported by a significant number of patients, upon further research, chronic VM may be included in a revised version of ICVD [5]. However, it may be more appropriate to make a coexisting diagnosis of persistent-postural perceptual dizziness (PPPD) if criteria for that condition are met [5, 7].

Additionally, there are other disorders closely related to VM. Migraine with brainstem aura (previously called basilar migraine) may also manifest with headache and vertigo; however, this diagnosis requires at least two focal neurologic brainstem symptoms in addition to visual, sensory, or dysphasic aura, and these criteria are met

Benign paroxysmal vertigo (of childhood) (ICHD-3, 2018)	**Vestibular migraine of childhood (ICVD, 2021)**	**Recurrent vertigo of childhood (ICVD, 2021)**
(A) At least five attacks fulfilling criteria B and C	(A) At least five episodes with vestibular symptoms of moderate or severe intensity lasting between five minutes and 72 hours	(A) At least three episodes with vestibular symptoms of moderate or severe intensity, lasting between 1 minute and 72 hours
(B) Vertigo occurring without warning, maximal at onset, and resolving spontaneously after minutes to hours without loss of consciousness	(B) A current or past history of migraine with or without aura	(B) None of the criteria B and C for vestibular migraine of childhood
(C) At least one of the following five associated symptoms or signs: 1. nystagmus 2. ataxia 3. vomiting 4. pallor 5. fearfulness	(C) At least half of the episodes are associated with at least one of the following three migraine features: 1. Headache with at least two of the following four characteristics: a. One-sided location b. Pulsating quality c. Moderate or severe pain intensity d. Aggravation by routine physical activity 2. Photophobia and phonophobia 3. Visual aura	(C) Age < 18 years
(D) Normal neurological examination and audiometric and vestibular functions between attacks	(D) Age < 18 years	(D) Not better accounted for by another headache disorder, vestibular disorder, or other conditions
(E) Not attributed to another disorder	(E) Not better accounted for by another headache disorder, vestibular disorder, or other conditions	

ICHD-3—the third edition of the International Classification of Headache Disorders; ICVD—the International Classification of Vestibular Disorders.

Table 2.
Criteria of benign paroxysmal vertigo, vestibular migraine of childhood, and recurrent vertigo of childhood by the international headache society and Barany society [9, 11].

by less than 10% of VM patients [9]. Therefore, while a subset of patients may fit the diagnostic criteria for both disorders, they are recognized as distinct entities.

ICHD-3 defines benign paroxysmal vertigo as an episodic syndrome that may be associated with migraine, which occurs primarily (but not exclusively) in healthy children. In 2021, the Barany Society proposed a new consensus document [11] with an aim to change the terminology from "benign paroxysmal vertigo" to definite and probable "VM of childhood" and "recurrent vertigo of childhood." Contrary to the ICHD-3 criteria, which do not specify an age limit, the ICVD criteria do (see **Table 2**). One of the reasons for the change of terminology was confusion about the term "paroxysmal," which is designated for vestibular symptoms lasting less than one minute. Additionally, while being thought to be a migraine precursor, the diagnosis of benign paroxysmal vertigo does not include any migrainous characteristics [11]. Migraine diagnosis is commonly absent due to a short clinical history of headaches and difficulties distinguishing children's symptoms [12]. However, a substantial proportion of children with episodic vertigo also display migraine features. New criteria distinguish these groups by employing a continuum ranging from probable and definite VM to recurrent vertigo of childhood, allowing for more precise research [11].

2.2 Epidemiology and comorbidity

The precise prevalence of VM is unknown; however, it is thought to be a very common condition, with an estimated prevalence of 1% to 2.7% in the general population [13, 14] and 11–13% in specialized dizziness and headache centers [15, 16]. Probable and definite VM was the most frequent episodic non-positional non-ischemic vestibular syndrome in primary care [17]. In both familial and non-familial cases of VM, there was a female predominance in most of the studies included in systematic review [13]. The mean age of onset of VM in patients with concurrent migraine and vertigo manifestation was found to be 22.7 ± 10.4 years, whereas patients with a non-simultaneous presentation of symptoms were somewhat older, with a mean age of onset for vertigo of 35.6 ± 12.4 years and 24 ± 8.9 years for migraine [18].

The frequent comorbidity of migraine with other vertigo-causing diseases complicates diagnosis and undoubtedly distorts data on VM prevalence. Comorbid conditions and disorders associated with VM include benign paroxysmal positional vertigo (BPPV), Ménière's disease (MD), motion sickness, Mal de Debarquement syndrome (MdDS), PPPD, and anxiety disorder [19, 20].

BPPV is one of the most common causes for short episodic dizziness and vertigo [21]. Migraine patients have a 2.5-fold increased risk of BPPV recurrence, whereas BPPV patients have an increased risk of migraine [22–24].

MD and VM share many similar clinical features. Patients with MD present with migraine twice as often as the control group [25, 26].

Motion sickness, visually induced motion sickness (VIMS), and corresponding disorders may co-occur, and the severity of symptoms may be increased by a variety of vestibular disorders, including VM [27].

MdDS is a syndrome defined by non-spinning vertigo with an oscillatory perception that occurs within 48 hours of the end of passive motion. The symptoms last for at least 48 hours and are temporarily alleviated by recurrence of passive motion [28]. Migraine headaches may develop alongside the onset of MdDS and worsen as the disease progresses [29].

PPPD is characterized by one or more symptoms of dizziness, unsteadiness, or non-spinning vertigo, which are present on most days for three months or more and

are exacerbated by upright posture, active or passive movement, and exposure to moving or complex visual stimuli. VM attacks are one of the most common precipitating conditions leading to the development of comorbid PPPD [30].

Cross-sectional studies conducted in specialized vertigo and dizziness centers revealed that nearly half of the dizzy patients had a psychiatric disorder compared to 20% of the general population [31–33]. Nevertheless, psychiatric comorbidities were not consistent across different vestibular disorders. VM had one of the highest rates for psychiatric comorbidity (49%). In addition, anxiety and phobic disorders were the most common among VM patients (32.6%) [31].

It must be highlighted that any vestibular disorder might be complicated by migraine. Therefore, the nonspecific co-occurrence of migraine and vestibular symptoms necessitates the consideration of differential diagnoses (see Chapter 2.5).

2.3 Pathophysiology

The pathophysiology of VM is multifactorial and not fully understood, as evidenced by the multitude of hypotheses proposed to explain VM. Suggested central mechanisms include a cortical spreading depression induced by an unknown trigger impacting the vestibular cortex and, as a result, affecting brainstem vestibular nuclei via vestibular-thalamic cortical pathways or a brainstem aura directly affecting these nuclei [34, 35]. Yet only 2–30% of individuals fulfill the migraine aura criteria, that is, vertigo lasting 5 to 60 minutes prior to the onset of the headache [36].

In addition, altered sensory processing and integration have been proposed as one of the contributing mechanisms for VM [24]. In order to maintain spatial orientation when the head position is changed, the brain must integrate vestibular and visual inputs. Inaccuracies in this process have been described in VM patients with spatial perception errors consistent with overestimation of the head position [24, 37]. Consequently, VM patients predominantly report dizziness when their heads are tilted in the same direction, which causes greater spatial orientation error, indicating that altered sensory processing and integration may be a cause of visuospatial symptoms in VM [24].

Alternatively, activation of the trigeminovascular system represents a peripheral mechanism. The inner ear, cochlear nucleus, and superior olivary complex are innervated by neurons of the trigeminal ganglion [38, 39]. It has been previously shown in experimental studies that chemical and electrical stimulations of the trigeminal ganglion cause a considerable increase in inner ear blood flow and alterations of vascular permeability, causing plasma protein extravasation and disruption of inner ear metabolism [40, 41]. The chemical stimulation in VM occurs by substances released during the cortical spreading depression (potassium, glutamic, and arachidonic acid), which activate the trigeminal pain receptors and neurons of the trigeminal nucleus [42]. Moreover, this notion is supported by painful electrical stimulation to the trigeminal nerve, triggering peripheral spontaneous nystagmus or enhancing pre-existing spontaneous nystagmus in migraine patients [43]. In addition, calcitonin gene-related peptide (CGRP) has been identified in sensory fibers that innervate vestibular nuclei and the inner ear, explaining the possible role of CGRP inhibitors in the treatment of VM [44].

There has been research associating migraine and vertigo to ovarian hormones, particularly estrogen and progesterone [45]. Vestibular symptoms in women can become more severe or begin around menopause, which may be explained by the modulating action of ovarian hormones on monoaminergic systems in various cerebral structures, including the vestibular and trigeminal nuclei.

VM has been documented in families with an autosomal dominant pattern and reduced penetrance in men [46]. Additionally, VM is related to familial hemiplegic migraine (FHM) and episodic ataxia type 2 (EA-2) in that both conditions frequently present with vertigo and migraine. Furthermore, VM is associated with mutations of CACNA1A (coding for neuronal calcium channel), ATP1A2 (coding for Na/K ATPase), and SCN1A (coding for voltage-gated sodium channel), which may occur in FHM and EA-2 [47].

2.4 Clinical characteristics

VM patients frequently experience vestibular symptoms following headache; fewer patients have vestibular symptoms right before headache, whereas some individuals never experience headache and vestibular symptoms simultaneously [24].

The most common vestibular symptoms include spontaneous vertigo, positional vertigo, head motion-induced vertigo, and visually induced vertigo [13, 24]. However, patients frequently employ a variety of different terminologies to describe these symptoms, reflecting the diverse internally perceived experiences. The majority of VM patients experience more than one vestibular symptom during an acute attack, with the most common being spinning vertigo (72%), disequilibrium and/or Mal de Debarquement (58%), and rocking/tilting sensation (43%), whereas most common migraine symptoms include headache (81%), visual aura (26%), and photophobia (23%) [48]. Vertigo is frequently triggered by head motion (44%), visual stimuli (41%), or positional change in the supine position (25%) followed by spontaneous vertigo [49]. Nausea is also common (55%), with almost 20% of all patients progressing to vomiting [48]. The duration of vestibular symptoms varies greatly, with approximately 30% of patients estimating that the symptoms continue for minutes, 30% for hours, and 30% for days. The remainder 10% describe fluctuating daily symptoms [24]. Additionally, one study found that visually induced dizziness (89%) and head motion-induced dizziness (66%) were extremely common during the interictal period [19].

Along with vestibular symptoms and migrainous headache, VM patients frequently experience unilateral or bilateral auditory symptoms such as tinnitus (52%), aural fullness (41%), mild and easily reversible low-frequency hearing loss (21%), and even otalgia (8.4%) [50]. However, because of their great incidence in other vestibular disorders, it was decided that auditory symptoms should not be included in the VM diagnostic criteria [5].

2.5 Differential diagnosis

Differential diagnoses for VM include MD, BPPV, vertebrobasilar transient ischemic attacks (TIAs), vestibular paroxysmia (VP), and episodic ataxia type 2 (EA2).

MD is characterized by recurrent spontaneous vertigo lasting 20 minutes to 12 hours and cochlear signs: sensorineural hearing loss, fluctuating tinnitus, and aural fullness [51]. VM and MD are the most difficult diagnoses to differentiate between, usually necessitating several follow-up visits to a specialist in order to make a clear distinction [52]. Patients meeting both definite VM and definite MD criteria have been described repeatedly in the literature, suggesting the possibility of an overlap syndrome and a shared pathophysiology [53]. Signs and symptoms such as aural pressure, tinnitus, migrainous headache, auras, and endolymphatic hydrops can occur in both of these disorders. Nevertheless, a low-frequency hearing loss is

the key finding that differentiates MD from VM [52]. Hearing loss in VM typically is bilateral and down sloping as opposed to unilateral or asymmetric and flat in MD [5]. The Barany Society recommends diagnosing MD even if migraine symptoms occur during episodes of vertigo. Additionally, only patients with different types of attacks who meet the diagnostic criteria for VM and MD (in respect to different attack types) should be diagnosed with both disorders [7].

BPPV is a common cause of paroxysmal (less than 1 minute) dizziness and vertigo, which occurs due to abnormal stimulation of the semicircular canals by otoliths [21]. The primary distinction between BPPV and VM is the duration of the vertigo episode: an acute episode of BPPV lasts less than one minute, whereas a typical episode of VM lasts 5 minutes to 72 hours [5]. Although residual dizziness and associated symptoms in BPPV may be misinterpreted as an active continuation of the vertigo episode, a thorough history taking and the use of repositioning maneuvers that result in semicircular canal-specific nystagmus with latency, as opposed to persistent nystagmus of moderate velocity in VM, may help in differential diagnosis [5, 54].

Although rare, vertebrobasilar TIAs can occur as isolated episodes of vertigo, lasting minutes to hours without additional brainstem symptoms; however, TIAs are not associated with headache and usually follow a course of increasing frequency (if evident cardiovascular risk factors are not corrected) rather than weeks or months of vertigo-free periods as in the case of VM [36, 55]. In suspected stroke cases, HINTS (head impulse test, nystagmus, and skew test) examination may be beneficial [54].

VP is distinguished by brief (less than a minute, usually a few seconds) episodes of transient vertigo that are unrelated to migraine attacks. Successful treatment with carbamazepine or oxcarbazepine may also aid in diagnosis [5, 54].

EA2 is a genetic autosomal dominant disorder that typically manifests at the age of 20 [55]. EA2 is characterized by a sudden onset of vertigo and associated with headache and ataxia [52, 55]. Vertigo episodes can last from hours to days and are usually brought on by emotional stress, physical exertion, and alcohol consumption [55]. The time between episodes can range from a few days to several years. As a result of the clinical similarities, the vertigo caused by EA2 cannot be reliably distinguished from VM [52, 55]. Additional changes in MRI consist of cerebellar atrophy of anterior vermis, and treatment with acetazolamide may attenuate or prevent attacks in up to 50 percent of patients [55]. The combination of these clues, together with typical triggers and a positive family history, may help in diagnosis.

2.6 Clinical and laboratory findings

The heterogenic nature of VM is reflected by great variation of vestibular and audiological findings between different investigators and even across patients. The neurologic examination is usually normal between acute attacks. Some studies have reported various oculomotor abnormalities interictally in up to 55% of individuals such as spontaneous, gaze evoked, central positional nystagmus, or smooth pursuit deficits; however, they were nonspecific [6, 56–59].

During vertigo episodes, up to 71% of patients had spontaneous nystagmus, with horizontal direction in 50%, down-beat in 12%, and up-beat in 10% [56]. In addition, it was found that the use of a visual fixation block can reveal low-velocity positional nystagmus, which is either horizontal, vertical, or torsional [36]. Furthermore, spontaneous horizontal nystagmus can be provoked by the headshaking test in 35% of VM patients during an attack [36]. Interestingly, one study found that severe spontaneous horizontal nystagmus with a speed greater than 12 degrees per second

was more sensitive and specific for MD, whereas spontaneous vertical nystagmus was more specific for VM [60]. A majority of VM patients show unidirectional nystagmus during repeated attacks, with only a small fraction displaying nystagmus direction reversal [56]. Additionally, an abnormal head impulse test may accompany spontaneous horizontal nystagmus in VM, indicating a unilateral deficit in the vestibuloocular reflex [36]. During episodes of VM, most patients have impaired stance and gait, with difficulties performing the Romberg test, tandem gait, or even standard gait [36].

There are no conclusive tests to confirm VM. The diagnosis is based on consensus criteria, and VM should be regarded as a diagnosis of exclusion. Nevertheless, due to a variety of phenotypes, VM may mimic other vestibular disorders, in which case additional testing might be beneficial; thus, instrumental findings will be further discussed. The absence of any structural lesions supports the diagnosis of VM and can reduce patients' anxiety.

Video head impulse testing (vHIT) performed interictally may demonstrate reduced gain of lateral semicircular canals; however, gain changes in addition to saccade frequency, amplitude, and duration are insignificant compared to normal controls [56]. In contrast, a majority of VM patients during acute vertigo display vHIT results consistent with peripheral dysfunction [56, 61].

Caloric testing is rarely useful in distinguishing VM patients from those with other causes of vertigo [20, 36]. Bithermal caloric testing is abnormal in approximately 15–20% of patients, with the most common deficits being a reduced unilateral response, directional preponderance, and, in some cases, a reduced bilateral response [36, 56].

On a firm surface, posturography findings do not differ between VM patients and control groups; however, on a foam surface, the mean center of gravity sway velocity with eyes open and closed is increased in VM patients [58]. Nonetheless, posturography results are insufficient to diagnose VM and must be interpreted in conjunction with other vestibular tests.

Vestibular-evoked myogenic potentials (VEMPs) have recently received additional attention in the differential diagnoses of VM and MD [62]. By testing for cervical VEMPs, it is possible to selectively measure the function of saccule, while ocular VEMPs reflect the function of utricle [63]. In a recent study, it has been found that the decrease in saccular function is greater than the decrease in utricular function in MD, whereas the degree and asymmetry of saccular and utricular dysfunctions in VM are low and tend to be equal in both labyrinths. As a result, different patterns of saccular and utricular dysfunctions may help differentiate VM from other causes of dizziness over time [62]. However, several other studies measuring VEMPs have yielded contradictory results [49, 64, 65]. Although the rate of VEMP abnormalities is higher in patients with vestibular disorders than in controls, there are as yet insufficient data to confirm that VEMPs can confidently distinguish between VM and MD [20, 36].

Even though many VM patients have mild auditory symptoms, audiometry results show that the majority of individuals have normal symmetric function or age-related symmetric high-frequency hearing loss, whereas only less than 8% of patients display asymmetry or loss of hearing in more than one frequency [56]. On the other hand, audiometric testing should be performed in patients who exhibit any auditory symptoms during or between attacks to confirm the subclinical or evident hearing loss, which is characteristic of MD. It should be noted that while initial testing may be negative, in the case of unknown diagnosis, follow-up audiometry could add additional value [66].

Endolymphatic hydrops, a characteristic finding of MD classically described in the literature, may also occur in VM. According to studies performing high-resolution

MRI of the inner ear, the presence of endolymphatic hydrops was found to be the most common in MD (79%), the least common in VM (12%), and intermediate in the VM-MD overlap syndrome (25%; i.e., when both criteria of VM and MD are met) [48]. Moreover, studies report a relatively high prevalence of endolymphatic hydrops in healthy controls [10]. As a result, while the rate of endolymphatic hydrops in VM is lower than that in MD, there is still controversy about the utility of distinguishing these two diseases based on the presence of hydrops. Alternatively, in cases of suspected VM, the main indication for neuroimaging is a new onset of isolated vertigo lasting minutes in those with additional vascular risk factors to exclude vertebrobasilar transient ischemic attacks [36].

2.7 Treatment of vestibular migraine

There are currently no evidence-based treatment guidelines for VM [67]. Furthermore, randomized controlled trials are scarce, and most treatment recommendations are based on either case series, individual case reports, or retrospective studies with no control groups. In addition, patient documentation of headache and vestibular symptoms could significantly improve the rate of correct diagnosis and be used to assess treatment efficacy in everyday practice; nevertheless, it is still underused.

2.7.1 Non-pharmacologic treatment

Education in VM is one of the most important aspects of disease management. Reassuring patients that the episodes are only temporarily disabling and not related to stroke and not associated with permanent hearing or vestibular function loss may significantly help to relieve VM-associated anxiety. Although follow-up studies show that as many as 90% of patients still suffer from VM after 9 years of initial diagnosis, the frequency of vertigo decreases in over 50% of patients [68]. As a result, cautious positive expectation formation is reasonable.

Identifying modifiable lifestyle factors that contribute to VM (e.g., sleep disturbances, stress, insufficient physical activity, food triggers) and encouraging patients to take an active role in their disease management is another key intervention during consultation. In addition, vestibular rehabilitation should be recommended as it may increase the threshold of a VM attack to some extent [54]. Vestibular rehabilitation typically consists of four groups of exercises (gaze stability exercises, habituation exercises, gait and balance training, and walking to improve endurance), and some studies, albeit with low-quality evidence, suggest that it may be more effective than pharmacologic intervention [54, 69]. Furthermore, some randomized controlled trials concerning alternative non-pharmacologic prophylaxis (e.g., acupuncture) are underway [70].

Lastly, studies with external trigeminal nerve stimulation (eTNS) and non-invasive vagus nerve stimulation (nVNS) for acute VM symptoms (including both vertigo and headache) have shown promising results, with no intolerable side effects and mean vertigo reduction of 61.3% with eTNS and 46.9% with nVNS [71, 72].

2.7.2 Treatment of acute attack

In the case of short, infrequent, and mild episodes of VM, it should be discussed with the patient whether pharmacologic treatment is indicated. On the other hand, if significant vertigo and/or nausea lasts longer than the time required for the drug to

Drug	Dosage	Notes
Dimenhydrinate [73]	25–50 mg, may be repeated every 6 h	May cause sedation, vision impairment, urinary retention
Diphenhydramine [73]	50–100 mg, may be repeated every 6 h	As above
Meclizine [73]	25–50 mg, may be repeated every 6 h	As above
Metoclopramide [74]	10 mg, may be repeated every 4 h	May cause QT prolongation, extrapyramidal effects
Almotriptan [75]	12.5 mg	May cause nausea, xerostomia, paraesthesia
Rizatriptan [76]	10 mg	As above
Zolmitriptan [77]	2.5 mg	As above
Methylprednisolone [78]	1000 mg i/v	Primarily used to decrease the severity of unusually long episodes

Table 3.
Treatment of acute symptoms in vestibular migraine.

start working (usually more than 30 minutes), pharmacologic intervention should be initiated [36]. Despite the lack of high-quality evidence on efficacy, acute VM treatment mainly consists of antivertiginous and antiemetic medications [36, 54, 55, 73]; see **Table 3**. In addition, benzodiazepines may be used to suppress vestibular system and consequently reduce the symptoms of acute VM. Triptans have been reported to reduce vertigo; however, most studies investigating triptans in the treatment of acute VM provide low-quality evidence [75–77]. The combination of different classes of aforementioned drugs may be used if nausea and/or vomiting is particularly severe. Additionally, analgesics should be added if headache is present. Finally, in treatment-resistant cases, intravenous methylprednisolone has been shown to be successful in some case reports [78].

2.7.3 Preventive treatment

Most medications for VM treatment are targeted at prophylaxis. Prophylaxis may be appropriate for patients who have long, frequent, and severe episodes or in cases of acute treatment failure. In addition, combining pharmacologic and non-pharmacologic treatment (e.g., vestibular rehabilitation) may be more effective than either treatment alone [73].

Due to a lack of trials focusing specifically on VM, treatment approaches are based on migraine research with the goal of reducing the frequency and severity of VM attacks. In practice, medication selection is based on side effects and comorbidities rather than on data of efficacy see **Table 4** [36, 54, 79]. Nonetheless, a recent meta-analysis has found that propranolol, followed by venlafaxine, resulted in the greatest short-term improvement in the dizziness handicap index [67].

To date, there have been only two randomized controlled trials in VM prophylaxis. One study found that flunarizine reduced the frequency and severity of vertigo without reduction in headache [81], while the other showed that vertigo and dizziness were significantly reduced in patients treated with propranolol or venlafaxine [80]. In addition, venlafaxine was superior to propranolol in the treatment of comorbid mood

Drug	Dosage	Notes
Propranolol [80]	40–160 mg/d	Useful in cases of hypertension, tachycardia, anxiety. May cause bronchial constriction, impotence. May worsen depression. RCT available.
Venlafaxine [80]	37.5–150 mg/d	Useful in cases of obesity, mood disorders. May cause fatigue, insomnia. RCT available.
Flunarizine [81]	10 mg/d	May cause weight gain, somnolence, nausea. RCT available.
Topiramate [73]	50–100 mg/d	Useful in cases of obesity and fatigue. May cause cognitive dysfunction, somnolence, paresthesia. Teratogenic.
Valproic acid [54]	500–1000 mg/d	May cause weight gain, somnolence, thrombocytopenia, hepatotoxicity. Teratogenic.
Amitriptyline [73]	25–75 mg/d	Useful in cases of insomnia. May cause constipation, sedation, weight gain, conduction block.

RCT—randomized-controlled trial.

Table 4.
Prophylactic treatment typically used in vestibular migraine.

disorders and is believed to be a preferred choice when VM coexists with persistent postural-perceptual dizziness, depression, or anxiety [80, 82].

New treatment strategies, for example, anti-CGRP medications or onabotulinumtoxin A injections, have been also employed in small studies. Anti-CGRP medications (monoclonal antibodies and gepants) have demonstrated moderate to significant VM improvement in 60% (n = 15) of patients [83]. Onabotulinumtoxin A injections have been shown to be effective in reducing vertigo and headaches in resistant VM cases, as reflected by improvements in the Headache Impact Test, Migraine Disability Assessment, Dizziness Handicap Inventory, Vertigo Symptom Scale, and even changes in functional brain connectivity [84].

Many patients prefer non-pharmacologic treatment to taking medications on a daily basis. In those cases, alternative methods such as relaxation training, biofeedback training, and cognitive behavioral therapy that have shown significant improvements could be suggested [73].

3. Conclusions

VM is a disorder characterized by migrainous headaches and associated vestibular symptoms. VM is believed to be a very common condition affecting up to 2.7% of the general population, whereas probable and definite VMs are reported to be the most frequent episodic non-ischemic vestibular syndrome in primary care. Furthermore, VM is frequently comorbid, with other vertigo-causing disorders. The diagnosis of VM is based on the consensus diagnostic criteria and the exclusion of other vestibular disorders. There is no single instrumental or laboratory test that can reliably confirm or reject the diagnosis of VM. Although no evidence-based guidelines exist for the treatment of VM, in clinical practice, a combination of painkiller and vestibular suppressant for acute VM attacks is commonly used.

Similarly, personalized prevention of migraine headaches has been shown to reduce the frequency of VM episodes.

Conflict of interest

The authors declare no conflict of interest.

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