



Vestibular migraine — an underdiagnosed cause of vertigo. Diagnosis and treatment

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ABSTRACT

Introduction. Migraine and vertigo are two common conditions. The main disorder connecting both these entities is vestibular migraine (VM).

State of the art. VM may affect 1–3% of the general population. It is a disabling disease of recurrent attacks of vestibular symptoms accompanied by migraine features which occur in patients with a current or previous history of migraine. The episodes can last minutes, hours or even days, and may occur without any concurrent headache, which can prompt misdiagnosis. VM often begins several years after a typical migraine, and the delay between onset of headache and vertigo may be long. The diagnosis is based on the patient's clinical history and can be challenging due to the lack of an established confirmatory diagnostic test or biomarkers. The mechanism of vestibular migraine remains unclear and is still under investigation, but it seems to be an interaction between trigeminal and vestibular systems. Due to the lack of specific trials, treatment recommendations are based on migraine guidelines. Several drugs seem to be effective, although there have been few randomised controlled trials in this area. Regardless of the published strict and detailed diagnostic criteria, this condition remains little known, and as a consequence is underdiagnosed and undertreated.

Clinical implications. Efforts should be made to educate medical communities and patients about this disease and to encourage neurologists and ENT specialists to cooperate. Every patient with vertigo of unknown origin should be directly asked about a past or present history of migraine, or migraine symptoms experienced during their vertigo episodes.

Future directions. There is a growing need for studies regarding the pathophysiology of VM as well as randomised trials to establish clear treatment recommendations and to improve management of this surprisingly common disorder.

Key words: migraine, vestibular migraine, headache, dizziness, vertigo, treatment

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Introduction

Migraine and vertigo are two very prevalent conditions in the general population [1]. Nevertheless, a link between those two disorders exists: vertigo is 2–3 times more common in migraine patients, especially migraine with aura patients, than in the general population (ranging from 30 to 50%) [2, 3]. In addition, vertigo is reported to coexist with other migraine symptoms during almost half of severe migraine attacks [3].

It is also worth noting that vertigo can be one of the aura symptoms in migraine with brainstem aura, as well as a part of benign paroxysmal vertigo in children [4].

However, the main disorder which connects vertigo with headache is vestibular migraine (VM) (previously known as migrainous vertigo or migraine-associated vertigo). VM is one of the most common neurological disorders causing vertigo and dizziness [5], but despite its high prevalence and published diagnostic criteria, it remains surprisingly little known, and

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thus underdiagnosed and undertreated [6]. The cause of this diagnostic challenge may be the wide spectrum of symptoms, the absence of headache in almost half of the attacks, as well as poor knowledge of diagnostic criteria and a lack of cooperation between the neurological and otolaryngological communities.

Epidemiology and demographic factors

Vestibular migraine affects approximately 1–3% of the general population and up to 30% of patients in specialised vertigo or headache centres [1, 7]. Moreover, benign paroxysmal vertigo of childhood is known to be an early manifestation of migraine. This has a 3% prevalence in children between the ages of 6 and 12 [8]. In one study, VM was diagnosed in 10.3% of migraineurs, while another determined its prevalence to be 21% in the migraine population [9, 10]. Chia et al. [11] reviewed 208 patients with benign recurrent vertigo diagnosed in a neuro-otology clinic, and found that 87% of them met ICHD criteria for migraine (62% with aura), and that among migraine patients with vertigo and migraine 70% met the VM criteria, which is a surprisingly high amount. Indeed, VM patients significantly more often experience migraine with aura, and vestibular symptoms are twice as likely to be reported in patients with migraine with aura compared to individuals without aura [2, 3]. Plus, according to Martinez et al. [12], almost 40% of patients with VM meet criteria for chronic migraine.

Importantly, the presence of migraine is extremely high in patients with recurrent unclassified vertigo (ranges from 60–80%) [11]. VM is more prevalent in females, with a gender ratio of from 1.5 up to 5.1 [13–15]. The average age at onset of VM is 38–50 [15–17]. It is worth noting that migraine headache precedes the onset of vestibular attacks, where the age at onset of migraine is 23–32 years, while the average age at onset of vertigo is 34–38 years [15, 18, 19]. Moreover, mostly in postmenopausal women, typical migraine attacks may be replaced by vestibular episodes [20]. Interestingly, the time gap between migraine and vestibular symptoms onset may be long, as patients can be headache-free for years before the vestibular symptoms starts [21]. The mean delay between the onset of headache and vertigo is 8–14 years [21, 22]. The majority of VM patients suffer from episodic migraine, and one in four of them have chronic migraine with medication overuse headache [15]. Most VM patients report a family history of the disorder. A history of migraine has been found in 50.8–70.2% of cases [16]. Notably, some patients also mention a family history of vertigo of no clear origin, and many of them refer to migraine precursors, especially motion sickness during childhood as well as cyclic vomiting, torticollis and/or episodic abdominal pain [8, 16, 23].

Some authors have suggested that vestibular symptoms in paediatric patients may predispose to the development of VM as adults [18]. One study revealed that up to 78% of VM patients had experienced carsickness in their lifetime [24]. Other prevalent comorbidities in VM patients are mood disorders

including anxiety, depression and sleep disturbance [7, 15, 25]. One study demonstrated that age under 40, female sex, mood disorders, and prior head trauma were all connected with significantly increased odds of developing VM [7]. VM is one of the most common precipitants of persistent postural perceptual dizziness, a newly defined disorder characterised by persistent dizziness and perceived instability, which is exacerbated in busy visual environments and in an upright position [26]. Approximately 40% of patients with VM have reported missing work because of their symptoms, which demonstrates the impact of this disease on daily activities and quality of life [1].

VM is an underdiagnosed disorder: a study in a tertiary vertigo centre demonstrated that although VM was finally diagnosed in 20.2% of patients, it was suspected by the referring doctor in fewer than 2% of patients [6]. Another study showed that only 20% of VM patients were correctly diagnosed after visiting a doctor [1]. Similarly, Formaister et al. [7] reported that only 10% of subjects meeting the criteria for VM were told that migraine was the cause of their dizziness.

Misdiagnosis increases when vestibular symptoms are not associated with headaches and half of the patients are supposed to have Meniere's Disease [15]. A survey among American neurologists and otorhinolaryngologists (ENT) revealed that a neurologist would diagnose VM in vertigo and headache patients more often than an ENT specialist. This finding is alarming because 19% of ENT specialists and 14.5% of neurologists have declared that they had never treated a patient with VM before [27].

Our experience is that more patients with VM are found in otolaryngological than in neurological departments, and that to set up a VM diagnosis a neurological consultation must be performed, as most ENT specialists are unfamiliar with migraine diagnostic criteria.

Pathophysiology of VM

Although the pathogenesis of VM is still unclear, genetic, inflammatory and neurochemical mechanisms have been proposed, mostly based on migraine pathophysiology, as migraine interacts with the vestibular system at many different levels [28–30]. It seems that both the central and peripheral vestibular systems are associated with its pathogenesis.

First of all, many studies have highlighted the overlap between vestibular and migraine pathways, as the caudal parabrachial nucleus receives both afferent peripheral trigeminal nociceptive and vestibular input, and also the trigeminal nerve affects the inner ear by cochlear vasculature innervations [31]. So, the cause of VM may be direct central activation of vestibular centres by the trigeminovascular system together with its effects on the inner ear [32]. It seems that vestibular symptoms come from the vestibular nuclei, which are simultaneously suppressed by inhibitory feedback from the cerebellar nodulus and uvula [33]. There is also evidence of otolithic

pathway abnormalities in individuals with VM [34]. It is also suggested that Purkinje cells in the paraflocculus could be inhibited after the occurrence of a migraine episode, which may be an important factor leading to vestibular migraine [35]. Magnetic resonance studies have shown an increased thalamic activation in VM patients during vestibular stimulation compared to healthy controls, as well as grey matter volume abnormalities of nociceptive and multisensory vestibular brain areas [36, 37]. In addition, a PET study during an attack revealed increased metabolism in the temporo-parieto-insular areas and bilateral thalami as well as decreased metabolism in the occipital cortex [38]. Another pathophysiological mechanism in VM may be a visuo-vestibular problem, as patients with VM have a longer duration of post-rotatory nystagmus compared to healthy controls or ordinary migraine patients [39]. Plus, a genetic component in the pathogenesis of VM has been identified [28, 40].

Diagnostic criteria

A VM diagnosis is based on clinical history. The International Headache Society (IHS) and the Bárány Society have published a consensus document with diagnostic criteria for vestibular migraine added to the ICHD-3 beta (Appendix, subchapter 'Episodic syndromes that may be associated with migraine') [4, 41] (Tab. 1). According to academic panels, VM was defined as an emerging condition needing further research for full validation [4].

The basic diagnostic criteria are presented in Table 1, but some terms need to be broadly described. First of all, vestibular symptoms (as defined by the Bárány Society's Classification of Vestibular Symptoms) include: spontaneous vertigo (internal — a false sensation of self-motion, or external — a false sensation that the visual surrounding is spinning or flowing); positional vertigo; visually-induced vertigo; head motion-induced vertigo; and head motion-induced dizziness with nausea. It is worth noting that to fulfill diagnostic criteria dizziness must be characterised by a sensation of disturbed spatial orientation, thus other forms of dizziness are currently not included in the classification of vestibular migraine [4]. Besides, vestibular symptoms do not include presyncope symptoms, mental confusion, depersonalisation, generalised weakness or fatigue. Moderate vestibular symptoms are defined as those interfering with, but not preventing, daily activities, while severe symptoms are those that stop daily activities. Although vestibular symptoms must last between five minutes and 72 hours, individuals with attacks lasting only for a few seconds may be included if the total period during which short attacks recur is one of at least five minutes [4]. Interestingly, Abouzari et al. compared patients with a diagnosis of VM by ICHD-3 to those who did not fulfill ICHD-3 criteria although they had concurrent vertigo and migraine. They found that both groups were very similar in their characteristics. Thus, adhering strictly to the ICHD-3 criteria, a nearly equivalent

Table 1. Diagnostic criteria for definite vestibular migraine [4, 41]

Diagnostic criteria for definite vestibular migraine	
A.	At least five episodes fulfilling criteria C and D
B.	Current or previous history of migraine with or without aura according to International Classification of Headache Disorders (ICHD-3)
C.	Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours
D.	One or more migraine features with at least 50% of the vestibular episodes: <ul style="list-style-type: none"> • Headache with at least two of the following characteristics: unilateral location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity • Photophobia and phonophobia • Visual aura
E.	Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder.
In order to diagnose probable VM, only one of criteria B and D is required to be fulfilled	

population that might benefit from migraine treatment would be missed. The authors concluded that the differences between cohorts represented selection bias, not meaningful features, so both groups may represent a spectrum of the same disease [42].

Clinical features

Vestibular symptoms

Most patients report internal vertigo (73%) and triggered vertigo, while external vertigo and positional vertigo are less frequent [16, 18]. Vertigo is often described as a spinning, rocking, tilting, swaying and falling sensation. According to other study, vestibular symptoms were mainly described as like feeling the ground slipping (40.6%), swaying (27.7%), rocking as on a boat (26.7%), or stepping into an empty space (24.8%) [15]. Less common vertigo sensations include floating, shimmering, tumbling, bobbing, swimming, sliding, and multi-directional motion [18]. One third of patients experienced unsteadiness [16]. It is worth noting that most patients report more than one vestibular symptom during VM episodes [16]. Almost half of individuals complain of persistent dizziness during the interictal period [16]. The majority of patients continue to have recurrent vertigo in the long-term evolution of VM, with possible slow progression of vestibulo-cochlear dysfunction and the development of mild persistent unsteadiness [43].

Headache and other migraine features

Although the core of the disease is migraine, and according to diagnostic criteria migraine features must be present for at least half of the time along with vestibular symptoms, in some VM patients, headache and vestibular symptoms never occur together.

In cases without headache, other associated features like age at onset and duration of episodes indicate that vertigo is a migraine equivalent. Headaches accompany VM episodes in almost 50% of patients; some report head fullness or head

pressure without headache [16]. Interestingly, headaches during VM episodes are usually less severe compared to a typical migraine-related headache, thus patients are more bothered by the vertigo than by the headache [23]. Balci et al. [44] confirmed that headache severity was higher in migraineurs than in vestibular migraineurs. During VM episodes, other migraine features are also observed, often without a headache, including nausea, photophobia, phonophobia, osmophobia, allodynia and vomiting [5, 18, 42]. If a patient complains of nausea and vomiting during their typical migraine headaches, they tend to experience similar symptoms during VM attacks more frequently [8]. The higher prevalence of migraine with aura (MA) seen in VM patients may reflect an association of MA with vascular disease and posterior circulation [22]. We would like to underline that patients with vertigo should be directly asked about migraine symptoms during their episodes, as they often do not volunteer them [5].

Other symptoms

Some patients report tinnitus, fullness of the ear, palpitations, and even mild and transient hearing loss, during their episodes [18]. Several authors have described an accompanying Alice in Wonderland Syndrome — a rare and fascinating sensory perception disorder, with the symptoms of visual distortions, extrapersonal misperceptions, or somesthetic distortions [16, 45]. Some studies show that VM patients are significantly more anxious and agoraphobic, have depression symptoms and insomnia, have higher sensitivity to separation, and are prone to seek medical reassurance [16, 46].

Duration of vestibular attacks

The duration of episodes varies from a few seconds up to several days [8, 18]. According to Vuralli et al. [15], vestibular symptoms were described as short attacks lasting for seconds by 60.4% of VM patients, but the total time with repeated short episodes lasted between hours and days. Notably, patients with short-lasting attacks tend to have several such attacks every day [15, 19].

Triggers and aggravating factors

The top VM triggers are stress, sleep deprivation, bright lights, missing meals and weather changes; these do not differ from typical migraine triggers [16, 47]. Some attacks may be started by daily head and body movements, menstruation and sensory stimuli [5, 15]. Visually induced vestibular symptoms have been described by 71.3% of patients, and positional motion-induced vestibular symptoms by 82.2% of patients [15].

Neuro-otological examination

A neurological as well as an otological examination is usually normal between episodes, although mild central deficits can exist, mostly in patients with a long history of VM [14, 43, 48]. During VM episodes, most patients have a central spontaneous or positional nystagmus [48, 49]. The nystagmus has no latency

period, is usually persistent and non-fatigable, and is markedly reduced by visual fixation [50, 51]. In addition, imbalance is a regular finding during acute attacks, including a positive Romberg test and gait ataxia [17, 49].

Differential diagnosis

VM must be differentiated particularly from Meniere's Disease (MD) and benign paroxysmal peripheral vertigo (BPPV). Regardless of the many differences between these disorders, one should remember that, at very early stages, most symptoms can be quite similar. Meniere's Disease is a disorder with attacks of coexisting vertigo, tinnitus, aural fullness, and hearing loss [32]. MD is known to be associated with migraine, as the lifetime prevalence of migraine is higher in the MD group compared to controls, and almost half of MD patients may experience headache, photophobia or aura during Meniere's attacks [32, 52]. Radtke et al. [52] reported that the lifetime prevalence of migraine was higher in the MD group (56%) than controls (25%, $p < 0.001$); they concluded that migraine and MD might share a similar pathophysiology.

In a very large group of 911 MD patients, Pyykkö et al. [53] found migraine in 20.9% of subjects while headache was reported by 42.9%. Notably, migraine patients complained of more severe MD symptoms than non-migraineur patients with MD. Shin et al. [54] discovered an incidence of VM in 35% of MD patients, mostly with a probable MD diagnosis. One study revealed that MD patients with VM have better hearing and more frequent vertigo episodes than individuals without VM [55]. Interestingly, some patients may experience the clinical features of both VM and MD during their attack. This has been described in the literature as 'VM/MD overlapping syndrome' [56].

Nevertheless, many differences between these two disorders exist, as individuals with MD suffer mostly from accompanying auditory symptoms, anxiety, and palpitations, while migraine sufferers experience typical headache, photo-/phonophobia or aura [57, 58]. Endolymphatic hydrops is the primary pathological finding in MD patients, although Gurkov et al. [59] using MRI found endolymphatic hydrops in 20% of patients with VM and auditory symptoms. Also, other vestibular tests do not completely segregate individuals with MD from those with VM [60, 61].

BPPV is another common cause of vertigo and should be considered in the differential diagnosis of VM patients. Notably, migraine is more common in patients with BPPV compared to controls, and having migraine is linked with an increased risk of developing BPPV [11, 14]. For differentiation, positional testing which stimulates vertiginous attacks and elicits nystagmus may be helpful, including the Dix-Hallpike manoeuvre or the supine roll test for the horizontal canal variant [62].

Other disorders that rarely may mimic VM, and should be taken into consideration during the differentiation process, include: vertebrobasilar TIA, vascular compression of

the eighth nerve, autoimmune inner ear disease, insufficient compensation of unilateral vestibular loss, schwannoma of the eighth nerve, persistent postural-perceptual dizziness, and anxiety disorder [16, 26, 62].

Laboratory findings

Although no pathognomonic laboratory test that can verify a VM diagnosis exists, investigations should be done to rule out any other diagnosis. Many authors report minor signs of peripheral and central vestibular dysfunction, including pathological results in caloric and video head impulse testing (vHIT) as well as in tonal audiometry, cervical and ocular vestibular evoked myogenic potentials (VEMP), posturography or magnetic resonance imaging (MRI), but the findings are ambiguous [61, 63–71].

It is worth emphasising that abnormal vestibular testing results are also found in patients with migraine without a history of vestibular symptoms [72]. In most patients, tonal audiometry is normal [73]. The most consistent laboratory finding in VM is a unilaterally reduced caloric response as well as bilateral caloric hyporesponsiveness, but only in a small percentage of patients.

Notably, individuals with VM are significantly more likely to have an emetic response to caloric stimulation than other patients [74]. Almost half of VM patients have saccadic movements in vHIT [10]. Some studies have revealed that cervical VEMP can be used as a diagnostic test to differentiate MD from VM, because VEMP responses are symmetrically reduced on both sides in VM patients [75]. One study has demonstrated an absence of unilateral or bilateral VEMP response at 90 dB normal hearing level in almost half of VM patients, which was significantly higher than in healthy controls [65], while another showed reduced VEMP amplitudes compared to controls, suggesting that not only peripheral but also central vestibular structures are affected in VM patients [63].

Moreover, patients with VM have shown compromised body balance in static posturography tests, and higher visual dependency and low stability of the postural control system when maintaining quiet standing [67, 76]. Brain MRI is unremarkable in VM sufferers, although some studies found that grey matter volume of some brain regions of patients with VM is significantly larger than in patients with migraine or healthy controls, mostly areas linked with assessment, integration and pain expectations as well as those associated with mood and anxiety [16, 77]. Interestingly, other authors have found grey matter volume reduction in the temporal gyrus, mid-cingulate, dorsolateral prefrontal area, insula, parietal and occipital cortex [78].

Vestibular migraine treatment

Due to the lack of well-conducted randomised clinical trials, VM treatment recommendations are mostly based on the

guidelines for migraine therapy or observational studies, retrospective cohorts and open-label trials, anecdotal experience, and expert opinion [79–81]. We should remember that inconsistent definitions of VM based on old diagnostic criteria were used in many of these studies, thus examined cohorts were quite heterogeneous. Also, because of the absence of control groups in most of the studies it is difficult to determine if the improvement is spontaneous or due to treatment. Generally, migraine prophylactic treatments were effective in 77% of VM patients [19]. Surprisingly, even patients with headache and dizziness who did not meet VM diagnostic criteria benefit from prophylactic treatment of VM [82]. Regardless of the wide range of drugs that can be prescribed for the prophylactic treatment of VM, their effectiveness seems to be similar. Domínguez-Durán et al. [83], in a prospective study, reported that after five weeks of treatment, topiramate, amitriptyline, propranolol, flunarisine and acetazolamid were all equally effective in reducing vestibular symptoms, headache and the number of crises. A recent systematic review and meta-analysis revealed that although all treatment options showed improvements in outcome parameters, due to heterogeneity and the lack of standardised reporting on outcomes, no preferred treatment modality could be determined [84].

All studies regarding VM acute or prophylactic treatment (based on the modern VM definition) are summarised in Table 2. Several older studies regarding treatment of so called migraneus vertigo, with inconsistent groups, also exist [85]. They showed the effectiveness of acetazolamide, topiramate, lamotrygine, pizotifen, flunarisine or amitriptyline in reducing vertigo or headache [85]. Apart from pharmacological treatment, other treatments include lifestyle modification, trigger avoidance, and vestibular rehabilitation, although results are inconsistent [86, 87]. Interestingly, recent trials of specific migraine neuromodulation devices (trigeminal external nerve stimulation and noninvasive vagus nerve stimulation) have reported improvements in vertigo and headache severity without any side effect in the majority of VM patients [88, 89]. Because in recent times monoclonal anti-CGRP antibodies have been approved for migraine prevention, the question arises as to whether they might be effective in VM sufferers. Theoretically, they could play a role in vestibular physiology, because CGRP is also detected in human cochlear and vestibular end organs, and possibly plays a role in motion sickness [90, 91]. But as yet there is too little data on this topic.

Clinical implications

Vestibular migraine is a common cause of vertigo, and may affect 1–3% of the general population. It is a disabling disease of recurrent attacks of vestibular symptoms accompanied by migraine features and it occurs in patients with a current or previous history of migraine. The episodes can last for minutes, hours or even days, and may occur without a concurrent headache, which can increase misdiagnosis. VM often begins

Table 2. Overview of studies evaluating different drugs and devices in acute and prophylactic VM treatment

Author/year	Type of study	Type of treatment	Drug/ device used	Population/number of participants	Results	Conclusion
Beh S.C. 2020 [88]	Prospective study	acute	e-TNS (Cefaly)	VM (n = 19)	↓ vertigo and headache severity	effective
Dominguez-Durán E. 2020 [83]	multicentre prospective study	prophylactic	acetazolamide amitriptyline flunarizine propranolol topiramate	VM (n = 31)	↓ intensity of vestibular symptoms, headache and number of crises No significant between-group differences	effective
Bayer O. 2019 [92]	double-blind, randomised placebo-controlled trial	prophylactic	metoprolol	VM+ PVM n = 130 Neuhauser criteria 2001	discontinued due to poor participant accrual, no treatment benefit of metoprolol over placebo	Inconclusive findings
Beh S.C. 2019 [89]	retrospective chart review	acute	(nVNS)	VM 14	↓ vertigo intensity (46.9%) ↓ headache intensity (63.3%)	effective
Celik O. 2019 [79]	Prospective study	prophylactic	propranolol	VM 38	↓ severity, frequency, and number of attacks and disability scores ↓ quality of life	effective
Kaya I. 2019 [93]	Prospective observational	prophylactic	verapamil 80 mg	VM+MD n = 17	↓ vertigo and headache attack frequency and severity	effective
Liu F. 2017 [94]	single-blinded randomised comparison trial	prophylactic	venlafaxine 37.5 mg, flunarizine 10 mg, valproic acid 1,000 mg	VM PVM n = 75	↓ decreasing number of vertiginous attacks, venlafaxine had advantage in terms of emotional domains, valproic acid less effective than venlafaxine and flunarizine to decrease vertigo severity.	effective
Salmito M.C. 2017 [95]	Retrospective study	prophylactic	amitriptyline, flunarizine, propranolol and topiramate	VM n = 47	Effective, but no statistically significant difference between responses of prophylactic drugs	effective
Çelebisoy, N. 2016 [96]	Retrospective study	prophylactic	acetazolamide	VM n = 39	↓ frequency and severity of vertigo and headache attacks, effect was more prominent for vertigo frequency and severity.	effective
Salviz M. 2016 [97]	Randomised control trial	prophylactic	venlafaxine 37.5–150 mg propranolol 40–160 mg	VM n = 64	equal effectiveness as prophylactic drugs for ameliorating vertiginous symptoms, venlafaxine may be superior to propranolol in ameliorating depressive symptoms.	effective
Cassano D. 2015 [98]	retrospective, multicentric, open-label investigation	acute	almotriptan 12.5 mg	VM n = 18 (27 vertigo attacks)	55% complete disappearance of vertigo, 28% reduction of 50%+ 16% less than 50% reduction.	effective
Teggi R. 2015 [99]	Observational study	Prophylactic	cinarizine 20 mg + dimenhydrinate 40 mg	VM n = 22 Lempert criteria	↓ vertigo attacks from 5.3 to 2.1 and headaches from 4.3 to 1.7 (p < 0.0001); ↓ monthly attacks from 3.8 to 0.4	effective
Taghdiri, F. 2014 [100]	Retrospective open label	Prophylactic	cinarizine 75 mg	VM n = 24		effective
Mikulec A. 2012 [101]	retrospective chart review	Prophylactic	caffeine cessation + nortriptyline or topiramate	VM n = 44	Topiramate reduced symptoms in 25% of patients, nortriptyline in 46% of patients.	effective
Neuhauser H. 2003 [102]	Randomised control trial	acute	Zolmitriptan 2.5 mg	VM 8 Control 9	Improvement in vertigo in 38% compared to 22% of controls (CI 3–60%)	effective

VM — vestibular migraine; PVM — probable vestibular migraine; eTNS — external trigeminal nerve stimulation; nVNS — noninvasive vagus nerve stimulation

several years after typical migraine and there may be a considerable delay between onset of headache and vertigo. The diagnosis is based on the patient's clinical history.

According to the recent consensus reached by the International Headache Society and the Barany Society, in order to fulfill VM diagnostic criteria, attacks of vestibular symptoms connected with at least one migraine feature must be observed. Diagnosing vestibular migraine is a challenge, as there is lack of established confirmatory tests or biomarkers. The mechanism of vestibular migraine remains unclear and is still under investigation, but there is apparent interaction between the trigeminal and vestibular systems. Due to the lack of specific trials, treatment recommendations are based on migraine guidelines and several drugs seem to be effective, although there have been few randomised controlled trials.

Regardless of the published strict and detailed diagnostic criteria, this condition remains little known, and as a result it is underdiagnosed and undertreated.

Efforts should be made to educate medical communities and patients about this frequent disease and to encourage neurologists and ENT specialists to cooperate. It is important that every patient with vertigo of unknown origin should be directly asked about a past or present history of migraine, and about any migraine symptoms experienced during their vertigo episodes.

In cases with coexisting migraine and vertigo, a VM diagnosis should always be considered.

Future directions

There is a need for future studies in order to fully validate the diagnostic criteria and better characterise this disorder. Moreover, well conducted randomised trials must be performed to establish clear treatment recommendations. Finally, efforts should be made to elucidate and understand the pathophysiology of VM, from both the clinical and the scientific perspectives, so as to improve management of this surprisingly common disorder.

Conflicts of interest: *None*

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