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REVIEW ARTICLE

Migraine and Its Association with Vestibulocochlear Symptoms

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Abstract

Migraine is a common and potentially disabling neurological disorder caused by complex neurobiological mechanisms involving multiple areas and different neuronal networks in the central and peripheral nervous system. It is considered a genetically based disorder, characterized by recurrent episodes of moderate to severe headaches, typically unilateral, and frequently accompanied by symptoms such as nausea, vomiting, photophobia, and phonophobia.

It is now recognized that migraine can present with vestibular manifestations, with episodic vestibular syndrome, characterized by spontaneous vertigo and motion sickness, being the most common presentations. Recent studies have shown that migraine can present with other otoneurological symptoms such as tinnitus, hearing loss, aural fullness, ear pain, and sinus-like symptoms. A higher prevalence of migraine has also been observed in patients with other vestibulocochlear disorders, including Ménière's disease, benign paroxysmal positional vertigo, and persistent postural perceptual dizziness.

This article offers a practical review of the current literature on vestibular migraine and its relationship with other vestibulocochlear spectrum disorders and symptoms.

Keywords: Vestibular Migraine, Migraine, Vestibular, Auditory Manifestations.

1. Introduction

Migraine is a common and potentially disabling neurological disorder characterized by a complex neurobiology involving different areas and networks of the central and peripheral nervous system (1). It is caused by the interaction between genetic and environmental factors and is clinically characterized by recurrent episodes of moderate to severe headaches, typically unilateral, and frequently accompanied by symptoms such as nausea, vomiting, photophobia and phonophobia. (2,3). Currently, the association with spontaneous episodic vertigo is well known; however, many other vestibulocochlear symptoms and manifestations can be associated with common migraine, including motion sickness, tinnitus, hearing loss, aural fullness, otalgia, and sinus symptoms (4). A higher prevalence of migraine has also been observed in patients with other vestibulocochlear disorders, including Ménière's disease (MD), benign paroxysmal positional vertigo (BPPV), and persistent postural perceptual dizziness (PPPD). (2,4).

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This article offers a practical review of the current literature on vestibular migraine and its relationship with other vestibulocochlear spectrum disorders and symptoms.

2. Vestibular Migraine and Its Classic Vertigo

According to the criteria established in 2012 by the Bárány Society and the International Headache Society, vestibular migraine (VM) requires for its diagnosis (4):

A. At least five episodes that meet criteria C and D.

B. Having a history of migraine without aura or Migraine with aura.

C. Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours.

D. At least half of the episodes are associated with at least one of the following three migraine characteristics:

1. Headache with at least two of the following four features:
   a) Unilateral location.
   b) Pulsating type.
   c) Moderate or severe intensity.
   d) Aggravation with regular physical activity.
2. Photophobia and phonophobia.
3. Visual aura.

E. Not better explained by another diagnosis.

Classic vertigo in VM may have the following characteristics (4):

1. Spontaneous vertigo, manifested as internal vertigo (false sensation of one's own movement) or external vertigo (false sensation that the visual environment is spinning or flowing).
2. Positional vertigo, which appears after a change in head position.
3. Visually induced vertigo, triggered by a complex or large visual stimulus in the moving visual field.
4. Vertigo induced by head movement, produced during head movement.
5. Motion sickness (motion sickness is characterized by an altered sense of spatial orientation; other forms of motion sickness are not currently included in the classification of vestibular migraine).

The duration of episodes is usually highly variable (4). About 30% of patients have episodes lasting minutes, 30% have attacks lasting hours, another 30% last several days, and in the remaining 10%, attacks last only a few seconds. These attacks tend to occur repeatedly with head movements, visual stimulation, or after changes in head position. In these patients, the duration of an episode is defined as the total period during which short attacks are repeated (5,6). At the other end of the spectrum, patients may take up to four weeks to fully recover from an episode, although the main episode rarely lasts longer than 72 hours (5,6).

Although the definition of VM requires the concomitant presence of headache attacks associated with vestibular manifestations, in almost 50% of cases the headache and vestibular symptoms do not coincide temporally, being asynchronous pictures (4).

Vestibular symptoms are classified as moderate when they interfere with, but do not prevent daily activities, and severe when they prevent the performance of these activities (4).

In these patients, the clinical history and physical examination must not only meet established criteria but also rule out and suggest the presence of another vestibular disorder, before attributing the symptoms to VM (4).

3. Vestibular Migraine and Abnormal Eye Movements

Different types of abnormal eye movements have been described in patients with VM during periods of acute attacks or during interictal periods (7).

During acute attacks, central positional nystagmus may be found more frequently, with an incidence of up to 100% of attacks (8). In most cases it is of the apogeotropic or geotropic of low intensity, lasting approximately 40 seconds or more (7,9). In a study by Polensek et al, spontaneous nystagmus was observed in 19% of patients and head impulse-induced nystagmus in 35%, while positional nystagmus occurred in 100% of symptomatic patients (8).

The study by Radtke et al. described interictal oculomotor abnormalities in patients with VM, during the initial follow-up phase in 16% of patients and subsequently in up to 41% after 9 years (10). These findings were also corroborated by another study by Cass et al. (11).

In the interictal periods, central positional nystagmus, induced by head impulse, evoked by gaze or spontaneous, among others, may be evident (7). The

incidence of abnormal eye movements has been shown to increase after years of follow-up as evidenced in the study by Radtke et al.

4. Common Migraine and Benign Paroxysmal Positional Vertigo

BPPV is the most common cause of vertigo, with an approximate lifetime prevalence rate of 2.4% (6). Idiopathic BPPV is more common in older adults, with a peak onset between 50 and 60 years of age; it is also more common in women, with a female - male ratio of 2–3:1 (6). The typical symptom of BPPV is recurrent episodes of vertigo lasting less than a minute and triggered by head movements or changes in position (12). BPPV is thought to result from the displacement of otoliths from the otolithic organs into the semicircular canals, although the cause of this alteration remains unknown (13–15). The Dix-Hallpike, McClure, or Yacovino maneuvers induce nystagmus in patients with suspected BPPV (16). After a clinical diagnosis of BPPV, canalicular repositioning maneuvers are used to treat patients immediately (13,17).

Migraine and BPPV are among the most prevalent diseases in otoneurological clinics; however, because they do not share a common pathophysiological association, there is a lack of information about the association between these two diseases (16,18,19). A higher incidence of BPPV has been demonstrated in migraine patients than in the non-migraine population. Among the hypotheses for this possible association is that during headache attacks, vascular changes in the otolithic maculae would favor the detachment of otoconia (16).

According to a study by Shih et al, patients younger than 45 years of age who present with BPPV have a 2.96 times higher risk of developing migraine than the population without BPPV (95% CI: 2.30-3.80, $p < 0.001$) (13). In addition, the ICHD-3 considers BPPV to be one of the precursor syndromes of migraine (4). As it is considered benign paroxysmal vertigo of childhood (13).

5. Vestibular Migraine and Ménière's Disease

MD is a clinical diagnosis, with a spectrum of cochleovestibular symptoms (20). Originally described by Prosper Ménière in 1861 with his classic symptom triad of vertigo attacks, tinnitus and hearing loss, what is often omitted is that the fourth symptom in his original description was migraine (53).

Its prevalence is approximately 50 to 200 cases per 100,000 people, being more common in women and in people between 40 and 60 years of age (21,22). It is worth noting that patients with MD have a prevalence of migraine twice as high, compared to the healthy population (23).

Although the precise etiology of MD is unknown and cannot be limited to a single pathophysiological theory, endolymphatic hydrops is the most consistent pathological finding in patients with this disease (although other theories exist) (21,22). Currently, it is postulated that MD and VM share pathophysiological bases, such as trigeminovascular involvement, since the labyrinthine artery has trigeminal innervation (24). Therefore, there could be a reciprocal association and a notable overlap between VM and MD (25).

Patients with VM may also experience tinnitus, aural fullness, and variable sensorineural hearing loss; however, the hearing loss caused by VM does not typically involve low-pitched tones (24,26) as it does in MD. Similarly, photophobia and headache are common during MD attacks (2,26,27) .

Both entities may share similar pathophysiological mechanisms, such as alterations in the inner ear microcirculation, trigeminovascular system dysfunction, and immunological phenomena. This clinical overlap may complicate the differential diagnosis and requires careful patient evaluation, considering the temporal evolution of symptoms, audiometric findings, and response to treatment, especially during the first year of symptom onset. This is particularly true for MD, since it may present with vestibular symptoms only in the early stages of the disease (20). When MD criteria are met, particularly hearing loss documented by audiometry, MD should be diagnosed, even when migraine symptoms occur during vestibular attacks; only patients who have two different types of attacks, one meeting criteria for VM and the other for MD, should be diagnosed with both disorders (26).

6. Vestibular Migraine and Persistent Postural Perceptual Dizziness

VM is one of the most common precipitants of PPPD, a disorder recently defined by the Bárány Society, characterized by persistent dizziness and perception of instability, constituting a chronic vestibular syndrome, so the symptoms must be present most days for three or more months. Symptoms are exacerbated by upright posture, movement (active or passive) and exposure to complex or moving visual stimuli (28).

PPPD may develop in a quarter or more of VM patients, so it is possible to consider the overlap of PPPD with VM by terming it "chronic vestibular migraine" (29); however, to date, VM is defined by ICHD-3 as an episodic condition without persistent symptoms (4). In migraine patients who present with persistent motion- and vision-induced dizziness, a dual diagnosis of migraine (vestibular or non-vestibular) with PPPD is suggested (2,30).

Against the attribution of PPPD symptoms as a manifestation of "chronic vestibular migraine" is the fact that most patients with PPPD do not have migraine but another associated trigger (e.g., BPPV, vestibular neuritis, MD, among others)(2,30,31). Currently, three distinct groups of patients with migraine and chronic dizziness can be considered:

1. Patients with recurrent attacks of episodic vertigo with headache, superimposed with chronic dizziness and motion sensitivity (VM + PPPD).

2. Patients with chronic dizziness and chronic headache that may come and go and who meet criteria for migraine with or without aura (PPPD + chronic migraine other than VM).

3. Patients with chronic dizziness and headache as part of a broader set of chronic somatic symptoms (e.g., diffuse myalgias, chronic fatigue, chronic dyspepsia, etc.) who are better diagnosed with a functional neurological disorder (2,30,31).

7. Common Migraine and Tinnitus

About 38–46% of migraine patients may experience tinnitus once during their lifetime (23). The recognition of tinnitus— a highly prevalent symptom—when associated with spontaneous and recurrent vertigo episodes, can be diagnostically challenging, mainly for distinction from other vestibular disorders (23). Long migraine attacks can mimic MD and short attacks are like BPPV episodes (32). Tinnitus has been reported in both disorders, making it difficult to accurately identify its origin; however, migraine patients are three times more likely to present with tinn itus (33,34). Similarly, in a survey assessing vestibulocochlear symptoms of migraine, 20% reported tinnitus (34).

Tinnitus in migraine patients can be explained pathophysiologically because migraine presents central pathological changes such as vascular mechanisms, inflammatory responses and neurochemical responses that lead to anatomical and functional changes within the inner ear and central nervous system; generated vasospasm in the small arterioles of the cochlea and labyrinth, which has been widely considered as a possible contributor to the appearance of tinnitus in migraine patients (33–35).

8. Common Migraine and Hearing Loss

Subjective hearing change during migraine attacks is common, although it is unclear whether this is a peripheral or perceptual process (36). A nationwide database study of approximately 13,000 patients found that those with migraine were more likely to have subjective hearing loss (25.0% *vs.* 16.6%, $p < 0.001$) and tinnitus (34.6% *vs.* 16.9%, $p < 0.001$) compared with patients without migraine (37). Furthermore, a recent meta-analysis concluded that a history of migraine is a risk factor for sudden sensorineural hearing loss, with a pooled hazard ratio of 1.84 (95% CI: 1.11–2.57; $p < 0.001$) (38). Other analyses have also shown that the history of migraine is 35% to 80% more likely to develop sudden hearing loss (39,40).

9. Common Migraine and Hyperacusis

The association between hyperacusis and chronic migraine is well documented and is often described in the migraine literature as "phonophobia (41). It is a symptom strongly associated with headache severity and has been shown to improve with migraine prophylaxis (42,43).

10. Common Migraine and Auditory Fullness

It has been reported that up to 63% of migraine patients may experience aural fullness (AF) during an acute pain attack (44); furthermore, these patients are at increased risk of cochlear disorders such as tinnitus, sudden deafness and sensorineural hearing loss, with an almost three-fold increase in likelihood (39,45). Interestingly, half of MD patients, who experience tinnitus, hearing loss and aural fullness, also have a history of migraine, making it likely that MD is in fact a migraine-related phenomenon as described in the VM and MD section(39).

In a study evaluating patients with AF and classic migraine symptoms, 54% of patients with AF met ICHD-3 criteria for migraine and all patients had symptomatic improvement with migraine prophylactics such as verapamil (82%) or nortriptyline (18%) (46). Migraine features commonly associated with AF were further described, with visual motion sensitivity (91%), head motion sensitivity (81%),

sinus/facial pressure (72%), and phonophobia (72%) being the most common (46).

Another study showed that 74% of patients with headache-related AF met most of the criteria for common migraine , and also that in patients with AP and normal imaging studies (no mass or third window) the AF is most likely related to migraine (47).

11. Common Migraine and Earache

Patients with common migraine are more likely to report otalgia than patients with other headache types, and patients with otalgia are more likely to report headache than patients without otalgia (48,49). In a retrospective review of patients seen by otorhinolaryngology who presented with otalgia without another identifiable cause, more than half had a history of headache and 65% met ICHD-3 criteria for migraine with or without aura (50). Otalgia was triggered by classic migraine triggers in most patients, and most patients had improvement in otalgia with classic migraine therapies (39).

12. Conclusion

Numerous studies demonstrate that migraine is implicated in the generation of numerous otological and neurootological symptoms. The pathophysiology of this disorder may be due to the functional involvement of both peripheral and central vestibulocochlear structures.

Its high prevalence and wide variety of presentations mean that migraine should be considered as a differential diagnosis in every patient who consults for vestibulocochlear symptoms.

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