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## Diagnosis and Treatment of Vestibular Migraine

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### Abstract

Vestibular migraine is a disorder associated with symptoms of vertigo, imbalance or dizziness. The duration of attacks varies from seconds to days but usually lasting minutes to hours. The pathophysiology of Vestibular Migraine is not well understood, however, studies have shown a dysfunctional thalamo-cortical apparatus may play a role in the etiology. Diagnostic tests are usually normal in patients afflicted with vestibular migraine, however, they can be used to rule out other vestibular disorders. A detailed physical exam and history suggesting recurrent vestibular symptoms and a history of migraine may point to the diagnosis. Management of vestibular migraine has shown to be effective using a combination of lifestyle modifications, vestibular rehabilitation and medications.

**Keywords:** Vestibular Migraine; Migraine Treatment; Dizziness; Vertigo; Migraine.

### 1. Introduction

Vestibular migraine is one of the most common vestibular disorders affecting up to 1% of the general population [1] and 11% of patients in specialized vestibular clinics [2]. Among patients with dizziness and disequilibrium from tertiary clinics, vestibular migraine is considered the most common neurotologic condition [1]. Familial occurrence of vestibular migraine with autosomal dominant inheritance has been documented in several families [3], however, women were often affected two to three times more than men [4]. Vestibular migraine is not only prevalent amongst adults, but is also identified amongst the pediatric population of children and adolescents [5].

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Vestibular migraine presents with vertigo and dizziness in the presence of migraine. It is still widely unknown and therefore under-diagnosed in patients [6]. Based on the diagnostic criteria for vestibular migraine, the frequency of diagnosis was 7% in a study group of 200 dizziness clinic patients and 9% in a group of 200 migraine clinic patients [7]. More studies should be done to understand the complexity of vestibular migraine and its accurate diagnosis.

## **2. Epidemiology**

Migraine and dizziness are two common disorders that affect the general population, with a lifetime prevalence of 13-16% for migraine [8] and 7% for dizziness [9]. With the rise of several clinical studies, researchers have recently in the last decades begun acknowledging an association between the two disorders [10], also otherwise known as vestibular migraine.

The lifetime prevalence of vestibular migraine is estimated to be 0.98% [11], though people can suffer from it at any age throughout their lives [12, 13]. Vestibular migraine also tends to occur later in life, especially in patients who have already suffered earlier from migraine headaches and in patients who have previously been headache-free for years [7,14]. In terms of gender distribution, females seem to present with vestibular migraines more frequently than males. According to multiple studies, for every 1 male who presents with vestibular migraine, 1.5 to 5 females is reported to suffer from it [7, 14, 12].

## **3. Pathophysiology**

Russo and colleagues [15] described the thalamus as a pivotal neuroanatomical structure responsible for migraine. They explored the functional response of vestibular neural pathways using whole-brain blood oxygen level dependent (BOLD) fMRI during caloric vestibular stimulation [15]. The thalamus is a key structure in transmitting sensory input from the brainstem to the cortex and signal perception depends on modulating effects of this gate [15]. The thalamus exerts supreme importance in pain processing and cortical excitability control [16]. The role of the mediodorsal thalamus in vestibular migraine pathophysiology could perhaps signify a dysfunctional vestibulo-thalamocortical network, which overlaps with the migraine circuit [17]. Further, processing between the thalamus and the cortex integrates vestibular, visual, proprioceptive, and somatosensory afferent inputs [15].

The “Spreading Depression” theory may also be a key in understanding migraine attacks. Cortical spreading depression (CSD) is a short-lasting depolarization wave that moves across the cortex at a rate of 3-5 mm/min. A brief phase of excitation signals the reaction, following nerve cell depression and an efflux of excitatory amino acids from nerve cells [18]. Upon reaching the vestibular cortex or even the brainstem vestibular nuclei, the wave of CSD can result in causing vestibular symptoms. Further, current evidence indicates that CSD is a possible primary event in the trigeminovascular system (TGVS) activation in migraine with aura, and perhaps also, migraine without aura. Dysfunctional brainstem nuclei involved in the central control of pain may favor central trigeminal hyperexcitability. Additionally, the relationship between abnormal cortical activity and abnormal brainstem function remains unclear [19].

#### 4. IHS Criteria

Vestibular migraine is a migraine disorder that presents with vertigo, dizziness, and balance disturbance, with the attack duration ranging from seconds to days [20]. Aura phenomena, especially visual and somatosensory symptoms common to migraines with aura, may be present before, during, or after the headache during an attack [20], but symptoms vary greatly between patients. The International Headache Society [21] has identified criteria for migraine with and without aura, however, it does not include vestibular migraine as a distinct entity.

##### **MIGRAINE WITHOUT AURA**

- A. At least 5 attacks fulfilling criteria B-D below:
- B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)
- C. Headache has at least 2 of the following characteristics:
  - 1. Unilateral location
  - 2. Pulsating quality
  - 3. Moderate or severe intensity (inhibits or prohibits daily activities)
  - 4. Aggravation by walking stairs or similar routine physical activity
- D. During headache, at least 1 of the following:
  - 1. Nausea and/or vomiting
  - 2. Photophobia and phonophobia
- E. Not better accounted for by another ICHD-3 diagnosis.

##### **MIGRAINE WITH AURA**

- A. At least 2 attacks fulfilling criteria B and C
- B. One or more of the following fully reversible aura symptoms:
  - 1. visual
  - 2. sensory
  - 3. speech and/or language
  - 4. motor
  - 5. brainstem
  - 6. retinal
- C. At least two of the following four characteristics:
  - 1. at least one aura symptom spreads gradually over 5 minutes, and/or two or more symptoms occur in succession
  - 2. each individual aura symptom lasts 5-60 minutes<sup>1</sup>
  - 3. at least one aura symptom is unilateral
  - 4. the aura is accompanied, or followed within 60 minutes, by headache
- D. Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded.

**Figure 1:** The International Classification of Headache Disorders, 3<sup>rd</sup> edition(beta-version)[21]

#### 5. Diagnostic Criteria

Vestibular migraine patients typically present with a combination of migraine and vestibular symptoms [22]. Further investigation must assess whether there exists a strong association between these disorders, or if they simply manifest independently. According to the criteria of the International Headache Society, the vestibular

symptoms in patients with migraine are unrelated. As a result, many patients with both migraine and vestibular symptoms do not have a specific diagnosis to account for their vestibular symptoms [11]. Further study should be done to understand whether these patients have a recognized independent vestibular disorder such as Ménière's disease, BPPV, or vestibular neuritis.

Challenges to diagnosis of vestibular migraine include variation in the nature of patients' dizziness, the clarity of distinct attacks, the length of these attacks, and the time lapsed between the appearance of migrainous and vestibular symptoms [23]. Because physical examinations and laboratory assessments do not yield any pathognomonic findings, vestibular migraines are often diagnosed through exclusion of alternative diagnoses. The lack of physical examination or laboratory irregularities that would otherwise point toward other diagnoses could suggest a diagnosis of vestibular migraine. Besides exclusion, the diagnostic criteria for vestibular migraine are widely centered around a patient's history [23]

It was not until Neuhauser and colleagues developed diagnostic criteria for what is now termed vestibular migraine, a disorder in which vestibular symptoms are judged as part of the migrainous disorder itself [11]. According to the American Academy of Neurology, nearly 1% of the general population meets these criteria, which is five to ten times higher than the prevalence of Ménière's disease [24]. Neuhauser and his colleagues' criteria for vestibular migraine have been re-assessed favorably in a recent long-term follow-up paper. A structured diagnostic interview using the criteria has been used in studies of the clinical features, epidemiology, genetics, pathophysiology, and treatment of vestibular migraine.

The degree to which balance symptoms in the absence of headaches affect the functioning of a patient is a common diagnostic factor of vestibular migraine. If vestibular symptoms interfere with a person's daily activities, the balance symptoms are considered moderate. If these symptoms more frequently restrict one from performing daily activities, they are deemed to be severe. Both moderate and severe symptoms are characteristic of patients with vestibular migraine. If vestibular symptoms neither interfere nor restrict one's daily activities, differential diagnoses should be considered [25].

The International Headache Society's current diagnostic criteria for vestibular migraine includes -- A) A current or past history of migraine with aura or migraine without aura. B) At least five episodes fulfilling criteria C and D. C) Vestibular symptoms of moderate or severe intensity, lasting from 5 minutes to 72 hours. D) At least 50% of the episodes are associated with one or more of the following three migrainous features: 1. Headache with at least two of the following features: Unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity; 2. Photophobia and phonophobia; 3. Visual aura. E) Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder [21].

Further, in a study done by Murdin and colleagues, a lifetime prevalence estimates of dizziness ranged between 17 and 30%, and for vertigo between 3 and 10% [26]. Studies in the past have established an association between migraine and vertigo [27, 28, 29]. Vertigo is two to three times more common in patients with migraine than in patients with tension-type headaches. Symptoms of migraine are shown to be enhanced amongst patients already suffering from vertigo. The lifetime prevalence of migraine (16%) and vertigo (7%) lead to an expected

comorbidity in 11% of the general population. According to a recent study identified by the German National Health Survey, individuals with migraine were more likely to have vertigo and vertigo with an accompanying headache [27,28, 29].

## **6. Clinical Presentation**

Symptoms of vestibular migraine are episodic in nature, but the duration of attacks is highly variable, ranging from seconds to days [11, 30]. Headache associated with migraine generally precedes the onset of vestibular migraine [14]. The clinical presentation of vestibular symptoms that often correlate with migraine includes-but is not limited to - dizziness; motion intolerance with respect to head, eyes, and/or body; spontaneous vertigo attacks (often accompanied by nausea and vomiting); diminished eye focus with photosensitivity; sound sensitivity and tinnitus; balance loss and ataxia; cervicgia (neck pain) with associated muscle spasms in the upper cervical spine musculature; confusion with altered cognition; spatial disorientation; and anxiety/panic [31].

Vertigo associated with vestibular migraine includes positional, head motion-induced, and visually induced vertigo [25]. In temporal association with balance symptoms, hearing loss or ringing in the ears may be experienced. However, these symptoms may require additional thorough evaluation to consider the possibility of a nonmigrainous comorbid otologic disorder [25]. Other clinical signs include lightheadedness, a swimming sensation, heavy headedness, a rising sensation, a tingling sensation, a rocking sensation, and excessive motion-sickness susceptibility [23].

With half or more of the dizziness episodes, at least one of the following symptoms manifest. A headache initially occurs displaying two or more of these characteristics - one-sided location, pulsing, moderate to severe pain intensity, or escalated pain brought upon by routine physical activity. Second, elevated sensitivity to both normal room lighting and sounds is notable. The person reports the necessity to stay in a room with minimal light and sounds, avoiding sunlight and all noise. Third, visual aura is experienced, such as visual scotoma or visual hallucination [25].

Another common sign that clinicians can use to help in accurately diagnosing vestibular migraines is nystagmus, or involuntary eye movement. A recent study from 2010 identified eye movement findings in vestibular migraine patients while symptomatic during an attack; symptomatic subjects were reported to have difficulty in exhibiting smooth eye pursuit [32]. Interestingly, all patients exhibited positional nystagmus, which either happened spontaneously or were stimulated by headshaking [32]. Horizontal headshaking was identified to be the most common direction that triggered nystagmus. Other less common types of positional nystagmus recorded in the study included vertical nystagmus and torsional nystagmus [32]. Spontaneous positional nystagmus was also commonly reported and the eye movements during a vestibular migraine attack were of low velocity and sustained [32]. Furthermore, symptoms of migraine may occur simultaneously with symptoms of vestibulopathy [11]. Vestibular migraine symptoms are often identical to those of other sources of dizziness, including and most notably Ménière's disease and Benign Paroxysmal Positional Vertigo (BPPV) [23].

## **7. Physical Exam/Testing**

Vestibular migraine is often challenging to diagnose when the patient is asymptomatic, as many symptoms overlap with other conditions. Discerning peripheral vs. central causes of vertigo with objective testing is crucial for accurate diagnoses. The Dix-Hallpike test and the head thrust may be performed for vestibular assessment. If these tests remain negative, central causes of the vertigo, such as, stroke, tumor, Multiple Sclerosis, or Migraine, must be thoroughly investigated. However, if the results of these exams are positive, BPPV should be considered and managed accordingly. The presence of nystagmus on physical exam can be a valuable tool for diagnosis. Patients on an asymptomatic day were reported to have normal eye movements and extraocular motion [32]. Studies have also found that ten to twenty percent of vestibular migraine patients tend to have unilateral hypo-excitability due to caloric stimulation. Additionally, patients usually have imbalance and show central vestibular findings during the acute phase of vestibular migraine [13].

It is important to note head shaking and positional changes that provoke nystagmus during vestibular migraine attacks, imply there may be multiple anatomic sites involved in vestibular migraine [32]. In fact, PET studies from various research studies indicate many sites in the brainstem are persistently activated during migraine attacks; these include the periaqueductal gray, dorsal raphe nucleus, and locus coeruleus. Blood oxygen level-dependent fMRI studies also detected other interconnected structures of the brain affected during a migraine attack; these include the substantia nigra, red nucleus, and hypothalamus [32].

## **8. Differential Diagnoses**

Vestibular migraine often shares similar symptoms with various conditions, specifically those related to balance disorders. Vestibular migraine may present with purely positional vertigo, thus mimicking (BPPV). Latency of onset and rotational nystagmus are two key characteristics of a positive Dix-Hallpike test that is used to diagnose BPPV [33]. Positional nystagmus in vestibular migraine is similarly persistent; however for BPPV the symptomatic episodes tend to be shorter with vestibular migraine (minutes to days rather than weeks) and more frequent (several times per year with vestibular migraine rather than once every few years with BPPV) [33].

Over half of patients with Meniere's Disease have a history of migraine, but migrainous headaches, photophobia, or aura are not typical of Meniere's Disease [34]. Additionally, some literature suggests an association between Meniere's Disease and Migraine Associated Dizziness (MAD). MAD exhibits multiple symptoms including motion intolerance, motion-provoked vertigo accompanied by lightheadedness, spontaneous intervals of vertigo lasting from seconds to days, tinnitus, as well as unilateral and bilateral perceived fluctuant hearing [13].

## **9. Treatment**

Treatment of vestibular migraine is most effective when offered early in the course of the illness. Avoiding triggers and reducing stress and anxiety is an important part of overall management [35]. A patient diary should be kept including severity, duration, and frequency of vestibular migraine attacks.

Triptans are 5HT-1B/1D receptor agonists developed to treat migraine headaches [36]. According to a retrospective study conducted in San Diego, the ability of triptans to relieve symptoms of migraine-associated vertigo, is directly correlated to its ability to alleviate headache [37]. Migraine therapy could be beneficial to patients with migraine-associated vestibular disorders. Sumatriptan is an effective medication in improving headache and vertigo [37]. Almotriptan (ALM) was suggested to be both effective and safe in reducing vertigo and headache, according to a small clinical study [39]. In this study, 12.5 mg of ALM was administered to 26 subjects with vestibular migraine, to which 83% of the patients benefitted. 55% of patients reported complete disappearance of vertigo, 28% reported over 50% reduction, and 16% reported below 50% reduction. Pain was significantly reduced within the first month of treatment, and confirmed in the next two months. A limitation of this study, as acknowledged, was the small population size on which the results were based. The efficacy of almotriptan should be further examined in large scale, randomized, controlled clinical trials.

Non-steroidal anti-inflammatory drugs, or NSAIDs are typically less expensive than triptans, and in some studies have shown to perform nearly as well in managing symptoms [40]. Available evidence suggests that ibuprofen in 200 and 400 mg doses are effective in reducing headache intensity and rendering patients pain free at 2 hours [43].

Migraine prophylactic treatment has been shown to be widely effective in treating migraine-related vertigo [41]. In a study involving patients undergoing this treatment, a post-treatment analysis shows 69.3% reporting satisfactory control of symptoms, and 81.8% reporting at least a 50% reduction in the frequency of vertiginous episodes. Beta-blockers, such as propranolol and metoprolol are recommended for non-asthmatic patients with hypertension [41]. They, along with other prophylactic medications targeting headache treatment, were shown to be effective in treating vertigo and dizziness [37].

Antiepileptics have been used for migraine prophylaxis in both adults and children. Examples of such drugs include, topiramate, valproic acid, leviteracetam, zonisamide and gabapentin. Topiramate is considered one of the first-line options for treatment of migraine in adults.

## **10. Recommendations**

Additionally, alongside medication and vestibular rehabilitation, certain lifestyle modifications can benefit patients [36]. These include limiting the risk factors associated with migraine such as those relating to diet, sleep, stress and anxiety. Patients should strive to adopt a balanced diet, develop a regular sleep pattern, and acquire daily exercise habits. Stress reduction through relaxation methods, biofeedback, and cognitive-behavioral therapy are highly recommended [38]. Such lifestyle changes are crucial in the prevention, management, and effective symptom reduction of vestibular migraine [42].

## **11. Conclusion**

In our practice at the University of Southern California, we notice several trends in patients with Migraines and vestibular symptoms. A good history categorizing the symptoms and signs associated with the syndrome will help guide diagnosis and treatment.

When a history and physical exam cannot determine if vestibular symptoms versus Vestibular Migraine, and there are no contraindications to therapy, we treat using standard FDA approved medications

We noticed that if a patient's migraine headache and its associated symptoms including photophobia, phonophobia, nausea and vomiting respond to treatment, migraine-induced vestibular symptoms should have some degree of response.

Patients may also present with Migraine and Benign Paroxysmal Positional Vertigo or Ménière's disease. Primary vestibular signs and symptoms will persist after successful treatment of migraine. After good control of migraines has been achieved, withdrawal of medication may induce a return of both migraine symptoms and vestibular symptoms.

## **12. Disclosure of Interest**

Paul-Henri Cesar, Lilian Massihi, Helen Banh, and Heidi Banh declare there is no conflict of interest regarding the publication of this manuscript.

## **13. Geolocation Information**

Los Angeles, CA

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