Vestibular migraine

Abstract

Vestibular migraine (VM) is a syndrome of episodic recurrent vertigo or dizziness in patients with a current or history of migraine. Previously known as migraine-associated vertigo, migraine-associated dizziness, migraine-related vestibulopathy, migrainous vertigo, the term vestibular migraine perhaps best emphasises the prominent vestibular symptoms upon a background of migraine. In 2012, the International Headache Society and the Barany Society representing the international neuro-otological community published a first consensus on diagnostic criteria for VM (Table 1) [1]. In 2012, the International Headache Society and the Barany Society representing the international neuro-otological community published a first consensus on diagnostic criteria for VM (Table 1) [1].

Pathophysiology

Current models of VM pathophysiology are based on evolving theories of migraine, such as activation and sensitisation of trigeminovascular pathways, as well as brain stem and diencephalic nuclei [10,11]. Based on neurophysiological data, thalamocortical dysrhythmia (TCD) – altered rhythmic activity between thalamus and cortex leading to abnormal information processing – is also considered key to migraine pathophysiology [12,13]. Imaging studies have showed increased activity in the thalamus – known to participate in multimodal sensory sensitisation [14] – during vestibular stimulation in patients with VM [15]. A longstanding hypothesis suggests that VM episodes result from repeating depolarisation [16], akin to ‘visual aura’, but this cannot account for the more prolonged episodes of dizziness or vertigo commonly seen in VM.

From a molecular perspective, descending pathways from the monoaminergic nuclei control the sensory trigeminal unit, and several neuropeptides, such as calcitonin gene-related peptide (CGRP) and pituitary adenylate cyclase-activating polypeptide (PACAP), have essential roles in the activation mechanisms of the trigeminovascular pathways [17]. A differential proinflammatory signature in VM (namely elevated IL-1β, CCL2, OCL22, and CXCL1 levels) appears to differentiate VM from other vestibular disorders such as Ménière’s disease [18]. VM, similar to migraine headache, is commonly familial and is widely assumed to have a genetic susceptibility, with combined epigenetic, and environmental contributions [19].

Clinical features

Triggers

In one of the largest studies to investigate triggers in 1027 migraineurs, the commonest trigger was emotional stress (79.7%) [20]. Others have identified sleep disorders (over-sleep, lack of sleep, or change in sleep pattern) to be a trigger in 81% [21], but these studies did not include VM as a specific subgroup. A history of anxiety and depression has also been associated with a significantly increased risk of developing VM [4]. Patients with VM often have a significant past history of adverse experiences. This can include a mental health disorder (anxiety in 70%, depression in 40%) [22], as well as adverse experiences associated with migraine and disorders causing nausea and/or dizziness (Figure 1).

Symptoms

The diagnosis of VM relies on the clinical history. Frequently, patients presenting with vertigo do not volunteer a history of migraine, so it is important to specifically enquire about this.
The duration of vertigo varies widely from seconds to days, with further episodes occurring after days, months or less commonly, years. Nausea and imbalance are frequent associated features. Vertigo may occur as a prodromal aura before a migraine headache, occur with the migraine headache or independently. Most episodes have no temporal relationship with the headaches. Susceptibility to motion sickness has found to be enhanced in patients with VM [23] and particular attention should be given to a pre-existing history of motion sickness such as being unable to read in the passenger seat of a car due to nausea [24].

Another prominent feature of VM is visually induced dizziness, where challenging visual inputs (i.e., moving images, shopping aisles) can provoke vertigo or spatial disorientation [25]. Positional vertigo can occur with VM and has been reported in 24% of patients, in contrast to 67% of patients describing spontaneous (non-positional) episodic vertigo [26]. Head motion-induced dizziness has been described as a unique feature of VM [22] but is seen in patients with unilateral and bilateral vestibulopathies also. Persistent, almost constant dizziness has been reported in 51.1% of patients with VM [22], but such patients have more likely transitioned into persistent postural-perceptual dizziness (PPPD) – a functional neurological disorder characterised by persistent non-vertiginous dizziness and/or unsteadiness [27].

Audiological symptoms are commonly reported by patients with VM and can include otalgia, tinnitus, aural fullness or pressure and subjective hearing change in more than two-thirds of patients [28]. Hyperacusis [29], and fluctuating hearing loss has also been described [30].

Additional clinical history should include any migraine-specific precipitants of vertigo attacks including menstrual cycle, sleep disturbance, emotional stress, sensory stimuli (e.g. bright lights, intense smells and noise) [31].

**Signs**
Clinical examination in patients with VM is typically normal, particularly in the inter-ictal phase. Non-specific, non-localising oculomotor deficits have been described in VM interictally, such as smooth pursuit deficits in 48% of patients, spontaneous nystagmus in 10% of patients, and central positional or gaze-evoked nystagmus in 28% of patients [32]. When present, these abnormalities are typically subtle and other central disorders should be excluded when these findings are identified. During an acute episode, spontaneous and positional nystagmus has been recorded in 70% of patients, as well as unsteadiness during the symptomatic period [33]. VM and BPPV can co-occur in the same individual, and VM can mimic BPPV where there may be recurrent episodes of positional nystagmus and vertigo that tend not to resolve with repositioning manoeuvres [34].

**Investigations**
There is no specific diagnostic test for VM. Vestibular function tests have shown
a range of mild abnormalities but there are no consistent trends observed. Mild sensorineurial hearing loss has been described especially affecting the lower frequencies, but in such cases Ménière’s disease should be strongly suspected. Central auditory processing deficits are reported in VM with prolonged latencies on auditory brainstem response testing [35], but such findings are not specific for VM.

Management

Management of VM is based on recognised approaches for migraine. A dizziness diary can be useful in assessing an individual’s response to treatment and guide the timing of preventative therapies.

Treatment for acute episode

There is no evidence-based approach for acute treatment of VM, although many specialists will advocate the use of antiemetic medication, particularly for the treatment of accompanying nausea or vomiting. Contrary to migraine, there is inconclusive evidence for triptan use in VM. Zolmitriptan has been shown to have some benefit in a small pilot randomised placebo-controlled trial [36] and Rizatriptan reduces vestibular-induced motion sickness in patients with VM [37]. Where there is an unremitting, prolonged, distressing episode of VM, treatment with intravenous methylprednisolone has been found to be effective [38].

Prophylactic treatments

A recent systematic review and meta-analysis assessing the efficacy of preventative treatments for VM identified that antiepileptic drugs, calcium channel blockers, tricyclic antidepressants, beta-blockers, serotonin and norepinephrine reuptake inhibitors, as well as vestibular rehabilitation demonstrated improvements in outcome parameters [39]. However, due to significant heterogeneity of studies and lack of standardised reporting outcomes a preferred treatment modality could not be determined. A similar conclusion was reached by another prospective multicentre study evaluating acetazolamide, amitriptyline, flunarizine, propranolol or topiramate for VM, finding all similarly reduced symptom severity and frequency [40].

A prospective randomised non-placebo-controlled trial suggested flunarizine is effective in reducing the severity and frequency of vertigo attacks in VM patients [41], with positive patient experiences [42]. Another prospective randomised trial compared the effectiveness of venlafaxine and propranolol in VM patients and found both were effective at controlling vertiginous symptoms, however venlafaxine was better at controlling depressive symptoms [43], as also shown in another study [44].

Dietary modification with reduction in migraine triggers may be beneficial, and lifestyle modification should be considered where other triggers such as stress and irregular sleep pattern may be contributory. Regular exercise can reduce stress and improve sleep [30] and reduce the intensity and frequency of VM [45]. Given that up to 65% of patients with VM may exhibit anxiety disorder [46] there may also be a role for cognitive behavioural therapy in these patients.

Vestibular rehabilitation is a useful non-medicinal pharmacological approach to treating VM, with a review reporting significant improvement in all outcomes measures, including headaches [47], although good quality randomised controlled trials are lacking.

Conclusion

Vestibular migraine is a common balance disorder with likely genetic, epigenetic and environmental factors contributing to its development. Diagnosis can be challenging due to symptom overlap with other disorders and lack of a specific diagnostic test. A thorough history and examination is essential together with relevant investigations to rule out other neurological or otological disorders. There is growing evidence that the shared pathophysiology involves central sensitisation, maladaptive neuroplasticity, thalamocortical dysrhythmia, abnormal sensory gating, and abnormal functional connectivity with dysmodulation of multimodal sensory processing. Life experiences may drive maladaptive neuroplasticity of large-scale networks involved in sensory, attention and emotion processing.

2. Nitesh Patel, Consultant ENT Surgeon, Whips Cross University Hospital, Whips Cross Road, Leytonstone, E11 1NR, UK. E. nitesh.patel@nhs.net

3. References

4. Correspondence to: Nitesh Patel, Consultant ENT Surgeon, Whips Cross University Hospital, Whips Cross Road, Leytonstone, E11 1NR, UK. E. nitesh.patel@nhs.net

5. Conflict of interest statement: None declared

6. Disclosure: Anish Bahra is ACNR’s Headache Editor. This article has been subject to our normal peer review process, being peer reviewed by two expert, external reviewers prior to acceptance by the journal’s Co-Editors. Provenance and peer review: Submitted and externally reviewed

7. Date first submitted: 2/1/2022

8. Date submitted after peer review: 14/4/2022

9. Acceptance date: 14/4/2022

10. Published online: 12/5/2022


12. 1 The sensation of self-motion when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement.

13. 2 The sensation of distorted or impaired spatial orientation without a false or distorted sense of motion.