Journal of Medicines Development Sciences, 2026, Volume 11, Issue 3
https://bbwpublisher.com/journal/JMDS
ISSN Online:2382-6371
ISSN Print:2382-6363

Vestibular Migraine and Its Emotional and Sleep Comorbidities: Shared Mechanisms and Integrated Care

Yanan Meng[1], Jing Zhang[2]*

[1]Department of Neurology, The Affiliated Hospital of Hebei University, Baoding 071000, Hebei, China
[2]Department of General Practice, The Affiliated Hospital of Hebei University, Baoding 071000, Hebei, China

***Corresponding author:** Jing Zhang, hdfyzj@hbu.edu.cn

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Abstract: Vestibular migraine (VM) is a common cause of recurrent vertigo. Many patients with VM also report anxiety, depression, insomnia, and obstructive sleep apnea, and these problems are more frequent than in the general population or in patients with migraine alone. Standard care still focuses on vertigo and headache, and the emotional and sleep dimensions of VM are often under-recognized. Anxiety, depression, and sleep disturbance appear to influence one another and VM in both directions, increasing attack frequency, reducing quality of life, and complicating differential diagnosis. This review summarizes epidemiological evidence on these comorbidities, discusses shared pathophysiological mechanisms, including altered central vestibular and limbic network activity, serotonergic and GABAergic/glutamatergic dysregulation, and neurogenic inflammation—and outlines a practical diagnostic approach based on structured symptom assessment and standardized instruments. We then propose an integrated management framework that combines pharmacological prophylaxis and acute treatment, vestibular rehabilitation, neuromodulation, cognitive behavioral therapy, mindfulness-based interventions, and lifestyle modification. Recognizing and treating these comorbidities as part of VM, rather than as separate problems, may improve symptom control and quality of life.

Keywords: Vestibular migraine; Anxiety; Depression; Sleep disorders; CGRP

Online publication: September 16, 2026

1. Introduction

VM is a common cause of episodic vertigo and is frequently accompanied by anxiety, depression, insomnia, and obstructive sleep apnea (OSA). These comorbidities are more prevalent in VM than in the general population or in migraine without vertigo [1], and they are not merely consequences of recurrent dizziness. A meta-analysis of adults with vestibular disorders found higher rates of anxiety and depression in VM than in healthy controls [2], and a cross-sectional study reported higher symptom scores and poorer quality of life in

VM than in migraine without vertigo or healthy controls [3]. Sleep is similarly affected: during non-rapid eye movement (NREM) sleep, cyclic alternating pattern (CAP) activation is abnormal, sleep quality is poorer, and sleep is more fragmented [4]. Together, these problems increase attack frequency and severity, reduce quality of life, and complicate diagnosis and treatment [5]. This review argues that VM, emotional distress, and sleep disturbance are best understood and treated as one clinical problem rather than three.

2. Epidemiology and Comorbidity Profile

Affective symptoms are common in VM. In one epidemiological sample, nearly half of patients with VM reported an anxiety or depressive disorder [6]. Among 2,801 patients with newly diagnosed migraine, those who met the criteria for VM had a higher burden of anxiety and depressive symptoms [7]. In a series of 74 patients with VM, 48.6% had clinically significant anxiety and 32.4% had depressive symptoms [8].

The association appears to be bidirectional. A long history, variable symptoms, and recurrent attacks may make patients with VM more vulnerable to anxiety and depression than patients with other causes of dizziness. Anxiety and depression, in turn, can increase distress, worsen symptom severity, and raise attack frequency, so that each problem sustains the other [9]. Whether these relationships are causal, and in which direction, remains uncertain.

Sleep problems are equally common. In a cross-sectional study of 530 patients with VM, 62.64% had a comorbid sleep disorder, and sleep disturbance severity correlated with the intensity of headache and vertigo [10]. The most frequent complaints were difficulty falling asleep and headache on awakening [11]. OSA is also overrepresented in vestibular disorders, and moderate-to-severe OSA appears to be more common in patients with VM than in comparison groups [12].

3. Possible mechanisms of comorbidity

Several mechanisms may link VM with emotional and sleep symptoms; none is yet established as a complete explanation.

3.1. Abnormalities in central nervous system circuits and networks

The vestibular nuclei and trigeminocervical complex are connected with limbic regions such as the amygdala and anterior cingulate cortex and with hypothalamic and brainstem reticular nuclei that regulate sleep and arousal [13]. These pathways allow vestibular signals to influence emotion and sleep, and vice versa. Vestibular input to the amygdala may contribute to fear and anxiety, while vestibular-hypothalamic connections may affect autonomic function and sleep timing. Patients with VM show increased CAP duration and index, a higher proportion of A3 sleep, and less A1 sleep, consistent with thalamocortical hyperarousal and fragmented sleep [4]. These changes may trigger attacks and vestibular symptoms and may also worsen mood through effects on neurotransmitter balance and the hypothalamic-pituitary-adrenal (HPA) axis.

3.2. Imbalance of neurotransmitter systems

Serotonin (5-HT) links VM, vestibular function, mood, and sleep. It modulates pain transmission, vestibular processing, emotional stability, and sleep rhythms [14], and altered 5-HT signaling has been implicated in VM attacks, vestibular symptoms, and comorbid anxiety and depression [15]. Norepinephrine and dopamine

dysfunction may also contribute, particularly through limbic and prefrontal circuits [16]. The γ-aminobutyric acid (GABA)-glutamate balance is a further point of convergence. Reduced GABAergic activity is associated with anxiety, and excessive glutamatergic activity with stress and hyperarousal [17]. In VM, this imbalance may increase sensitivity to pain and vestibular input and may predispose patients to anxiety and insomnia.

3.3. Neuroinflammatory mechanisms

VM and migraine without vertigo share inflammatory and metabolic abnormalities, but inflammation appears more pronounced in VM [18]. Calcitonin gene-related peptide (CGRP), a key migraine neuropeptide, drives neurogenic inflammation and has been implicated in VM attacks; migraine-related inflammation may also impair inner ear function [19]. CGRP, TNF-α, and IL-1β promote vasodilation and plasma extravasation and may reach limbic and sleep-regulating centers through neural pathways or by crossing the blood-brain barrier. They could therefore contribute to anxiety, depression, and sleep disruption. Evidence in VM remains indirect.

4. Clinical presentation and diagnostic workup

4.1. Symptom overlap and diagnostic challenges

VM overlaps with anxiety disorders, depression, and primary sleep disorders. Recurrent vertigo, photophobia, phonophobia, and movement intolerance resemble the somatic symptoms of anxiety [1], while low mood and anhedonia can be mistaken for interictal fatigue, cognitive slowing, and functional impairment [1, 20]. Insomnia can both trigger and follow attacks, especially nocturnal or morning attacks [1]. Ménière's disease and persistent postural-perceptual dizziness (PPPD) should be excluded.

The diagnostic criteria for VM are recurrent moderate-to-severe vestibular symptoms lasting 5 minutes to 72 hours, with a history of migraine and migraine features during episodes (headache, photophobia, phonophobia, or visual aura) [23].

Anxiety presents with disproportionate tension and worry, fear or dread, irritability, poor concentration, restlessness, tremor, muscle tension, rapid breathing, repeated requests for reassurance, and heightened vigilance; depression presents with low mood, slowed thinking, memory and concentration problems, guilt, and psychomotor change [21]. Panic attacks, which often lead patients to seek care in emergency departments, involve sudden intense fear, loss of control, and autonomic symptoms such as palpitations, dyspnea, dizziness, choking, and impending doom [21]. Other somatic symptoms include insomnia, pain, fatigue, sweating, palpitations, chest tightness, dyspnea, throat or nasal obstruction, nausea, vomiting, abdominal pain, diarrhea, urinary frequency or urgency, eating disturbance, and sexual dysfunction [21].

Sleep disorders are assessed by insomnia symptoms (difficulty initiating sleep, frequent or early awakening, poor quality, reduced total sleep time, and daytime impairment) and by OSA symptoms (snoring with apneas, daytime sleepiness, non-restorative sleep, fatigue, and insomnia) [22].

4.2. Application of standardized assessment tools

(1) Vestibular assessment. The diagnosis of VM should follow the criteria of the International Headache Society and the Bárány Society and be supported by history and vestibular testing, including the video head impulse test (vHIT), caloric testing, and vestibular evoked myogenic potentials (VEMPs) [23,24]. The Dizziness Handicap Inventory (DHI) [25] and the Vestibular Migraine Patient Assessment Tool and

Handicap Inventory (VM-PATHI) [26] quantify vestibular disability.

(2) Anxiety and depression. Validated questionnaires should be used routinely to screen for clinically significant anxiety and depression. The Generalized Anxiety Disorder-7 (GAD-7) [27] and Patient Health Questionnaire-9 (PHQ-9) [28] are widely used for screening, while the Hamilton Anxiety Rating Scale (HAMA) [29] and Hamilton Depression Rating Scale (HAMD) [30] provide clinician-rated severity.

(3) Sleep. Sleep quality and sleep-disordered breathing require both subjective and objective assessment. The Pittsburgh Sleep Quality Index (PSQI) [31] and Insomnia Severity Index (ISI) [32] assess subjective sleep quality and insomnia severity. Questionnaires are insufficient for OSA, and polysomnography (PSG) remains the diagnostic standard. Combining the PSQI or ISI with PSG when clinically indicated gives a more complete picture and helps tailor treatment to sleep as well as to VM.

5. Integrated management strategy

5.1. Pharmacotherapy

Preventive drugs include topiramate, sodium valproate, flunarizine, and propranolol; these drugs have been associated with fewer vertigo attacks and better patient-reported outcomes [33]. Amitriptyline is useful when insomnia and anxiety coexist [33]. Selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) are used when serotonergic dysfunction is suspected [21], but evidence specific to VM is limited. Agomelatine may improve sleep and mood together, and mirtazapine is an alternative [34]. Orexin receptor antagonists improve sleep continuity without suppressing REM sleep [34]. These drugs may reduce both attack frequency and comorbid symptoms, although the evidence is not consistent.

Acute treatment includes NSAIDs or triptans for pain [35], antiemetics, and vestibular suppressants. Triptans may disturb sleep, so benefit and risk need to be weighed. Rimegepant has been reported to relieve headache, vertigo, unsteadiness, nausea, photophobia, and phonophobia during acute attacks [36]. Vestibular suppressants and benzodiazepines should be used only briefly because they may delay compensation [37].

5.2. Vestibular rehabilitation therapy

Vestibular rehabilitation therapy (VRT) is a core non-pharmacological treatment. Individualized programs aim to promote central compensation, improve balance and gait, reduce dizziness-related disability, and, indirectly, lessen anxiety and avoidance. In a prospective 8-week trial in patients with VM, individualized VRT improved Dynamic Gait Index and DHI scores and reduced falls on the Modified Clinical Test of Sensory Interaction and Balance [38]. Programs should target visual dependence, desensitize patients to movement-provoked dizziness, correct stiff postural strategies, and gradually restore daily activities.

5.3. Physical therapy and neuromodulation

Non-invasive brain stimulation, including repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS), has been studied in VM. Stimulation is usually directed at the vestibular cortex, which processes vestibular input, or at the prefrontal cortex, which regulates emotion. The aim is to modulate cortical excitability and thereby reduce migraine symptoms and improve sleep [39]; evidence remains preliminary. When OSA is present, continuous positive airway pressure (CPAP) reduces nocturnal hypoxemia and sleep fragmentation and may also lower the frequency of migraine-like headache [39].

5.4. Cognitive behavioral therapy and mindfulness-based stress reduction

Cognitive behavioral therapy (CBT) has established efficacy in insomnia and anxiety and can reduce catastrophic thinking, fear, and avoidance in VM [40]. Mindfulness-based stress reduction (MBSR) may improve distress tolerance and emotional regulation, although VM-specific evidence is limited.

5.5. Lifestyle management

Sleep hygiene, regular physical activity, regular meals, and avoidance of dietary triggers such as caffeine and tyramine may reduce vertigo and headache [41]. Relaxation training, including diaphragmatic breathing and progressive muscle relaxation, can lower autonomic arousal. Gradual aerobic exercise—brisk walking, swimming, or gentle yoga—may improve mood and reduce attack frequency through effects on neurotransmitter systems and neuroinflammation [42], although the evidence is indirect.

6. Conclusion

Anxiety, depression, and sleep disturbance are not incidental to VM. They share neural circuits, neurotransmitter systems, and inflammatory pathways with migraine and vestibular dysfunction, and they interact with VM in both directions. Limiting assessment and treatment to vertigo and headache leaves these mechanisms unaddressed. Structured screening with validated scales, together with functional vestibular assessment, should be part of routine care. Treatment that combines pharmacotherapy, vestibular rehabilitation, and psychological or behavioral interventions is more likely to produce durable symptom control and meaningful gains in quality of life than treatment aimed at vertigo alone.

Funding

Baoding Municipal Science and Technology Program (Project No.: 2541ZF270)

Disclosure statement

The authors declare no conflict of interest.

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