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REVIEW

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Migraine management for the otolaryngologist

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Abstract

Objective: To characterize migraine pathophysiology, presentation, and current treatment strategies, specifically in regard to vestibulocochlear manifestations of migraine.

Methods: Narrative review of available literature.

Results: Migraine disorder can be described as a spectrum of otologic manifestations, with vestibular migraine now recognized with fully-fledged diagnostic criteria. Otologic manifestations are theorized to be due, in part, to trigeminal innervation of the inner ear structures and calcitonin gene-related peptide (CGRP) expression within the labyrinth. Patients can experience vertigo, aural fullness, enhanced tinnitus, and hearing loss without the characteristic migraine headache, leading to under recognition of these symptoms as migraine-related. Meniere's disease, mal de débarquement syndrome, persistent postural perceptual dizziness, and recurrent benign paroxysmal positional vertigo have close associations to migraine and may exist on the migraine spectrum. Migraine treatment consists of two goals: halting acute attacks (abortive therapy) and preventing attacks (prophylactic therapy). Abortive medications include triptans, corticosteroids, anti-histamines, and anti-emetics. Pharmacologic prophylaxis in conjunction with lifestyle modifications can decrease frequency and severity of symptoms and include tricyclic antidepressants, calcium channel blockers, anti-epileptic medications, selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, beta-blockers, gepants, and monoclonal antibodies to CGRP. Promising evidence is emerging regarding the ability of migraine medications to positively treat the various otologic symptoms of migraine.

Conclusion: Migraine disorder manifesting with primarily cochleovestibular symptoms can be challenging to diagnose and manage for practicing clinicians. Patients with various vestibulopathies that are closely related to migraine may benefit from migraine treatment. Lifestyle choices and prophylactic medications are key to satisfactorily preventing acute migrainous attacks and improve function.

Kaitlyn A. Brooks and Karen Tawk contributed equally to this study.

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KEYWORDS
lifestyle modifications, migraine diagnosis and evaluation, migraine spectrum, prophylactic medications, tinnitus, vestibular migraine, vestibulocochlear migraine

1 | INTRODUCTION

Migraine disorder, estimated to affect 15%–18% of the population,[1] is a leading cause of disability worldwide in young and middle-aged adults.[2,3] Originally considered a vascular headache disorder, the understanding of migraine and its variable presentations have evolved tremendously.[4] A spectrum of disease, migraine disorder, and its associated neural dysfunction can cause numerous cochleovestibular complaints, bringing this neurological disease into the purview of otolaryngologists (Figure 1). An estimated 5%–7% of patients seen in neurotology outpatient clinics are vestibular migraneurs,[5] which is under diagnosed[6–8] due to the underrecognized intersection of migraine and other vertigo-causing pathologies. It is imperative that otolaryngologists feel confident diagnosing and managing this disabling disease.

2 | EPIDEMIOLOGY AND EFFECT ON PATIENTS

Vestibular migraine impacts an estimated 1%–2.7% of the population[9–11] and accounts for 25% of patients with vertigo.[8] Median age of vestibular migraine onset is quoted between 30 and 40 years, and women are affected three times more than men.[12–14] Patients who suffer from vestibular migraine are more disabled with lower health-related quality of life than migraineurs without vestibular symptoms.[10,12,15,16]

3 | PATHOPHYSIOLOGY

Migraine attacks are thought to occur in multiple phases: premonitory phase, trigeminal activation, cortical spreading depression, and sustained inflammation.[17] Activation of the trigeminal neurovascular complex is considered a forefront mechanism underlying migraine[1] and is mediated by endogenous signaling neuropeptides inducing neurogenic and meningeal inflammation.[8,17] Cortical spreading depression is a slowly propagating wave of neuronal depolarization accompanied by ion and electrical charge fluctuation followed by a period of depressed activity[17]; this phenomenon is thought to be responsible for migrainous auras and could play a role in hypersensitivity to surrounding stimuli.[8]

3.1 | Pathophysiology specific to vestibular migraine

The exact mechanism for vestibular migraine remains debated, but it is likely a peripheral manifestation of a central process in which the vestibular system is the end-organ affected. Leading theories include neurovascular inflammation secondary to an initial trigeminal neurovascular complex activation propagated to the inner ear,[18–20] genetic abnormalities in neuronal membrane ion channels,[21] and hyperactivation of cortical sensory processing sites due to cortical spreading depression.[18,21] Calcitonin gene-related peptide (CGRP) is expressed in trigeminal neurons and efferent synapses of hair cells in the cochlea and semicircular canals. This neuropeptide is currently thought to play a role in nociception and cause vasodilation.[22] Functional magnetic resonance imaging has identified that chronic migraineurs have abnormal activity at the amygdala, thalamus, and temporal cortex, even when not experiencing an attack,[23] suggesting pathology regarding nociceptive processing.

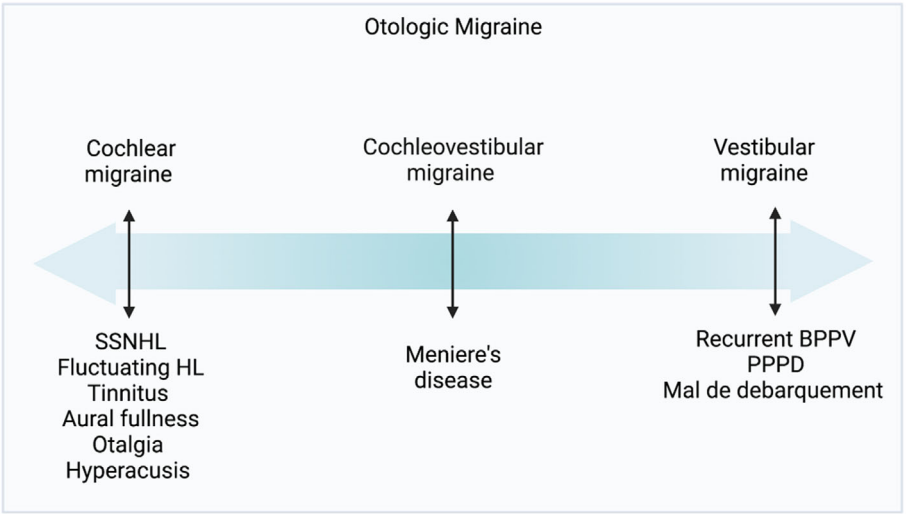

FIGURE 1 Spectrum of migraine inner ear disease. Created with BioRender.com.

Family studies focusing on vestibular migraine report autosomal dominant inheritance with incomplete penetrance but with high penetrance reported in women.[19,20] Familial prevalence rates of vestibular migraine have been shown to be 4–10 times higher than that of the general population.[24] Vestibular migraine is suspected to be a complex multigenic disease with significant variability in presentation.[8,25]

TABLE 1 Bárány Society and International Headache Society consensus criteria for vestibular migraine.[26]

Bárány Society Vestibular Migraine criteria	
Vestibular migraine	
A	At least five episodes with vestibular symptoms of moderate or severe intensity, lasting 5–72 h
B	Current or previous history of migraine with or without aura according to ICHD-3
C	One or more migraine features with at least 50% of the vestibular episodes:
1	Headache with at least 2 of the following: unilateral, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity
2	Photophobia and phonophobia
3	Visual aura
D	Not better accounted for by another vestibular or ICHD diagnosis
Probable vestibular migraine	
A	At least five episodes with vestibular symptoms of moderate or severe intensity, lasting 5–72 h
B	Only one of B or C criteria from VM diagnosis is fulfilled
C	Not better accounted for by another vestibular or ICHD diagnosis

Abbreviation: ICHD, International Classification of Headache Disorders.

4 | DIAGNOSIS AND SYMPTOMATOLOGY

4.1 | Vestibular migraine diagnostic criteria

Vestibular migraine is defined as recurrent, episodic vertigo caused by migraine and excludes dizziness due to other organic vestibular and non-vestibular causes.[8,26] This disorder is characterized by episodic vertigo with associated migraine features in patients with a history of migraine. The Bárány Society diagnostic criteria are listed in Table 1.[26] Vestibular symptoms can include spontaneous vertigo (internal or external), positional vertigo, visually-induced vertigo, and head motion-induced vertigo with and without nausea.[26] Patients can experience vestibular symptoms with and without headache, and may even experience aura without the characteristic headache.[27] Vestibular migraine is juxtaposed against common migraine due to misleading patient reports of attenuated headache, scalp tingling, head pressure, sinus pressure, or absence of headache altogether. Moderate vertiginous symptoms interfere with daily activities but do not prohibit function. Vestibular symptoms are classified as severe if daily activities cannot be executed.[26] The current criteria may be too stringent and possibly misclassifies patients with vestibular migraine, but do not meet the criteria for migraine headaches.[28]

For patients who experience migraine episodes for at least 15 days a month with interictal symptoms, the term chronic migraine is used. Patients with interictal vestibular symptoms can be considered akin to chronic migraineurs, but with vestibular symptoms rather than headaches. Vestibular symptoms are more common in patients with chronic migraine symptoms.[15] Figure 2 shows the type and frequency of symptoms experienced by patients with vestibular migraine.[5,29,30] Episodic vertigo episodes can be spontaneous, positional, visual, or a mixed picture.[5,8] Over half of vestibular migraine patients experience episodic spontaneous and positional vertigo.[5]

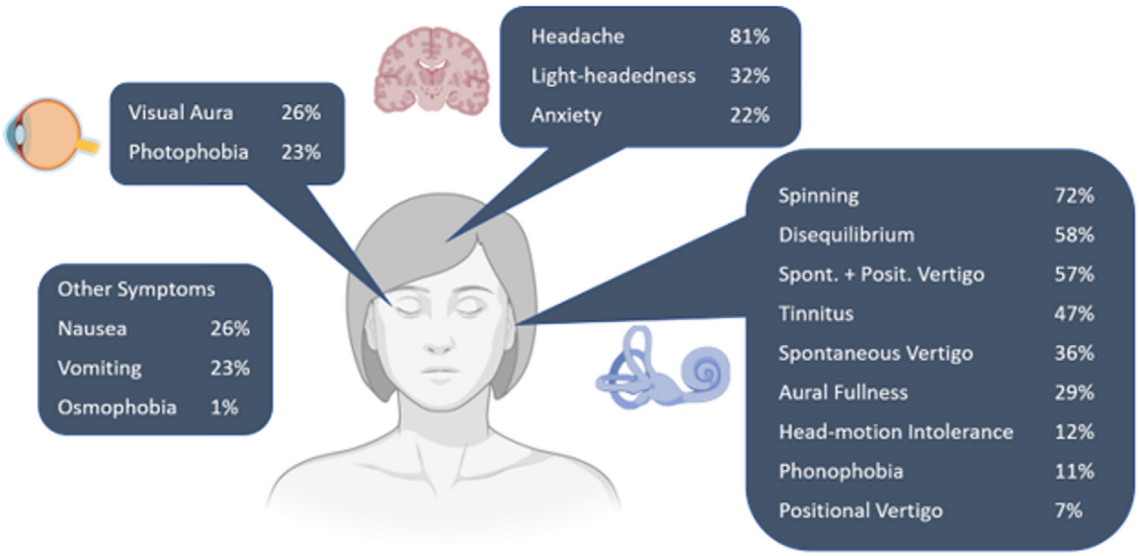

FIGURE 2 Symptomatology among vestibular migraine patients. Data from Young et al.,[5] Preysner et al.,[29] Lepcha et al.[30] Created with BioRender.com.

Patients can also experience interictal head motion and visually-stimulated dizziness.[6]

4.2 | Cochlear migraine

Aural symptoms, such as increased tinnitus, aural pressure, and subjective hearing loss,[5,6,12] can accompany vestibular migraine in a bilateral or unilateral configuration, raising the question whether vestibular migraine should be termed vestibulocochlear migraine. Lai and Liu[31] described a subset of patients with long-term, unilateral fluctuating hearing loss associated with aural fullness and tinnitus that did not meet the criteria for vestibular migraine.[31] This group of patients also reported migraine features and family histories pertinent for migraine. Shi et al.[12] demonstrated that tinnitus, otalgia, and hearing loss were connected among vestibular migraine patients.[12] Similarly, Ma et al.[32] detailed a close relationship between migraine, sudden sensorineural hearing loss, deafness, and tinnitus.[32] Goshtasbi et al.[33] found that 35.6% of patients with migraine reported tinnitus.[33] Twenty-one percent of patients with vestibular symptoms exhibited unilateral or bilateral objective hearing loss; these patients experienced a higher rate of aural-related symptoms and shorter periods of vertigo.[12] Cochlear migraine was introduced in 2018 to explain the relationship between migraine and auditory symptoms. Although vestibular migraine is fully recognized with diagnostic criteria, cochlear migraine requires further study. Studies dedicated to explaining and characterizing the combined vestibulocochlear manifestations of migraine are continuing to emerge.

5 | EVALUATION

The history of present illness should include prominent vestibular symptoms, timing, personal and family history of migraine, and headache symptoms. A comprehensive neuro-otologic assessment should be performed for any patient presenting with vertigo. For patients who suffer from vestibular migraine, otoscopy is typically normal, though some patients with cochlear migraine, on our clinical observation, will have dilation of the capillaries over the malleus on the involved side. Patients actively experiencing an acute vertiginous attack will likely have low-amplitude spontaneous, positional, or mixed-pattern nystagmus upon examination.[5,34] Neurological exams between attacks are typically normal,[18,34] although nystagmus has been reported.[35] Audiologic assessment is warranted for patients who report aural symptoms or subjective hearing changes.[36] As previously mentioned, hearing loss on pure tone audiometry has been reported in up to 21% of patients with vestibular symptoms.[12,35,37] Hearing loss pattern is typically mild, sensorineural, and centered in the low-frequency range; it can present both unilaterally, which is more common, and bilaterally.[12] Most patients who suffer from VM will have normal audiometric testing results and normal results on caloric and vestibular evoked myogenic potential testing.[5]

Psychiatric co-morbidities, notably generalized anxiety, major depressive, and panic disorders, have been shown to be related to vestibular symptoms.[6,38] Patients with vestibular migraine experience higher levels of anxiety,[38,39] which can lead to a positive feedback loop of continued heightened awareness of vestibular symptoms and anxiety.[8] Screening for anxiety in patients with vestibular migraine has been proposed to identify patients who would benefit from prophylactic migraine treatment that has been repurposed from psychiatric treatment.

6 | MIGRAINE AND OVERLAP WITH OTHER VESTIBULOPATHIES

6.1 | Mal de débarquement syndrome

Mal de débarquement syndrome (MDDS) is a distinct type of disequilibrium characterized by the sensation of rocking or swaying, as if on a boat, typically starting after prolonged exposure to passive motion following travel by air, boat, or car.[40] Symptoms need to be present for at least 1 month.[41] The sensation improves with passive motion. MDDS is closely associated with migraine and may even be part of the migraine disorder spectrum[40,42]; the pathognomonic symptom, rocking and swaying sensation, worsens during acute vestibular migraine attacks.[43] Patients who experience both MDDS and vestibular migraine exhibit increased severity of attacks and disability.[43] Patients with MDDS have been shown to respond well to migraine therapy.[40]

6.2 | Benign paroxysmal positional vertigo

Benign paroxysmal positional vertigo (BPPV) is characterized by fleeting, seconds-to-minutes-long vertigo occurring with head position changes. The general consensus of causative mechanism is semicircular canal otolith mispositioning[44] and is usually idiopathic or secondary to other vestibular pathology. Co-incidence of migraine disorder in patients with BPPV of unknown etiology is three times higher than in the general population, and onset of BPPV at a younger age is strongly associated with a migraine history.[45,46] It has been suggested that the two diseases may have similar pathophysiology and that BPPV may be on the disease presentation spectrum for migraine with vestibulocochlear symptoms.[44,47,48]

6.3 | Meniere's disease

Meniere's disease (MD), or endolymphatic hydrops, is an inner ear disorder caused by increased intralabyrinthine pressure.[49–51] Symptoms of MD include aural pressure, fluctuating hearing loss, and low-pitched tinnitus accompanied by episodic vertigo lasting 20 min–12 h,[50] which falls within the accepted time frame for VM attacks. Audiometric testing reveals a unilateral or asymmetric low-frequency

sensorineural hearing loss, and hearing loss on audiometry has been suggested as a method for distinguishing MD as a diagnosis over vestibular migraine.[52] Asymmetric hearing loss, tinnitus, and aural fullness have been found to be more prevalent in MD than in VM.[52] It has been demonstrated that patients with MD respond well to migraine therapy.[53]

6.4 | Persistent postural perceptual dizziness

Persistent postural perceptual dizziness (PPPD) is a chronic vestibular dysfunction hallmarked by visually-induced vertigo, space motion discomfort, and subjective chronic dizziness.[54] A majority of patients with PPPD suffer from migraine.[55] Co-morbid anxiety and depression are considered to play key roles in the pathogenesis of PPPD and often require treatment for optimal treatment outcomes. Vestibular migraine may interact with PPPD as a trigger or perpetuating co-morbidity[56]; treatment for PPPD and co-morbid vestibular migraine requires a holistic biopsychosocial approach with medical therapy targeted at migraine and co-morbid psychiatric diagnoses.[57] Vestibular rehabilitation (VR) with habituation exercises has been shown to be beneficial.[58,59]

7 | VESTIBULAR MIGRAINE TREATMENT

Vestibular migraine is challenging to treat with no widespread consensus because of the lack of evidence-based treatment and randomized controlled clinical trials.[9,18,60] As a result, the current treatment options for vestibular migraine rely on those used for migraine headaches.[18,61,62] Abortive medications are used to alleviate symptoms during an acute attack, whereas prophylactic medications are considered in patients with frequent and/or severe attacks that impact their quality of life.

7.1 | Abortive therapy

The goal of abortive therapy is to halt or lessen the severity of an acute vestibular attack. There are few studies investigating abortive medications and their efficacy in vestibular migraine attacks. Triptans (selective serotonin receptor 5-hydroxytyramine receptor one agonists) have been shown to be effective in managing migraine with vestibular symptoms (Table 2).[37,63,64] In our clinical practice, we have not found triptans to be as effective against vertigo, especially for those with chronic symptoms.

Other medications used for acute migrainous vertigo include those routinely used for acute vertigo from other causes. Despite inconclusive evidence detailed in McCoul et al.,[65] systemic corticosteroids have been broadly used in acute vestibular neuritis and sudden sensorineural hearing loss.[65] Based on a 65-year systematic review, corticosteroids are as effective as migraine abortive medications for acute attacks.[66] Prakash and Shah detail improvement of migrainous vertigo after intravenous methylprednisolone administration in acute and chronic vertigo recalcitrant to other medications. Antiemetics (ondansetron) and antihistamines (meclizine, diphenhydramine) are commonly employed despite sparse well-established evidence of their efficacy in treating vestibular migraine.[22,67] Similarly, antidopaminergic medications (metoclopramide) are effective in treating migrainous nausea.[68]

7.2 | Pharmacological prophylaxis

The goal of prophylactic medication for vestibular migraine is to reduce attack frequency and severity. Most of the medications used are extrapolated from the prophylactic medications for treating migraine headaches with no evidence of superiority of any medication over others.[67] These treatments include beta-blockers (propranolol, metoprolol),[69–71] antiepileptics (topiramate, lamotrigine, valproic acid),[72] calcium channel blockers (verapamil, flunarizine, lomerizine, cinnarizine),[30,71,73,74] tricyclic antidepressants (amitriptyline, nortriptyline),[71,72] serotonin-norepinephrine reuptake inhibitors (SNRI) (venlafaxine),[69] and selective serotonin reuptake inhibitors (SSRI) (paroxetine).[75] Flunarizine and cinnarizine are not currently available in the United States. If dizziness is not controlled with one class of medication, another class should be considered to achieve symptomatic control for a few months before tapering. Some have suggested maintenance therapy for a year prior to tapering off medication.[76] The algorithmic approach to vestibular migraine prophylactic management is detailed in Figure 3. Outcomes from prophylactic management of VM have been encouraging, with many cohort studies reporting a majority of patients with symptom resolution, reduction in frequency of attacks, and satisfactory symptom control.[30,35,69,77,78] VM patients anecdotally are very sensitive to medication changes and side effects; common side effects are detailed in Table 3. The American College of Obstetricians and Gynecologists recommends avoiding prophylactic migraine treatment in pregnancy.[79]

7.3 | New and emerging therapies

Gepants (CGRP receptor antagonists) are a new class of medication with the potential to absolve and possibly prevent acute attacks. Rimegepant, ubrogepant, and zavegepant have been shown in clinical trials to be efficacious for acute attacks; rimegepant and atogepant both have promising preventative applications.[100] Trials to show non-inferiority of gepants when compared to triptans have not occurred yet, and studies assessing the efficacy of gepants in patients with vestibular migraine are yet to be conducted despite promising animal studies.[101,102]

Russo et al.[103] were the first to report the efficacy of anti-CGRP monoclonal antibodies (erenumab, fremanezumab, or galcanezumab) on vestibular migraine, particularly when started early in the disease course. They found that nearly 80% of the patients experienced at least a concurrent 50% reduction in migraine days, vertigo days, and migraine disability assessment scores.[103] Furthermore, Lasmiditan, a

TABLE 2 Triptan dosing and efficacy in acute VM attack.

Abortive therapy				
Medication	**Dose**	**Study**	**Study type**	**Effect found**
Almotriptan	12.5 mg PO within 1 hour of attack	Cassano et al.	Retrospective, multicenter open-label ($n = 18$)	55% complete vertigo resolution, 28% of patients reported over 50% reduction of vertigo
Sumatriptan	Unspecified	Bikhazi et al.	Retrospective survey ($n = 111$)	Excellent efficacy at improving headaches (4) and vertigo (3) on a numerical scale from 1 to 4
Zolmitriptan	2.5 mg PO during attack	Neuhauser et al.	RCT ($n = 10$)	38% of patients had improvement in vertigo at 2 hours following treatment versus 22% for placebo, results inconclusive due to limited power

Note: Sources include Cassano et al.,[64] Bikhazi et al.,[37] and Neuhauser et al.[63]
Abbreviation: PO: per os.

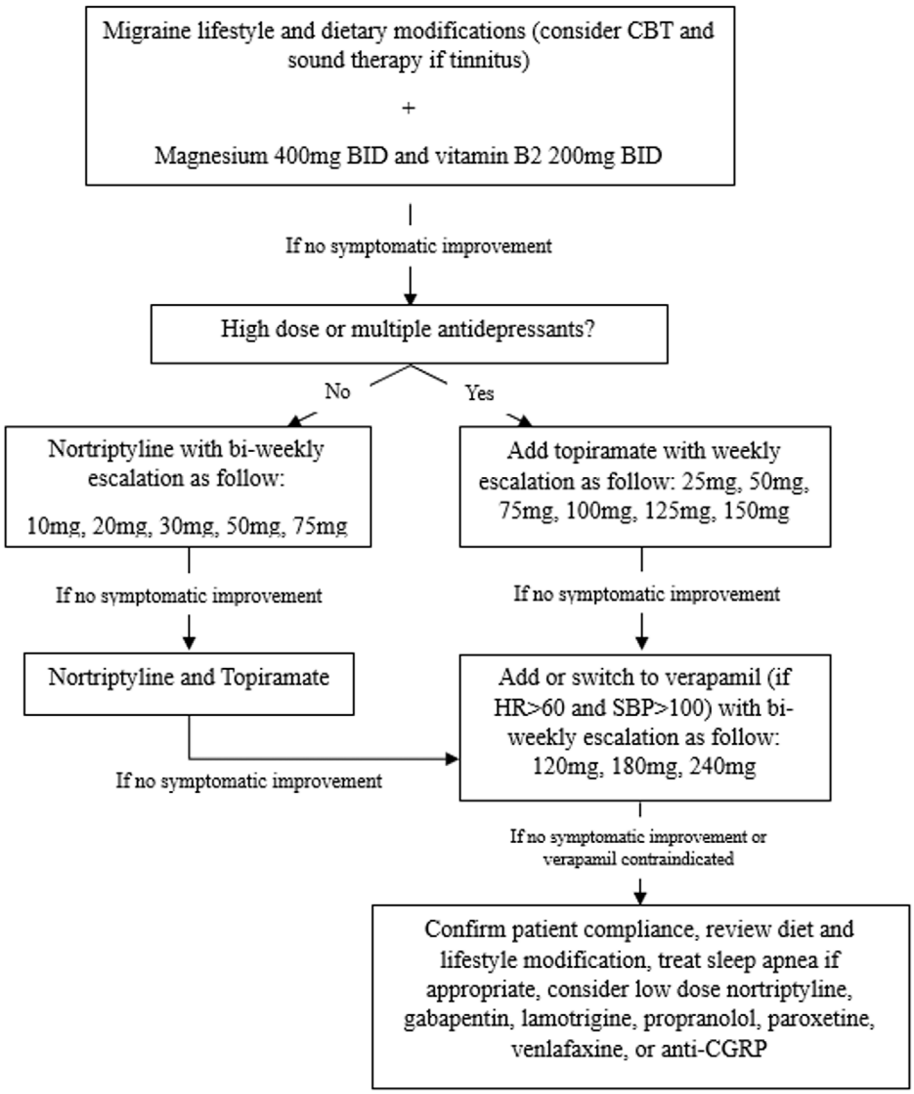

FIGURE 3 Sample algorithmic approach to otologic migraine prophylactic management. †CGRP: calcitonin-gene related peptide; HR, heart rate; SBP, systolic blood pressure.

highly selective 5-hydroxytryptamine receptor 1F agonist, was approved by the US Food and Drug Administration for the treatment of acute migraine and might be a promising therapy for vestibular migraine by blocking trigeminally-mediated responses, such as CGRP release.[104]

7.4 | Non-pharmacological prophylaxis

Many patients with vestibular migraine seek non-pharmacological treatment due to medication side effects. Non-pharmacological-approaches focus on managing migraine triggers, which can be

TABLE 3 Side effects and contraindications of migraine prophylactic medications.

Medication	Side-effects	Contraindications
Antiepileptics		
Topiramate	Cognitive slowing, weight loss, depression, paresthesia	Pregnancy
Lamotrigine	Rash, nausea, dizziness	Cardiac arrhythmias
Valproic acid	Nausea, vomiting, hair loss, easy bruising, tremor, weight gain, hepatotoxicity	Pregnancy
Gabapentin	Somnolence, cognitive slowing	Pregnancy
Calcium channel blockers		
Flunarizine, cinnarizine (not FDA approved)	Headache, lightheadedness, flushing, parkinsonism (rare)	Pregnancy
Verapamil	ECG abnormalities at doses above 480 mg/day, edema, gastrointestinal discomfort, dull headache, gingival hyperplasia	Underlying heart disease (relative)
Tricyclic anti-depressants		
Amitriptyline	Hypersomnolence (higher than nortriptyline), orthostasis, mild QTC prolongation, dry mouth, constipation, tachycardia, palpitations, weight gain, blurred vision, urinary retention, lower seizure threshold at high doses, sexual dysfunction	Underlying heart disease (relative)
Nortriptyline	Hypersomnolence (less than amitriptyline), orthostasis, dry mouth, mild QTC prolongation, constipation, tachycardia, palpitations, weight gain, blurred vision, urinary retention, lower seizure threshold at high doses	Underlying heart disease (relative)
SNRIs and SSRIs		
Venlafaxine	Nausea, hypertension, dizziness, dry mouth, sexual dysfunction	-
Paroxetine	Orthostasis, sexual dysfunction, weight gain, drowsiness	Pregnancy
Beta-blockers		
Propranolol	Fatigue, depression, sexual dysfunction, hypoglycemia, hyperkalemia	Severe bronchospastic disease
Metoprolol	Fatigue, depression, sexual dysfunction	Severe bronchospastic disease (less than propranolol)
Angiotensin II receptor antagonists		
Candesartan	Back pain, dizziness, and cold- or flu-like symptoms	None

Note: Sources include Ko et al.,[80] Beumer and Hardonk,[81] Singh et al.,[82] Skinner et al.,[83] Reid et al.,[84] Castellino et al.,[85] Deacon and Barnett,[86] Shorr et al.,[87] Capella et al.,[88] Chouza et al.,[89] Anderson et al.,[90] Stahl et al.,[91] Wenzel-Seifert et al.,[92] Hirsch et al.,[93] Arif et al.,[94] Abou-Khalil,[95] Biton et al.,[96] Nasreddine and Beydoun,[97] Cohen et al.[98] and Tomson et al.[99]

categorized into five main categories: stress (psychological or physical), hormonal changes (menstrual cycle, menopause, hormone replacement therapy, etc.), changes in sleep (too much or too little sleep, interrupted sleep, obstructive sleep apnea), diet (skipping meals; eating certain foods such as caffeine, monosodium glutamate, glutamate, tyramine, and histamine; and dehydration), and intense stimulations (bright lights, intense sound, visual motion, intense smells, weather changes, heat, and atmospheric pressure changes) (Table 4).[5,6,8,105–107] A personalized treatment plan to control or avoid patients' identified triggers may reduce the frequency and severity of their vestibular migraine symptoms. Additionally, dietary supplementation such as magnesium (400 mg PO twice daily) and riboflavin (vitamin B2) (200 mg PO once daily) has been proven helpful in preventing migraine.[108–112] Roberts et al.[113] found that lifestyle modifications in patients with definite vestibular migraine, specifically restful sleep, are associated with improvement in dizziness handicap inventory (DHI) and headache. It should be noted, however, that 32% of patients were excluded due to their request for pharmacological treatment or failure to follow up.[113]

7.5 | Vestibular rehabilitation

VR is a non-pharmacologic approach that involves gaze stability exercises, habituation exercises, gait and balance training, and walking to improve endurance, with the aim of alleviating dizziness and balance dysfunction in patients with vestibular disorders including vestibular migraine.[59,114] VR can improve functional outcomes in various vestibulopathies,[115,116] including in patients who suffer from vestibular symptoms due to migraine.[58,117] Patients with migraine undergoing

TABLE 4 Dietary and physiologic migraine triggers.

Migraine triggers	Recommendations
Stress (conflict at home/work, death of relative, physical pain, infection, other illness, etc.)	• Exercise, start with 5 minutes, gradual 1–2 minutes/week increase, goal 20–30 minutes 3–5 times a week • Practice guided meditation
Sleep (too much sleep, too little sleep, interrupted sleep, shifting sleep schedule, different sleep schedule on weekends, etc.)	• Maintain the same regular sleep schedule on weekdays and weekends • Use guided meditation on nightly basis before sleep • Avoid looking at screens and turn lights down 1 hour prior to sleep to not suppress melatonin secretion • Diagnose/treat sleep apnea
Diet (skipping meals, eating certain foods, and dehydration)	• Maintain a strict eating schedule by eating on time, even if not hungry • Drink ≥70 oz. of water per day, more with exercise or when outdoors in the heat • Follow the migraine diet by eliminating caffeine, avoiding byproducts of food aging or fermentation (red wine, aged cheeses, yeast, etc.) which contain tyramine (also in overly ripened fruit, processed protein/meat, etc.), monosodium glutamate (MSG) (soy sauce, canned soups, pickled foods, chips, canned foods, salad dressings, etc.), and histamine-containing foods (citrus fruit and nuts)

VR may report persistent or worsening symptoms, particularly after a session of therapy. Although we recommend stopping VR in patients who experience worsening symptoms with therapy, one study showed improvement in patient-reported outcome measures and objective performance measures for vestibular migraineurs who completed a 6-month program.[58] Additional investigation is crucial to determine the impact of vestibular suppressant medications and the timing of their administration on the outcomes of VR.

7.6 | Treatment of MD as a migraine variant

Current strategies for treatment of MD as shown in Figure 4 include lifestyle changes (low-sodium diet, good hydration, caffeine restriction), medication trial (betahistine and diuretics),[118,119] and intratympanic steroid injection. Surgical or ablative therapies include endolymphatic sac surgery, intratympanic gentamicin, labyrinthectomy, and vestibular neurectomy, as detailed in Figure 4.[118–121]

Migraine diet and lifestyle modifications and medications as treatment for MD patients have sparked a debate among otolaryngologists. Proponents assert that MD, also referred to as "cochleovestibular migraine," encompasses vestibular and cochlear migraine symptoms. As a result, some argue that patients with MD may benefit from migraine treatments.[53,106,122,123] Migraine treatment options include the migraine diet and lifestyle modifications that focus on avoiding migraine triggers that potentially provoke MD attacks (Table 4). Importantly, in contrast to the belief that a salt-restricted diet is beneficial, the key may lie in maintaining adequate hydration. Prophylactic medications previously discussed can be started at a low dose (10 mg of nortriptyline or 25 mg of topiramate) and increased weekly or bi-weekly, following the steps outlined in Figure 3.[122] Oral or intravenous steroids could be considered in managing acute MD attacks by preventing the spreading cortical depression secondary to migrainous central phenomena.[66] Finally, patients who have identified barometric changes as a migraine trigger may benefit from tympanostomy tubes.[124,125]

7.7 | Treatment of other migraine-related otologic symptoms

7.7.1 | Tinnitus

A significant percentage of migraine patients experience enhanced tinnitus,[5,33] and clinicians should educate patients with persistent, bothersome or fluctuating tinnitus about management strategies. Clinicians should also recommend cognitive behavioral therapy (CBT) with a potential consideration of sound therapy (ST).[36] Abouzari et al.[126] showed that an 8-week course of CBT, which included emotional awareness, stress management, and sleeping techniques among others, and personalized pitch-matched ST, led to a notable improvement in tinnitus handicap inventory scores.[126] Other studies have shown that customized ST and CBT are helpful in tinnitus management.[127,128] Migraine diet, lifestyle modifications, and medications may also have potential in treating tinnitus. Sullivan et al.[129] demonstrated the effectiveness of nortriptyline in reducing the severity of tinnitus.[129] In a recent network meta-analysis including 36 randomized controlled trials with 2761 participants, Chen et al.[130] reported that pharmacological interventions that act on the brain (amitriptyline, acamprosate, and gabapentin) and those with anti-inflammation effects (steroids and melatonin) exhibit a significant reduction in tinnitus severity and response rate when compared to the placebo or waiting-list control groups.[130] In our clinical practice, we have found improvement in the fluctuation of tinnitus and reduction of loud tinnitus in patients treated with a migraine regimen and control of migraine triggers. Other keys to tinnitus management include dietary modifications such as having regular eating habits and avoiding migraine food triggers, maintaining a regular sleep schedule, gaining control over stress, and use of supplements such as magnesium and vitamin B2 (Figure 3).[131,132]

7.7.2 | Persistent postural perceptual dizziness

Currently, there is no definitive explanation for how pharmacological and non-pharmacological approaches work in treating PPPD.[133,134]

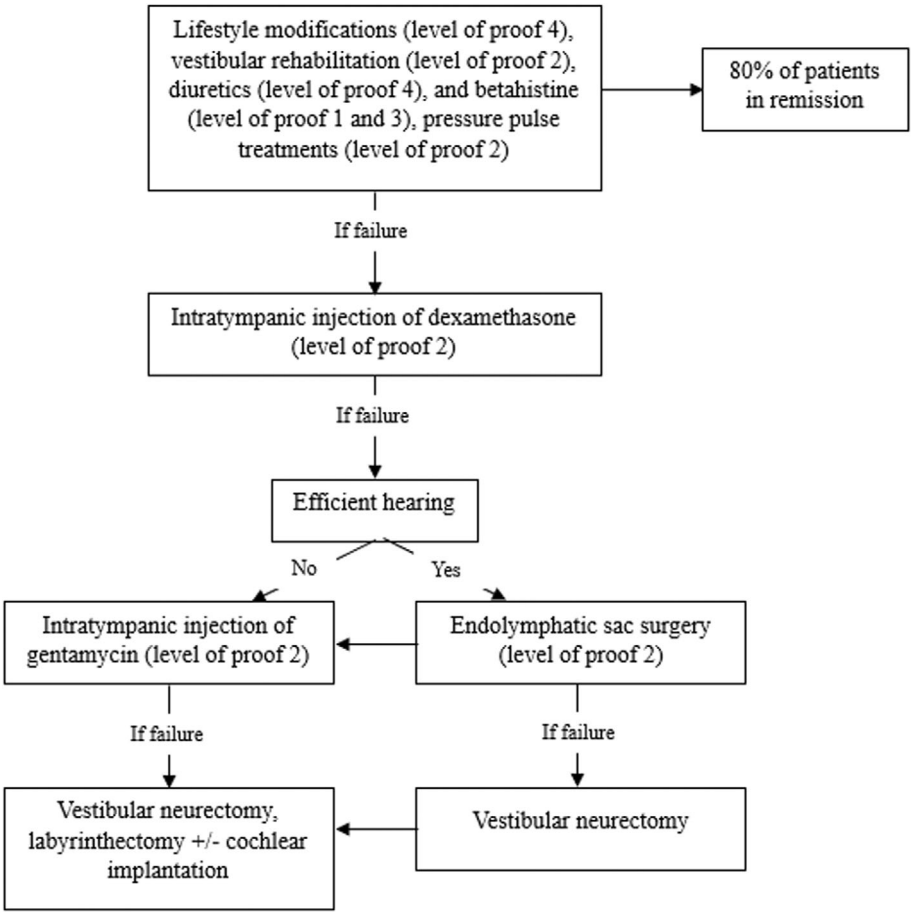

FIGURE 4 The international consensus algorithm to treat Meniere's disease.[118,119]

Main therapeutic approaches include CBT, SSRIs, SNRIs, and VR with the aim of breaking the maladaptive cycle.[135] Staab et al.[136] reported that sertraline (25 –200 mg) significantly reduced DHI scores and psychological distress in patients with PPPD. Yu et al.[137] compared the use of sertraline alone to sertraline plus CBT and found that adding CBT alongside sertraline significantly enhanced the effectiveness of sertraline in reducing scores from the DHI, Hamilton Anxiety Rating Scale, and Hamilton Depression Rating Scale. In addition, CBT reduced the dosage of sertraline used in the experiment group.[137] Finally, since several studies show an association between PPPD and migraine headaches, it may be prudent to expand PPPD treatment to include migraine treatment.[55,138,139]

7.7.3 | Treatment of sudden sensorineural hearing loss (SSNHL)

According to clinical guidelines, when patients present with SSNHL, clinicians should administer corticosteroids as soon as possible and consider intratympanic steroid injection between two to 6 weeks from onset if recovery is incomplete.[140] Concurrent oral and intratympanic steroids may have a stronger positive effect than either alone[141]; however, Plontke et al.[142] demonstrated little to no effect difference for combination oral and intratympanic steroids versus unimodal administration. An association between SSNHL and migraine has been described; Abouzari et al.[111] found that migraine prophylactic treatment may be efficacious in treating SSNHL. They found that patients with SSNHL who received oral steroids, intratympanic dexamethasone injections, migraine lifestyle modifications, as well as a combination of nortriptyline and topiramate, demonstrated greater improvements in thresholds at the low frequencies and pure tone average and required fewer intratympanic injections when compared to those who received oral steroids and intratympanic dexamethasone only.[111] Moreover, Goshtasbi et al. showed that patients with long-term SSNHL had statistically significant improvement in hearing thresholds, low-frequency pure tone averages, speech-frequency pure tone averages, word recognition scores, and speech recognition thresholds after receiving migraine prophylactic medication including nortriptyline, topiramate, and/or verapamil, lifestyle changes, and intratympanic steroid injections. These findings serve as a positive indicator for the use of migraine treatment for long-term hearing loss.[143]

8 | CONCLUSION

There is increasing evidence regarding the association between migraine and various otologic symptoms, which carries significant

implications for the development and investigation of therapies to address these challenging cochleovestibular presentations. An ideal treatment would be integrative neurosensory rehabilitation including migraine diet and lifestyle modifications, supplements (magnesium and vitamin B2), and migraine prophylactic medications. Further randomized placebo-controlled clinical trials are required to provide higher evidence regarding migraine treatment's effectiveness on various symptoms of otologic migraine.

CONFLICT OF INTEREST STATEMENT

Dr. Brooks, Dr. Tawk, and Dr. Hobson do not have any relevant financial disclosures. Dr. Djalilian holds equity in Neuromed Care LLC, MindSet Technologies, Elinava Technologies, and Cactus Medical LLC. He is a consultant to NXT Biomedical.

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