Chapter 8
Vestibular Migraine

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Introduction

While the association between migraine headaches and vestibular symptoms has long been noted in the literature [1–5], the term "vestibular migraine" was first used by Boenheim [6] in 1917 and recently reused by Dieterich and Brandt [3] in 1999. Vestibular migraine is the current nomenclature used by International Headache Society (IHS) to define vestibular symptoms associated with migraine headache. It was in 2013 that for the first time the International Headache Society [7] and the Bárány Society defined the criteria for the diagnosis of vestibular migraine as a distinct diagnostic entity. Vestibular migraine (VM) is also referred to in the literature as migrainous vertigo, migraine associated vertigo, definite migrainous vertigo, and probable migrainous vertigo.

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Diagnostic criteria for VM were initially described by Neuhauser et al. [5] in 2001. The original definitions included definite and probable migrainous vertigo and focused on the co-occurrence of International Headache Society (IHS) criteria migraine and symptoms of vertigo. In patients considered to have definite migrainous vertigo, the headaches and vertigo occurred at the same time. Patients considered to have probable migrainous vertigo had vertigo symptoms that behaved like migraine—the episodes occurred in response to environmental, physiologic or food triggers typically associated with migraine, or merely responded to treatment with medications for prophylaxis of migraine. Using these criteria, a positive predictive value of 85% was found in a follow-up study conducted over 9 years. These definitions were accepted with some variation into the International Classification of Headache Disorders (ICHD) in 2013 where the accepted terminology is vestibular migraine.

Diagnostic Criteria for Vestibular Migraine

The diagnostic criteria described by the International Headache Society in collaboration with Bárány Society in the ICHD-3 beta version [7] describe vestibular migraine as:

A. At least five episodes fulfilling criteria C and D
B. A current or past history of migraine without aura or migraine with aura
C. Vestibular symptoms of moderate or severe intensity, lasting between 5 min and 72 h.
D. At least 50% of episodes are associated with at least one of the following three migrainous features:
    1. headache with at least two of the following four characteristics:
        (a) unilateral location
        (b) pulsating quality
        (c) moderate or severe intensity
        (d) 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.

Simply stated, the above currently used criteria are vestibular symptoms that last 5 min to 72 h [7] which co-occur at least 50% of the time with classic migraine headache.

In the Bárány Society's Classification of VM, vestibular symptoms that qualify for a diagnosis of vestibular migraine include:

(a) Spontaneous vertigo:
    (i) Internal vertigo (a false sensation of self-motion);
    (ii) External vertigo (a false sensation that the visual surround is spinning or flowing);
(b) Positional vertigo, occurring after a change of head position;
(c) Visually induced vertigo, triggered by a complex or large moving visual stimulus;
(d) Head motion-induced vertigo, occurring during head motion;
(e) Head motion-induced dizziness with nausea (dizziness is characterized by sensation of disturbed spatial orientation).

Other forms of dizziness are currently not included in the classification of vestibular migraine. The criteria for VM have been intentionally created to be very restrictive to improve the quality of the epidemiologic and drug-efficacy studies in which they will be used.

Epidemiology of Vestibular Migraine

Neuhauser et al. [8] estimated that vestibular migraine has a lifetime prevalence of approximately 1% in the German population. The prevalence of HIS migraine in the US has been assessed at 13–18% and 25–33% of these individuals will experience vertigo along with their other migraine symptoms at some time [9–11]. This makes MV much more common than benign paroxysmal positional vertigo which has a lifetime prevalence [12] of 2.4%, Ménière's disease with a life time prevalence of 0.19% [13], and vestibular neuritis whose incidence is 3.5 per 100,000 [14].

Like migraine headache, vestibular migraine is 1.5–5 times more common in women than in men [1, 3, 5, 15]. While VM can occur at any age the most common patients are males and females in early mid-life for whom migraine prevalence is highest. These patients stand out in the clinic because they tend to be younger than most patients with senile BPPV, Ménière's disease, or senile multi-factorial dizziness. Epidemiological data have shown that migraine-related syndromes are the most common cause of vertigo and dizziness in children [16, 17].

Symptoms

As outlined in the criteria above, symptoms other than headache which are attributable to VM include spontaneous vertigo, positional vertigo, visually induced vertigo, head motion-induced vertigo, and head motion-induced dizziness with nausea [7]. The criteria exist as inclusion criteria to allow study of epidemiology

and drug efficacy in groups of patients whose clinical manifestations are so manifest that there can be little dispute about their inclusion. As is always the case in medicine, however, not all patients who will benefit from treatment in the trenches of clinical care will meet criteria established in the literature.

Forms of dizziness that are currently not included in the VM criteria but are experienced by patients include lightheadedness, heavy headedness, rocking, swimming sensations, rising or falling sensations, tingling sensation, distortions of spatial awareness, and excessive motion-sickness susceptibility [18]. Aside from patients with Mal de Debarquement syndrome who have a very particular history, the complaint of "rocking" can be attributed directly to central mechanisms. Whereas symptoms originating from a lesion in one labyrinth may result in a tonic deviation in one direction, the reversing rocking sensation is central and migrainous in origin in most cases. The rare exception to this rule is the rocking osscillopscia that can occur in rare patients with dura pulsing against an open labyrinth.

Patients often have extreme motion sensitivity, especially on back roads, escalators or elevators where visual-vestibular coordination is challenged. Head motion sensitivity is typical and is often severe enough to provoke a history suggestive of BPPV but that cannot be demonstrated on Dix–Hallpike testing.

In a study of a large German population, Neuhauser et al. reported that 67% of the participants with VM had spontaneous rotational vertigo, whereas 24% had positional vertigo [8]. Only 24% of the participants with vestibular migraine complained of headache concomitantly with vestibular symptoms. Head motion intolerance is a frequent complaint and has been reported by 31–77% of patients with vestibular migraine [1, 2].

The greatest numbers of VM patients who do not meet current criteria, however, are those who do not have headaches that meet criteria for migraine, migraine with aura or even VM as defined by the IHS. They typically relate a history of vestibular symptoms which started with concurrent typical or atypical headache symptoms such as generalized head or ear pressure, sinus pressure, or pressure or pain in the neck. These are all sites in the head and neck to which pain can be referred within the trigemino-cervical complex. Symptoms of headache often do not coincide at all with episodes of vertigo. Nonetheless patients with this history will respond to migraine treatment.

The majority of patients with VM do also have migraine headaches, however, these headaches may not be concurrent with the onset of their vestibular symptoms. Most patients think of their migraine headaches as an episode of occurrence within their lifetime rather than an inherited susceptibility they never lose, and which can manifest many different symptoms. Patients also do not understand that any migraine symptom may be triggered by the same triggers headaches are. A typical story, for example, is a patient with a past history of migraine who presents decades later with vestibular symptoms that are provoked by stress, weather change, and food triggers that used to provoke headache. Headaches are absent or present only as a vague head pressure.

Some patients have never had what they consider to be migraine headaches. Instead they have had sinus pressure, allergy headaches, frequent sinus infections, or head pressure.

Similar to migraineurs, patients can have a pattern of seasonal symptoms observed over years that is concurrent with allergy symptoms.

Other clues to the presence of migraine mechanisms presenting as vestibular symptoms are available to the careful historian. The ability to trigger even long-lasting symptoms of vertigo with a brief provocation is a common characteristic in VM. The duration of symptoms in VM is highly variable; symptoms can range from continuous for months at a time to momentary and occur many times per day. Overall, only 10–30% of patients with vestibular migraine fulfill VM diagnostic criteria [3, 5]. These patients respond to VM treatment.

In addition to different forms of dizziness, patients with VM may suffer from photophobia, phonophobia, osmophobia, visual, and other auras. These symptoms may support or help establish the diagnosis when formal criteria for vestibular migraine are not met [2, 18]. Auditory symptoms such as vague hearing disturbances, tinnitus, and aural pressure have been found in 38% of patients with vestibular migraine, but hearing is usually only mildly and transiently affected [19].

Diagnosis of Vestibular Migraine

The diagnosis of VM is clinical and relies heavily on a detailed history, but a careful neurotologic examination and formal vestibular evaluation can also be helpful. Vestibular testing has given insights into the ways VM may present in different individuals. Between attacks, the neurotologic exam and laboratory testing are generally normal but some abnormalities such as subtle saccadic pursuit dysfunction, persistent positional nystagmus, directional preponderance on rotational testing, and increased vestibular ocular reflex (VOR) time constant have been reported [1, 20, 21]. These findings tend to stand out because they occur in young individuals in whom central findings are not expected. Many patients cannot tolerate optokinetic stimulus or head shake even on a day they consider themselves asymptomatic, suggesting that, as in migraine headache, the brain remains sensitive to stimuli even between attacks. The most common finding seen on electronystagmography is nausea provoked by optokinetic testing and an inability to complete all 4 caloric irrigations because of excessive nausea.

During the acute vestibular migraine attacks, patients may present with spontaneous nystagmus, positional nystagmus or a combination of spontaneous and positional nystagmus [22]. Indeed, a unilateral reduction of peripheral vestibular function occurs in about 25% of patients and vestibulo-ocular asymmetry has been reported in about half of patients [23]. Inferior vestibular nerve dysfunction manifested as reduced cervical vestibular-evoked myogenic potential (cVEMP) testing has also been observed [24, 25]. These highly sensitive patients will often complain

they remained ill for many hours after the completion of testing; this triggerability of migraine mechanisms to strong stimuli is a hallmark of migraine disease.

It has been demonstrated that patients with VM have dramatically lower thresholds (greater sensitivity) to motion in certain planes than do both normal people and migraineurs without vertigo. This work may lead to the development of diagnostics specific to some mechanisms of VM [26–28].

Pathophysiology

Vestibular migraine refers to symptoms of dizziness and vertigo that can develop as a result of migraine mechanisms acting in different locations: at the cortex of the brain, in the brainstem, or in the labyrinth itself.

Cortex

In migraine, symptoms may be generated at the cortex from spreading depression over the area of the vestibular cortex [29, 30]. Vestibular symptoms generated in this way may occur in isolation or as an aura symptom of an associated headache, as much as visual scotoma may be experienced as an aura preceding a migraine headache [29, 30]. Symptoms created in this way are usually experienced as vertigo with a sense of self-motion and may be fleeting or last up to 20 min. Dizziness is considered a common aura symptom among patients experiencing migraine with aura.

Brainstem

The laterality of a migraine attack can be seen with functional imaging which will demonstrate increased metabolic activity in the trigemino-cervical complex on the side of the migraine episode. Functional imaging also demonstrates scattered areas of hyperactivity in the brainstems of migraineurs in highly variable patterns [31, 32]. Symptoms of vertigo may occur because of abnormal activity in the vestibular nuclei and vestibular pathways.

If the vestibular nuclei are affected, then symptoms of vertigo may occur because of derangements of processing of normal vestibular input. These changes may result in extreme sensitivity to head movement. Patients with symptoms generated in this way do not have an uncompensated labyrinthine lesion on clinical or laboratory examinations and generally do not respond to vestibular suppressant medications.

Inner Ear

Symptoms of vertigo may occur in patients with vestibular migraine because of direct effects on the inner ear. Direct injury to the labyrinth is possible in migraine as seen with caloric and cVEMP testing. The direct mechanism of this injury is unknown but likely relates to the innervation of the blood vessels of the inner ear by unmyelinated C fibers originating from the ophthalmic division of the trigeminal nerve (V1). These are the same C fibers, which innervate the cortical and dural blood vessels, and contain inflammatory neuropeptides which are released at the beginning of a migraine attack. The release of these neuropeptides causes inner ear plasma extravasation, blood flow changes, and abnormal firing of vestibular afferents, which are all potential mechanisms for symptom generation in VM [33–36].

Vestibular Migraine and Ménière's Disease

There is some speculation that Ménière's disease (MD) may be a complication of migraine in susceptible individuals. There is a relationship between MD, migraine, and VM. A co-occurrence of Ménière's and VM is often seen just as a co-occurrence of Ménière's disease and migraine headache is often seen. The prevalence of migraine in patients with Ménière's disease is 56%. This is much higher than the 13% prevalence of migraine in the general population.

It can be difficult to distinguish patients with endolymphatic hydrops from those with VM. Even with the presence of aural symptoms, it may be difficult since auditory symptoms like hearing disturbances, tinnitus, and aural pressure have also been found in 38% of VM patients [1, 19]. The inner ear and intracranial blood vessels share the same innervation. So, the neuropeptides known to be important in the generation of migraine symptoms may play a role in the Ménière's disease pathophysiology and it is not surprising that Ménière's disease and VM share their response to similar food triggers, stress, weather changes, and allergy. Patients with bilateral aural symptoms that fluctuate in unison are manifesting disease in the central nervous system that affects both labyrinths and will respond to migraine management.

Treatment of Vestibular Migraine [37]

The treatment of VM is the same as the treatment of migraine headache, and takes its guidance from the frequency, duration, and severity of symptoms. In migraine management a decision must be made about the strategy of treatment: will it be abortive at the time of attacks, preventive to prevent attacks, or will both strategies

be necessary? In VM abortive medications work poorly compared to agents available for treatment of head pain. In addition, vestibular suppressant medications help only 20% of VM patients. Therefore, a strategy of migraine prevention is preferred in VM.

For most patients preventive treatment involves the reduction of migraine triggers such as trigger foods through diet modification as well as the elevation of threshold for triggering of migraine with preventive migraine medication. Environmental, physiologic, and dietary triggers may add up on a particular day to push a patient over their personal migraine threshold. Migraine symptoms, whether vestibular or headache related, may occur whenever the threshold is exceeded (Fig. 8.1). Reducing trigger loads commonly results in less frequent and less severe breakthrough symptoms.

Food Triggers in Migraine

There is a common misconception that if a person is sensitive to a food item they will know it. This is not true except for the strongest triggers. Many food triggers may not be potent enough to cause migraine alone, but may increase an individual's migraine threshold modestly for days. In combination with other partial triggers, a patient may be pushed over their personal migraine threshold and experience episodic or continuous symptoms. Patients with VM should therefore be encouraged to

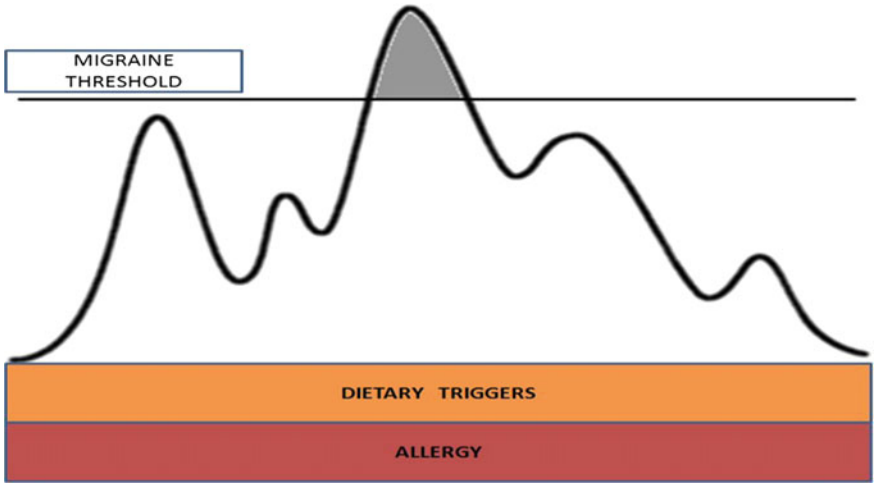

Fig. 8.1 Everybody is born with a threshold for migraine headache. Some have lower threshold due to inherited ion channelopathies. Crossing the threshold results in headache, dizziness, or other migraine symptoms. One way to treat these patients is to elevate the threshold with preventive medications and to decrease known migraine triggers. In this way the threshold is not exceeded and symptoms will not occur

reduce known migraine food triggers in their diet. Many patients can control symptoms in this way and avoid prophylaxis with anti-migraine medications. There is a **14%** treatment success in VM with caffeine cessation alone [38].

Food triggers of migraine fall into two general groups (Table 8.1) [39]:

- Foods that contain complex products of food aging and fermentation: wines, aged cheeses, fresh baked bread, yogurt, etc.
- Foods that contain chemicals that are potent CNS neurostimulants: caffeine, chocolate, tyramine in aged cheese, MSG, etc.

Table 8.1 Dietary migraine triggers [39]: patients should eliminate foods on this list or reduce their consumption to once weekly

Accent seasoning	Fresh bread	Pizza dough
Aged meats	Frozen yogurt	Plant protein
Anchovies		Processed meats
Autolyzed yeast	Garbanzo beans	Protein concentrates
Avocados	Gelatin	Protein fortified items
	Glutamic acid	Provolone
Bacon	Grapefruits and juice	
Bagels	Gravy	Raisins
Bananas	Gruyere cheese	Raspberries
Beef jerky		Ready-to-eat meals
Blue cheese	Hams	Red plums
Bouillons	Heavy alcohol drinks	Red vinegar
Breadcrumbs	Hot dogs	Red wine
Brewers yeast	Hydrolyzed protein	Restaurant food
Brick cheese		Rice protein
Brie cheese	Iced tea	Romano cheese
Broad Italian beans		Roquefort cheese
Broth	Kombu (seaweed extract)	
Buttermilk		Saccharin
	Lemons and juice	Salami
Calcium caseinate	Lima beans	Salty snacks
Camembert cheese	Limes and juice	Sauerkraut
Canned meats	Lentils	Sausage
Carrageenan	Liverwurst	Seasoned salt
Caviar	Low calorie foods	Smoked fish
Champagne	Low fat foods	Smoked meats
Cheap buffets	Lunchmeats	Snow peas
Cheddar cheese		Sodium caseinate
Cheese spread	Malt extract	Soft pretzels
Chicken livers	Malted barley	Soups

(continued)

Table 8.1 (continued)

Chinese food	Maltodextrin	Sour cream
Chocolate	Marinated meats	Soy products
Clementines	Mozzarella cheese	Soy protein
Coffee	MSG	Soy protein concentrate
Coffee cake	Muenster cheese	Soy protein isolate
Coffee substitutes		Soy sauce
Cola	Natural flavors	Stilton cheese
Croutons	Navy beans	Sulfites
Cultured items	Nitrates	Sweet n' Low
Cured meats	Nitrites	
	Nut butters	Tea
Dark alcohol drinks	Nutrisweet	Tenderized meats
Dates	Nuts	Textured protein
Decaf coffee		Tyramine
Decaf tea	Olives	
Doughnuts	Onions	Ultra-pasteurized items
Dried fruits with sulfites	Oranges and juice	
	Papayas	Vegetable protein
Enzyme modified items	Parmesan cheese	Veggie burgers
	Passion fruit	
Fava beans	Pate	Whey protein
Fermented items	Pea pods	Wild game
Fermented meats	Pepperoni	
Feta cheese	Pickled fish	Yeast
Figs	Pickles	Yeast extract
Flavored snacks	Pineapples and juice	Yogurt
Flavorings	Pinto beans	
Fresh beef liver	Pizza	

Some common foods such as peanuts and banana have unknown causes of chemical provocation of migraine. The use of a patient handout with dietary recommendations is recommended. Patients with frequent symptoms are encouraged to reduce food triggers on the list to only once per week. This allows them the freedom to function socially while reducing their dietary trigger load substantially.

The first goal of dietary trigger reduction is to achieve intermittent rather than daily symptoms. When a week passes and a MV episode occurs, the patient can look back over the previous 36 h and consider what may have triggered their attack; was it environmental, physiologic or dietary? Typically, a common pattern will emerge for each patient that points out their strongest triggers, those they should continue to avoid, and they can add other foods back with careful observation.

Sometimes the patient's strongest triggers are not dietary and lifestyle management to avoid stress, fatigue, dehydration, or hunger are employed to avoid symptoms.

Medication Treatment of Vestibular Migraine

Abortive treatment is rarely helpful in the treatment of VM because symptoms do not respond to treatment or are too brief or frequent for abortive medications to be useful.

Some patients with VM respond to centrally acting promethazine during acute episodes better than meclizine or diazepam. This suggests a central mechanism of action and origin of symptoms. Overall only 20% of patients with VM respond to peripheral vestibular suppressants during vertigo episodes. Medication treatment, therefore, must necessarily focus on elevation of the migraine threshold to prevent recurrent attacks.

Preventive Medications for Vestibular Migraine

Preventive medications are highly effective and useful in individuals whose symptoms are frequent or severe enough to warrant daily preventive medication. Medications useful for preventive therapy include tricyclic antidepressants, β-blockers, sodium channel blockers, calcium channel blockers, and long acting benzodiazepines.

Migraine is thought to be an inherited disorder of defective ion channels in the brain; therefore, many preventive drug therapies are ion channel antagonists. The best choice of an initial medication is best determined by the patient's current medications for other medical problems, their general health, and their willingness to accept side effects.

Patients are encouraged to start on medications early, and to tolerate side effects if they are mild, and to continue diet modification so that their symptoms can be interrupted, preventing chronification that may lead to resistance to treatment. After symptoms have been controlled for several months a weaning of medication to determine the smallest effective dose is reasonable.

The drugs below are presented by class in the order of their preference.

Tricyclic Antidepressants

Nortriptyline and amitriptyline have the highest response rates in patients with VM. Despite a high side-effect profile, these drugs are typically very well tolerated since

they are effective at very low doses in VM. Nortriptyline is better tolerated than amitriptyline with similar efficacy especially in elderly patients. Nortriptyline is a medium potency sodium and calcium channel blocker, in addition to being a serotonin and norepinephrine reuptake inhibitor (SNRI).

An initial nightly dose of 20 mg is often helpful. Thirty percent of patients at this dose will have some symptoms of slow waking in the mornings. These patients are instructed to take their medications earlier in the evening. If the patient has a definite but incomplete clinical response to the medication, the dose may be escalated in 10 mg increments to 30 then 40 or 50 mg. A few patients respond to higher doses of Nortriptyline. As expected, some patients have an excellent clinical response but have intolerable side effects. These patients should discontinue the medication and a trial of a sodium or calcium channel blocker can be started. Nortriptyline should be avoided in patients with bipolar disorder as it may exacerbate bipolar swings.

Patients can generally take low dose Nortriptyline successfully alongside other antidepressant medications but there is a small risk of serotonin syndrome. This should be discussed with the patient. At higher doses dry mouth and sedation can limit tolerance of therapy. Nortriptyline has a high response rate and good tolerance in older patients. Selective serotonin reuptake inhibitors (SSRIs) have less proven benefit in control of vestibular migraine and migraine headache.

Anticonvulsants

As a class, anticonvulsants are sodium channel blockers and have demonstrated a 25% response rate in VM patients. The most commonly used anticonvulsant is **topiramate**. It is a combination carbonic anhydrase inhibitor and sodium channel blocker. Treatment is started with 25 mg tablets and escalates by 25 mg weekly to an initial dose of 50 mg BID. Patients may require 100 mg BID. The most common side effects are associated with the carbonic anhydrase inhibitor properties and include taste disturbance (especially prominent with carbonated beverages), decreased appetite with moderate weight loss, and numbness and tingling of the extremities. Cognitive side effects of Topiramate can be limiting in up to 24% of patients but can be avoided in many patients with slow escalation as described. A 24-h release formulation of Topiramate has recently become available and has only a 4% incidence of cognitive side effects. Topiramate may be the best starting point for therapy in young female patients.

While its exact mechanism of action is unknown, gabapentin can be effective if the side effects of Topiramate are limiting. Patients are started at a low dose (300 mg a day) with weekly escalation to a first target dose of 300 mg TID. This can be increased as tolerated to 1200 mg TID or until side effects (usually sedation) appear. This agent has frequent dosing but a low side-effect profile.

Calcium Channel Blockers

Calcium channel blockers are the best-tolerated regimen for many patients. Verapamil 80 mg three times daily is often effective, and has the highest response rate but has a short half life requiring frequent dosing. Patients are instructed to taper their medication upward by taking 80 mg daily the first week, twice daily the second week then three times daily. Diltiazem CD 120 mg a day increasing as tolerated to as high as 240 mg twice daily is also effective. Constipation and hypotension are some times limiting side effects of calcium channel blockers. Avoid giving calcium channel blockers to patients already on beta-blocker therapy for hypertension. Patients should be cautioned about orthostasis which may limit utility.

Beta Blockers

Propranolol has long been used for migraine prophylaxis. Propranolol LA 60 mg per day may be increased as needed up to 160 mg per day. Propranolol should be avoided in patients with reactive airway disease, diabetes, and depression. Young men seem to have the highest response rate to beta blockers but limitations of exercise performance can be seen in athletes.

Benzodiazepines

Clonazepam at low doses is unusually effective in some patients who have symptoms of rocking at their presentation. One quarter to 1 mg twice daily can significantly reduce symptoms in these patients. This effect is seen even in patients who do not exhibit anxiety. Clonazepam can also be used in patients where stress is a major contributing factor to their illness. It should only be used as a bridge until life stressors are addressed with mental health professionals.

Allergy Treatment

Some patients with VM present a long history of symptoms with a distinctly seasonal pattern that suggests allergy as a triggering factor. It has been established there is a direct correlation between the degree of atopy and the intensity and frequency of migraine headache, as well as a headache response to treatment with allergy immunotherapy [40]. A treatment response among patients with VM has also been seen but has not been carefully studied.

Allergy testing for inhalant allergens and foods may be fruitful in establishing a connection of specific sensitivities to the patient's seasonal pattern. Testing may also detect unknown sensitivities to foods, which should be added to the list of

foods to be avoided. Routine treatment of allergy with antihistamines may be helpful.

Patient Follow-up

Effective treatment of VM requires significant time and counseling. Patients need to understand the migraine origins of their problem, and if they do not, to accept the rigors of a clinical trial in search of a solution to their problem. It is helpful to have materials prepared and easily accessible to them to completely enroll them into the work that is necessary to be successful. Many patients are understandably hesitant to take medications or modify their already healthy diets, or insist on natural treatments. It is important for patients to view their problem from the migraine paradigm to see the real health of their diet. Chronification of symptoms is seen in many patients who have experienced symptoms for years and may lead to resistance to treatment. For this reason, patients should be seen every 6–8 weeks to review changes in the frequency, intensity, and duration of symptoms of dizziness that have occurred in response to prescribed medications and adherence to the migraine diet. Reporting on headache symptoms in the same way is important because headache resolution is a marker of treatment response and may precede vertigo resolution. A partial response to treatment should lead to a recommendation of dose escalation if there are no limiting side effects.

Patients with concurrent Ménière's disease should have treatment directed at control of their hydrops. Surgical or chemical labyrinthectomy is avoided if headache or central symptoms of vertigo have not been controlled to avoid problems with post-labyrinthectomy compensation.

Many practitioners refer VM patients to their neurology colleagues. Most neurologists do not have a strong interest in seeing or managing these patients as they may not meet IHS criteria for the headache component of their symptoms, or even meet the ICHD vestibular migraine criteria. Neurologists typically see patients at 3–6 month follow-up intervals, which can lead to chronification of symptoms.

Summary

Migrainous vertigo is the most common disorder causing dizziness and vertigo among patients who seek care from otolaryngologists. Its mechanisms of actions have yet to be clearly understood but it responds to treatment using therapeutic strategies developed for the care of migraine headache sufferers. Symptoms of vertigo are varied and may originate in widely varying patterns among patients.

Migraine mechanisms can cause dizziness in patients because of dysfunction in the labyrinth, in the brainstem and at the cortex of the brain. A clear classification of VM has been adopted by the International Headache Society to allow high quality studies to improve patient care.

Recommendations

A care strategy for patients with vestibular migraine is now necessary in otolaryngologic practice. Because this plan necessarily involves migraine management, and because migraine is not currently a part of the otolaryngology curriculum, this chapter may serve as the starting point for creation of such a plan.

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