Introduction

Vestibular migraine is a condition marked usually by the presence of vertigo in association with migraine headache. This topic is quite relevant to the practicing otolaryngologist. This is because an all too common patient chief complaint at an otolaryngology office visit is “dizziness”, and it is important to always have a high suspicion for migraine as part of the differential when delineating the cause of this ambiguous symptom. Given that this is a neurological disease in origin, the proper diagnosis of vestibular migraine will help ensure the proper treatment for the patient as well as to avoid any unnecessary testing and, most importantly, treatment.

Terminology

This entity referred to here as “vestibular migraine” can be found under many names in the literature. Some of the most common terms used for this condition include the above as well as “migraine-associated vertigo” and “migraine vestibulopathy”. Other less common terms include “migraine-related vertigo”, “migrainous vertigo”, and “migraine dizziness”. Familiarity with these terms will help decrease confusion of subject matter when one comes across the condition in the literature and text.

Distinct Entities From Vestibular Migraine

There exists several disease processes with similar symptomatology and presentations that could easily be confused with vestibular migraine. Some of the main ones include Meniere’s disease and benign paroxysmal positional vertigo (BBPV). Historically in textbooks and primary literature, authors considered basilar migraine to be the same disease as vestibular migraine. It has only been in the past 10 to 15 years that a more definitive distinction between the two conditions has been accepted by authority figures on the subject.
Benign recurrent vertigo of childhood is a distinct disease characterized by recurrent brief attacks of vertigo with complete recovery in between episodes in an otherwise healthy child. With this condition, there is often a strong family history of migraine, making the distinction between it and vestibular migraine more difficult. In addition to intermittent balance disturbances, episodic ataxia (particularly the subtype episodic ataxia 2) can often present with or without both migraines and vertigo.

**Epidemiology**

Many would consider vestibular migraine not to be a distinct disease by itself but rather a manifestation of migraine, perhaps even the most predominant symptom for some who are afflicted by it. Simply speaking, it occurs when there is a disturbance in the central or peripheral branch of the vestibular system with timing before, during, or after a migraine occurs. Vestibular migraine is actually estimated to be the second most common cause of true “vertigo”, falling second only to BPPV.

Migraines themselves have an overall prevalence of about 10% in the general population, with 11-17% of women affected and 4-6% of men. Older patients tend to be less affected by migraines, and along those lines 20% of women ages 30-49 years old have migraines. In regards to “dizziness”, it is estimated that between 23-29% of the population experience significant morbidity from either vertiginous or non-vertiginous causes of this symptom.

Based on the prevalence of dizziness and migraine separately, by pure chance approximately 3-4% of the population would experience both of these symptoms. Using stricter criteria on what is vertigo-associated migraine, it is still thought to be prevalent in about 1% of the population. For comparison, only 1 in 500 people are considered to have Meniere’s disease, meaning vestibular migraine is 5 times more common.

**Pathophysiology**

One cannot discuss the mechanism behind vestibular migraine without first talking about that of migraine headache. Though there is still ample debate amongst experts about the exact mechanism of migraines, several credible theories have been proposed and will be discussed below. The pathophysiologic processes specifically for vestibular migraine are simply extensions of these theories.

The first mechanism of migraine to discuss is the most historically accepted of the theories known as cerebral vasospasm. The vasospasm theory asserts an explanation for both why people experience the pain associated with migraines as well as the aura symptoms. The theory is that a migraine trigger event such as stress or certain food consumption causes neurons to fire which control the vascular tone around cerebral blood vessels, decreasing blood flow to the cortex. This hypoxic event is thought to be the cause for the aura event occurring prior to the headache. Furthermore, once the cerebral vasculature relaxes after this constrictive event, the distension of these blood vessels from reperfusion is believed to stretch the neuron fibers innervating the meningeal vasculature which are primarily in the trigeminal nerve distribution, thus producing the headache that the person experiences.

The vasospasm theory has been explored through measurements of blood flow in the occipital cortex during migraines (i.e. decreased flow was seen). Another example for this is the decreased retinal artery blood flow experienced in patients with retinal migraines during their attacks with the associated
symptom mainly being visual disturbance. Support for the vasospasm theory partially lies in the fact that beta-blockers and calcium channel blockers that are used commonly to prevent migraines also happen to decrease the cerebral vascular tone.

In regards to vestibular migraine, one of the current theories is that migraine-induced vasospasm and subsequent decrease in regional blood flow to the inner ear via the internal auditory artery from the anterior inferior cerebellar artery (AICA) could cause transient ischemia, and thus transient or permanent peripheral vestibular dysfunction. Similarly, it may be that the individual small branches of the vestibular system vasculature (e.g. internal auditory artery) spasm during this phenomenon.

Another major theory behind migraine genesis and probably the most recently popular is that of cortical spreading depression. Also a proposed cause for both the aura and headache pain of migraines, cortical spreading depression is thought to occur when a trigger event depolarizes hyperexcitable neurons in a wave-like fashion across the brain. This “spreading depression” may be what illicits the sensation of the aura. Furthermore, these hyperexcitable neurons may also irritate the meningeal blood vessels, thus causing the pain of the migraine. One can extrapolate that should this process concentrate within the vestibular processing centers of the cortex, the resulting symptom could be vertigo.

A third and final theory for this discussion revolves around the disruption of ion channels in the brain. Beta blockers and calcium channel blockers effect their action in the cells primarily upon calcium channels within the cell membrane. Several inherited disorders with calcium channel mutations result in migraine headaches and related symptoms. For example, episodic ataxia type 2, a disease marred by ataxia lasting for hours along with migraine and a high propensity for vertigo, is associated with a mutation in the voltage-gated calcium channel gene CACNA1A. Another related disorder with the same mutation is familial hemiplegic migraine type 1 which includes hemiparesis as one of its auras in addition to vertigo and ataxia.

**Symptomatology**

Some of the most common symptoms found in vestibular migraine patients include what one would expect with classic migraine such headache pain, photophobia, phonophobia, and gastrointestinal upset (nausea, vomiting, motion sickness). Additionally, visual and other auras like auditory hallucinations can be found. Auditory symptoms such as tinnitus and hearing loss have been inconsistently reported throughout the literature with some authors finding them common and others finding them scarce.

In order to establish a diagnosis of vestibular migraine, the otolaryngologist must decipher whether or not the patient has experienced true vertigo. Often the patients present with the chief complaint of “dizziness” which is a non-specific term which could manifest itself many ways. True vertigo is defined as a sensation of the patient or the room spinning or circular movement of either. If the room or objects appear to undulate up and down, this may be termed as oscillopsia (a subtype of vertigo) which often presents as the primary symptom of superior semicircular canal dehiscence syndrome.

Other symptoms that a patient may generically term as “dizziness” include a light-headed sensation, a lack of mental clarity, or loss of balance. The origins of these symptoms may be vestibular,
yet they may also be psychogenic, cardiac (e.g. orthostatic hypotension), CNS-related (transient ischemic attack), or drug-related by nature to only name a few. The inability for the patient to accurately tell the physician exactly the type or cause of their dizziness lies in the fact that patients are not routinely trained in learning to distinguish between them like the physician is.

For patients with vestibular migraines, most of them experience similar frequencies and presentations of their symptoms such as vertigo and photophobia. The vertigo typically lasts minutes to hours (but can be seconds to weeks). It most commonly occurs spontaneously. In other words, it is not commonly triggered by positional changes, though more rarely can be positional. This fact can make it difficult to distinguish between vestibular migraine and BPPV. Additionally, strong visual stimuli such as car chase scenes on television or repeating patterns on rugs may trigger the vertigo.

In a related study conducted by Neuhauser et al. (2001) in dizziness and migraine clinics, the authors found the most common duration of the vertigo in vestibular migraine patients to be between 5-60 minutes (33%). The second most common associated symptom experienced by these patients was photophobia (70%) followed by phonophobia (64%). One noteworthy fact to mention is that only 94% of these patients diagnosed with vestibular migraine actually had a migraine headache associated with their episodes of vertigo. Yet, they all experienced at least a remote history of migraines.

**Testing**

Whenever a “dizzy” patient comes into the office, most otolaryngologists are lucky to have at their disposal several relatively accessible and highly useful resources for testing the patient. It is prudent to first remember to always do a thorough ear, nose, and throat exam before proceeding with advanced testing and imaging. There is no specific test to absolutely confirm vestibular migraine as the diagnosis, but one must utilize available testing to ensure that other causes of dizziness and vertigo are ruled out before settling on vestibular migraine as the diagnosis.

Initially, the examiner should start with in-office neurologic tests requiring no additional sophisticated equipment. In addition to the standard neurologic exam, the vestibulo-ocular reflex test assesses the visual compensations the brain makes for head rotation, and the Romberg test evaluates the body’s ability to sense positioning which may give insight into a balance disorder causing dizziness. Body swaying after eye closure within one minute of testing indicates a positive Romberg test. Probably the most important exam test for the otolaryngologist to perform on the “dizzy” patient is the Dix-Hallpike test. The Dix-Hallpike is a positioning maneuver which will possibly illicit vertigo as well as nystagmus and is usually diagnostic for benign paroxysmal positional vertigo if positive.

Beyond the capabilities of the standard patient exam room, there are several more sophisticated tests that one should consider for a patient experiencing vertigo. Audiogram represents one of the first-line tests in the otolaryngologist’s repertoire and can help to distinguish whether hearing loss is associated with the vertigo, a finding which could point the diagnosis towards Meniere’s disease. Other less commonly used but helpful tests include auditory brainstem response, videonystagmography (VNG), water calorics, and vestibular-evoked myogenic potentials (VEMPs). VEMPs measure myogenic potentials from the sternocleidomastoid muscle via the vestibulocolic reflex and the inferior vestibular nerve after a “clicking” sound is presented to the ipsilateral ear. If the above tests do not lead
to any diagnosis, and the physician is concerned that there may be a central process causing the patient’s symptoms, he can obtain an MRI of the brain to rule out a lesion such as acoustic neuroma that is potentially contributing to the presentation.

In the past 10 years, evaluation of nystagmus has become an important consideration when diagnosing a patient with vestibular migraine. Traditional testing for nystagmus involved using electronystagmography (ENG) where electrodes were placed around the eyes and recorded eye movements. Videonystagmography (a.k.a. video-oculography or VOG)) has more or less replaced ENG due to the fact that with VNG one wears more comfortable, infrared goggles instead of the electrodes on the face and that VNG can detect torsional nystagmus which ENG cannot.

One study by von Brevern et al. (2005) sought assess the type of nystagmus experienced by patients with vestibular migraine. Among the 20 patients that they evaluated, they found pathological spontaneous or positional nystagmus in 70% of the patients during an acute vestibular migraine attack. From this, they inferred that spontaneous nystagmus indicates a central (i.e. cortical) origin of the nystagmus whereas positional would indicate a peripheral (i.e. vestibular system) origin. Of note, 70% of these people in the study also had a positive Romberg sign, and none of the patients experienced hearing loss.

**Diagnosis**

Establishing a diagnosis of vestibular migraine for a patient presents a formidable challenge to the physician for many reasons. First, there exists no pathognomonic exam signs, biomarkers, blood, or lab tests to confirm it, and there is significant symptom overlap between this disorder and the other causes of vertigo. Furthermore, there is no definitive agreed upon criteria by authorities in the field for how to diagnose it. In fact, the diagnostic criteria for basilar migraine is the only criteria put forth by the International Headache Society that includes “vertigo” as a symptom. Ultimately then, to diagnose vestibular migraine, one must first rule out other causes of vertigo.

In 2005, Neuhauser and Lempert published a set of guidelines (seen above) to assist physicians in making this diagnosis. These guidelines are sub-divided into a “definite” vestibular migraine section and a “probable” section. A person must experience at least “moderate severity” vestibular symptoms (which they define as interfering with daily life) in order to fit into either of these categories. In the “definite” category, one must have a history of current or previous migraine headaches whereas this is not required to be considered “probable vestibular migraine”. Important to note is that both “definite” and “probable” categories indicate that other causes of the vertigo should be ruled out, thus indicating that vestibular migraine is mostly a diagnosis of exclusion.

**Differential Diagnosis**

Although one could include virtually any cause of vertigo in the differential diagnosis, there are three main conditions discussed in the literature that must be ruled out before concluding someone has vestibular migraine. Basilar migraine, a subtype of migraine headache where the pain is primarily experienced in the occipital area of the head, used to be considered apart of the same process as vestibular migraine, and historically the literature and textbooks lumped them as the same disease. In
addition to the occipital migraines, symptoms include vertigo in 60% of the patients as well as ataxia, paresthesias, and dysarthria.

These “basilar” symptoms typically arise from impairment related to the cerebellum and brainstem area. For basilar migraine, the International Headache Society has published a set of diagnostic criteria. In it they specify a requirement of at least two “posterior circulation symptoms” (such as listed above) lasting between 5 to 60 minutes which are immediately followed by a migraine. This requirement helps the physician to distinguish between basilar and vestibular migraines, given that this specific temporal and symptomatic relationship is not typically seen in vestibular migraine patients.

The next condition to strongly consider in the differential for vertigo is Meniere’s disease. Meniere’s presentation is typically comprised of vertigo lasting between 20 minutes and 24 hours, often with associated tinnitus and unilateral, low frequency, fluctuating hearing loss. Though vestibular migraine patients may present with hearing loss too, the loss does not typically progress to a profound level and is not usually permanent as it often is in Meniere’s. Despite these differences, the reality of the situation is that the distinction between Meniere’s and vestibular migraine can be quite difficult to make due to the significant symptom overlap between the two. A substantial portion of Meniere’s patients also experience migraine. Furthermore, Radke et al. (2002) found that up to 45% of Meniere’s patient experience migraine-related symptoms such as visual aura during their vertigo attacks. In consideration of these similarities, in 2013 even Lempert (one of the biggest proponents of defining vestibular migraine) and his colleagues suggested that if a patient meets criteria for Meniere’s disease, specifically based on audiogram findings, then the diagnosis should be Meniere’s and not vestibular migraine.

Benign paroxysmal positional vertigo is a vertiginous disorder caused by misplaced canaliths within the semicircular canal and is considered to be the most common cause of vertigo by far. Compared to BPPV, in vestibular migraine the Dix-Hallpike does not usually produce nystagmus or vertigo. Also with vestibular migraine, the vertigo typically occurs earlier in life with shorter duration and more frequent episodes. Although the vertigo in vestibular migraine can be positional causing the distinction between the two to be difficult, unlike in BPPV, the positional nystagmus of vestibular migraine patients does not fatigue and cannot be aligned with a specific semicircular canal.

Treatment

Though a few specific therapies for vestibular migraine patients will be discussed, the majority of the treatment lies with the same regimens used for migraine headaches. Abortive therapy is aimed at treating an acute attack of headache or vertigo. Some of the drugs used include Fioricet and Fiorinal which are combination pills that have butalbital as well caffeine and either acetaminophen or aspirin in them. The triptans like sumatriptan can be tried but there has been no evidence to suggest that they lessen symptoms. A placebo trial by Neuhaußer et al. (2003) tested the drug zolmitriptan for vestibular migraine attacks and results were inconclusive at best, partially owing to the small sample size of 10 patients they studied. Overall, there are simply few options available for abortive treatment of vestibular migraine attacks.

The next arm of treatment consists of prophylactic medications. Various categories of drugs are used including antihypertensives like beta blockers (e.g. metoprolol) and calcium channel blockers
(verapamil) as mentioned earlier. Tricyclic antidepressants like nortriptyline are typically used to treat depression but are also a viable option for migraine prophylaxis. Lastly, commonly used anticonvulsants such as topiramate and lamotrigine are often used with varying success.

Whether or not preventive or abortive therapy is successful, it is important for the physician to know how to treat the associated symptoms of a vestibular migraine attack as these can be equally debilitating. The primary treatment of the phonophobia and photophobia a patient experiences is to remove the offending stimuli. This is why migraine patients often prefer to lay down in a dark, quiet room during an attack. As for the treatment of severe nausea and vomiting, the ultimate goal is to suppress the vestibular system. This is accomplished by a variety of medications, such as the antiemetics ondansetron and promethazine. Promethazine is especially an effective option given that it can be given as a suppository if needed, and its sedating properties may be desired for patient to get rest. One can also trial antihistamines like dimenhydrinate and meclizine or a short course of benzodiazepines. Given that gastric motility is typically slowed during migraine attack, metoclopramide (a dopamine antagonist) can help to speed up motility to promote absorption of abortive and other drugs.

Probably the most important aspect of a patient’s treatment regimen is proper counseling and follow up for trigger avoidance. The patient should keep with them a “symptom diary” where they record the onset of their symptoms and what they were doing and consuming before these occurred. Just like with migraine headaches, there are several dietary and environmental triggers the patient try to avoid if possible. Some environmental triggers like a woman’s hormone fluctuation or weather changes can obviously not be avoided. Yet others like stress, dehydration, and sleep deprivation can with proper lifestyle changes.

Though often quite difficult to enact and harder to sustain, dietary modifications can make a significant difference in a vestibular migraine patient’s quality of life. Caffeine and alcohol consumption are some of the most common triggers. Other common food triggers include aged or ripened cheeses and foods high in monosodium glutamate (i.e. MSG). It is important to note that not all of these triggers effect every person but rather different individuals have their own variety of triggers, and a symptom diary can help keep track of these so they can be better avoided.

In addition to trigger avoidance and medications, both of which can be used for vestibular migraine and migraine headaches, physical therapy has been shown by Whitney et al. (2000) to be particularly effective in treating patients specifically with vestibular migraine. In this retrospective case series of 39 patients, the participants were asked to fill out several questionnaires before undergoing therapy. Some of the questionnaires included a 0-100 symptom severity index, dizziness handicap inventory, dynamic gait index, and recorded number of falls.

Next, they subjected the patients to several sessions of physical therapy which included balance and gait training, strength and stretching, and habituation exercises. After this was completed, they had the patients fill out the questionnaire’s again and compared the difference in the scores. The results of this study demonstrated that the patient had statistically significant improvement in almost all category scores. For example, the number of patients reporting more than one fall in the past 4 weeks declined by
Overall, they found that 35 of the 39 patients experienced improved scores and concluded that vestibular rehab should be offered to all patients with vestibular migraine.

**Conclusion**

In the end, one can think of vestibular migraine as a common manifestation of migraines as well as a deceivingly common cause of vertigo. It is important to recognize that there is still significant work to be done to better define this entity and further ways to diagnose it. If a physician suspects a patient to have the disorder, then he should consider initiating migraine therapy including trigger avoidance, and a positive response will help confirm the diagnosis. To help the physician with this process, the International Headache Society reportedly will be publishing more definitive criteria for diagnosis of vestibular migraine in 2014. Until further diagnostic tools come to light, the physician must rely on keen history taking skills and a trial of migraine therapy to confirm the diagnosis of vestibular migraine.

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