Chapter 22
Complex Dizziness

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Introduction

The first step in evaluating dizziness is to obtain a clear history and classify a patient's complaint into a single subjective perception (e.g., vertigo, presyncope, disequilibrium, etc.) [1, 2]. This will usually lead to a clear diagnosis and path for further evaluation and management. Presyncope and syncope, for example, are largely cardiogenic and, less often, neurogenic [3]. Though patients with complaints of presyncope due to orthostatic hypotension are commonly referred for otologic evaluation, there is seldom a significant otologic component. In contrast, the illusion of motion, vertigo, has been heavily attributed to vestibular disorders. However, non-otologic disorders may also present with vertigo, and more mild or chronic vestibular dysfunction may present with dizziness without vertigo. Furthermore, careful inquiry into patient symptoms often reveals multiple types of dizziness [2, 4]. In a national survey, the presence of multiple descriptions of dizziness has been found to be the norm in adults in the United States, even in patients with otherwise typical presentations of a vestibular disorder [1].

The evaluation and management of patients with complex presentations of dizziness require a solid understanding of the numerous systems that contribute to one's sense of balance. Maintaining balance requires adequate detection of environmental stimuli, integration of multisensory neural input, and execution of ocular, musculoskeletal response and autonomic responses [5, 6]. Central vestibular pathways are integrated with pathways responsible for autonomic control, as well as for the generation, perception, and regulation of emotional and affective states [7]. Thus, any pathology arising within the peripheral vestibular, ophthalmologic, cardiovascular, musculoskeletal, or central nervous systems may contribute to a complex presentation

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of dizziness. The psychologic consequences of experiencing alarming symptoms, such as vertigo and imbalance, may exaggerate or color the reported complaints [8].

This chapter will discuss vestibular disorders with overlapping symptoms, comorbid conditions, and clinical features that may help establish the appropriate diagnoses in patients presenting with complex dizziness.

Commonly Recognized Balance Disorders with Overlapping Symptoms

While most balance disorders have classic presentations, there can be significant overlap among many of these. Recurrent, spontaneous vertigo carries a large differential diagnosis, and many conditions may present similarly, especially in their initial *forme fruste* phase. This is particularly true if a precise, thorough history is not obtained.

Benign Paroxysmal Positional Vertigo

Patients presenting with classic symptoms and examination findings of benign paroxysmal positional vertigo (BPPV) are usually promptly diagnosed [9]. Patients with BPPV will, however, describe their vertigo duration in highly variable and inaccurate ways. First, many will describe the duration by the cumulative number of hours, days, weeks, or years that they are affected rather than by the duration of each episode of vertigo (i.e., less than a minute). Second, many patients avoid movements that provoke their vertigo or change positions quickly enough that they do not know its true duration. Third, BPPV may feel so severe; the vertigo likely feels longer than it truly is.

Questioning on the positional provocation may similarly lead to diagnostic confusion. Provocation of vertigo by *rolling over* in bed, typical for BPPV, may in fact be resulting from *getting out* of bed, which is commonly due to orthostatic hypotension. As both BPPV and orthostatic hypotension are most commonly found in elderly individuals, both may be present. Furthermore, many conditions are associated with the development of BPPV, including diabetes, trauma (and postsurgical), osteoporosis, vascular, psychiatric, MD, migraine, vestibular neuritis, and autoimmune disorders [10, 11]. Thus, it is not uncommon to have a presentation of BPPV along with other types of dizziness.

Residual dizziness is common after the treatment or resolution of the positional vertigo [12, 13]. Diverse explanations, including delayed compensation and underlying otolithic organ dysfunction, have been proposed as responsible mechanisms.

Semicircular Canal Dehiscence

Sound-induced (i.e., Tullio's phenomenon) or pressure-induced vertigo accompanied by autophony is pathognomonic for semicircular canal dehiscence (SCD); however, this may also present with spontaneous, ill-defined dizziness [14]. Tullio's phenomenon may occur in the absence of SCD with endolymphatic hydrops and perilymph fistula [15–18]. Clinicians must also be aware of more esoteric disorders with sound-induced dizziness, such as vestibular atelectasis, reflex epilepsy, and long QT syndrome [19–21].

Otosclerosis

Otosclerosis may be accompanied by complaints of dizziness. Though the diagnosis of otosclerosis is usually suggested by an air-bone gap on audiometry, labyrinthine dysfunction can occur without footplate fixation [22]. Vestibular complaints in the presence of otosclerosis can be highly variable. Eza-Nuñez and colleagues reported the most common presentations to be Ménière's syndrome (30%), spontaneous recurrent vertigo without hearing fluctuation (27.5%), positional vertigo (32.5%), and chronic unrelapsing imbalance (7.5%) [23]. Stapedotomy may both lead to improvement with and onset of vestibular complaints [24]. Endolymphatic hydrops is more common in patients that have undergone stapedotomy than those with unoperated otosclerosis [25].

Vestibular Migraine and Ménière's Disease

As these two conditions often have similar presenting symptoms and patients are commonly without objective findings between episodes, they can be rather difficult to distinguish. Ménière's disease (MD) may present with an isolated attack of vertigo lasting less than 30 min, with minimal or no auditory complaints [26, 27]. Conversely, vestibular migraine (VM) may present with a violent attack of vertigo lasting a few hours and is accompanied by aural fullness, tinnitus, and no history of migraine headaches [26, 28]. The diagnosis of MD and VM is based on the clinical history, and there is no definitive diagnostic test to differentiate the two diseases [29–31]. Focused questioning on a history of headaches photophobia/phonophobia and visual stigmata (e.g., seeing lights) will often be productive, as patients will tend to neglect such events that occurred in the remote past and had no clear connection to their dizziness. Similarly, a family history of vertigo and seeing lights, as well as migraine headaches, should be solicited.

Adding to the potential confusion on the distinction between MD and VM is the lack of clear consensus on the diagnostic criteria. Numerous authors and expert panels have promulgated diagnostic criteria. Whereas the criteria for MD are rather uniform (Table 22.1) [28], those for VM are not. Neuhauser et al. published the first diagnostic criteria for VM in 2001 and updated these in 2009 [31, 32]. However, in 2012, the International Headache Society (IHS) and Bárány Society proposed and validated slightly different criteria for the diagnosis of VM (Table 22.2) [26, 30]. The IHS/Bárány Society criteria have been found to be more specific but less

Table 22.1 Diagnostic criteria for Ménière's disease jointly formulated by the Classification Committee of the Bárány Society, The Japan Society for Equilibrium Research, the European Academy of Otology and Neurotology, the Equilibrium Committee of the American Academy of Otolaryngology-Head and Neck Surgery, and the Korean Balance Society

Definite Ménière's disease
Two or more spontaneous episodes of vertigo, each lasting 20 min to 12 h
Audiometrically documented low- to mid-frequency sensorineural hearing loss in one ear, defining the ear on one occasion before, during, or after one episode of vertigo
Fluctuating aural symptoms (hearing, tinnitus, or fullness) in the affected ear
Not better accounted for by another vestibular diagnosis
Probable Ménière's disease
Two or more episodes of vertigo or dizziness, each lasting 20 min to 24 h
Fluctuating aural symptoms (hearing, tinnitus, or fullness) in the affected ear
Not better accounted for by another vestibular diagnosis

Adapted from Lopez-Escamez et al. [28]

Table 22.2 Diagnostic criteria for vestibular migraine jointly formulated by the Committee for Classification of Vestibular Disorders of the Bárány Society and the Migraine Classification Subcommittee of the International Headache Society

Definite vestibular migraine
At least five episodes with vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h
Current or previous history of migraine with or without aura according to the ICHD
One or more migraine features with at least 50% of the vestibular episodes
Headache with at least two of the following characteristics: one-sided location, pulsating quality, moderate or severe pain intensity, aggravation of routine physical activity
Photophobia and phonophobia
Visual aura
Not better accounted for by another vestibular or ICHD diagnosis
Probable vestibular migraine
At least five episodes with vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h
Only one of the criteria B and C for vestibular migraine is fulfilled (migraine history or migraine features during the episode)
Not better accounted for by another vestibular or ICHD diagnosis

Adapted from Lempert et al. [26]
Abbreviations: *ICHD* international classification of headache disorders

sensitive for the diagnosis of VM than Neuhauser's criteria due to the type, duration, and intensity of vertigo specified in the IHS criteria [33].

Radtke et al. evaluated the validity of Neuhauser et al.'s criteria for definitive vestibular migraine (dVM) and probable vestibular migraine (pVM) and demonstrated high reliability, with a positive predictive value of 85% [52]. Patients meeting criteria for MD were excluded from the study, but 13% of patients diagnosed with dVM and 7% of patients diagnosed with pVM developed bilateral sensorineural hearing loss and met AAO-HNS criteria for bilateral MD. These patients had symptomatology atypical for MD including symmetric and mild hearing loss, vertigo attacks of long duration (≥3 days), imbalance with head motion, or recurrent brief attacks of spontaneous or positional vertigo. The authors thus attributed these symptoms to VM with cochlear involvement. Cochlear dysfunction and hearing loss should be rare in VM and relatively mild [35, 36]. The degree of hearing loss that should be attributed to VM alone versus comorbid MD has not been established. The rate of overlapping VM and MD is likely underestimated as patients diagnosed with MD are typically excluded from studies investigating VM [27, 34].

Neff et al. compared the presentations of patients diagnosed with MD, VM, and both MD and VM (MDVM) [27]. Neuhauser's criteria were used for VM diagnosis [32]. Symptom overlap between groups was significant, and there was no single clinical feature or vestibular test reliably differentiated between groups. Increased age, male gender, vertigo lasting hours, and SNHL were more significantly associated with the MD group; a younger age, female gender, and recurrent moderate to severe headaches were more significantly associated with the VM group. Chronic subjective dizziness (CSD) was more commonly associated with VM than MD. Aural symptoms, decreased performance on audiometry, and evidence of peripheral vestibular deficit were more prevalent in the MD and MDVM group than the VM alone group. The presence of a peripheral vestibular deficit (e.g., positive head thrust) was unable to separate MDVM and VM patients. Patient with MDVM had a significantly higher prevalence of family history of vertigo and a greater rate of perceived hearing loss than both MD and VM alone.

Lopez-Escamez et al. investigated accompanying symptoms of vestibular attacks in 268 patients with VM (84 patients), pVM (65 patients), and MD (119 patients) using the IHC criteria for VM [37]. This study also demonstrated significant symptom overlap but identified no specific clinical feature unique to VM, pVM, or for MD. Aural symptoms (tinnitus, aural fullness, hearing loss) and vomiting were more frequently seen in patients with MD than with VM during vestibular attacks. Female gender, photophobia, phonophobia, visual auras, palpitations, and anxiety were more frequently seen in patients with VM. The frequency of headaches (all types) and a migraine risk score (calculated based on headache features) were higher in patients with VM than patients with pVM and MD. A recent systematic review of the literature comparing the clinical presentations of MD and VM showed findings consistent with the studies discussed above [30]. MD patients tend to have greater age of onset and increased prevalence of hearing loss, tinnitus, aural fullness, abnormal nystagmus, abnormal caloric testing and VEMP testing results, and endolymphatic hydrops. VM patients demonstrated a higher frequency of headaches, photophobia, and auras.

Magnetic resonance imaging (MRI) has demonstrated gray matter volume reduction in patients with VM compared to controls; however, there is a paucity of literature describing distinguishing MRI findings between VM and MD. The structural and functional MRI changes in VM are similar to findings previously described for migraine headache; however, these changes extend to areas involved in multisensory vestibular control and central vestibular compensation [38]. Endolymphatic hydrops is more commonly identified in patients with MD; however, hydrops has been identified on MRI in patients with symptoms meeting criteria for VM but not for MD [39, 40]. Gurkov et al. reported several patients meeting criteria for both definite VM and definite MD failed to show hydrops on MRI [40]. It is unclear if these patients truly had both disorders, if the patients were misdiagnosed with VM, if hydrops was incidentally associated with VM, or if hydrops was independently associated with VM-related labyrinthine injury.

Vestibular testing has been used to differentiate VM and MD, although impact of these tests on management and outcomes is controversial [27]. Caloric weakness and VEMP abnormalities have not been consistently found to be more common in MD versus VM [27, 41–44] [27, 30, 45–47]. Cervical-VEMP (cVEMP) asymmetry ratios for 500 Hz:1000 Hz tone bursts and cVEMP amplitudes were found to be lower in patients with MD compared to VM and control patients [44, 48]; however, other studies failed to show a difference in amplitudes or latency [46, 47]. Ocular-VEMP (oVEMP) testing has demonstrated more abnormalities, including longer latencies, lower amplitudes, and larger asymmetry ratios in patients with MD [44, 46, 49]. Rotary chair has been unable to reliably differentiate VM from MD [27, 44]. Video head impulse testing has also been recently described to demonstrate differences in vestibulo-ocular reflex gain; however, formal investigations of its utility are warranted [43, 50].

Less Established Balance Disorders

Vestibular Paroxysmia

Neurovascular compression of the vestibular nerve as a cause of episodic vertigo was first described by Janetta in 1975 [51, 52]. In 1994, Brandt introduced the term “vestibular paroxysmia” (VP), but its use has largely been limited to the European literature [53]. In 2016, an international consensus definition and criteria for VP were published (Table 22.3) [54]. VP is thought to be a rare condition in which ephaptic discharges are produced secondary to segmental, pressure-induced compression by blood vessels in the cerebellopontine angle, most commonly a loop of the anterior inferior cerebellar artery [52]. Episodes of rotational or non-rotational vertigo last seconds to a few minutes and can be accompanied by hearing loss, tinnitus, and hypo- or hyperacusis. Vertigo may be triggered by a change in head position or hyperventilation; however, most attacks are spontaneous. High-resolution constructive interference in steady state

Table 22.3 Diagnostic criteria for vestibular paroxysmia

Vestibular paroxysmia
At least ten attacks of spontaneous spinning or non-spinning vertigo
Duration less than 1 min
Stereotyped phenomenology in a patient
Response to a treatment with carbamazepine/oxcarbazepine
Not better accounted for by another diagnosis
Probable vestibular paroxysmia
At least five attacks of spinning or non- spinning vertigo
Duration less than 5 min
Spontaneous occurrence or provoked by certain head-movements
Stereotyped phenomenology in a patient
Not better accounted for by another diagnosis

Adapted from Strupp et al. [54]

magnetic resonance imaging (MRI) may support the diagnosis by demonstrating neurovascular compression; however, neurovascular compression may be identified on MRI in up to 30% of asymptomatic patients [55]. Both microvascular decompression and medical therapy (e.g., carbamazepine) have been reported to improve the severity and reduce the duration of symptoms; however, clinical trials are lacking [52].

Persistent Postural-Perceptual Dizziness

Patients with episodic vestibular symptoms or vestibular insults may experience chronic non-vertiginous disequilibrium after or in between vertiginous attacks. This phenomenon was traditionally attributed to either uncompensated peripheral vestibular deficits or a nonorganic, psychogenic etiology. The term "persistent postural-perceptual dizziness" (PPPD) and its diagnostic criteria were introduced in 2015, using common features of several previously described conditions: space motion discomfort (SMD), visual vertigo (VV), and phobic postural vertigo (PPV), which was later refined as chronic subjective dizziness (CSD) syndrome [56]. It has been promoted as a unique disorder and has been described to affect patients between vertiginous episodes of MD or VM (Table 22.4) [27, 56, 57]. PPPD manifests as non-vertiginous dizziness or non-spinning vertigo that is present on most days for at least 3 months and is exacerbated by movement, upright posture, visual motion, and other complex visual stimuli [56, 58]. PPPD may also be precipitated by disorders that cause vertigo or other non-vertiginous dizziness, such as vestibular neuritis, BPPV, VM, syncope, traumatic brain injury, and panic attacks [59]. Differentiating VM and PPPD may be challenging as the 2012 International Headache Society (IHS) criteria for VM and PPPD overlap [8, 26] [60–62]. Comorbid conditions may include anxiety, depression, VM, or neurotologic disorders with a compensated

Table 22.4 Diagnostic criteria for persistent postural-perceptual dizziness

One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo are present on most days for 3 months or more
Symptoms last for prolonged (hours-long) periods of time, but may wax and wane in severity
Symptoms need not be present continuously throughout the entire day
Persistent symptoms occur without specific provocation but are exacerbated by three factors:
Upright posture
Active or passive motion without regard to direction or position
Exposure to moving visual stimuli or complex visual patterns
The disorder is precipitated by conditions that cause vertigo, unsteadiness, dizziness, or problems with balance including acute, episodic, or chronic vestibular syndromes, other neurologic or medical illnesses, or psychological distress
When the precipitant is an acute or episodic condition, symptoms settle into the pattern of criterion A as the precipitant resolves, but they may occur intermittently at first and then consolidate into a persistent course
When the precipitant is a chronic syndrome, symptoms may develop slowly at first and worsen gradually
Symptoms cause significant distress or functional impairment
Symptoms are not better accounted for by another disease or disorder

Adapted from Staab et al. [56]

vestibular deficit. Selective serotonin reuptake inhibitors and serotonin norepinephrine reuptake inhibitors have been reported to reduce the symptoms of PPPD in open-label studies [57]. The habituation form of vestibular and balance rehabilitation therapy has been shown to be beneficial in reducing symptoms in preliminary studies [59].

Interplay with Non-vestibular Disorders

Psychiatric Disorders

Psychiatric comorbidity and somatization are more often seen in both adult and pediatric patients experiencing dizziness and vertigo [63–65]. Anxiety and palpitations may accompany episodic vertigo with vestibular disorders. These symptoms have been found to be more common in VM than in MD [37]. Patients with MD or VM have been shown to have higher rates of psychiatric comorbidity than patients with BPPV or vestibular neuritis. Neuroticism and panic in patients with vestibular disorders and comorbid anxiety may exacerbate the symptoms experience [8]. Anxiety associated with vertical height intolerance has been shown to adversely affect gaze control and reduce gaze stability on visual targets, potentially contributing to vestibular symptoms [66]. Psychiatric consultation is recommended in patients presenting with bothersome dizziness and comorbid psychiatric conditions.

Central Nervous System Disorders

Central nervous system (CNS) disorders affecting the pathways involving brainstem or cerebellum may be comorbid or difficult to differentiate from peripheral vestibular lesions [67]. Vertigo, imbalance, nausea, vomiting, ataxia, and multidirectional nystagmus that is not suppressed by visual fixation may be present in isolation or with other focal neurological signs, suggesting a central vestibular disorder. The combination of head impulse, nystagmus type, and test of skew (HINTS) is likely the most accurate means of distinguishing stroke from an acute peripheral event [68, 69]. Central vestibular disorders, such as cerebrovascular disease and multiple sclerosis, are discussed in detail elsewhere in this text.

Susac's Syndrome

Susac's syndrome is thought to be an autoimmune disease of the small arteries of the brain, inner ear, and retina [70]. The classic triad includes encephalopathy with or without focal neurological signs, retinal artery branch occlusions, and hearing loss. The latter is typically bilateral, low-frequency sensorineural hearing loss, and it may be accompanied by vestibulopathy. MRI of the brain is key to making the diagnosis, particularly in the absence of retinal manifestations.

Cervicogenic Vertigo

Vertigo and dizziness have been associated with cervical pathology. Several terms have been used to describe this phenomenon, including "cervical" or "cervicogenic" dizziness or vertigo. This entity lacks a validated diagnostic test and is controversial among balance specialists [71]. Several etiologies have been described, including vertigo associated with vertebral artery impingement as well as cervical injuries, such as whiplash [72]. Rotational vertebral artery syndrome (RVAS), or "Bow-Hunter syndrome," has been described as a rare etiology in which vascular compression occurs at the atlantoaxial level, most commonly by a cervical osteophyte [71, 73]. Treatment addresses the vascular compression, as physical therapy may exacerbate symptoms and decrease blood flow.

Cervical vertigo associated with degenerative cervical spine disease and injury is more commonly described, and its etiology is based on the somatosensory input hypothesis [72]. Cervical proprioceptive pathways integrate in the vestibular nuclei and modulate peripheral vestibular discharges and posture. Somatosensory signals alone may generate vertigo or nystagmus [72]. Dysfunction of the joints, muscles, and ligaments may cause pain, limited range of motion, dizziness, imbalance, motion sickness, aural fullness, phono- and photosensitivity, and nausea [74].

Symptoms are associated with cervical movement and may be episodic, lasting minutes to hours, mimicking other episodic vestibular disorders. One may mistake cervical motion-triggered dizziness with dizziness triggered only by head and body position, such as BPPV or orthostatic hypotension. Dizziness is often comorbid with cervical disorders in the elderly population as this population also suffers from vascular, osteoarticular, and degenerative cervical disease [72]. Proprioceptive and vestibular function deteriorates in older adults, and sensory input from cervical pain stimuli is altered; the sense of head position may thus be impaired, resulting in dizziness [75, 76]. Cervical vertigo is considered by many to be a diagnosis of exclusion, and the differential diagnosis must include presbystasis and multisensory imbalance in patients with peripheral neuropathy or visual deficits.

Trauma may also affect cervical proprioception and create a mismatch in sensory input with proprioceptive, visual, and vestibular inputs [71]. Patients with whiplash-associated disorder after cervical trauma infrequently report true vertigo; however; light- or heavy-headedness and imbalance are commonly encountered. Although vestibular therapy may be helpful, the beneficial role of physical therapy to improve neck mobility and relieve pain and spasm must not be underestimated [77]. Cervical disk decompression may be an option for patients with associated radiculopathy or severe degenerative disease [78, 79].

Ophthalmologic Conditions

Ophthalmologic conditions are not commonly the dominant etiology for dizziness; however, conditions affecting visual input and integration can produce several symptoms that mimic a vestibulopathy [80]. Ocular disorders have been reported to be responsible for instability, disequilibrium, or vertigo in both the adult and pediatric populations [81]. Patients with binocular visual dysfunction in conditions such as vertical heterophoria, vergence insufficiency, or latent strabismus may report dizziness, light-headedness, nausea, anxiety, vertigo, headache, and motion sickness [80, 81]. These conditions warrant ophthalmologic referral.

An intact visual system is necessary for central nervous system compensation to vestibular pathology. Advanced cataracts, macular degeneration, and poorly corrected refraction can prevent central compensation to simple vestibulopathies [82–84].

Oscillopsia, or the illusion of motion of the visual surroundings, may be mistaken by clinicians for vertigo. Patients may describe oscillopsia as their visual field jumping, wobbling, or becoming difficult to focus, particularly while walking [85]. In addition to bilateral vestibular hypofunction, oscillopsia may be secondary to brainstem and cerebellar lesions, head tremor (pseudonystagmus), voluntary nystagmus, ocular flutter, or superior oblique myokymia [86]. Oscillopsia must not be confused with the development of vertigo or unsteadiness associated with complex or moving visual stimuli, as seen in PPPD and other vestibular disorders [56, 86].

Cardiovascular Disease

Cardiovascular disorders are a common cause of dizziness; however, isolated vertigo is an uncommon manifestation of cardiac disease. Orthostatic hypotension is typically associated with presyncope and "light-headedness," but patients may report vertigo or the illusion of motion during tilt-table testing [87]. Vertigo has also been reported to be a primary manifestation of a cardiac arrhythmia [88]. Sound has been reported as a trigger of long QT syndrome, mimicking Tullio's phenomenon. Culić et al. reported that 8% of patients with acute myocardial infarction reported vertigo as one of their presenting symptoms [89]. These forms of "cardiogenic" vertigo are hypothesized to be secondary to asymmetric perfusion of the right- and left-sided vestibular structures in the inner ear and cerebellum during an episode of global hypoperfusion caused by cardiovascular disease [88]. The vertebral arteries may demonstrate underlying asymmetry or atherosclerosis that may predispose patients to focal ischemia vestibular structures. Vertigo may thus be more commonly attributed to cardiovascular disease than would be expected. Given the episodic nature of cardiac arrhythmias, these attacks of cardiogenic vertigo can mimic more common vestibular disorders.

Endocrine Disorders

A range of endocrine disorders can present with atypical forms of dizziness. Diabetes mellitus (DM) is associated with a high rate of dizziness [90]. Even in the absence of symptomatic dizziness, DM often causes vestibular dysfunction, especially utricular hypofunction [91]. In addition to the otologic impact, DM insidiously leads to multisystem organ dysfunction in the cardiovascular system, brain, peripheral nerves, and eyes. As organ involvement differs by individual, the nature of the underlying vestibular complaints can vary tremendously. As indicated above, DM may both contribute to the pathogenesis and the clinical course of common vestibular disorders, such as BPPV and MD [92–94]. There is evidence that the effects of DM on the vestibular system are mediated by hypertension [93]. Hypothyroidism is similarly associated with vestibular pathology, including MD [95–99].

Obesity and Obstructive Sleep Apnea

Obesity has long been linked to benign intracranial hypertension (BIH, aka pseudotumor cerebri) [100]. BIH commonly presents with dizziness and pulse-synchronous tinnitus, as well as headaches [101]. The presentation of headaches and dizziness can mimic migraine [102]. BIH has also been associated with the development of

MD [103]. Obesity has been linked to superior semicircular canal dehiscence [104]. Though related to obesity, obstructive sleep apnea has been independently associated with hearing loss, tinnitus, increased intracranial pressures, idiopathic dizziness and vertigo, and possibly MD [105–110]. Recognition of the role of obesity and OSA is important, as treatment with weight loss and CPAP may improve functional outcomes [111].

Medications and Medication Withdrawal

Dizziness is commonly reported with a wide range of medication therapy [112]. This is particularly true in elderly patients that are taking numerous medications with overlapping effects (i.e., polypharmacy) [113]. Many have direct, central nervous system effects or side effects. Many affect the balance system secondarily, particularly in the presence of underlying pathology, such as cardiac ischemia or dysrhythmias and VM. Abrupt discontinuation of selective serotonin reuptake inhibitors is known to cause dizziness, which may be positionally provoked [114].

Aging

Advancing age brings progressive degeneration of all organ systems, making dizziness and falls common in the elderly [115–117]. This often leads to a vicious cycle that includes a more sedentary lifestyle and generalized deconditioning and further compounds the symptoms resulting from vestibular dysfunction, CNS pathology, and vision loss [118]. Treatment of numerous vestibular and non-vestibular conditions may be needed to effect clinical improvement [119, 120]. Since many conditions afflicting the elderly cannot be treated, some authors have advocated forgoing exhaustive diagnostic evaluations in favor of focusing on establishing a prognosis and managing treatable issues (e.g., BPPV and polypharmacy) [121, 122].

Conclusions

Patients with complex dizziness and vestibular symptoms overlapping multiple disorders are challenging to diagnose. Episodic vertigo carries a broad differential diagnosis. A careful history including type, duration, and triggers of dizziness, associated with headaches, medical comorbidities, family history, and neurotologic examination is recommended. There is no single clinical feature or test to reliably differentiate the underlying conditions in patients with overlapping symptoms. Comorbid PPPD may complicate the diagnosis and may generate an additional sensation of dizziness between episodes of vertigo. Non-vestibular pathologies such as

central nervous system, cardiac, ophthalmologic, and musculoskeletal disorders can generate vertigo and non-vertiginous dizziness by affecting multisensory input, central integration, or vestibular motor output. Clinicians must be cognizant of atypical presentations of vestibular disorders and understand the impact of comorbid psychiatric and medical disorders on the clinical manifestations of balance disorders.

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