

Review Article

Migraine and Vertigo

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Migraine and vertigo are common disorders in medicine, affecting about 14–16% and 7–10%, respectively, of the general population. Recent epidemiologic studies indicate that 3.2% of the population have both migraine and vertigo. Vertigo may occur in up to 25% of patients with migraine. Migraine is the most frequent vascular disorder causing vertigo in all age groups. Migraine leads to various central or peripheral vestibular syndromes with vertigo such as migrainous vertigo, basilar-type migraine, benign paroxysmal vertigo of childhood, and other vertigo syndromes related to migraine. Migrainous vertigo is the most common cause of spontaneous recurrent vertigo. Diagnostic criteria for migrainous vertigo have been proposed but are not included in the most recent International Headache Society classification of migraine. On the other hand, there are statistical associations between migraine and vertigo syndromes including benign paroxysmal positional vertigo, Meniere's disease, persistent cerebellar symptoms, anxiety-related dizziness, and motion sickness. Vertigo can also act as a migraine trigger. Although some mutations in the CACNA1A gene have been identified in some familial cases, the mechanism of migraine-associated vertigo is still obscure. Treatment includes vestibular suppressants for acute attacks and migraine prophylaxis for patients with frequent attacks.

1. Introduction

Headache and dizziness are two of the most frequent symptoms occurring in the general population. Both migraine and vertigo are common in the general population with lifetime prevalences of about 16% for migraine and 7% for vertigo [1]. They are also two clinical disorders that tend to occur together. Although a concurrence of the two conditions can be expected in about 1.1% of the general population by chance alone, a recent epidemiologic study reveals that the actual comorbidity is 3.2%. Vertigo may occur in up to 25% of patients with migraine [2, 3]. According to several epidemiological studies, it seems that migraine is the most frequent vascular disorder causing vertigo in all age groups [4, 5].

Migraine may be associated with many vestibular symptoms including episodic vertigo, chronic motion sensitivity, and nonspecific dizziness. The principal clinical vestibular syndromes associated with migraine can be classified as migrainous vertigo (or vestibular migraine), basilar-type migraine, benign paroxysmal vertigo of childhood, and other vertigo/dizziness syndromes related to migraine. On

the other hand, there are statistical associations between migraine and vertigo syndromes including benign paroxysmal positional vertigo, Meniere's disease, persistent cerebellar symptoms, anxiety-related dizziness, and motion sickness. Vertigo can also act as a migraine trigger. Although migrainous vertigo is the most common cause of spontaneous recurrent vertigo, it is presently not included in the most recent International Headache Society classification of migraine [6–9]. In this review, the epidemiology of vertigo due to migraine and the clinical vestibular syndromes associated with migraine are discussed.

2. Method

The articles and abstracts for this review were found by searching in MEDLINE/PubMed. A Medline literature search was conducted with, individually or in combination, the search terms vestibular migraine, migraine-associated vertigo, migraine-associated dizziness, migrainous vertigo, migraine and vertigo, migraine and disequilibrium, and headache and vertigo. Additional articles were obtained from the reference lists of retrieved articles.

3. Epidemiology

Migraine is estimated to occur in 18% to 29% of women, 6% to 20% of men, and 4% of children. Vertigo and dizziness also rank among the most common complaints in medicine, affecting approximately 20% to 30% of the general population. The lifetime prevalence of vertigo in adults aged 18–79 years is 7.4%, the 1-year prevalence is 4.9% and 1-year incidence is 1.4% [1, 10]. Although vertigo is a rare primary complaint of children, it makes up a little less than 1% of all children visiting hospital during a 5-year period [4, 11]. Thus, both migraine and vertigo are common in the general population with lifetime prevalences of about 16% for migraine, 7% for vertigo, and 3.2% for comorbidity of the two conditions. Conversely, vertigo may occur in up to 25% of patients with migraine. Migraine is associated with various vertigo syndromes such as migrainous vertigo, basilar-type migraine, benign paroxysmal vertigo of childhood, and other vertiginous syndromes [2, 9, 12].

Migrainous vertigo (MV), which is vertigo directly caused by migraine, is relatively frequent in migraine patients especially in migraine with aura, and it affects more than 1% of the general population, about 10% of patients in dizziness clinics, and at least 9% of patients in migraine clinics [2, 13]. The lifetime prevalence of MV is 0.98%, and the 1-year prevalence is 0.89% in adult population [9, 12]. There is a female preponderance with a reported female-to-male ratio of 1.5–5:1 in adults with MV [10]. MV may occur at any age and is the most common diagnosis in children presenting vertigo, and the prevalence of MV is estimated at 2.8% of children between ages 6–12 years [10, 11]. Although vertigo in childhood is a complaint consisting of a wide spectrum of diagnoses, migraine, middle ear infections, and benign paroxysmal vertigo of childhood are found to be the most frequent presenting diagnosis in childhood vertigo in most studies [4, 11, 14–16].

Although basilar-type migraine (BTM) affects adolescents more frequently than adults, incidence figures are lacking. Benign paroxysmal vertigo of childhood is a paroxysmal, nonepileptic, recurrent event characterized by subjective or objective vertigo that occurs in neurologically intact children. It is not uncommon in children, and is the most frequent syndrome occurring in 38% among childhood periodic syndromes [17].

On the other hand, several dizziness and vertigo syndromes occur more frequently in migraineurs than in controls [5, 8]. Conversely, migraine and motion sickness are three-times more common in patients with BPPV of unknown cause than in the general population [18, 19]. The lifetime prevalence of migraine is also increased in patients with Meniere's disease compared with controls, 56% versus 25% [20]. Motion sickness is significantly higher in individuals who suffered from migraine [21]. There is an increased prevalence of migraine headache among patients with depression and anxiety disorders as compared to the general population [22].

4. Vertigo Syndromes Related to Migraine

Vertigo is an illusory sensation of movement and occurs in asymmetric involvement of the vestibular system. When the vertiginous sensation is one of horizontal environmental spin or of clear self-rotation, the lesion is peripheral, mostly in the vestibular endorgan. Autonomic symptoms such as sweating, pallor, nausea, and vomiting are also commonly associated with peripheral vertigo [3]. An illusion of linear movement and tilting suggests isolated involvement of utricle and saccule, respectively, and their central connections. Central vertigo that makes up only about 25 percent of diagnoses of patients presenting with vertigo is generally associated with severe imbalance, additional neurologic signs, less prominent movement illusion, and nausea and central nystagmus [23]. The patient's history and clinical findings are the keys to differentiation of peripheral or central vertigo, and to diagnosis of causes. The most common causes of vertigo are BPPV, vestibular neuritis, Meniere's syndrome, and vascular disorders, respectively. Migraine from a vascular cause leads to various central or peripheral vestibular syndromes [23, 24].

It is well known that vertiginous syndromes and migraine can coexist. Vertigo and dizziness can be related to migraine in various ways: causally, statistically, or, quite frequently, just by chance. Migraine may be associated with many vestibular symptoms including episodic vertigo, chronic motion sensitivity, and nonspecific dizziness. On the other hand, patients with migraine may present with vertigo/dizziness syndromes more often than patients without migraine [2, 5, 12, 13]. The principal clinical vestibular syndromes associated with migraine can be classified as below:

- (a) migrainous vertigo (or vestibular migraine),
- (b) basilar-type migraine,
- (c) benign paroxysmal vertigo of childhood,
- (d) other vertiginous syndromes related to migraine.

4.1. Migrainous Vertigo. Vertigo is frequently associated with migraine, and sometimes it is the cardinal symptom of migraine. This type of migraine is called "migrainous vertigo," "vestibular migraine," "migraine-associated vertigo," or "migraine-related vertigo" [9, 12]. Migrainous vertigo (MV) is relatively common but underdiagnosed in the general population, and it has considerable personal and healthcare impact. MV is a vestibular disorder caused by migraine, which presents with attacks of spontaneous or positional vertigo lasting seconds to days and migrainous symptoms during attack; however, the patients do not fulfil the criteria of the International Headache Society for basilar-type migraine. It is relatively more frequent in migraine with aura than without aura [5, 9, 25]. Although MV is the most common cause of spontaneous recurrent vertigo, some clinical diagnostic criteria have been proposed but are not included in the most recent International Headache Society classification of migraine [6]. MV can be diagnosed according to the following criteria given in Table 1 [9].

TABLE 1: Diagnostic criteria for migrainous vertigo [9].

(1) Recurrent vestibular symptoms such as rotatory/positional vertigo, other illusory self or object motion, and head motion intolerance
(2) Migraine according to criteria of the International Headache Society
(3) At least 1 of the following migrainous symptoms during at least 2 vertiginous attacks: <i>migrainous headache, phonophobia, photophobia, visual, or other auras</i>
(4) Exclusion of other causes

MV may occur at any age, usually begins in the first 3 decades of life, with a peak in the fourth decade in men and a “plateau” between the third and fifth decade in women. The natural course of MV is not well known. Benign paroxysmal vertigo of childhood can be an early manifestation of MV in children presenting with vertigo. In most patients, migraine headaches begin earlier in life than MV. Vertigo is occasionally coincident with headache, but more often occurs as an isolated symptom. Migraine-related vertigo is characterized by varying motion illusions and motion sensitivity, often with nausea. Vestibular symptoms associated with migraine are often described as spinning, slow or fast rotation, rocking, tilting, swaying, swimming, to-and-fro oscillation, or floating and head motion intolerance. Attack duration often varies from seconds to days [1, 25–28]. Spell frequencies also vary from 1 per month to >40 per month. Most of the patients have vertigo attacks lasting minutes or hours and most are completely free of dizziness between attacks. Imbalance and nausea typically accompany the vertigo. However, in half of the cases, vertigo occurs without an association with headache, but some migrainous features such as photophobia or auras may be present [1, 27]. Like migraine headaches, stress, sleep deprivation, and hormonal changes may also trigger MV. Although auditory symptoms are usually not seen in patients with MV, some patients have also auditory symptoms of tinnitus, episodic hearing loss, or aural fullness during the attack [28–32]. In neurologic examination during acute attacks, central spontaneous or positional nystagmus and, less commonly, unilateral vestibular hypofunction can be found. In the symptom-free interval, vestibular testing adds little to the diagnosis as findings are mostly minor and nonspecific [27, 29, 32]. However, in the study by Dieterich and Brandt, most of the patients with MV showed mild central ocular motor signs such as vertical (48%) and/or horizontal (22%) saccadic pursuit, gaze-evoked nystagmus (27%), moderate positional nystagmus (11%), and spontaneous nystagmus (11%) in the symptom-free period [30]. Some of the patients with migraine may present with benign paroxysmal positional vertigo. The following features such as short-duration symptomatic episodes and frequent recurrences, manifestation early in life, migrainous symptoms during episodes with positional vertigo, and atypical positional nystagmus help to distinguish migrainous positional vertigo from BPPV [19]. On the other hand, the features of MV resemble vertebrobasilar insufficiency (Table 2). In differential diagnosis between vertigo of posterior circulation ischemia and migrainous vertigo, motion sickness, motion sensitivity, photophobia, and phonophobia are principal differential features to MV. Furthermore, abnormal blood

TABLE 2: Differential diagnosis of migrainous vertigo [7].

Disease	Duration of vertigo
Migrainous vertigo	Minutes to hours, or days (rarely)
Vertebrobasilar insufficiency	Minutes (usually)
Meniere’s disease	Hours
Labyrinthitis	Days
BPPV	Seconds

BPPV, benign paroxysmal positional vertigo.

pressure, abnormal blood fat, pathoglycemia, and arteriosclerosis are usually found in the posterior circulation ischemia [7, 13, 26].

The mechanism of MV is still obscure. The epidemiological link between migraine and vestibular syndromes suggests shared pathogenetic mechanisms between the vestibular nuclei, the trigeminal system, and thalamocortical processing centers providing the basis for the development of a pathophysiological model of migrainous vertigo. It has been believed that vertigo in migraine may arise from disorders such as cortical spreading depression, regional changes in brain perfusion, release of neurotransmitters and paroxysmal dysfunction of ion channels anywhere along the peripheral and/or central vestibular structures at the labyrinth, brainstem, and cerebral cortex [29, 31, 32]. On the other hand, it is also speculated that a migrainous aseptic inflammation is thought to create a central sensitivity that spreads from the trigeminal to the vestibular system [33]. Migrainous vertigo is sometimes inherited as an autosomal dominant trait. With regard to the pathogenesis of autosomal inherited familial migraine with vertigo, no mutations were found in the voltage-gated calcium channel gene and CACNA1A gene [34]. Although findings demonstrate that migrainous vertigo is genetically heterogeneous and complex, it has been recently reported that locus for cases with familial migrainous vertigo maps to chromosome 5q35 [35].

Treatment of migrainous vertigo currently parallels that of migraine headache including dietary changes, medication, physical therapy, lifestyle adaptations, and acupuncture. Mild symptoms or brief or infrequent spells may be left untreated. The long-lasting (at least 30 minutes) and frequent symptoms need vestibular suppressants such as meclizine, dimenhydrinate, diazepam, or promethazine during vertigo attacks, and treatment of acute migraine attack including intravenous methylprednisolone, zolmitriptan, and a migraine prophylactic drug. Vestibular suppressants often reduce symptoms, but do not abort vertigo, and

TABLE 3: Diagnostic criteria for basilar-type migraine (IHS).

(a) At least 2 attacks fulfilling criteria (b)–(d)

(b) Aura consisting of at least two of the following fully reversible symptoms, but no motor weakness:

- (1) dysarthria
- (2) vertigo
- (3) tinnitus
- (4) hypacusia
- (5) diplopia
- (6) visual symptoms simultaneously in both temporal and nasal fields of both eyes,
- (7) ataxia
- (8) decreased level of consciousness
- (9) simultaneously bilateral paraesthesias

(c) At least one of the following:

- (1) at least one aura symptom develops gradually over ≥ 5 minutes and/or different aura symptoms occur in succession over ≥ 5 minutes
- (2) each aura symptom lasts ≥ 5 and ≤ 60 minutes

(d) Not attributed to another disorder

have sedating side effects. The prophylactic medication may be effective for treating MV and its associated symptoms; therefore, patient's response to medical therapy may also provide guidance in the diagnostic process of MV [1, 26, 36]. On the other hand, although migraine with aura was associated with increased risk of major cardiovascular and cerebrovascular disorders, and death due to ischemic events, preventive medications for migraine with aura or antiplatelet therapy might reduce the risk of vascular disorders [26, 36–38]. Although treatment efficacy in MV has not been validated by properly controlled clinical trials so far, the observational studies show marginal improvement with migraine prophylactic medications such as nortriptyline, verapamil, or metoprolol [39]. Lomerizine, a calcium channel antagonist, may be effective as a treatment for migraine-associated vertigo [40]. Although prophylactic low-dose valproic acid decreases the frequency of headache and vestibular symptoms, it can be used satisfactorily for patients with MV [41]. In a recent study, topiramate was also found to be effective in reducing the frequency and the severity of vertigo and headache attacks in MV [42]. However, patients with migraine-associated vertigo and dizziness can also benefit from physical therapy intervention [43]. Avoidance of migraine triggers, stress management, and biofeedback may also play a role in preventive strategies [1].

4.2. Basilar-Type Migraine. Basilar-type migraine (BTM) was first described by Bickerstaff in 1961, and originally the terms “basilar artery migraine” or “basilar migraine” were used but, since involvement of the basilar artery territory is uncertain, the term basilar-type migraine is preferred. BTM, as a subtype of migraine with aura, is characterized by recurrent headaches, usually occipital, associated with aura symptoms localizing to the vascular territory of the basilar artery. BTM is the most common migraine “variant,” representing 3%–19% of migraine, and affects all age groups and both sexes [3, 38, 44]. This syndrome affects adolescents more frequently than adults. The International

Headache Society criteria for BTM require the presence of 2 or more preceding aura symptoms (Table 3). The aura generally lasts less than 1 hour, and is usually followed by a headache that may be occipital. A typical hemianoptic field defect can rapidly expand to involve all visual fields, leading at times to temporary blindness. The visual aura is usually followed by vertigo, tinnitus, decreased hearing, diplopia, ataxia, dysarthria, bilateral paresthesia, and impaired cognition. The headache can be associated with nausea and projectile vomiting. BTM should be diagnosed only when no motor weakness occurs. The bilateral nature of neurologic findings in basilar-type migraine helps to differentiate it from more typical migraine. The relationship between BTM and MV refers to a distribution of severity across the disease spectrum of migraine-related vertigo. BTM presents the most severe form, and MV is the mildest form in clinical manifestation and brainstem involvement [1, 38, 44]. Electroencephalography between or during attacks of BTM may reveal occipital spike discharges. In the differential diagnosis one should consider the pathology of posterior fossa, diseases with recurrent vertigo, temporal or occipital lobe epilepsy, occipital neuralgia, vascular disease, CADASIL, MELAS, and alternative hemiplegic migraine with cerebellar syndrome [38, 44]. It is most likely that vertigo in BTM is due to a functional vestibular tone imbalance caused by an asymmetric involvement of bilateral vestibular neuronal activity during the attack [1, 30].

In the prophylaxis of BTM, sodium valproate, topiramate and calcium channel antagonists, and especially in the prophylaxis of vertigo, betahistine chloride are used. In a recent study assessing the efficacy and safety of topiramate for prophylaxis of BTM in children and adolescents, preventive therapy with topiramate resulted in reducing the overall migraine frequency and the frequency of attacks of BTM at both 25 mg and 100 mg doses without serious adverse events [45]. Triptans are contraindicated in BTM because of theoretical risks of vasospasm and stroke [1, 38, 45].

TABLE 4: Diagnostic criteria for benign paroxysmal vertigo of childhood (IHS).

(a)	At least 5 attacks fulfilling criterion (b)
(b)	Multiple episodes of severe vertigo, occurring without warning and resolving spontaneously after minutes to hours
(c)	Normal neurological examination and audiometric and vestibular functions between attacks
(d)	Normal electroencephalogram

4.3. Benign Paroxysmal Vertigo of Childhood. Benign paroxysmal vertigo of childhood, as a subtype of childhood periodic syndromes in migraine, is characterized by onset between 1 and 4 years, abrupt randomly occurring attacks of vertigo and imbalance often with nausea and vomiting that lasts for 30 seconds to 20 minutes, usually unaccompanied by headache. Episodes begin suddenly with a sensation of anxiety and fear, and may be associated with pallor, nausea, or diaphoresis. Older children usually describe a sensation of vertigo. Sleep may occur after an episode of vertigo. Consciousness is not lost. Nystagmus as an objective evidence of vestibular dysfunction may be present during attack. These children are healthy between attacks. This syndrome often subsides by adolescence or evolves into migraine headaches. These patients generally have a positive family history of migraine and a history of motion sickness. Electroencephalography and neuroimaging are normal (Table 4). This syndrome can be considered a migraine equivalent or a migraine precursor, and could be due to the same vascular and/or biochemical mechanisms responsible for the migraine. There is strong evidence for close relationship between benign paroxysmal vertigo of childhood, spasmodic torticollis, BPPV, and migraine [1, 46, 47].

In prophylaxis of cyclic vomiting syndromes of childhood that are precursors to migraine, especially with antihistamines such as cyproheptadine in children 5 years or younger and amitriptyline for those older than 5 years can be effective at decreasing the frequency and severity of attacks [38, 47]. And, triptans, as abortive therapy, can be used in acute therapy [48].

4.4. Other Vertiginous Syndromes Related to Migraine. Patients with migraine may also present with benign paroxysmal positional vertigo, Meniere's disease, psychogenic dizziness, motion, sickness, and other vestibular disturbances more often than patients without migraine. However, persistent cerebellar syndrome may develop in the course of familial hemiplegic migraine. Dizziness also may be due to orthostatic hypotension, anxiety disorders, or major depression, all of them have an increased prevalence in migraineurs [1, 7, 25–27, 31].

BPPV is characterized by sudden, severe attacks of either horizontal or vertical vertigo, or a combination of both, precipitated by certain head position changes and movements. BPPV can be caused by either canalolithiasis or cupulolithiasis, and can theoretically affect each of the 3 semicircular canals. Migraine has been found to be closely associated with BPPV, and is 3-times more common in patients with BPPV. Patients with MV can present with any combination of recurrent spontaneous vertigo, migrainous positional vertigo, or head

motion intolerance. Migrainous isolated episodic positional vertigo mimics BPPV. The following factors help to distinguish migrainous positional vertigo from BPPV: short-duration symptomatic episodes and frequent recurrences, manifestation early in life, migrainous symptoms during episodes with positional vertigo, and atypical positional nystagmus [3, 18, 19, 49]. Ishiyama and colleagues and Lempert and colleagues also found an increased incidence of migraine in patients with BPPV and higher recurrence rates of BPPV after successful positioning in patients with migraine [19, 50]. It has been suggested that spasm of the inner ear arteries or some other mechanisms due to migraine may be possible causative mechanisms [51]. BPPV and Meniere's disease may also be triggering migraine headaches [52].

The lifetime prevalence of migraine is increased in patients with Meniere's disease. The frequent occurrence of migrainous symptoms during Meniere's attacks suggests a pathophysiologic link between the two diseases [20]. Attacks are usually accompanied by either headache, photophobia, or aura in 45% of patients with Meniere's disease. Although hearing loss may be a feature of migraine, fluctuating acoustic symptoms are key symptoms for diagnosis of Meniere's disease. Since their clinical features may overlap, it can be difficult to distinguish between Meniere's disease and MV [1, 20].

Persistent cerebellar symptoms may develop in the course of familial hemiplegic migraine. Familial hemiplegic migraine, spinocerebellar ataxia type 6, and episodic ataxia type 2 are allelic disorders associated with mutations in the CACNA1A gene, which is caused by the alteration of the alpha 1A voltage-dependent calcium channel subunit [53]. On the other hand, benign paroxysmal torticollis of infancy is also a disorder characterized by recurrent episodes of head tilt secondary to cervical dystonia often accompanied by vomiting, pallor, and ataxia, settling spontaneously within hours or days. Episodes begin within the first year of life and resolve by 5 years. Head tilt becomes less prominent after infancy, replaced by vertigo and eventually by migraine headaches. Benign paroxysmal torticollis of infancy may also be an age-sensitive and migraine-related disorder [1, 7, 54].

Motion sickness is a common form of physiologic dizziness usually caused by prolonged vestibular stimulation. Patients with migraine are generally more prone to motion sickness, and attacks of motion sickness during childhood may be the first symptom of migraine. The mechanisms of MV and motion sickness remain unknown. A concurrence of motion sickness and allodynia in migraine patients supports the importance of central mechanisms of sensitization for migraine-related vestibular symptoms [55]. However, it is speculated that innate hypersensitivity of the vestibular

system may be an underlying mechanism of motion sickness in migraine [56]. Dizziness may also be due to orthostatic hypotension, anxiety disorders, or major depression which all have an increased prevalence in patients with migraine [25].

Conversely, it has also been reported in some patients that vestibular stimuli can trigger migraine attacks. Induced vertigo can act as a migraine trigger, a finding with implications for the diagnosis of patients with episodic vertigo and migraine headache. While such patients may have BTM or MV, alternatively, another disorder causing episodic vertigo such as BPPV or Meniere's disease may trigger migraine headaches [1, 52].

5. Conclusion

Vertigo, as a common complaint in medicine, is an illusory sensation of movement, and occurs in asymmetric involvement of the vestibular system by various causes. Migraine is the most frequent vascular disorder causing vertigo in all age groups. MV, which is vertigo directly caused by migraine, occurs in up to 25% of patients with migraine, especially in migraine with aura, and presents with attacks of spontaneous or positional vertigo, dizziness lasting seconds to days, and migrainous symptoms during the attack. Although MV is the most common cause of spontaneous recurrent vertigo, it is presently not included in the International Headache Society classification of migraine. BTM and benign paroxysmal vertigo of childhood are also vertigo syndromes associated with migraine. BPPV and Meniere's disease are statistically related to migraine, but the possible pathogenetic links have not been established. Moreover, migraineurs suffer from motion sickness and head motion intolerance more often than controls. Persistent cerebellar symptoms may develop in the course of familial hemiplegic migraine. Dizziness may also be due to orthostatic hypotension, anxiety disorders, or major depression which all have an increased prevalence in patients with migraine. However, vestibular stimuli can also trigger migraine attacks.

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