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Original Article

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Clinical Features of Migraine, Vestibular Migraine, and Tension-Type Headache and Their Vestibular Evoked Myogenic Potential Study

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Abstract

Background: Migraine, vestibular migraine (VM), and tension-type headache (TTH) are commonly associated with dizziness, vertigo, and postural instability, which increases patients' risk of falling and contributes to anxiety and depression. However, the vestibular pathophysiology underlying these primary headache disorders remains unclear. This study aimed to assess the saccular and utricular functions using vestibular evoked myogenic potentials (VEMPs), to investigate the peripheral and central vestibular involvement across these headaches.

Methods: A total of 353 patients diagnosed with migraine, VM, or TTH, based on the International Classification of Headache Disorders, third edition (beta version, ICHD-3β), were recruited from the Dizziness and Headache Clinic at People's Hospital of Weifang between December 2019 and September 2022. All participants underwent standardized clinical assessments and demographic data collection. VEMP tests were performed using 95 dB air-conducted sound stimuli to evaluate peripheral and central vestibular functions prior to enrollment.

Results: Sleep disturbances and psychiatric comorbidities (i.e., anxiety and depression) were significantly more prevalent in TTH patients compared to those with VM and migraine. VM patients also demonstrated higher rates of psychiatric comorbidities than migraine patients. The average headache duration in VM patients was 7.14 years, which was notably longer than the average dizziness duration of 4.03 years. Transient vertigo was reported in 22% of VM patients and 17.65% of TTH patients. The prevalence of occipital and/or neck pain was significantly higher in VM patients than in migraine patients. Absent ocular VEMP (oVEMP) responses, both unilateral and bilateral, were found at a significantly higher rate in VM patients compared to migraine patients. Additionally, cervical VEMP (cVEMP) asymmetry ratios (ARs) were significantly higher in VM patients compared to TTH patients, and marginally higher than in migraine patients (P = 0.05). Prolonged cVEMP latencies (right p13, n23, and interpeak intervals) were observed in both VM and migraine compared to TTH. Left-sided latencies were significantly prolonged in migraine than TTH.

Conclusions: Psychiatric comorbidities were most pronounced in TTH, followed by VM and migraine. Both VM and TTH were associated with transient vertigo, exposing patients to drop-attack risk. The significantly higher occipital and/or neck pain reported in VM than in migraine may suggest the cervical neurovascular involvement in its pathophysiology. VEMP results indicate peripheral vestibular dysfunctions in VM patients and lower brainstem involvement in both VM and migraine patients, with the right-sided abnormalities more severe than the left-sided ones.

Keywords: Vestibular evoked myogenic potentials; Ocular vestibular evoked myogenic potentials; Cervical vestibular evoked myogenic potentials; Migraine; Vestibular migraine; Tension-type headache

Introduction

Migraine, vestibular migraine (VM), and tension-type headache (TTH) are the most common primary headaches and are often associated with gait imbalance, sleep disorders, anxiety, and depression. Migrainous vertigo is one of the most common presenting complaints in headache and vertigo clinics. VM attacks can manifest as a broad spectrum of vertigo or dizziness, lasting from seconds to days, and may occur spontaneously, positionally, or be triggered by specific stimuli. However, the underlying pathophysiological mechanisms remain under debate [1].

Cervical vestibular evoked myogenic potentials (cVEMPs) are short-latency inhibitory reflexes recorded from the ipsilateral sternocleidomastoid (SCM) muscle, whereas ocular VEMPs (oVEMPs) are excitatory reflexes recorded from the contralateral inferior oblique muscle [2]. These responses are typically elicited by air-conducted (AC) click sound stimulation delivered through headphones [3-5].

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Delayed VEMP reflex latencies have been attributed to central pathology, typically involving the brainstem, whereas absent responses or reduced amplitudes are more indicative of peripheral causes [6, 7]. VEMP signals are transmitted to the vestibular nuclei via the vestibular nerves. cVEMPs originate from the saccule and are conveyed through the inferior vestibular nerve. oVEMPs arise from the utricle and are transmitted via the superior vestibular nerve. In the central nervous system, cVEMPs are mediated by the vestibular nuclei and run through the uncrossed medial vestibulospinal tract (MVT) in the lower brainstem and spinal cord, descending to the motor neurons of the SCM muscle, which eventually completes a vestibulocollic reflex (VCR). In contrast, oVEMPs are mediated by the vestibular nuclei afferent pathway primarily in the ascending medial longitudinal fasciculus (MLF), projecting contralaterally to the oculomotor nuclei and completing the vestibulo-ocular reflex (VOR) [8-10]. oVEMPs also ascend through other otolithic pathways, including the central ventral tegmental tract, the ascending tract of Deiters, and the ipsilateral vestibulo-thalamic tract, extending from the vestibular nuclei to the upper brainstem and thalamus [3, 11].

The medullary lesions that affect the descending MVT, or the spinal accessory nucleus, can impair cVEMPs. Similarly, brainstem lesions involving the MLF, the crossed ventral tegmental tract, oculomotor nuclei, or the interstitial nucleus of Cajal can lead to abnormal oVEMPs [9]. A central lesion causing abnormal responses in both cVEMPs and oVEMPs is likely localized to the vestibular nerve root entry zone or the vestibular nuclei [12, 13]. Thus, oVEMPs and cVEMPs provide information about both ascending and descending vestibular pathways in the brainstem, as well as the peripheral vestibular function [14, 15] by assessing the VCR and VOR [3, 11]. These tests can further facilitate understanding of the underlying pathophysiology of migraine, VM, and TTH.

In this study, we analyzed unilaterally and bilaterally absent VEMP responses, prolonged latencies of negative and positive peaks, prolonged interpeak intervals, and asymmetry ratios (ARs) of VEMPs in participants under 60 years of age. Peak amplitudes from both sides were analyzed, with the side showing reduced amplitude considered pathological. Statistical analysis was performed using SPSS 26 software. In addition, we evaluated demographic and clinical characteristics, including age, sex, sleep disorders, anxiety and depression, hypertension, diabetes, abortive treatment usage, and clinical features related to headache, dizziness, and/or vertigo.

To date, few studies have focused on the function of the otolith organs and their associated pathways across the three major primary headaches. This study applied both cVEMP and oVEMP testing to assess subclinical vestibular dysfunction, including peripheral and central vestibular components, in patients with migraine, VM, and TTH.

Materials and Methods

Patients

Between December 2019 and September 2022, a total of 353 participants (aged 7 - 60 years) were enrolled in this prospective study from the outpatient population referred to the Headache and Dizziness Clinic at Weifang People's Hospital. Diagnoses were made according to The International Classification of Headache Disorders, 3rd edition beta version (ICHD-3β) [16] by the Classification Committee of the International Headache Society and Barany's Society. Participants were divided into three groups: 1) 118 patients with migraine (mean age 38.47 ± 13.28 years; range 8.8 - 60 years); 2) 150 patients with VM (mean age 40.77 ± 14.10 years; range 7 - 60 years); and 3) 85 patients with TTH (mean age 47.08 ± 10.94 years; range 13 - 60 years). All diagnoses were confirmed by neurologists, written informed consent was obtained from all participants, and the study was approved by the Human Research Ethics Committees of Weifang Brain Hospital in accordance with the Declaration of Helsinki (approval No. 2021YX059).

Exclusion criteria

Patients were excluded if any of the following conditions were met: age over 60 years; conductive hearing loss in either ear ≥ 30 dB [17]; previous cerebellar or brainstem stroke; severe cardiac, renal, or hepatic diseases; acoustic neuroma or other posterior fossa lesions; history of vestibular neuritis; autoimmune diseases; obstruction of external auditory canal; otitis media; temporal bone surgery; Meniere's disease; or unilateral tympanic membrane perforation [18]. Patients with hypertension, diabetes mellitus, or hyperlipidemia were included only if their blood pressure, glucose, and lipid profiles were controlled within normal ranges through conservative treatments.

Physical examination

All participants underwent a comprehensive ocular, ontological, and neurological examination, which included a medical history check, clinical evaluation, routine blood tests, liver and kidney function tests, thyroid function test, and blood glucose and lipid test, to exclude severe disorders. Brain magnetic resonance imaging (MRI) was performed to exclude neurological conditions such as acute brain ischemia, posterior lesions, cerebrovascular and carotid artery stenosis ≥ 50%. Anxiety and depression were assessed using the Hospital Anxiety and Depression Scale (HADS), while the sleep disturbances were evaluated by the Pittsburgh Sleep Quality Index (PSQI) [19]. Familial history is identified as the presence of a first- or second-degree relative affected by similar conditions [20].

Audio-vestibular workup

Patients with headache and vestibular symptoms underwent electrophysiological testing on headache-free and vertigo-free days. Air-conduction threshold (ACT) testing was conducted at 250, 500, 1,000, 2,000, 4,000, 6,000, and 8,000 Hz, while bone conduction threshold (BCT) testing was conducted at 500, 1,000, 2,000, and 4,000 Hz frequencies (Interacoustics

AC 40 Clinical Audiometer; Assens, Denmark) [21, 22]. Hearing loss is defined as a pure-tone average (PTA) greater than 30 dB hearing level (HL), averaged across 0.5, 1, 2, and 4 kHz in one ear. To confirm the absence of conductive hearing loss, the air-bone gap was required to be less than 10 dB across frequencies 1 to 4 kHz [23].

oVEMP test

For the oVEMP recordings, patients were instructed to fix their gaze approximately 30° upwards in a superomedial direction, as this position has been reported to elicit the largest responses [24, 25]. The recording electrode was placed over the contralateral inferior oblique muscle (centered under the pupil and 3 mm below the eye), with the reference electrode placed under the recording electrode and a ground electrode placed on the forehead. Stimulation was given with 500 Hz, 0.1 click sounds at 95 dB via air-conduction headphones.

The reference latency peaks were defined as the first negative peak at 10 ms (n10) and the subsequent positive peak at 15 ms (p15). The n10 and p15 latency, interpeak latency (ms), peak-to-peak amplitude (mV), and VEMP AR (%) were analyzed. The VEMP AR was calculated by the Jongkees formula: VEMP AR (%) = (Au - Aa)/(Aa + Au) × 100, where Au is the n10-p15 amplitude on the unaffected (healthy) side and Aa is the amplitude on the affected (lesion) side [26]. An oVEMP AR ≥ 35% was considered abnormal based on our established clinical normative data. This threshold is consistent with Colebatch et al, who reported that amplitude ARs were typically < 35% in individuals under 60 years of age [27]. Responses were considered present if the peak-to-peak amplitude exceeded 1.0 mV. If responses were not detected in two consecutive runs of the unrectified trace, the oVEMP was recorded as absent. Parameters including unilaterally or bilaterally absent VEMP responses, prolonged latencies, prolonged interpeak intervals (ms), and bilateral amplitude differences (mV), were analyzed. If the cases with VEMP AR ≥ 35%, the side with the lower response was considered the lesion side.

cVEMP test

For cVEMP testing, participants were seated upright and instructed to rotate their head towards the contralateral shoulder to activate the (SCM muscle. Electromyographic (EMG) activity was recorded from surface electrodes. Specifically, the positive electrode was placed on the upper third of the SCM belly, the reference electrode was placed on the SCM tendon right above the clavicle, and the ground electrode was placed on the forehead. Acoustic stimulation (500 Hz, 0.1 click sounds at 95 dB) was given through the ICS Medical Insert Earphones (ER 3A/5A, 300 ohms; ICS Medical, IL, USA). Participants were instructed to maintain SCM contraction during stimulation and relax between recordings.

EMG activities were recorded ipsilaterally from the SCM. The positive/negative polarity of the waveform consisted of an early positive peak at approximately 13 ms (p13) and a subsequent negative peak at around 23 ms (n23). The responses were considered present if the peak-to-peak amplitude exceeded 5.0 mV, and absent if no response was detected in two consecutive runs of the unrectified trace. The p13 and n23 latencies, interpeak latency (ms), peak-to-peak amplitudes (mV), and VEMP AR were analyzed.

Statistical analyses

All statistical analyses were performed using SPSS Statistics 26 software (SPSS Inc., Chicago, IL). The measurement data were analyzed using one-way analysis of variance (ANOVA) and *post hoc* tests using the least significant difference (LSD). For categorical data, Chi-square test was used; if there was significance, then column proportions were compared using two-sample z-test, with Bonferroni adjustment applied to control for multiple comparisons. A P-value of < 0.05 was considered statistically significant.

Results

Patient demographics

The mean age of TTH patients was significantly higher than patients with migraine and VM ($P < 0.001$), while no significant age difference was found between the migraine and VM patient populations. In terms of sex distribution, the proportion of male patients in TTH was significantly higher compared to VM ($P < 0.01$), and also higher in the migraine group than in VM ($P < 0.05$) (Table 1).

In terms of associated comorbidities, the prevalence of sleep disturbances was significantly higher in TTH patients compared to VM and migraine ($P < 0.001$), and no difference was found between the latter two groups. Similarly, anxiety and depression were most prevalent in TTH ($P < 0.001$), followed by VM, which had significantly higher rates than migraine ($P < 0.05$).

No significant differences were observed in the hypertension and diabetes history among the three groups. However, the use of abortive treatments (analgesic and infusion) was significantly higher in the migraine patient population, followed by VM and then TTH. All pairwise comparisons were statistically significant with $P < 0.001$.

Triggering factors were reported significantly less common in TTH patients than in VM ($P < 0.02$) and migraine ($P < 0.05$), with no significant difference between the latter two groups. No significant differences were observed between migraine and VM patient populations in terms of visual aura. Lastly, a positive familial history of headache was more commonly reported by migraine patients compared to those with TTH ($P < 0.05$).

Symptom profiles and headache attack characteristics

Headache duration was longest in the migraine group (9.21 ± 0.79 years), followed by the VM group (7.14 ± 0.70 years, P <

Table 1. Patient Demographic for the Three Primary Headaches

	Migraine (n = 118)	VM (n = 150)	TTH (n = 85)	P-value		
				1 vs. 2	1 vs. 3	2 vs. 3
Age (years), mean ± SD	38.47 ± 13.28	40.77 ± 14.10	47.08 ± 10.94	P > 0.05	P < 0.001	P < 0.001
Male sex, (%)	37 (31.36%)	31 (20.67%)	34 (40%)	P < 0.05	P > 0.05	P < 0.01
Sleep disorder (%)	44 (37.29%)	63 (42%)	56 (65.88%)	P > 0.05	P < 0.001	P < 0.001
Depression and anxiety (%)	20 (16.95%)	42 (28%)	47 (55.29%)	P < 0.05	P < 0.001	P < 0.001
Hypertension (%)	9 (7.63%)	22 (14.67%)	14 (16.47%)	P > 0.05	P > 0.05	P > 0.05
Diabetes (%)	3 (2.54%)	2 (1.33%)	3 (3.53%)	P > 0.05	P > 0.05	P > 0.05
Abortive treatment (%)	89 (75.42%)	49 (32.67)	7 (8.23%)	P < 0.001	P < 0.001	P < 0.001
Triggers (%)	43 (36.44%)	56 (37.33%)	18 (21.18%)	P > 0.05	P < 0.05	P < 0.02
Visual aura (%)	27 (22.88%)	24 (16%)		P > 0.05		
Familiar history (%)	15 (12.71%)	14 (9.33%)	4 (4.71%)	P > 0.05	P < 0.05	P > 0.05

Continuous variables: mean ± standard deviation (SD); count variables: %. 1: migraine group, 2: VM group, 3: TTH group. TTH: tension-type headache; VM: vestibular migraine.

0.05 vs. migraine) and the TTH group (4.40 ± 0.67 years, P < 0.001 vs. migraine; P < 0.02 vs. VM). Notably, in VM patients, the duration of headache was significantly longer than the duration of dizziness (4.03 ± 0.44 years) (P < 0.01), suggesting a later onset of vestibular involvement relative to headache onset. Transient vertigo was reported at a comparable rate in the VM and TTH groups, suggesting similar patterns of transient vestibular instability. Additionally, 74.12% of TTH patients reported a variety of dizziness and/or transient vertigo episodes (Table 2).

In terms of attack frequency, no significant difference between the migraine group and the VM group was found in the proportion of patients experiencing headache or vertigo more than four times per month or between one and four times per month (P > 0.05). However, infrequent attack (i.e., less than once per month) was significantly less common in the migraine group than the VM group (P < 0.001), suggesting that the headache attack in VM patients may include a higher proportion of individuals with infrequent attacks. During the month of clinic evaluation, 19.49% and 2.67% of migraine and VM patients reported increased headache frequency (P < 0.001).

Pain localization patterns were found to be different among groups. Occipital and neck pain in VM (24%) was significantly higher than in migraine (7.63%, P < 0.001). Simultaneous occipital and frontal pain was reported in 4.67% of VM patients but was absent in migraine group (P < 0.02). Hemicrania pain was more frequently reported in migraine (18.64%) than in VM (4%, P < 0.001).

Absent VEMP responses

Absent oVEMP responses (unilateral and bilateral) were significantly more frequent in VM (45.33%) compared to 28.81% in the migraine group (P < 0.01), with no significant differences between VM and TTH or between migraine and TTH. Specifically, unilateral absent oVEMP responses were also significantly higher in VM than in migraine (P < 0.05). These results indicate a greater likelihood of peripheral utricle dysfunction in VM patients (Table 3).

In contrast, absent cVEMP responses were rare, with no significant differences found among the three groups (P > 0.05). No statistical tests were performed due to the low number of cases. Lastly, no group differences were observed in abnormal oVEMP or cVEMP ARs (i.e., AR ≥ 35%).

VEMP parameters

oVEMP latency, interpeak intervals, and ARs presented no statistically significant differences among the groups. However, cVEMP testing revealed that the right-sided p13 and n23 latencies and interpeak intervals in both migraine and VM were significantly prolonged compared to TTH. Moreover, the right-sided AR cVEMP in VM was marginally higher than migraine (P = 0.05) and significantly higher than TTH (P < 0.05), indicating unilateral peripheral saccule dysfunction in VM. On the left side, interpeak p13-n23 latency of cVEMP was significantly prolonged in the migraine than the TTH group (P < 0.05) with no significant difference between the VM and TTH groups, indicating a possible lower brainstem involvement in migraine. No significant differences were found in cVEMP amplitudes (Table 4).

Discussion

Overview of cVEMP and oVEMP physiology

The vestibular apparatuses are small organs located deep within the temporal bone, making direct assessment challenging and often inaccessible by conventional imaging like MRI. However, recent advancements have made non-invasive assessment of all five vestibular end organs possible. High frequency (approximately 5 Hz) canal function can be evaluated

Table 2. Description of the Vertigo and/or Headache Complaints of the Three Groups

Clinical variables	Migraine (n = 118)	VM (n = 150)	TTH (n = 85)	P-value		
				1 vs. 2	1 vs. 3	2 vs. 3
Disease duration, years, mean ± SD						
Headache	9.21 ± 0.79	7.14 ± 0.70	4.40 ± 0.67	P < 0.05	P < 0.001	P < 0.02
Dizzy/vertigo		4.03 ± 0.44**				
Duration of attack						
0 - 4 h						
Pain	68 (57.63%)	100 (66.67%)		P > 0.05		
Vertigo or dizzy		105 (70%)				
≤ 1 min		33 (22%)	15 (17.65%)			P > 0.05
4 - 24 h						
Pain	36 (30.51%)	39 (26%)		P > 0.05		
Vertigo or dizzy		21 (14%)				
> 24 h						
Pain	36 (30.51%)	11 (7.33%)		P > 0.05		
Vertigo or dizzy		9 (6%)				
TTH						
Unsteady gait			5 (5.88%)			
Continuous pain			68 (80%)			
Continuous dizzy			44 (51.76%)			
TTH accompanied dizzy and/or vertigo			63 (74.12%)			
Attack rate						
> 4 times a month						
Pain	30 (25.42%)	34 (22.67%)		P > 0.05		
Vertigo/dizzy		50 (33.33%)				
1 - 4 times a month						
Pain	38 (32.20%)	41 (27.33%)		P > 0.05		
Vertigo/dizzy		35 (23.33%)				
Less than once month						
Pain	27 (22.88%)	71 (47.33%)		P < 0.001		
Vertigo/dizzy		59 (39.33%)				
Increased frequency						
Pain	23 (19.49%)	4 (2.67%)		P < 0.001		
Vertigo/dizzy		6 (4%)				
Location of pain						
Temple	52 (44.07%)	53 (35.33%)		P > 0.05		
Occiput/neck	9 (7.63%)	36 (24%)		P < 0.001		
Occiput/forehead	0	7 (4.67%)		P < 0.02		
Occiput/temple	9 (7.63%)	8 (5.33%)		P > 0.05		
Temple/forehead	9 (7.63%)	14 (9.33%)		P > 0.05		
Hemicranial	22 (18.64%)	6 (4%)		P < 0.001		
Indefinite	17 (14.41%)	26 (17.33%)		P > 0.05		

**P < 0.01, the duration between headache and vertigo in VM. 1: migraine group, 2: VM group, 3: TTH group. TTH: tension-type headache; VM: vestibular migraine.

Table 3. Absent Vestibular Evoked Myogenic Potential Response, Either Bilaterally or Unilaterally

Group	Migraine (n = 118)	VM (n = 150)	TTH (n = 85)	P-value		
				1 vs. 2	1 vs. 3	2 vs. 3
Absent oVEMP at 95 dB nHL (including bilateral and unilateral)	34 (28.81%)	68 (45.33%)	34 (40.0%)	P < 0.01	P > 0.05	P > 0.05
Bilateral	13 (11.02%)	22 (14.67)	14 (16.47%)	P > 0.05	P > 0.05	P > 05
Unilateral	21 (17.80%)	46 (30.67%)	20 (23.53%)	P < 0.05	P > 0.05	P > 0.05
Absent cVEMP at 95 dB nHL	5 (4.24%)	10 (6.67%)	1 (1.18%)	P > 0.05	P > 0.05	P > 0.05
Bilateral	2	3	1			
Unilateral	3	7	0			
AR oVEMP ≥ 35%	22 (18.64%)	27 (18.0%)	15 (17.65%)	P > 0.05	P > 0.05	P > 0.05
AR cVEMP ≥ 35%	9 (7.63%)	18 (12.0%)	7 (8.24%)	P > 0.05	P > 0.05	P > 0.05

1: migraine group, 2: VM group, 3: TTH group. AR: asymmetry ratio; cVEMP: cervical vestibular evoked myogenic potential; nHL: normal hearing level; oVEMP: ocular vestibular evoked myogenic potential; TTH: tension-type headache; VM: vestibular migraine.

by three-dimensional video head impulse testing (vHIT) [28], low frequency (approximately 0.002 - 0.004 Hz) horizontal canal function by caloric testing [29], and otolith functions by VEMPs, specifically, oVEMPs for the utricle and cVEMPs for the saccule otolith. When combined with audiometry, these tests can produce a characteristic disease profile that enables diagnosis of the underlying vestibular disorder.

The utricle, horizontal canal, and superior canal transmit afferent signals through the superior division of the vestibular nerve and are evaluated by oVEMP. In contrast, the saccule and posterior canal transmits afferents via the inferior division and are assessed by cVEMP. A small subset of the saccular afferents travels via Voit's nerve to the superior division [30].

The cVEMP response to loud acoustic stimulation con-

Table 4. Comparisons of oVEMP and cVEMP Among the Three Groups

Parameters	Migraine (n = 118)	VM (n = 150)	TTH (n = 85)	P-value		
				1 vs. 2	1 vs. 3	2 vs. 3
oVEMP						
AR oVEMP	22.61 ± 17.92	22.08 ± 17.55	19.65 ± 14.30	P > 0.05	P > 0.05	P > 0.05
r-n10 latency	10.88 ± 1.66	10.94 ± 1.44	11.22 ± 1.61	P > 0.05	P > 0.05	P > 0.05
r-p15 latency	14.87 ± 1.68	14.81 ± 1.61	14.94 ± 1.73	P > 0.05	P > 0.05	P > 0.05
r-interpeak	4.00 ± 0.97	3.87 ± 0.99	3.78 ± 0.92	P > 0.05	P > 0.05	P > 0.05
r-amplitude	2.51 ± 2.01	2.22 ± 2.01	2.22 ± 2.08	P > 0.05	P > 0.05	P > 0.05
l-n10 latency	11.01 ± 1.85	10.85 ± 1.62	11.13 ± 1.62	P > 0.05	P > 0.05	P > 0.05
l-p15 latency	14.92 ± 1.76	14.74 ± 1.66	14.94 ± 1.68	P > 0.05	P > 0.05	P > 0.05
l-interpeak	3.95 ± 0.96	3.89 ± 0.90	3.87 ± 0.90	P > 0.05	P > 0.05	P > 0.05
l-amplitude	2.40 ± 2.47	2.09 ± 2.17	1.88 ± 1.74	P > 0.05	P > 0.05	P > 0.05
cVEMP						
AR cVEMP	14.64 ± 13.56	17.83 ± 13.53	14.21 ± 12.04	P = 0.05	P > 0.05	P < 0.05
r-p13 latency	15.27 ± 2.51	14.99 ± 2.98	14.24 ± 2.83	P > 0.05	P = 0.01	P < 0.05
r-n23 latency	20.25 ± 2.90	19.92 ± 3.14	19.01 ± 3.06	P > 0.05	P < 0.005	P < 0.05
r-interpeak	4.98 ± 2.14	4.98 ± 1.73	4.77 ± 1.78	P > 0.05	P < 0.005	P < 0.05
r-amplitude	25.75 ± 28.61	21.65 ± 20.68	20.27 ± 16.30	P > 0.05	P > 0.05	P > 0.05
l-p13 latency	15.32 ± 2.64	15.07 ± 2.62	15.34 ± 2.81	P > 0.05	P > 0.05	P > 0.05
l-n23 latency	20.40 ± 2.90	19.95 ± 2.95	19.91 ± 3.05	P > 0.05	P > 0.05	P > 0.05
l-interpeak	5.08 ± 1.95	4.88 ± 1.79	4.56 ± 1.63	P > 0.05	P < 0.05	P > 0.05
l-amplitude	28.35 ± 40.22	21.76 ± 19.94	20.90 ± 17.74	P > 0.05	P > 0.05	P > 0.05

1: migraine group, 2: VM group, 3: TTH group. latency, ms; AR VEMP, %; amplitude, mV. AR: asymmetry ratio; cVEMP: cervical vestibular evoked myogenic potential; l: left; oVEMP: ocular vestibular evoked myogenic potential; r: right; TTH: tension-type headache; VM: vestibular migraine.

sists of a biphasic waveform, characterized by an early positive peak (p13) followed by a negative peak (n23), recorded from the ipsilaterally SCM muscle. This reflex represents a brief inhibition of the SCM muscle and originates from activation of the saccule via the inferior vestibular nerve [6]. In contrast, oVEMP responses are characterized by an initial negative peak (n10) followed by a positive peak (p15), recorded from the contralateral inferior oblique muscle [31]. oVEMPs reflect excitatory activation of the utricle and represent a crossed VOR pathway [3-5]. The short latencies observed in both cVEMP and oVEMP suggest involvement of the oligosynaptic pathway [27].

The otolith organ consists of two receptors: the saccular and utricular maculae. The saccule is primarily sensitive to vertical acceleration, while the utricle responds to horizontal acceleration [32]. Utricular stimulation can result from either lateral linear acceleration or tilt due to gravity. Otolith-ocular reflexes, driven by utricular activation, may induce either tilt reflexes or a translational linear VOR (LVOR) in the horizontal plane [31].

Both utricular and saccular afferents project to neurons in the vestibular nuclei of the brainstem [33, 34]. Utricular signals ascend primarily via the contralateral MLF in the upper medulla and pons, and are also transmitted through other ascending otolithic pathways, including the central ventral tegmental tract, the ascending tract of Deiters, and the ipsilateral vestibulo-thalamic [3, 11]. On the other hand, saccular signals predominantly descend via the ipsilateral MVT.

Although there are overlaps among the otolithic projections, saccular afferents primarily target the lateral, particularly the spinal (inferior) and superior vestibular nuclei. Utricular afferents project toward the medial, the superior vestibular nucleus, and the rostral portion of the spinal vestibular nucleus [27]. Vestibular afferents ultimately terminate either in the vestibular nuclei or the vestibulocerebellum [35]. Functionally, the saccular projection plays a role in the VCR mediated by activating the inferior vestibular nerve, which projects through the medial and lateral vestibulospinal tracts (VSTs) to the motor neurons of the accessory spinal nuclei, reaching the neck muscles. Additional projections to the spinal cord also travel through the reticulospinal pathways [36]. In contrast, utricular signals contribute to the VOR via the superior vestibular nerve, ascending through the contralateral MLF and other dorsomedial brainstem pathways to reach the ocular motor nuclei and efferent to the extraocular muscles [37].

In monkeys, saccular signals project primarily to the cerebellar uvula, with a less dense projection to the nodulus, whereas utricular signals project strongly to the nodulus and weakly to the flocculus, paraflocculus, and uvula [33]. Cortical representations of otolith stimulation involve a multisensory network within the posterior insular cortex, the middle and superior temporal gyri, and the inferior parietal cortex in both hemispheres [38].

Demographic and psychiatric comorbidities

In this study, the mean age of TTH patients (mean 47.08 years) was significantly higher than that of migraine patients (mean 38.47 years) or VM patients (mean 40.77 years). The sex distribution also differed significantly among the groups, with a higher proportion of male patients in the TTH group, followed by migraine, and lowest in VM. No significant differences were observed in patients' hypertension and diabetes history. Age is known to affect cVEMP responses, particularly by reducing the amplitude across stimulus frequencies [39]. For high-frequency stimuli such as air- or bone-conducted 500 Hz tones, response amplitudes decrease by approximately 12% per decade after the age of 20, whereas responses to low-frequency stimuli (< 100 Hz) are less affected [6]. Generally, in individuals under 60, age-related effects on VEMP amplitudes and latencies are less pronounced [38]. Thus, to minimize potential confounding, participants older than 60 years old were excluded. Notably, while aging can lead to absent responses in older adults, it does not significantly affect amplitude symmetry between sides [40, 41].

The prevalence of sleep disorders in TTH was significantly higher than VM and migraine. Similarly, comorbid anxiety and depression were most common in TTH, followed by VM, and least prevalent in migraine. The use of abortive treatment was highest in the migraine group, followed by VM and TTH. This pattern may reflect differences in perceived symptom severity and treatment-seeking behavior across headache subtypes.

Vestibular symptoms can occur during headache attacks, but are often experienced during headache-free intervals, lasting from minutes to days [42]. These episodes may arise spontaneously or be positionally triggered, which can exhibit features of both central and peripheral vestibular dysfunctions [1, 43].

Although TTH is typically associated with milder pain intensity, patients with TTH reported a lower quality of life because of the higher burden of psychotic comorbidities. Vertigo and postural instability could contribute to heightened anxiety and fear, potentially explaining the greater prevalence of anxiety and depression observed in TTH than VM and migraine [44]. The overall patterns of psychotic comorbidity followed the sequence: TTH > VM > migraine.

Clinical features and vertigo profiles

In this study, the duration of headache symptoms was significantly longer in the migraine group (9.21 years) than in the VM (7.1 4years) and TTH (4.40 years) groups. This pattern presents consistency to the trend in symptom onset, with migraine patients presenting at younger ages than those with VM or TTH. In VM patients, the mean headache history of 7.14 years was significantly longer than the dizziness/vertigo history of 4.03 years, indicating a temporal shift from headache to vestibular symptoms over time.

Regarding headache frequency, 47.33% of VM patients reported headache attack frequency below once per month, which was significantly higher compared to 22.88% from the migraine group. During the month of clinical evaluation, only 2.67% of VM patients reported an increase in headache attack frequency, significantly lower than the 19.49% reported by mi-

graine patients. These results indicate a higher proportion of infrequent and stable headache presentations in the VM population. Transient vertigo was reported in 22% of VM patients and 17.65% of TTH patients. In the TTH group, 5.88% reported gait instability, 51.76% experienced continuous dizziness, and 74.12% reported either variable dizziness or vertigo symptoms. Although the peripheral otolith system appeared to be less affected in TTH than in VM or migraine, patients with TTH experienced notable disequilibrium, which may be caused by peripheral canal lesions [45] and proprioceptive dysfunctions [46, 47].

Occipital and/or neck pain was significantly more prevalent in VM than in migraine. Additionally, simultaneous occipital and frontal pain was reported in 4.67% of VM patients but was not observed in the migraine group. These findings suggest that while migraines involve trigeminovascular pathways primarily, VM might have both trigeminovascular and cervical neurovascular mechanisms involved, consistent with prior studies [48]. Hemicranial pain was reported in 18.64% of migraine patients, significantly higher than in VM patients (4%, $P < 0.001$), which is well aligned with the well-established pattern of lateralized pain and visual aura in migraine [49].

Interpretation of VEMP results

Brainstem auditory evoked potential (BAEP) responses have shown abnormalities in absolute or interpeak latencies in migraine without auras (MoA) and VM, suggesting a potential early involvement of auditory nerve or brainstem dysfunctions [50, 51]. Despite the increasing number of studies on VEMP responses in VM patients in recent years, the findings remain inconsistent. Particularly, there are limited studies on the subclinical abnormality of peripheral otolith organs and their afferent and descending pathways in migraine, VM, and TTH.

Previous studies on VEMPs in migraine and VM have shown different results. Rahsan et al reported reduced cVEMP response rates in migraine patients with MoA, while no statistically significant differences were found in cVEMP latencies or interpeak intervals, nor oVEMP response rate across the MoA, VM, and healthy control groups [52]. Similarly, Kandemir et al observed no significant differences in cVEMP latency, amplitude, or asymmetry among patients with VM, MoA, and TTH when compared to normal controls [53]. In contrast, Zaleski et al reported a significantly higher proportion of bilaterally absent oVEMPs responses in VM than in the healthy controls. Additionally, oVEMP response amplitudes were significantly reduced and often asymmetric in the VM group [54]. Other studies have supported the presence of central [55] and peripheral [56-58] vestibular dysfunction in VM patients, noting that abnormal oVEMPs responses may reflect increased vulnerability within the ascending utricular ocular pathway [59, 60]. These inconsistences suggest that migraine-related mechanisms may act on the vestibular system at multiple levels, including both peripheral and central pathways [61]. Makowiec et al observed normal cVEMP and abnormal oVEMP responses in VM patients, suggesting that this VEMP pattern could serve as a biomarker for VM [49]. Taylor et al, however, reported similar cVEMP latencies and amplitudes in VM patients and healthy controls, concluding that VM may involve more central mechanisms rather than peripheral vestibular impairment [62]. Boldingh et al reported uni- or bilaterally absent cVEMPs in 44% of VM patients and 25% of their migraine patients, compared to 3% of the healthy controls [63]. Hong et al stated that although no significant cVEMP asymmetry was present, 60% of the VM patients had bilaterally absent cVEMP responses [64]. Other studies have also reported normal latency cVEMPs with reduced amplitude in migraine patients [65, 66], and Yetiser et al also recorded with a unilaterally reduced amplitude of P13 in migraine patients with normal cVEMP latency [67].

In our study, the overall absent oVEMP response rate in VM patients (45.33%) was significantly higher than that in migraine (28.81%). Specifically, the unilateral absent was 30.67% in VM, significantly exceeding the rate in the migraine group, indicating a peripheral utricle involvement in VM. However, no significant differences were found among groups regarding the absolute latencies of n10, p15, or the n10-p15 interpeak interval, suggesting that the utricular afferent pathways within the brainstem, particularly the MLF, were likely unaffected.

Absent cVEMP responses were similarly rare across all groups, which were only observed in 4.24% of the migraine patients, 6.67% of the VM patients, and 1.18% of the TTH patients. However, the cVEMP AR was significantly higher in VM compared to TTH and marginally higher in migraine patients. This suggests unilateral saccule dysfunction in VM.

Our study showed that both migraine and VM patients exhibited greater right-sided prolongation in cVEMP parameters, including p13 latency, n23 latency, and interpeak intervals compared to TTH patients. Additionally, left-sided interpeak latency of cVEMP was prolonged in the migraine group relative to the TTH group, but not in VM patients. These results suggest greater involvement of the brainstem (mainly involving vestibular-spinal tract) in migraine and VM, with a severity gradient of migraine > VM > TTH. The reason for this apparent right-sided predominance remains unclear and requires further investigation. The precise mechanism underlying cochleovestibular (end-organ) involvement in VM remains unclear. However, Vass et al proposed that trigeminovascular modulation of inner ear blood flow, a known phenomenon in migraine, may contribute to vestibular dysfunction in VM [68]. In contrast, oVEMP latency did not differ significantly among the three groups, indicating no evidence of upper medulla and pons involvement in the utricular afferent pathway. Our observations on the cVEMP and oVEMP asymmetries provide additional evidence to the concept that migraine and VM are lateralized disorders, which potentially rooted in developmental lateralization [69].

Similar VEMP abnormalities have been observed in patients with multiple sclerosis and brainstem infarctions [70], reinforcing the diagnostic value of cVEMPs for lower brainstem involvement and oVEMPs for upper brainstem lesions [3, 71-76].

In patients with lateral medullary infarction (LMI), cVEMP abnormalities on the ipsilesional side typically involve the descending vestibule-spinal track, which often present as prolonged latencies. Topographical studies suggest that caloric paresis tends to reflect more rostrally located infarctions, while

absent or delayed cVEMPs are more commonly associated with caudally located ones [77]. Additionally, contralesional or bilateral cVEMP abnormalities in unilateral LMI may result from the disruption of commissural modulation between vestibular nuclei [76]. Recently, isolated vestibular nuclear infarctions showed decreased or absent of both cervical and ocular VEMP responses during ipsilesional stimulation [13].

To summarize, VM patients demonstrated signs of peripheral otolith dysfunction. This was reflected by a higher rate of absent oVEMP responses, especially unilateral, and greater cVEMP amplitude asymmetry compared to migraine patients and TTH patients. Both migraine and VM groups presented evidence of low brainstem involvement proved by prolonged right-sided p13, n23, and interpeak latencies in cVEMP, and prolonged left-sided p13-n23 interpeak latency compared to TTH patients. These findings suggest subclinical involvement of the VST, potentially mediated by trigeminovascular or cervical neurovascular mechanisms.

Asymmetry in the oVEMP responses was observed in patients with VM, which supports a growing body of evidence suggesting that the migraine brain functions asymmetrically [78]. Anatomical studies have demonstrated reciprocal connections between the vestibular nuclei and the trigeminal nucleus caudalis (TNC) [79]. Neurons in the TNC participate in several peripheral mechanisms of migraine, including neurogenic inflammation, endothelium-mediated vasodilation, and peripheral sensitization [80].

It is plausible that migraine-related processes impact the utricle, saccule, vestibular nerve, or central otolith pathways. The distinct clinical and electrophysiological profiles observed between migraine and VM suggest that underlying pathophysiological differences exist. Further research will be needed to clarify how different systems are involved and contribute to the clinical manifestation.

Conclusions

Psychic comorbidities such as anxiety and depression showed a graded pattern across headache types, with the highest prevalence observed in TTH, followed by VM, and the lowest in migraine. Both VM and TTH patients reported transient vertigo episodes, which may increase the risk of imbalance and drop attacks. In VM patients, vestibular symptoms tended to gradually replace headache symptoms over time. Lastly, occipital and/or neck pain was more frequently reported in VM than in migraine, suggesting a greater involvement of cervical pathways in VM pathophysiology.

The high rate of absent oVEMP responses and elevated cVEMP ARs in VM patients indicated peripheral utricle and saccule dysfunctions. In contrast, no significant group differences were observed in oVEMP latencies, suggesting that upper medulla and pons brainstem were likely spared. However, both migraine and VM patients demonstrated prolonged right-sided p13, n23, and interpeak latencies in cVEMP, as well as prolonged left-sided interpeak latencies. These results indicated subclinical involvement of the lower brainstem, primarily the VST. Notably, the right-sided abnormalities were more prominent.

The presence of unilateral VEMP abnormalities and predominant right-sided lower brainstem involvement in both migraine and VM patients supports the notion of lateralized dysfunction within the vestibular system. These findings align with broader clinical and electrophysiological evidence suggesting asymmetric neural processing, possibly mediated through trigeminovascular and cervico-vascular pathways.

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Conflict of Interest

The authors declare no conflict of interest.

Informed Consent

Written informed consents were obtained from participants.

Author Contributions

AJZ: study design, cases collection, literature retrieval, manuscript writing, and final revision; LQY: vestibular function test; AYZ: literature retrieval and cardiovascular assessment; XZC: statistical analysis; LA and YL: cases collection.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

Abbreviations

AC: air-conducted; ACT: air-conduction threshold; AR VEMP: asymmetry ratio of VEMP; BCT: bone conduction threshold; BAEP: brainstem auditory evoked potential; cVEMP: cervical vestibular evoked potential; EMG: electromyographic; HADS: Hospital Anxiety and Depression Scale; ICHD-3β: International Classification of Headache Disorders, 3rd edition beta version; LMI: lateral medullary infarction; LVOR: linear vestibulo-ocular reflex; MoA: migraine without auras; MVT: medial vestibulospinal tract; MLF: medial longitudinal

fasciculus; MRI: magnetic resonance imaging; oVEMP: ocular vestibular evoked potential; PSQI: Pittsburgh Sleep Quality Index; PTA: pure-tone average audiometric threshold; SCM: sternocleidomastoid; TNC: trigeminal nucleus caudalis; TTH: tension-type headache; VM: vestibular migraine; vHIT: video head impulse test; VCR: vestibulocollic reflex; VOR: vestibulo-ocular reflex; VST: vestibulospinal tract

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