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ORIGINAL ARTICLE

Vestibular evoked myogenic and auditory brainstem evoked potentials in a female migraine population

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Background and purpose – The purpose of the present study was to evaluate ocular vestibular evoked myogenic potential (oVEMP), cervical vestibular evoked myogenic potential (cVEMP), and brainstem auditory evoked potential (BAEP) response characteristics and to understand the pathophysiology of vestibular dysfunction in female migraineurs with vertigo symptoms. We also aimed to assess the electrophysiological diagnostic significance of the VEMP responses in vestibular migraine (VM).
Methods – 23 patients with migraine without aura (MoA), 23 patients with VM, and 20 sex-and age-matched healthy controls, a total of 66 female participants were enrolled in this study. The outcome parameters were asymmetry ratios (ARs), amplitudes of oVEMP, cVEMP, N1P1, P13N23, and the respective latencies (mean ± SD). From the BAEP graphs, absolute and interpeak interval latencies of waves were analyzed.
Results – 30.4% of the MoA group and 21.7% of the VM group had uni- or bilaterally absent cVEMP responses which were statistically significant only in the MoA group (p=0.035) in comparison to control group. Both groups displayed statistically insignificant absent or asymmetrical responses for oVEMP (13.1%). Cervical VEMP P13 and N23 latency, peak-to-peak amplitude, interaural latencies, and amplitude ARs did not show any significant difference between MoA and VM patients and healthy controls. No significant difference was detected among the three groups in the oVEMP and BAEP parameters.
Conclusion – Although absent cVEMP responses were more common in MoA and VM patients than in healthy individuals, the

Vestibularisan kiváltott myogen és auditív agytörzsi kiváltott potenciálok női migrénes populációban
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Háttér és cél – A jelen vizsgálat célja az volt, hogy értékelje az ocularis vestibularis kiváltott myogen potenciál (oVEMP), a nyaki vestibularis kiváltott myogen potenciál (cVEMP) és az agytörzsi auditív kiváltott potenciál (BAEP) válasz jellemzőit, valamint hogy segítsen megérteni a vestibularis diszfunkció patofiziológiáját a szédüléses tünetekkel járó migrénben szenvedő nőknél. Célunk volt továbbá, hogy értékeljük a VEMP-válaszok elektrofiziológiai diagnosztikai jelentőségét a vestibularis migrénben (VM).
Módszerek – A vizsgálatba 23 aura nélküli migrénes (MoA) beteget, 23 VM-es beteget és 20, nemben és életkorban illesztett egészséges kontrollt, összesen 66 női résztvevőt vontunk be. A kimeneti paraméterek az aszimmetriaarányok (AR), az oVEMP, cVEMP, N1P1, P13N23 amplitúdói és a megfelelő latenciák (átlag ± SD) voltak. A BAEP-gráfokból a hullámok abszolút és csúcsok közötti intervallumlatenciáit elemeztük.
Eredmények – A MoA-csoport 30,4%-ának és a VM-csoport 21,7%-ának egy- vagy kétoldali cVEMP-válaszai hiányoztak, ami statisztikailag csak a MoA-csoportban volt szignifikáns (p = 0,035) a kontrollcsoporthoz képest. Mindkét csoport statisztikailag nem szignifikáns hiányzó vagy aszimmetrikus oVEMP-válaszokat mutatott (13,1%). A nyaki VEMP P13 és N23 latencia, a csúcs-csúcs amplitúdó, az interaurális latenciák és amplitúdó-AR-ek nem mutattak szignifikáns különbséget a MoA- és a VM-betegek, valamint az egészséges kontrollok között. Az oVEMP- és a BAEP-paraméterek tekintetében nem volt szignifikáns különbség a három csoport között.
Következtetés – Bár a hiányzó cVEMP-válaszok gyakoribbak voltak a MoA- és a

VEMP and BAEP test results should not be used in the differential diagnosis of VM and MoA.

VM-betegeknél, mint az egészségeseknél, a VEMP és BAEP vizsgálati eredményeket nem szabad felhasználni a VM és a MoA differenciáldiagnózisában.

Keywords: migraine, vestibular migraine, vestibular evoked myogenic potential, vertigo, migraine without aura

Kulcsszavak: migrén, vestibularis migrén, vestibularis kiváltott myogen potenciál, vertigo, aura nélküli migrén

Migraine without aura (MoA) is a primary headache disorder characterized by unilateral, recurrent, and pulsatile headaches associated with nausea, vomiting, and phono-photophobia without aura[1]. Patient with migraine frequently has vestibular complaints, such as dizziness, unsteadiness, or head motion intolerance[2, 3]. Many studies have identified subclinical vestibular dysfunction in migraineurs who do not complain of vestibular symptoms[4].

Vestibular migraine (VM) is a clinically common disease that presents recurrent dizziness/vertigo, with or without headache. VM is one of the most common causes of episodic vertigo in adults, with a lifetime prevalence of 1%[5, 6].

The pathophysiology of VM remains unclear. Altered neural activity in the trigeminal vascular system (TVS) is one of the initial mechanisms underlying migraine. Calcitonin gene-related peptide (CGRP) and substance P are neuropeptides expressed in the TVS. These neuropeptides cause vasodilation and inflammation, which exposes the throbbing pain of migraine. It has also been reported that some neuropeptides, such as CGRP and serotonin, may be involved in the VM pathway. This pathway starts from the TVS, goes to the brainstem and vestibular nuclei, and connects the contralateral thalamus and cortical pain-related areas. Besides this pathway, nociceptive centers of the brain are also associated with pain centers and vestibular nuclei[7, 8]. Part of the pathway that modulates neuronal hyperexcitability remains obscure. While some studies have found a higher incidence of central vestibular dysfunction in patients with VM[9], others have reported a higher incidence of peripheral vestibular dysfunction[10–13].

Cervical and ocular vestibular evoked myogenic potentials (cVEMP/oVEMP) have been widely used to analyze vestibular dysfunction in patients with MoA and VM. These are short-latency, vestibular-dependent reflexes recorded from the sternocleidomastoid (SCM) and the inferior oblique (IO) extraocular muscles. Electromyographic responses derived from vestibular labyrinths can be evoked by sound delivered through headphones, vibration applied to the skull, or electrical stimulation. It has been reported that they reflect otolith function rather than semicircular canals[14-16].

The cVEMP, representing the vestibulo-collic reflex, originates from the saccule. It is transmitted to the inferior vestibular nerve and descends via the vestibulospinal tract through the lower brainstem to the motor neurons of the SCM muscle[17, 18]. oVEMP, a manifestation of the vestibulo-ocular pathway, appears to be mainly utricular in origin. It is transmitted to the superior vestibular nerve and ascends via the medial longitudinal fasciculus through the upper brainstem to the oculomotor nuclei[17-19].

Since oVEMP and cVEMP provide information about both ascending and descending vestibular pathways in the brainstem, combined VEMP measures have been studied in several peripheral and central vestibular disorders[20, 21]. Delayed reflex latencies have been attributed to central pathology, whereas the absence of responses and reduced amplitudes have been accepted to localize peripheral causes[22, 23]. VEMP findings in the literature with regard to MoA and VM do not appear to be homogenous.

Auditory symptoms are generally considered to be less common than vestibular symptoms in migraine[24]. Specific auditory symptoms such as phonophobia and hearing loss and tinnitus suggest impairment of auditory pathways in migraine cases[25]. Brainstem auditory evoked potential (BAEP) is an important neurophysiological method for evaluating peripheral and central nerve functions from the cochlea to the brainstem[26]. BAEP responses were reported to have some abnormalities in the form of absolute or interpeak latencies or both in MoA and VM patients, thereby demonstrating that these abnormalities might be the earliest indicator of the auditory nerve and/or brainstem dysfunctions[24, 27].

The present study aimed to analyze the auditory and vestibular profile differences of patients with VM and MoA through BAEP and VEMP testing and to help understand the pathophysiology of vestibular dysfunction. We also aimed to assess the electrophysiological diagnostic significance of the VEMP responses.

Methods

Participants

Between May 2020 and August 2020, 66 female participants (aged 20-56 years) were enrolled in this prospective, controlled study. The subjects were divided into 3 groups: The first group consisted of 23 female VM patients (mean age 40.15±10.47 years; range, 20– 60 years) based on the criteria of the International Classification of Headache Disorders, 3rd edition (ICHD-3)[28] and the International Classification of Vestibular Disorders (ICVD) of the Barany Society[29].

The second group consisted of 23 female MoA patients (mean age 41.56 ± 7.84 years; range, 29– 54 years) based on the criteria of the ICHD-3. The third group, the control group, consisted of 20 healthy female subjects age-matched to the patients' group (mean age 38.85 ± 9.89 years; range, 25-65 years). Patients with headache and vestibular symptoms underwent electrophysiological tests on headache-free and vertigo-free days.

All participants underwent a thorough neurological workup, that is, history taking, clinical examination, and a basic audiological evaluation, including pure tone audiometry (250-8000 Hz) (Interacoustics AC 40 Clinical Audiometer; Assens, Denmark) to rule out any hearing loss, and brain magnetic resonance imaging (MRI) performed to exclude other neurological problems.

The exclusion criteria included the history of prolonged noise exposure, ototoxic medication, ear discharge, otosclerosis, head or ear trauma, diabetes mellitus, and hypertension or ischemic heart disease. The control group showed no neurological or vestibular symptoms.

The study protocol was approved by the local ethics committee (reference number 2020/514/177/34; approval date May 13th, 2020). Informed consent was obtained from all participants.

Audio-vestibular workup

BAEP and VEMP recordings were performed using an EMG/EP measuring machine (MEB-2300K, Nihon Kohden, Tokyo, Japan) while the subjects were seated, in a dim and quiet environment.

BAEP recordings were performed using a montage consisting of Cz-ipsilateral mastoid (M1) and Cz-contralateral mastoid (M2) derivations. Auditory stimuli presented to each ear separately via earphones were clicks with a duration of 0.1 ms, a frequency of 10 Hz, and an intensity of 60 dB higher than the hearing threshold initially established for each subject. The responses were analyzed with a 100–3000 Hz bandpass filter and a sweep time of 10 ms. Two hundred responses were averaged in each run and two runs were performed for each ear. Absolute latencies of waves I, III, and V and interpeak latencies (IPL) of waves I–III, III–V, and I–V were noted.

VEMPs were recorded following stimulation with a 500Hz tone burst (1ms rise/fall time, 2 ms plateau) presented through headphones (air-conducted-AC-sound) at an intensity of 95 dB NHL and a stimulus presentation repetition rate of 5 Hz. The electrode impedances were less than 5 kΩ.

cVEMP test

The active electrode was placed on the upper one-third of the SCM muscle, ipsilateral to the sound stimulation, with the reference electrode over the sternum and the ground electrode on the forehead. Patients were tested in a seated position. While the subjects turned their heads to the counter-lateral side to contract the SCM, the responses were recorded from the ipsilateral SCM. A total of 200 sweeps were averaged. Myogenic signals were amplified and band-pass-filtered at 20-2000 Hz. The procedure was repeated twice on both sides.

The results of VEMP were evaluated by the existence of the initial successive positive and negative polarities termed P13 and N23 based on their respective latencies. If they were not detected in two consecutive runs of the unrectified trace at 95 dB stimulation, the result was accepted as the absence of VEMP. In the unrectified trace, interpeak (P13-N23) latency and amplitude, and after rectification, the absolute peak latencies and amplitudes of P13 and N23 were measured. Intersite differences in P13 and N23 latencies were calculated. Interaural P13-N23 amplitude asymmetry ratio (AAR) was calculated as follows: (larger response - smaller response) / (larger response + smaller response) × 100. Greater than 30 % asymmetry was accepted as abnormal. VEMP parameters were compared among the three groups.

oVEMP test

The active electrode was placed ~1 cm below the center of the inferior eyelid contralateral to the sound stimulation, with the reference electrode located 2 cm below the active electrode and ground electrode on the forehead. Patients were tested in a seated position. During the recording, the participants were asked to keep their heads at a midline position and look upward to a fixed point of 30° above the horizontal line.

Myogenic signals were amplified and band pass-filtered between 30 Hz and 3000 Hz. 200 stimuli were applied to each ear twice.

The unrectified signals from 200 trials were averaged from the oVEMP traces. The first negative and positive responses were designated as the N10 and P15 waves, respectively. In the unrectified trace, the interpeak (N1-P1) latency and amplitude, and after rectification, the absolute

Table 1. *Comparison of BAEP results between left and right ears in the three groups*

	MoA group			VM group			Control group				
	Left	Right	Pa value*	Left	Right	Pb value*	Left	Right	Pc value*	Pd value**	Pe value**
PL I	1.34 ± 0.12	1.37 ± 0.13	0.851	1.21 ± 0.14	1.17 ± 015	0.889	1.73 ± 0.11	1.66 ± 0.11	0.645	0.331	0.811
PL III	3.58 ± 0.14	3.65 ± 0.16	0.668	3.35 ± 0.21	3.35 ± 0.23	0.568	3.62 ± 0.17	3.72 ± 0.15	0.335	0.167	0.220
PL V	5.26 ± 0.14	5.39 ± 0.19	0.309	5.15 ± 0.27	5.25 ± 0.31	0.354	5.21 ± 0.22	5.21 ± 0.21	0.565	0.472	0.110
IPL I-III	2.14 ± 0.35	2.10 ± 0.26	0.578	2.18 ± 0.31	2.12 ± 0.32	0.484	2.07 ± 0.19	2.08 ± 0.30	0.840	0.184	0.430
IPL III-V	1.91 ± 0.32	1.86 ± 0.36	0.879	1.83 ± 0.28	1.80 ± 0.28	0.891	1.83 ± 0.26	1.67 ± 0.30	0.088	0.370	0.188
IPL I-V	4.05 ± 0.33	3.97 ± 0.47	0.862	4.03 ± 0.41	3.92 ± 0.25	0.260	3.89 ± 0.30	3.75 ± 0.40	0.064	0.159	0.123

p value* Dependent samples Wilcoxon signed- rank test; p value** Independent samples Kruskall-Wallis Test.
MoA: migraine without aura, VM: vestibular migraine, Pa: comparison between left and right ears in the MoA group, Pb: comparison between left and right ears in the VM group, Pc: comparison between left and right ears in the control group, Pd, comparison of left ears among the groups, Pe: comparison of right ears among the groups

peak latencies of N1 and P1 and the amplitude of N1 were measured. Intersite differences in the latencies were also calculated. The interaural N1 AAR was calculated using the same method that was mentioned for cVEMP; greater than 30% asymmetry was accepted as abnormal. VEMP parameters were compared among the three groups.

Statistical analysis

Data were entered into Excel and analyzed with SPSS 15.0 for Windows (SPSS Inc., Chicago, IL, USA). Numbers (n) and percentages (%) were used to describe categorical data. Continuous data are expressed as mean ± standard deviation (SD) values. Three groups were compared in the analysis: VM, MoA, and control. Wilcoxon signed-rank and Kruskal Wallis tests were used to compare group differences in the VEMP and BAEP variables. Of note, the absence of a VEMP response was assigned an amplitude of zero microvolts, and latency was considered as missing data. The associations of VEMP response rates with both MoA and VM were compared separately with the those of control group using Fisher's exact test. Statistical significance was set up at $p<0.05$.

Results

Demographics

All participants were female to avoid a gender bias. There was no statistical difference in age between the study and control groups. The duration of the disease was 13.7±9.05 years for the MoA group and 10.43±6.85 years for the VM group; there was no statistical difference in disease duration between MoA and VM groups. None of the groups had any patients with sensorineural or conductive hearing loss in the pure tone audiometry test.

BAEP results

There were no statistically significant differences in any groups between the right and left peak latency differences of waves I, III, V, and IPL I-III, III-V, and I-V waves **(Table 1)**. In addition, there were no significant differences between the groups in terms of right and left sides.

VEMP results

cVEMP findings

Seven patients in the MoA group (4, 17.4% unilateral; 3, 13% bilateral) demonstrated absent cVEMP responses, while responses could not be obtained for five patients in the VM group (2, 8.7% unilateral; 3, 13% bilateral) and one in the control group (1, 5% unilateral; 0.0% bilateral). A statistically significant low response rate was observed in the MoA group (Fisher' exact test, p=0.035) **(Table 2)**.

No statistically significant differences were observed for any of the peak latencies, interpeak intervals, and interaural latency differences between the patient and control groups. In addition, P13-N23 interpeak and rectified P13 and N23 amplitudes of cVEMP in paitents VM and

Table 2. *VEMP responserates in patients and healthy controls*

	VM (n:23)	MoA (n:23)	Controls (n:20)
cVEMP			
Bilateralresponse, n (%)	18 (78.3%)	16 (69.16%)	19(95%)
Unilateralresponse, n (%)	2 (8.7%)	4 (17.4%)	1(5%)
No response, n (%)	3 (13%)	3 (13%)	0 (0%)
oVEMP			
Bilateralresponse, n (%)	20 (86.9%)	20 (86.9%)	19 (95%)
Unilateralresponse, n (%)	2 (8.6%)	2 (8.6%)	1 (5%)
No response, n (%)	1 (4.5%)	1 (4.5%)	0 (0%)

VM: vestibular migraine, MoA: migraine without aura, cVEMP: cervical vestibular evoked myogenic potential, oVEMP: ocular vestibularevoked myogenic potential

MoA did not differ significantly from those of healthy controls. Moreover, the amplitude ARs did not differ between the groups (p> 0.05) **(Table 3)**.

oVEMP findings

Three patients in the VM group (2, 8.6% unilateral; 1, 4.5% bilateral), three patients in the MoA group (2, 8.6% unilateral; 1, 4.5% bilateral) and one in the control group (1, 5% unilateral; 0, 0% bilateral) showed no oVEMP responses. There was no statistically significant difference between the groups in terms of oVEMP response rate **(Table 2)**.

In oVEMP, there were no significant differences in the peak, interpeak, and interaural latencies among the groups. In addition, no statistically significant difference was found when comparing the amplitude of all waveforms and ARs between patients and healthy controls (p> 0.05) **(Table 4)**.

Discussion

In the present study, we found no significant differences in the VEMP and BAEP parameters between the patient and control groups. However, low cVEMP response rates were observed in the MoA group.

Despite the increasing amount of published data on VM-related VEMP studies in recent years, the findings appear to be contradictory, and migraine-related published data are limited as well. While some studies have found a higher incidence of central vestibular dysfunction in VM patients[9, 30], others have reported peripheral

Table 3. *cVEMP results of patients and healthy controls*

Parameters	VM	MoA	Controls	p*
Leftside				
Latency P13 (ms)	14.33±2.75	14.45±2.45	13.93±1.81	0.960
Latency N23 (ms)	20.61±3.00	30.8+-2.80	20.65±2.40	0.396
P13-N23 interpeak latency (ms)	4.21±1.19	3.84±1.06	4.09±1.56	0.613
P13-N23 amplitude (μV)	10.713±7.12	13.34±10.10	17.13±16.39	0.313
P13rectified amplitude (μV)	4.64±3.64	5.42±5.65	7.57±11.81	0.880
N23 rectified amplitude (μV)	4.22±3.16	4.41.±4.12	5.54±4.49	0.628
Right side				
Latency P13 (ms)	14.46±2.41	14.16±2.73	14.23±2.32	0.562
Latency N23 (ms)	20.38±2.48	19.78±2.81	20.78±3.06	0.356
P13-N23 interpeak latency (ms)	4.00±1.48	4.11±1.60	4.40±1.84	0.356
P13-N23 amplitude (μV)	12.93±11.93	12.2±9.22	16.47±18.05	0.890
P13 rectified amplitude (μV)	5.45±6.11	4.85±3.92	7.12±7.27	0.606
N23 rectified amplitude (μV)	4.66±4.87	3.98±4.08	6.27±7.35	0.356
Interside difference				
Interaural latency diff, P13	2.13±1.89	2.21±1.87	1.78±1.13	0.983
Interaural latency diff, N23	1.67±1.13	2.06±1.40	2.70±2.22	0.511
P13-N23 amp asymmetry ratio, %	29.53±22.88	21.87±15.91	29.63±26.10	0.868

* Kruskal-Wallis Test. VM: vestibular migraine, MoA: migraine without aura

Table 4. *oVEMPResults of patients and healthy controls*

Parameters	VM	MoA	Controls	P value*
Leftside				
Latency N1(ms)	10.79±2.17	10.35±1.71	10.81±1.88	0.752
Latency P1(ms)	15.00±1.99	14.19±1.71	15.00±1.88	0.405
N1-P1 interpeak latency (ms)	4.21±1.19	3.84±1.06	4.09±1.56	0.627
N1-P1 amplitude (μV)	1.64±1.34	1.82±1.77	2.69±5.70	0.908
N1 rectified amplitude (μV)	1.10±1.24	0.95±1.20	1.08±2.39	0.766
Right side				
Latency N1 (ms)	10.92±2.40	10.35±1.63	10.11±1.39	0.632
Latency P1 (ms)	14.92±2.32	14.46±1.95	14.72±1.60	0.875
N1-P1 interpeak latency (ms)	4.00±1.48	4.11±1.60	4.40±1.84	0.910
N1-P1 amplitude (μV)	4.57±5.42	2.38±2.53	4.56±4.56	0.262
N1 rectified amplitude (μV)	1.84±2.57	1.05±1.38	1.94±3.48	0.726
Interside difference				
Interaural latency diff, N1	1.82±1.42	1.46±0.97	1.29±1.05	0.880
Interaural latency diff, P1	1.79±1.55	1.66±1.36	1.26±1.04	0.637
Amp. Asymmetry ratio, %	44.96±30.06	43.30±30.92	36.90±27.92	0.861

*Kruskal-Wallis Test. VM: vestibular migraine, MoA: migraine without aura

vestibular dysfunction in VM patients[10, 11, 31]. Such variance suggests that the migraine mechanism may act on the vestibular system at various levels[32]. Although the pathophysiology is not clear, altered neural activity within the trigeminovascular system and vestibular hyperexcitability are considered the primary mechanisms of vestibular dysfunction in patients with migraine[33, 34].

We found no significant differences in cVEMP or oVEMP parameters between patient and control groups. The findings in the literature on VM and cVEMPs do not appear homogenous. Some authors reported abnormalities in latency, amplitude, and the presence or absence of a response[2, 17, 35–37]. Our results are concordant with the results of the studies performed by *Taylor* et al. and *Kandemir* et al. revealing cVEMPs with similar latencies and amplitudes in patients with VM and healthy controls[13, 38]. Although we could not demonstrate the diagnostic significance of c- and oVEMP, some researchers have considered VEMP findings to be effective markers in VM diagnosis. *Makowiec* et al. reported that patients with VM exhibited normal cVEMP and abnormal oVEMP responses, suggesting that such a VEMP pattern might be a biomarker of VM[39]. Additionally, many investigators have reported that VM patients often manifest oVEMP but not cVEMP abnormalities. They reported that higher rates of abnormal oVEMPs may suggest greater vulnerability within the ascending utricular ocular pathway in patients with VM[40–42]. Whereas, Taylor et al. detected no significant c/oVEMP abnormalities in VM patients, which is consistent with our results[13]. Based on these previously published reports in the literature and the results of the present study, we think that the variability of VEMP results of VM patients prevent these responses from being used as a definitive biomarker.

The VEMP profiles of patients and controls in our study differed in the bilateral presence of c- and oVEMP responses. A high absent cVEMP responses in migraineurs was observed compared to the controls in our study, although the difference was not statistically significant. In line with our findings, *Hong* et al. stated that neither an abnormality in latencies nor a cVEMP asymmetry was present but 60% of the patients had bilaterally absent cVEMP responses[12]. A significant low response rate was also observed in the MoA group in the present study, but there was no statistically significant difference among the groups concerning the VEMP response rate. This finding may suggest a subclinical dysfunction within the descending saccular pathway in patients with MoA and might be related to pathophysiological similarities between MoA and VM. Moreover, Taylor et al. concluded that peripheral vestibular function is usually preserved in VM and that central mechanisms must be the cause of vertigo[13]. There are several hypotheses about absent VEMP responses, such as reduced serotonergic control of the saccular reflex pathways in the brainstem and insufficiency of glutamate, the major neurotransmitter of the vestibular system[35, 39, 42]. Although VEMP results in patients with MoA are contradictory, *Boldingh* et al. re-

ported uni- or bilaterally absent cVEMPs in 44% of their patients with VM and 25% of their patients with migraine as compared to 3% of the healthy controls[43].

Various vestibular function test studies have been conducted in patients with migraine during the interictal period. Several studies have reported vestibular abnormalities in the form of involvement of peripheral or central vestibular pathways or both[3, 43]. One study reported dysfunction in the vestibulo-ocular reflex, whereas another indicated underlying dysfunction in the vestibulospinal system. These findings suggest that migraineurs without vestibular symptoms exhibit vestibular abnormalities, generally indicating subclinical vestibulopathy[44, 45]. *Allena* et al. recorded normal latency cVEMPs with reduced amplitude, which suggested reduced serotonergic control of the VEMP pathways[46]. *Yetiser* et al. also recorded normal latency with a unilaterally reduced amplitude of P13 in 30 female migraine patients[47]. *Moallemi* et al. reported no meaningful difference between migraine patients and a healthy group in cVEMP asymmetry measures. Furthermore, they claimed that unilateral headaches in migraine patients do not result in abnormalities in VEMP side difference measures[48]. *Kandemir* et al. also reported a normal interictal cVEMP profile in migraineurs which is consistent with our results[38].

Although oVEMP abnormalities are reported more frequently in VM, cVEMP abnormalities have been reported to be more reliable than oVEMP in assessing vestibular dysfunction in migraineurs indicating subclinical vestibulo-collic pathway dysfunction[2]. In a study evaluating the diagnostic value of cVEMP in VM and migraine, the absence of VEMP responses was found to be numerically higher in the migraine group than in the VM group. The increased rate of absent VEMPs was associated with hypoperfusion of the sacculo-collic reflex pathway in migraine patients. In addition, it was concluded that the VEMP reflex responses appear to be insufficient for the differential diagnosis of VM and migraine[49]. However, some authors reported an abnormal interictal oVEMP profile in migraineurs, suggesting pathology within the vestibulo-ocular reflex[4]. They reported that oVEMP is a more reliable measure than cVEMP to evaluate vestibular function in migraineurs. Moreover, the significantly prolonged oVEMP latencies in their study suggested an underlying functional abnormality in the central vestibular system.

There was no statistically significant difference in the absolute and interpeak latencies obtained for BAEP among the groups consistent with some previous reports[50]. *Dash* et al. evaluated the audiovestibular functions in 50 cases of migraine with or without vertigo[24]. They reported that all patients showed some abnormalities in the form of prolonged absolute latency or prolonged interwave peak latencies or both consistent with the findings of *Zhang* et al.[27]. These results demonstrated that BAEP abnormalities might be the earliest indicator of impending auditory involvement in migraine. Moreover, in the studies of Zhang et al. compared with the migraine group, the peak latencies of I, III, and V waves in the VM group were prolonged, but the V wave changes were still within the normal range, indicating that brainstem dysfunction was more serious in VM patients than in migraine patients and VM patients have both central nervous system damage and peripheral nerve damage. Prolongation of wave V latency in VM patients has been indicated as a physiological dysfunction in the auditory system up to the brainstem level in another study[37].

In conclusion, the VEMP and BAEP tests are easy, noninvasive, and convenient to use in daily clinical practice with minimal discomfort. In our study, we provide evidence of the possible involvement of the descending saccular pathways in MoA and VM, as shown by the higher absent cVEMP responses in migraineurs than in healthy individuals. However, based on the findings of the present study, it is possible to state that VEMP and BAEP findings should neither be used in the differential diagnosis of VM and MoA. Futher studies are needed to determine whether MoA and/or VM are disorders of central or peripheral pathology.

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