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Evaluating vertebrobasilar insufficiency and Meniere's disease: Insights from cervical vestibular evoked myogenic potential and video head impulse test

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A vertebrobasilaris elégtelenség és a Meniere-kór értékelése: A nyaki vestibularis kiváltott myogen potenciál és a videós fejimpulzustesztből nyert felismerések

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Background and purpose – This prospective study aimed to investigate differences in video head impulse test (vHIT) and cervical vestibular evoked myogenic potential (cVEMP) findings between patients with vertebrobasilar insufficiency (VBI) and Meniere's disease (MD) who experience episodic vertigo attacks.

Methods – A total of 27 patients with VBI and 37 patients with MD were enrolled into the study in a tertiary referral center. Inclusion criteria consisted of patients with a minimum of two previous vertigo attacks, unaccompanied by any neurological symptoms during an attack. All patients underwent horizontal canal h-vHIT and c-VEMP assessments following pure sound audiometric examinations. First, vHIT and cVEMP results for low and high flow sides in VBI patients were analyzed. Subsequently, data from the low-flow side in VBI patients and the affected side in MD patients were compared.

Results – The mean vHIT values for low and high-flow volume sides in VBI patients were 0.68 and 0.88, respectively. In MD patients, mean vHIT values for affected and healthy sides were measured as 0.77

Háttér és cél – Ennek a prospektív vizsgálatnak a célja a videós fejimpulzusteszt (vHIT) és a cervicalis vestibularis kiváltott myogen potenciál- (cVEMP-) eredmények különbségeinek vizsgálata volt olyan vertebrobasilaris elégtelenségben (VBI) és Meniere-kórban (MD) szenvedő betegek között, akiknél epizodikus szédüléssel rohamok jelentkeznek.

Módszerek – A vizsgálatba összesen 27 VBI-beteget és 37 MD-beteget vontunk be egy tercier szakorvosi központban. A felvételi kritériumokat akkor teljesítették a betegek, ha legalább két korábbi szédüléssel rohamuk volt, és a roham alatt nem volt semmilyen neurológiai tünetük. Minden betegnél a tiszta hang audiometriai vizsgálatokat követően horizontális csatornás (h)-vHIT- és cVEMP-értékelést végeztünk. Először a vHIT- és a cVEMP-eredményeket elemeztük a VBI-betegek alacsony és magas áramlási oldalára vonatkozóan. Ezt követően a VBI-betegek alacsony áramlási oldalának és az MD-betegek érintett oldalának adatait hasonlítottuk össze.

Eredmények – Az átlagos vHIT-érték a VBI-betegek alacsony és magas áramlási térfogatú oldalain 0,68, illetve 0,88 volt. Az MD-betegeknél az érintett és az egészséges oldal

and 0.87, respectively. Abnormal results were observed in 66.7% of VBI patients and 51.4% of MD patients, with no statistically significant difference between the findings ($p > 0.05$). Upon examining the affected side, c-VEMP responses were absent in 41% of MD patients and 48% of VBI patients, with no statistically significant difference between the groups ($p > 0.05$).

Conclusion – vHIT and cVEMP assessments can be utilized as supplementary tools to radiologic investigations for the clinical diagnosis and follow-up of VBI. However, no significant differences were observed between vHIT and cVEMP findings in patients with MD and VBI.

Keywords: vertebrobasilar insufficiency, Meniere's disease, video head impulse test, vestibular evoked myogenic potential, vestibular system

átlagos vHIT-értékei 0,77 és 0,87 voltak. A VBI-betegek 66,7%-ánál és az MD-betegek 51,4%-ánál rendellenes eredményeket figyeltünk meg, és az eredmények között nem volt statisztikailag szignifikáns különbség ($p > 0,05$). Az érintett oldal vizsgálatakor a cVEMP-válaszok az MD-betegek 41%-ánál és a VBI-betegek 48%-ánál hiányoztak, a csoportok között nem volt statisztikailag szignifikáns különbség ($p > 0,05$).

Következtetés – A vHIT- és a cVEMP-értékelések a radiológiai vizsgálatokat kiegészítő eszközként használhatók a VBI klinikai diagnózisában és utánkövetésében. Mindazonáltal, a vHIT- és a cVEMP-leletek között nem észleltünk szignifikáns különbségeket az MD- és a VBI-betegekénél.

Kulcsszavak: vertebrobasilaris elégtelenség, Meniere-betegség, video-fejimpulzusteszt, vestibularis kiváltott myogen potenciál, vestibularis rendszer

Meniere's disease (MD) is a complex inner ear disorder characterized by idiopathic episodes of vertigo, accompanied by fluctuating hearing loss, tinnitus, and a sensation of aural fullness. With a community incidence of approximately 190 per 100,000 individuals¹, this condition has garnered significant attention within the medical community. The diagnostic and therapeutic guidelines, initially established in 1972 by the American Academy of Otolaryngology Committee on Hearing and Equilibrium (AAOHN-CHE), were updated in 2015 to reflect the current understanding of the disease². The diagnostic criteria primarily rely on subjective data, with two key classifications emerging²:

Definite Meniere Disease: a minimum of two spontaneous episodes of vertigo, each lasting between 20 minutes and 12 hours; audiometrically documented low-to-medium frequency sensorineural hearing loss in the affected ear, occurring at least once prior to, during, or after one of the vertigo episodes; fluctuating aural symptoms (fullness, hearing, tinnitus) localized in the affected ear; and absence of a more fitting vestibular diagnosis.

Probable Meniere Disease: a minimum of two episodes of dizziness or vertigo, each lasting between 20 minutes and 24 hours; fluctuating aural symptoms (fullness, hearing, or tinnitus) in the affected ear; and presence of a more fitting vestibular diagnosis.

Ischemia in the vertebrobasilar system (VBS) primarily manifests in two forms: vertebrobasilar insufficiency (VBI) and stroke resulting from posterior circulation disorders³. The National Institute of Neurological Disorders

and Stroke (NINDS) defines VBI as transient ischemia in the VBS⁴. Comprised of two vertebral arteries and one basilar artery, the VBS supplies blood to critical brain structures, including the pons, medulla oblongata, cerebellum, occipital cortex, mesencephalon, thalamus, and vestibulocochlear system. Atherosclerosis is the most common cause of VBI and VBS occlusion, followed by major vascular atherosclerosis, embolism, and arterial dissection⁵. Less frequently, migraine, coagulopathy, and substance abuse may contribute to VBI⁵. Classic symptoms include episodic vertigo, sudden hearing loss, dysarthria, hemiparesis, diplopia, dysphagia, ataxia, and falling attacks^{6, 7}. However, VBI may occasionally present with sudden hearing loss, vertigo, tinnitus, episodes of falls, and associated symptoms such as vomiting and nausea, exclusively affecting the vestibulocochlear system without other accompanying symptoms. Occlusion in this area can lead to hypoperfusion or transient ischemic attack, yielding episodic manifestations. These transient attacks necessitate the differentiation of VBS symptoms from other vestibular system diseases, with Meniere's disease being one such condition presenting similar clinical findings. **Table 1** shows the main clinical features of MD and VBI.

The video head impulse test (vHIT) serves as an objective method for evaluating the vestibulo-ocular reflex (VOR) arising from semicircular canals, building upon the head impulse test (HIT) first described by *Halma-gyi* and *Curthoys* in 1988⁸⁻¹⁰. vHIT provides a numerical assessment of VOR gain, as well as an evaluation of re-

fixation saccades in patients with vestibular insufficiency. Vestibular evoked myogenic potential (VEMP) measures electromyogenic muscle activities stemming from vestibular otolith organs following vestibular system stimulation, with cervical VEMP (cVEMP) representing the muscle response from the saccule to the sternocleidomastoid muscle (SCM) and oVEMP representing the muscle response from the utricle to the ocular system. The vestibulo-collic reflex originating from the saccule involves the saccule, inferior vestibular nerve, vestibular nuclei, vestibulospinal tract, accessory nuclei, and SCM muscle. Various stimuli, such as air-conducted audio stimulants, bone conduction stimulants, forehead taps, or galvanic stimulation, can be used to activate the vestibular system, allowing for VEMP recordings.

This study aims to investigate the differences in vHIT and cVEMP findings between patients with VBI (occurring in young-to-middle-aged individuals) and MD who present with episodic vertigo and drop attacks. By identifying unique patterns in these vestibular tests, we hope to improve diagnostic accuracy and facilitate tailored treatment approaches for these two clinically overlapping conditions.

Materials and methods

This study was conducted through a collaboration between the Otorhinolaryngology, Neurology, and Radiology departments at University of Health Sciences, Umraniye Training and Research Hospital from January 2019 to January 2023. All participating patients provided informed consent. The study received approval from the Ethics Committee of the University of Health Sciences, Umraniye Training and Research Hospital (Approval date: 21.07.2016, Approval number: 11060).

Patients

A total of 27 patients diagnosed with VBI and 37 patients diagnosed with unilateral MD, based on audiologic and radiologic examinations, were selected from the pool of patients presenting to the otorhinolaryngology and neurology outpatient clinics with recurrent vertigo episodes but without accompanying neurologic findings. MD diagnosis followed the 2015 revised diagnostic criteria of the Barany Society.

Exclusion criteria included patients aged under 18 years or over 60 years, those with previous or current middle ear disease, prior ear surgery or ablative therapy

Table 1. Diagnostic clinical features of MD and VBI

	MD	VBI
Vertigo	+ (It usually lasts 20 minutes to 12 hours, but not more than 24 hours)	+
Hearing loss	+ (Documented sensorineural hearing loss from low to mild frequencies during or after an episode of vertigo)	-
Ringling in the ear	+	-
Feeling of fullness in the ear	+	-
HINTS	Normal (Positive head impulse test on the side of the lesion, nystagmus in the horizontal axis without changing direction, no skew deviation)	Abnormal (Normal head impulse test, directional nystagmus, skew deviation)

MD: Meniere disease, VBI: vertebrobasilar insufficiency, HINTS (Head Impulse Test, Nystagmus, Test of Skew Deviation)

(steroids or gentamicin), additional vestibular diseases, migraine, vestibular migraine, vision loss, musculoskeletal system diseases, diabetes mellitus, hypo or hyperthyroidism, and presbyvestibulopathy. Furthermore, patients with MD demonstrating a total vertebral flow <200 mL/min in vertebral artery transcranial color duplex or bilateral MD were also excluded. Cranial MR imaging was used to rule out other pathologies that might cause vestibular system symptoms.

Only patients with at least two previous vertigo attacks, unaccompanied by neurologic symptoms during the vertigo episode, were included in the study. Comprehensive clinical histories were obtained from all patients, and neurotologic and neuroophthalmological examinations were conducted. Bilateral transcranial color duplex sonography, h-vHIT (horizontal canal vHIT), and cVEMP were performed for all patients after pure sound audiometric examinations.

Image analysis

An experienced radiologist conducted all transcranial color-coded duplex sonography examinations using a high-frequency (7.5 MHz) linear probe on a Doppler USG system (Toshiba Aplio 300, Toshiba Medical Systems, Tokyo, Japan). Patients were evaluated in the supine position with their neck extended. The vertebral artery was investigated longitudinally between the transverse foramina. A spectral Doppler flow pattern of the vertebral artery was obtained during the acquisition time. The vertebral artery diameter was measured, and the spectral

flow pattern contours were manually delineated for blood flow volume estimation. The duplex US device automatically calculated blood flow volume in each vertebral artery using the following formula: blood flow volume (cm^3/min) = time average velocity (cm/s) \times (diameter/2)² (cm^2) \times 60. Vertebrobasilar insufficiency was defined as the total blood flow volume in bilateral vertebral arteries being less than 200 mL/min according to transcranial color duplex sonography¹¹⁻¹⁴.

vHIT Analysis

Horizontal vHIT was employed to assess the horizontal semicircular canal function. The EyeSeeCam system (Interacoustics a/s, Middelfart, Denmark) was used to record vHIT, which consisted of lightweight glasses with a small video camera and a half-silver mirror reflecting the patient's left eye image. The glasses were tightly secured to the patients' heads. Patients were instructed to fixate on a target placed on a wall 1.2 meters away. Calibration was performed prior to each recording. During the test, random head impulses were applied along both lateral semicircular canal axes, approximately 15-20° on the lateral side of the midline in the horizontal plane at 150°-200°/s. Fifteen recordings were made separately for each side. VOR Gain at 40, 60, and 80 ms was recorded, with the mean VOR Gain at 60 ms being taken for evaluation. Normal values for VOR Gain were accepted as 0.8-1.2⁹.

cVEMP Analysis

cVEMP recordings were conducted using an evoked potentials machine (Eclipse EP- 25/VEMP; Interacoustics, Denmark). The active electrode was placed in the middle of the same sternocleidomastoid muscle, the reference electrode on the upper two-thirds of the SCM, and the grounding electrode in the middle of the forehead. The test was performed once in a silent environment with the patient awake and sitting. Ipsilateral recordings were obtained with stimulation from the right and left ears. Electrode impedance was maintained at <5 k Ω . Acoustic

stimulation of 100 dB for 0.1 ms was delivered separately to each ear at 5 Hz. The EMG signal was filtered in the range of 10 to 500 Hz, and averaged over a 100 ms interval. As a result of cVEMP, a total of 200 results were averaged. P1 and N1 negative/positive polarity peak waves were measured. P1 and N1 peak latencies and P1-N1 inter-peak amplitudes were calculated.

vHIT and cVEMP data for the affected and healthy sides of patients with MD were calculated separately. Moreover, vHIT and cVEMP data for the low-flow and high-flow sides were calculated separately and recorded according to the vertebral flow states of the patients with VBI.

The study data were examined under two categories. In the first analysis, vHIT and cVEMP results obtained from the low-flow and high-flow sides of patients with VBI were statistically analyzed; additionally, the healthy and affected sides of patients with MD were statistically analyzed. In the second analysis, the data of the low-flow side of patients with VBI and the affected side of patients with MD were statistically compared.

Statistical analysis

Statistical analysis was performed using SPSS version 20 program (IBM Corporation, New York, NY). Descriptive statistical analyses were conducted. The Chi-square test was employed to compare qualitative variables. Quantitative variables were examined for normal distribution using variance analysis. The independent sample t-test was utilized to compare quantitative variables with normal distribution, while the Mann-Whitney U test was used to compare quantitative variables without normal distribution. A p-value of <0.05 was considered statistically significant.

Results

The study included 64 patients, with a mean age of 49.5 years for patients with VBI and 45.3 years for patients with MD (Table 2).

Table 2. Patient characteristics

	VBI	MD	P value
	n: 27	n: 37	
Sex (M/F); n	9/18	13/24	0.54
Low Flow & Disease Side (R/L)	10/17	15/22	0.06
Age (year)	Mean \pm SD (range)	49.52 \pm 7.39 (35-60)	45.27 \pm 8.74 (28-60) 0.06

VBI: vertebrobasilar insufficiency, MD: Meniere disease, M: male, F: female, R: right, L: left

Table 3. *h-vHIT results for both groups. There was no difference between Meniere's disease and VBI in terms of h-vHIT VOR Gain results ($p>0.05$). In addition, in VBI patients, the VOR Gain results for the affected side were lower compared to the healthy side ($p<0.05$)*

h-vHIT Gain		VBI n: 27	MD n: 37	p-value
Low Flow & Disease Side	Mean±SD (Range)	0.68 ± 0.18 (0.29–1.02)	0.77 ± 0.24 (0.29–1.02)	0.08
High Flow & Healthy Side	Mean±SD (Range)	0.88 ± 0.17 (0.62–1.14)	0.87 ± 0.19 (0.45–1.20)	0.87
p-value		*0.001	0.10	

VBI: vertebrobasilar insufficiency, MD: Meniere disease, SD: standard deviation

Vertebral artery flow volumes were assessed using transcranial color duplex sonography. The right vertebral artery flow volume was 71.11 ± 44.06 mL/min, the left vertebral artery flow volume was 96.30 ± 47.73 mL/min, and the total vertebral artery flow volume was 168.41 ± 28.50 mL/min. The total vertebral artery flow volume for patients with MD was measured as 240 ± 20 mL/min.

In patients with VBI, the low-flow and high-flow sides were evaluated separately based on vertebral flow state. The mean vertebral artery flow volume on the high-flow side that fed the basilar artery was 121.48 ± 33.59 mL/min, and the mean vertebral artery flow volume on the low-flow side was 45.93 ± 21.35 mL/min.

Hearing assessment was conducted using pure sound audiometric examination. In patients with VBI, the mean pure sound audiometry results for the low-flow and high-flow sides were 18 ± 5.4 dB HL and 15 ± 7.2 dB HL, respectively. In patients with MD, the affected and healthy ear hearing thresholds were evaluated separately, with measurements of 39.5 ± 23.0 dB HL and 18.8 ± 14.9 dB HL, respectively.

A total of 64 bilateral h-vHIT records were obtained for both groups. In patients with VBI, the mean vHIT results for low-flow and high-flow volume sides were 0.68 ± 0.18 and 0.88 ± 0.17 , respectively, based on the arterial flow volume state ($p<0.05$). In patients with MD, the mean vHIT results for the affected and healthy sides were 0.77 ± 0.24 and 0.87 ± 0.19 , respectively ($p>0.05$) (Table 3).

In both groups, considering a cut-off value of 0.8, 66.7% of test results were abnormal in the low-flow side for patients with VBI, and 51.4% of test results were ab-

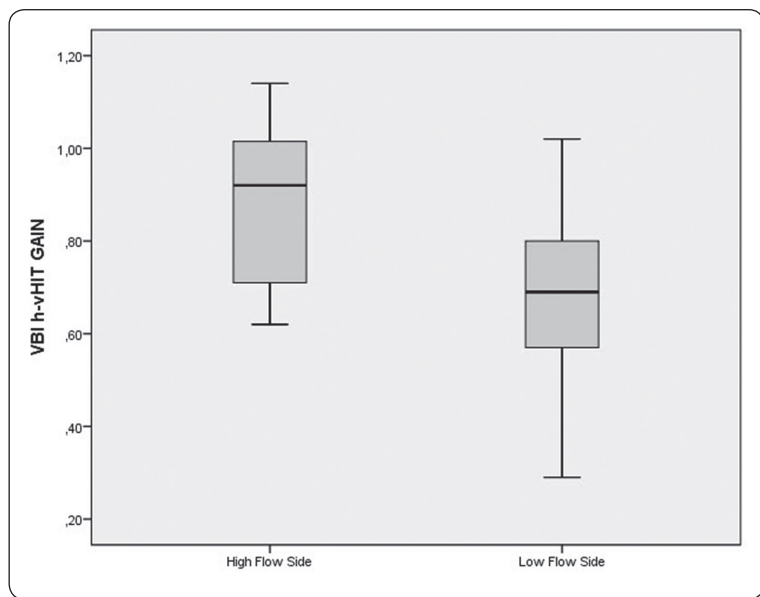


Figure 1. *vHIT results according to high and low flow in vertebrobasilar insufficiency patients. Low flow was found to have low vHIT gain on the current side ($p<0.05$)*

normal in the affected side for patients with MD ($p>0.05$) (Figure 1). Moreover, 37.0% of test results were abnormal in the high-flow side for patients with VBI, and 37.8% of test results were abnormal in the healthy side for patients with MD ($p>0.05$) (Figure 2).

cVEMP responses were examined in both groups. For the affected side, cVEMP responses were not obtained in 41% ($n=15$) of patients with MD and 48% ($n=13$) of patients with VBI ($p>0.05$) (Table 4).

In cVEMP examinations, P1-N1 peak latencies and P1-N1 amplitude values were analyzed. When comparing the affected sides of MD and VBI patients, no significant difference was found in terms of cVEMP findings, while maintaining the integrity of meaning ($p>0.05$) (Table 4).

Discussion

Drop attack is characterized by spontaneous episodes of sudden falls lasting seconds or minutes, often without loss of consciousness. This serious symptom is frequently accompanied by dizziness, vomiting, and nausea. Drop attack etiology includes heart diseases (syncope variants) (12%), VBI (8%), both heart disease and VBI (8%), seizures (7%), inner ear diseases (Meniere's disease) (5%), and psychological problems (1%)¹⁵.

VBI and MD share similar symptoms, making it difficult to differentiate between the two diseases. Both conditions present with episodes of drop attacks, vomiting, nausea, dizziness, and sudden hearing loss (fluctuating).

Lee W et al.¹⁶ reported a patient with VBI experiencing recurrent vertigo attacks. The patient's attacks occurred once per month, lasting approximately 20 minutes. During the attack, the patient had horizontal right-beat nystagmus and catch-up saccades in the HIT test. Other cranial nerve examinations were normal. MRI revealed occlusion in the left vertebral artery and right anterior inferior cerebellar artery (AICA). The patient had no response on the right side in the cVEMP examination¹⁶.

Central or peripheral vertigo may result from ischemia because the AICA supplies both the brainstem and the vestibular system. Conversely, the medial branch of the posterior inferior cerebellar artery (mPICA) provides blood to the cerebellar nodulus and uvula. Peripheral vertigo may occur in ischemia related to this artery because these parts are connected to the ipsilateral vestibular nucleus. Lee H et al.'s study¹⁷ examined 240 patients with cerebellar infarction and identified 25 patients with ves-

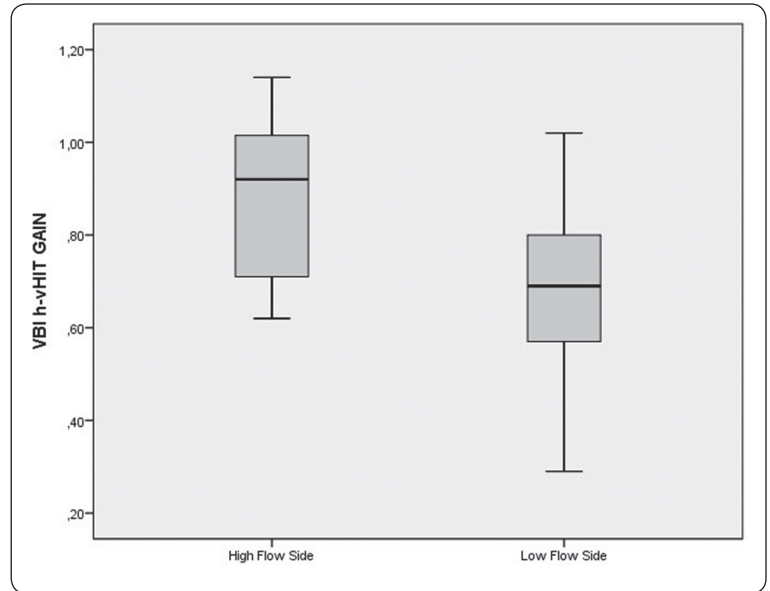


Figure 2. Patient and healthy side vHIT results in "Meniere's patients". There was no difference between vHIT gains on both sides ($p > 0.05$)

tibular neuritis. Twenty-four (96%) of the 25 patients had infarction in the mPICA territory and one patient (4%) in the AICA territory¹⁷. Hearing loss may accompany acute vestibular syndrome due to AICA ischemia, and these findings may present as episodic attacks.

Patient with migraine frequently has vestibular complaints. Many studies have identified subclinical vestibular dysfunction in migraineurs who do not complain of vestibular symptoms. In a study by Inan et al. was shown that vestibular system involvement may be present in migraine patients¹⁸.

The inner ear (vestibular apparatus and cochlea) is supplied by the labyrinthine artery, which originates from the AICA. The labyrinthine artery is an end artery. Since the brainstem and cerebellum are mostly supplied

Table 4. cVEMP results of both groups

c-VEMP	Low Flow & Disease Side		p-value	High Flow & Healthy Side		p-value
	VBI	MD		VBI	MD	
	Mean \pm SD (Range)	Mean \pm SD (Range)		Mean \pm SD (Range)	Mean \pm SD (Range)	
P1 Latency (seconds)	17.21 \pm 1.54 (14.67–19.67)	16.26 \pm 1.62 (14.33–20.33)	0.87	13.99 \pm 0.57 (13.0–14.67)	14.26 \pm 0.97 (11.67–15.67)	0.19
N1 Latency (seconds)	26.06 \pm 2.11 (23.0–29.67)	25.60 \pm 1.38 (24.0–28.33)	0.58	24.11 \pm 1.25 (22.0–25.67)	23.32 \pm 1.35 (20.6–25.67)	0.12
P1-N1 Amplitude (μ V)	21.20 \pm 24.61 (0.0–69.09)	32.33 \pm 39.11 (0.0–135.5)	0.49	47.38 \pm 38.81 (0.0–157.5)	71.71 \pm 55.68 (0.0–151.9)	0.85

VBI: vertebrobasilar insufficiency, MD: Meniere disease, SD: standard deviation

by anastomosis arteries, they are less affected by ischemia and infarction than the inner ear¹⁹. This explains the isolated acute vestibular syndrome and hearing loss in VBI^{20, 21}.

Few studies have investigated the functional outcomes of VBI on the vestibular system. *Guo et al.* found canal paresis in caloric tests in 50% of patients in their study²². *Baki et al.* reported decreased cVEMP latencies and amplitudes in patients with VBI²³. In our study, bilateral or unilateral cVEMP responses were not obtained in 44.4% of all patients. On the low-flow side, this rate was 48%. Additionally, the average h-vHIT gain was measured below the cut-off value of 0.8 in 66.7% of patients. When examining the low and high-flow sides separately in patients with VBI, both vHIT and cVEMP data on the low-flow side showed significant abnormalities compared to the high-flow side.

Permanent damage to audiovestibular structures occurs due to endolymphatic hydrops in MD, resulting in unilateral or bilateral vestibular insufficiency. In our study, 41% of patients with unilateral MD had no cVEMP responses when examining cVEMP data. *Egami et al.* identified cVEMP abnormalities in 29.8% of patients with unilateral MD in their study²⁴. In later studies, *Chen et al.* found this rate as 40%²⁵. The mean vHIT gain was 0.77 when the horizontal canal cut-off vHIT gain value was accepted as 0.8. *Heuberger et al.* found a vHIT gain of 0.7 in a study involving 35 patients with single-sided MD²⁶. In subsequent studies, *Lee SU et al.* found a vHIT of 0.7²⁷, and *Rubin et al.* found a vHIT of 0.78²⁸.

In our study, there was no statistically significant difference between the cVEMP and h-vHIT findings of patients with MD and VBI. The peripheral vestibular system was affected in both diseases, and abnormalities were observed in vHIT and cVEMP examinations.

In conclusion, the findings of our study suggest that both VBI and MD can affect the peripheral vestibular system, leading to abnormalities in vHIT and cVEMP examinations. Due to the similarity in symptoms and vestibular test results, it can be challenging to differentiate between these two conditions based solely on clinical presentation and vestibular testing. Furthermore, future

investigations could benefit from extending the vHIT analysis to assess the function of all semicircular canals, rather than focusing solely on the horizontal canal, as this may provide a more comprehensive evaluation of vestibular function in patients with VBI and MD. Future studies could further explore the role of ABR findings, the HINTS protocol, and other diagnostic clinical features in differentiating between VBI and MD. Incorporating these additional assessments alongside vHIT and cVEMP may provide a more comprehensive understanding of the underlying pathophysiology and improve diagnostic accuracy. Moreover, comparing the results of these tests with the findings of vHIT and cVEMP could offer valuable insights into the relationship between various diagnostic methods and their clinical utility in distinguishing these two conditions. A comprehensive diagnostic approach, including detailed medical history, imaging, and other diagnostic tests, is essential to accurately identify and manage these disorders. Further research is needed to better understand the relationship between VBI, MD, and vestibular function, as well as to develop more reliable methods for differentiating between these conditions.

Conclusion

In summary, vHIT and cVEMP can be valuable tools for the clinical diagnosis and follow-up of VBI when used alongside radiological investigations. However, our study found no significant differences between vHIT and cVEMP findings in patients with MD and VBI. This indicates that additional diagnostic methods beyond these tests are necessary for accurately differentiating between the two diseases. Further research and the development of more precise diagnostic tools will be essential in improving the differentiation and management of VBI and MD.

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