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VESTIBOLOGY

Evaluation of video head impulse test during vertiginous attack in vestibular migraine

Valutazione dell'head impulse test eseguito durante l'attacco vertiginoso acuto nella emicrania vestibolare

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SUMMARY

Objective. The aim of this study is to evaluate vestibular functions with video head impulse test (VHIT) and to understand the value of VHIT in differential diagnosis in patients with vestibular migraine (VM) during dizziness attack.

Materials and methods. Two groups were enrolled in this study. The first consisted of 84 vestibular migraine patients, and second group of 74 healthy subjects. VHIT was applied to patients with VM during vertigo attack and the results were compared with the VHIT values applied to subjects in the control group.

Results. The mean vestibulo-ocular reflex (VOR) in all semicircular canals in the VM group was lower than healthy individuals, but the results were not statistically significant. Refixation saccades were found in 52.3% of VM patients and in 10.2% of healthy individuals.

Conclusions. When patients with VM were evaluated with VHIT during vertiginous attack, VOR gain values were not different from healthy individuals, but the number of catch-up saccades were higher in VM patients, which indicates peripheral vestibular involvement. For differential diagnosis in patients with VM, vestibular tests should be performed during the vertigo attack. When evaluating VHIT results, the presence of refixation saccades should also be evaluated.

KEY WORDS: vertigo, migraine, head impulse test

RIASSUNTO

Obiettivo. *Lo scopo di questo studio è valutare la funzione vestibolare con il Video Head Impulse Test (VHIT) e valutare il valore della VHIT nella diagnosi differenziale dei pazienti con emicrania vestibolare (VM) durante l'attacco vertiginoso acuto.*

Materiali e metodi. *Ci sono due gruppi arruolati in questo studio. Il primo Gruppo è composto da 84 pazienti con emicrania vestibolare e il secondo gruppo da 74 soggetti sani. La VHIT è stata applicata ai pazienti con VM durante l'attacco di vertigini e i risultati sono stati confrontati con i valori VHIT applicati ai soggetti nel gruppo di controllo.*

Risultati. *Il guadagno medio del riflesso oculo vestibolare (VOR) in tutti i canali semicircolari nel gruppo VM era inferiore rispetto agli individui sani, ma i risultati non erano statisticamente significativi. I movimenti saccadici sono stati riscontrati nel 52,3% nei pazienti con VM e nel 10,2% negli individui sani.*

Conclusioni. *Quando i pazienti con VM sono stati valutati con VHIT durante un attacco vertiginoso, i valori di guadagno di VOR non erano diversi dagli individui sani, ma il numero di movimenti saccadici di recupero era più alto nei pazienti con VM, il che indica un coinvolgimento vestibolare periferico. Per la diagnosi differenziale nei pazienti con VM, i test vestibolari devono essere eseguiti durante l'attacco acuto di vertigini. Quando si valutano i risultati VHIT, deve essere valutata anche la presenza di movimenti saccadici di rifissazione.*

PAROLE CHIAVE: *vertigini, emicrania, head impulse test*

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Introduction

Migraine is an episodic headache disease with diagnostic criteria described by the International Headache Society (IHS). Vestibular symptoms are more common in patients with migraine than in the general population. In about half of patients, dizziness has also been reported [1,2]. Vestibular migraine (VM) is considered as a separate entity because of case-controlled studies [3]. It is the most common cause of vertigo after benign paroxysmal positional vertigo [3]. The lifetime prevalence is estimated to be 1% [4]. It can be seen at any age and is more common in women [5] with the highest prevalence in middle-aged women [2]. There is no objective diagnostic test or biomarker for vestibular migraine. Diagnostic criteria were described by Neuhauser in 2001 [6]. In 2012, the Barany Society and International Headache Society published the first consensus on diagnostic criteria [3].
Symptoms are variable in patients with VM; the most frequent is recurrent episodes of spontaneous vertigo, but positional vertigo and vertigo triggered by head movements may also be seen. Vestibular symptoms may occur during a migraine headache attack or in the attack-free period [5,7]. Thus, VM is difficult to diagnose, as vestibular symptoms are also common in patients with migraine.
Vestibular tests may show abnormal findings during or immediately after vertigo attacks [5]. It is not known exactly how vestibular functions are affected in vestibular migraine. Vestibular functions can be measured indirectly by the physiological vestibulo-ocular, vestibulo-spinal and vestibulo-colic reflexes. Measurement methods are electronystagmography, videonystagmography, vestibular evoked myogenic potentials and video head impulse test (VHIT). VHIT provides objective evaluation for all semicircular canals, and is a physiological and easy-to-apply test. With VHIT, vestibulo ocular (VOR) gain (ratio between head velocity and eye velocity), compensatory eye movements (covert and overt saccades) and the time required for correcting gaze can be determined. VHIT can provide information that help to better understand the pathophysiology of VM. In the literature, studies reporting VHIT results in VM patients are examinations made during the vertigo attack-free period. To our knowledge, there is no study in the literature evaluating the results of VHIT measurements during a vertigo attack for VM patients. The aim of this study is to evaluate vestibular functions during vertigo attack in vestibular migraine patients with VHIT and to understand the value of VHIT in differential diagnosis.

Materials and methods

This case control study was carried out in the Vestibular Diseases Unit. VHIT test results of patients with VM and healthy control group were evaluated. VM patients were included in the first group. 84 patients were identified with a retrospective evaluation who were diagnosed with VM according to Barany Society and International Headache Society criteria between September 2018 and September 2020. The diagnosis of VM was made in collaboration with the authors, who are ENT and Neurology specialists. The second group consisted of healthy volunteers without dizziness or migraine headache complaints. There were 84 patients in the VM group, mean age was 40 (18-65), 72 were female (85.7%) and 12 were male (14.3%). There were 78 individuals in the healthy control group. The mean age of individuals in this group was 38 (22-63), 68 were female (87.1%) and were male (12.9%). Individuals under 18 years of age, those with current or previous neurological disease and patients using vestibular sedative drugs were excluded from the study. Healthy individuals did not have a history of episodic or chronic headache, no hearing problems or dizziness complaints, did not use any medications, and had normal ENT physical examination. The groups were homogeneous in terms of gender ($p = 1.000$) and age ($p = 0.998$). A detailed history was taken from all patients, and standard ENT and neurological examinations were performed. All patients were evaluated on the day of vertigo attack.
Subjects in both groups were examined with VHIT by two investigators (by an ENT physician with experience in neurotology, and an audiologist with experience in VHIT applications). The investigators were blinded to this process. VHIT Ulmer was used for VHIT testing (VHIT ULMER, Synapsys, Marseille, France). In the VHIT test, the subject was placed on the examination chair 90 cm away from the VHIT device (the distance between the lens of the camera and the patient's external orbital cantus). Three fixation dots were placed on the wall 1 m away from the device, one in the central gaze axis and the others at ± 15-20°. Patients were asked to keep their neck muscles relaxed, open their eyes wide and not blink. Head impulses were applied to stimulate each semicircular canal. For lateral canal stimulation, passive lateral head impulses were applied. For vertical canal stimulation, the patient's head was turned left (for right anterior - left posterior stimulation) and right (for left anterior - right posterior stimulation), 30-40° lateral from the midline and fixed to the fixation points. Next, unexpected passive head movements were applied at an amplitude of 10-15°, a duration of 150-200 msec and a peak speed of 150-200°/sec. At least 20 impulses were delivered for each canal.
VHIT gain was calculated as the ratio of the area under the curve of eye movement versus the curve of the head movement. Gain was classified as 1 when the graph of head move-

Table I. Results of VOR gains in both groups (data was expressed in mean ± SD, median (min-max) and compared with Mann Whitney test).

	VM Group N = 84	Control Group N = 78	P
Right anterior SCC			
Mean ± SD	0.97 ± 0.12	0.98 ± 0.095	0.39
Median (min-max)	0.98 (0.56-1.16)	0.99 (0.67-1.23)	
Left anterior SCC			
Mean ± SD	0.88 ± 0.15	0.97 ± 0.088	0.43
Median (min-max)	0.95 (0.52-1.29)	0.98 (0.74-1.28)	
Right lateral SCC			
Mean ± SD	0.85 ± 0.11	0.91 ± 0.085	0.57
Median (min-max)	0.92 (0.52-1.08)	0.94 (0.64-1.12)	
Left lateral SCC			
Mean ± SD	0.87 ± 0.13	0.90 ± 0.091	0.45
Median (min-max)	0.90 (0.24-1.05)	0.92 (0.64-1.07)	
Right posterior SCC			
Mean ± SD	0.90 ± 0.11	0.92 ± 0.088	0.35
Median (min-max)	0.94 (0.48-1.06)	0.95 (0.64-1.16)	
Left posterior SCC			
Mean ± SD	0.89 ± 0.11	0.92 ± 0.0928	0.45
Median (min-max)	0.91 (0.53-1.10)	0.93 (0.67-1.08)	

VOR: vestibulo-ocular reflex; VM Group: vestibular migraine group; SCC: semicircular canal.

ment and eye movement was equal (i.e., normal condition). If the eye movement during head movement is less than necessary to fix the gaze on the target, the gain will be less than 1 and the second eye movement will occur; correction saccade (CS). This corrective eye movement puts the gaze on the target. If the impulse delivered to the head does not provide an appropriate stimulus, the device will automatically reject this impulse. Possible VHIT results: gain (normal, lower, or higher than normal), presence and number of correction saccades. In VHIT analysis, VOR gain and presence of refixation saccades were evaluated. Normal VOR gain was taken as > 0.8 for the lateral canal and > 0.7 for the vertical canals. Mann-Whitney Test was used to determine the significance of any difference for VOR gain values between groups. A p value < 0.05 was considered significant. The number and percentage of saccades depending on VOR gains in both groups (VM group and control group) was analysed by chi-square test (Tab. II).

Results

In patients with VM, 4 (4.7%) of 84 patients with VM described migraine with aura, while 80 (95.3%) described migraine without aura. The number of patients who described spontaneous vertigo was 29 (34.5%), while 55 (65.5%) patients described positional vertigo. While migraine was present for less than 5 years in 24 patients (28.5%), it was present for more than 5 years in 60 patients (71.5%). The frequency of migraine headache was one or more attacks per week in 12 patients (14.2%), 1-3 attacks per month in 34 patients (40.5%) and less than once a month in 38 (45.2%) patients. While headache and vertigo were observed together in 16 patients (19%), in 27 patients (32.2%) vertigo was seen just before or just after the headache. In 41 patients (48.8%), the relationship was uncertain. Vertigo was present for less than 5 years in 73 patients (87%), and more than 5 years in 11 patients (13%).

VHIT analysis

Table I shows the average VOR gains for each semicircular canal in both groups. In the VM group, the mean VOR gains in all semicircular canals were lower than the healthy group, but the results were not significant. Refixation saccades were seen in 44 patients (52.3%) in the VM group, and only in 8 patients (10.2%) in the healthy group, all of these were covert saccades. This difference was significant (Tab. II). The amplitudes and latencies of the saccades seen in the VM group are shown in Table III.

Discussion

The fixation of the visual image in the retina during head

Table II. The number and percent of saccades depending on VOR gains in both groups.

	VM group (n = 84)		Control group (n = 78)		
	n	%	n	%	
No saccades	40	47.6	70	89.8	$p < 0.001^c$
Saccades with normal VOR gain	24	28.5	8	10.2	$p < 0.001^c$
Saccades with low VOR gain	20	23.8	0	0	$p < 0.001^c$

VOR: vestibulo-ocular reflex; VM Group: vestibular migraine group; [c]: chi-square test.

Table III. Amplitude (°/s) and latency (msn) of saccades in VM group.

VM group	Amplitude (°/s)	Latency (msn)
Saccades with normal VOR gain	203 (116-250)	121 (63-190)
Saccades with low VOR gain	151 (111-189)	139 (84-188)

VM Group: vestibular migraine group; VOR: vestibulo-ocular reflex.

movement is achieved by the VOR. The relationship between head and eye movements is called VOR gain [8,9]. If, due to vestibular hypofunction, the velocity of the eyes is lower than head velocity, the eyes pause in the orbit, delay and the eye slips off the target. In this case, eye movements called catch-up saccades (CS) are used to stabilise the gaze to compensate for the impaired eye movement [10,11]. CSs can be covert or overt depending on whether they are seen during head movements or after head movements are terminated [12]. VHIT, one of the newest tests in the vestibular laboratory, evaluates VOR by measuring eye deviations during rapid head acceleration in each semicircular canal (SCC) plan. VHIT calculates vestibulo-ocular reflex gain, determines the presence and amplitude of covert and overt catch-up saccades and records eye and head movements [8-13]. Unlike the caloric test, it is a physiological test, each semicircular canal can be evaluated, gives a more comprehensive assessment and is better tolerated by the patient [2,14,15].

In migraine, not only headache but also other complaints such as dizziness, hearing loss and tinnitus can be found. These are thought to be due to chemical changes in synaptic transmission and subsequent imbalance between neural excitation and inhibition [16]. VM is the second most common cause after BPPV in many studies evaluating patients with vertigo complaints [3,7]. VM occurs with central or peripheral vestibular dysfunction during headache attacks [17]. There are some hypotheses about how the inner ear disorders occur in patients with migraine. Sterile inflammatory response in intracranial vessels with activation in the trigeminovascular system [18], cortical spreading depression affecting the brain stem [3], peripheral vestibular dysfunction because of vasospasm in the internal auditory artery [19] and plasma extravasation because of trigeminal neurogenic inflammation in the labyrinth have been invoked [20]. Positron emission tomography (PET) and blood oxygenation level dependent functional magnetic resonance (fMR) studies have shown structural and functional changes in brain areas responsible for central vestibular control and vestibular compensation in patients with VM [21]. Vestibular findings are thought to be explained by a wave of depression that spreads from the occipital lobe to the cerebral cortex [1].

The fact that the clinical appearance of VM varies according to age creates difficulties in differential diagnosis. There is no laboratory test or biomarker that gives a definitive diagnosis for VM [2]. Therefore, vestibular tests in patients with VM are investigated to obtain information that will help in differential diagnosis [2]. Most patients with VM do not have any complaints during the attack-free periods. Nystagmus can be detected on physical examination during an attack. Performing vestibular tests during the vertiginous attack may be more useful for differential diagnosis.

Studies evaluating VHIT results in patients with VM have shown VHIT abnormalities (low gain and/or saccades) in 11-36% of cases [2,15,22]. Only gain abnormalities were detected between 8-11% [15,23,24]. In a study on 31 patients with VM, Salmito found similar VOR gain values for lateral canals between the VM and control groups, but found higher VOR gain values for vertical canals in the VM group [5]. Salmito argued that this difference in vertical canals may be due to artifacts, incorrect head positioning, improper positioning of the VHIT device, or head impulses at different velocities. In the study by Mahringer, VHIT had abnormal results in 11% of patients with VM [25]. In Blödow's study, 9% abnormal gain was found in patients with VM, and the average VOR gain in these cases was 0.73 ± 0.04 [23]. In two studies by Elsherif, VHIT abnormality in the VM group was 36% in 25 patients and 26.3% in 80 patients [1,22]. In the mean VOR gains between the VM and control groups, a statistically significant difference was found only in the right posterior canal measurements, and was found to be lower

in the group with VM. Kang detected abnormal VHIT findings in 9 (11%) of 81 VM patients [15]. In a previous study, the rate of covert saccades in patients with migraine was found to be significantly higher than the control group, and it was thought that this high saccade rate may be related to the connection between the trigeminal nerve and vestibular nuclei. Like VHIT, recent studies propose using functional head impulse test (fHIT) to evaluate VM patients [26,27]. The results of percentage of correct answers in fHIT can support the diagnosis of VM.

It has been argued that VM occurs because of a deterioration in the rate of spontaneous neural firing at the level of the vestibular nuclei with the decrease in inhibitory inputs from the cerebellar flocculus to the vestibular nuclei [22]. VHIT has been found to be useful in evaluating the response to medical treatment in patients with VM. Kang reported that cervical vestibular evoked myogenic potential test (cVEMP), ocular vestibular evoked myogenic potential test (oVEMP) and sensory organization test (SOT) are not useful when evaluating the response to medical treatment in patients with VM, but that VHIT results are useful. Complete recovery was observed in 82% of patients with normal VHIT gains compared to 44% in patients with abnormal VHIT gains [15]. VHIT has also been found to be useful in differential diagnosis of VM and Meniere's disease [23,28].

In all these studies, VHIT was performed in patients with VM during the period between attacks (attack-free periods). In the current study, the VHIT test was performed on the day of vertigo attack. In the VM group, 24 (28.5%) patients had normal VOR gain and 20 (23.8%) had low VOR gain. The total VHIT abnormality rate was found to be 52.3%. In the control group, only 8 patients (10.2%) had normal VOR gain with saccades.

The episodic character of VM means that tests are performed in the asymptomatic period in most patients. To our knowledge, the present study is the only study in the literature evaluating VHIT results during vertigo/dizziness attack in VM. The results show that there is no significant difference in VOR gains during attacks compared with results obtained in the attack-free periods in patients with VM or in healthy normal subjects. However, the high rate of refixation saccades seems to be more important than VOR gains in the evaluation of VHIT results, which indicates peripheral vestibular involvement.

Conclusions

VHIT should be among the vestibular tests performed for differential diagnosis in patients with vertigo-dizziness complaints. When evaluating the VHIT results, not only VOR gain rate, but also the presence of refixation saccades should be assessed. Vestibular tests should be performed on patients with VM, if possible, during the vertigo attack.

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Conflict of interest statement

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Authors' contributions

AK and ECA have equally contributed to the manuscript.

Ethical consideration

This study was approved by Ethics Committee of University of Acıbadem University (approval number/protocol number 2020-18/4).

The research was conducted ethically, with all study procedures being performed in accordance with the requirements of the World Medical Association's Declaration of Helsinki.

Written informed consent was obtained from each participant/patient for study participation and data publication.

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