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# The Video Head Impulse Test and the Oculomotor Test in Patients with Vestibular Migraine

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**Received:** 27 June 2024; **Revised:** 27 July 2024; **Accepted:** 14 November 2024; **Published:** 6 December 2024

**Abstract:** To compare the results of the Oculomotor Random Saccade test, Positional test and Video Head Impulse Test (vHIT) patients with Vestibular Migraine (VM) and in healthy volunteers and to determine the sensitivity of these tests in revealing peripheral vestibular hypofunction. A retrospective examination was made of the data of 33 patients (mean age:44.72±13.73 years) diagnosed with VM in a tertiary level neuro-otological clinic between June 2020 and December 2022. The oculomotor random saccade test, positional tests, vHIT gain and asymmetry results were compared with those of 33 healthy volunteers and statistically analysed. Gaze nystagmus was observed in 18.18% of VM patients. The positional tests were abnormal in 15.1% of the VM patients. The mean velocity was seen to be increased 18.2% patients in VM. The right-side accuracy values were determined to be <80% in 4 (12.2%) patients in VM. The two groups were similar in terms of vHIT vestibulo-ocular reflex (VOR) gain values. Vestibular deficit of VOR gain <0.7 was determined in 30.3% of the VM patients. VOR gain asymmetry of >20% was observed in 11 (33.3%) VM patients (p>0.05). Abnormalities in the oculomotor saccade test results and differentiated asymmetry values with reduced VOR gain values may be observed in patients with VM. It should be kept in mind that vHIT results may be affected in patients with VM.

**Keywords:** BPPV; Vertigo; Vestibular Migraine; Vestibular System; Vestibulo-Ocular Reflex

## 1. Introduction

Vestibular Migraine (VM) is a type of migraine that manifests with episodic vestibular symptoms [1, 2] and has a reported prevalence of 1–2.7% of the adult population. It has been reported that VM mainly affects females aged from late 30s to mid-40s, and that patients usually have complaints of motion sickness and headache since childhood [3]. A family history of migraine is common, and a history of anxiety/depression has been reported to increase the risk of VM [4]. The combination of VM and Benign Paroxysmal Positional Vertigo (BPPV) has also been reported in the literature [1, 5, 6]. Although the pathogenesis of VM is not clear, it is thought that the reciprocal connections between vestibular nuclei and the structures modulating trigeminal nociceptive inputs may play a role [7, 8]. The reciprocal connections between trigeminal and vestibular nuclei were defined in another human study. Trigeminal activation has been shown to create nystagmus in patients with migraine but not in healthy control subjects, and this has been linked to a reduced signal transmission threshold between the two systems [9]. At the same time, the trigeminovascular system innervates the inner ear [10].

Diagnosis of the disease is mainly based on the clinical history. A joint committee representing the Headache

Society and Barany Society defined both definitive and potential disease diagnostic criteria for the disease named VM, which is associated with the emergence of vestibular symptoms of migraine itself [1]. Although abnormalities in vestibular tests are common in VM, they are not specific to VM. Various vestibular tests have been used in different studies, but the results have been inconsistent. The neurological examination is generally normal in the symptom-free period between attacks. Moreover, central vestibular ocular motor abnormalities, including gaze-evoked nystagmus, saccadic pursuit, central positional nystagmus, and dysmetric or slow saccades [11, 12] have been associated with VM [6] in 8.6%–66% of patients [11, 13].

Spontaneous or positional nystagmus together with pathological nystagmus has been reported in more patients (70%) during an acute attack. These types of findings obtained during an acute attack indicate central vestibular dysfunction in 50% of cases, peripheral vestibular dysfunction in 15%, and indeterminate location of involvement in 35% [14]. Therefore, the reliability of vestibular tests used in the diagnosis of VM and progression of treatment remains a matter of debate. In conclusion, abnormal ocular motor findings are common in VM, but there are no pathognomonic or specific findings for VM, and these abnormalities are interpreted in the context of the patient history and examination findings [3].

The aim of this study was to examine and compare the oculomotor test and vHIT findings of patients with VM evaluated in our clinic with the complaint of dizziness with those of a healthy age-matched control group.

## 2. Method

The clinical data of 33 patients (Group 1) who were evaluated between October 2020 and October 2022 and were definitively diagnosed with VM according to the 2018 criteria of the International Headache Society's Headache Classification Committee were retrospectively analyzed. All the patients were aged <65 years with hearing within normal limits.

The study exclusion criteria were defined as not type A tympanogram and/or hearing loss determined in the audiological tests, the presence of BPPV, Meniere disease, neuritis or central vestibular pathology, structural abnormalities determined on brain magnetic resonance imaging (MRI) and/or electroencephalogram (EEG), any psychiatric or neurological disease, or a history of ear surgery. For comparison of the data of Group 1, a control group was formed of 33 healthy adults (23 females, 10 males) (Group 2) who had provided informed consent with the required legal permission (KA21/486) in another previous project. All the patients and control group subjects underwent vHIT, pure tone audiometry, tympanometry, cranial MRI, EEG, and excluding the caloric test, completed the vestibular test battery. The demographic data of age and gender were recorded together with right and left gaze peak velocity (%), accuracy (%), and latency (ms) values in the oculomotor and saccade tests, dynamic positional test results, and the right and left ear lateral, posterior, and anterior semi-circular canal VOR gain and asymmetry values in vHIT. These values were analysed and compared between the groups.

## 3. Audiovestibular Test Protocols

### 3.1. Videonystagmography (VNG)

The pure tone average (PTA) was determined as the average of the 0.5, 1, 2, and 4 kHz air conduction thresholds. A PTA value of <25 dB was accepted as normal. The oculomotor test sequence included spontaneous or positional nystagmus, gaze-evoked nystagmus, and smooth pursuit. Less than 3° nystagmus can be accepted as in the normal range in this test. The stimulus used in the light bar for randomized saccade is a random visual target for 60 seconds at different angles and varying frequencies in the horizontal plane in the range of −20°, 0°, and +20°. All the measurements were recorded of the mean gains of pursuit movements towards left and right of the horizontal jerk stimulus at frequencies between 0.2 and 0.7 Hz. The latency, accuracy and velocity parameters were evaluated on the recorded saccade curve. The VOR gain values of the semicircular canals and the rates of gain asymmetry between the two ears were also evaluated on VNG.

### 3.2. vHIT Test

This test was performed using a vHIT device (Otometrics, ICS Impulse USB, GN A/S Denmark). The patient was seated on a chair and instructed to look straight ahead at a fixed visual target 1 meter in front of the eyes, to avoid blinking and to keep the neck muscles relaxed. An experienced technician administered at least 20 high

velocity, sudden and unseen impulses to the head on each side (at angles of 10–20°, head impulses lasting 150–200 ms, with the highest rate of >150°/s in horizontal head impulse movements and >100°/s in vertical head impulse movements). Using the integral software of the device, the immediate VOR gain was calculated as eye velocity (°/s)/head velocity (°/s). Abnormal vHIT results are defined as immediate VOR gain <0.8 in 60 ms for horizontal canals, or regression VOR gain <0.7 for vertical canals, or when corrective saccades were seen in each semicircular canal. When evaluating the vHIT results, as there was no affected ear in the control group, the difference between the ears was checked in both groups and when there was no difference, the mean VOR value for the left and right ears was used to compare the general status of the canals. When there was a difference between the ears, the values were evaluated in respect of the 'relatively affected' ear.

## 4. Statistical Evaluation

Data obtained in the study were analysed statistically using Statistical Package for the Social Sciences (SPSS) vn. 22.0 software (SPSS Inc., Chicago, IL, USA). Descriptive statistics were stated as mean±standard deviation, median, minimum and maximum values for continuous variables and number (n) and percentage (%) for categorical variables. The variables of both groups showed normal distribution and were homogenous, so the Independent Samples t-test and Pearson Chi-square analysis were used when comparing differences between the groups. The results were examined in a 95% confidence interval. A value of $p<0.05$ was accepted as statistically significant.

## 5. Results

The patients in Group 1 comprised 28 females and 5 males (F/M: 5.6/1), and Group 2 comprised 23 females and 10 males. Patient age was mean 44.73±13.79 years in Group 1 and 47.91±12.92 years in Group 2, with no significant difference determined between the groups ($p=0.97$).

### 5.1. VNG

In the gaze test, nystagmus was observed in 6 (18.18%) patients in Group 1, of which 4 were horizontal and 2 were vertical nystagmus. Gaze nystagmus was not observed in any of the control subjects in Group 2 (Table 1). BPPV was determined as a comorbidity in 5 (13.1%) of the current study patients with VM, and these patients were excluded from the study. In both groups, no latency abnormality was determined in the oculomotor saccade test, with no significant difference determined between the groups (Table 2). The mean velocity was seen to be increased (>400 °/s) in 6 (18.2%) patients in Group 1. Velocity was <400°/s in all the Group 2 subjects. The mean saccade accuracy values were 96±1.23 on the left side and 88.3±1.04 on the right side in Group 1. Although not at a level of statistical significance the right-side accuracy values were determined to be <80% in 4 (12.2%) patients in Group 1 and in 2 (6.06%) subjects in Group 2.

**Table 1.** Abnormal findings in the vestibular tests.

| | | **Group 1**<br>**n (%)** | **Group 2**<br>**n (%)** |
|---|---|---|---|
| Gaze test | Horizontal | 4 (12.2%) | 0 |
| | Vertical | 2 (6.1%) | 0 |
| Saccade test | Velocity (>400 °/s) | 6 (18.2%) | 0 |
| | Accuracy | 4 (12.2%) | 2 (6.1%) |
| | Latency | 0 | 0 |
| vHIT | Gain | 10 (30.3%) | 1 (3.03%) |
| | Asymmetry | 11 (33.3%) | 1 (3.03%) |

### 5.2. vHIT

The vHIT results of both groups, when the left and right lateral canal VOR gains in Group 1 were examined, normal distribution was observed for the lateral canals of both ears (right lateral canal: $t=0.164$, $p>0.05$; left lateral canal: $t=0.158$, $p>0.05$). The mean VOR gain values in Group 1 and Group 2 were seen to be extremely close to each

**Table 2.** Oculomotor saccade test results of groups.

| Saccade Test | | Group 1 Mean±SD | Group 1 Median (Min.–Max.) | Group 2 Mean±SD | Group 2 Median (Min.–Max.) | p# |
|---|---|---|---|---|---|---|
| **Velocity** | **L** | 353.24±48.06 | 348 (282–483) | 350.54±42.28 | 360 (261–417) | 0.81 |
| | **R** | 329.45±47.12 | 322 (257–460) | 327.39±42.27 | 329 (250–417) | 0.85 |
| **Accuracy** | **L** | 96±12.38 | 96 (81–134) | 93.12±7.66 | 91 (79–119) | 0.26 |
| | **R** | 88.3±10.4 | 86 (75–121) | 85.42±5.17 | 85 (76–100) | 0.16 |
| **Latency** | **L** | 199.76±25.13 | 198 (142–257) | 204.36±34.92 | 195 (162–276) | 0.54 |
| | **R** | 200.48±27.78 | 201 (154–272) | 202.18±30.48 | 200 (153–272) | 0.81 |

L: Left ear; R: Right ear; Mean±SD: Mean±Standard deviation; #Independent samples test; Statistical significance p<0.05.

other (Figure 1) and no statistically significant difference was determined (p>0.05). In both groups, no statistically significant difference was determined between the ears in respect of the lateral canal VOR gains (p>0.05) (Table 3). When the VOR gains in both groups were categorised as normal, low, and high, the posterior and anterior VOR gain was determined to be more evidently low (36.3%) on VM, but only the posterior VOR was found to be significantly low (p<0.05).

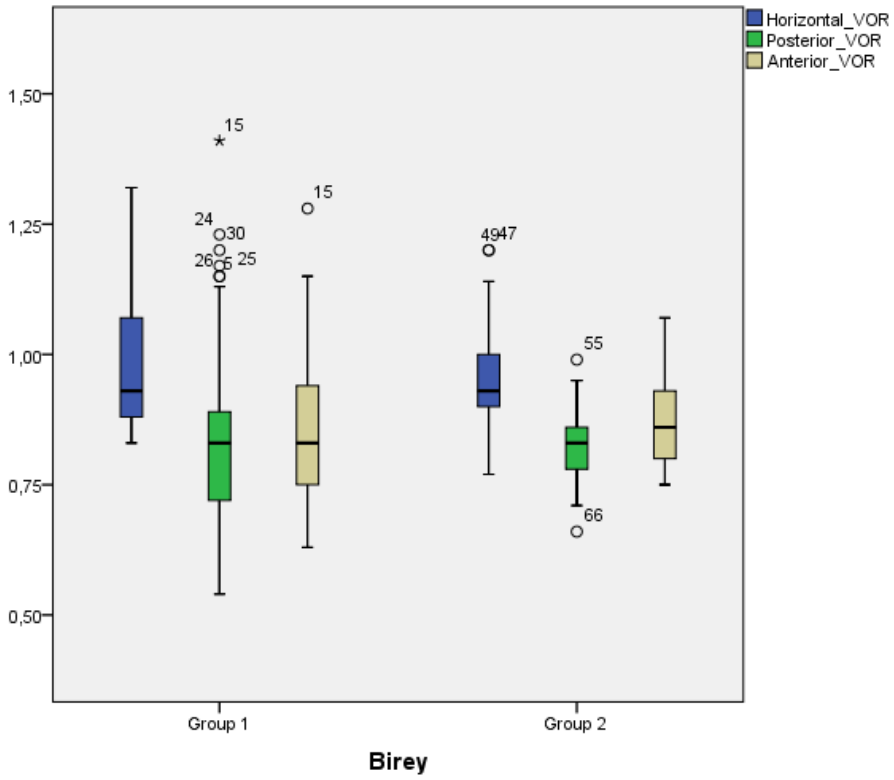


**Figure 1.** The VOR values of the groups.

In Group 1, the rate of asymmetry was 13.57±5.8 for the lateral canal, 10.85±7.7 for the LARP canal, and 9.66±8.2 for the RALP canal. In Group 2, the rate of asymmetry was 12.54±5 for the lateral canal, 9.98±6.42 for LARP, and 8.4±5.2 for RALP. A rate of asymmetry >20% was determined in a total of 6 patients in Group 1; lateral canal (n:4), LARP canal (n:3), RALP canal (n:4), and in 4 patients in Group 2; lateral canal (n:2), LARP canal (n:3). No statistically significant difference was determined between the asymmetry rates in both groups (p>0.05).

**Table 3.** VOR gain results in vHIT.

| | Group 1 | | Group 2 | | |
|---|---|---|---|---|---|
| | **Mean±SD** | **Median (Min.–Max.)** | **Mean±SD** | **Median (Min.–Max.)** | **p#** |
| **Lateral VOR** | 0.99±0.13 | 0.93 (0.83–1.32) | 0.94±0.09 | 0.86 (0.71–1.06) | 0.11 |
| **Lateral VOR Asymmetry** | 13.57±5.8 | 14 (1–27) | 12.54±5 | 13 (3–22) | 0.44 |
| **Posterior VOR** | 0.85±0.21 | 0.82 (0.54–1.4) | 0.82±0.6 | 0.78 (0.7–0.94) | 0.41 |
| **LARP Asymmetry** | 10.85±7.6 | 10 (0–35) | 9.97±6.4 | 9.97 (0–22) | 0.61 |
| **Anterior VOR** | 0.85±0.15 | 0.83 (0.63–1.23) | 0.87±0.8 | 0.89 (0.7–1.15) | 0.59 |
| **RALP Asymmetry** | 9.66±8.2 | 7 (1–35) | 8.4±5.2 | 9 (1–22) | 0.46 |

LARP: Left Anterior Right Posterior; RALP: Right Anterior Left Posterior. Mean±SD: Mean±Standard deviation; Min-Max: Minimum-Maksimum; #Independent samples test; Statistical significance p<0.05.

In Group 1 participants, Left Horizontal (LH) Covert 6.2%, Overt 12.1%, Covert+Overt 18.2%, Right Horizontal (RH) Covert 18.2%, Overt 24.2%, Covert+Overt 18.2%, Left Anterior (LA) Covert 15.2%, Overt 3%, Right Posterior (RP) Covert 12.1%, Overt 18.2%, Covert+Overt 21.2%, Right Anterior (RA) Covert 18.2%, Overt 12.1%, Left Posterior (LP) Covert 12.1%, Overt 12.1%, Covert+Overt 6.1% saccades were observed. Significant difference was determined between the two groups in respect of capture saccades in all planes (p<0.05). It was observed that the presence of capture saccades and VOR gains was statistically similar in both groups (p>0.05). However, in some VM patients, capture saccades were seen despite normal VOR gain levels, but this finding was not statistically significant (p>0.05).

The PR scores of the groups calculated in vHIT are shown in **Table 4**. Although the PR scores in all the canals were higher in Group 1, a significant difference was only determined in the PR score of the right lateral canal.

**Table 4.** Comparisons of the PR scores of the groups.

| | **Group 1 Mean±SD (Min.–Max.)** | **Group 2 Mean±SD (Min.–Max.)** | **p** |
|---|---|---|---|
| L horizontal PR (%) | 7.69±25.4 (0.00–100) | 0 | 0.092 |
| R Horizontal PR (%) | 12.45±29.3 (0.00–100) | 0 | 0.021* |
| L Posterior PR (%) | 2.87±16.2 (0.00–92.0) | 0 | 0.325 |
| P Posterior PR (%) | 8.27±21.6 (0.00–97.0) | 1.15±4.7 (0.00–24.0) | 0.073 |
| L-R Anterior PR | 0 | 0 | |

Mean±SD: mean±standard deviation; Min-Max: Minimum-Maximum.; *Independent Samples t-test; Statistical significance p<0.05.

## 6. Discussion

Vestibular migraine is accepted as the second most common cause of recurrent spontaneous vertigo attacks after BPPV. Lifetime frequency in the general population is approximately 1%. VM constitutes approximately 7% of patients seen in clinics with dizziness, and 9% of patients seen in migraine clinics [15]. In a recent study of young patients referred by doctors to a dizziness clinic, VM was suspected in only 1.8%, but a diagnosis of VM was made in 20.2% [16]. It has been stated that VM is seen 1.5–5-fold more in females than males [15]. It has been suggested that there is a genetic cause of VM and that an autosomal dominant inheritance model is present with less penetration in males [17]. The female to male ratio was determined to be 5.6:1 in the current study. Although VM is accepted as a central vestibular disease [18], peripheral vestibular end organs can also be affected [19]. However, the functional abnormalities and affected characteristics of vestibular end organs are not clear, and there is no clinical test that can reliably predict this disease at an early stage.

BPPV should also be considered in the differential diagnosis because it has often been associated with migraine [4]. The majority of VM patients have been reported to have central oculomotor dysfunctions such as saccadic pursuit, spontaneous or gaze nystagmus, and positional nystagmus, including in the symptom-free period [11, 20]. In a study by, Bir et al. (2003) found electronystagmography abnormalities in 58% of patients with VM. In contrast,

these rates have been reported as 8%--17% in other studies including migraine and vertigo patients [13, 21]. In the current study, central oculomotor dysfunction was determined in 10 (30.3%) and peripheral oculomotor dysfunction in 15 (45.4%) VM patients. These pathologies were pathological gaze in 6 patients and pathological saccade test in 9 patients. In addition, central oculomotor dysfunction (pathological gaze) was determined in 2 (6.06%) control group subjects. Slowing of saccadic pursuit can be observed with the use of drugs affecting the central nervous system, in cases of sleep deprivation and fatigue, in diseases involving the basal ganglia, cerebellar diseases, as well as in conditions such as myasthenia gravis and internuclear ophthalmoplegia [22]. In addition to these conditions, lack of concentration or distraction during saccadic pursuit may have contributed to the pathological results observed in two individuals from the control group.

In a study involving individuals with migraine in the interictally period, the authors reported that the smooth pursuit 0.2 Hz gain and the latency (ms) parameter of the saccade test were statistically different from individuals without migraine, while the velocity parameter was similar between the two groups [23]. Consequently, it was suggested that the presence of mild otoneurological abnormalites was probably due to the effect on the oculomotor function of vestibulocerebellar origin. In the current study, gaze nystagmus was determined in 6 (18.1%) VM patients, although it remained within normal limits (<3°) [23]. In a recent retrospective study [24], the VNG findings of 35 VM patients were examined, and a statistically significant difference was reported in the Dix Hallpike test and vertical smooth pursuit of the VM patients compared to the control group.

It has been stated that maturation of the latency, velocity, and accuracy parameters in the oculomotor randomised saccade test is completed in adolescence and there are no age-related changes. A delay in latency may be associated with the presence of a lesion in the basal ganglions, brain stem, cerebellum, peripheral oculomotor nerves, or the eye muscles [25]. Nystagmus in the positional test was observed in 54 (67.5%) of the VM patients, and spontaneous nystagmus without fixation in 25 (31.3%) [26]. Some type of abnormal oculomotor test result was observed in 82.5% of the VM patients, of which low gain pursuit was determined in 40%, saccadic pursuit in 28.4%, and asymmetric low gain pursuit in 8.8%. Saccadic dissymmetry in the saccade test was reported in 4 (4.9%) patients. In the control group of 40 healthy individuals, low gain pursuit was observed in 17.5%. The results of the current study showed an increase in the velocity parameter of the saccade test in 6 (18.1%) VM patients. The data of two of these patients were excluded from the study as they were determined to have BPPV accompanying VM. In addition, the right-side accuracy values were determined to be <80% in 4 (12.1%) VM patients and in two control group subjects.

As VM is a chronic disorder, it is possible that central oculomotor dysfunction worsens over time. In patients with VM, mild central oculomotor dysfunction with horizontal and/or vertical saccadic tracking may be observed in the asymptomatic range. It was reported that although the degree of central oculomotor dysfunction did not worsen during the follow-up period, its prevalence increased from 20% to 63%, and the presence of central oculomotor dysfunction during follow-up had a positive predictive value of 90.5% for the diagnosis of VM [25]. Central oculomotor dysfunction is valuable in predicting VM in patients with recurrent vertigo attacks, and prophylactic migraine treatment may reduce the development and progression of central oculomotor dysfunction. Attention has also been drawn to the fact that the development and progression of central oculomotor dysfunction can be reduced with prophylactic migraine treatment.

Interictal ocular motor abnormalities may increase over time. The most common abnormality has been reported as central positional nystagmus [12]. The presence of saccadic pursuit in VM has been reported at varying rates (3%–57%) [12, 13, 25]. This can be attributed to differences in test procedures (bedside evaluation versus eye movement recordings) and differences in patient compliance [21]. Therefore, the smooth pursuit test was not included in the current study evaluations as it may not be able to be seen to be pathological in patients with migraine and no vestibular complaints. vHIT includes an objective evaluation of almost all the semi-circular canals separately and is the first test to be performed physiologically (with high frequencies and head movements). The sensitivity of vHIT in VM has been discussed in many studies in literature, but no definitive conclusion has been reached. The reason for this may be the inconsistency of two vHIT parameters (VOR gain and capture saccades). Low VOR gain has been reported in 9–11% of VM patients [27, 28]. When compared with the low sensitivity of VOR gain, saccades have been frequently shown in VM [29, 30]. However, as saccades is a less definable and less measurable parameter than VOR gain, it can be ignored when no significant gain loss is observed in vHIT reports [31]. In addition, although the reason is unknown, vHIT in VM shows less abnormality than the caloric test [27]. In a study

involving 41 patients with VM, cervical and ocular vestibular evoked myogenic potential (cVEMP, oVEMP), vHIT, caloric test and videonystagmography tests were performed. The authors reported that 73% of patients with VM had abnormal vestibular test findings and 42% of patients with VM showed pathologic results in oculomotor tests Abnormal results were reported to be determined at the rate of 32% of the VM patients tested with vHIT [32]. Thus, it was emphasised that abnormal vestibular and oculomotor functions are commonly seen in patients with VM. As it could not be performed on the control group for ethical reasons, the caloric test evaluation was excluded from this study, which could be evaluated as a handicap. Pathological results in the oculomotor tests were determined in 45.4% of the VM patients.

Oculomotor tests can be obtained pathologically in VM patients. It is reported that refixation saccades were found in 52.3% of VM patients and 10.2% of healthy individuals who underwent vHIT testing during a vertigo attack in VM patients [33]. The authors emphasized that the VOR gain values of the VM patients during a vertigo attack were no different from those of the healthy control subjects, but there was a higher number of capture saccades showing peripheral vestibular involvement in the VM patients. In the current study, the VOR gains in all the semicircular canals were lower in the VM patients than in the control group, but the difference was not statistically significant.

The PR score is one of several quantitative saccadic parameters that can show vestibular improvement [34]. In a retrospective study of 11 patients with VM, it was reported that the saccade detection rate on vHIT was higher than the abnormal VOR gain. The authors suggested that the VOR gain combined with the PR score would provide an appropriate discrimination between common vertigo disorders and that the PR score is more sensitive than the gain in assessing physiologic status, vestibular compensation and disease progression [35]. In the current study, significant PR score elevation was determined in the VM patients.

In our retrospective study, we consider the absence of the cervical vestibular evoked myogenic potentials test and the caloric test as a limitation. In future studies on vestibular migraine, these tests can be evaluated together with the vHIT and VNG test battery.

## 7. Conclusion

The interaction between VM and vestibular disorders may be a difficult scenario for diagnosis and treatment, and especially in patients with recurrent vertigo, the diagnosis must be reviewed, and multidirectional otoneurological evaluation is mandatory. Research is ongoing to increase the effectiveness of the highly tolerable VNG test battery and vHIT in reaching a diagnosis, especially in VM patients who may also have comorbid vestibular problems during the attack period. In the vestibular evaluations of VM patients, especially during an attack, oculomotor saccade abnormalities, reduced VOR gain, and/or capture saccades may be often seen. These changes can be caught with careful examination by both laboratory and clinical practitioners of VNG and vHIT outputs especially in respect of capture saccades. It can be considered that combining some data of the tests applied in this study with the patient history will provide a valuable tool in VM cases that are difficult to diagnose.

## Author Contributions

Conceptualization, N.T. and A.F.B.; methodology, N.T.; software, Ö.K.; validation, N.T., Ö.K. and A.F.B.; formal analysis, N.T.; investigation, N.T.; resources, N.T. and Ö.K.; data curation, N.T., Ö.K. and A.F.B.; writing—original draft preparation, N.T.; writing—review and editing, A.F.B.; visualization, Ö.K.; supervision, A.F.B.; project administration, N.T.; funding acquisition, A.F.B. All authors have read and agreed to the published version of the manuscript.

## Funding

The authors declared that this study has received no external financial support.

## Institutional Review Board Statement

Approved by the Ethics Committee of Health Sciences (Approval No: KA22/452 and 11 Nov 2022 date of approval).

## Informed Consent Statement

Written informed consent has been obtained from the patients who agreed to take part in the study to publish this paper.

## Data Availability Statement

The data will be available upon request to the corresponding author.

## Conflicts of Interest

The authors have no conflicts of interest to declare.

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