



Video head impulse test (vHIT) in migraine dizziness

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ABSTRACT

Background and objective: Migraine is an extremely prevalent primary headache disorder that frequently associates parallel symptoms such as dizziness, tinnitus and hearing loss. Our aim is to investigate differences in video head impulse (vHIT) results with patients suffering from vestibular migraine (VM) and healthy people, taking into consideration mean values of vestibule ocular reflex (VOR) gain, occurrence of the compensatory saccades latency and amplitude. According to the results, determine the usefulness of vHIT in vestibular migraine diagnostics.

Methods: A total number of 120 subjects were enrolled in the study, 80 of them were vestibular migraine patients (VM), while the other 40 were a control group of age matched healthy subjects. History was taking during the evaluation; videonystagmography and the video head impulse test were done.

Results: The rate of saccades is much more higher in the VM group compared to the healthy subjects group, only 7.5% of the VM group showed a low VOR gain with compensatory saccades denoting a vestibular deficit.

Conclusion: The refixation saccades are an important sign that could underlie different vestibular problems. vHIT result can contribute to the completion of full mosaic of vestibular migraine diagnostics.

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1. Introduction

The vestibule ocular reflex (VOR) is a simple, accurate, and fast reflex arc with a latency of 8–12 ms (Maranhão and Maranhão-Filho, 2012), and it consists of three main neuronal arcs (Bronstein and Lempert, 2009). The first goes from the vestibular end organs to the vestibular nuclei in the brain stem, the second from the latter to the oculomotor nuclei, and the third reaches the extraocular muscle. Its function is to maintain focus on a stationary target during head movement (Maranhão and Maranhão-Filho, 2012). The flocculus in the cerebellum is essential for the VOR, because it is the part of the brain responsible for the integration of information from the labyrinth, the visual cortex, and the muscles involved in head movements (Balaban and Watanabe, 1984) in order to facilitate rapid extraocular muscle responses and enable a clear image of the fixation target (du Lac et al., 1995).

Like any neural circuit in the brain, the VOR requires a positive feedback system in order to accommodate behavioral changes. Theories ascribe the site of learning for the VOR to the flocculus in the cerebellum (Ito, 1972, 1982) or the brain stem (Lisberger, 1994), with the latter now being the more widely accepted of the two. The complex theoretical pathophysiological link between migraine and

the vestibular system (Bisdorff et al., 2010) is thought to be a combination of neurological and vascular events (Tajti et al., 2011). These may include cortical spreading depression (CSD) and the activation of the trigeminovascular system with neurogenic inflammation, and the interaction of these events and their sequence is still not clear (Dalkara et al., 2006). However, CSD can not explain the peripheral vestibulopathy that occurs during an acute attack of vestibular migraine (Boldingh et al., 2011). A second more logical proposition is the trigeminovascular theory, which holds the vasoactive neuropeptide, innervate the labyrinthine vessels, which may explain the peripheral vestibular deficit in VM patients (Seemungal et al., 2015). The brain uses the feedback system to compensate for any gaze or prediction error occurring via the central creation of saccadic eye movements independent of VOR gain.

vHIT can show: (1) VOR gain, which is the ratio between head and eye velocity. (2) Any compensatory eye movement during head movement (covert saccades) or after its stoppage (overt saccades). (3) The time required to correct any gaze or prediction error and to fixate on a target. (4) The vestibular–saccades interaction mechanism. In VM patients who are neurologically and otologically free of symptoms except for migraine headache, the vHIT may have a specific profile that might facilitate a better understanding of VM pathophysiology.

2. Materials and methods

This was a case–control observational study that was conducted

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at the Audio Vestibular Clinic of Alexandria University Main Hospital in Egypt, from 01 August 2015 to 31 July 2017. The relevant institutional review board approved the study. All the patients provided written informed consent to participation.

2.1. Subjects

There were two groups in the study, a vestibular migraine (VM) group and a group of healthy subjects. The VM group consisted of 80 consecutive patients with a mean age of 39.06 years (SD 7.43 years), of which 69 (86.3%) were female and 11 (13.8%) were male. All patients met the Barany criteria for definite vestibular migraine (Lempert, 2012). The healthy group (n = 40) consisted of subjects who reported no dizziness, and had a mean age of 36.95 years (SD 6.65 years), of which 27 (67.5%) were female and 13 (32.5%) were male. All VM patients with central pathology other than migraine were excluded from the study.

2.2. Methods

A detailed history was obtained from each patient via a flexible structured interview. Pure tone threshold sensitivity was assessed. Videonystagmography (VNG) examination using an ICS Otometric System was performed. All VM patients were examined within 1 week of their last dizzy spell. Spontaneous eye movements with and without visual fixation in the neutral eye position and positioning manoeuvre-based method were assessed.

The oculomotor tests performed included the random paradigm saccade, the tracking test, the optokinetic nystagmus (OKN) in the horizontal plane at 20°/sec target speed test, and the assessment of gaze-evoked nystagmus via a red LED light bar. VOR evaluation was performed using an ICS Impulse 3-Dimensional vHIT system (GN Otometrics, Taastrup, Denmark). In this test, the patient wears tight-fitting goggles equipped with a high velocity camera and a mirror to reflect the right eye image, and the fixation target is a dot on the wall 1-m from the patient. The same right-handed physician performed all the vHIT examinations in the study. Calibration was achieved via laser dots on which the subject had to fixate for 1 s in each direction. Subjects were instructed to relax their neck muscles, to not blink, and to keep their eyes open to the best of their ability, to enable the software to maintain focus on their pupil.

Lateral canal stimulation was achieved via a passive lateral head impulse, with 10–20° and a peak velocity of 200°/sec, while the head was in a neutral position inclined downwards by approximately 20°. Vertical canal stimulation was performed while the subject's head was rotated to the left for right anterior left posterior stimulation, and to the right for left anterior right posterior stimulation, 30–40° from the fixation target. Then, unpredictable passive backward and forward movements of the head of 10–15° amplitude with a peak head velocity of 150°/sec. At least 20 impulses were performed for each canal. vHIT analysis depends mainly on VOR gain and the presence of refixation saccades. Normal VOR gain is specified by the manufacturer (ICS otometric vHIT) as > 0.8 for the lateral canals and > 0.7 for the vertical canals (Yollu et al. (2016)). Refixation saccades are categorized as either covert saccades (occurring during a head movement) or overt saccades (occurring after a head movement) (Yollu et al. (2016)).

2.3. Descriptive statistics

A comprehensive data collection sheet was used for each patient, including details of the patient's history, VNG examination results, and vHIT results. Data analysis was performed using IBM SPSS version 22. The Mann–Whitney test was used to compare mean VOR gain in the two groups.

3. Results

3.1. History analysis

Forty-one of the 80 patients in the VM group (51.3%) were classified as sufferers of migraine with aura (visual aura), 30 (37.5%) were classified as “migraine only”, and 9 (11.3%) were classified as chronic migraine patients according to the international headache society (IHS) (2013). Of the total of 80 migraine patients, 67 (83.8%) complained of true vertigo. Forty-two (62.7%) of these 67 patients complained of spontaneous vertigo, 25 (37.3%) complained of positional vertigo, and the vertigo lasted for seconds in 31.3%, minutes in 41.3%, and hours in 11.3%. Attacks of vertigo were associated with other intermittent symptoms relating to dizziness such as spinning in 38.8%, unsteadiness in 31.3%, head motion intolerance in 15%, and chronic dizziness in 13.8%. Thirteen out of 80 (16.3%) complained of other dizziness complaints as spinning and unsteadiness. Migraines had occurred within 1 week of the examination in 38 patients (47.5%), within 1 month in 27 (33.8%), and over 1 month in 15 (18.8%).

3.2. VNG test results

Of the 80 patients in the VM group, 54 (67.5%) exhibited nystagmus in positional testing, and 25 (31.3%) exhibited spontaneous nystagmus in fixation testing with vision denied. The nystagmus was horizontal in 40/80 patients (50.0%), up beating in 33 (41.3%), torsional in 4 (5%), and down beating in 3 (3.8%). Of the 80 VM patients, 66 (82.5%) exhibited some form of abnormal oculomotor test result. Of these, 32 patients (40%) had low gain pursuit, 23 patients (28.4%) had saccadic pursuit and 7 patients (8.8%) had asymmetric low gain pursuit. The saccade test showed that 4 patients (4.9%) had saccadic dysmetria. Four patients (4.9%) showed asymmetric OKN.

Of the 40 healthy subjects, 7 subjects (17.5%) showed low gain pursuit test. No gaze nystagmus was observed in both study groups.

3.3. Video head impulse test analysis

3.3.1. VOR gain

Table 1 shows the mean VOR gain in each semicircular canal in both groups. The right head impulse exhibited higher VOR gain than the Left for the lateral and anterior canals in both groups. In the migraine group, the VOR gains in the lateral, posterior, and

Table 1

The mean VOR gain for each semicircular canal in the VM group and in normal healthy subject.

| | Cases (n = 80) | Control (n = 40) | U | P |
|----------------------|----------------|------------------|--------------------|---------------------|
| LL | | | | |
| Mean ± SD. | 0.84 ± 0.13 | 0.89 ± 0.15 | 1278.0 | 0.072 |
| Median (Min. – Max.) | 0.87 (0.5–1.1) | 0.89 (0.4–1.3) | | |
| LA | | | | |
| Mean ± SD. | 1.1 ± 0.3 | 1.2 ± 0.3 | 1508.0 | 0.607 |
| Median (Min. – Max.) | 1(0.5–1.8) | 1.2(0.8–1.8) | | |
| LP | | | | |
| Mean ± SD. | 1.2 ± 0.3 | 1.2 ± 0.3 | 1526.50 | 0.680 |
| Median (Min. – Max.) | 1.2(0.4–1.8) | 1.2(0.8–1.7) | | |
| RL | | | | |
| Mean ± SD. | 0.9 ± 0.3 | 1 ± 0.3 | 1450.50 | 0.403 |
| Median (Min. – Max.) | 0.9(0.4–1.8) | 0.9(0.4–1.8) | | |
| RA | | | | |
| Mean ± SD. | 1.2 ± 0.3 | 1.3 ± 0.3 | 1412.0 | 0.293 |
| Median (Min. – Max.) | 1.2(0.3–1.8) | 1.2(0.9–1.8) | | |
| RP | | | | |
| Mean ± SD. | 0.98 ± 0.25 | 1.10 ± 0.27 | 978.0 ^a | <0.001 ^a |
| Median (Min. – Max.) | 0.89 (0.8–1.8) | 1.03 (0.4–1.8) | | |

Quantitative data was expressed in mean ± SD, median (Min. - Max.) and compared using **Mann Whitney test**.

^a Statistically significant at p ≤ 0.05.

vertical canals were lower than they were in the healthy group although not statistically significantly. In the migraine group, the VOR gain in the right posterior canal was significantly lower than it was in the healthy group.

3.3.2. Refixation saccades

Refixation saccades were present with normal VOR gain in 3 subjects (7.5%) in the control group without any dizziness complaints. The amplitude of these saccades was $>50^\circ/\text{sec}$ with latency ranging from 100 to 250 ms. The same type of saccades were present in 15 subjects in the migraine group. Refixation saccades were present in the lateral plane with low VOR gain in 6 patients in the VM group, with latency ranging from 100 to 350 ms (Table 2).

4. Discussion

Vestibular examination of VM patients during a dizzy spell usually suggests dysfunction in the central vestibular system, either in the vestibular nuclei in the brain stem or in the multisensory vestibular cortex (Harno et al., 2003). In the current study, 67.5% of the VM group exhibited a non-paroxysmal type of positional nystagmus. It has been reported that the horizontal type of this nystagmus is caused by abnormal vestibular signals from the lateral canals. The vertical type of this nystagmus is thought to be due to lesions in the vestibulocerebellum (flocculonodulus) leading to disinhibition of the vestibular nuclei in the brainstem (Kim et al., 2005). It is unlike benign paroxysmal positional nystagmus. The latter has a paroxysmal form, has a latency, and is usually associated with a sensation of vertigo (Dumas et al., 1994). The majority of patients in the VM group exhibited abnormalities in the oculomotor tests. These results are concordant with studies reporting saccadic pursuit in 50% of migraine sufferers (Cutrer and Baloh, 1992).

In the majority of VM patients (73%), VOR gain was normal in the vHIT, and refixation saccades were not present. This suggests that the VOR reflex arc is intact in VM patients. In the current study, the mean VOR gains of the rightward head impulses were higher than those of the leftward head impulse in the horizontal and anterior semicircular canals. It has been suggested that monocular analysis by the right eye is the cause of this asymmetry and/or the medial rectus muscle is stronger than the lateral rectus muscle with regard to inducing eyeball movement (Weber et al., 2008). In contrast, 26% of the VM group exhibited abnormal vHIT results. This percentage exceeds that reported by Kang et al. (2016), who detected VOR abnormalities in approximately 11% of VM sufferers. In the current study, 26% of the VM group exhibited vHIT abnormalities, in the form of refixation saccades with normal VOR gain (18%), and low VOR gain with refixation saccades in (7.5%). The presence of covert saccades despite normal VOR gain is a correction of eye drifts to the target position. They exhibited some variations from saccades described in unilateral or bilateral vestibular loss, including saccades associated with normal VOR gain, covert saccades, saccadic amplitude of more than half the amplitude of head movement, and saccades occurring in the lateral head impulses only.

It has been suggested that the presence of refixation saccades with normal VOR gain is part of the process of aging of the vestibular system (Anson et al., 2016). In the current study, 3/40 (7.5%) of the healthy subjects showed this type of saccades without any dizziness

complaints. Yollu et al. (2016) reported significantly higher covert saccades in migraine patients compared to healthy controls, and suggested that these saccades were associated with the connection between the trigeminal nerve and the vestibular nuclei. These covert saccades could be explained by a disturbance in the spontaneous neural firing rate at the level of the vestibular nuclei (disturbed inhibitory input from the cerebellar flocculus to the vestibular), as a part of the migrainous central spreading depression. However, low VOR gain and refixation saccades were seen in 7.5% of the VM patients in the current study. This finding suggests vestibular deficits.

5. Conclusion

Refixation saccades are more important and reliable in the interpretation of vHIT than VOR gain. vHIT profiles in VM patients; suggesting that vestibular migraine pathophysiology is central in origin, and may be associated with vestibular deficits at the level of brainstem. vHIT results can complement other vestibular tests, facilitating a more complete set of vestibular migraine diagnostics.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.joto.2017.12.002>.

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Table 2

The frequency of saccades depending on the VOR gain in the VM group.

| | Frequency | Percent |
|-------------------------------|-----------|---------|
| No saccades | 59 | 73.8 |
| Saccades with normal VOR gain | 15 | 18.8 |
| Saccades with low VOR gain | 6 | 7.5 |