Clinical Vestibular Finding and Cognitive Performance Among Vestibular Migraine Patients

Original Article

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ABSTRACT

Background: Patients with vestibular migraine (VM) describe having both vestibular and migraine symptoms. Patients with migraines have been known to have imbalance, intolerance to head movement, rotational vertigo, and positional vertigo as dizzy symptoms.

Objective: To evaluate patients' cognitive function and characterize the clinical vestibular characteristics of vestibular migraine.

Subjects and Methods: The 10^6 people in this study were split into two groups: 53 adults with VM in the cases group and 53 age and gender-matched normal adults in the control group. Every subject underwent a comprehensive history taking, a basic audiological evaluation, video nystagmography (VNG), water-based caloric irrigation, the Mini-Mental State Examination (MMSE), the Subjective Visual Vertical Test (SVV), the Cervical Vestibular Erupted Myogenic Potentials Test (c-VEMP), and the Symbol Digits Modality Test (SDMT).

Result: In eighteen (34%) of the cases with upbeating tortional nystagmus in the Dix-Hallpike position (unilateral and bilateral), positional nystagmus was the most common finding during the VNG test. Nine cases (17%) of horizontal nystagmus had positional nystagmus, which was either constant in direction or changed with changes in head position. Seven instances (15%) exhibited unilateral caloric hypofunction during caloric testing. The static SVV values at the upright posture showed a statistically significant difference between the two groups (P=0.001). When VM patients were compared to their normal controls, there was a statistically significant delay in P13, N23 latency, and amplitude. In VM patients, C-VEMP anomaly represents the vestibulospinal reflex consequences of migraine. When compared to the control group, the MMSE and SDMT results indicated lower scores, which indicate cognitive impairment.

Conclusions: Vestibular migraine (VM) cannot be diagnosed with a typical fixed profile in vestibular testing; instead, patients with VM exhibit spatial disorientation and dysfunction of the otolithic pathway. The duration of migraines is an unavoidable risk factor that may be linked to a decline in cognitive function.

Key Words: CVEMP, MCI, MAD, MMSE, SDMT, SVV.

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BACKGROUND

Chronic migraine is a disorder marked by recurrent headache attacks lasting four to seventy-two hours, with a pulsating quality. The headaches can be moderately to severely intense, aggravated by normal physical activity, and frequently accompanied by nausea, vomiting, photophobia, or phonophobia. The headaches can also be associated with aura or not[1].

The phrases vestibular migraine, migrainous vertigo, migraine-associated vertigo, and migraine-associated balance disturbance are most frequently used to characterize the co-occurrence of migraine and vestibular symptoms. Vestibular migraine diagnostic criteria have been developed by the International Headache Society (IHS), the Bárány-Society (International Society for Neuro-Otology), ENT doctors, and other experts[2].

Vertigo in vestibular migraineurs can occur either spontaneously or in response to a change in position. It can also be rotational or non-rotational[3].

Positional or illusion of movement vertigo can develop from vertigo when there are gait abnormalities and heightened sensitivity to motion, especially head motions[4].

"The mental action or process of acquiring knowledge and understanding thought, experience, and the senses" is the definition of cognition[5].

The ability to store multiple pieces of information simultaneously and retrieve them quickly when needed for

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further processing is known as "brain working memory." A wide range of cognitive problems result from working memory injury, and the patient is unable to use his own knowledge to think correctly in various contexts[6].

Neuropsychological testing can be used to learn more about the patient's memory, executive functioning, behavior, and cognitive abilities[7].

In migraineurs, subjective cognitive deterioration is not unusual. Even while cognitive symptoms are not thought to be among the primary symptoms of migraine, many migraineurs frequently report intellectual impairment, especially memory and attention problems. During the premonitory and headache phases of a migraine episode, as well as the postdrome, cognitive problems are common. Outside of migraine attacks, some migraineurs also report having cognitive problems[8].

Treatments for acute attacks don't always work to reduce cognitive symptoms. Disability from migraine attacks is also a result of cognitive dysfunction, namely executive function impairment. Indeed, in terms of severity and attack-related handicap, cognitive symptoms came in second only to pain, making them a pertinent target for migraine attack management[8].

SUBJECTS AND METHODS

The Otorhinolaryngology department and Cairo University's Ethics Committee accepted this case control study (number-131-2021). It involved 10^6 Egyptian participants, split up as follows: The study group consisted of 53 cases, ages ranging from 23 to 58 years. Of the patients, 26 (49.1%) were male and 27 (50.9%) were female, with dizziness-related migraine; all patients met the inclusion criteria. The control group, consisting of 53 subjects, was made up of healthy individuals whose age and gender matched those of the cases.

Inclusion criteria

Adults of either gender who can move their neck freely and have normal vision or corrected vision of 1 or higher, or who meet the diagnostic criteria for a vestibular migraine with or without aura (ICHD-3, 2013) and may be diagnosed with a vestibular migraine.

- Exclusion criteria:
- People who are older than 65.
- Any prior otological or neuromuscular illness history or present.
- Individuals having lesions in the ocular muscles or oculomotor nerves.

Individuals who have been diagnosed with cognitive impairments (such as dementia) or mental health issues (such as anxiety and depression)

- Individuals with any general illness, such as diabetes, that is known to impair balance.

Methods

A) Equipment

1. Sound treated room (Amplisilence Model E).
2. One-channel audiometer Itera II (Madsen Corporation, USA), calibrated according ISO standards 389-1. Head phones TDH 39 and bone vibrator radio-ear B71.
3. Tympanometry: Zodiac 901 (Madsen Corporation, USA). Calibrated according IEC 60645-5.
4. Evoked potentials system: Eclipse (Interacoustic-Denmark), (Neurosoft Ltd, Russia).
5. Video-nystagamography (cyclope –Indian) .

B) Procedure

a) On a single clinic visit, each study participant underwent the following tests

1. Complete history taking(complete history of migraine attacks including age at which attacks begin, radiation, frequency, side, length of attack, severity measured on a visual analog scale) & a comprehensive history of vertigo include duration of the attack, frequency, character, severity & history of general diseases)
2. Otological examination: to rule out diseases of the middle or external ears.
3. Basic Audiological Assessment: a) Pure Tone Audiometry which uses pulsed stimulus to perform PTA for octave frequencies 250–8000 Hz for air conduction and 500–4000 Hz for bone conduction.

b) Using Arabic spondaic words[9], determine the Speech Recognition Threshold (SRT).Using phonetically balanced Arabic words, the word discrimination score (WDS) was calculated.

c) Immittancemetry: single-frequency tympanometry evaluating the ipsilateral and contralateral auditory reflex threshold at frequencies of 500, 1000, 2000, and 4000 Hz using a probe tone of 226 Hz.

4. Videonystagmography (VNG): All patients underwent VNG examinations during an acute episode of vertigo, which was not always accompanied by a headache.

Recording VNG subtests in order to identify any abnormalities in gaze or oculomotor testing, as well as to rule out peripheral vestibular lesions. Included are the following: location tests, positioning tests (to rule out BPPV), occulomotor testing (smooth pursuit, saccade,

and optokinetic tests), spontaneous nystagmus, and caloric testing (to rule out peripheral vestibulopathy).

5. Vestibular Evoked Myogenic Potentials (VEMP):

Electrode montage

Active electrodes are positioned across the middle or upper part of the SCMs during the same session as the VNG testing. According to Sheykholeslami *et al*. (2000), reference and ground electrodes are positioned above the upper sternum and forehead midline, respectively.

Recording and stimulus parameters

The ER-3A insert earphones were used to provide a 500 Hz tone burst stimulus, which was presented monoaurally at an intensity of 90 dBnHL. The stimulus was presented with a rise time of 1 ms, a fall time of 1 ms, and a plateau duration of 2 ms. The strength of the stimulus was 95 dBnHL. 5 Hz was the stimulus rate. The 1-1000 Hz filter produced at least 200 sweeps. 50 milliseconds was the analysis window. To guarantee repeatability, averaged signals from two trials were acquired.

Depending on whether the P13-N23 biphasic response was present or missing, VEMP responses were classified as either present or absent. if the P13-N23 biphasic response is present or not. The following parameters were assessed for the cVEMP response: P13 latency (measured in milliseconds), N23 latency (measured in milliseconds), P13-N23 peak to peak amplitude (measured in ultraviolet light), and inter-aural amplitude difference ratio (IAAD).

Subjective Visual Vertical Test

Following VEMP and VNG testing, at the same visit. The patient positioned the vertical visual bar stimulus produced by a laser beam in a dark room using a remote control. Meanwhile, the light band position divergence (measured in angles) from the gravitational vertical was recorded by the computer. The computer automatically determined the means deviations after the test was run six times.

Symbol Digits Modality Test (SDMT)[35]

The SDMT is a cheap, easy test. This series of nine symbols is a great tool for physicians to evaluate for organic cerebral dysfunction. The test subject is given a sheet of paper with the key printed on top (nine abstract symbols and nine matching integers). Throughout the test, the individual has access to the key. Below the key is a list of 120 symbols, each printed as a square. The squares with symbols on them are topped by empty squares. For ninety seconds, patients are instructed to try writing the proper number under the matching sign as quickly as they can.

Mini-Mental State Examination (MMSE)[36]

Is a tool that can be used as a screening tool to distinguish patients with normal cognitive function from those who have cognitive impairment. According to Pangman *et al*.[10], the MMSE is a fully structured scale with 30 points divided into 7 categories: orientation to the place, orientation to the time, registration, attention and concentration, recall, and language.

Statistical methods

- The statistical software for social science (SPSS version 24) was utilized for data analysis, and Microsoft Excel 2013 was used for data entry.
- The summary of normal quantitative data was based on simple descriptive statistics (arithmetic mean and standard deviation); the summary of abnormal quantitative data was based on median and interquartile range; and the summary of qualitative data was based on frequencies.
- Cross tabulations show bivariate relationships, and proportion comparisons was carried out using Fisher's exact tests and the chi-square test as necessary.
- For comparing quantitative data that was normally distributed, T-independent was utilized, and for skewed data, Mann-Whitney.
- To determine statistical significance, the *P value* was computed; a value of less than 0.05 will be regarded as statistically significant .
- The Pearson correlation coefficient was used to determine whether there was a significant link between the quantitative variables. As R is less than 0.5, moderate when R is between 0.5 and 0.7, and strong when R is greater than 0.7, the correlation is poor.

RESULTS

In this case control study, 10^6 participants were involved. 53 participants with VM and 53 age and gender matched normal subjects were separated. (Tables 1,2).

According to (Table 3), the VNG findings indicate that during oculomotor testing, 93% of individuals had normal oculography tests and 7% of cases had anomalies (such as low gain and asymmetry in pursuit and optokietic and delayed latency in saccade).

According to (Table 4), this study revealed that nine cases (17%) had positional nystagmus (in the form of horizontal and vertical upbeating nystagmus), seven cases (15%) had unilateral canal weakness caloric test, and eighteen cases (34%) had postioning nystagmus (in the form of upbeating tortional at Dix-Hallpike Position). Regarding the positioning and positioning test as well as the caloric test, there was a highly statistically significant difference between the patients and the control group.

There was no statistically significant variation in the VNG findings for the intensity of the migraines as shown in (Table 5)

Regarding VEMP, (Table 6) demonstrates statistically significant variations in p13 latency in the right ear and N13-P23 amplitude in the left ear between patients and controls.

However there was no statistically significant variation in the C-VEMP findings as regards the intensity of the migraines as shown in (Table 7)

However in (Table 8) indicates that there was a highly statistically significant difference in SVV tilt in Rt CW and CCW tilts between the patients and the normal controls.

Concerning SVV tilt result, there was no significant difference in SVV tilts regarding the severity of the migraine as shown in (Table 9)

Considering cognitive Assessment finding in This study, there was highly significance difference between MMSE score and SDMT scores in cases compared to control group (*p value* < 0.001) as shown in (Tables 10,11)

While there were significance correlation of MMSE and SDMT scores in the cases regarding the onset of migraine duration as shown in (Figure 1)

Tables (1-2): Age and gender distribution among cases and control groups.

	Cases				Control				*P value*
	Mean	SD	Minimum	Maximum	Mean	SD	Minimum	Maximum	
Age	45.26	9.37	23.00	58.00	42.64	10.22	22.00	59.00	0.171

Chi square test ** Unpaired t test P value is significant when $P < 0.05$

Count		Cases		Control		*P value*
		%	Count	%		
Gender	F	27	50.9%	27	50.9%	1
	M	26	49.1%	26	49.1%	

Chi square test ** Unpaired t test P value is significant when $P < 0.05$

Table 3: Pursuit, Saccade and Optokientic findings in cases (n=53).

Count		Cases N=53	
		%	
Pursuit	Normal	49	93%
	Abnormal	4	7%
Saccade	Normal	49	93%
	Abnormal	4	7%
Optokinetic	Normal	49	93%
	Abnormal	4	7%

Table 4: Comparison between cases and control regarding positioning, positional and caloric test

Count		Cases N=53		Control N=53		*P value*
		%	Count	%		
Postioning test	Abnormal	18	34.0%	0	0.0%	< 0.001*
	Normal	35	66.0%	53	100.0%	
Positional test	Abnormal	9	17.0%	0	0.0%	0.003*
	Normal	44	83.0%	53	100.0%	
Caloric test	Abnormal	14	26.4%	0	0.0%	< 0.001*
	Normal	39	73,6%	53	100%	

**P-value* <0.05 is statistically significant.

Table 5: Comparison of saccade, pursuit, optokinetic, positional, positioning and caloric finding regarding severity of the migraine.

		Severity of the migraine				
		Severe		Not severe		*P value*
		Count	%	Count	%	
Pursuit	Normal	30	96.8%	19	86.4%	0.295
	Abnormal	1	3.2%	3	13.6%	
Saccade	Normal	30	96.8%	19	86.4%	0.295
	Abnormal	1	3.2%	3	13.6%	
Optokinetic	Normal	30	96.8%	19	86.4%	0.295
	Abnormal	1	3.2%	3	13.6%	
Postioning	Positive	13	41.9%	5	22.7%	0.146
	Negative	18	58.1%	17	77.3%	
Positional	Positive	4	12.9%	5	22.7%	0.464
	Negative	27	87.1%	17	77.3%	
Caloric	Abnormal	6	19.4%	8	36.4%	0.166
	Normal	25	80.6%	14	63.6%	

**P-value* <0,05 is statistically significant.

Table 6: Comparisons of cVemp result between cases and controls.

	Cases (N=53)				Control (N=53)				*P value*
	Mean	SD	Minimum	Maximum	Mean	SD	Minimum	Maximum	
P13 latency (RT)	14.08	2.38	11.00	21.40	12.67	1.02	11.00	15.30	< 0.001*
N23 latency (RT)	23.05	3.12	18.00	35.90	23.39	1.01	22.00	27.40	0.451
N13-P23 amplitude (RT)	42.05	12.02	22.50	191.90	36.38	7.65	28.80	305.00	0.301
P13 latency (LT)	14.11	2.41	11.00	22.30	13.13	1.54	11.00	16.30	0.054
N23 latency (LT)	23.99	1.86	22.00	29.50	23.72	1.67	19.70	27.40	0.431
N13-P23 amplitude (LT)	35.98	8.57	20.00	84.60	31.97	2.09	28.10	36.70	0.002*

**P-value* <0.05 is statistically significant.

Table 7: Comparison between c-VEMP results in cases regarding severity of the migraine disease.

	Severity of migraine disease				
	Severe		Not severe		*P value*
	Mean	Standard Deviation	Mean	Standard Deviation	
P13 (Rt)	13.63	1.96	14.72	2.78	0.100
N23 (Rt)	22.88	2.39	23.29	3.98	0.648
N13-P23(Rt)	40.69	13.12	43.96	10.27	0.335
P13 (Lt)	14.21	2.75	13.98	1.88	0.742
N23 (Lt)	24.05	1.93	23.90	1.80	0.762
N1-P1 (Lt)	36.94	8.67	34.63	8.43	0.337

P value is significant when $P < 0.05$.

Table 8: Comparsion of SVV between the cases and the control .

	Cases (N=53)				Control (N=53)				P value
	Mean	SD	Minimum	Maximum	Mean	SD	Minimum	Maximum	
Right CW tilt	0.35	0.25	0.142	0.98	0.42	0.23	0.20	0.90	< 0.001*
Right CCW tilt	0.26	0.21	0.24	0.90	0.37	0.16	0.013	0.89	< 0.001*
Left CW tilt	0.32	0.18	0.15	0.90	0.24	0.17	0.23	0.60	0.051
Left CCW tilt	0.36	0.22	0.20	0.90	0.32	0.18	0.12	1.00	0.460

**P-value* <0.05 is statistically significant.

Table 9: Comparison between SVV test finding in the cases regarding severity of the disease

	Severity				
	Severe N=35		No V=18		*P value*
	Mean	Standard Deviation	Mean	Standard Deviation	
Right CW tilt	0.35	0.21	0.31	0.19	0.508
Right CCW tilt	0.26	0.12	0.27	0.21	0.373
Left CW tilt	0.32	0.19	0.33	0.18	0.328
Left CCW tilt	0.36	0.22	0.30	0.23	0.948

**P-value* 0.05 is statistically significant.

Table 10: Comparison between cases and control regarding MMSE and SDMT scores.

	Cases				Control				*P value*
	Mean	SD	Minimum	Maximum	Mean	SD	Minimum	Maximum	
MMSE	25.04	3.09	19.00	29.00	30.00	0.00	27.00	30.00	< 0.001*
SDMT	37.83	5.15	27.00	48.00	50.00	0.00	45.00	50.00	< 0.001*

**P value* is significant when $P < 0.05$

Table 11: Comparison between MMSE and SDMT scores in the cases regarding severity of the disease.

	Severity				
	Severe N=35		No n=18		*P value*
	Mean	Standard Deviation	Mean	Standard Deviation	
MMSE	25.42	2.98	24.50	3.23	0.290
SDMT	38.68	5.02	36.64	5.22	0.157

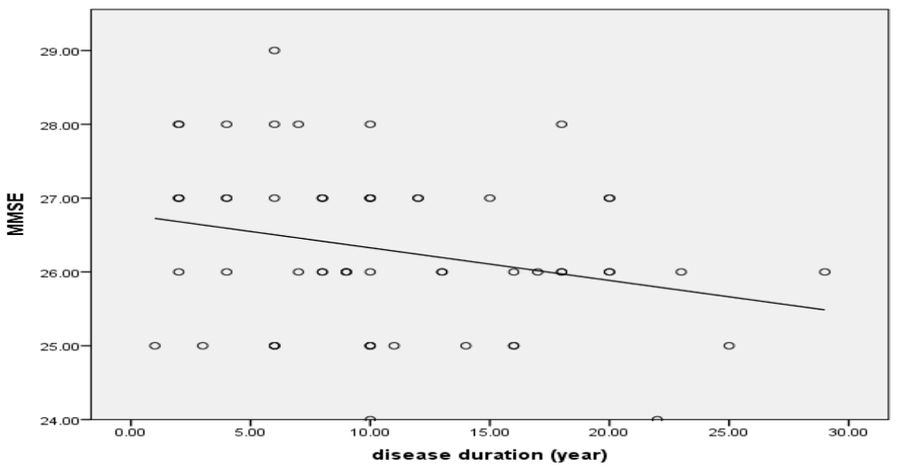

Fig. 1: Correlation between MMSE scores in the cases regarding onset of migraine duration

DISCUSSION

With a high prevalence and morbidity, migraine is one of the most prevalent neurologic illnesses, affecting over one billion people annually worldwide. VM is a disorder that warrants attention due to the significant occurrence of both migraine and dizziness in the general population. There is proof that migraineurs may have cognitive impairments regardless of whether cognitive dysfunction occurs during migraine attacks or during free time[11].

The results of VNG testing varied across VM patients in the current study. The VNG test commonly revealed positional nystagmus and positive placement, neither of which could pinpoint the pathology's location. The patients also have ocular motor anomalies and calorie weakness. At this stage, the question of what mechanism(s) causes vestibular system dysfunction as determined by VNG arises. Peripheral vestibular impairment may be temporary or permanent as a result of migraine-induced vasospasm and the ensuing reduction in regional blood flow to the inner ear via the internal auditory artery from the anterior inferior cerebellar artery (AICA).

In a study of 65 VM patients, Servillo *et al*.[12] discovered that 35.5% of VM patients had positive results from positional and positioning tests, and 13.5% of patients experienced gaze-evoked nystagmus.

On the other hand, Kenig *et al*.[13] discovered that 35% of VM patients had aberrant ocular movement. Thirty patients (75%) had a vertical nystagmus component, and ten percent of patients had spontaneous nystagmus. Central vestibular abnormalities can result from either a transient or permanent vestibular disorder, or from vascular insufficiency from chronic ischemia that damages the brain's higher centers. Long-term, more ischemic changes can also cause abnormalities in the brain's high-level cortical function.

It's unclear exactly why vestibular system malfunction and trouble maintaining a fixed profile in VNG tests occur in persons with VM. Numerous theories have been put out regarding the vascular and neurological systems involved in the pathophysiology of migraines. A number of neurotransmitters have been implicated in the pathophysiology of migraines, and it has also been suggested that spreading depression affects brainstem regions. When the multimodal cortical areas are engaged in the vestibular signal processing, spreading depression can result in vestibular symptoms such as unsteadiness or even positional vertigo[14].

Asymmetric vestibular system involvement in VM patients may be indicated by the significant difference in p13 latency and N13-P23 amplitude between cases and controls. This could imply vestibulo-spinal tract affection, such as retrolabyrinthine lesion, vestibular nerve, or brainstem lesions[15].

Elmoazen *et al*.[16] demonstrated noticeably longer P13 of cVEMP in migraine and VM patients compared to controls, which is consistent with our findings. However, Dabbous *et al*.[17] found that 7 out of 35 migraine cases showed delayed latency, suggesting a disturbance in the vestibulo-spinal reflex (VSR). Compared to the controls, there was a statistically significant decrease in IAAD%. While in agreement with our results, Ghali & Kolkaila (2005) found a significant delay in VEMP waves as numerous theories have been put up to explain migraineurs' vestibular impairment. The first theory links the growing depression that affects brainstem regions. Vestibular dysfunction in migraine may possibly be explained by internal auditory artery vasospasm, which is comparable to retinal vasospasm in retinal migraine[18].

Moreover, Murofushi *et al*. (2015)[15] examined 221 VM patients and proposed that, when assessing the extended latencies of VEMP, P13 latency is preferable to N23 latency since N23 latency has a higher standard deviation of normal values than P13.

The wide range of absolute amplitude measurements, which is largely dependent on the EMG level of sternocleidomastoid muscle contraction, can be used to explain the variability in amplitude results between studies. In contrast, Kim *et al*.,[19] & Baier *et al*.,[20] found that amplitudes were reduced with VM while the latencies were within normal range.

The interpretation and rehabilitation of these patients' symptoms may benefit from the addition of SVV tests to the evaluation process for VM patients. In addition to helping distinguish between central and peripheral vestibular illnesses, measurements of SVV are helpful in the clinical test battery for identifying probable anomalies in the utricle and the superior vestibular nerve's pathways. During vestibular therapy, SVV can also be utilized to track both good and negative changes in recovery and compensation[21].

The mean SVV tilts in the SVV testing showed a statistically significant difference. These results point to possible anomalies in the utricle and the superior vestibular nerve's pathways related to aberrant sensory processing and integration for spatial perception in vestibular migraineurs.

Similar to our findings, Jamie *et al*.[22] discovered that 54% of VM patients had aberrant mean SVV tilt. They found that in patients with VM, absolute mean SVV tilt and response variance are frequently aberrant. These results provide credence to theories indicating anomalous intralabyrinthine integration in the cerebellar nodular pathways and vestibular nuclei.

Fei *et al*.[23] measured the mean SVV tilt when the subject was seated upright in the time between attacks. They discovered that there was a significant difference (P=0.006) in SVV between VM patients and normal controls when they were upright. The reason for any anomaly in SVV

in individuals with VM was interpreted as a functional problem of the cerebellum or high-level cortical areas, or as a result of insufficient vestibular compensation.

Conversely, Ariel *et al*.[24] examined the inaccuracies in upright perception among a cohort of 27 VM patients by contrasting them with a cohort of 27 healthy counterparts. For both healthy controls and VM patients, SVV errors were within the normal range, which is 2° from true vertical. The SVV precision of the VM patients and the healthy controls did not differ significantly. The difference may have resulted from the stage of the illness at when the patients were enrolled and tested.

There is evidence that VM patients may come with cognitive loss, although the cognitive screening of VM patients has not been thoroughly studied[25]. Both during migraine episodes and during free periods, there is cognitive impairment[26].

The MMSE was used to measure subjects' cognitive performance in the current study. VM patients' scores decreased significantly compared to the control group, indicating that they had reduced cognitive function.

According to Wang *et al*.[27] there was a greater impact on the MMSE scores of the VM patients group compared to the control group. When VM patients and controls underwent the MMSE, Kalaydjian *et al*.[28] found a highly significant decrease in the VM group's MMSE scores when compared to the controls. They attributed this cognitive impairment to the vasomotor disturbances brought on by chronic ischemia during recurrent headaches.

this was in contrast to Demrihan M & Celebisoy N[29] who found that there was no significant difference ($p > 0.05$) between the cognitive test results of the MD and VM patients and the healthy controls. They clarified that cognitive symptoms might linger into the postdrome and are common during the premonitory and headache phases of a migraine attack. Outside of migraine attacks, some migraineurs also report having cognitive problems. Thus, for an improved assessment, use cognitive questionnaires as a follow-up.

According to Baars *et al*.[30] there was no difference in MMSE scores between the group of VM patients and the controls. A migraine diagnosis had no impact on cognitive decline or performance. These discrepancies could be the result of many methodological problems, such as disparities in migraine assessment techniques. Inconsistencies may also arise from differences in clinical parameters such as age, gender, migraine kinds, use of headache medications, food, sleep, or physical activity.

Subjects in both groups completed the SDMT in this study, and the group of VM patients had statistically significantly lower scores than the control group. The primary theory to explain the pathophysiology of cognitive decline linked to migraine is vasomotor abnormalities, which is why VM patients were at risk of cognitive decline. Long-term vasomotor dysfunction in migraineurs causes cerebral vasospasms, which lower blood flow volumes in perforating arterial branches and cause brain degeneration, particularly in deep white matter[31].

This was in accordance with Calandre *et al*.[32] who assessed the cognitive functioning of patients with VM and looked into potential irregularities that might be connected to the long-term nature of the illness. When comparing the SDMT scores of the VM group to the control group, they discovered cognitive impairment in individuals with vestibular migraines.

When the VM group and controls underwent the SDMT, Mulder *et al*.[33] discovered a highly significant decline in the VM group's SDMT scores.

However, other authors disagree with our findings. Gaist *et al*.[34] reported no statistically significant change in SDMT ratings between the VM group and the controls. This might be because there were less participants in our study group than in Gaist *et al*.'s group, who tested a greater number of patients.

Other authors, however, disagree with our results. Between the VM group and the controls, there was no statistically significant difference in SDMT evaluations, according to Gaist *et al*.[34]. This could be the result of fewer participants in our study group compared to the group of Gaist *et al*., who tested a larger number of patients.

CONCLUSION

It is clear from this that MV is a distinct clinical entity and that this illness may be diagnosed using the Structured Interview method. Vestibular migraine (VM) cannot be diagnosed with a typical fixed profile in vestibular testing; instead, patients with VM exhibit spatial disorientation and dysfunction of the otolithic pathway. The duration of migraines is an unavoidable risk factor that may be linked to a decline in cognitive function.

According to classification of International Headache Society (IHS): there’s vestibular migraine and probable vestibular migraine. The severity of the symptoms must be moderate or severe. Acute episodes might last anywhere from five minutes to seventy-two hours.

RECOMMENDATION

The routine assessment of migraine complications should include the evaluation of the cognitive function. We recommend early implementation of MMSE and SDMT; which are simple tools; to be done for screening of MCI in migraine patients earlier in age.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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