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Original article

A study of the correlation between Migraine and Vestibular Vertigo

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ABSTRACT

The pathophysiology of Vestibular Migraine is puzzling dilemma. The simultaneous process of the two symptoms doesn't initiate a definitive causal relationship.

Objectives: This study aimed to investigate whether Audiovestibular evaluation can help in differentiation of the migraine-associated vertigo.

Materials and methods: Participants of this study had an attack of Dizziness associated with Migraine or after attacks of Migraine only. They were subjected to questionnaire to confirm the criteria for Migraine (International Classification of Headache) and characteristics of vertiginous attacks. Videonystagmography and Caloric tests were done.

Results and conclusion: Comparison between Control and study group revealed no statistically significant difference. The association of Vertigo and Migraine is certainly insufficient to make the dizziness of Migraine related Vertigo. The rationale is to improve management of this disease.

1. Introduction

Migraine is a benign and recurring syndrome of unilateral throbbing headache, associated with nausea, vomiting and phonophobia.[1] Diagnostic criteria for Migraine were lacking until 1988 when the International Headache Society (IHS) Classification for headache was published.[2] Both disorders Migraine and Vertigo are common disorder in the general population. Recent population-based studies using the 1988 HIS diagnostic criteria for Migraine have consistently estimated the vestibular migraine rates to be 4–6.5% in men and 11.2–18.2 in women in both US and Europe.[3,4]

Crevits and Bosman[5] considered Vestibular Migraine to be the second most frequent cause of recurrent vertigo after BPPV, and the first cause of recurrent spontaneous vertigo, affecting 1% of the adult population. Various terms have been used to date to refer to this clinical entity: Migraneous vertigo, Migraine-associated vertigo or dizziness, migraine-related or Migraine induced vestibular pathology.

In 2008, a group of neurologists and Otolaryngologists set up a Committee to develop an International Classification of Vestibular Disorders, in order to be able to establish clinical diagnostic criteria for the most frequent disorders following the IHS model for the definition of headaches (International Classification of Headache Disorders [ICHD].[6] The Committee has initially developed definitions for the signs and symptoms to unify the terminology.[7] Furman et al.[8] reported that Vestibular Migraine became a subtype of migraine. The recently developed diagnostic criteria allowed more understanding of the clinical aspects of vestibular migraine.

This study aimed to investigate whether Audiovestibular evaluation can help in differentiation of the migraine-associated vertigo.

2. Subjects and Methods

In this prospective study participants were referred due to attacks of Vertigo. All patients were subjected to careful history taking: Structured interview focused on vertigo characteristics and migraine headache according to (IHS) criteria. The final approved diagnostic criteria were the product an accord between the HIS Classification Committee and the Committee for Classification of Vestibular Disorders of the Barany Society. We selected these final approved diagnostic criteria to be followed in our study, according to International Classification of Headache Disorders (ICHD).[9]

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The exclusion criteria for testing were: Evidence of chronic otitis media and/or previous surgery of the middle ear; Therapy with ototoxic drugs and/or chemotherapy. Diseases of the central nervous system. All patients underwent a neurological examination and magnetic resonance imaging (MRI) of the central nervous system.

Each subject was then submitted to an audiovestibular test battery which included:

- Basic Audiological Evaluation.
- Vestibular tests: Videonystagmography (VNG including spontaneous nystagmus, smooth pursuit & saccade) and also Dix-Hallpike and finally Air Caloric was done.

We considered central signs as having at least one of the followings.

Disorganized pursuit and reduction of pursuit gain; Saccades dysmetria with undershoot or over-shot or asymmetrical latency or velocity; Rebound nystagmus; Visual fixation suppression of nystagmus lower than 60%; Pure vertical or torsional spontaneous or positional nystagmus; Positional nystagmus when bilateral, beating to uppermost or lowermost ear, showing no latency, low frequency, lack of fatigability and habituation, without concomitant vertigo.

All the results of the study group compare to the control group, it consisted of 12 subjects who complained of dizziness attacks only with no history of Migraine or severe headache. Any vascular, neurological or ear disorders were excluded.

All categorical variables were tested using Multivariate analysis logistic regression analysis to test which factors were the most likely to discriminate patients with benign recurrent vertigo.

3. Results

Subjects who full fill the diagnostic criteria of Vestibular Migraine were classified into three groups according to duration of Migraine. Group I (duration of Migraine $1 \leqslant 5$ yrs.) represented 46.4% of the subjects, mean age 35.9 ± 5.8, group II (duration of Migraine $5 \leqslant 10$ yrs.) include 28.6% of patients, mean age 35.2 ± 4.6, group III (duration $\geqslant 10$ yrs.) represented 25%, mean age 39.7 ± 9 There was no significant difference between groups as regards to age (see Figs. 1 and 2).

3.1. Side of Migraine

In all patients migraines headache had been present before the onset of vertigo. Left sided Migraine more presented than right side in all groups. Comparison of left (68%) and right sided (32%) Migraine subjects in the three studied groups revealed no significant difference (Table 1).

3.2. Character of Vertigo

The character of dizziness in the control group mostly is sense of imbalance, and no effect on activity. While sense of imbalance and spinning are equal in the study group. No significant difference between study groups and the duration of migraine has no effect in the study group (Table 2). There was significant difference between study and control groups as regards to sense of rotation.

3.3. Degree of the attack

Vestibular symptoms included: spontaneous vertigo, positional vertigo, visually induced vertigo, head motion-induced dizziness with nausea. Vertigo was rated as moderate if vertigo interfered with but did not prohibit daily activities and as severe if daily activities could not be continued. (Table 3) showed that ten out of the 28 (36%) couldn't specify the effect of dizziness on their activities. In the three groups of patients represented different degrees either first degree with no effect on activity, or second degree where stoppage of activity occurred only during episode, the third degree bed ridden, can't work, can't drive. Most of the patients in the three groups reported first and second degree of the effect. There was no statistically significant difference between the three groups.

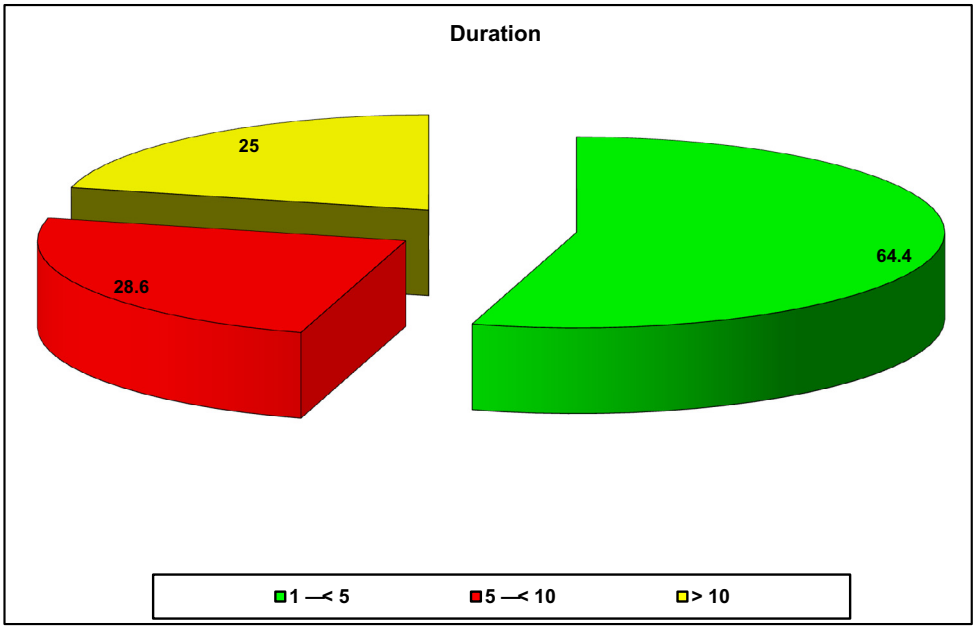

Fig. 1. Showed classification of studied groups according to duration.

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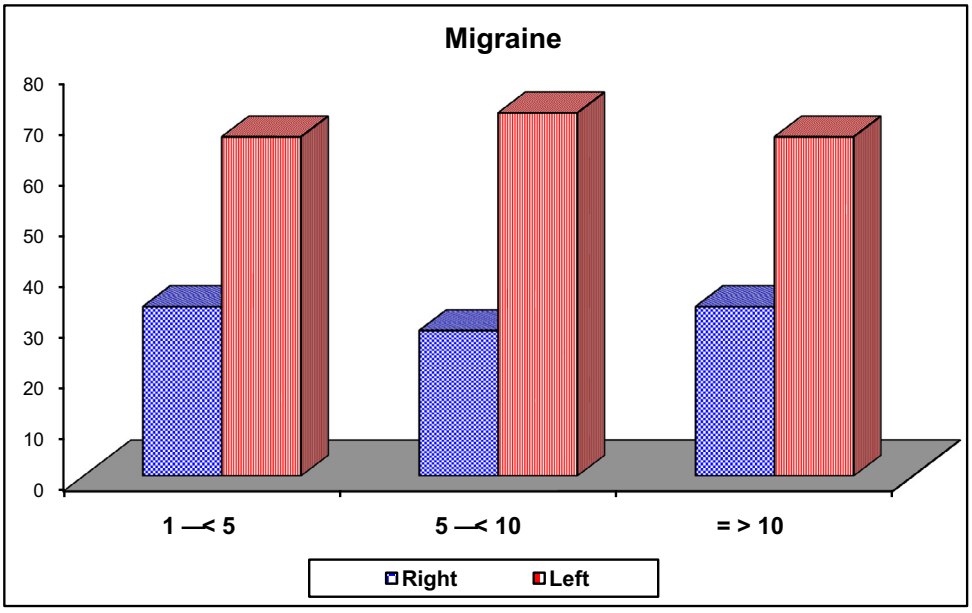

Fig. 2. Showed right and left sided migraine in the studied group.

Table 1
Comparison of left and right sided Migraine in the VM study groups.

Migraine		1–<5	5–<10	=>10	Total
Right	N	5	2	3	10
	%	33.3%	28.6%	33.3%	32.0%
Left	N	9	5	4	18
	%	66.7%	71.4%	66.7%	68.0%
Total	N	14	7	7	28
	%	100.0%	100.0%	100.0%	100.0%
Chi-square	X^2	0.053			
	P-value	0.974			

Table 2
Comparison of character of dizziness in the VM study groups and control group.

			Disease			Control	X^2	P-value
			1–<5	5–<10	=>10			
Sense of imbalance	Yes	N	9	4	1	7	0.234	0.629
		%	69.2%	50.0%	14.3%	58.3%		
	No	N	4	4	6	5		
		%	30.8%	50.0%	85.7%	41.7%		
X^2			5.495					
P-value			0.064					
Sense of rotation	Yes	N	5	4	5	2	3.889	0.049
		%	38.5%	50.0%	71.4%	16.7%		
	No	N	8	4	2	10		
		%	61.5%	50.0%	28.6%	83.3%		
X^2			1.978					
P-value			0.372					
Sense of lightheadedness	Yes	N	2	1	0	1	0.053	0.818
		%	15.4%	12.5%	0.0%	8.3%		
	No	N	11	7	7	11		
		%	84.6%	87.5%	100.0%	91.7%		
X^2			1.163					
P-value			0.559					
Black outs	Yes	N	1	1	0	0	0.902	0.342
		%	7.7%	12.5%	0.0%	0.0%		
	No	N	12	7	7	12		
		%	92.3%	87.5%	100.0%	100.0%		
X^2			0.891					
P-value			0.641					

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Table 3
Comparison of the degree of the dizziness attacks in the VM study groups and control group.

Degree		Disease			Control	Chi-square	
		1–<5	5–<10	=>10		X^2	P-value
I	N	5	3	2	8		
	%	50.0%	37.5%	40.0%	72.7%		
II	N	5	4	2	3		
	%	50.0%	50.0%	40.0%	27.3%		
III	N	0	1	1	0		
	%	0.0%	12.5%	20.0%	0.0%	1.997	0.736
Total	N	10	8	5	11		
	%	100.0%	100.0%	100.0%	100.0%		
Chi-square	X^2	1.997					
	P-value	0.736					

I = Has no effect on activity.
II = Stoppage of activity occurs only during episode.
III = Bed ridden, can't work, can't drive.
N.B: 5 subjects couldn't specify their degree of hearing loss.

Table 4
Comparison of associated factors in the VM Study groups.

			Disease			Control	Chi-square	
			1–<5	5–<10	=>10		X^2	P-value
Sense of pressure	Yes	N	0	0	2	0	0.902	0.342
		%	0.0%	0.0%	28.6%	0.0%		
	No	N	13	8	5	12		
		%	100.0%	100.0%	71.4%	100.0%		
X^2			6.462					
P-value			0.040					
Tinnitus	Yes	N	1	0	1	1	0.017	0.896
		%	7.7%	0.0%	14.3%	8.3%		
	No	N	12	8	6	11		
		%	92.3%	100.0%	85.7%	91.7%		
X^2			1.160					
P-value			0.560					
Hearing loss	Yes	N	1	0	1	0	0.902	0.342
		%	7.7%	0.0%	14.3%	0.0%		
	No	N	12	8	6	12		
		%	92.3%	100.0%	85.7%	100.0%		
X^2			1.160					
P-value			0.560					
Nausea & Vomiting	Yes	N	4	4	4	3	1.143	0.285
		%	30.8%	50.0%	57.1%	25.0%		
	No	N	9	4	3	9		
		%	69.2%	50.0%	42.9%	75.0%		
X^2			1.526					
P-value			0.466					
Blurring of Vision	Yes	N	0	1	0	0	0.440	0.507
		%	0.0%	12.5%	0.0%	0.0%		
	No	N	13	7	7	12		
		%	100.0%	87.5%	100.0%	100.0%		
X^2			2.593					
P-value			0.274					
Loss of Consciousness	Yes	N	0	0	0	0		
		%	0.0%	0.0%	0.0%	0.0%		
	No	N	13	8	7	12		
		%	100.0%	100.0%	100.0%	100.0%		
X^2			–					
P-value			–					

3.4. Associated Symptoms

The highest association was nausea and vomiting in the three groups and recorded with high percent in group III, followed by auditory symptoms (tinnitus and hearing loss) in group I and III. Sense of pressure was only reported in two patients in group III. Only one patient in group II had blurred vision. None of our patients had loss of consciousness. There was no significant difference between groups as regards these associated symptoms. In the control group, three out of 12 subjects had nausea only and one complained of tinnitus. There was no significant difference between study and control groups (Table 4).

3.5. Vestibular tests

Saccade test, there was delayed latency with increase in the duration of Migraine, but it did not reach level of significance (Table 5). For Smooth Pursuit at different phases, no significant dif-

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ference was found between the three studied groups (Table 6). Moreover, the same results were recorded for Optokinetic test for either right or left (Table 7). Most of our patients had a unilateral canal paresis, although it did not reach significant level. It was clearly observed and related to side of Migraine (Tables 8 and 9).

Comparing the results of the control and study subgroups revealed no significant difference.

4. Discussion

The detailed phenotypic characterization and analysis of episodic vestibular Migraine is going to be a key process for the development of accurate management.

4.1. Duration of Migraine

In this study we classified our patients' complaint of dizziness. Also, they had Migraine for variable duration from 1 year to more than 10 yrs. We classified patients according to duration to study its effect on vestibular finding. Also to check if the findings change over time. Our results revealed no significant differences in the duration of illness among the three groups. These were consistent with the results of Lee et al.[10] who did not support the hypothesis that repeated migraines cause permanent damage to the cochlea and vestibule. Pagnin et al.[11] emphasized that in spite of the long duration of vestibular symptoms before diagnosis none of their patients had another transformation of the vestibular disturbance into headache or into another migraine equivalent; therefore, vestibular symptoms in patients with migraneous vertigo appear to represent the last delayed manifestation of their migraneous habit. Hence, EMV does not seem to be temporary conversion, but rather a definite conversion of migraines symptoms.

We were unable to find clinically significant differences between VM patients with different duration and Vestibular Function Test results. That is the clinical manifestations and prognosis were similar in all three groups, regardless of the VFT findings.

4.2. Pathophysiology

In our study, there was no significant difference in the degree of vertigo in the studied groups and this also applied for the associated symptoms and character of vertigo. This was explained by Cutrer and Baloh[12] that the primary abnormality in Migraine may be a disinhibition of the brainstem pathways subserving sensory inputs. Dieterich and Brandt[13] have suggested that vertigo symptoms may be a, "brainstem aura" which is a spreading wave of depression of neural activity possibly accompanied by changes in blood flow. The observation that the symptoms of imbalance and motion intolerance occur with headache rather than being a proceeding aura symptom, provides credence to the belief that they result from abnormal central processing rather than vasospasm.[14] Analogous to the increased sensitivity in migraine to visual and auditory stimuli, an alteration in sensitivity to vestibular inputs may be present in MRV (Migraine related Vertigo).[15] Teggi et al.[16] in their experience reported that subjects with migraine and vertigo rely more on visual rather than on proprioceptive cues in balance control. As a possible explanation that neuromediator release during the algic phase of migraine produces an increase in the threshold of somatosensorial cues in central nervous system.

Parker and Radtk et al.[17,18] reported that the repeated Circulation problems (e.g. vasospasm-induced ischemia of the labyrinth and plasma extravasation during migraine attacks may result in permanent damage of the cochlea and vestibule. In addition, the clinical manifestation of these patients may be similar to typical vestibular dysfunction. Furthermore, the evidence of ion-channel dysfunction and calcium channel disturbances of the inner ear and its central connections also offer a promising hypothesis for the diagnosis and treatment of migraine associated vestiblopathy.[19]

Specifically, the vascular, neurogenic inflammations, and central neural mechanisms that have been implicated as peripheral and central triggers of migraine are all present in central vestibular pathways and the inner ear.[20,21] Recent studies of vestibular psychophysics[22] and motion sickness susceptibility[23,24] in vestibular migraine are yielding new insights. The large overlap between migraine pathways and vestibular pathways is consistent with the view that vestibular migraine is a migraine variant with vestibular manifestations.

4.3. Vestibular Results and Caloric stimulation

The results of our study showed no significant difference in the occulomotor vestibular tests. However, Teggi et al.[16] Showed abnormal occulomotor findings indicating brainstem or cerebellar dysfunction. Findings showed consistently decreased number of occulomotor alterations compared with Diteerich and Brandt[13] which were not different from the results of Celebisoy et al.[25]. Different inclusion criteria may explain the different results. Majority (68%) of our patients had a unilateral canal paresis (left side), although it did not reach significant level, which can be explained by small number of the sample. It was clearly observed and related to the side of Migraine. Our results agreed with the results of[26] that Electronystagmographic abnormalities (canal Paresis and directional preponderance) occurred in 55% of migraneous patients with vertigo.

Boldingh et al. 2015[27] and Bir et al.[26] reported vestibular abnormalities in patients with vestibular Migraine and no difference between Vestibular Migraine and Migraneous patients. Dash et al.[1]

Table 5
Comparison of saccade latency test in the VM study groups.

Saccade latency	1–<5	5–<10	⩾10
Range	170–225	174–226	181–287
Mean ± SD	194.77±15.65	200.25±18.81	217.14±39.11
F. test	1.978		
p. value	0.159		

Table 6
Comparison of Smooth Pursuit gain test in the VM study groups.

		1–<5	5–<10	⩾10	F. test	p. value
SP. 1	Range	0.47–1.06	0.57–1.04	0.66–1.0	0.015	0.985
	Mean ± SD	0.87 ± 0.15	0.88 ± 0.15	0.87 ± 0.13		
SP. 1	Range	0.55–1.69	0.58–1.04	0.65–1.04	0.877	0.428
	Mean ± SD	1.0 ± 0.24	0.92 ± 0.15	0.89 ± 0.13		
SP. 1	Range	0.49–1.07	0.58–1.15	0.72–1.03	0.230	0.796
	Mean ± SD	0.92 ± 0.15	0.89 ± 0.17	0.95 ± 0.11		

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Table 7
Comparison of Optokinetic gain test in the VM study groups.

		1–<5	5–<10	⩾10	F. test	p. value
OPK right	Range	0.49–1.17	0.45–1.26	0.69–1.01	0.029	0.972
	Mean ± SD	0.90 ± 0.18	0.88 ± 0.23	0.89 ± 0.13		
OPK left	Range	0.68–1.08	0.52–1.05	0.57–1.07	0.700	0.506
	Mean ± SD	0.89 ± 0.12	0.82 ± 0.16	0.88 ± 0.16		

Table 8
Comparison of Caloric test in the in both sides of VM study groups.

		1–<5	5–<10	⩾10	F. test	p. value
Caloric right	Range	7–25	5–30	7–37	0.421	0.663
	Mean ± SD	16.5 ± 6.50	16.85 ± 9.58	20.8 ± 10.68		
Caloric left	Range	5–19	7–25	9–28	1.691	0.214
	Mean ± SD	11.13 ± 4.61	14.71 ± 2.94	17.0 ± 7.31		

Table 9
Comparison of Caloric test between study& control groups.

		Disease	Control	T. test	P. value
Caloric Right	Range	0.80–37	5.0–26	0.009	0.924
	Mean ± SD	13.99 ± 8.16	14.25 ± 6.51		
Caloric Left	Range	5–35	7–25	0.010	0.921
	Mean ± SD	14.0 ± 7.56	14.25 ± 6.06		

said that clinical symptoms and cochleovestibular findings in cases of migraine with and without vertigo revealed no statistically significant difference. Different percentage of difference in caloric stimulation were reported: Teggi et al.[16]and Celebisoy et al.[25] emphasized that only 20% of patients had side difference of caloric stimulation, while Kuritsky et al.[28] (50%) and Olsson[29] (45%), Dieterich and Brandt[13] (12.6%).

Results of Boldingh et al.[27] supported ours as they reported no differences in the distribution of central and peripheral vestibular signs between Vestibular Migraine and Migraines patients. This may indicate that subclinical vestibular dysfunction is an integral part of migraine pathology in general and not solely in Vestibular Migraine. Pagnini et al.[11] emphasized the similarity of temporal succession of symptoms, type of headache and vertigo spells, habitus, absence of any other definitive vestibular pathology. Also, the negativity of general and neurological investigations that we noticed among patients led to consider such patients homogenous enough to be grouped into a new entity that we called "Epigone migraine vertigo" a term that means "borne after".

Our results point to the conclusion that VFT results in VM patients may be non-specific and clinically non-significant. This may be due to big gap of duration among patients with vestibular Migraine. The results may change overtime, this issue may be clearly important and requires verification. Therefore, appears that treatment strategy should be focused on the migraine itself rather than the status of vestibular function.

5. Conclusion

The pathophysiology of Vestibular Migraine is a puzzling dilemma. The simultaneous presence of the two symptoms does not indicate a definite causal relationship**.** VM patients showed that Vestibular Function test results either Peripheral or central vestibular may not reflect the true pathology. The challenge now is to better understand the pathophysiology of vestibular migraine from both a clinical and basic science perspective to enable improved rational management of this disorder.

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