Neurology Asia 2022; 27(2) : 403 – 409

Can vestibular migraine development be predicted in patients with new onset migraine headaches?

[1]Neşe Çelebisoy, [2]Ayşın Kısabay Ak, [3]Ceyla Ataç, [1]Hüseyin Nezih Özdemir, [1]Figen Gökçay, [1]Gülsüm Saruhan Durmaz, [3]Dilek Top Kartı, [4]Hülya Ertaşoğlu Toydemir, [4]Vildan Yayla, [5]Ayşe İlksen Çolpak Işıkay, [5]İrem Erkent, [2]Aysegul Seyma Sarıtas, [6]Pınar Özçelik, [6]Gülden Akdal, [7]Şebnem Bıçakcı, [8]Eylem Ozaydın Göksu, [9]Feray Güleç Uyaroğlu

[1]Department of Neurology, Ege University, Izmir; [2]Department of Neurology, Celal Bayar University, Manisa; [3]Department of Neurology, Bozyaka Education and Research Hospital, University of Health Sciences Izmir; [4]Department of Neurology, Bakırköy Dr. Sadi Konuk Education and Research Hospital, University of Health Sciences Istanbul; [5]Department of Neurology, Hacettepe University, Ankara; [6]Department of Neurology, Dokuz Eylül University Izmir; [7]Department Of Neurology, Çukurova University, Adana; [8]Department of Neurology, University of Health Sciences Antalya Education and Research Hospital Antalya; [9]Department of Neurology, University of Health Sciences Izmir Tepecik Education and Research Hospital, Izmir, Turkey

Abstract

Objective: This study aims to determine the clinical features associated with the development of vestibular migraine (VM) in patients with migraine headaches. *Methods:* A cross-sectional, multicenter study was performed in nine tertiary neurology clinics. Patients with migraine without vestibular symptoms were classified as having migraine only (MO) and compared with patients with VM to determine any differences in clinical features, associated disorders, past medical history, and family history of migraine headaches. Moreover, we investigated the features that might predict the development of VM. *Results:* Two hundred forty-four patients with MO and 461 patients with VM were included. The age of onset of headache attacks was later in life for patients with VM ($p<0.001$). Migraine without aura (MwoA) was significantly more common than migraine with aura (MwA) in patients with VM ($p=0.016$). All associated features of migraine headaches were significantly more frequent in patients with MO than patients with VM ($p<0.005$). The same was true for all triggers, including fasting, sleep disturbances, menstruation, stress, flickering lights, and smartphones/computer games ($p<0.005$). A family history of migraine headaches was more common in MO patients ($p=0.002$). However, a previous history of motion sickness was significantly more common in patients with VM ($p<0.001$), as was aural fullness/tinnitus accompanying attacks ($p<0.001$). Logistic regression analysis indicated that aural fullness/tinnitus accompanying attacks and a previous history of motion sickness were risk factors for the development of VM.
Conclusion: Patients with migraine reporting aural symptoms accompanying attacks and motion sickness in their past medical history are at increased risk of vestibular attacks fulfilling the diagnosis of VM later in life.

Keywords: Vestibular migraine, aural fullness/tinnitus, motion sickness, vertigo

INTRODUCTION

Migraine is a chronic neurological disorder with a 1-year prevalence of 12% in the general population[1], wherein women are more commonly affected than men. The annual and lifetime prevalence is 18% and 33% in women and 6% and 13% in men respectively.[2] It is characterized by recurrent episodes of moderate to severe headaches with reversible neurological and systemic symptoms. Headache is often unilateral, pulsating and aggravated by physical activity. The most common associated symptoms are photophobia, phonophobia, nausea, vomiting, and cutaneous allodynia. Additional symptoms like vertigo, dizziness, and tinnitus can be present.[3]

Vestibular migraine (VM) is characterized

Address correspondence to: Neşe Çelebisoy, Ege University Department of Neurology, Izmir, 35000 Turkey. emails: nese.celebisoy@ege.edu.tr
Date of Submission: 26 December 2021; Date of Acceptance: 15 February 2022
https://doi.org/10.54029/2022whv

by migraine-related vestibular symptoms, and it is accepted as the most common cause of spontaneous episodic vertigo.[4] Diagnostic criteria have been jointly developed by the International Headache Society and the International Barany Society for Neuro-otology.[5,6] A 1-year prevalence of 2.7% has been reported in a population-based study.[7] In patients with recurrent vertigo, a significantly high prevalence of migraine ranging from 60% to 80% has been reported.[8,9] Conversely, VM has been diagnosed in 10%–20% of patients in headache clinics.[10,11] An overlap between vestibular and migraine pathways is the proposed pathophysiological mechanism.[12]

Patients with VM report different types of vertigo, including spontaneous vertigo, positional vertigo, and unsteadiness. In a study from a headache clinic, unsteadiness was present in 91% of the patients, and 82% had balance problems.[13] Nausea is a very common symptom that accompanies these attacks. Photophobia, phonophobia, osmophobia, and visual or other auras have also been documented.[14] Moreover, auditory symptoms including hearing loss, tinnitus, and aural pressure related to acute attacks, have been reported in 20%–40% of VM patients.[14,15] Hearing loss is usually mild and transient.[16] Attacks can be provoked by menstruation, sleep disorders, stress, specific foods, and sensory stimuli, such as scintillating lights, intense smells, or noise.[17]

The duration of episodes can range from minutes to days. Vertigo can precede, occur during, or follow headache. In some patients, vertigo and headache can occur as unrelated attacks.[18] A susceptibility to motion sickness is commonly reported in patients with VM.[19] Vertigo induced by visual scenes and moving objects (visually induced vertigo) persisting in between attacks has also been reported.[20]

In this multicenter study comprising patients from different parts of Turkey, the clinical features of patients with migraine without vestibular symptoms [migraine only (MO)] and patients with VM were compared to determine any significant differences in terms of demographic features, associated disorders, past medical history, and family history of migraine headaches. Moreover, features that might predict the development of VM were investigated.

METHODS

This cross-sectional multicenter study included patients diagnosed as having MO and VM according to the ICHD-3 criteria[6] who had a follow-up period of at least one year. This study was performed in eight tertiary neurology clinics between 2015 and 2020. The study protocol was approved by the Ege University Medical School Ethics Committee (reference number: 99166796-050.06.04) and approval from the local ethics committees of all the participating centers was granted. Written informed consent was obtained from all participants.

A structured questionnaire was used for data collection. All patients were interviewed by a senior neurologist. Migraine was classified as migraine with or without aura (MwA or MwoA, respectively) depending on the presence or absence of visual, sensory, speech and/or language, motor, and brainstem or retinal symptoms accompanying or followed by headache. The age of onset of migraine headaches and vertigo attacks; headache and vertigo attack frequency; pain intensity determined using a visual analog scale (VAS) measured in centimeters from 0 to 10; and associated symptoms (nausea, vomiting, photophobia, phonophobia, osmophobia, allodynia, ear fullness/tinnitus, hearing loss) were recorded.

Data on trigger factors, such as fasting, sleep disturbances, menstruation, stress, flickering lights, and smartphones/computer games, were obtained. A history of motion sickness and any family history of migraine were also recorded. A psychiatry consultation was made when necessary for comorbid anxiety and depression.

Cranial magnetic resonance imaging and pure tone audiometry were performed in all patients.

Statistical analysis

The SPSS 21 for Windows software package (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Continuous variables were analyzed as mean and standard deviation or the median and interquartile range, and categorical variables were analyzed as frequency and percentage. The distribution of the independent variables was compared using the chi-square test and Fisher's exact test. Receiver operating characteristic (ROC) curve analysis was performed to identify potential cutoff quantitative values associated with the development of VM.

Potential risk factors associated with the development of VM were analyzed via univariate and multivariate logistic regression analyses. Univariate odds ratio and 95% confidence limits were first calculated, and variables with $p<0.20$

were included in the logistic regression analysis as independent variables. Backward stepwise selection (Wald) was used to calculate multivariate odds ratios. In all statistical tests, p-values below 0.05 were considered statistically significant.

RESULTS

This study involved 244 patients with MO (30 men (12.3%) and 214 women (87.7%)) and 461 patients with VM (69 men (15%) and 392 women (85%)). The mean ages of the MO and VM groups were 36.5 (range, 13–52) years and 42±1 (range, 17–74) years, respectively.

In the MO group, 48 patients (19.6%) had MwA and 196 patients (80.4%) had MwoA. Of the 48 patients, 34 presented with visual aura symptoms and 14 patients presented with sensory aura symptoms. In the VM group, 51 patients (11.1%) had MwA and 410 patients (88.9%) had MwoA. Forty-two patients presented with visual aura symptoms and nine patients presented with sensory aura symptoms. Overall, MwoA was significantly more common than MwA in patients with VM (p=0.016). Table 1 shows the clinical features of the two groups.

The age of onset of migraine headaches was 25.5±8.4 years in the MO group and 28.4±9.4 years in the VM group, showing a statistically significant difference (p<0.001). The mean age at onset of vertigo attacks was 38.9±10.4 (range, 22–66) years, and the interval between the onset of migraine headaches and subsequent vertigo attacks was 125±60 (range, 6–360) months in patients with VM.

All associated features of migraine headaches, including nausea, vomiting, photophobia, phonophobia, osmophobia, and allodynia, were significantly more common in patients with MO (p<0.005). Similarly, all trigger factors, including fasting, sleep disturbances, menstruation, stress, flickering lights, and smartphones/computer games, were significantly more common in the MO group (p<0.005). A family history of migraine headaches was also more common in MO patients (p=0.002). A previous history of motion sickness was significantly more common in patients with VM (p<0.001). Similarly, aural fullness/tinnitus was significantly more common in patients with VM (p<0.001) (Table 1).

Based on the ROC analysis, age of onset of headache >26 years (specificity: 60.25%, 95% CI: [53.8–66.4] sensitivity: 55.1, 95% CI: [50.4–59.7], AUC: 0.586; p<0.0001) and history of migraine headaches >48 months (specificity: 36.07%, 95% CI: [30.8–42.4], sensitivity: 76.36, 95% CI: [72.2–80.2], AUC: 0.581; p<0.0001) were risk factors for the development of VM.

On univariate analysis, age of onset of headache >26 years (OR: 1.860, 95% CI: [1.357–2.549]; p<0.001), migraine history >48 months (OR: 1.822, 95% CI: [1.299–2.555]; p<0.001), aural fullness/tinnitus during headache attacks (OR: 1.370, 95% CI: [1.010–1.935]; p=0.064), and previous history of motion sickness (OR: 6.262, 95% CI: [4.194–9.174]; p<0.001) were risk factors for the development of VM.

On multivariate analysis, aural fullness/tinnitus accompanying attacks (OR: 2.29, 95% CI: [1.29–4.06], p=0.005) and previous history of motion sickness (OR: 17.25, 95% CI: [8.77–33.93]; p<0.001) were risk factors for the development of VM. (Table 2).

Comorbid anxiety/depression was present in 40.2% of patients with MO and 38.8% of patients with VM. The difference between the groups was not statistically significant (p>0.05).

DISCUSSION

Migraine onset most commonly occurs during late adolescence or early adulthood, with 75% of migraineurs experiencing the onset of migraine before the age of 35.[21] VM can develop at any age.[20,22] In most patients, migraine begins earlier in life than vestibular symptoms.[18,22] Similarly, the interval between migraine headaches and vertigo attacks was around 10 years in our patients with VM.

A few studies have specifically found MwA prevalence as 5% for MwA and 8% for MwoA.[23] In a study of over 700 patients with migraine, 56% had MwoA, 23.3% had MwA and both types were noted in 20.6%.[24] VM has been reported to occur more often in patients with MwoA.[25] Similar results were obtained in our study. MwA was significantly more common in patients with MO (19.6%) than in patients with VM (11.1%) (p=0.016).

In patients with MO, photophobia (94%), phonophobia (91%), and dizziness (72%) are frequently associated with attacks. Nausea occurs in over half of all patients.[26] Approximately one-third of patients present with symptoms of vomiting and 16% of patients have diarrhea during attacks.[25] Approximately 70% of patients have visual non-aura symptoms and about one-third have osmophobia or hyperosmia.[27,28]

Over 70% of patients have cutaneous allodynia, which is the perception of pain when non-painful

Table 1: Demographic and clinical features of patients with migraine only (MO) and vestibular migraine (VM)

Variables	MO (n: 244)	VM (n: 461)	p value
Sex (number) (%)			
Female	214 (87.7)	392 (85.0)	0.331
Male	30 (12.3)	69 (15.0)	
Migraine type (number) (%)			
MwA	48 (19.6)	51 (11.1)	0.016
MwoA	196 (80.4)	410 (88.9)	
Age of onset (years)			
Headache	25.5±8.4	28.4±9.4	<0.001
Vertigo		38.9 ± 10.4	
Attack frequency (attack number/month)			
Headache	3.5±0.5	4.03±0.6	0.037
Vertigo	-	4.17±1.45	
Intensity (VAS) (cm)			
Headache	7.3±1.2	7.4±1.4	0.208
Vertigo		6.7±1.7	
Associated symptoms (number) (%)			
Nausea	156 (64.0)	240 (52.0)	<0.001
Vomiting	115 (47.0)	161 (35.0)	<0.001
Photophobia	214 (87.7)	270 (58.7)	<0.001
Phonophobia	222 (91.0)	219 (47.6)	<0.001
Osmophobia	92 (37.7)	87 (18.9)	<0.001
Allodynia	100 (41.0)	94 (20.4)	<0.001
Aural fullness/tinnitus	22 (9.0)	161(35.0)	<0.001
Triggers (number) (%)			
Fasting	115 (47.1)	141 (30.6)	<0.001
Sleep disturbances	93 (38.1)	139 (30.2)	<0.032
Menstruation	125 (58.4)	143 (35.8)	<0.001
Stress	197 (80.7)	231 (50.1)	<0.001
Flickering lights	104 (42.6)	113 (24.5)	<0.001
Smart phones/computer games	67 (27.5)	71 (15.4)	<0.001
History of motion sickness (number) (%)	38 (15.6)	246 (53.4)	<0.001
Family history of migraine (number) (%)	106 (43.4)	147 (31.9)	0.002
Comorbid anxiety/depression (number) (%)	98 (40.2)	179 (38.8)	0.730

MwA: migraine with aura; MwoA: migraine without aura; VAS: visual analog scale

stimuli are applied to the skin.[29,30] In our patients with MO, phonophobia was commonly reported in 91%, followed by phonophobia in 87.7%. Nausea was present in 64% of the patients, whereas 47% reported vomiting during the attacks. Allodynia was reported in 41% of the patients and osmophobia in 37.7%. Moreover, aural fullness/tinnitus was present in 9% of the patients.

Aside from vertigo, patients with VM also report photophobia, phonophobia, and osmophobia. In a previous study, photophobia was reported in 44% of patients and phonophobia was reported in 39%. The prevalence of associated nausea and vomiting was 60% and 18% respectively. Tinnitus was reported in 11% of the patients and aural fullness in 9%.[14] In our patients with VM, photophobia was present in 58.7% and phonophobia in 47.6%. Moreover, nausea was present in 52% of the patients and vomiting in 35% during the attacks. Allodynia was also reported in 20.4% of the patients and

Table 2: Results of the logistic regression analysis performed to identify potential risk factors for the development of vestibular migraine (VM)

Variables	Univariate odds ratio (95% CI) p value	Multivariate odds ratio (95% CI) p value
Age of onset of headache (>26 years)	OR: 1.860 (1.357–2.549) p<0.001	OR:1.01 (0.55–1.85) p=0.969
Migraine history (>48 months)	OR: 1.822(1.299–2.555) p<0.001	OR:1.18 (0.60–2.32) p=0.635
Aural fullness/ tinnitus during headache attacks	OR: 1.370 (1.010–1.935) p=0.064	OR: 2.29 (1.29–4.06) p=0.005
Previous history of motion sickness	OR: 6.203 (4.194–9.174) p<0.001	OR: 17.25 (8.77–33.93) p<0.001

osmophobia in 18.9%. Aural fullness/tinnitus accompanying attacks was present in 35% of the patients.

The comparison of accompanying symptoms revealed that, except for aural fullness/tinnitus, all accompanying symptoms were significantly more frequent in patients with MO (p<0.005), but hearing-related symptoms were significantly more common in patients with VM (p<0.001).

With regard to the trigger factors, over 75% of migraineurs reported experiencing specific migraine triggers, the most common being stress, missed meals, sleep disturbances, and menstruation.[31] Odors, neck pain, light, and alcohol use were also reported, though less frequently. Our patients with MO reported all triggers, including fasting, sleep disturbances, menstruation, stress, flickering lights, and smartphones/computer games, more frequently than patients with VM did (p<0.005).

The majority of migraineurs have a family history of migraine, suggesting that genetic predisposition plays a major role.[32] Similarly, a positive family history of migraine has been found in 70% of patients with VM.[14] In our group, a family history of migraine was significantly more frequent in patients with MO (43.4%) than in patients with VM (31.9%) (p=0.002). However, both figures were lower than those in previous reports.

A previous history of motion sickness is well known in patients with migraine[33]; this has been reported in 43% of patients with VM.[14] In our patients, the rate was 15.6% in those with MO and 53.4% in patients with VM, significantly higher in the VM group (p<0.001).

A few studies have compared patients with MO and VM, and they deal with different aspects of the disorders. In addition to three studies investigating vestibular and audiologic findings[34-36], three studies focused on clinical features[37-39], like ours. Similar to our results, in the study investigating the lifetime prevalence of car sickness, patients with VM were more susceptible to car sickness than those with migraine or Meniere's disease.[37]

In the second study, it was found that both patients with migraine and VM were more anxious and apprehensive and had more panic symptoms and illness phobias than healthy controls, and that patients with VM were even more anxious and more agoraphobic than patients with migraine.[38] In our group, anxiety and depression were common comorbid psychiatric disorders in both MO and VM patients, but a significant difference between groups was not present (p>0.05).

In the third study that compared symptom characteristics, it was reported that the symptomatology of patients with migraine and those with VM was remarkably similar, indicating a significant overlap in clinical presentation between the two groups.[39]

However, in the previous studies, the risk factors for the development of VM were not analyzed. In our subjects, aural fullness/tinnitus accompanying attacks and a previous history of motion sickness were found to be risk factors on multivariate logistic regression analysis.

The main limitation of this study is the low number of patients with MO in comparison with VM despite its higher prevalence. Further studies including vestibular tests such as video head impulse test and vestibular evoked myogenic

potentials can possibly contribute to prediction of development of vestibular features in patients with MO.

In conclusion, patients with migraine who presented with auditory symptoms accompanying attacks as well as who have symptoms of motion sickness in their past medical history appear to be at risk for developing vestibular attacks later in life that fulfill the diagnosis of VM. The age of onset of headache attacks is later in life in patients with VM compared with those with MO. Patients with VM mainly present on a background of migraine headache without aura. Attack triggers and attack accompaniments, such as nausea, vomiting, photophobia, phonophobia, osmophobia, and allodynia, are more common in patients with MO than in those with VM. Furthermore, comorbid anxiety/depression is common in both groups.

ACKNOWLEDGEMENTS

We are grateful to Ege University Planning and Monitoring Coordination of Organizational Development and Directorate of Library and Documentation for their support in editing and proofreading service of this study. The authors would like to thank Atilla Atasever for his contribution for the statistical analysis.

DISCLOSURE

Ethics approval: The study protocol was approved by Ege University Medical School Ethics Committee (reference number: 99166796-050.06.04) and approval from the local ethics committees of all the participating centers was gathered.

Financial support: None

Conflice of interests: None

REFERENCES

1. Lipton RB, Bigal ME, Diamond M, Freitag F, Reed ML, Stewart WF. Migraine prevalence, disease burden, and the need for preventive therapy. *Neurology* 2007; 68:343-9. https://doi.org/10.1212/01.wnl.0000252808.97649.21.
2. Dodick DW. Migraine. *Lancet* 2018;391:1315-30. https://doi.org/10.1016/S0140-6736(18)30478-1.
3. International Headache Society. The international classification of headache disorders, 3rd edition. *Cephalalgia* 2018; 38:1-211. https://doi.org/10.1177/0333102417738202.
4. Dieterich M, Obermann M, Celebisoy N. Vestibular migraine: the most frequent entity of episodic vertigo. *J Neurol* 2016; 263:S82-S89. https://doi.org/10.1007/s00415-015-7905-2
5. Lempert T, Olesen J, Furman J, *et al*. Vestibular migraine: diagnostic criteria. *J Vest Res* 2012; 22:167-72. https://doi.org/10.3233/VES-2012-0453.
6. Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, 3rd edition. *Cephalalgia* 2018; 38:1-211. https://doi.org/10.1177/0333102417738202.
7. Formeister EJ, Rizk HG, Kohn MA, Sharon JD. The epidemiology of vestibular migraine: a population-based survey study. *Otol Neurotol* 2018; 39:1037-44. https://doi.or/10.1097/MAO.0000000000001900
8. Lee H, Sohn SI, Jung DK, *et al*. Migraine and isolated recurrent vertigo of unknown cause. *Neurol Res* 2002; 24:663-665. https://doi.org/10.1179/016164102101200726.
9. Cha YH, Lee H, Santell LS, Baloh RW. Association of benign recurrent vertigo and migraine in 208 patients. *Cephalalgia* 2009; 29:550-5. https://doi.org/10.1111/j.1468-2982.2008.01770.x.
10. Cho SJ, Kim BK, Kim BS, *et al*. Vestibular migraine in multicenter neurology clinics according to the appendix criteria in the third beta edition of the international classification of headache disorders. *Cephalalgia* 2016; 36:454-62. https://doi.org/10.1177/0333102415597890.
11. Yollu U, Uluduz DU, Yilmaz M, *et al*. Vestibular migraine screening in a migraine diagnosed patient population, and assessment of vestibulocochlear function. Clin Otolaryngol 2017; 42:225-33. https://doi.org/10.1111/coa.12699.
12. Furman JM, Marcus DA, Balaban CD. Vestibular migraine: clinical aspects and pathophysiology. Lancet Neurol 2013; 12:706-15. https://doi.org/10.1016/S1474-4422(13)70107-8.
13. Cohen JM, Bigal ME, Newman LC. Migraine and vestibular symptoms—identifying clinical features that predict "vestibular migraine". *Headache* 2011; 51:1393-7. https://doi.org/10.1111/j.1526-4610.2011.01934.x.
14. Teggi R, Colombo B, Albera R, *et al*. Clinical features, familial history and migraine precursors in patients with definite vestibular migraine: the VM phenotypes projects. *Headache* 2018; 58:534-44. https://doi.org/10.1111/head.13240.
15. Neff BA, Staab JP, Eggers SD, *et al*. Auditory and vestibular symptoms and chronic subjective dizziness in patients with Meniere's disease, vestibular migraine, and Meniere's disease with concomitant vestibular migraine. *Otol Neurotol* 2012; 33:1235-44. https://doi.org/10.1097/MAO.0b013e31825d644a.
16. Radtke A, von Brevern M, Neuhauser H, Hottenrott T, Lempert T. Vestibular migraine: long-term follow-up of clinical symptoms and vestibulo-cochlear findings. *Neurology* 2012; 79:1607-14. https://doi.org/10.1212/WNL.0b013e31826e264f.
17. Lempert T, von Brevern M. Vestibular migraine. *Neurol Clin* 2019;37:695-706. https://doi.org/10.1016/j.ncl.2019.06.003.
18. Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo, and migrainous vertigo. *Neurology 2001;* 56:436-41. https://doi.org/10.1212/WNL.56.4.436.

19. Murdin L, Chamberlain F, Cheema S, *et al*. Motion sickness in migraine and vestibular disorders. *J Neurol Neurosurg Psychiatry* 2015; 86:585-7. http://dx.doi.org/10.1136/jnnp-2014-308331.
20. Cass SP, Furman JM, Ankerstjerne K, Balaban C, Yetiser S, Aydogan B. Migraine-related vestibulopathy. *Ann Otol Rhinol Laryngol* 1997; 106:182-9. https://doi.org/10.1177/000348949710600302.
21. Stewart WF, Roy J, Lipton RB. Migraine prevalence, socioeconomic status, and social causation. *Neurology* 2013; 81:948-55. https://doi.org/10.1212/WNL.0b013e3182a43b32.
22. Dieterich M, Brandt T. Episodic vertigo related to migraine. *J Neurol* 1999; 246:883-92. https://doi.org/10.1007/s004150050478.
23. Rasmussen BK, Olesen J. Migraine with aura and migraine without aura: an epidemiological study. *Cephalalgia* 1992; 12:221-8. https://doi.org/10.1046/j.1468-2982.1992.1204221.x.
24. Matías-Guiu J, Porta-Etessam J, Mateos V, Diaz-Insa S, Lopez-Gil A, Fernandez C. One-year prevalence of migraine in Spain: a nationwide population-based survey. *Cephalalgia* 2010; 31:463-70. https://doi.org/10.1177/0333102410382794.
25. Dieterich M, Brandt T. Episodic vertigo related to migraine. *J Neurol* 1999; 246:883-92. https://doi.org/10.1007/s004150050478.
26. Silberstein SD. Migraine symptoms: results of a survey of self-reported migraineurs. *Headache* 1995; 35:387-96. https://doi.org/10.1111/j.1526-4610.1995.hed3507387.x.
27. Friedman DI, Evans RW. Are blurred vision and short-duration visual phenomena migraine aura symptoms? *Headache* 2017; 57:643-7. https://doi.org/10.1111/head.13042.
28. Kelman L. The place of osmophobia and taste abnormalities in migraine classification: a tertiary care study of 1237 patients. *Cephalalgia* 2004; 24:940-6. https://doi.org/10.1111/j.1468-2982.2004.00766.x.
29. Bigal ME, Ashina S, Burstein R, *et al.* Prevalence and characteristics of allodynia in headache sufferers: a population study. *Neurology* 2008; 70:1525-33. https://doi.org/10.1212/01.wnl.0000310645.31020.b1.
30. Selby G, Lance JW. Observations on 500 cases of migraine and allied vascular headache. *J Neurol Neurosurg Psychiatry* 1960; 23:23-32. https://doi.org/10.1136/jnnp.23.1.23.
31. Kelman L. The triggers or precipitants of the acute migraine attack. *Cephalalgia* 2007; 27:394-402. https://doi.org/10.1111/j.1468-2982.2007.01303.x.
32. Peck KR, Johnson YL, Smitherman TA. Migraine. Handb. Clin Neurol 2016;138:283-93. https://doi.org/10.1016/B978-0-12-802973-2.00016-1.
33. Furman, JM, Marcus, DA. Migraine and motion sensitivity. *Continuum* (Minneap Minn) 2012; 18:1102-17. https://doi.org/10.1212/01.CON.0000290483.03247.57.
34. Boldingh MI, Ljøstad U, Mygland A, Monstad P. Comparison of interictal vestibular function in vestibular migraine vs migraine without vertigo. *Headache* 2013;53:1123-33. https://doi.org/10.1111/head.12129.
35. Kırkım G, Mutlu B, Olgun Y, *et al*. Comparison of audiological findings in patients with vestibular migraine and migraine. *Turk Arch Otorhinolaryngol* 2017; 55:158-61. https://doi.org/10.5152/tao.2017.2609.
36. Xue J, Ma X, Lin Y, Shan H, Yu L. Audiological findings in patients with vestibular migraine and migraine: history of migraine may be a cause of low-tone sudden sensorineural hearing loss. *Audiol Neurotol* 2020; 25:209-14. https://doi.org/10.1159/000506147.
37. Chang TP, Hsu YC. Vestibular migraine has higher correlation with carsickness than non-vestibular migraine and Meniere's disease. *Acta Neurol Taiwan* 2014; 23:4-10.
38. Kutay Ö, Akdal G, Keskinoğlu P, Balci BD, Alkin T. Vestibular migraine patients are more anxious than migraine patients without vestibular symptoms. *J Neurol* 2017; 264:37-41. https://doi.org/10.1007/s00415-017-8439-6.
39. Abouzari M, Goshtasbi K, Moshtaghi O, Donald T, Harrison WL, Hamid RD. Association between vestibular migraine and migraine headache: yet to explore. *Otol Neurotol* 2020; 41:392-6. https://doi.org/10.1097/MAO.0000000000002528.