Original Article / *Özgün Araştırma*

DOI: 10.4274/haseki.galenos.2021.6439
Med Bull Haseki 2021;59:25-31

The Relationship Between the Long-Term Non-Rehabilitated Benign Paroxysmal Positional Vertigo and Vestibular Hypofunction

Uzun Süre Rehabilite Edilmemiş Benign Paroksismal Pozisyonel Vertigo ve Vestibüler Hipofonksiyon İlişkisi

Dastan Temirbekov, Erdal Sakallı, Gamze Kılıç*

İstanbul Aydin University Faculty of Medicine, Department of Otorhinolaryngology, İstanbul, Turkey
**Erdem Hospital, Clinic of Physical Therapy and Rehabilitation, İstanbul, Turkey*

Abstract

Aim: Recurrent Benign paroxysmal positional vertigo (BPPV) and vestibular hypofunction (VH) can be co-diagnosed in some patients. We aimed to sight the relationship between the recurrent BPPV and VH, and to evaluate the change in the VH prevelance according to recurrent BPPV duration.

Methods: We conducted a retrospective chart review of 416 patients who were diagnosed with recurrent BPPV. Demographic features of the patients, co-diagnosis frequency of recurrent BPPV and VH, and the change in the VH prevelance according to recurrent BPPV duration were recorded. Patients were divided into two groups for having BPPV attacks for more than 12 months or less than 12 months. Both groups were compared in terms of VH prevalence.

Results: VH was co-diagnosed in 61.7% of patients with BPPV. The age of the recurrent BPPV patients with VH was significantly higher than the patients without VH ($p<0.05$). VH positivity was directly correlated with the duration of recurrent BPPV. VH prevelance was significantly higher in the group that the BPPV duration was above 12 months than in the group that the recurrent BPPV duration was under 12 months ($p<0.05$).

Conclusion: VH is common in patients with recurrent BPPV. There is may be a causal relationship between BPPV and VH.

Keywords: Benign paroxysmal positional vertigo, peripheral vestibulopathy, vestibular hypofunction, BPPV, vestibulopathy

Öz

Amaç: Benign paroksismal pozisyonel vertigo (BPPV) ve vestibüler hipofonksiyon (VH) bazı hastalarda birlikte görülebilir. Çalışmamızda, BPPV ve VH arasındaki ilişkiyi gözlemlemeyi ve tekrarlayan BPPV süresine göre VH görülme sıklığındaki değişimi araştırmayı amaçladık.

Yöntemler: BPPV tanısı almış 416 hastanın geriye dönük dosya incelemesi yapıldı. Hastaların demografik özellikleri, BPPV ve VH birlikteliğindeki sıklık ve de BPPV süresine göre VH görülme sıklığındaki değişim kaydedildi. Hastalar 12 aydan daha uzun süredir tekrarlayan ve son 11 ay içinde gelişen ve/veya tekrarlayan BPPV'li olmak üzere iki gruba ayırıldı. Her iki gruptaki hastalar VH sıklığı açısından karşılaştırıldı.

Bulgular: BPPV'li hastaların %61,7'sinde ayrıca VH tanısı konuldu. VH ile birlikte olan BPPV'li hastaların yaş ortalaması, VH ile birlikte olmayan BPPV'li hastaların yaş ortalamasına göre anlamlı olarak yüksekti (PC 0,05). VH ile birlikte olan veya olmayan BPPV'li hastaların cinsiyet dağılımında anlamlı fark yoktu ($p<0,05$). VH pozitifliği ile tekrarlayan BPPV süresi doğru orantılı idi. Tekrarlayan BPPV süresi 12 aydan uzun olan gruptaki VH sıklığı, BPPV süresi 12 aydan kısa olan gruba göre anlamlı olarak yüksekti ($p<0,05$).

Sonuç: Uzun süredir tekrarlayan BPPV'li hastalarda VH görülmesi yaygındır. VH ile BPPV'nin bir sebep-sonuç ilişkisi olabilir.

Anahtar Sözcükler: Benign paroksismal pozisyonel vertigo, periferik vestibülopati, vestibüler hipofonksiyon, BPPV, vestibülopati

Address for Correspondence/Yazışma Adresi: Erdal Sakallı, İstanbul Aydin University Faculty of Medicine, Department of Otorhinolaryngology, İstanbul, Turkey
Phone: +90 533 441 77 10 E-mail: erdalkbb1979@hotmail.com ORCID: orcid.org/0000-0003-1582-1816
Received/Geliş Tarihi: 29.07.2020 **Accepted/Kabul Tarihi:** 12.11.2020

Introduction

Peripheral vestibular dysfunction is one of the most common reasons for applying to the hospitals. The most common cause of peripheral vestibular dysfunction is benign paroxysmal positional vertigo (BPPV) (1). BPPV is characterized by short (seconds to minutes) recurrent episodes of positional vertigo and dizziness provoked by changes in head position. Displaced otoconia (canalolithiasis or cupulolithiasis) are thought to cause BPPV by mechanically stimulating the vestibular receptors within the semicircular canals (2,3).

The second most common cause of peripheral vestibular dysfunction is vestibular hypofunction (VH), which can be described as a disturbance of the vestibulo-ocular reflex (VOR) in one or both of the inner ears (4). The VOR assists in maintaining gaze stability, which allows the eyes to maintain focus on a target while the head is moving. As the VOR is disturbed in VH, tracking of moving objects becomes difficult, which is an important symptom of VH (1,5).

Among patients with BPPV or VH who have not undergone appropriate medical treatment, intentional movement restriction and hypodynamics are often experienced due to fear of falling (6). Previous studies have shown that leading a sedentary lifestyle and hypodynamics have negative effects on the balance system, while sports and movement have positive effects on it (7,8). In our clinical experiences, we often observe VH symptoms in BPPV patients even after successful canalith repositioning. Thus, we speculated that prolonged movement restriction may cause secondary VH.

We aimed to investigate the frequency of co-diagnosis of BPPV and VH and also to find the relationship between the VH prevelance and recurrent BPPV duration.

Methods

Patients who were admitted to our audio-vestibular center between January 2016 and January 2020 and underwent detailed vestibular examination were recruited for this study. The study protocol was approved by the Ethics Committee of İstanbul Aydın University, Faculty of Medicine (number: 2020/240). Patients between 18 and 65 years of age who were diagnosed with BPPV were included in the study. The diagnosis of BPPV was made according to the 2015 Barany Society criteria (9). The onset of dizziness of every subject was determined by examining the history of the selected patients. The patients were divided into two groups depending on for how long the recurrent BPPV had been troubling, as less than 12 months (control group) and 12 months or more (study group). The both groups patients were investigated in terms of VH. During the evaluation of VH, the Dizziness Handicap Inventory Questionnaire (validated Turkish version) was completed by patients according to their anamnesis (10). Videonystagmography (VNG) recorded vestibular examination, including saccade test, tracking test, optokinetic test, gaze test, Dix-Halpike maneuver, air-stimulated binaural bithermal caloric test, fixation suppression test, baseline shift, gain asymmetry were taken into consideration. VNG equipment from Otometrics (ICS Chartr 200; Taastrup, Denmark) and a computerized system were used for VH diagnosis. PC-VNG software was used for automatic analysis of the recordings. The following criterias were used to diagnose VH.

A. Chronic vestibular syndrome with the following symptoms:

1. Unsteadiness when walking or standing plus at least one of 2 or 3.

2. Movement-induced blurred vision or oscillopsia during walking or quick head/body movements and/or.

3. Worsening of unsteadiness in darkness and/or on uneven ground.

B. No symptoms while sitting or lying down under static conditions

C. Bilateral weakness in the caloric response was considered based on the 2017 Barany Society criteria (11): Bilaterally reduced or absent angular VOR function documented by reduced caloric response (sum of bithermal maximum peak Slow-phase velocity on each side <6◦/sec).

D. Unilateral weakness in the caloric response was quantified according to the Jongkees formula. A value greater than or equal to 25% was pathological according to our normative data. Additionally, the directional preponderance (DP) was measured using the formula DP=[(RW+LC)-(LW+RC)]/(RW+LW+RC+LC)x100. Normal absolute values in our lab are below a DP of 30%.

E. Not better accounted for by another disease

The both groups patients were statistically compared in terms of VH frequency. The demographic characteristics of the patients, relationship between BPPV duration and VH prevelance, involved semicircular canal, and involved ear were investigated and analyzed.

Patients with any additional otologic pathologies that can cause VH (including endolymphatic hydrops, history of labyrinthitis, vestibular neurinitis, trauma, and meningitis), patients with additional neurologic or orthopedic pathologies, patients less than 18 or more than 65 years of age, patients with incomplete anamnesis and/or questionnaire, and patients who had not undergone VNG recorded vestibular examination were excluded from the study. Patients whose anamnesis suggested that VH developed before BPPV occurred were also excluded from the study.

Statistical Analysis

Mean, standard deviation, median, lowest and highest frequency, and ratio values were used as descriptive statistics of the data. The distribution of variables was measured using the Kolmogorov-Smirnov test. In the analysis of quantitative independent data, the Mann-Whitney U test was used. The chi-square test was used for the analysis of qualitative independent data. The Statistical Package for the Social Sciences (SPSS) 22.0 program was used in the analysis.

Results

Of the 416 patients diagnosed with BPPV who matched our study criteria, 257 (61.7%) had VH, and 159 (38.2%) did not have VH. The demographic features and VNG records of the BPPV patients are shown in Table 1. The age of the patients with VH was significantly higher than the age of the patients without VH ($p<0.05$). There was no significant difference in gender distribution between the BPPV patients with and without VH ($p<0.05$). There was no significant difference between the groups in terms of saccade positivity, gaze positivity, tracking test positivity, and optokinetic test positivity ($p>0.05$) (Table 2). In the patients with VH, the duration of the BPPV was significantly longer than in the patients without VH ($p<0.05$) (Figure 1). We found that the duration of BPPV was directly correlated with VH positivity (Figure 2). In the study group (BPPV duration ≥12 month), VH positivity was significantly higher than in the control group (BPPV duration <12 month) ($p<0.05$) (Figure 3). The age of the patients with bilateral VH was significantly higher than the age of the patients with unilateral VH ($p<0.05$). There were no statistically significant differences in patients with bilateral or unilateral VH ($p<0.05$) in terms of gender distribution, duration of BPPV, and optokinetic test results (Table 3).

Discussion

BPPV has been shown to be related with otologic disorders, including sudden idiopathic hearing loss, vestibular neuritis, and Meniere's disease (12-14). BPPV may develop after head trauma or inner ear surgery (stapes surgery, cochlear implantation, or when repairing superior canal dehiscence) (15). Patients with an acute

Table 1. Demographic features and videonystagmography records of the BPPV patients

		Min-Max			Median	Mean ± SD/N-%		
Age (years)		7.0	-	91.0	50.0	49.0	±	16.0
Sex	Female					280		67.5%
	Male					136		32.8%
BPPV time (months)		0.1	-	420.0	5.3	30.7	±	60.8
Saccade	(-)					383		92.3%
	(+)					33		8.0%
Gaze	(-)					416		100.2%
	(+)					0		0.0%
Tracking	(-)					298		71.8%
	(+)					118		28.4%
Optokinetic	(-)					299		72.0%
	(+)					117		28.2%
ROLL test	(-)					389		93.7%
	(+)					27		6.5%
DIX hallpike right	(-)					123		29.6%
	(+)					293		70.6%
DIX hallpike left	(-)					136		32.8%
	(+)					280		67.5%
Vestibular hypofunction	(-)					159		38.3%
	(+)					257		61.9%
-	Right					107		25.8%
-	Left					101		24.3%
-	Bilateral					49		11.8%

Min: Minimum value, Max: Maximum value, ± SD/N: Standart deviation/number, BPPV: Benign paroxysmal positional vertigo

Table 2. Comparison of the patients regarding BPPV duration and VH positivity											
		Vestibular Hypofunction (-)				Vestibular Hypofunction (+)				p	
		Mean ± SD/n-%			Median	Mean ± SD/n-%			Median		
Age (years)		46.3	±	16.5	47.5	50.6	±	15.4	51.0	0.015	m
Sex	Female	110		69.2%		170		66.1%		0.521	X²
	Male	49		30.8%		87		33.9%			
BPPV time (months)		17.7	±	43.9	1.0	38.8	±	68.1	12.0	0.000	m
BPPV time (monhts)	≤12 >12	120 39		75.5% 24.5%	1.0	139 118		54.1% 45.9%		0.000	X²
BPPV	(-) (+)	0 159		0% 100%		0 257		0.0% 100%		1.000	X²
Saccade	(-)	148		93.1%		235		91.4%		0.547	X²
	(+)	11		6.9%		22		8.6%			
Gaze	(-)	159		100.0%		257		100.0%		1.000	X²
	(+)	0		0.0%		0		0.0%			
Tracking	(-)	114		71.7%		184		71.6%		0.982	X²
	(+)	45		28.3%		73		28.4%			
Optokinetic	(-)	115		72.3%		184		71.6%		0.872	X²
	(+)	44		27.7%		73		28.4%			
ROLL test	(-)	146		91.8%		243		94.6%		0.272	X²
	(+)	13		8,2%		14		5,4%			
DIX hallpike right	(-)	55		34.6%		68		26.5%		0.077	X²
	(+)	104		65.4%		189		73.5%			
DIX hallpike left	(-)	60		37.7%		76		29.6%		0.085	X²
	(+)	99		62.3%		181		70.4%			

Min: Minimum value, Max: Maximum value, ± SD/N: Standart deviation/number, m: Mann-whitney u test, x2: Chi-square test, BPPV: Benign paroxysmal positional vertigo, VH: Vestibular hypofunction

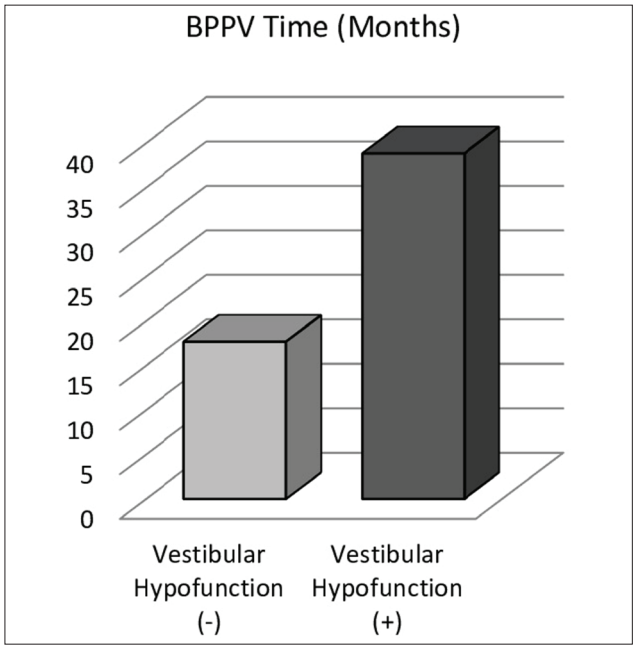

Figure 1. The duration of BPPV between the patients with and without vestibular hypofunction
BPPV: Benign paroxysmal positional vertigo

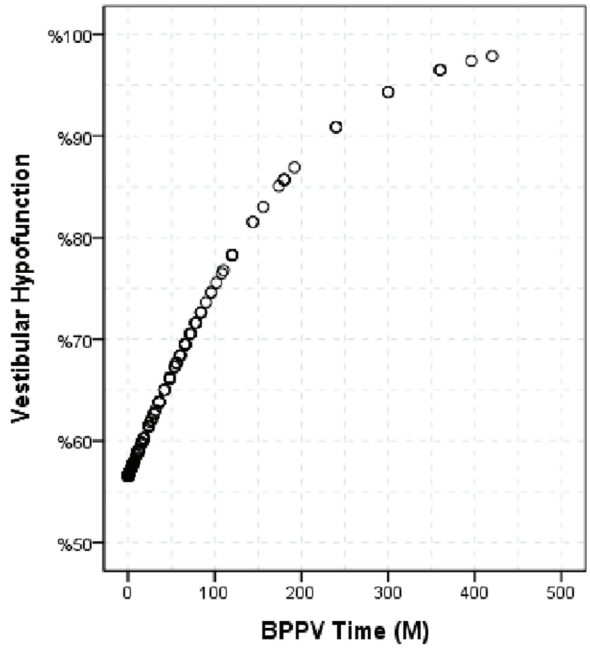

Figure 2. The correlation between duration of BPPV and VH
BPPV: Benign paroxysmal positional vertigo, VH: Vestibular hypofunction

and/or chronic unilateral peripheral vestibulopathy can develop recurrent posterior canal BPPV in the same ear (16,17).

In a recent study, it was reported that patients with a history of BPPV and otologic disease were more likely to have VH than those without a history of otologic disease (18). However, the relationship between VH and BPPV has not been comprehensively described in the literature. There is only one study, conducted by Summer et al. (19) in 2012, in which the rate of co-diagnosis of VH and BPPV,

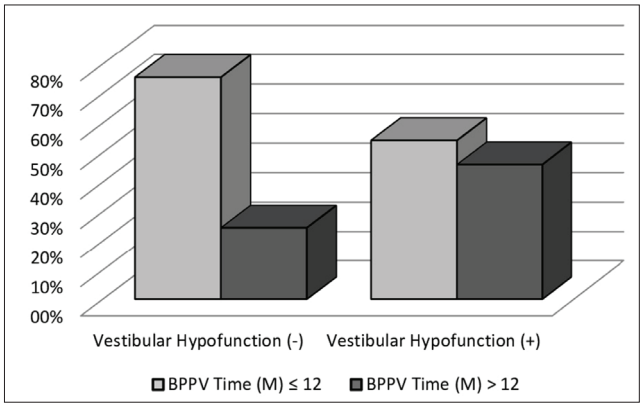

Figure 3. Comparison of vestibular hypofunction prevelance between the patients who have BPPV more than 12 months and less than 12 months
BPPV: Benign paroxysmal positional vertigo

the frequency of admission to physical therapy, and the relationship with patients' ages were determined. They retrospectively reviewed 500 medical records of patients diagnosed with VNG. They found that 38% of their patients had a single diagnosis, and 6.6% of their patients had a co-diagnosis. They also found a positive relationship between VH-BPPV co-diagnosis and increased age. We also found significantly higher ages in BPPV patients with VH than in patients without VH (19). However, it is possible that the increase in co-diagnosis with age may simply be due to the increased incidence of both VH and BPPV in the elderly.

Physical activity including head and body movements is important factor for recovering from vestibular dysfunctions. On the other hand, prolonged dizziness caused by vestibulopathies including BPPV is a risk factor for developing anxiety (20). Therefore, patients with untreated BPPV tend to be less active as a result of balance problems and anxiety (6,20). Morimoto et al. (6) reported a study in which they provided objective measures of physical activity in patients with chronic unilateral vestibular hypofunction. They found direct correlation between physical activity and postural stability (6).

In the study by Summer M. and San Lucas (19), they did not determine the time course of VH and BPPV. It is possible that patients may have had VH years prior to VNG testing and developed BPPV near the time of VNG testing.

Table 3. Demographic features and videonystagmography records of the patients regarding unilateral and bilateral VH

		Vestibular hypofunction unilateral			Vestibular hypofunction bilateral				p	
	Mean ± SD			Median	Mean ± SD			Median		
Age (years)		51.9	±	15.2	52.0	45.3	±	15.4	44.0	0.009	m
Sex	Female	136		65.4%		34		69.4%		0.594	X²
	Male	72		34.6%		15		30.6%			
BPPV Time (months)		37.7	±	67.6	12.0	43.7	±	70.5	12.0	0.803	m
Saccade	(-)	191		91.8%		44		89.8%		0.648	X²
	(+)	17		8.2%		5		10.2%			
Gaze	(-)	208		100.0%		49		100.0%		1.000	X²
	(+)	0		0.0%		0		0.0%			
Tracking	(-)	151		72.6%		33		67.3%		0.464	X²
	(+)	57		27.4%		16		32.7%			
Optokinetic	(-)	151		72.6%		33		67.3%		0.464	X²
	(+)	57		27.4%		16		32.7%			
ROLL test	(-)	200		96.2%		43		87.8%		0.020	X²
	(+)	8		3.8%		6		12.2%			
DIX-hallpike right	(-)	57		27.4%		11		22.4%		0.479	X²
	(+)	151		72.6%		38		77.6%			
DIX-hallpike left	(-)	67		32.2%		9		18.4%		0.056	X²
	(+)	141		67.8%		40		81.6%			

Min: Minimum value, Max: Maximum value, ± SD/N: Standart deviation/number, m: Mann-whitney v test, x2: Chi-square test, BPPV: Benign paroxysmal positional vertigo

In our study, we recruited only BPPV patients. Out of the 416 BPPV patients, 257 (61.7%) had VH, and 159 (38.2%) did not have VH. These remarkably different results may be due to the fact that our clinic is a referral center, and specific patients are admitted oftenly. For the same reason, we have had many BPPV cases that have not been treated for a long time.

We analyzed the correlation between the duration of recurrent BPPV and the frequency of VH in order to investigate the duration of recurrent BPPV as a risk factor for VH. It was remarkable that we found a positive correlation. Since our audio-vestibular center is a tertiary center, we noted that most of our patients had previous examinations and treatments in other clinics and had not benefited or had recurrences. Thus, we excluded patients when it was revealed that they had developed VH before BPPV. However, this exclusion was provided based on the patients' history. Conversely, Summer M. and San Lucas (19), reported that they found a significant association between peripheral vestibular dysfunction and non-referral to physical therapy after BPPV treatment. This data suggests that non-rehabilitated BPPV may contribute to the occurrence of VH.

In order to distinguish long- and short-term BPPV, we chose a relative period of 12 months. Statistical analysis could not prove its usefulness as a cut-off time. If a patient has multiple sources of dizziness and only one source is treated, it is likely that the treatment will be incomplete. Accurate identification of BPPV and VH comorbidities will provide an appropriate treatment strategy. In cases of co-diagnosis of BPPV and VH, it is recommended that BPPV, which can be resolved in one to three sessions, be treated first (21). Then, rehabilitation procedures can be initiated to treat VH.

The mean age of patients with VH was found to be significantly higher compared to those without VH (Table 2). It is known that vestibular response decreases with increasing age and old age is a risk factor for VH. But, considering the follow-up periods of our patients, the average age of the group with VH is 50 years, the average follow-up period is 38 months (three years), while the average age of those without VH is 46 years and the average follow-up period is 17 months (1 year). In other words, we see that the ages at which the first BPPV attack developed in both groups are close to each other. Therefore, although there appears to be a statistical age difference between the patient group with VH and the group without VH, we believe that this did not affect negatively our study results.

In conclusion, VH, which is very common in the elderly population, quite often diagnosed together with BPPV. Further investigations are needed regarding possible causal relationship of these conditions. We speculate that VH can develop because of prolonged movement restriction due to fear of falling. From this point of view early rehabilitation of BPPV can prevent VH development.

Authorship Contributions

Concept: D.T., Design: D.T., Data Collection or Processing: G.K., Analysis or Interpretation: E.S., Literature Search: D.T., E.S., Writing: E.S

Conflict of Interest: There is not any financial and personal relationships with other people or organisations. Also there is not any funding source.

Informed Consent: Consent form was filled out by all participants.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Sakalli E, Temirbekov D, Celikyurt C, Kılıç G, Practical diagnosis and management of peripheral vertigo. Praxis of Otorhinolaryngology 2017;5:57-62.
2. Mandalà M, Salerni L, Nuti D. Benign Positional Paroxysmal Vertigo Treatment: a Practical Update. Curr Treat Options Neurol 2019;21:66.
3. Nuti D, Masini M, Mandalà M. Benign paroxysmal positional vertigo and its variants. Handb Clin Neurol 2016;137:241-56.
4. Walther LE. Current diagnostic procedures for diagnosing vertigo and dizziness. GMS Curr Top Otorhinolaryngol Head Neck Surg 2017;16:Doc02.
5. Uffer DS, Hegemann SC. About the pathophysiology of acute unilateral vestibular deficit - vestibular neuritis (VN) or peripheral vestibulopathy (PVP)? J Vestib Res 2016;26:311-7.
6. Morimoto H, Asai Y, Johnson EG, et al. Objective measures of physical activity in patients with chronic unilateral vestibular hypofunction, and its relationship to handicap, anxiety and postural stability. Auris Nasus Larynx 2019;46:70-7.
7. Krasnoff J, Painter P. The physiological consequences of bed rest and inactivity. Adv Ren Replace Ther 1999;6:124-32.
8. Leong HT, Fu SN, Ng GY, Tsang WW. Low-level Taekwondo practitioners have better somatosensory organisation in standing balance than sedentary people. Eur J Appl Physiol 2011;111:1787-93.
9. von Brevern M, Bertholon P, Brandt T, et al. Benign paroxysmal positional vertigo: Diagnostic criteria. J Vestib Res 2015;25:105-17.
10. Canbal M, Cebeci S, Duyan GÇ, Kurtaran H, Arslan İ. A study of reliability and validity for the Turkish version of dizziness handicap inventory. Turk. J. Family Med. Prim. Care 2016;10:19-24.
11. Strupp M, Kim JS, Murofushi T, et al. Bilateral vestibulopathy: Diagnostic criteria Consensus document of the Classification

Committee of the Bárány Society. J Vestib Res 2017;27:177-89.

12. Balatsouras DG, Ganelis P, Aspris A, Economou NC, Moukos A, Koukoutsis G. Benign paroxysmal positional vertigo associated with Meniere's disease: epidemiological, pathophysiologic, clinical, and therapeutic aspects. Ann Otol Rhinol Laryngol 2012;121:682-8.
13. Kim MB, Ban JH. Benign paroxysmal positional vertigo accompanied by sudden sensorineural hearing loss: a comparative study with idiopathic benign paroxysmal positional vertigo. Laryngoscope 2012;122:2832-6.
14. Türk B, Akpinar M, Kaya KS, Korkut AY, Turgut S. Benign Paroxysmal Positional Vertigo: Comparison of Idiopathic BPPV and BPPV Secondary to Vestibular Neuritis. Ear Nose Throat J 2019. DOI: 10.1177/0145561319871234
15. Kao WT, Parnes LS, Chole RA. Otoconia and otolithic membrane fragments within the posterior semicircular canal in benign paroxysmal positional vertigo. Laryngoscope 2017;127:709-14.
16. Karlberg M, Hall K, Quickert N, Hinson J, Halmagyi GM. What inner ear diseases cause benign paroxysmal positional vertigo? Acta Otolaryngol 2000;120:380-5.
17. Meghji S, Murphy D, Nunney I, Phillips JS. The Seasonal Variation of Benign Paroxysmal Positional Vertigo. Otol Neurotol 2017;38:1315-8.
18. Sloane P, Blazer D, George LK. Dizziness in a community elderly population. J Am Geriatr Soc 1989;37:101-8.
19. Summer M, San Lucas. Co-diagnosis frequency of peripheral vestibular disorders and physical therapy. Loma Linda University Electronic Theses, Dissertations & Projects.97. 2012.
20. Azevedo Da Silva M, Singh-Manoux A, Brunner EJ, Kaffashian S, Shipley MJ, Kivimäki M, et al. Bidirectional association between physical activity and symptoms of anxiety and depression: the Whitehall II study. Eur J Epidemiol 2012;27:537-46.
21. Herdman SJ. Canalith repositioning maneuver. Otolaryngol Head Neck Surg 1994;111:691-2.