B-ENT, 2015, **11**, 211-218

Do viral infections have a role in benign paroxysmal positional vertigo?

D. Hanci[1], *S. Ulusoy*[2], *N. B. Muluk*[3] *and C. Cingi*[4]

[1]ENT Specialist Doctor in Okmeydanı Training and Research Hospital, ENT Clinics, Istanbul, Turkey; [2]TR Ministry of Health, Taksim Ilkyardım Training and Research Hospital, ENT Clinics, Istanbul, Turkey; [3]Professor in Kırıkkale University, Medical Faculty, ENT Department, Kırıkkale, Turkey; [4]Professor in Eskisehir Osmangazi University, Medical Faculty, ENT Department, Eskisehir, Turkey

Key-words. Benign paroxysmal positional vertigo (BPPV); viruses; season

Abstract. *Do viral infections have a role in benign paroxysmal positional vertigo? Objectives*: To investigate the role of viral infection in benign paroxysmal positional vertigo (BPPV).
Methods: In this retrospective study, 483 patients with BPPV were included in the study group. The control group consisted of 461 healthy subjects. In both groups, serologic analysis of viral agents (HSV1, HSV2, Herpes zoster, EBV, CMV, adenovirus, influenza, and parainfluenza virus) was performed.
Results: With the exception of influenza and parainfluenza, all viral serology values were higher in the BBPV group than the control group. We also observed seasonal variation. The BPPV group exhibited elevated values for HSV1 and adenovirus in March and May, for Herpes zoster, adenovirus, and influenza in April, for HSV1 in June, and for HSV1 and CMV in September, compared to the control group. In October, the BPPV group showed increased values for all of the viruses studied, compared to the control group.
Conclusion: BPPV is associated with positive viral serology, particularly during certain months of the year, mainly in spring and autumn. Viral infection might promote BPPV attacks due to the development of vestibulopathy or induce secondary BPPV via viral infection-related neurolabyrinthitis.

Introduction

Among patients experiencing dizziness, 65% may receive a vestibular etiologic diagnosis such as benign paroxysmal positional vertigo (BPPV). The diagnosis of BPPV is based on medical history and Dix-Hallpike test findings.[1] Typical BPPV is characterized by episodes of rotational vertigo of less than one minute in duration, which are brought on by changes in head position. The condition is thought to be caused by free-floating otoconial debris that is displaced from the utricle into the semicircular canals.[2] The Dix-Hallpike test is the standard by which the diagnosis of posterior semicircular canal BPPV is made.[3] During this test, a person is brought from sitting to a supine position, with the head turned 45 degrees to one side and extended about 20 degrees backward. Once supine, the eyes are typically observed for about 30 seconds. If no nystagmus ensues, the person is brought back to sitting. There is a delay of about 30 seconds again, and then the other side is tested.[4]

There are two forms of BPPV, canalithiasis and cupulolithiasis. Canalithiasis is the more common of the two, which occurs when otoconia move within the semicircular canal, causing vertigo and nystagmus that resolves within 60 seconds. Cupulolithiasis occurs when otoconia adhere to the cupula, causing vertigo and nystagmus that persist for a longer period of time.[5] A theory explaining cupulolithiasis was proposed by Harold Schuknecht in 1962. He observed basophilic particles or densities that were adherent to the cupula, and postulated that the posterior semicircular canal (PSC) was rendered sensitive to gravity by attachment or impingement of these abnormal dense particles upon the cupula. This situation is analogous to the attachment of a heavy object to the top of a pole. The extra weight makes the pole unstable and thus harder to maintain in the neutral position.[5] A theory explaining canalithiasis was proposed by Epley in 1980.[6,7] He found that the symptoms of BPPV seemed more consistent with free-moving densities (canaliths) in the posterior semicircular canal (PSC) than with fixed densities attached to the cupula. While the head is upright, the particles sit in the PSC at a gravity-determined position. When the head is tilted back into a supine

position, the particles are rotated by approximately 90° along the arc of the PSC. After a momentary inertial lag period, gravity pulls the particles down the arc.[6,7]

Karlberg, *et al*.[8] reported 4 patients who presented with BPPV and sensorineural hearing loss (SSNHL). The caloric test was normal in all subjects; however, in our patients the caloric test was mildly abnormal in two patients, which may indicate other or additional pathogenesis in these cases[8]. Caloric test is performed while patients lying supine (on the back), head elevated 30 degrees, cool and warm air or water is infused into the ear canal. The air or water cools or warms the fluid in the horizontal semicircular canal, which is on the other side of the eardrum. This creates a current (cooler fluid is more dense and moves toward the ground while warmer fluid is less dense and moves away from the ground) with the end result being the creation of nystagmus. Cooling of the inner ear fluid creates nystagmus away from the tested ear and warming of the inner ear fluid creates nystagmus toward the tested ear[9]. Viral infection or viral reactivation within the inner ear could cause inflammation and damage critical inner ear structures, and has long been suspected as a possible cause of both acute-unilateral-vestibulopathy and of SSNHL. The simultaneous involvement may be explained by a possible "patchy pattern" in the nerves or labyrinth, such as is seen in other viral diseases and Bell's palsy, caused by HSV I or Herpes zoster oticus (Ramsey-Hunt syndrome).[8,10] In the present study, we investigated the role of viral infection in 483 BPPV patients by serologic study, compared with 461 healthy controls.

Materials and methods

This retrospective study was conducted in Acıbadem Fulya Hospital, ENT Clinics, Istanbul, Turkey, between 2009-2010. All procedures were performed in accordance with the Declaration of Helsinki.[11] Ethics Committee approval was granted by Bakırköy Dr. Sadi Konuk, Training and Research Hospital, Clinical Research Ethics Committee on May 5, 2014; Number: 2014/07/28.

Subjects

A total of 483 patients who were diagnosed with BPPV were included in the study group (Group 1). The mean age of the subjects was 47.1±11.7 years, ranging from 12.0 to 74.0 years. The study group included 245 males (50.7%) and 238 females (49.3%). The Dix-Hallpike exam was performed; right-sided involvement was detected in 314 patients (65.0%), and left-sided involvement in 169 (35.0%). Patients were treated by Epley maneuver, with a mean of 2.4±0.8 treatments performed (maximum of 4.0). The control group (Group 2) comprised 461 healthy subjects without BPPV who were relatives of the BPPV patients. The mean age of the control individuals was 39.9±9.7 years, ranging from 18.0 to 74.0 years. The control group included 237 males (49.2%) and 224 females (48.5%).

The following exclusion criteria were applied: a diagnosis other than BPPV with respect to the vertigo, malignant diseases, head and neck trauma, epilepsy, or known neurological diseases. Patients who met any of these criteria were not included in the study.

Epley maneuver

Epley maneuver was performed as following: First, while sitting up, the patients head is turned about 45 degrees to the side that normally provokes the vertigo. Then the patient is quickly laid down backwards with their head just over the edge of the examining table. This position usually provokes strong vertigo. The head is kept in this position for about 30 seconds and then turned 90 degrees to the opposite side. After another 30 seconds, the head and the body are turned together in the same direction so that the body is pointing towards the side, and the head is pointing down toward the ground at a 45 degree angle. After 30 seconds in this position, the patient is brought upright again. This is repeated as many as five or six times until neither vertigo nor nystagmus are elicited when the head is brought into the bad ear down position.[12]

Methods

Patient history was taken, including all diseases, balance problems, motion sickness, migraine, previous vertigo, nausea, vomiting, hearing loss, and eye problems (blurred vision, double vision, eyeglass usage). An ENT examination was performed, including the Dix-Hallpike exam.

In both groups, a serologic analysis of viral agents was performed via IgM antibody in a venous

blood sample. Analysis was performed on the first day of the BPPV attack and the measurement was repeated on the 14th day. Increased titration of IgM antibody was coded as (1), and unchanged IgM antibody titration was coded as (0).

The following viral agents were evaluated: Herpes simplex virus 1 (HSV 1), Herpes simplex virus 2 (HSV 2), Herpes zoster, Epstein Barr Virus (EBV), Cytomegalovirus (CMV), Adenovirus, Influenza virus, and Parainfluenza virus.

Statistical analysis

SPSS 16.0 software (SPSS Inc., Chicago, 2007) was used for the statistical analyses, including Mann Whitney U Test and Chi-square test. $P<0.05$ was defined as statistically significant.

Results

Table 1 summarizes the relevant data from the patient histories. In the BPPV group, the incidences of balance problems (37.3%), motion sickness (21.3%), migraine (27.5%), previous vertigo (54.0%), nausea (98.7%), and vomiting (76.6%) were found to be significantly higher than that in the control group, by Chi-square test ($P<0.05$).

Viral serology results are shown in Table 2. With the exception of influenza and parainfluenza, the serology results for all of the other viruses studied were significantly higher in the BBPV group than the control group, by Chi-square test ($P<0.05$). HSV1 was detected in 24.6%, adenovirus was detected in 16.3%, and CMV was detected in 7.5% of the patients with BPPV.

Table 3 and Figure 1 show the viral serology results separated by month (January to December). Monthly variability is observed. The BPPV group exhibited significantly higher values than the control group ($P<0.05$) for HSV1 in June; for HSV1 and adenovirus in March and May; for HSV1 and CMV in September; for Herpes zoster, adenovirus, and influenza in April; and for HSV1, HSV2, Herpes zoster, EBV, CMV, adenovirus, influenza and parainfluenza virus in October. When performing an analysis for each virus, in different months of the year, and in the patient and control groups separately, statistically significant differences were found in the CMV values of the BPPV group ($P<0.05$), which were higher in September (23.8%) and October (15.6%).

It was observed that there was a statistically significant difference in age between the experimental and control groups, as defined by Mann Whitney U test ($P=0.000$). There was no significant difference in the gender distribution between the two groups ($P=0.833$, $X^2=0.044$).

Discussion

Benign paroxysmal positional vertigo (BPPV) is defined by brief attacks of rotational vertigo and concomitant rotatory-linear nystagmus, which are triggered by rapid neck extension and lateral head tilt toward the affected ear. Cupulolithiasis of the

Table 1

Medical history

	BPPV group		Control group		*P**
	n (%)	n (%)	n (%)	n (%)	
	Present (+)	Absent (-)	Present (+)	Absent (-)	
Balance problems	180 (37.3)	303 (62.7)	9 (2.0)	450 (98.0)	$P=0.000$, $X^2=1.829$
Motion sickness	103 (21.3)	380 (78.7)	21 (4.6)	439 (95.4)	$P=0.000$, $X^2=57.950$
Migraine	133 (27.5)	350 (72.5)	24 (5.2)	435 (94.8)	$P=0.000$, $X^2=84.322$
Hearing loss**	4 (0.8)	477 (99.2)	0 (0.0)	459 (99.8)	$P=0.145$, $X^2=2.122$
Previous vertigo	261 (54.0)	222 (46.0)	5 (1.1)	456 (98.9)	$P=0.000$, $X^2=3.268$
Nausea	471 (98.7)	12 (2.5)	6 (1.3)	455 (97.4)	$P=0.000$, $X^2=8.735$
Vomiting	369 (76.6)	113 (23.4)	2 (0.4)	459 (99.6)	$P=0.000$, $X^2=572.200$

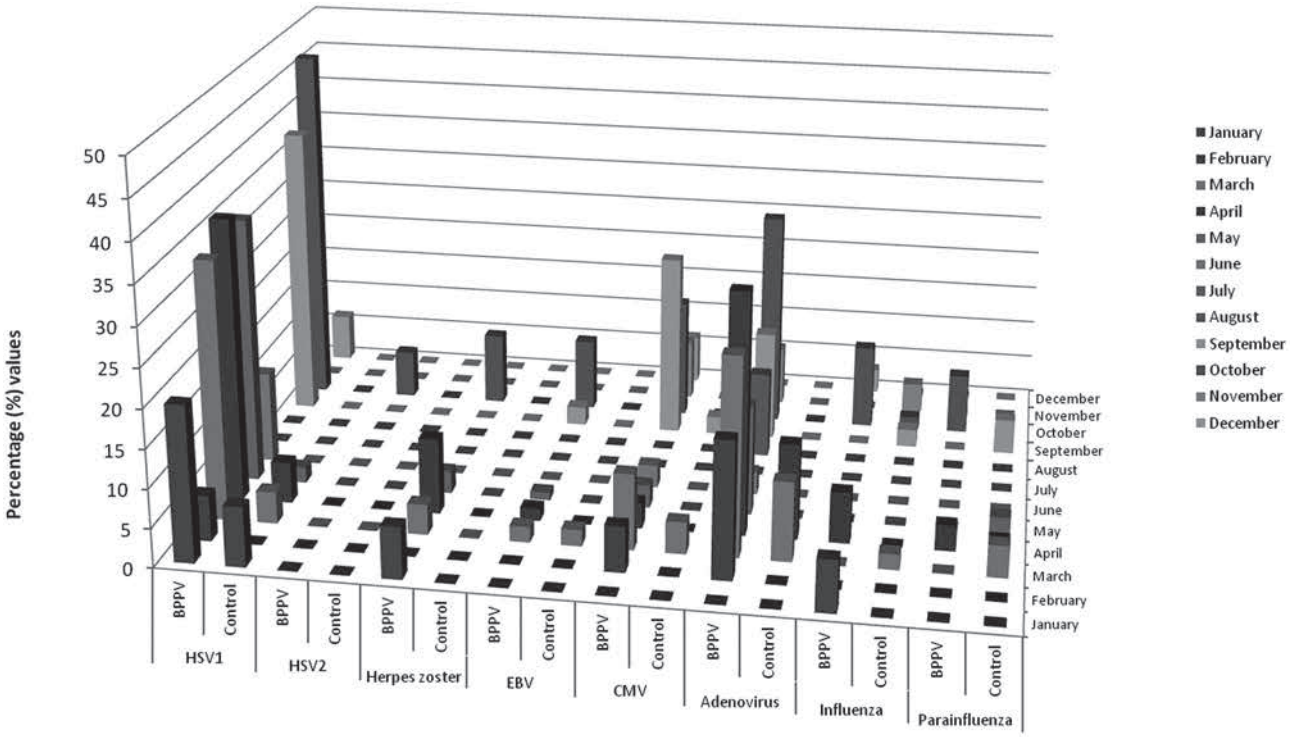
* *P*-value shows the results of Chi-square test.

** There are missing values. Because 2 participants in the control group did not fill this item.

Table 2
Viral serology*

	BPPV group		Control group		*P**
	n (%)	n (%)	n (%)	n (%)	
	Serology (+)	Serology (-)	Serology (+)	Serology (-)	
HSV 1	119 (24.6)	364 (75.4)	8 (1.7)	453 (98.3)	P=0.000, X^2=106.174
HSV 2	6 (1.2)	477 (98.8)	0 (0.0)	461 (100.0)	P=0.016, X^2=5.757
Herpes zoster	18 (3.7)	465 (96.3)	0 (0.0)	461 (100.0)	P=0.000, X^2=17.514
EBV	10 (2.1)	473 (97.9)	1 (0.2)	459 (99.8)	P=0.000, X^2=7.017
CMV	36 (7.5)	447 (92.5)	3 (0.7)	458 (99.3)	P=0.000, X^2=27.559
Adenovirus	79 (16.3)	404 (83.6)	12 (2.6)	449 (97.4)	P=0.000, X^2=51.271
Influenza	13 (2.7)	470 (97.3)	6 (1.3)	455 (98.7)	P=0.128, X^2=2.311
Parainfluenza	7 (1.4)	476 (98.6)	7 (1.5)	453 (98.5)	P=0.927, X^2=0.008

* *P*-value shows the results of Chi-square test.



* Percent of BPPV patients and controls who show positive serology for virus infection. Values are separated and displayed by month of the year, to elucidate seasonality.

Figure 1
Viral agents*

posterior semi-circular canal in one inner ear is responsible of this specific type of positional vertigo.[13] The most common underlying causes of BPPV are previous traumatic head injury and post-viral labyrinthitis.[14]

Most commonly, BPPV involves the posterior semicircular canal and is characterized by the following typical findings upon Dix-Hallpike exam: up-beating, rotational, geotropic nystagmus affecting one side, with a latency of 5-10 s and

Table 3

Viral serology results by months (January to December)**

		January	February	March	April	May	June	July	August	September	October	November	December	P*
		%	%	%	%	%	%	%	%	%	%	%	%	
HSV1	BPPV	20.0	5.9	33.3	36.4	34.3	11.8	0.0	0.0	38.1	46.9	0.0	6.2	P=0.913 X^2=0.012
	Control	7.7	0.0	4.1	5.3	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	P=0.006 X^2=7.472
	*P**	>0.05	>0.05	*P*=0.000 X^2=13.755	>0.05	*P*=0.000 X^2=35.541	*P*=0.047 X^2=3.947			*P*=0.000 X^2=20.354	P=0.000 X^2=39.362		>0.05	
HSV2	BPPV	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	6.2	0.0	0.0	P=0.142 X^2=2.155
	Control	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	
	*P**					>0.05					P=0.041 X^2=4.160			
Zoster	BPPV	6.7	0.0	3.9	9.8	2.9	0.0	0.0	0.0	0.0	9.4	0.0	0.0	P=0.405 X^2=0.695
	Control	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	
	*P**	>0.05		>0.05	*P*=0.016 X^2=5.857	>0.05					P=0.012 X^2=6.341			
EBV	BPPV	0.0	0.0	2.0	1.6	1.0	0.0	0.0	0.0	2.4	9.4	0.0	0.0	P=0.096 X^2=2.765
	Control	0.0	0.0	2.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	P=0.243 X^2=1.364
	*P**			>0.05	>0.05	>0.05				>0.05	P=0.012 X^2=6.341			
CMV	BPPV	0.0	5.9	9.8	3.3	2.9	2.9	0.0	0.0	23.8	15.6	7.7	6.2	P=0.007 X^2=7.338
	Control	0.0	0.0	4.1	0.0	0.0	0.0	0.0	0.0	2.3	0.0	0.0	0.0	P=0.380 X^2=0.771
	*P**		>0.05	>0.05	>0.05	>0.05	>0.05			*P*=0.003 X^2=8.831	P=0.001 X^2=10.924	>0.05	>0.05	
Adenovirus	BPPV	0.0	17.6	25.5	31.1	14.3	2.9	0.0	11.1	14.3	28.1	7.7	0.0	P=0.140 X^2=2.182
	Control	0.0	0.0	10.2	12.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	P=0.001 X^2=11.634
	*P**		>0.05	*P*=0.047 X^2=3.956	*P*=0.013 X^2=6.054	*P*=0.000 X^2=15.486	>0.05		>0.05	*P*=0.010 X^2=6.679	P=0.000 X^2=21.048	>0.05		
Influenza	BPPV	6.7	0.0	0.0	6.6	0.0	0.0	0.0	0.0	0.0	10.9	0.0	3.1	P=0.183 X^2=1.770
	Control	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	2.3	1.5	3.8	0.0	P=0.609 X^2=0.262
	*P**	>0.05		>0.05	P=0.050 X^2=3.836	>0.05				>0.05	P=0.027 X^2=4.860	>0.05	>0.05	
Parainfluenza	BPPV	0.0	0.0	0.0	3.3	0.0	0.0	0.0	0.0	0.0	7.8	0.0	0.0	P=0.122 X^2=2.391
	Control	0.0	0.0	4.1	1.8	2.0	0.0	0.0	0.0	4.5	0.0	0.0	0.0	P=0.330 X^2=0.948
	*P**			>0.05	>0.05	>0.05				>0.05	P=0.022 X^2=5.242			

* *P* value shows the results of Chi-square test.

** In empty box, no positive serology results were found in either group.

duration of up to 10-20 s. Less common types of BPPV include horizontal semicircular canal (HSCC) BPPV and anterior SCC BPPV, which are diagnosed by the roll test and the Dix-Hallpike exam, respectively.[15]

Spontaneous resolution is observed in many cases of BPPV within several months. The main benefit of performing a canal repositioning maneuver is that it shortens the time for recovery from this form of dizziness, which can be quite limiting to patients. Pharmacologic therapy with vestibular suppressant medication, such as antihistamines or benzodiazepines, is not recommended for BPPV.[3]

In the present study, we investigated the occurrence and possible role of viral infection in BPPV patients compared with healthy controls. A total of 483 BPPV patients were evaluated by Dix-Hallpike exam. Male and female patients composed 50.7% and 49.3% of the sample, respectively. In 65.0% of the patients, right-sided involvement was observed and in 35.0% of the patients, left-sided involvement was detected. Patients were treated with multiple applications of the Epley maneuver. In the BPPV group, occurrence of balance problems (37.3%), motion sickness (21.3%), migraine (27.5%), previous vertigo (54.0%), nausea (98.7%), and vomiting (76.6%) were found to be significantly higher than in the control group. Although BPPV often co-occurs with migraine,[16] no direct pathophysiological link between migraine and BPPV has been established.[17] In our study, we also detected migraine and BPPC co-incidence, consistent with previously published results.

Porta-Etessam, *et al*.[18] studied a group of 70 patients with a diagnosis of episodic migraine, with or without aura. For the diagnosis of instability, they investigated the occurrence of psychophysiological dizziness, presyncopal symptoms, benign paroxysmal positional vertigo (BPPV), migraine-associated recurrent vertigo (MARV), and Meniere's disease. They observed orthostatism or syncope (44.3%), instability (possibly due to bilateral vestibular hypofunction) (15.7%), MARV (8.6%), and BPPV (14.2%) in their patients. The presence of BPPV was observed in older patients (40 years), whereas MARV was seen in younger patients (35 years). They also reported that benign paroxysmal vertigo is a childhood condition, and that age is a risk factor for BPPV. In our study, the mean age of the BPPV group was 47.1±11.7 years, consistent with the results of Porta-Etessam, *et al*.[18]

In the present study, serologic analysis of viral agents was performed in both patients and control individuals. With the exception of influenza and parainfluenza, the serology detected higher values for the other viruses (HSV1, HSV2, Herpes zoster, EBV, CMV and adenovirus) in the BBPV group patients than the control group. HSV1 was detected in 24.6%, adenovirus was detected in 16.3%, and CMV was detected in 7.5% of the patients with BPPV.

We also evaluated the viral serology results according to month (January to December). Compared to the control group, the BPPV group showed elevated HSV1 and adenovirus values in March and May, HSV1 values in June, and HSV1 and CMV values in September. In April, Herpes zoster, adenovirus and influenza values were higher in the BPPV group. In October, all of the virus measurements (HSV1, HSV2, Herpes zoster, EBV, CMV, adenovirus, influenza and parainfluenza virus) were increased in the BPPV group compared to the control group.

Several inner ear diseases, including Ménière's disease, neurolabyrinthitis, and vestibular neuronitis, are considered to be causes of secondary BPPV (s-BPPV). Because these inner ear diseases may have different pathophysiologies, the mechanisms responsible for s-BPPV may differ from those in iatrogenic BPPV (i-BPPV). To date, few studies have examined the clinical differences between i-BPPV and s-BPPV.[19]

Unilateral vestibulopathy exhibited the highest rate of postcanal involvement (78.6%). Herpes simplex virus-1 seems to be the most likely cause; it can result in focal involvement of a superior vestibular nerve.[20] In our study, HSV1 incidence was observed to increase mainly in March, May, June, September, and October, during the spring and autumn seasons. Viral infection-related neurolabyrinthitis is a possible cause of s-BPPV.

In a study by Lee *et al*.,[21] 718 BPPV patients were reviewed, and 69 of them had existing inner ear diseases and consequently s-BPPV, rather than i-BPPV. The diseases associated with s-BPPV in this study were idiopathic sudden sensory hearing loss (ISSHL, 50.7%), Ménière's disease (MD, 28.9%), and unilateral vestibulopathy, such as acute vestibular neuronitis and herpes zoster oticus (20.2%). The authors concluded that the mean duration of treatment for BPPV with ISSHL or unilateral vestibulopathy was longer than that of

other groups. The different pathophysiologies of s-BPPV cases that are associated with different inner ear diseases may explain its diverse clinical features and courses. In our study, an increase in Herpes zoster was detected in April and October, similar to the increased incidence of HSV1 infections observed in spring and autumn.

In an electron microscopic study by Pulec and Patterson,[22] 42 patients with intractable vertigo were treated by surgical removal of segments of the superior and inferior vestibular nerves. The authors reported that the vestibular nerves were found to be histologically normal in lesions that primarily involve the inner ear, such typical early Meniere's disease, benign paroxysmal postural vertigo (BPPV), and mild labyrinthine concussion. Vestibular nerve degeneration was present in advanced Meniere's disease, severe labyrinthine concussion, and with vascular loops in the internal auditory canal. In Herpes zoster oticus, the Scarpa ganglion is involved. Viruses were detected in the nuclei of vestibular nerve cells in a patient with delayed hydrops.

Our results showed that BPPV and migraine may co-occur. In our study, positive viral serology was found in BPPV patients and associated with certain months of the year, mainly the spring and autumn seasons. In October, all of the viral agents were detected in BPPV patients, in contrast to the control group. We cannot absolutely say that viral agents cause BPPV. However, our results are consistent with the possibility that viral infection and subsequent development of vestibulopathy promotes BPPV attacks or that viral infection-related neurolabyrinthitis causes secondary BPPV.

Conclusion

No demonstrable cause-and-effect relationship between viral infections and BPPV can be concluded from our study, but our results are consistent with possible association. We observe that viral infectious serology values are higher in BPPV patients than in the control group, especially in spring and autumn months.

Acknowledgements

Except data collection, the preparation of this paper, including design and planning, was supported by the Continuous Education and Scientific Research Association. There was no grant or funding, only scientific support.

References

1. Kollén L, Frändin K, Möller M, Fagevik Olsén M, Möller C. Benign paroxysmal positional vertigo is a common cause of dizziness and unsteadiness in a large population of 75-year-olds. *Aging Clin Exp Res*. 2012;24(4):317-323.
2. Schuknecht HF, Ruby RR. Cupulolithiasis. *Adv Otorhinolaryngol*. 1973;20:434-443.
3. Halker RB, Barrs DM, Wellik KE, Wingerchuk DM, Demaerschalk BM. Establishing a diagnosis of benign paroxysmal positional vertigo through the dix-hallpike and side-lying maneuvers: a critically appraised topic. *Neurologist*. 2008;14(3):201-204.
4. Hain CT. Dix-Hallpike test. Available at: http://www.dizziness-and-balance.com/disorders/bppv/dix%20hallpike.htm. Accessed July 9, 2015.
5. Aw ST, Todd MJ, Aw GE, McGarvie LA, Halmagyi GM. Benign positional nystagmus: A study of its three-dimensional spatio-temporal characteristics. *Neurology*. 2005;64(11):1897-1905.
6. Li JC, Epley J. Benign Paroxysmal Positional Vertigo Clinical Presentation. Medscape. Available at: http://emedicine.medscape.com/article/884261-overview#a0104. Accessed December 9, 2014.
7. Epley JM. New dimensions of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 1980; 88(5):599-605
8. Karlberg M, Halmagyi GM, Büttner U, Yavor RA. Sudden unilateral hearing loss with simultaneous ipsilateral posterior semicircular canal benign paroxysmal positional vertigo: a variant of vestibulo-cochlear neurolabyrinthitis? *Arch Otolaryngol Head Neck Surg*. 2000;126(8):1024-1029.
9. No authors listed. Caloric testing. Available at: http://www.balancemd.net/Caloric_Testing.html. Accessed July 9, 2015.
10. Arbusow V, Schulz P, Strupp M, Dieterich M, von Reinhardstoettner A, Rauch E, Brandt T. Distribution of herpes simplex virus type 1 in human geniculate and vestibular ganglia: implications for vestibular neuritis. *Ann Neurol*. 1999;46(3):416-419.
11. WMA Declaration of Helsinki - Ethical Principles for Medical Research Involving Human Subjects. Available at: http://www.wma.net/en/30publications/10policies/b3/index.htm. Accessed December, 9 2014.
12. No authors listed. Benign Paroxysmal Positional Vertigo. John Hopkins Medicine. Available at: http://www.hopkinsmedicine.org/neurology_neurosurgery/centers_clinics/vestibular/conditions/benign_paroxysmal_positional_vertigo.html. Accessed July 9, 2015.
13. Sauron B, Dobler S. Benign paroxysmal positional vertigo. Diagnosis, course, physiopathology and treatment. *Rev Prat*. 1994;44(3):313-318.
14. Andaz C, Whittet HB, Ludman H. An unusual cause of benign paroxysmal positional vertigo. *J Laryngol Otol*. 1993;107(12):1153-1154.

15. El-Saied S, Joshua BZ, Segal N, Kraus M, Kaplan DM. Sudden hearing loss with simultaneous posterior semicircular canal BPPV: Possible etiology and clinical implications. *Am J Otolaryngol*. 2014;35(2):180-185.
16. Handelsman JA. Migraine and Vertigo. The ASHA Leader. 2009, October 13. Available at: http://www.asha.org/publications/leader/2009/091013/f091013b.htm Accessed April 2, 2013.
17. Faralli M, Cipriani L, Del Zompo MR, Panichi R, Calzolaro L, Ricci G. Benign paroxysmal positional vertigo and migraine: analysis of 186 cases. *B-ENT*. 2014;10(2):133-139.
18. Porta-Etessam J, García-Cobos R, Cuadrado ML, Casanova I, Lapeña T, García-Ramos R. Neuro-otological symptoms in patients with migraine. *Neurologia*. 2011; 26(2):100-104.
19. Ban JH, Lee JK, Jin SM, Lee KC. Glycerol pure tone audiometry and glycerol vestibular evoked myogenic potential: representing specific status of endolymphatic hydrops in the inner ear. *Eur Arch Otorhinolaryngol*. 2007;264(11):1275-1281.
20. Gianoli G, Goebel J, Mowry S, Poomipannit P. Anatomic differences in the lateral vestibular nerve channels and their implications in vestibular neuritis. *Otol Neurotol*. 2005;26(3):489-494.
21. Lee NH, Ban JH, Lee KC, Kim SM. Benign paroxysmal positional vertigo secondary to inner ear disease. *Otolaryngol Head Neck Surg*. 2010;143(3):413-417.
22. Pulec JL, Patterson MJ. Vestibular nerve pathology in cases of intractable vertigo: an electronmicroscopic study. *Am J Otol*. 1997;18(4):475-483.

Dr. Nuray Bayar Muluk
Birlik Mahallesi
Zirvekent 2. Etap Sitesi, C-3 blok, No: 62/43
06610 Çankaya / Ankara, Turkey
Tel.: +90 312 4964073 , +90 535 6655718, +90 532 7182441
Fax: +90 312 4964073
E-mail: nbayarmuluk@yahoo.com
nurayb@hotmail.com