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OTOLOGY

Dizziness handicap and clinical characteristics of posterior and lateral canal BPPV

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Abstract

Purpose Benign paroxysmal positional vertigo (BPPV) is diagnosed and divided into subtypes based on positioning vertigo and nystagmus. Whether these subtypes entail any significant differences in patient-reported symptoms; is yet not known. Such differences may have clinical and therapeutic consequences. Our aim was to assess dizziness handicap and clinical characteristics of posterior and lateral canal BPPV.

Methods This prospective observational multicentre study analysed consecutive patients with BPPV, confirmed by standardized procedures including videonystagmography under diagnostic manoeuvres in a biaxial rotational chair. Patients were screened for other neurological and otological disorders.

Outcomes Dizziness handicap inventory (DHI), posterior vs. lateral canal involvement. Factors: age, gender, positional nystagmus intensity (maximum slow-phase velocity), symptom duration, 25-hydroxyvitamin D-level and traumatic aetiology.

Results 132 patients aged 27–90 (mean 57, SD 13) years were included. Higher DHI scores were associated with lateral canal BPPV [95% CI (1.59–13.95), $p=0.01$] and female gender [95% CI (0.74–15.52), $p=0.03$]. Lateral canal BPPV was associated with longer symptom duration [OR 1.10, CI (1.03–1.17), $p=0.01$] and lower 25-hydroxyvitamin D-levels [OR 0.80, CI (0.67–0.95), $p=0.03$]. There was no correlation between DHI scores and nystagmus intensity.

Conclusions This study suggests that patients with lateral canal BPPV have increased patient-perceived disability, lower vitamin D-levels and longer duration of symptoms. This subtype might therefore require closer follow-up. Patient-perceived disability is not related to positional nystagmus intensity.

Keywords Lateral canal BPPV · Posterior canal BPPV · Videonystagmography · Biaxial rotational chair · Quality of life

Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo, and the incidence increases with age [1]. BPPV is demonstrated to have great impact on patients' physical, emotional and functional well-being, and hence, on their dizziness-related quality of life (DRQoL) [2–4]. BPPV patients have been shown to rate their quality of life on the same level as in patients with macular degeneration, hepatitis B and HIV/AIDS [5]

The accepted cause of BPPV is free-floating particles within the lumen of the semi-circular canals (canalolithiasis) or otolithic debris adherent to the cupula (cupulolithiasis). While canalolithiasis has been demonstrated intraoperatively [6, 7], cupulolithiasis is mainly documented in temporal bone studies [8]. 25-Hydroxyvitamin D deficiency may be associated with increased risk of BPPV and increased recurrence rates [9, 10]. Recent studies concerning subtypes of BPPV and their variations have broadened the understanding of the disease and the variation in nystagmus patterns. This has improved diagnostic accuracy and enhanced appropriate treatment.

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The size of otoconia and the paths they take, as well as wall interactions and particle–particle interactions may all affect the vestibular nerve response and the oculomotor response [11–13]. Cupular volume displacement is frequently assumed to be proportional to the sensed rotation rate and eye movements [12]. Despite growing knowledge with regard to the different subtypes of BPPV and their specific nystagmus findings, there is limited knowledge of the effect BPPV subtypes may have on DRQoL, and how this is reflected in patient-reported symptoms. This study investigates two different subtypes of BPPV: canalolithiasis of the posterior canal BPPV (PC-BPPV) and canalo- and cupulolithiasis of the lateral canal BPPV (LC-BPPV). To our knowledge there are no previous studies on BPPV subgroups and nystagmus intensity, in relation to DRQoL.

The aim of this study was to investigate whether patient-reported symptoms and other clinical characteristics of BPPV are related to subtype and canal-specific nystagmus findings.

In particular, we aimed to use consistent positioning manoeuvres under standardized test conditions in a biaxial rotational chair, to document and analyse nystagmus findings using a video-based recording system (videonystagmography). We also wanted to use the dizziness handicap inventory (DHI), which is a validated tool for measuring DRQoL.

Materials and methods

Ethics

The Regional Committee for Medical and Health Research Ethics of Western Norway approved the study in advance. Participation was based on written informed consent. The study was performed in accordance with the ethical standards of the 1964 Declaration of Helsinki. The study was registered at clinicaltrials.gov.

Design and setting

This was a prospective observational multicentre study conducted at two university hospitals from June 2013 to June 2016. The Department of Otorhinolaryngology and Head and Neck Surgery at Haukeland University Hospital runs a specialized laboratory for investigation of vestibular disorders, which examines approximately 1200 patients yearly, as well as a national centre for education and research on vestibular disorders. The Department of Otorhinolaryngology and Head and Neck Surgery at Oslo University Hospital runs a similar facility for vestibular examinations.

Subjects

Consecutive patients referred with a history suggestive of BPPV were considered for inclusion, which was based on confirmed active BPPV according to international diagnostic criteria [14] (Table 1). Exclusion criteria were a history of neurological disease including migraine, inner ear disease other than BPPV, multiple canal BPPV, anterior canal BPPV and pregnancy. Anterior canal BPPV was excluded, as this involves a controversial and small group of BPPV patients. Multiple canal BPPV was also excluded as their nystagmus pattern varies and the group is diverse. A requirement made to patients was that they had not consumed alcohol, sedating drugs, tranquilizers or vestibular suppressants 48 h prior to testing. To rule out central disorders, 57 patients (40%) underwent head MRI. Indications for MRI were previous head trauma and lack of response to treatment. In some cases, head MRI had been taken prior to referral. Audiograms had to be within normal limits related to age and gender reference [15] or show symmetrical presbyacusis. Subjects included did not have spontaneous nystagmus when fixating with the unrecorded eye or nystagmus during gaze testing or after 10 s headshake with videonystagmography

Table 1 BPPV diagnostic criteria according to the consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society [14]

	Posterior canalithiasis	Lateral canalithiasis	Lateral cupulolithiasis
Recurrent attacks of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position	x	x	x
Vertigo duration	< 1 min	< 1 min	Not specified
Diagnostic maneuver	Dix-Hallpike or Semont diagnostic maneuver	Supine roll test	Supine roll test
Nystagmus latency	One or few seconds	Brief or none	Brief or none
Nystagmus direction	Torsional upbeat	Geotropic direction changing	Apogeotropic direction changing
Nystagmus duration	Typically < 1 min	< 1 min	> 1 min
Not attributable to another disorder	x	x	x

(VNG, Interacoustics, Denmark). The hospital clinical laboratory measured serum 25-hydroxyvitamin D-levels.

Procedure

On the day of the examination, the patient's history was verified by interview and filling out symptom questionnaires. The subjects underwent a physical exam as well as a standardized examination for positional nystagmus with video recording in a biaxial chair (TRV chair, Interacoustics). Recording was done during Supine test, Dix-Hallpike and Pagnini McClure. The rotational chair used in this study can be rotated around a patient-fixed vertical axis mounted in a frame that can be rotated around an earth-fixed horizontal axis. During rotations, the patient is secured in the chair with a four-point harness, with headrest, headband and leg straps. This setup ensures exact and reproducible positioning manoeuvres without any active movements on the part of the patient, it also eliminates movements in the spinal column that may cause pain and discomfort in some subjects. Bilateral Dix-Hallpike and bilateral supine roll test were performed as shown in Fig. 1. The same setup and equipment were used in both participating departments [16]. The testing always started towards the most symptomatic side.

Further assessment included an ear, nose, and throat examination, otoneurological examination, and head impulse testing and pure tone audiometry.

During positional testing, patients used a standard VNG mask with an infrared camera recording eye movement in three planes using a dark pupil tracking system (Synapsys, Marseille, France). Eye movements were inspected in real-time on a wall-mounted monitor. After each diagnostic manoeuvre, eye movements were recorded for 30 s with a sampling rate of 25 Hz. The transition time of movement from one position to another was approximately 3 sec. Recordings were taken from the right eye. The subjects were asked to keep their eyes open and to blink as little as possible while looking straight ahead. All nystagmography recordings were reviewed to detect discrepancies due to blinking, unsatisfactory pupil detection or other artefacts.

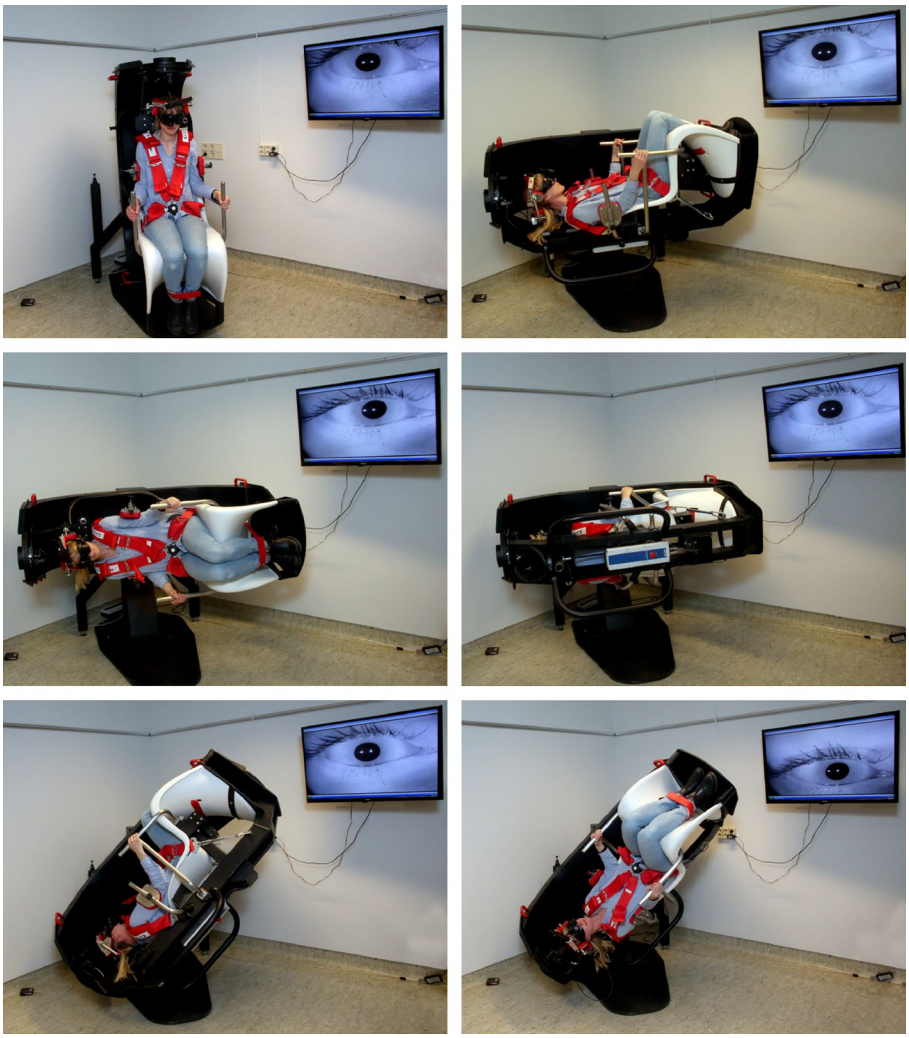

Fig. 1 From left to right. Upright, supine, supine roll test right, supine roll test left, Dix-Hallpike left, Dix-Hallpike right

Nystagmus intensity was defined as the maximum slow-phase velocity (SPV_{max}), measured in degrees per second after each diagnostic manoeuvre (Fig. 1). Nystagmus seen during the transition time of movement from one position to another was ignored. The VNG-files were imported into a LabVIEW program developed for this study, and two of the authors did a blinded evaluation of the VNG-signals, selecting and measuring the area with highest slow-phase velocity. In case of discrepancies, two of the other authors independently reviewed the recordings. In cases of lateral canal BPPV, the horizontal component was used for further analysis. In cases of posterior or anterior canal BPPV, the vertical and torsional components were considered. Due to limitations in the VNG technology, the slow phase of torsional nystagmus could not be measured but was quantified visually by the authors as the mean of three independent observations on a visual analogue scale (VAS), anchored in both ends with words descriptive of the maximum and minimum extremes of nystagmus.

Patient-reported symptoms were collected via DHI questionnaire in conjunction with the consultation. A few forms contained missing items; to correct for this and to obtain a corrected total score, the mean score of the answered items was multiplied by 25 (total number of questions). In general, missing items were few. The questionnaire we used was a version adapted to Norwegian with verified internal reliability and validity [17]. To make interpretation of the DHI scores more intuitive, we used the following grading system: mild disability (0–30 points), moderate disability (31–60 points) and severe disability (61–100 points) [18].

Diagnosis

The diagnosis of BPPV and subtypes was based on criteria published by the Committee for Classification of Vestibular Disorders of the Barany Society [14].

Statistics

A multivariable linear regression model was used to identify factors associated with dizziness-related quality of life using DHI score as dependent variable (continuous, ranging from 0–100) and BPPV subtype, age, gender, symptom duration, traumatic aetiology, 25-hydroxyvitamin D-level and positional nystagmus intensity (SPV_{max}) as factors. Backward stepwise elimination of the least significant factor was performed until only significant factors remained. We performed a binomial logistic regression analysis to identify predictors of lateral vs. posterior canal BPPV. Age, gender, DHI, $SPV_{max,}$ symptom duration, vitamin D and head trauma were included as factors. To use all available information from the data Mplus version 8 was used to estimate linear and logistic regression models, with the estimator set to full information maximum likelihood (FIML) [19].

We performed a power analysis based on DHI results from earlier reports [20, 21] using a mean DHI of 60 with SD ± 8 and a minimal significant change of five points. With a power of 80% and a significance level of 5% the power analysis resulted in a minimum of 42 participants in each group. Symptom duration was defined as the time between symptom onset as reported by the patient, and inclusion. For the analysis of association between symptom severity (DHI score) and nystagmus velocity, we divided the patients into two groups. When analysing SPV as a factor we used horizontal slow-phase velocity in patients with lateral canal BPPV and vertical slow-phase velocity for patients with posterior canal BPPV. We used STATA version 15.0 for statistical analysis. The significance level was set to $\alpha = 0.05$.

Results

Demographics and clinical characteristics

The study included 132 patients aged 27–90 years (mean 57 years, SD 13 years). The main clinical features are shown in Table 2. A majority of the patients (78%, $n = 103$) were women. The most common BPPV subtype was PC-BPPV (66%, $n = 87$) followed by LC-BPPV (34%, $n = 45$). Previous BPPV episodes that had resolved spontaneously or after treatment were reported by 34 (26%) patients. Head trauma prior to BPPV onset was reported by 31 (23%) patients. The right side was affected more often (61%) than the left (39%). Stress was a provoking factor for dizziness spells in 49 (37%) patients. BPPV was most common in the fifth ($n = 32$, 24%) and sixth decades of life ($n = 41$, 31%).

LC-BPPV was associated with longer symptom duration [OR 1.10, 95% CI (1.03–1.17), $p = 0.01$] and lower 25-hydroxyvitamin D-levels [OR 0.80, 95% CI (0.67–0.95), $p = 0.03$] and higher DHI scores [OR 2.08, 95% CI (1.08–4.02), $p = 0.03$] (Table 3).

Dizziness handicap

DHI scores are shown in Table 2 and ranged from 0 to 74 points (mean 40, SD 17). Dizziness handicap was mild in 31%, moderate in 57% and severe in 12% of patients. Higher dizziness handicap was associated with LC-BPPV [95% CI (1.59–13.95), $p = 0.01$] and female gender [95% CI (0.74–15.52), $p = 0.03$] (Table 4). DHI scores were not associated with age, symptom duration, traumatic aetiology, nystagmus intensity or serum 25-hydroxyvitamin D.

Table 2 Baseline characteristics of patients with BPPV ($N=132$)

Characteristics	Posterior canal BPPV $N=87$	Lateral canal BPPV $N=45$
Gender		
Female	72	31
Male	15	14
Age year (range)	27–90	27–78
Mean ± SD	56.9 ± 14.0	56.7 ± 11.9
Involved side		
Right	57	24
Left	30	21
Bilateral	0	0
Traumatic BPPV		
Yes	24	7
No	63	38
Canalolithiasis	87	19
Cupulolithiasis	0	26
Dur. of sympt. (range in weeks)	1–208	0–780
Mean ± SD	23.3 ± 38.9	75 ± 139.4
Vit. D nmol/l (range)	10–120	20–96
Mean ± SD	68.5 ± 20.5	60.0 ± 18.6
DHI score (range)	6–72	0–74
Mean ± SD	37.7 ± 16.3	44.1 ± 17.2
MRI		
Yes	25	25
No	62	20
Nystagmus SPV_{max}[a] (range)	2.2–66.8	2–103
Mean ± SD	16.2 ± 13.1	15.3 ± 19.2
Recurrent		
Yes	22	12
No	63	33

Dur. of sympt subjective duration of symptoms prior to investigation, *BPPV* benign paroxysmal positional vertigo, SPV_{max} maximum slow-phase velocity

[a]Posterior canal BPPV vertical component, lateral canal BPPV horizontal component

Discussion

The main finding of this study is that lateral canal BPPV differs from posterior canal BPPV with respect to symptom duration, dizziness handicap and serum 25-hydroxyvitamin D-levels. There was no correlation between dizziness handicap and the intensity of positional nystagmus at the time of diagnosis.

The longer symptom duration in patients with BPPV of the lateral canal (75 weeks) than that of the posterior canal (23 weeks) is in contrast to previous findings [22, 23]. However, the latter studies evaluated only the most recent episode in patients with acute BPPV, while in our research, we asked the patients to report the first onset of dizziness symptoms. In our study we did not exclusively select patients with acute vertigo. The natural course of BPPV includes improvements, that may be spontaneous or due to treatment, as well as relapses. Our findings imply that the total time course of vertigo symptoms is longer in patients with lateral canal BPPV. The reason for longer symptom duration in lateral canal BPPV could be anatomical. Lateral canalolithiasis would be expected to resolve spontaneously in many cases simply by lying on the healthy side in bed. However, it might recur more easily when turning to the diseased side. Thus, a large accumulation of debris in the utricle could easily cause prolonged symptoms by entering and exiting the lateral canal over a long period before dissolving in the endolymph. In contrast to this, recurrence of posterior canalolithiasis might be less likely due to the ostium of the common crus being located superiorly in the utricle. Another reason could be that clinicians are more familiar with diagnosing and treating posterior canal BPPV than lateral canal BPPV, and patients who do not recover spontaneously goes untreated

Table 3 Binomial logistic regression analysis of factors associated with subtypes of BPPV. Factors predicting lateral ($n=45$) versus posterior canal BPPV ($n=87$)

	Lateral canal BPPV					
	Unadjusted			Final model		
	OR	95% CI	Sig.	OR	95% CI	Sig.
Age						
Years	0.99	0.97, 1.02	0.65			
Gender						
Female	0.56	0.25, 1.24	0.16			
DHI group						
Mild, moderate, severe	2.19	1.18, 4.08	**0.01**	2.08	1.08, 4.02	**0.03**
Nystagmus intensity[a]						
°/s	0.99	0.97, 1.02	0.56			
Symptom duration						
per week	1.01	1.00, 1.02	**0.01**	1.10	1.03, 1.17	**0.01**
Serum vitamin D						
nmol/l	0.98	0.96, 1.00	**0.04**	0.80	0.67, 0.95	**0.03**
Head trauma prior to BPPV onset	0.49	0.20, 1.23	0.13			

OR and Sig.: odds ratio and p values. Significant P values marked in bold. Unadjusted: binomial logistic regression. Final model: multivariable binominal logistic regression analysis with backward stepwise elimination of least significant factor until $p<0.05$ for all remaining factors. Nystagmus intensity: maximum slow-phase velocity of nystagmus after diagnostic maneuver (Dix-Hallpike or Pagnini-McClure)

[a]For posterior canal BPPV vertical component, for lateral canal BPPV horizontal component

Table 4 Linear regression analysis of factors associated with patient-reported dizziness in 132 BPPV patients

	Dizziness handicap inventory					
	Unadjusted			Final model		
	B	95% CI	Sig.	B	95% CI	Sig.
Age	− 0.11	− 0.34, 0.12	0.33			
Gender						
Female	6.22	− 1.32, 13.77	0.11	8.13	0.74, 15.52	**0.03**
Affected canal						
Lateral	6.37	0.09, 12.65	**0.05**	7.77	1.59, 13.95	**0.01**
Nystagmus intensity[a]						
°/s	0.04	− 0.16, 0.25	0.67			
Symptom duration						
per week	0.01	− 0.03, 0.04	0.67			
Serum vitamin D						
nmol/l	0.02	− 0.14, 0.18	0.92			
Head trauma prior to BPPV onset	2.32	− 4.71, 9.35	0.52			

B and Sig.: unstandardized coefficients and p values. Significant p values marked in bold. Unadjusted: simple linear regression. Final model: multivariable regression analysis with backward stepwise elimination of least significant factor until $p<0.05$ for all remaining factors. Nystagmus intensity: maximum slow-phase velocity of nystagmus after diagnostic maneuver (Dix-Hallpike or Pagnini-McClure)

[a]For posterior canal BPPV vertical component, for lateral canal BPPV horizontal component

over longer periods, as BPPV persists in 30–33% of the untreated patients [24, 25].

Lateral canal BPPV in our study was associated with higher dizziness handicap compared to the posterior canal. Studies on dizziness-related quality of life in different subtypes of BPPV are scarce. Kim et al. [26] in their study found a relation between lateral canal BPPV and increased symptoms in terms of functional, emotional, and physical limitations. However, the study comprised a very limited number of patients with lateral canal BPPV ($n=18$), and the

number of patients in the respective groups canalolithiasis and cupulolithiasis of the lateral canal varied from section to section in the paper. Furthermore, the criteria for inclusion were not set following accordance with international guidelines. A possible explanation for higher dizziness handicap may be that dysfunction of the lateral canals may lead to more symptoms during daytime when the head is mostly in the upright position. Another explanation could be frustration due to longer symptom duration.

BPPV in general has been shown to be associated with relatively high scores in DHI, averaging around 42 and standard deviations around 19 points [2], which is similar to our results. This is higher than the average for patients with some other unilateral vestibular disorders like vestibular schwannomas, that in a study of 538 patients by Carlson et al. was positively skewed with a median score of 10 points (IQR 2–26), or in patients followed up in 1–6 months after vestibular neuritis [27]. It is on level with some studies of patients with Menière's disease [28]. This indicates that BPPV is associated with considerable dizziness handicap. A recent study by Whitney et al. [18] demonstrated that perception of handicap is correlated with functional performance in patients having vestibular disease.

Several studies indicate that 25-hydroxyvitamin D deficiency increases the risk of BPPV, and that this may be due to otoconial degeneration associated with changes in the calcium metabolism [29–31]. Low 25-hydroxyvitamin D levels also seem to be associated with recurrence of BPPV [9]. Research on osteopenic rats, has shown that otoconia in these rats has decreased density and increased size, thus making the otoconia more easily dislodgeable [29]. Our study showed a correlation between lateral canal BPPV and low levels of 25-hydroxyvitamin D. We have not found other studies of 25-hydroxyvitamin D in different BPPV subtypes.

We found no association between dizziness handicap and the intensity of positional nystagmus during diagnostic manoeuvres. To our knowledge, there are no prior studies on the correlation between dizziness handicap and nystagmus intensity. In theory, one would expect dizziness handicap to be greater in patients with stronger nystagmus. However, the findings at the time of diagnosis may not be representative of the entire course of the disorder. The Dizziness Handicap Inventory is used to evaluate symptoms during the last 4 weeks before diagnosis. Nystagmus intensity and symptom severity may vary during this period.

In our experience, some patients with a clear positional nystagmus do not report vertigo, while others report strong positional vertigo even though nystagmus is weak. This is in agreement with the findings in the present study and could be due to differences in central processing of vestibular stimuli as well as to differences in coping mechanisms.

Head trauma is known to be associated with BPPV representing 8.5% [32]–18% [33]. In our sample 23% had suffered from a head trauma less than 3 months prior to the onset of BPPV.

Another finding was that stress was a provoking factor for dizziness spells in 37% of the patients in our study. Two recent studies suggested that inflammatory mediators and oxidative stress contributes to the pathogenesis of BPPV [34, 35].

In our material, we found a higher incidence of right-side involvement; this is in accordance with other studies [36, 37]. Von Brevern et al. conducted a PubMed literature search where they found that the right side was affected 1.41 times more often than the left side [38], and proposed that the predominance of the right-sided involvement could be caused by the habit of the sleeping on the right side.

Posterior canalolithiasis is already known to be the most common variety of BPPV [39], this is also a finding in our study (66%). The prevalence of lateral canal involvement (34%) was in line with previous estimates ranging from 5–35.3% [40–42]. In our sample 58% of the patients with lateral canal affection were diagnosed with cupulolithiasis. West et al. [41] reported findings of 32% of patients with lateral canal involvement and 39% with cupulolithiasis. They argued that cupulolithiasis would have been insufficiently diagnosed without repositioning devices. We believe that the high rate of cupulolithiasis in our study was a result of patients with BPPV being insufficiently diagnosed without repositioning devices.

The strengths of this study were its prospective design, the use of a mechanical chair that ensured reproducible diagnostic maneuvres in pre-set positions, the rigorous use of international diagnostic criteria for the BPPV subtypes, as well as the use of video documentation and computerized videonystagmography to analyse the direction and velocity of positional nystagmus. The chair also enabled correct positioning of patients with decreased mobility and neck problems. Recent investigations have found that mechanical chairs are valuable for patients who are difficult to diagnose and treat [43]. These chairs facilitate consistency of speed, angle, and amplitude of the diagnostic manoeuvres, which is crucial when evaluating the latency and intensity of nystagmus [44, 45].

Limitations of the study were mainly related to generalizability. The results are valid for secondary or tertiary care patients, but not necessarily for patients seen in general practice or in emergency departments.

Conclusion

In this study, we found that lateral canal BPPV was associated with longer symptom duration and higher disability compared to patients with posterior canal involvement. The former patients may require closer follow-up.

Patient-reported handicap was not related to positional nystagmus intensity.

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Compliance with ethical standards

Conflict of interest The authors have no conflicts of interest to disclose.

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