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Bilateral simultaneous presentation of posterior canal benign paroxysmal positional vertigo

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Main Article

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Abstract

Objective. To outline the clinical picture of bilateral posterior canal benign paroxysmal positional vertigo.

Methods. A total of 573 patients with posterior canal benign paroxysmal positional vertigo were classified as having unilateral, or true or pseudo bilateral, posterior canal benign paroxysmal positional vertigo, and were treated with the Epley manoeuvre. Statistical significance was set at $p < 0.05$.

Results. Of the patients, 483 had unilateral and 90 (15.7 per cent) had bilateral presentation. Of the latter, 72 patients had pseudo bilateral posterior canal benign paroxysmal positional vertigo. Comparisons of site of involvement, male to female ratio and the incidence of associated problems in unilateral, and true and pseudo bilateral posterior canal benign paroxysmal positional vertigo did not reveal any statistically significant differences ($p = 0.828$, $p = 0.200$, $p = 0.142$). Comparisons of the number of manoeuvres required to provide symptom relief and the rate of recurrence were significant ($p < 0.05$).

Conclusion. Identification of true and pseudo bilateral posterior canal benign paroxysmal positional vertigo is important given the differences in aetiology and treatment outcome. Treatment of patients with true bilateral posterior canal benign paroxysmal positional vertigo requires several therapeutic manoeuvre attempts, and patients should be warned about recurrence.

Introduction

The most common type of benign paroxysmal positional vertigo (BPPV) is posterior canal BPPV. The posterior canal, which has the lowest anatomical position, is the most gravity-dependent part of the vestibule of the inner ear, in both upright and supine positions. Posterior canal BPPV is characterised by a transient, up-beating, torsional nystagmus when the patient is brought to a head-hanging position from a sitting position. After a short latency period, up-beating nystagmus with a limited duration and intensity, and with a torsional component clockwise on the left and counter-clockwise on the right head-hanging position, are observed. Diagnosis is confirmed with the reversal of nystagmus, which is in the same plane but in the opposite direction when the patient returns to the upright sitting position.

Canalolithiasis has been implicated as the main pathogenetic mechanism. Free-travelling otoconia inside the membranous labyrinth gravitates into the canal, close to the ampulla. In the Dix–Hallpike position, otoconia, under the influence of gravity, pulls the cupula, producing an excitatory response.[1] Epley's repositioning and Semont's liberatory manoeuvres become the primary treatment in patients with posterior canal BPPV. The aim is to take the debris out of the canal and into the vestibule, which can only exit at the end without the cupula.

The bilateral occurrence of posterior canal BPPV is a challenging condition for treatment planning. It could reflect true involvement of both sides, or unilateral posterior canal BPPV may mimic bilateral posterior canal BPPV (pseudo bilateral). The mechanism of pseudo bilateral posterior canal BPPV seems to be obscure. It was first described by Steddin and Brandt in 1994.[2] These authors hypothesised that travelling otoconial debris in the affected ear will be displaced from a perpendicular position, and will move gravitationally close to the cupula. This causes an artificial and transient cupulolithiasis by deflecting the cupula in the ampullopedal direction when the head-hanging manoeuvre is applied to the non-involved side, and will evoke inhibitory positional nystagmus which is directed toward the lower unaffected ear.[2,3] This inhibitory nystagmus has a lower amplitude and frequency than excitatory nystagmus. However, it may have a down-beating component and longer duration.[1,2,4] Nevertheless, it is unclear why some cases of unilateral posterior canal BPPV rarely mimic a bilateral case. According to these authors, it could be related to inappropriate positioning of the head during testing of the healthy side. They suggest that, to differentiate the true and pseudo cases, maximal stimulation of

the under-most posterior canal by ampullofugal deflection of the cupula during the head-hanging test is possible only when a lateral tilt similar to Semont's manoeuvre has been performed (head rotated by 45° to one shoulder with a tilt to the opposite side). However, the density or dispersal of the travelling debris could also be different in some cases.

There are some features that can be used to differentiate pseudo bilateral and true bilateral posterior canal BPPV. The severity of nystagmus during head-hanging manoeuvre is usually asymmetric in patients with pseudo bilateral cases, being more pronounced on the pathological side. It is reasonable to wait until nystagmus disappears and carefully observe the intensity of nystagmus to identify true BPPV.[5] Additionally, down-beating reversal of nystagmus is expected when the patient returns to a sitting position from a head-hanging position in patients with canalolithiasis of the posterior canal. This sign will never be seen on the non-pathological side in bilateral BPPV cases. Three-dimensional analysis of nystagmus is helpful to differentiate true and pseudo bilateral posterior canal BPPV;[4] however, this may require a technical setup. Positional nystagmus during the lying down test or the straight head-hanging manoeuvre is purely vertical, without any torsional component, in patients with true bilateral posterior canal BPPV. In this test, both posterior canals are simultaneously stimulated, and torsional nystagmus from the two posterior canals cancel one another out, resulting in purely vertical nystagmus. Reports indicate the diagnostic role of vertical nystagmus during the lying down or straight head-hanging test.[6–8] However, one may erroneously suspect a central nervous system disorder, as this vertical nystagmus is generally more persistent than other types of positional nystagmus.[8] Finally, symptomatic relief after Epley's manoeuvre applied on the side with more intense manifestation is evidence of pseudo bilateral BPPV, which is unlikely in true bilateral cases. Observation of vertiginous symptoms in patients during the Dix–Hallpike manoeuvre on the pathological side will also give some clues of true bilateral BPPV.[9]

Although most cases respond to Epley's manoeuvre, recurrence is likely to occur. Bilateral presentation of posterior canal BPPV has been reported to be more difficult to treat than unilateral posterior canal BPPV, and the recurrence rate is high. Bilateral posterior canal BPPV is suspected when the head-hanging manoeuvre is positive on both sides.[1] Trauma is the leading cause. Care should be taken when determining the differential diagnosis. This study aimed to outline the clinical picture of this rare bilateral, simultaneous presentation of posterior canal BPPV, to analyse the treatment outcome, follow up and recurrence pattern.

Materials and methods

The authors assert that all procedures contributing to this work complied with the ethical standards of the relevant national and institutional guidelines, and with the Helsinki Declaration of 1975, as revised in 2008.

A total of 913 patients were diagnosed with BPPV among 1167 patients who underwent videonystagmography for positional symptoms from 2008 to 2021. A total of 573 patients with posterior canal BPPV were included in the study. A flow diagram is shown in Figure 1. Those patients with a history of head or ear surgery, visual disturbance, cervical spine lesions, or muscular or neurological diagnoses, were excluded.

All patients were analysed with videonystagmography (MicroMed, Chatham, Illinois, USA) during the Dix–Hallpike, lying down positional and head-bending (lean and bow) tests. Patients were classified into three groups. The first group included patients with unilateral posterior canal BPPV. The second group included patients with bilateral posterior canal BPPV whose side of involvement based on the severity of nystagmus was clear following the head-hanging and lying down positional tests. The third group included patients with bilateral posterior canal BPPV whose side of involvement was not clear in any of the tests.

Patients with unilateral posterior canal BPPV were treated with the Epley manoeuvre. In patients with a bilateral presentation, the more severe side was treated first. If the intensity of nystagmus was the same on both sides, the treatment side was randomly selected. The Epley manoeuvre was repeated if the patient still had positional nystagmus one week following the last session. The number of manoeuvres required for a four-week symptom-free interval was noted in each patient.

The case was defined as persistent if the patient needed more than one session of treatment on the same side. Recurrence was defined in patients with no symptoms for at least four weeks following the last therapeutic manoeuvre. The recurrence rate was documented in each group. The numbers of patients who had no recurrence during the follow-up period were determined in each BPPV group, and assigned as non-recurrent groups.

Average patient age at the time of initial hospital admission, gender and associated problems (migraine, diabetes, hypertension, osteoporosis, coronary disease, thyroid disorders, arthrosis, anaemia, asthma, cancer, hepatitis and so on) were documented in patients with recurrent and non-recurrent posterior canal BPPV.

The chi-square test was used to statistically compare the percentages of two groups. The one-way analysis of variance test was used to statistically analyse the distribution of more than two groups. Statistical significance was set at $p < 0.05$. Statistical analysis was conducted using SPSS software, version 26 (IBM, Chicago, Illinois, USA).

Results

The average patient age was 41.96 ± 13.26 years and the average follow-up duration was 57.61 ± 18.97 months. A total of 483 patients had unilateral posterior canal BPPV presentation (255 left-sided, 228 right-sided), and 90 patients (15.7 per cent) had bilateral presentation on the head-hanging manoeuvre. Of the 90 patients with bilateral presentation, 72 had either nystagmus with asymmetric intensity on both the head-hanging manoeuvre or torsional up-beating positional nystagmus during the straight head-hanging manoeuvre. Therefore, the pathological side was predictable (pseudo bilateral posterior canal BPPV) in 80 per cent of patients (39 left-sided, 33 right-sided). However, 18 of the 90 patients with bilateral presentation had nystagmus with equal severity on both sides during the head-hanging manoeuvre or had pure vertical nystagmus during the lying down positional test, and the pathological side was not identified (true bilateral posterior canal BPPV). Therefore, the incidence of true and pseudo bilateral posterior canal BPPV was 3.1 per cent (18 out of 573) and 12.6 per cent (72 out of 573), respectively.

Comparison of the side of involvement in unilateral and pseudo bilateral posterior canal BPPV cases did not reveal a statistically significant difference ($p = 0.828$). Comparison of the male to female ratio in unilateral and bilateral (true and pseudo) posterior canal BPPV also did not reveal any

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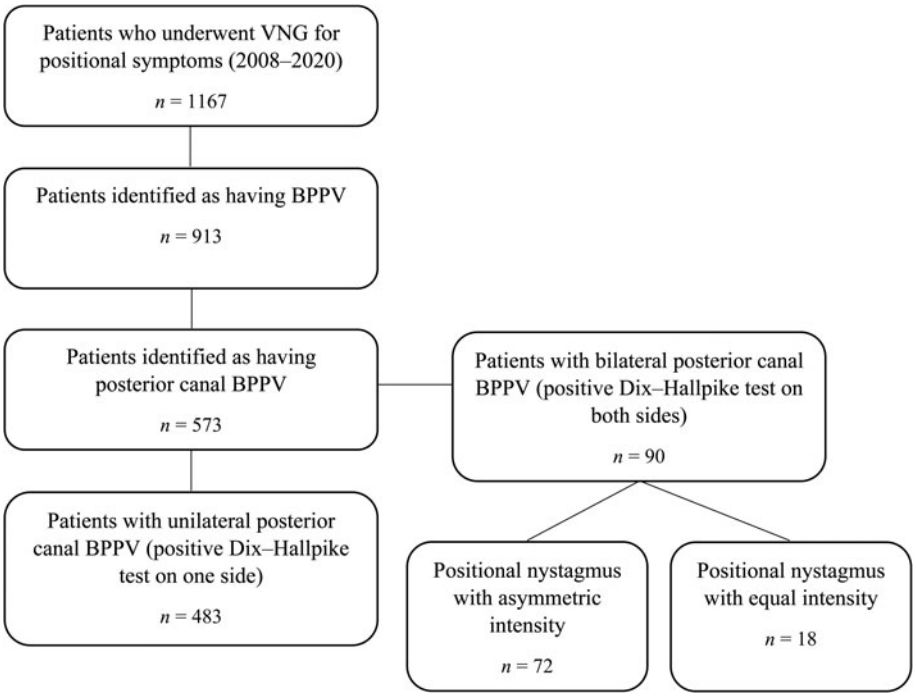

Figure 1. Flow diagram of the study groups. VNG = videonystagmography; BPPV = benign paroxysmal positional vertigo

statistically significant ($p = 0.200$). Eighty patients in the unilateral group (80 out of 483; 16.5 per cent) and 17 patients in the pseudo bilateral group (17 out of 72; 23.6 per cent) had associated problems (migraine, diabetes, hypertension, osteoporosis, coronary disease, thyroid disorders, arthrosis, anaemia, asthma, cancer, hepatitis and so on). None of the patients in the true bilateral group had an associated problem. Therefore, comparison of the rate of associated problems in the unilateral and pseudo bilateral groups was not statistically significant ($p = 0.142$).

Nine patients in the unilateral group (9 out of 483; 1.8 per cent), five patients in the pseudo bilateral group (5 out of 72; 6.9 per cent) and three patients in the true bilateral group (3 out of 18; 16.7 per cent) had a history of trauma a few days before their BPPV diagnosis. The difference was statistically significant ($p < 0.05$).

The average (mean ± standard deviation) number of manoeuvres required to provide symptom relief in patients with unilateral, pseudo and true bilateral posterior canal BPPV was 1.45 ± 0.71, 1.61 ± 0.81 and 2.27 ± 0.57, respectively. All of the patients with true bilateral posterior canal BPPV required multiple sessions of treatment. Comparison of the average number of treatment sessions between groups was statistically significant ($p < 0.05$).

Of all the posterior canal BPPV patients, 66.8 per cent (383 out of 573) had no recurrence at all following the therapeutic manoeuvres. However, the overall recurrence rate in the study group was 33.2 per cent (190 out of 573). The recurrence rates in the three groups were as follows: unilateral posterior canal BPPV = 31.3 per cent (151 out of 483), pseudo bilateral posterior canal BPPV = 43 per cent (31 out of 72) and true bilateral posterior canal BPPV = 44.4 per cent (8 out of 18). Time of recurrence ranged between three months and eight years. Comparison of the recurrence frequency in patients with bilateral (true and pseudo) and unilateral posterior canal BPPV was statistically significant ($p < 0.05$) (Table 1).

There were 49 males and 102 females with recurrent unilateral posterior canal BPPV, and 12 males and 27 females with recurrent bilateral (pseudo and true) posterior canal BPPV. Comparison of the male to female gender ratio in patients with recurrent and non-recurrent unilateral and recurrent and non-recurrent bilateral posterior canal BPPV was not statistically significant ($p = 0.841$).

Twenty-five patients with recurrent unilateral posterior canal BPPV and four patients with recurrent bilateral (pseudo and true) posterior canal BPPV were aged over 60 years. Comparison of the number of older patients with recurrent and non-recurrent unilateral and recurrent and non-recurrent bilateral posterior canal BPPV was not statistically significant ($p = 0.329$).

Thirty-two patients with recurrent unilateral and 11 patients with recurrent bilateral (pseudo and true) posterior canal BPPV had associated problems. Comparison of the rate of associated problems in patients with recurrent and non-recurrent unilateral and recurrent and non-recurrent bilateral posterior canal BPPV was not statistically significant ($p = 0.351$). Table 2 demonstrates the comparative data of patients with recurrent and non-recurrent unilateral and bilateral (pseudo and true) posterior canal BPPV in terms of age, associated problems and gender.

Discussion

The reported incidence of simultaneous involvement of different canals varies greatly in previously published studies, ranging from 6.8 per cent to 20 per cent of all BPPV cases. This high percentage of multiple canal involvement has been artificially influenced by the number of bilateral posterior canal cases that may reflect the difficulty in distinguishing true from pseudo bilateral cases.[10] Pollak and Michael have reported that the incidence of bilateral posterior canal BPPV is 6.9 per cent of all cases of posterior canal BPPV.[11] However, Imai *et al.* have reported that bilateral posterior canal BPPV accounted for 1.4 per cent of cases,[4] and Hotta *et al.* reported a 2.2 per cent incidence.[3] The incidence of true and pseudo bilateral posterior canal BPPV in this study was 3.1 per cent and 12.6 per cent, respectively. Pseudo bilateral posterior canal BPPV is more common than true bilateral

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Table 1. Comparative data of patients with unilateral, pseudo and true bilateral posterior canal BPPV

Groups	Unilateral posterior canal BPPV	Pseudo bilateral posterior canal BPPV	True bilateral posterior canal BPPV	*P*-value
Site of involvement			N/A	0.828
– Left/right ear ratio	1.1	1.2		
– Left ear involvement (*n*)	255	39		
– Right ear involvement (*n*)	228	33		
Patients' sex				0.200
– Female/male ratio	1.7	2.1	3.5	
– Females (*n*)	304	49	14	
– Males (*n*)	179	23	4	
Associated problems (*n* (%))	80/483 (16.5)	17/72 (23.6)	N/A	0.142
Trauma (*n* (%))	9/483 (1.8)	5/72 (6.9)	3/18 (16.7)	<0.05
Number of manoeuvres (mean ± SD)	1.45 ± 0.71	1.61 ± 0.81	2.27 ± 0.57	<0.05
Recurrence rate (% (*n*))	31.3 (151/483)	43 (31/72)	44.4 (8/18)	<0.05

BPPV = benign paroxysmal positional vertigo; N/A = not applicable; SD = standard deviation

cases. In patients with a bilateral presentation of posterior canal BPPV, care should be taken to isolate true bilateral cases.

Studies on comparative analysis of the clinical characteristics of unilateral, true and pseudo bilateral posterior canal BPPV are very few. In contrast to unilateral posterior canal BPPV, which is often idiopathic, trauma is the main aetiological factor in patients with bilateral posterior canal BPPV.[12,13] Katsarkas reported that the incidence of bilateral BPPV was significantly higher among post-trauma patients compared to those with idiopathic BPPV (14.3 per cent *vs* 6.3 per cent).[13] Our findings were compatible with previous reports indicating that patients with bilateral BPPV had a significantly higher incidence of trauma than did those with unilateral BPPV. However, no other prominent factor has been identified. Vibert *et al.* confirmed clinical osteoporosis in 24 of 32 older women with BPPV (75 per cent). Six of the study group had bilateral BPPV.[14] Comparisons of the male to female ratio, the incidence of associated problems, and patient age of over 60 years were not statistically significant between unilateral and pseudo bilateral posterior canal BPPV, and between patients with recurrent and non-recurrent unilateral and recurrent and non-recurrent bilateral posterior canal BPPV, in the present study. Analyses of older age, sex and the presence of associated problems do not indicate a predisposition to a bilateral occurrence and the risk of recurrence.

Bilateral occurrence of true posterior canal BPPV is oftentimes refractory to repositioning manoeuvres. Understanding the correlation between the provocative head movements and the resulting symptoms with the site of involvement is usually difficult in patients with bilateral involvement. Simultaneous recovery of both sides with one manoeuvre is unlikely, as Epley's manoeuvre, performed to remove the otoconia from the canal in the affected ear, leads to ampullopedal displacement of the otoconia in the contralateral ear.[11] More therapeutic sessions are required for patients with bilateral posterior canal BPPV, and it takes a longer time to achieve symptom resolution, when compared to those with unilateral posterior canal BPPV. Recurrence is high when compared to unilateral cases. Pollak and Michael reported a 47 per cent recurrence rate in bilateral cases during a mean follow up of 9.5 ± 5 years.[11] Comparisons of the average number of manoeuvres required to provide symptom relief and the recurrence ratio, in patients with unilateral, pseudo and true bilateral posterior canal BPPV, were statistically significant in the present study.

Table 2. Comparative data of patients with recurrent unilateral, pseudo and true bilateral posterior canal BPPV

Groups	Unilateral posterior canal BPPV	Pseudo & true bilateral posterior canal BPPV	*P*-value
Recurrence according to age (*n*)			0.329
– Patients aged >60 years	25	4	
– Patients aged <60 years	126	35	
Recurrence according to associated problems* (*n*)			0.351
– Patients with associated problems	32	11	
– Patients without associated problems	119	28	
Patients' sex in recurrent cases			0.841
– Female/male ratio	2.1	2.3	
– Females (*n*)	102	27	
– Males (*n*)	49	12	

*For example, migraine, diabetes, hypertension, osteoporosis, coronary disease, thyroid disorders, arthrosis, anaemia, asthma, cancer, hepatitis. BPPV = benign paroxysmal positional vertigo

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Multiple therapeutic manoeuvres without favourable outcomes can be disabling in some patients, and occasionally surgery becomes the only treatment of choice. Plugging surgery eliminates the response of the posterior canal ampulla to angular acceleration in the plane of the canal, by preventing endolymph flow, but it is not clear whether the surgery causes fixation of the cupula. When the canal is occluded, canaliths were assumed to be immobilised within the canal between the site of occlusion and the cupula. Therefore, the intervention is expected to be effective, particularly in patients with canalolithiasis. Nevertheless, patients with cupulolithiasis may be symptomatic following surgery.[15] One of the drawbacks of the surgery is vertical oscillopsia and persistent reduction of the vertical vestibulo-ocular reflex. Another reservation regarding surgery is the recurrence of symptoms in about 40 per cent of patients, indicating the involvement of other canals.[16] Vestibular function is expected to improve within 6–12 months by way of central vestibular compensation due to the complementary function of other canals. In addition, partial recovery of the vestibulo-ocular reflex with time indicates that it is just loss of function, rather than the surgery causing complete ablation. However, bilateral occlusion surgery will lead to bilateral vestibulo-ocular reflex deficit.[16] Nevertheless, bilateral occlusion may not be necessary for some patients, as the other side may improve spontaneously or may resolve following more conservative treatment.[3]

One of the strengths of the study is that the Dix–Hallpike manoeuvre is always applied bilaterally in our vestibular testing room, even if the patient has unilateral BPPV. Therefore, there is no uncertainty regarding whether the contralateral side is tested. The study is limited by the fact that it is based on retrospective chart review; some particular points, such as the role of trauma or the presence of associated problems, could have been overlooked in some patients during the interview.

- Bilateral posterior canal benign paroxysmal positional vertigo (BPPV) could reflect true bilateral involvement or unilateral posterior canal BPPV mimicking bilateral involvement
- Clinical characteristics, therapeutic outcome and recurrences rates of pseudo and true bilateral posterior canal BPPV are not well described
- In this study, the number of manoeuvres required for symptom relief in unilateral, pseudo and true bilateral posterior canal BPPV cases varied significantly ($p < 0.05$)
- Trauma incidence was high in bilateral cases ($p < 0.05$), and recurrence rate was different in bilateral (true and pseudo) and unilateral posterior canal BPPV cases
- Differentiation of true and pseudo bilateral posterior canal BPPV is important, as aetiology and treatment outcomes differ
- Treatment of true bilateral posterior canal BPPV involves several therapeutic manoeuvre attempts, and there is risk of recurrence

Conclusion

It is crucial to differentiate true bilateral and pseudo bilateral posterior canal BPPV given the differences in treatment outcome. Some patients with posterior canal BPPV may have positional nystagmus on the healthy side that is short-lived, less severe and which has a lower amplitude. The mechanism is unclear. Some authors claim 'inhibitory' positional nystagmus.

The lying down positional or straight head-hanging tests are very helpful to differentiate true from pseudo bilateral cases. Positional up-beating vertical nystagmus is torsional in cases of pseudo bilateral posterior canal BPPV. If it is clockwise, it indicates a left ear problem and if it is counterclockwise it indicates an issue with the right ear. However, in the case of bilateral true posterior canal BPPV, positional nystagmus seen during the lying down test is purely vertical. The most prominent sign for true posterior canal BPPV which fulfils the diagnosis is the presence of reverse nystagmus when the patient returns to an upright sitting position from a head-hanging position. However, one should be mindful that reverse nystagmus sometimes cannot be seen in unilateral cases. Positional nystagmus on the healthy side in patients with pseudo bilateral posterior canal BPPV does not have any prognostic impact on the outcome, and those patients can be treated as having unilateral posterior canal BPPV. However, several attempts of therapeutic manoeuvres are required for patients with true bilateral posterior canal BPPV.

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