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Benign Paroxysmal Positional Vertigo Associated With Meniere's Disease: Epidemiological, Pathophysiologic, Clinical, and Therapeutic Aspects

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Objectives: We studied the demographic, pathogenetic, and clinical features of benign paroxysmal positional vertigo (BPPV) associated with Meniere's disease.

Methods: The medical records of patients with BPPV associated with Meniere's disease were reviewed. In all patients, results of a complete otolaryngological, audiological, and neurotologic evaluation, including nystagmography, were available. Patients with idiopathic BPPV were used as a control group.

Results: Twenty-nine patients with both disorders were found and were compared with 233 patients with idiopathic BPPV. The patients with BPPV associated with Meniere's disease presented the following features, in which they differed from the patients with idiopathic BPPV: 1) a higher percentage of female patients; 2) a longer duration of symptoms; 3) common involvement of the horizontal semicircular canal; 4) a greater incidence of canal paresis; and 5) more therapeutic sessions needed for cure and a higher rate of recurrence.

Conclusions: The BPPV associated with Meniere's disease differs from idiopathic BPPV in regard to several epidemiological and clinical features, may follow a different course, and responds less effectively to treatment.

Key Words: benign paroxysmal positional vertigo, canalith repositioning procedure, hearing loss, Meniere's disease, nystagmography, vertigo.

Benign paroxysmal positional vertigo (BPPV) is the commonest clinical entity encountered in a neurotology clinic on an outpatient basis, with a lifetime prevalence of 2.4%.[1] It can be defined as transient vertigo induced by a rapid change in head position, associated with a characteristic paroxysmal positional nystagmus. The nystagmus may be torsional, vertical, or horizontal and is characterized by findings such as latency, crescendo, transience, reversibility, and fatigability.[2]

Clinical and laboratory research has shown that BPPV is caused by vestibular lithiasis, from otoliths originating from a degenerating utricular macula. Either free-floating otoliths within the semicircular canals (canalolithiasis) or otoliths attached to, or impinging upon, a cupula (cupulolithiasis) provoke an abnormal deflection to the cupula, inducing vertigo and nystagmus in the plane of the involved semicircular canal (SCC).[3] Although in most cases of BPPV the posterior SCC is involved, BPPV of the horizontal SCC also occurs, at a rate ranging from 5% to 30%, according to various reports.[3,4] More rarely, involvement of the anterior SCC may be observed.[5] Disease of multiple canals, either bilaterally or on the same side, also occurs, but represents only a small fraction of cases of BPPV.

In most cases, BPPV occurs spontaneously, but it may be secondary to various other conditions, including head trauma, viral neurolabyrinthitis, Meniere's disease, and vertebral-basilar ischemia, or it may be the result of surgery and prolonged bed rest.[6] It seems that any inner ear disease that detaches otoconia and yet does not totally destroy SCC function can induce secondary BPPV. Reports that idiopathic and secondary cases of BPPV differ in several respects imply that the pathology or pathophysiology of secondary BPPV may differ quantitatively or qualitatively from that of idiopathic BPPV. However, few studies have focused on secondary BPPV, which may be an underdiagnosed entity.

Meniere's disease is another common vestibular entity characterized by episodes of vertigo, fluctuating hearing loss, tinnitus, and ear fullness.[7] Be-

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nign paroxysmal positional vertigo may be associated with Meniere's disease and may occur at any stage of this disease. On the other hand, Meniere's disease may be considered as one of the causes of persistent vertigo in patients with BPPV, posing difficulties in obtaining the right diagnosis and aggravating the prognosis.[6] The aim of this study was to investigate a group of patients who presented with BPPV in conjunction with Meniere's disease that was diagnosed and treated in the neurotology unit of an otolaryngology department during the past 5 years. The demographic, clinical, pathogenetic, and nystagmographic features and treatment outcomes of this group were studied and compared with those of a group with idiopathic BPPV.

METHODS

During the past 5 years, 345 patients examined at the neurotology unit of our department received diagnoses of BPPV. Among them, 29 patients had had a previous diagnosis of Meniere's disease. The clinical records of these patients were retrospectively reviewed. The patient's age on initial diagnosis of BPPV, sex, and duration of symptoms were recorded. To evaluate the severity of the vertiginous symptoms, we used the following scale[8]: 1 — slight vertigo in the provoking position without autonomous symptoms; 2 — severe vertigo with nausea; 3 — severe vertigo with severe nausea, vomiting, or hypotension. Patients with any clinical, laboratory, or imaging findings suggesting a disorder of the central nervous system were excluded. Patients with idiopathic BPPV examined and treated during the same period were used as a control group. The protocol of the study was reviewed and approved by the local Institutional Review Board.

In all patients, a complete otolaryngological, audiological, and neurotologic evaluation was performed, including pure tone audiometry, measurements of acoustic immitance, and, occasionally, auditory brain stem response testing. Eye movements were recorded by electronystagmography or videonystagmography by use of a standard test protocol of visual and vestibular stimulation, described elsewhere.[9] The obtained nystagmographic data were compared with data from 78 randomly selected patients with idiopathic BPPV who underwent testing under similar conditions.

The diagnosis of Meniere's disease was based on the guidelines of the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS).[10] Most of these patients had already undergone disease-specific tests in the past, such as the glycerol test and electrocochleography. Patients hospitalized during an acute episode of Meniere's disease were not included, but are being studied separately in another ongoing prospective study.

All 345 patients underwent the Dix-Hallpike maneuver and the supine roll test. The Dix-Hallpike maneuver was considered positive for posterior (or anterior) SCC BPPV when vertigo was provoked, accompanied by a burst of torsional-vertical two-component nystagmus with the typical characteristics of latency, crescendo, and transience. The supine roll test was considered positive for horizontal SCC BPPV when intense vertigo was provoked, accompanied by horizontal geotropic (canalolithiasis) or apogeotropic (cupulolithiasis or canalolithiasis of the short arm of the horizontal SCC) paroxysmal nystagmus.[11]

Posterior SCC BPPV was treated by the modified Epley canalith repositioning procedure,[2] and horizontal SCC BPPV was treated by the barbecue maneuver or the Gufoni maneuver.[12] Repeat treatment sessions were performed every 2 or 3 days, in case of failure or incomplete remission of the symptoms, to a maximum of 7 sessions. Assessment of the success of the treatment included both the patient's report of relief from vertigo for at least 2 months and a negative Dix-Hallpike test or supine roll test result. In case of recurrence of symptoms, canalith repositioning procedures were repeated according to the same plan. Follow-up data were available for most patients for more than 1 year.

Continuous variables were expressed as mean ± SD, and categorical variables were expressed as frequencies and percentages. The significance of any difference between groups was evaluated by *t*-test for independent samples. The χ^2 test was used to evaluate any potential association between categorical variables, and the Fisher exact probability test was used for comparisons with small samples. Odds ratios and 95% confidence intervals were calculated for the estimation of treatment results.

RESULTS

We found 29 patients with Meniere's disease associated with BPPV, indicating a prevalence of 8.4% of this clinical entity in patients with BPPV. The demographic and clinical features of the patients are shown in Table 1. From the remaining 316 patients with BPPV, another 83 patients (24.0%) were found to have secondary BPPV due to other possible pathogenetic factors. The etiologic factors included cochleovestibular disease, such as vestibular neuritis or chronic otitis media (31 patients; 8.9%), head trauma (33 patients; 9.6%), and other causes, such as surgery or prolonged bed rest (19 patients; 5.5%).

TABLE 1. DEMOGRAPHIC AND CLINICAL FEATURES OF PATIENTS WITH BPPV WITH MENIERE'S DISEASE AND OF PATIENTS WITH IDIOPATHIC BPPV

	BPPV With Meniere's Disease (N =.29)		*Idiopathic BPPV (N = 233)*	*p*
Gender				<0.001
Male	2 (6.9%)		95 (40.8%)	
Female	27 (93.1%)		138 (59.2%)	
Age (years)				
Mean	55.6 ± 9.5		53.1 ± 9.9	0.12
Range	37-74		25-86	
Mean (±SD) duration of BPPV symptoms (months)	7.3 ± 8.3		3.8 ± 5.8	<0.001
Mean (±SD) duration of Meniere's disease (years)				
Total cases	11.8 ± 7.7			
0-5 years (N = 3)	2.2 ± 1.2			
6-9 years (N = 11)	6.7 ± 1.9			
≥10 years (N = 15)	17.4 ± 6.6			
Side of involvement	Meniere's	BPPV*		0.41
Right	14 (48.3%)	14 + 3 (58.6%)	122 (52.3%)	
Left	11 (37.9%)	11 + 1 (41.4%)	95 (40.8%)	
Bilateral	4 (13.8%)	3R, 1L†	16 (6.9%)	
Semicircular canal involved				<0.05
Posterior	22 (75.9%)		181 (77.7%)	
Horizontal	7 (24.1%)		25 (10.7%)	
Anterior or multiple			27 (11.6%)	
Vertigo severity‡				0.66
1	1 (3.5%)		25 (10.7%)	
2	25 (86.2%)		187 (80.3%)	
3	3 (10.3%)		21 (9.0%)	

*Benign paroxysmal positional vertigo (BPPV) involvement was always unilateral and on same side as Meniere's disease.

†In 3 patients with bilateral Meniere's disease right ear, and in 1 patient left ear, was involved with BPPV.

‡1 — slight vertigo; 2 — severe vertigo with nausea; 3 — severe vertigo with nausea, vomiting, or hypotension.

Accordingly, pure idiopathic BPPV was found in 233 patients (67.5%). The demographic and clinical features of the patients of the control group with idiopathic BPPV are also shown in Table 1 and are compared statistically to those of the study group.

Twenty-seven patients (93.1%) with Meniere's-associated BPPV were female (mean age, 56.0 ± 9.8 years; range, 37 to 74 years), and 2 (6.9%) were male (53 and 56 years of age). The mean time from the onset of the symptoms of BPPV was 7.3 months (range, 1 to 36 months). The neurologic examination findings were normal in all patients, but to exclude a central disorder, some of them were given brain magnetic resonance imaging, which also yielded normal findings.

Four patients (13.8%) had bilateral Meniere's disease, and the remaining 25 (86.2%) had involvement ipsilateral to the SCC responsible for the BPPV. All patients with bilateral involvement had unilateral BPPV, 3 on the side with more severe disease, and 1 on the other side. Audiological evaluation of the patients with Meniere's-associated BPPV proved that 26 of them (89.7%) had stage 2 or 3 disease according to the AAO-HNS[10] classification, with hearing loss (4-tone average of thresholds at 0.5, 1, 2, and 3 kHz) varying between 26 and 70 dB. Only 3 patients (10.3%) from this group were in an early stage of the disease, presenting with a completely reversible hearing loss.

In 22 patients (75.9%), vertigo was provoked unilaterally during the Dix-Hallpike test, and in the remaining 7 patients (24.1%), vertigo was provoked during the supine roll test, implying involvement of the horizontal SCC. In 6 patients (20.7%), the observed nystagmus was geotropic (canalolithiasis), and in 1 patient (3.4%), the nystagmus was persistent apogeotropic (cupulolithiasis). No patient was found to have involvement of the anterior SCC or multiple canals. Twenty-seven patients (11.6%) from the group with idiopathic BPPV had multiple SCC or anterior SCC BPPV.

In the majority of patients (86.2%), the vertigo was intense, with accompanying nausea (grade 2). Statistically significant differences in the study group, as compared to the idiopathic BPPV group, were found regarding the following variables: gen-

TABLE 2. NYSTAGMOGRAPHIC FINDINGS IN PATIENTS WITH BPPV WITH MENIERE'S DISEASE AND IN PATIENTS WITH IDIOPATHIC BPPV

Nystagmographic Findings	*BPPV With Meniere's Disease (N = 29)*	*Idiopathic BPPV (N = 78)*	*p*
Spontaneous nystagmus	4	3	0.15
Canal paresis	17	19	<0.005
Bilateral	3	6	
Unilateral	14	13	
Diseased ear	14	10	
Normal ear		3	
Directional preponderance	8	12	0.24
Toward diseased ear	1	4	
Toward normal ear	7	8	
Canal paresis and directional preponderance	3	5	0.78
Central findings		1	0.59

der (more were female), duration of symptoms (longer duration), and SCC involvement (more common horizontal SCC involvement and absence of multiple canal or anterior SCC BPPV).

We performed videonystagmography in all patients with the exception of 2 (4.7%) who were submitted to electronystagmography. The results are shown in Table 2, and are compared statistically with the results of the group of 78 examined patients with idiopathic BPPV. In 4 patients (13.8%) with Meniere's disease associated with BPPV, a mild spontaneous nystagmus of peripheral type was found, without optic fixation. These patients also had canal paresis. A similar finding was observed in 3 patients (3.8%) of the idiopathic BPPV group. Canal paresis occurred at a higher percentage in the group with BPPV and Meniere's disease ($p < 0.005$): 17 patients (58.6%), as opposed to 19 patients (24.3%) in the idiopathic BPPV group. No central nystagmographic findings were found, with the exception of 1 patient (1.3%) with idiopathic BPPV who had impaired fixation suppression of the vestibulo-ocular reflex.

The modified Epley procedure was performed in all patients with Meniere's disease and posterior SCC involvement and proved successful in 20 of them (68.9%) — after 1 (5 patients; 17.2%), 2 (12 patients; 41.4%), or 3 (3 patients; 10.3%) treatment sessions (Table 3). In the 7 patients (24.1%) with horizontal SCC involvement, the Gufoni maneuver was applied appropriately, resulting in successful treatment of 6 patients (20.7%). A mean of 1.9 treatment sessions was needed in patients with posterior SCC involvement, and 2.3 sessions in patients with horizontal SCC BPPV. The rates of treatment success in patients with BPPV associated with Meniere's disease and patients with idiopathic BPPV were almost similar, with an odds ratio of 1.37 (95% CI, 0.38 to 4.99; $p = 0.62$) in favor of patients with typical BPPV. However, the patients with Meniere's disease needed more therapy sessions for treatment of the disease, because in only 20.7% of them was treatment initially successful, versus 78.5% in the idiopathic group. In 27 patients (93.1%), follow-up data were obtained at 12 months, and 12 of them presented recurrence of BPPV (44.4%). In contrast, the recurrence rate in the idiopathic group was only 13.3% ($p < 0.001$).

DISCUSSION

Although the association of BPPV and Meniere's disease has occasionally been reported,[13-16] there are still several unsettled issues concerning the true incidence of this combination, the possible pathogenetic mechanisms, the specific clinical characteristics, the difficulties in diagnosis, and the appropriate treatment and prognosis of BPPV in the background of Meniere's disease.

The incidence of Meniere's disease among patients with BPPV has been reported within the wide range of 0.5% to 30%. In earlier studies, low incidence

TABLE 3. TREATMENT RESULTS IN PATIENTS WITH BPPV WITH MENIERE'S DISEASE AND IN PATIENTS WITH IDIOPATHIC BPPV

	N (%)	*Success of First Treatment*	*Success of Repeat Treatment*	*Failure*
BPPV with Meniere's disease				
Posterior canal BPPV	22 (75.9%)	5 (17.2%)	15 (51.7%)	2 (6.9%)
Horizontal canal BPPV	7 (24.1%)	1 (3.5%)	5 (17.2%)	1 (3.4%)
Total	29 (100%)	6 (20.7%)	20 (68.9%)	3 (10.3%)
Idiopathic BPPV				
Posterior canal BPPV	181 (77.7%)	156 (66.9%)	10 (4.3%)	15 (6.4%)
Horizontal canal BPPV	25 (10.7%)	16 (6.9%)	7 (3.0%)	2 (0.9%)
Anterior canal BPPV	14 (6.0%)	11 (4.7%)	3 (1.3%)	
Multiple canal BPPV	13 (5.6%)		12 (5.2%)	1 (0.4%)
Total	233 (100%)	183 (78.5%)	32 (13.7%)	18 (7.7%)

rates were reported; eg, Katsarkas and Kirkham[13] found that of 255 patients with BPPV, only 3 (0.8%) had Meniere's disease. Baloh et al[14] found that only 5 patients (2%) had Meniere's disease among 240 patients with BPPV. More recently, Karlberg et al[17] found only 16 patients with Meniere's disease in a group of 2,847 patients (0.5%). However, Hughes and Proctor[18] reported that 45 of 151 (29.8%) patients with BPPV had associated Meniere's disease, which is the highest incidence reported so far. A possible explanation of the difference is their inclusion of patients who had vestibular function tests on more than one occasion, resulting in selection of patients with recurrent disorders, such as Meniere's disease. Li et al[19] found 15 cases of confirmed BPPV among 150 hospitalized patients with Meniere's disease (10%), and Lee et al[20] found 20 patients with Meniere's disease in a group of 718 patients with BPPV (3%). Ganança et al,[21] in a group of 1,946 patients, found 1,033 cases of BPPV (53.1%), 841 cases of Meniere's disease (43.2%), and 72 cases of BPPV associated with Meniere's disease (3.7%). On the other hand, Paparella[22] reported that in 500 patients with Meniere's disease, approximately 65% to 70% experienced BPPV between attacks of the disease.

We found 29 patients with Meniere's disease in a group of 345 patients with BPPV (8.4%). The larger proportion of patients with Meniere's disease and associated BPPV observed recently may be explained by the increased awareness of BPPV in the past decade due to the development of the canalith repositioning procedures, so that the Dix-Hallpike test and the supine roll test might now be more often performed in patients with known Meniere's disease. Also, timely and accurate diagnosis of BPPV is now widely performed, increasing the population of patients with diagnosed BPPV. Finally, significant progress in diagnosis of horizontal SCC BPPV, which is quite common in Meniere's disease, has been recently achieved. Discrepancies in the reported incidence between studies might be due to differences in the study design, different patient populations, and/or use of various inclusion and exclusion criteria and criteria for diagnosis. It should be noted that Meniere's disease is quite difficult to diagnose, despite published criteria, and older studies[13,14,18] obviously did not use the AAO-HNS 1995 criteria, whereas more recent studies[17] used additional measures, such as positive electrocochleographic findings.

One issue that should be discussed is whether a true causative effect exists between Meniere's disease and BPPV, or whether their association is merely coincidental. In the present study and in several previous ones,[17,18,21] the fact that almost all patients had BPPV in the same ear as their Meniere's disease indicates a causal relationship between the two disorders. It has been hypothesized that the presence of endolymphatic hydrops may result in destruction of the maculae of the utricle and saccule, either through compromise of the vascular supply or through detachment of otoconia into the endolymph.[6,17] Additionally, Manzari[23] suggested that BPPV may be induced not only by changes in endolymphatic fluid, but also by structural alterations of the vestibular aqueduct. A prolonged course of Meniere's disease may enhance detachment of otoconia through macular fibrosis.[24] Accordingly, an increased rate of BPPV in patients with advanced Meniere's disease should be expected, as was evident in the present study (only 3 patients found in an early stage of the disease) and in previous reports.[17,19]

The above pathogenetic mechanisms have been further supported by temporal bone studies. Morita et al[25] found significant differences in the incidence of cupular and free-floating deposits in the posterior and lateral SCCs between temporal bones with and without Meniere's disease. They observed that the incidence of these deposits was associated with the duration of disease, rather than with aging. Although most authors support the idea that BPPV is secondary to Meniere's disease,[17,18,21] Paparella[22] proposed that Meniere's disease secondary to BPPV is possible as well, because loose otoconia could cause a decrease in endolymphatic absorption, resulting in endolymphatic hydrops.

Several authors have stated that secondary BPPV has specific clinical characteristics that differ from those of idiopathic BPPV.[6,17] We found this to be true in our patients with BPPV secondary to Meniere's disease. The features that distinguish this clinical entity from idiopathic BPPV are briefly discussed below.

The first feature is female predominance. Although this feature follows the current epidemiology of both Meniere's disease and idiopathic BPPV,[7,15] in patients with the combination of the two diseases the percentage of female patients is even higher. In the present study, it exceeds 93%, which is in accordance with previous reports.[17,19,20]

Second is the longer duration of symptoms. A probable explanation could be either different pathogenetic mechanisms, or difficulties in treatment.

Horizontal SCC involvement appears to be a cardinal feature of secondary BPPV. We found 7 patients with horizontal SCC BPPV, accounting for a rate of 24.1%, as compared to 10.7% in patients with idiopathic BPPV. Lee et al[20] found that 65%

of their patients had horizontal SCC BPPV. However, in previous studies this finding was not observed.[17,21] There is not a clear explanation for a horizontal SCC predilection or the discrepancies between studies. This finding may be attributed to the recently achieved progress in the diagnosis of horizontal SCC BPPV and to a different pathogenetic mechanism. Buckingham,[26] after studying photographs of macrosections of human temporal bones at the level of the utricular macula, reported that because of the adjacent location of the ampulla of the horizontal SCC, loose otoliths could more easily slide onto the horizontal SCC cupula when a supine patient turns to one side or the other. Hence, the horizontal SCC could be more susceptible to lithiasis than the posterior canal. We might thus assume that anatomic factors predominate in Meniere's-associated BPPV over the gravity factor, which is responsible for the predilection for the posterior SCC of idiopathic BPPV.[15]

The finding of more frequent canal paresis in our patients is not surprising, since this is a common finding in patients with Meniere's disease, especially in the advanced stage.[9]

Finally, the treatment of patients with BPPV associated with Meniere's disease appears to be less effective and more time-consuming than that of patients with idiopathic BPPV. We had success with the first canalith repositioning procedure in only 20.7% of our patients, whereas 68.9% needed 2 or more sessions, and in 10.3% treatment was not successful. The corresponding percentages in the patients with idiopathic BPPV were 78.5%, 13.7%, and 7.7%. Li et al[19] needed 3 or 4 sessions for the treatment of 66.7% of their patients and 5 sessions for the treatment of 26.7%. Also, Gross et al[24] reported 9 patients with definite Meniere's disease who had intractable BPPV. However, better results have been reported in other studies.[20,21]

High recurrence rates have been reported in most previous studies[27-30] and in the present study, as well. Even authors who reported successful treatment results[21,27] found recurrence rates as high as 50%. Several factors may explain the possible worse treatment results and higher rate of recurrence in patients with both BPPV and Meniere's disease.[6,19,24] First, repeated hydropic distention may reduce the elasticity of the membranous labyrinth and result in partial collapse or adhesions of the SCC, which therefore may exhibit partial obstruction. Accordingly, multiple canalith repositioning procedures would be needed for effective treatment, and still a higher rate of failure is possible. Second, partial obstruction may also be due to a dilated saccule or adhesion of otoliths to the membranous labyrinth. Finally, periodic hydropic distention, which is observed during the natural course of Meniere's disease, may result in repeated release of otoconia and manifestation of BPPV attacks.

In conclusion, patients with BPPV associated with Meniere's disease differed from patients with idiopathic BPPV in the following ways: 1) a higher percentage of female patients; 2) a longer duration of symptoms; 3) more frequent involvement of the horizontal SCC; 4) a greater incidence of canal paresis; and 5) poorer treatment results and a higher rate of recurrence. The above findings may imply that BPPV associated with Meniere's disease differs from idiopathic BPPV in terms of several demographic and clinical features, that it may follow a different course, and that it responds less effectively to treatment.

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