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Original Article

Benign Paroxysmal Positional Vertigo Secondary to Acute Unilateral Peripheral Vestibulopathy: Evaluation of Cardiovascular Risk Factors

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BACKGROUND: Lindsay–Hemenway syndrome was first described as an acute unilateral peripheral vestibulopathy followed by positional vertigo. A vascular etiology was proposed. An association between cardiovascular risk factors and benign paroxysmal positional vertigo secondary to acute unilateral peripheral vestibulopathy has been described with contradictory evidence. The study aimed to evaluate the prevalence of cardiovascular risk factors in patients with benign paroxysmal positional vertigo secondary to acute unilateral peripheral vestibulopathy and analyze differences in prior history of benign paroxysmal positional vertigo, affected semicircular canals, and response to repositioning maneuvers between patients with idiopathic benign paroxysmal positional vertigo and secondary to acute unilateral peripheral vestibulopathy.

METHODS: We performed a retrospective, descriptive study of all cases of benign paroxysmal positional vertigo between January/2017 and June/2020, with or without a history of acute unilateral peripheral vestibulopathy within the previous year. Cases secondary to trauma or oto-neurological causes and acute unilateral peripheral vestibulopathy without confirmatory tests and cases with auditory symptoms were excluded.

RESULTS: In total, 242 cases were obtained; 158 idiopathic benign paroxysmal positional vertigo and 84 secondary to acute unilateral peripheral vestibulopathy. No statistically significant differences were found in relation to age: 61.2 ± 14.6 versus 62.4 ± 16.2 years ($P = .55$), sex: female 78.5% versus 73.8% ($P = .41$), presence of cardiovascular risk factors: 52.5% versus 54.8% ($P = .67$), prior history of benign paroxysmal positional vertigo: 22.2% versus 27.7% ($P = .43$), affected semicircular canals ($P = .16$) or number of repositioning maneuvers ($P = .57$).

CONCLUSION: Associations between age, cardiovascular risk factors, and benign paroxysmal positional vertigo secondary to acute unilateral peripheral vestibulopathy have been described with conflicting evidence. This is the first study to evaluate cardiovascular risk factors specifically for Lindsay–Hemenway syndrome, and we did not observe any differences between idiopathic benign paroxysmal positional vertigo cases and those secondary to acute unilateral peripheral vestibulopathy.

KEYWORDS: Lindsay–Hemenway syndrome, vestibular neuritis, benign paroxysmal positional vertigo, acute vestibular syndrome, vertigo, acute unilateral peripheral vestibulopathy

INTRODUCTION

Lindsay–Hemenway syndrome was described by John R. Lindsay and William G. Hemenway in 1956 as an acute unilateral peripheral vestibulopathy (AUPVP) followed by prolonged positional vertigo with peripheral characteristics, and suggested that the etiology was ischemia of the anterior vestibular artery.[1] Since the anterior vestibular artery irrigates the territory of the utricle, a portion of the saccule, and the ampullae of the anterior and horizontal semicircular canals (SC), ischemia of this artery can cause AUPVP. With the consequent ischemic damage to the utricle, the utricular macula degenerates and otoconia can detach and migrate toward the ampullae of the SCs. Because the posterior SC is supplied by the posterior vestibular artery, it is spared and is therefore functional. Hence, free-floating otoconia in this canal lead to benign paroxysmal positional vertigo (BPPV) of the posterior SC.[2]

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Clinically, it presents as an acute vestibular syndrome, that is acute onset of dizziness, unsteadiness, vertigo, nystagmus with peripheral features, and nausea and/or vomiting, lasting at least 24 hours to several days, followed by BPPV of the posterior canal. Acute unilateral peripheral vestibulopathy without neurological or auditory symptoms is traditionally called vestibular neuritis.[3-5] However, there is no definitive way to differentiate if the AUPVP is due to ischemia of the anterior vestibular artery or if it is an inflammatory process of the vestibular nerve itself, hence, vestibular neuritis. Only histopathological studies would provide such evidence.[6-8]

If the etiology is in fact ischemia, it is logical to assume that patients with cardiovascular risk factors (CRFs) would be at greater risk of developing Lindsay–Hemenway syndrome. Drawing a parallel with hearing, a recent systematic review describes that sudden sensorineural hearing loss could be an independent risk factor for developing a stroke, especially in patients above 50 years.[9] It has also been suggested that elevated homocysteine levels, smoking, and elevated alcohol consumption may increase the risk of sudden sensorineural hearing loss.[10,11] As a result, the question arises as to whether the etiology of Lindsay–Hemenway syndrome is vascular in origin, and whether cardiovascular risk factors are more frequent as compared to patients with BPPV without AUPVP. One might think that Lindsay–Hemenway syndrome would be more frequent in men, older patients, those with cardiovascular risk factors, or with a prior history of stroke or transient ischemic attack (TIA) as compared to patients with idiopathic BPPV.

Therefore, we conducted a study to evaluate the presence of cardiovascular risk factors in patients with BPPV after presenting with AUPVP and to evaluate the differences in prior history of BPPV, affected SC, and response to repositioning maneuvers between patients presenting with BPPV with and without AUPVP.

METHODS

A retrospective, descriptive study was performed including all cases of BPPV from January 2017 to June 2020, with or without a history of AUPVP within the previous year. Acute unilateral peripheral vestibulopathy cases were defined as acute onset of dizziness, unsteadiness and/or vertigo, with nystagmus with peripheral features, and nausea and/or vomiting, lasting at least for 24 hours, a clinical presentation compatible with vestibular neuritis. Benign paroxysmal positional vertigo cases secondary to trauma or otoneurological etiologies, cases with AUPVP but lacking confirmatory tests of vestibular hypofunction, and cases with auditory symptoms were excluded. Vestibular hypofunction was defined as unilateral weakness cut-off at 25% side difference on caloric testing and/or video head impulse (VHIT) testing with a gain ≤ 0.8 for the horizontal canal. Benign paroxysmal positional vertigo was diagnosed based on the diagnostic criteria of the Bárány Society.[12]

In this time period, we identified 1222 cases of BPPV. After eliminating incomplete records and excluding cases according to the aforementioned exclusion criteria, we obtained 242 cases, of which 158 were idiopathic BPPV, and 84 presented AUPVP within the previous year. The clinical records were reviewed to obtain demographic data, history of cardiovascular risk factors, affected SC, and repositioning maneuvers. The study was approved by the local ethics committee, ID 200724005.

Statistical Analysis

A descriptive analysis of all data was performed and was reported as means, percentages, medians, and standard deviations. The comparison of quantitative variables was performed using Student's *t*-test and for categorical variables, using chi-square test, to assess differences between the two groups. A $P < .05$ was considered statistically significant.

RESULTS

Demographic characteristics were similar between both groups, and no statistically significant differences were identified with regard to age or sex between patients with idiopathic BPPV and BPPV with AUPVP. The average age in the group with idiopathic BPPV was 61.2 ± 14.6 years, and in the group of BPPV with AUPVP, 62.4 ± 16.2 years ($P = .55$). The majority of patients in both groups were female, with 78.5% and 73.8% for BPPV and BPPV with AUPVP, respectively ($P = .41$). Regarding the group of BPPV with AUPVP, the average unilateral hypofunction observed with caloric testing was 44 ± 14.4% (range: 28%-80%). Only 3 patients in this time period had VHIT testing with low gains, and the average unilateral horizontal canal gain for the affected canal was 0.75. Interestingly, while 81% of the BPPV cases were consistent with vestibular hypofunction ipsilaterally, some patients (n = 16) had BPPV on the contralateral side (n =13/16 were posterior canal). We did not observe an association between the degree of unilateral hypofunction and the canal involved, but the posterior canal was most frequently involved in general, even when the degree of hypofunction was high. Multicanal involvement was not more frequent when a greater degree of hypofunction was present in this study.

Table 1. Cardiovascular Risk Factors for Both Groups

	Idiopathic BPPV (n = 158)	BPPV Secondary to AUPVP (n = 84)	*P*
Cardiovascular risk factor	52.5%	54.8%	.67
Arterial hypertension	39.9%	47.6%	.25
Diabetes mellitus	17.7%	10.7%	.15
Dyslipidemia	15.2%	17.9%	.60
Prior stroke or TIA	4.5%	8.4%	.22
Active smoking	17%	15.6%	.85
≥2 risk factors	17%	19%	.73

AUPVP, acute unilateral peripheral vestibulopathy; BPPV, benign paroxysmal positional vertigo; TIA, transient ischemic attack.

Table 2. Quantity of Repositioning Maneuvers for Both Groups

	Idiopathic BPPV	BPPV Secondary to AUPVP	*P*
Prior history of BPPV	22.2%	27.7%	.34
Repositioning maneuvers	97.4%	98.8%	.57
0	1.2%	1.2%	
1	67.9%	60.9%	
2	17.9%	18.3%	
≥3	12.8%	19.5%	

AUPVP, acute unilateral peripheral vestibulopathy; BPPV, benign paroxysmal positional vertigo.

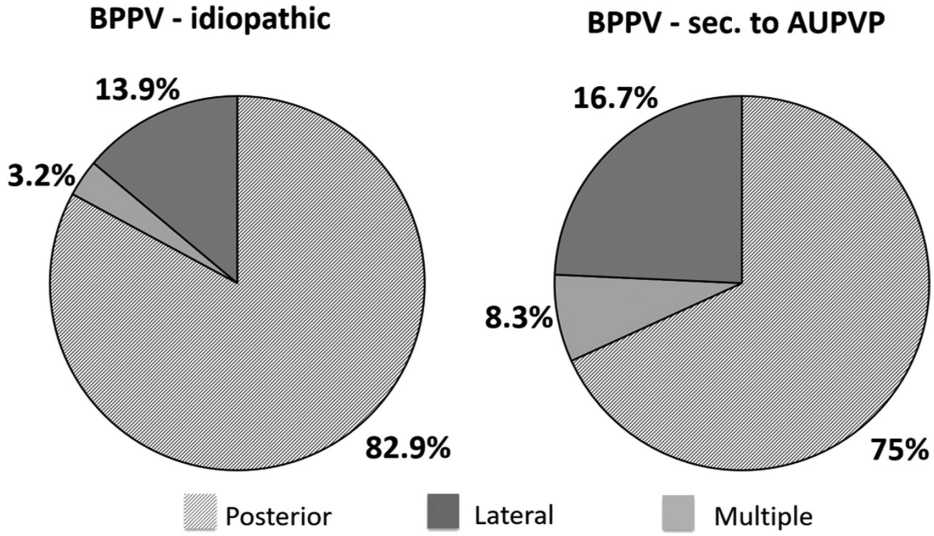

Figure 1. Semicircular canals involved in BPPV for patients with and without AUPVP. The posterior canal was most often involved in both cases, followed by the lateral, or horizontal, canal, and then, multiple canals. There were no significant differences between the canals involved in cases of idiopathic BPPV and secondary to AUPVP. BPPV, benign paroxysmal positional vertigo; AUPVP, acute unilateral peripheral vestibulopathy.

With regards to cardiovascular risk factors (Table 1), there were neither statistically significant differences between the two groups with regards to the presence of hypertension, diabetes mellitus, dyslipidemia, history of stroke, TIA, or active smoking nor there were significant differences when assessing whether two or more risk factors were present. Overall, 52.5% and 54.8% of patients with BPPV and BPPV with AUPVP, respectively ($P = .67$), had at least one CRF.

Neither did the number of repositioning maneuvers required between the two groups differ nor did the history of previous BPPV or the affected SC (Table 2). In both groups, with one maneuver, more than half of the cases were resolved, while ≈18% required a second maneuver. When reviewing the SC affected by BPPV (Figure 1), we observed that the most frequent SC involved was the posterior canal in both groups, followed by the horizontal canal. There was no statistically significant difference between the SC involved in both groups ($P = .16$). In the idiopathic group, 82.9% involved the posterior canal, 13.9% were cases of horizontal canal, and 3.2% involved multiple canals. For the secondary BPPV group, 75% involved the posterior canal, 16.7% were lateral canal, and 8.3% involved multiple canals. Here, we would like to emphasize that the vascular theory suggests that it would be a posterior canal BPPV post-utricular damage, because the posterior SC would be spared as it irrigated from the posterior vestibular artery and would be undamaged in cases of Lindsay–Hemenway syndrome. Yet, in our cohort, we observed that in cases of BPPV with AUPVP, there were not only cases of posterior canal but also cases of horizontal or multicanal BPPV as well.

We also evaluated the time elapsed between the onset of the AUPVP and the appearance of secondary BPPV (Figure 2). The vast majority presented concomitantly (within a month), at 72.6%. As time goes by, BPPV becomes less and less likely to appear, only appearing in 4.8% of cases after 10-12 months post-AUPVP.

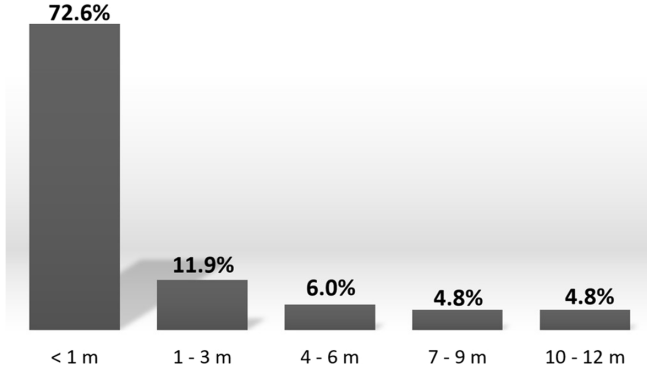

Figure 2. Timeline for the occurrence of BPPV following AUPVP. Most of the cases were detected within 1 month from the onset of the AUPVP, with a median of 7 days. BPPV, benign paroxysmal positional vertigo; AUPVP, acute unilateral peripheral vestibulopathy.

DISCUSSION

While BPPV is most commonly idiopathic, secondary causes of BPPV have been described and include head trauma, inner ear diseases, vestibular migraine, autoimmune or inflammatory disorders, etc.[13-15] The prevalence of secondary BPPV, however, is variable in the literature and has been described in 10%-30% of cases.[16,17] More specifically, BPPV secondary to AUPVP has been reported in 1.8%-22% (Table 3). Lindsay–Hemenway suggested a possible ischemic etiology in the territory of the anterior vestibular artery as a potential cause of AUPVP, with subsequent BPPV of the posterior SC. In their publication, they showed histopathologic findings of the temporal bone in one patient, 13 years after the initial acute vestibular syndrome. She died of an apparent myocardial infarction, and an autopsy was performed at that time. They described a "mass of convoluted vessels varying in size from one relatively large vessel to many very small vascular channels, many of which lay closely related to and within the vestibular nerve at the ganglion of Scarpa" within the internal auditory meatus.[1] However, the other cases presented in their series do not have any histopathological findings. Until there are more anatomopathological studies in patients with posterior SC BPPV secondary to AUPVP, it is not possible to conclude whether the cause is inflammatory or vascular.

Recently, evidence has shown that CRFs are potentially involved in sudden sensorineural hearing loss[10,11] and that sudden hearing loss could be a risk factor for developing a stroke.[9] A parallel could be

Table 3. Vestibular Neuritis as a Secondary Cause of BPPV

Study	BPPV — Secondary to VN	BPPV —Idiopathic	Prevalence	Time Between VN and BPPV	Onset of BPPV*	Vestibular Assessment	Findings
Türk et al 2021[19]	44	154	22.2%	<18 months	Early and late	Clinical ± calorics (n = 17/44)	Patients with BPPV secondary to VN were younger, involve posterior SC only, and require more treatments versus idiopathic BPPV
Lee et al 2015[17]	17	253	6.1%	-	Early	Clinical and calorics (>25% side difference)	VN cases were excluded from the study since they compared idiopathic BPPV versus secondary to SSNHL
Balatsouras et al 2014[18]	22	284	5.2%	22 days (average) Range, 3-75 days	Early	Clinical and calorics (>25% side difference)	Patients with BPPV secondary to VN: younger, involvement of the posterior SC only; poorer treatment results and higher rate of recurrence of BPPV
Kim et al 2011[25]	20	-	15.3%	40.6 ± 30.7 months (average)	?	Clinical and calorics (>25% side difference)	No details were provided about SC involved.
Mandalà et al 2010[23]	5	-	9.8%	Range 3 months to 6 years	Early and late	Clinical and calorics	All BPPV were posterior SC, in the same ear as had been affected by VN. BPPV developed within 3 months: 3/5, between 4 and 12 months: 1/5; between 2 and 6 years: 1/5
Lee et al 2010[16]	12	597	1.8%	17.9 days (average) Range 0-50 days	Early	Clinical and calorics (>25% side difference)	BPPV secondary to VN had the highest rate of posterior SC involvement (78.6%) versus other secondary causes and idiopathic BPPV
Huppert et al 2006[24]	14	-	14%	0 months-7 years	Early and late	Clinical and calorics (>25% side difference)	Study based on a questionnaire. BPPV developed within 3 months: 8/14, between 4 and 12 months: 2/14; between 3 and 7 years: 4/14

*Early onset: ≤3 months, late onset >3 months.
SC, semicircular canal; SSNHL, sensorineural hearing loss; VN, vestibular neuritis.

made with the labyrinth. If we assume the cause is vascular, one can rely on the presence of CRFs to suspect this etiology, although the current evidence is contradictory. In our study, we did not observe a significant difference in the prevalence of arterial hypertension, diabetes mellitus, dyslipidemia, prior history of stroke, TIA, or active smoking between patients with idiopathic BPPV and BPPV secondary to AUPVP.

In our cohort, we did not observe any significant differences with regard to sex or age between both groups. Benign paroxysmal positional vertigo, either idiopathic or secondary to AUPVP, tends to be more common in women and this finding is consistent with the literature.[18,19] For age, however, there are discrepancies. We did not observe any differences in age for both groups, yet Türk et al[18] and Balatsouras et al[19] reported that patients with BPPV secondary to AUPVP were younger. Nevertheless, our sample size for secondary BPPV is larger, and this discrepancy remains to be clarified in prospective studies.

The most common presentation of BPPV we observed was for the posterior SC, either idiopathic or following an event of AUPVP. Yet, cases of horizontal SC or multiple SC were also detected. These findings differ from those of Türk et al[18] and Balatsouras et al[19] who reported involvement of the posterior SC only in cases of BPPV secondary to AUPVP. Lee et al[16], on the other hand, have similar results to ours describing that 78.6% of their BPPV cases secondary to unilateral vestibulopathy (mostly vestibular neuritis; n = 12, and herpes zoster oticus; n = 2) were for the posterior SC, followed by the horizontal canal (14.3%). There is no definitive explanation as to why only the posterior canal would be affected, the horizontal canal macula may be partly damaged and may respond to the presence of otoconia as well.

Clinically, it is not possible to determine whether AUPVP is due to ischemia of the anterior vestibular artery or inflammation of the superior vestibular nerve. While it is known that AUPVP due to compromise of the superior vestibular nerve is the most frequent presentation, superior and inferior divisions of the nerve can be affected concomitantly in approximately 28.5%.[20] In this case, all vestibular end-organs can be affected in variable patterns, including the posterior SC and the saccule. We cannot eliminate the possibility that patients in our cohort had AUPVP involving both branches of the vestibular nerve, and we also do not have any ocular vestibular evoked myogenic potential testing for this group of patients to further assess utricular pathway involvement.[21]

We observed that BPPV appears soon after the onset of AUPVP, and most cases were detected within one month. Similar results have been described in the literature.[22-24] When seeing a patient with AUPVP, one must suspect BPPV[25] if patients report symptoms that are

triggered by changes in head or body position, especially within a month of onset. If patients report positional symptoms, the clinician must look for BPPV actively since patients with AUPVP tend to report worsening symptoms with rapid head movement, and hence, clinicians must be careful in taking the patients' history and performing the physical examination.

Limitations of our study include the fact that it is a retrospective study and hence has inherent limitations regarding the clinical data available. In addition, we did not evaluate other risk factors such as homocysteine, obesity, or the degree or type of dyslipidemia. We did not include patients with isolated inferior vestibular neuritis, and we did not have any data regarding vestibular evoked myogenic potential testing.

CONCLUSION

In conclusion, BPPV secondary to AUPVP is not uncommon and usually presents within one month of onset. We found no statistically significant differences in relation to CRFs or clinical characteristics between idiopathic BPPV and BPPV secondary to AUPVP. Acute unilateral peripheral vestibulopathy is a cause of secondary BPPV, and while an ischemic origin of the anterior vestibular artery is a possible cause, it should not be considered an independent etiology of BPPV. Until further research can differentiate a vascular from inflammatory etiology for AUPVP followed by BPPV, AUPVP should be considered a secondary cause of BPPV that can affect any SC, generally the posterior SC.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of the Faculty of Medicine of the Pontificia Universidad Católica de Chile (approval No: 200724005).

Informed Consent: N/A

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Author Contributions: Concept – S.W.; Design – S.W.; Supervision – S.W. Data Collection and/or Processing – S.W., J.B, J.I., V. S., F.G.H.; Analysis and/or Interpretation –S.W., V.S., F.G.H.; Literature Review – S.W., J.B., V.S.; Writing – S.W., V.S., J.B., J.I., F.G.H.; Critical Review – S.W., V.S., J.B., J.I., F.G.H.

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