Clinical Features of Secondary Benign Paroxysmal Positional Vertigo and the Impact of Psychological Factors on Residual Symptoms Following Repositioning: Postprint

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Abstract

Background Benign Paroxysmal Positional Vertigo (BPPV) is a common clinical disorder characterized by self-limiting episodes and recurrent attacks, which demonstrates favorable response to canalith repositioning procedures. However, residual symptoms may persist following successful reduction. While numerous studies have investigated factors influencing BPPV treatment outcomes in recent years, comparative analyses of treatment efficacy across specific etiologies and determinants of residual symptom development remain inadequately explored.

Objective To analyze the clinical characteristics of secondary BPPV, investigate the influence of etiological classification on disease recurrence rates, and identify factors affecting residual symptom development following BPPV reduction.

Methods A cohort of 340 patients diagnosed with BPPV who presented to the Vertigo Clinic of the Department of Otolaryngology-Head and Neck Surgery and the inpatient neurotology service of the First Hospital of Jilin University between April 2019 and April 2021 were enrolled, all of whom consented to undergo canalith repositioning procedures. Patients were stratified into primary BPPV (primary group) and secondary BPPV (secondary group) based on the presence or absence of underlying pathologies. The secondary group was further subdivided into five subgroups according to etiology: Sudden Sensorineural Hearing Loss (SSNHL), Vestibular Migraine (VM), Ménière's Disease (MD), Vestibular Neuritis (VN), and other causes (including craniocerebral and otologic surgery, Ramsay Hunt syndrome, temporal bone fracture, etc.). Clinical characteristics and prognostic outcomes were compared between the primary and secondary groups. Follow-up was conducted through three months post-initial reduction, after which patients were classified into residual symptom

and non-residual symptom groups based on the presence or absence of residual symptoms during the follow-up period. Inter-group differences in clinical characteristics and Self-Rating Anxiety Scale (SAS) and Self-Rating Depression Scale (SDS) scores at various time points (at diagnosis, 4 weeks post-treatment, and 3 months post-treatment) were evaluated, and independent predictors of residual symptom development following BPPV reduction were analyzed.

Results Among 340 patients, 184 (54.1%) comprised the primary group and 156 (45.9%) the secondary group. The secondary group exhibited younger age at onset, higher proportion requiring more than two reduction procedures, lower single-procedure success rate, elevated 3-month recurrence rate, increased incidence of residual symptoms, and higher SAS and SDS scores at diagnosis compared with the primary group ($P<0.05$). In secondary BPPV patients, age differed significantly across etiologies ($P<0.05$); patients with other etiologies (such as craniocerebral and otologic surgery, Ramsay Hunt syndrome, temporal bone fracture, etc.) were younger than those with SSNHL, VM, MD, and VN ($P<0.05$). No statistically significant differences were observed across etiologies within the secondary BPPV group regarding gender, affected semicircular canal, number of reduction procedures, single-procedure success rate, 3-month recurrence rate, residual symptom incidence, or SAS and SDS scores at various time points ($P>0.05$). At 3 months post-initial reduction, 133 patients exhibited residual symptoms while 207 did not. Residual symptoms predominantly manifested as head heaviness (59.4%, 79/133), unsteadiness (24.06%, 32/133), and head-neck discomfort (9.77%, 13/133), with 6.77% (9/133) experiencing two or more concurrent symptoms. The residual symptom group demonstrated higher proportions of secondary etiology and requirement for more than two reduction procedures compared with the non-residual symptom group ($P<0.05$); the residual symptom group exhibited lower proportion of concurrent disease recurrence and higher SAS and SDS scores at diagnosis, 4 weeks post-treatment, and 3 months post-treatment compared with the non-residual symptom group ($P<0.01$). Multivariate logistic regression analysis identified SAS score at diagnosis (OR=1.231, 95%CI=1.117~1.357, $P<0.001$) and SDS score at diagnosis (OR=1.209, 95%CI=1.113~1.314, $P<0.001$) as risk factors for residual symptom development following BPPV reduction.

Conclusion Secondary BPPV is characterized by higher incidence of residual symptoms and elevated recurrence rates following reduction, with anxiety and depression representing significant factors influencing residual symptom development.

Full Text

Clinical Characteristics of Secondary Benign Paroxysmal Positional Vertigo and the Influence of Psychological Factors on Residual Symptoms After Canalith Repositioning

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Abstract

Background: Benign paroxysmal positional vertigo (BPPV) is a common clinical disease characterized by self-limiting and recurrent episodes. Manual canalith repositioning procedures yield good therapeutic efficacy, though some patients experience residual symptoms post-treatment. While numerous studies have examined factors influencing BPPV treatment outcomes, comparisons between specific etiologies and factors affecting residual symptom development require further investigation.

Objective: To analyze the clinical features of secondary BPPV, investigate how etiological classification affects recurrence rates, and identify risk factors for residual symptoms following canalith repositioning.

Methods: A total of 340 patients diagnosed with BPPV at the Vertigo Clinic of the Department of Otorhinolaryngology Head and Neck Surgery and Inpatient Otology Department of the First Hospital of Jilin University between April 2019 and April 2021 were enrolled, all agreeing to undergo manual repositioning treatment. Patients were divided into primary BPPV (primary group) and secondary BPPV (secondary group) based on the presence or absence of underlying diseases. The secondary group was further subdivided into five subgroups according to etiology: sudden sensorineural hearing loss (SSNHL), vestibular migraine (VM), Ménière's disease (MD), vestibular neuritis (VN), and others (including cranial and ear surgery, Hunt syndrome, temporal bone fracture, etc.). Clinical features and prognosis were compared between the primary and secondary groups. Follow-up continued until three months after the initial repositioning, after which patients were classified into residual symptom and non-residual symptom groups based on symptom occurrence during follow-up. Differences in clinical characteristics and Self-rating Anxiety Scale (SAS) and Self-rating Depression Scale (SDS) scores at different time points (at diagnosis, 4 weeks post-treatment, and 3 months post-treatment) were compared between groups, and independent risk factors for residual symptoms were analyzed.

Results: Among 340 patients, 184 (54.1%) were in the primary group and 156 (45.9%) in the secondary group. Compared with the primary group, the secondary group had younger age at onset, higher proportion requiring more than two repositioning procedures, lower success rate after single repositioning, higher 3-month recurrence rate, higher incidence of residual symptoms, and higher SAS and SDS scores at diagnosis (all $P<0.05$). Among secondary BPPV patients, age differed significantly across etiologies ($P<0.05$), with patients in the "other causes" subgroup being younger than those with SSNHL, VM, MD, and VN ($P<0.05$). No significant differences were observed across secondary

BPPV etiologies in gender, semicircular canal involvement, number of repositioning procedures, single-procedure success rate, 3-month recurrence rate, residual symptom incidence, or SAS/SDS scores at any time point (P>0.05).

Three months after initial repositioning, 133 patients developed residual symptoms and 207 did not. Residual symptoms primarily manifested as head heaviness (59.4%, 79/133), instability (24.06%, 32/133), and head/neck discomfort (9.77%, 13/133), with 6.77% (9/133) experiencing two or more symptoms simultaneously. The residual symptom group had higher proportions of secondary etiology and patients requiring >2 repositioning procedures (P<0.05), lower proportion of disease recurrence (P<0.01), and higher SAS and SDS scores at diagnosis, 4 weeks, and 3 months compared with the non-residual symptom group (P<0.01). Multivariate logistic regression analysis revealed that SAS score at diagnosis (OR=1.231, 95%CI=1.117-1.357, P<0.001) and SDS score at diagnosis (OR=1.209, 95%CI=1.113-1.314, P<0.001) were independent risk factors for residual symptom development.

Conclusion: Secondary BPPV is characterized by higher incidence of residual symptoms and recurrence after canalith repositioning. Anxiety and depression significantly influence the occurrence of residual symptoms post-repositioning.

Keywords: Benign paroxysmal positional vertigo; Vestibular diseases; Residual symptoms; Psychological factors; Root cause analysis

Introduction

Benign paroxysmal positional vertigo (BPPV) is a common peripheral vestibular disorder in otology, with a lifetime prevalence of 2.4% [1]. Typical episodes involve brief vertigo, floating sensations, and instability triggered by head position changes relative to gravity, accompanied by characteristic nystagmus [2]. BPPV is classified as primary or secondary based on etiology. Primary BPPV, accounting for 50%-70% of cases, may be associated with degenerative changes in the otolithic organs. Secondary BPPV results from other inner ear or systemic conditions including sudden sensorineural hearing loss (SSNHL), vestibular migraine (VM), Ménière's disease (MD), vestibular neuritis (VN), surgery, head trauma, Hunt syndrome, and other disorders [3]. While most patients improve after canalith repositioning procedures, some develop residual symptoms (RS) manifesting as head heaviness, instability, head/neck discomfort, and anxiety about recurrent vertigo without typical positional vertigo or nystagmus [4].

Meta-analyses have identified female sex, hypertension, diabetes, hyperlipidemia, osteoporosis, and vitamin D deficiency as risk factors for BPPV recurrence [5]. This study collected data from BPPV patients at the First Hospital of Jilin University to further investigate the etiological classification of secondary BPPV, its impact on recurrence rates, and risk factors for residual symptoms after repositioning, aiming to improve precise diagnosis, treatment,

and prognostic analysis.

Methods

Study Design and Participants

This study enrolled 340 patients diagnosed with BPPV at the Vertigo Clinic of the Department of Otorhinolaryngology Head and Neck Surgery and Inpatient Otology Department of the First Hospital of Jilin University between April 2019 and April 2021. All patients agreed to undergo manual canalith repositioning, complete questionnaires, and participate in follow-up, and provided informed consent. Inclusion criteria: diagnosis of BPPV according to the 2017 BPPV Diagnosis and Treatment Guidelines [3]. All patients underwent detailed history taking regarding current symptoms and past medical history (including vertigo, dizziness, vestibular-visual symptoms, postural symptoms, autonomic symptoms, tinnitus, hearing loss, headache, photophobia, phonophobia, surgical history, and trauma). Secondary BPPV diagnosis was established based on diagnostic criteria for SSNHL, VM, MD, VN, Hunt syndrome, and other conditions. Exclusion criteria: microcirculation disorders due to hypertension, diabetes, or other factors; refusal or inability to cooperate with standardized repositioning maneuvers; severe systemic diseases; illiteracy or inability to complete scales or cooperate with follow-up. This study was approved by the Ethics Committee of the First Hospital of Jilin University (Approval No. 2021-527) and complied with the Declaration of Helsinki.

Patient Grouping

Patients were divided into primary BPPV (primary group) and secondary BPPV (secondary group) based on the presence or absence of underlying diseases. The secondary group was further subdivided into five subgroups by etiology: SSNHL, VM, MD, VN, and others (including cranial and ear surgery, Hunt syndrome, temporal bone fracture, etc.). Additionally, based on residual symptom occurrence during follow-up, patients were classified into residual symptom and non-residual symptom groups.

Treatment Protocol

All patients received standardized canalith repositioning procedures: the Epley maneuver for posterior canalithiasis, Semont liberation for posterior cupulolithiasis, Barbecue rotation for horizontal canalithiasis, Gufoni maneuver for horizontal cupulolithiasis, and modified Epley for superior canal BPPV. Secondary BPPV patients received concurrent treatment for their primary diseases according to relevant guidelines (e.g., SSNHL, MD). Patients with positive positional tests during follow-up underwent repeat repositioning, while those with residual symptoms received symptomatic treatment including medication and vestibular rehabilitation training. Patients with elevated SAS and SDS scores

received anti-anxiety/depression treatment in consultation with the Psychiatry Department.

Outcome Measures

Baseline indicators: Before treatment, patient age, sex, affected semicircular canal, presence of primary disease, and SAS/SDS scores were recorded. The SAS is a standard scale for assessing anxiety severity and treatment response (<50 normal, 50-60 mild anxiety, 61-70 moderate anxiety, >70 severe anxiety). The SDS reflects subjective depressive symptoms and treatment changes (<53 normal, 53-62 mild depression, 63-72 moderate depression, >72 severe depression) [6].

Follow-up indicators: All patients were followed up at 2 days, 4 weeks, and 3 months post-treatment regardless of recurrence or residual symptoms. Follow-up included symptom assessment, positional testing, and collection of SAS/SDS scores at 4 weeks and 3 months. Based on patient reports, positional test results, and nystagmus findings, we calculated: single-procedure success rate (proportion achieving symptom control without recurrence or residual symptoms after one maneuver), 3-month recurrence rate (proportion with recurrence during follow-up), number of repositioning procedures, residual symptom incidence (proportion developing residual symptoms during follow-up), timing of residual symptom onset, and predominant symptoms.

Statistical Analysis

Data were analyzed using SPSS 25.0 software. Normally distributed continuous variables were expressed as mean ± standard deviation ($\bar{x}\pm s$) and compared between two groups using independent samples t-tests; multiple groups were compared using one-way ANOVA. Categorical variables were expressed as percentages and compared using ^{2} tests or Fisher's exact test, with Bonferroni correction for multiple comparisons. Variables showing statistical and clinical significance in univariate analysis were entered as independent variables into a logistic regression model with residual symptom occurrence as the dependent variable. Stepwise regression using the likelihood ratio test was performed, calculating odds ratios (OR) and 95% confidence intervals (CI) to assess associations. $P<0.05$ was considered statistically significant.

Results

Comparison of Clinical Features Between Primary and Secondary Groups

Among 340 patients, 184 (54.1%) were in the primary group and 156 (45.9%) in the secondary group. The secondary group had younger age at onset, higher proportion requiring more than two repositioning procedures, lower single-procedure success rate, higher 3-month recurrence rate, higher residual

symptom incidence, and higher SAS and SDS scores at diagnosis compared with the primary group (all P<0.05). No significant differences were found between groups in sex, affected semicircular canal, or SAS/SDS scores at 4 weeks and 3 months (P>0.05) .

Comparison of Clinical Features by Etiology in Secondary BPPV

Among secondary BPPV patients, age differed significantly across etiologies (P<0.05), with patients in the "other causes" subgroup being younger than those with SSNHL, VM, MD, and VN (P<0.05). No significant differences were observed across etiological subgroups in sex, affected semicircular canal, number of repositioning procedures, single-procedure success rate, 3-month recurrence rate, residual symptom incidence, or SAS/SDS scores at any time point (P>0.05) .

Comparison of Clinical Features Between Residual Symptom and Non-Residual Symptom Groups

Three months after initial repositioning, 133 patients developed residual symptoms and 207 did not. Residual symptoms primarily manifested as head heaviness (59.4%, 79/133), instability (24.06%, 32/133), and head/neck discomfort (9.77%, 13/133), with 6.77% (9/133) experiencing two or more symptoms. The residual symptom group had higher proportions of secondary etiology and patients requiring >2 repositioning procedures (P<0.05), lower proportion of disease recurrence (P<0.01), and higher SAS and SDS scores at diagnosis, 4 weeks, and 3 months compared with the non-residual symptom group (P<0.01). No significant differences were found in age, sex, or affected semicircular canal between groups (P>0.05) .

Logistic Regression Analysis

Multivariate logistic regression analysis was performed with residual symptom occurrence as the dependent variable and five clinically significant indicators from univariate analysis as independent variables (see variable assignment in). Results showed that SAS score at diagnosis (OR=1.231, 95%CI=1.117-1.357, P<0.001) and SDS score at diagnosis (OR=1.209, 95%CI=1.113-1.314, P<0.001) were independent risk factors for residual symptom development after BPPV repositioning .

Discussion

The precise pathogenesis of BPPV remains unclear. The widely accepted mechanisms involve dislodged otoconial debris from the utricle entering the semicircular canals (canalithiasis) or adhering to the cupula (cupulolithiasis), with head movements causing endolymph flow that alters vestibular hair cell firing rates and produces vertigo [7]. This requires both otoconial detachment and intact or partially intact semicircular canal function. Elderly women often develop

BPPV due to vitamin D deficiency [8], altered sex hormone levels, and calcium metabolism disturbances affecting otoconial production and absorption, with the posterior canal being most commonly affected due to its dependent anatomical position [9], consistent with our findings.

Canalith repositioning maneuvers represent the most effective BPPV treatment, providing rapid symptom relief. One domestic study reported a 79% single-procedure efficacy [10], whereas our secondary group showed only 42.9% success, with most requiring multiple procedures and showing higher recurrence rates. This may relate to inadequate control of primary diseases, severe inner ear dysfunction, or pronounced inflammatory responses, with potential for re-detachment of otoconial fragments post-repositioning. Studies demonstrate that anxiety, depression, and fear significantly reduce repositioning efficacy [11], and our results show most secondary group patients had mild depression at diagnosis. The mechanism may involve impaired neuroinflammatory and endocrine responses from negative emotions, with similar inflammatory pathways [e.g., interleukin (IL)-1β] triggering recurrent vascular spasm and otoconial displacement [12]. Stress from anxiety and depression may also activate the hypothalamic-pituitary-adrenal axis dysfunction [13] or cause 5-hydroxytryptamine (5-HT) dysfunction. The vestibular nuclear complex contains numerous 5-HT receptors, and 5-HT deficiency may significantly impact neuronal electrophysiology and vestibular nuclear function [14]. Secondary BPPV patients with underlying inner ear disease have lower overall antioxidant capacity and poorer tolerance to stress responses, predisposing to BPPV occurrence and recurrence.

Clinically, 5%-19% of SSNHL patients develop BPPV during their disease course. LEE et al. [15] found BPPV can occur within 24 hours of SSNHL onset, with approximately 83.3% occurring simultaneously. BPPV primarily occurs in SSNHL patients with profound hearing loss, usually ipsilaterally. WU et al. [16] proposed that inner ear circulatory disturbance or viral infection in SSNHL patients impairs vestibular organ blood supply, disrupting otoconial metabolism and causing detachment. VM commonly presents with spontaneous and positional vertigo accompanied by positional nystagmus [17]. SUGAYA et al. [18] suggested secondary BPPV in VM results from migraine-induced labyrinthine artery spasm causing utricular and saccular dysfunction and otoconial detachment. VM patients often experience anxiety and insomnia, which may indirectly contribute to secondary BPPV. OUYANG et al. [19] found these patients often involve the horizontal canal, whereas our study showed posterior canal predominance, possibly due to our smaller sample size lacking representativeness.

MD with secondary BPPV is not uncommon. The mechanism may involve endolymphatic hydrops disrupting inner ear blood supply, causing fibrosis of the utricular and saccular maculae and promoting otoconial detachment [20]. Detached otoconial particles may also affect longitudinal endolymph flow and absorption, worsening hydrops [21], creating a vicious cycle of recurrent attacks. VN often relates to viral infection; anatomically, involvement of the utricular

branch of the vestibular nerve can impair utricular function and promote otoconial detachment. Superior vestibular nerve injury causes horizontal semicircular canal dysfunction and reduced gain, potentially preventing typical positional vertigo when otoconia enter the horizontal canal due to functional loss, explaining why VN-related BPPV commonly affects the posterior canal. Secondary BPPV etiologies are diverse; current evidence suggests post-maxillofacial surgery BPPV relates to mechanical forces, intraoperative positioning, and inner ear blood supply disruption [22]; Hunt syndrome involves varicella-zoster virus affecting vestibular and geniculate ganglia, damaging vestibular organs and the utricle; head trauma can damage otoconial and otolithic membrane proteins, causing utricular detachment [23]. These patients are younger than other etiological subgroups, and their brief vertigo episodes may be overlooked, leading to missed diagnosis.

Recent studies show 13%-61% of patients develop head heaviness, instability, head/neck discomfort, and other symptoms with negative positional tests and nystagmus after successful repositioning, i.e., residual symptoms [24]. Mechanisms may include incomplete repositioning with residual subthreshold otoconial fragments [25], delayed vestibular function recovery with inadequate central compensation [26], or vestibular nuclear degeneration reducing neural signal conduction [27]. Secondary BPPV involving inner ear pathology can affect the superior vestibular nerve and labyrinthine artery, causing severe vestibular dysfunction and higher residual symptom probability. Multiple repositioning procedures may constitute repeated vestibular organ injury, increasing damage and residual symptom risk [28], supporting our findings. Our study also examined psychosocial factors, revealing higher SAS and SDS scores at diagnosis and all follow-up time points in patients with residual symptoms, with diagnostic SAS and SDS scores being independent risk factors. The mechanism warrants further investigation but may involve inflammatory responses, hypothalamic-pituitary-adrenal axis dysfunction, and dizziness-induced circuit disturbances in hippocampal and amygdala pathways affecting neurotransmitter release and reuptake, exacerbating anxiety and depression. Although secondary etiology showed statistical significance in univariate analysis for residual symptoms, it was excluded from the regression model, suggesting its effect was attenuated or replaced by other factors, requiring validation after controlling for confounders. Only two patients experienced both recurrence and residual symptoms during follow-up, possibly because recurrent BPPV with positive positional tests and nystagmus may obscure residual symptom diagnosis. Studies indicate that multiple repositioning procedures, older age, and longer disease duration are independent recurrence risk factors [29], with recurrence and residual symptoms having similar clinical presentations but different management approaches, necessitating careful differentiation to avoid missed diagnosis and ensure precise treatment.

Limitations: This study focused on psychological factors affecting residual symptom development; future research should incorporate multiple variables while controlling for confounders.

References

[1] KIM H J, PARK J, KIM J S. Update on benign paroxysmal positional vertigo[J]. J Neurol, 2021, 268(5): 1995-2000. DOI: 10.1007/s00415-020-10314-7.

[2] BHATTACHARYYA N, BAUGH R F, ORVIDAS L, et al. Clinical practice guideline: benign paroxysmal positional vertigo[J]. Otolaryngol Head Neck Surg, 2008, 139(5 suppl 4): S47-81. DOI: 10.1016/j.otohns.2008.08.022.

[3] KONG Weijia. Guidelines for diagnosis and treatment of benign paroxysmal positional vertigo[J]. Chin J Otorhinolaryngol Head Neck Surg, 2017, 52(3): 173-177.

[4] SIM E, TAN D, HILL K. Poor treatment outcomes following repositioning maneuvers in younger and older adults with benign paroxysmal positional Vertigo: a systematic review and meta-analysis[J]. J Am Med Dir Assoc, 2019, 20(2): 224.e1-224.e23. DOI: 10.1016/j.jamda.2018.11.019.

[5] LI S C, WANG Z J, LIU Y, et al. Risk factors for the recurrence of benign paroxysmal positional Vertigo: a systematic review and meta-analysis[J]. Ear Nose Throat J, 2022, 101(3): NP112-NP134. DOI: 10.1177/0145561320943362.

[6] DUAN Quanquan, SHENG Li. Clinical validity of Self-rating Anxiety Scale and Self-rating Depression Scale[J]. Chin J Ment Health, 2012, 26(9): 676-679. DOI: 10.3969/j.issn.1000-6729.2012.09.007.

[7] YETISER S. Review of the pathology underlying benign paroxysmal positional vertigo[J]. J Int Med Res, 2020, 48(4): 300060519892370. DOI: 10.1177/0300060519892370.

[8] YANG B Y, LU Y X, XING D M, et al. Association between serum vitamin D levels and benign paroxysmal positional vertigo: a systematic review and meta-analysis of observational studies[J]. Eur Arch Otorhinolaryngol, 2020, 277(1): 169-177. DOI: 10.1007/s00405-019-05694-0.

[9] XUE Yiwen, ZHOU Bin, YU Cheng, et al. Risk factors for recurrence of benign paroxysmal positional vertigo in the elderly[J]. Chin J Gerontol, 2020, 40(15): 3261-3264. DOI: 10.3969/j.issn.1005-9202.2020.15.041.

[10] LOU Y, CAI M, XU L G, et al. Efficacy of BPPV diagnosis and treatment system for benign paroxysmal positional vertigo[J]. Am J Otolaryngol, 2020, 41(3): 102412. DOI: 10.1016/j.amjoto.2020.102412.

[11] WEI W, SAYYID Z N, MA X L, et al. Presence of anxiety and depression symptoms affects the first time treatment efficacy and recurrence of benign paroxysmal positional Vertigo[J]. Front Neurol, 2018, 9: 178. DOI: 10.3389/fneur.2018.00178.

[12] GÜÇLÜTÜRK M T, ÜNAL Z N, İSMI O, et al. The role of oxidative stress and inflammatory mediators in benign paroxysmal positional Vertigo[J]. J Int

Adv Otol, 2016, 12(1): 101-105. DOI: 10.5152/iao.2015.1412.

[13] FUCHS E, FLÜGGE G. Chronic social stress: effects on limbic brain structures[J]. Physiol Behav, 2003, 79(3): 417-427. DOI: 10.1016/s0031-9384(03)00161-6.

[14] SMITH P F, DARLINGTON C L. A possible explanation for dizziness following SSRI discontinuation[J]. Acta Otolaryngol, 2010, 130(9): 981-983. DOI: 10.3109/00016481003602082.

[15] LEE N H, BAN J H. Is BPPV a prognostic factor in idiopathic sudden sensory hearing loss?[J]. Clin Exp Otorhinolaryngol, 2010, 3(4): 199-202. DOI: 10.3342/ceo.2010.3.4.199.

[16] WU Ziming, ZHANG Suzhen, ZHOU Na, et al. Analysis of utricular and saccular function in sudden sensorineural hearing loss patients with vertigo[J]. J Audiol Speech Pathol, 2005, 13(6): 397-399. DOI: 10.3969/j.issn.1006-7299.2005.06.005.

[17] KIM S K, HONG S M, PARK I S, et al. Association between migraine and benign paroxysmal positional Vertigo among adults in South Korea[J]. JAMA Otolaryngol Head Neck Surg, 2019, 145(4): 307-312. DOI: 10.1001/jamaoto.2018.4016.

[18] SUGAYA N, ARAI M, GOTO F. Is the headache in patients with vestibular migraine attenuated by vestibular rehabilitation?[J]. Front Neurol, 2017, 8: 124. DOI: 10.3389/fneur.2017.00124.

[19] OUYANG Tangpeng, XU Xianrong, ZHAI Lihong, et al. Clinical characteristics of vestibular migraine complicated with benign paroxysmal positional vertigo[J]. J Audiol Speech Pathol, 2021, 29(6): 604-608. DOI: 10.3969/j.issn.1006-7299.06.002.

[20] KUTLUBAEV M A, XU Y, HORNIBROOK J. Benign paroxysmal positional vertigo in Meniere's disease: systematic review and meta-analysis of frequency and clinical characteristics[J]. J Neurol, 2021, 268(5): 1608-1614. DOI: 10.1007/s00415-019-09502-x.

[21] WANG Jinchao, YANG Li, ZHANG Jin, et al. Ocular vestibular evoked myogenic potentials in Ménière's disease with benign paroxysmal positional vertigo[J]. J Clin Otorhinolaryngol Head Neck Surg, 2021, 35(2): 120-124, 130. DOI: 10.13201/j.issn.2096-7993.2021.02.007.

[22] SHU Fan, ZHANG Hongzheng, CAI Jieqing, et al. Clinical characteristics and efficacy of secondary benign paroxysmal positional vertigo after middle ear surgery[J]. J Clin Otorhinolaryngol Head Neck Surg, 2020, 34(1): 79-82. DOI: 10.13201/j.issn.1001-1781.2020.01.019.

[23] YANG C J, LEE J W, KIM S J, et al. Development of a murine model of traumatic benign paroxysmal positional vertigo: a preliminary study[J]. Acta Otolaryngol, 2017, 137(1): 29-34. DOI: 10.1080/00016489.2016.1217043.

[24] TIRELLI G, NICASTRO L, GATTO A, et al. Repeated canalith repositioning procedure in BPPV: effects on recurrence and dizziness prevention[J]. Am J Otolaryngol, 2017, 38(1): 38-43. DOI: 10.1016/j.amjoto.2016.09.009.

[25] DISPENZA F, MAZZUCCO W, MAZZOLA S, et al. Observational study on risk factors determining residual dizziness after successful benign paroxysmal positional vertigo treatment: the role of subclinical BPPV[J]. Acta Otorhinolaryngol Ital, 2019, 39(5): 347-352. DOI: 10.14639/0392-100X-2247.

[26] FARALLI M, LAPENNA R, GIOMMETTI G, et al. Residual dizziness after the first BPPV episode: role of otolithic function and of a delayed diagnosis[J]. Eur Arch Otorhinolaryngol, 2016, 273(10): 3157-3165. DOI: 10.1007/s00405-016-3947-z.

[27] GIOMMETTI G, LAPENNA R, PANICHI R, et al. Residual dizziness after successful repositioning maneuver for idiopathic benign paroxysmal positional Vertigo: a review[J]. Audiol Res, 2017, 7(1): 178. DOI: 10.4081/audiores.2017.178.

[28] D'SILVA L J, STAECKER H, LIN J, et al. Retrospective data suggests that the higher prevalence of benign paroxysmal positional vertigo in individuals with type 2 diabetes is mediated by hypertension[J]. J Vestib Res, 2016, 25(5/6): 233-239. DOI: 10.3233/VES-150563.

[29] CHEN J B, ZHANG S L, CUI K, et al. Risk factors for benign paroxysmal positional vertigo recurrence: a systematic review and meta-analysis[J]. J Neurol, 2021, 268(11): 4117-4127. DOI: 10.1007/s00415-020-10175-0.

Author Contributions: ZHOU Xinyang conceptualized the study, performed formal analysis, and wrote the original draft; YU Shujian managed data, handled project administration, and provided resources; WANG Qian conducted investigation, developed methodology, and created software tables; YU Hong supervised, validated, and reviewed the manuscript.

Conflict of Interest: The authors declare no conflict of interest.

Note: Figure translations are in progress. See original paper for figures.