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Original Article

Risk factors for persistent postural-perceptual dizziness after successful manual repositioning in patients with benign paroxysmal positional vertigo: A nomogram model

Lanlan Li[1], Huijun Yu[2], Changguo Zhang[3]

ABSTRACT

Objectives: To identify risk factors for persistent postural-perceptual dizziness (PPPD) within one year after successful manual repositioning in patients with benign paroxysmal positional vertigo (BPPV) and to develop a nomogram model.

Methodology: Clinical records of 140 patients diagnosed with BPPV and achieving successful manual repositioning in the Department of Neurology, Huzhou Third Municipal Hospital from January 2022 to December 2024 were retrospectively reviewed and analyzed. According to the occurrence of PPPD during the one-year follow-up, patients were divided into the PPPD group (n=40) and the non-PPPD group (n=100). Univariate analysis and multivariate logistic regression were used to identify risk factors for PPPD after successful manual repositioning of BPPV. The nomogram model was constructed using the "rms" package of R software (version 4.0.1).

Results: Disease duration ≥ 7 days (OR = 3.596, 95% CI: 1.275–10.142; P = 0.016), residual dizziness (OR = 2.687, 95% CI: 1.056–6.840; P = 0.038), and anxiety (OR = 2.872, 95% CI: 1.096–7.528; P = 0.032) were independent risk factors for PPPD within one year after successful manual repositioning of BPPV, whereas standardized vestibular rehabilitation training was an independent protective factor (OR = 0.148, 95% CI: 0.044–0.492; P = 0.002). The nomogram yielded an area under the receiver operating characteristic curve of 0.827 (95% CI: 0.747–0.907) and a bootstrap-corrected C-index of 0.813 (95% CI: 0.730–0.896). The calibration curve showed good agreement between the predicted and observed probabilities, and decision curve analysis indicated a potential net benefit across threshold probabilities ranging from 0.09 to 0.83.

Conclusion: Long disease duration (≥ 7 days), residual dizziness, and anxiety were independently associated with higher odds of PPPD within one year after successful manual repositioning of BPPV, whereas standardized vestibular rehabilitation training was independently associated with lower odds. The constructed nomogram may assist in preliminary risk stratification, but further external validation is required.

KEYWORDS: Benign Paroxysmal Positional Vertigo, Risk Factors, Manual Repositioning, Nomogram, Persistent Postural-Perceptual Dizziness.

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INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most common peripheral vestibular disorder in clinical practice, characterized by transient vertigo and characteristic nystagmus induced by changes in head position.[1] BPPV accounts for up to 34.22% of vertigo-related diseases, and its incidence increases with age.[2] Manual repositioning, the current first-line treatment for BPPV, has the advantages of simple operation and definite efficacy, and can rapidly relieve vertigo symptoms, with a success rate of 80%–90%.[3] However, approximately 7%–50% of patients still experience non-rotatory dizziness, unsteady gait, a floating sensation, and other discomforts after treatment.[4,5] Some of these patients have persistent symptoms for more than three months, meeting the diagnostic criteria for persistent postural-perceptual dizziness (PPPD).[4–6]

PPPD is a functional vestibular disorder characterized by persistent non-rotatory dizziness, postural instability, and hypersensitivity to visual stimuli, often secondary to vestibular lesions.[6,7] Patients frequently experience limited activity and an increased risk of falls due to persistent symptoms, which severely impair quality of life and even lead to psychological problems such as anxiety and depression.[8] At present, studies on the independent factors influencing PPPD after repositioning are limited. Therefore, it is necessary to establish a reliable predictive tool for early identification of high-risk patients and targeted intervention.[8–10] This study aimed to retrospectively analyze clinical data from patients with successful manual repositioning for BPPV, identify independent predictors of PPPD, and develop a nomogram prediction model. The results may help clarify the pathogenesis of PPPD after BPPV repositioning, provide a scientific basis for the early clinical identification of high-risk populations and for individualized prevention and intervention strategies, reduce the incidence of PPPD, and improve patient prognosis.

METHODOLOGY

Clinical records and available routine follow-up data of 140 patients diagnosed with BPPV who achieved successful manual repositioning in the Department of Neurology, Huzhou Third Municipal Hospital, between January 2022 and December 2024 were retrospectively reviewed. Based on the occurrence of PPPD during the one-year follow-up period, patients were divided into the PPPD group (n = 40) and the non-PPPD group (n = 100).

Ethical Approval: The ethics committee of our hospital approved this study with the number: 2026-033, Date: February 12th 2026.

Diagnostic criteria for BPPV: Patients met the diagnostic criteria for benign paroxysmal positional vertigo in the Guidelines for the Diagnosis and Treatment of Benign Paroxysmal Positional Vertigo (2017) issued by the Otorhinolaryngology Head and Neck Surgery Branch of the Chinese Medical Association:[11]

- Recurrent transient vertigo or dizziness induced by changes in head position relative to gravity (usually lasting no more than one minute).
- Vertigo and characteristic positional nystagmus during positional testing.
- Exclusion of other diseases, such as vestibular migraine, vestibular paroxysmia, central positional vertigo, Ménière's disease, vestibular neuritis, labyrinthitis, superior semicircular canal dehiscence syndrome, posterior circulation ischemia, orthostatic hypotension, psychogenic vertigo, etc.

Diagnostic criteria for PPPD: Patients with PPPD met the Diagnostic Criteria for Persistent Postural-Perceptual Dizziness (PPPD): Consensus of the Vestibular Disorders Classification Committee of the Bárány Society (2020),[12] requiring satisfaction of all five criteria below:

Criterion A: One or more symptoms of dizziness, unsteadiness, or non-rotatory vertigo are present on most days for ≥ three months.

- Symptoms are prolonged, often lasting for hours, but the severity may fluctuate.
- Symptoms do not need to be present continuously for 24 hours per day.

Criterion B: The persistent vestibular symptoms arise without a clear trigger but may be exacerbated by three factors:

- Upright posture.
- Active or passive movement, regardless of direction or position.
- Exposure to moving visual stimuli or complex visual environments.

Criterion C: Symptoms are usually triggered by conditions that cause dizziness/vertigo or balance disturbance, including acute/paroxysmal/chronic vestibular syndromes, other neurological or medical disorders, and psychiatric diseases. When PPPD is precipitated by an acute or episodic disorder, dizziness, unsteadiness, or non-spinning vertigo persists after the precipitating condition has resolved and subsequently meets the symptom pattern and duration specified in Criterion A. Initially, symptoms may be intermittent and then gradually become persistent. When the trigger is a chronic syndrome, symptoms have an insidious onset and progressive worsening.

Criterion D: Symptoms cause significant distress or functional impairment.

Criterion E: Symptoms are not better explained by another disorder.

Inclusion criteria:

- Met the diagnostic criteria for BPPV.
- Achieved successful manual repositioning: complete disappearance of positional nystagmus and vertigo within one week after treatment with the Epley maneuver, Semont maneuver, or Barbecue roll maneuver according to BPPV subtype.
- Aged 18–80 years.
- Had sufficient clinical and follow-up information available for outcome assessment.

Exclusion criteria:

- Positive neurological localizing signs.
- A history of head trauma, or diagnosed with other vertigo-related diseases including sudden deafness, vestibular migraine, Ménière's disease, vestibular neuritis, vestibular paroxysmia, central vertigo, etc.
- Incomplete medical or follow-up records that precluded determination of the study variables or one-year PPPD outcome.

Data Collection: The following variables were retrospectively extracted from existing medical records and routine follow-up documentation: demographic characteristics, including age and sex;

Clinical characteristics: disease duration (time from first onset of BPPV symptoms to successful repositioning, categorized as < 7 days and ≥ 7 days), BPPV subtype (posterior semicircular canal / horizontal semicircular canal), presence or absence of residual dizziness (defined as non-rotatory dizziness, lightheadedness,

or unsteadiness reported by patients one week after successful manual repositioning, with resolution of positional nystagmus; categorized as yes/no), and comorbidities (including hypertension, diabetes mellitus, and osteoporosis);

Psychological status: HAM-A scores documented as part of routine clinical care within one week after successful manual repositioning were retrospectively extracted from the medical records. A total HAM-A score ≥ 8 was used to define the presence of anxiety symptoms.

Vestibular rehabilitation training: Information regarding vestibular rehabilitation was retrospectively extracted from medical records and routine follow-up documentation. The rehabilitation program documented in routine clinical care included gaze stability training (VOR × 1), balance training (single-leg standing for 30 s per set, three sets), and habituation training (Brandt-Daroff exercises, 10 times per set, two sets). The recommended training schedule was approximately 30 minutes per session, three times

Table-I: Comparison of general information between the two groups.

Variables	*Non-PPPD group (n=100)*	*PPPD group (n=40)*	Z/χ^2	*P*
Age (years)	61.5 (55, 68)	66.5 (56.8, 71.3)	−1.509	0.131
Gender			0.187	0.666
Male	24 (24.0)	11 (27.5)		
Female	76 (76.0)	29 (72.5)		
Disease duration ≥ 7 days			15.341	<0.001
Yes	49 (49.0)	34 (85.0)		
No	51 (51.0)	6 (15.0)		
BPPV classification			0.006	0.936
Posterior semicircular canal	87 (87.0)	35 (87.5)		
Horizontal semicircular canal	13 (13.0)	5 (12.5)		
Hypertension			0.688	0.407
Yes	35 (35.0)	17 (42.5)		
No	65 (65.0)	23 (57.5)		
Diabetes			0.737	0.391
Yes	12 (12.0)	7 (17.5)		
No	88 (88.0)	33 (82.5)		
Residual dizziness			14.795	<0.001
Yes	18 (18.0)	20 (50.0)		
No	82 (82.0)	20 (50.0)		
Osteoporosis			0.000	1.000
Yes	5 (5.0)	2 (5.0)		
No	95 (95.0)	38 (95.0)		
Anxiety			15.120	<0.001
Yes	16 (16.0)	19 (47.5)		
No	84 (84.0)	21 (52.5)		
Vestibular rehabilitation training			27.451	<0.001
Yes	95 (95.0)	24 (60.0)		
No	5 (5.0)	16 (40.0)		

per week for four consecutive weeks. Adherence was determined from available outpatient or telephone follow-up documentation. An actual completion rate ≥ 80% was classified as standardized vestibular rehabilitation training; otherwise, training was classified as non-standardized.

Follow-up and Outcome Assessment: One-year outcome data after successful manual repositioning were retrospectively obtained from available routine outpatient and telephone follow-up records. Information regarding persistent dizziness, unsteadiness, non-spinning vertigo, and completion of vestibular rehabilitation training was extracted from these records. The occurrence of PPPD within one year was determined retrospectively based on the available follow-up documentation and the Bárány Society diagnostic criteria described above. Patients who met the diagnostic criteria for PPPD within the one-year follow-up period were classified into the PPPD group, whereas the remaining patients were classified into the non-PPPD group.

Statistical Analysis: Data were analyzed using SPSS version 26.0. The normality of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables with non-normal distributions were presented as median (interquartile range) and compared between groups using the Mann–Whitney U test. Categorical variables were presented as n (%) and compared between groups using the chi-square test or Fisher's exact test, as appropriate. Variables with $P < 0.05$ in univariate analysis were included in a multivariate binary Logistic regression (forward: LR method) to identify independent predictors of PPPD, and the results were expressed as odds ratios (ORs) with 95% confidence intervals (95% CIs). Before multivariable analysis, multicollinearity among the candidate predictors was assessed using tolerance values and variance inflation factors (VIFs). A tolerance value >0.20 and a VIF <5 were considered indicative of the absence of substantial multicollinearity. $P<0.05$ indicated a statistically significant difference. The nomogram was developed using R (version 4.0.1) and the "rms" package. The concordance index (C-index) was calculated using the "rms" package, and calibration and receiver operating characteristic (ROC) curves were plotted to evaluate the model's predictive performance. Internal validation was performed using 1,000 bootstrap resamples, and the bootstrap-corrected C-index with its 95% confidence interval was calculated. Decision curve analysis was performed to assess the potential clinical utility of the nomogram by estimating its net benefit across a range of threshold probabilities and comparing it with the treat-all and treat-none strategies.

Supplementary Table-S1. Assessment of multicollinearity among variables included in the multivariable logistic regression model.

Predictor	*Tolerance*	*VIF*
Disease duration ≥ 7 days	0.905	1.104
Residual dizziness	0.892	1.122
Anxiety	0.901	1.110
Vestibular rehabilitation training	0.853	1.172

Note: Multicollinearity among the candidate predictors was assessed using tolerance values and variance inflation factors (VIFs). A tolerance value >0.20 and a VIF <5 were considered indicative of the absence of substantial multicollinearity.

RESULTS

One hundred forty patients were included in the analysis, of whom 40 developed PPPD within one year after successful manual repositioning.Table-I. The one-year incidence proportion of PPPD was therefore 28.6% (40/140 × 100%). The proportions of patients with disease duration ≥ 7 days, residual dizziness, and anxiety in the PPPD group were significantly higher than those in the non-PPPD group ($P < 0.05$). There were no significant differences in age, gender, BPPV classification, hypertension, diabetes, or osteoporosis between the two groups ($P > 0.05$).

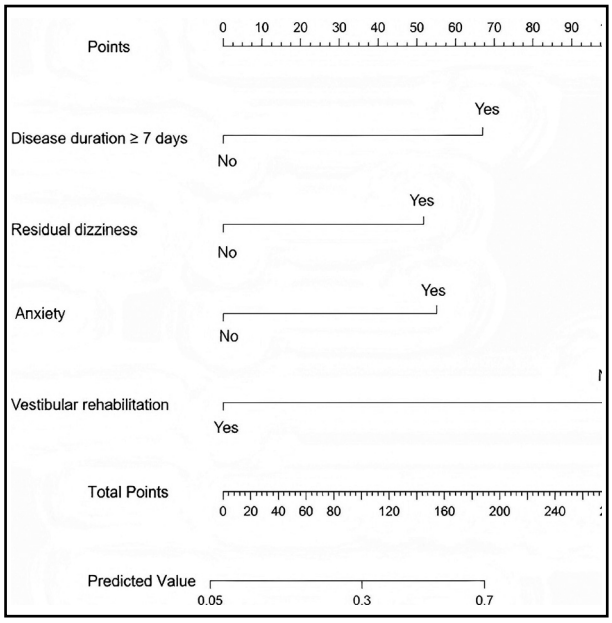

Fig.1: Nomogram for predicting PPPD after manual repositioning in BPPV patients. Each level of the predictive variable corresponds to a specific score. The total score was generated by summing the scores of each predictive variable. The total score corresponds to the probability of PPPD occurrence after manual repositioning for BPPV.

Table-II: Multivariate logistic regression analysis of PPPD after successful manual repositioning for BPPV.

Independent variables	*B*	*OR (95% CI)*	*P*
Disease duration ≥ 7 days	1.280	3.596 (1.275-10.142)	0.016
Residual dizziness	0.988	2.687 (1.056-6.840)	0.038
Anxiety	1.055	2.872 (1.096-7.528)	0.032
Vestibular rehabilitation	-1.910	0.148 (0.044-0.492)	0.002

Taking the occurrence of PPPD as the dependent variable (occurrence = 1, nonoccurrence = 0), variables with statistical significance in Table-I were included as independent variables in multivariate logistic regression analysis: disease duration ≥ 7 days (yes = 1, no = 0), residual dizziness (yes = 1, no = 0), anxiety (yes = 1, no = 0), and vestibular rehabilitation training (yes = 1, no = 0). Multicollinearity assessment showed that tolerance values ranged from 0.853 to 0.905 and VIFs ranged from 1.104 to 1.172, indicating no substantial multicollinearity among the variables included in the multivariable model (Supplementary Table-S1). The results showed that disease duration ≥ 7 days (OR = 3.596, 95%CI: 1.275 – 10.142), residual dizziness (OR = 2.687, 95%CI: 1.056 – 6.840), and anxiety (OR = 2.872, 95%CI: 1.096 – 7.528) were independent risk factors for PPPD, whereas vestibular rehabilitation training (OR = 0.148, 95% CI: 0.044 – 0.492) was a protective factor (all P < 0.05) (Table-II).

Independent predictors, including disease duration ≥ 7 days, residual dizziness, anxiety, and vestibular rehabilitation training, were incorporated to construct the nomogram model. The total score was calculated by summing the points assigned to each variable, and the corresponding probability of PPPD after successful manual repositioning for BPPV was determined based on the total score (Fig.1).

The discriminative performance of the nomogram for predicting PPPD within one year after successful manual repositioning in patients with BPPV was evaluated using receiver operating characteristic curve analysis. The area under the ROC curve (AUC) was 0.827 (95%CI: 0.747 – 0.907), with a sensitivity of 55.0%, specificity of 95.0%, positive predictive value (PPV) of 81.5%, and negative predictive value (NPV) of 84.1%, indicating favorable predictive efficacy (Fig.2). The model was subjected to 1000 bootstrap resamples for internal validation. The concordance index (C-index) was 0.813 (95% CI: 0.730–0.896), indicating excellent discrimination and accuracy in predicting PPPD. A calibration curve was plotted (Fig.3), which was close to the ideal curve, demonstrating good consistency of the model.

Clinical decision curve analysis (DCA) was performed based on the prediction model. DCA provided insight into the range of predicted risks for PPPD occurrence after manual repositioning in BPPV patients. The results indicated that the risk prediction model for PPPD after manual repositioning in BPPV patients achieved a favorable net benefit within the threshold probability range of 0.09 – 0.83 (Fig.4).

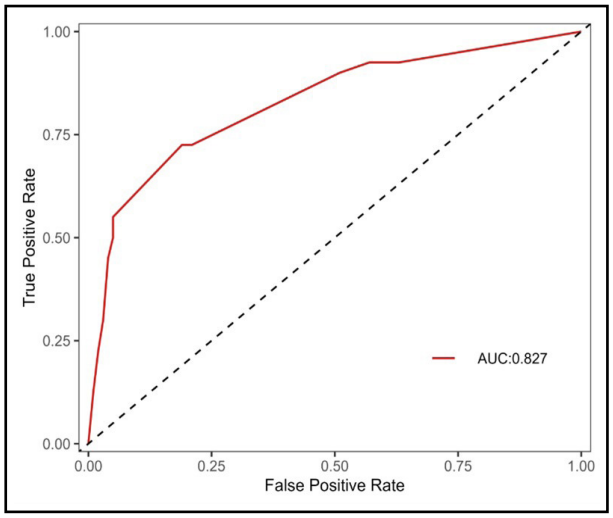

Fig.2: ROC curve and AUC of the predictive model.
ROC: Receiver operating characteristic;
AUC: Area under the curve

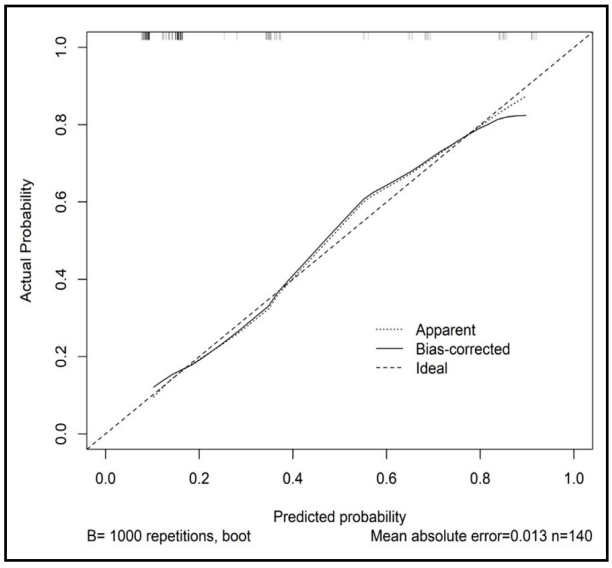

Fig.3: Calibration curve of the prediction model.

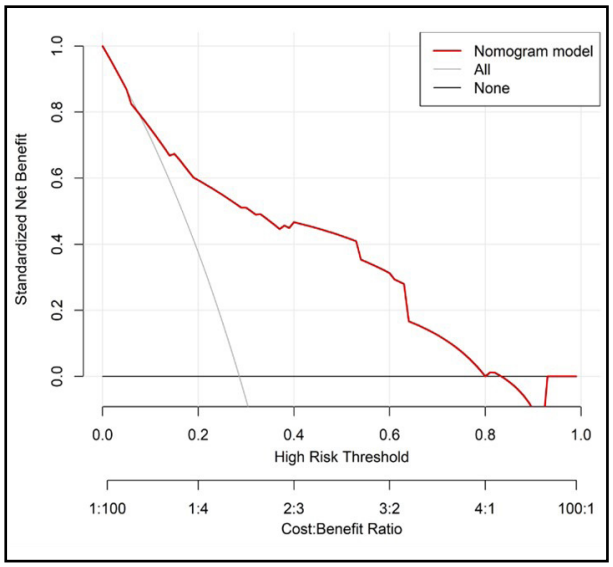

Fig.4: DCA of the nomogram model.
DCA: decision curve analysis.

DISCUSSION

This study identified long disease duration (≥ 7 days), residual dizziness, and anxiety as independent risk factors for PPPD within one year after successful manual repositioning in patients with BPPV. Standardized vestibular rehabilitation training was independently associated with lower odds of PPPD. The constructed nomogram showed potentially useful discrimination for preliminary risk stratification.

In this retrospective cohort of 140 patients who achieved successful manual repositioning for BPPV, 40 patients developed PPPD during the one-year follow-up, corresponding to a one-year incidence proportion of 28.6%. This proportion was within the broad range of persistent symptoms reported in previous studies.[5,13] Notably, the incidence in this cohort was slightly higher than the 3.9%–22.4% reported by Gambacorta et al.[14] Possible reasons for this difference include the study's one-year follow-up period, which may better capture delayed-onset cases than some cross-sectional studies. As a regional neurological referral center, our institution admits patients with relatively long disease duration and greater disease complexity, which may lead to an enrichment of high-risk populations. A study by Acle-Cervera et al. also noted that BPPV should not be regarded as a completely cured condition after successful repositioning, and that more than 70% of patients still had various vestibular symptoms after discharge,[15] which is highly consistent with the results of this study.

Multivariate regression analysis revealed that patients with disease duration ≥ 7 days had 3.596-fold higher odds of developing PPPD than those with disease duration < 7 days. These results are consistent with the theory of Özgirgin et al. that persistent abnormal input from peripheral vestibular signals can induce maladaptive plastic changes in the central nervous system.[16] If acute vestibular disorders are not corrected in a timely manner, the central nervous system will develop a compensatory strategy that inhibits vestibular input and relies instead on vision and proprioception to maintain balance. This mechanism is adaptive in the acute phase; once consolidated, the abnormal neural response pattern persists even after the peripheral lesion has resolved, which is precisely the core pathophysiological feature of PPPD. Other studies have also suggested that delayed diagnosis from symptom onset is a key predictor of persistent residual dizziness and subsequent progression to PPPD.[17]

The present study indicated that patients with residual dizziness within one week after successful repositioning had 2.687-fold higher odds of subsequent PPPD than those without residual dizziness. Approximately half of the patients in the PPPD group still reported non-rotatory dizziness or unsteadiness at one week after repositioning, whereas this proportion was only 18% in the non-PPPD group. The pathophysiological mechanism of residual dizziness remains unclear. Possible mechanisms include incompletely repositioned otoconia debris scattered outside the semicircular canals, mechanical perturbation of vestibular hair cells during repositioning maneuvers, and persistent low-level stimulation of the vestibular center by abnormal otolith gravity signals.[16,18] A recent interventional study by Shiozaki et al. demonstrated that early vestibular rehabilitation targeting residual dizziness significantly improved postural control and Dizziness Handicap Inventory scores,[19] providing evidence that residual dizziness serves as a modifiable target. Clinically, residual dizziness can be considered a phenotypic marker of the transition from acute peripheral vestibular lesions to chronic functional vestibular disorders, and its identification is crucial for stratified intervention.

Anxiety was independently associated with a higher likelihood of developing PPPD within one year after successful manual repositioning (OR = 2.872, 95% CI: 1.096–7.528). Previous studies have clearly identified anxiety induced by BPPV as an independent predictor of PPPD.[15] A recent review showed that patients with PPPD exhibit functional connectivity remodeling among visual-vestibular, sensorimotor, and emotional networks, characterized by weakened prefrontal cortex regulation of the limbic system and enhanced inhibition of vestibular input by visual input.[20] Notably, the HAM-A scores used in this study were documented within one week after successful repositioning, reflecting patients' psychological status during the early post-repositioning period rather than emotional disturbances arising during the subsequent chronic course. This suggests that clinicians should pay attention to patients' psychological status in the late acute phase rather than responding passively

until the chronic phase. A study of BPPV patients with residual dizziness showed that anxiety and depression were independent risk factors for progression to PPPD, and that study also found abnormal blood pressure to be associated with transformation risk.[21] The higher OR reported in that study may be partly related to its restriction to patients with residual dizziness, suggesting that the association between anxiety and subsequent PPPD may be more pronounced in patients with persistent residual symptoms.

Standardized vestibular rehabilitation training was independently associated with lower odds of PPPD, with patients who adhered to standardized training showing approximately 85% lower odds of PPPD than those with non-standardized training. This protective effect may be attributed to rehabilitation training, which promotes the absorption and settlement of residual otoconial debris through repeated head movements.[19] Repeated exposure to dizziness-inducing postures and visual scenes leads to vestibular adaptation, habituation, and sensory substitution, thereby reshaping the central integration of multisensory information.[16] Regular training reduces patients' somatic attention and anticipatory anxiety, interrupting the abnormal emotion-posture coupling response. Some studies have emphasized that treatment of residual dizziness should not rely on passive waiting but on active intervention to promote central compensation.[17] The results of this study provide quantitative support for this view from the perspective of PPPD prevention. The nomogram integrated long disease duration (≥ 7 days), residual dizziness, anxiety, and standardized vestibular rehabilitation training to estimate the probability of PPPD within one year after successful manual repositioning. The model showed good discrimination, with an area under the receiver operating characteristic curve of 0.827 and a bootstrap-corrected C-index of 0.813. The calibration curve indicated reasonable agreement between the predicted and observed probabilities, while decision curve analysis suggested a potential net benefit across threshold probabilities ranging from 0.09 to 0.83. However, the sensitivity was only 55.0%, indicating that some patients who subsequently developed PPPD could not be identified by the selected cutoff. In addition, the model was developed using a relatively small, single-center retrospective cohort and underwent internal validation only. Therefore, it should be considered a preliminary risk-stratification model rather than an established clinical decision tool, and its performance requires confirmation in larger external and prospective cohorts.

Strengths: This study focused on the development of PPPD after successful manual repositioning rather than on BPPV recurrence alone. It integrated clinically accessible vestibular, psychological, and rehabilitation-related variables into an intuitive nomogram for estimating the one-year probability of PPPD. In addition, the model was evaluated using discrimination, calibration, bootstrap internal validation, and decision curve analysis.

Limitations: First, this is a single-center study with a relatively small sample size and no external validation; the model's generalizability needs to be verified in subsequent multicenter studies. Second, anxiety assessment relied only on a single HAM-A measurement, without dynamic tracking of its evolution and interaction with symptoms. Third, the definition of residual dizziness was based on clinical complaints and lacked objective evidence, such as video Head Impulse Test (vHIT) or Vestibular Evoked Myogenic Potentials (VEMP). Finally, confounding factors such as medication and lifestyle interventions were not systematically recorded during follow-up, which may have affected the results.

This study has clear clinical implications. A transformation from BPPV to PPPD after repositioning is a dynamic process involving biological and psychological factors: a delayed disease course provides the pathological basis, residual dizziness serves as a transitional state, anxiety drives the vicious cycle, and vestibular rehabilitation training exerts blocking effects at multiple stages. Therefore, in clinical practice, efforts should be made to reduce diagnostic delay in BPPV, focus on patients who complain of residual dizziness after repositioning, identify and intervene early for anxiety, and actively promote standardized vestibular rehabilitation programs. The nomogram constructed in this study may assist in the preliminary identification of patients at higher risk of PPPD. However, its clinical applicability and potential role in individualized management require confirmation through external and prospective validation.

CONCLUSION

Long disease duration (≥ 7 days), residual dizziness, and anxiety were identified as independent risk factors for PPPD within one year after successful manual repositioning in patients with BPPV, whereas standardized vestibular rehabilitation training was identified as an independent protective factor. The nomogram incorporating these three independent risk factors and standardized vestibular rehabilitation training as an independent protective factor may help estimate the probability of PPPD within one year after successful manual repositioning in patients with BPPV. The nomogram may serve as a preliminary risk-stratification tool, but its clinical applicability requires further evaluation in larger, multicenter, prospective cohorts with external validation.

Recommendations: Future studies should address these limitations by including comprehensive risk factors and externally validating the model in larger populations.

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manuscript. It was not used for study design, data collection, statistical analysis, interpretation of the results, figure generation, or formulation of the scientific conclusions. All authors reviewed and approved the final manuscript and take full responsibility for its content.

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Authors' contributions:

LL: Literature search, study design and manuscript writing.
HY and CZ: Data collection, data analysis and interpretation. Critical Review.
LL: Manuscript revision and validation and is responsible for the integrity of the study.
All authors have read and approved the final manuscript.