Received: 27 August 2021 | Revised: 29 December 2021 | Accepted: 30 December 2021

DOI: 10.1002/lio2.735

ORIGINAL RESEARCH

Laryngoscope Investigative Otolaryngology

Presence of exacerbating factors of persistent perceptual-postural dizziness in patients with vestibular symptoms at initial presentation

Kayoko Kabaya MD, PhD[1] | Hitomi Tamai MD[1] | Akina Okajima MD[1] | Toshiya Minakata MD, PhD[1] | Masaki Kondo MD, PhD[2] | Meiho Nakayama MD, PhD[1] | Shinichi Iwasaki MD, PhD[1]

[1]Department of Otolaryngology, Head and Neck Surgery, Nagoya City University Graduate School of Medical Sciences, Aichi, Japan

[2]Department of Psychiatry and Cognitive-Behavioral Medicine, Nagoya City University Graduate School of Medical Sciences, Aichi, Japan

Correspondence
Kayoko Kabaya, Department of Otolaryngology, Head and Neck Surgery, Nagoya City University Graduate School of Medical Sciences, 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya, Aichi 467-8601, Japan.
Email: kabayakayoko@gmail.com

Funding information
the Japan Agency for Medical Research and Development (AMED); the Ministry of Education, Culture, Sports, Science and Technology, Grant/Award Numbers: 17K11338, 18K09370, 20K11161

Abstract

Objective: To investigate the presence of exacerbating factors of persistent perceptual-postural dizziness (PPPD) in patients with vestibular symptoms during the early period after vestibular symptoms onset, and to examine possible predictive factors for developing PPPD later.

Methods: One hundred and fifty-five consecutive patients with vestibular symptoms who presented less than 90 days from the onset were included in this study. They filled out the Niigata PPPD Questionnaire (NPQ) that consists of 12 questions on the exacerbating factors of PPPD. The NPQ scores of patients who developed PPPD were compared with those of patients who did not develop PPPD during the follow-up.

Results: Seventy-eight of the155 patients (50.3%) showed positive NPQ scores (≥27 points). High NPQ scores were found in patients diagnosed with psychogenic dizziness and vestibular neuritis. During the follow up for an average of 543.3 days after the initial presentation, eight patients (10.3%) developed PPPD. Seven of these eight patients (87.6%) showed positive NPQ scores and all of them had all three exacerbating factors of PPPD at their initial presentation. The NPQ scores of the patients who developed PPPD (40.6 ± 11.6) were significantly higher than those of the patients who did not develop PPPD (26.4 ± 18.3; p <.05).

Conclusion: Approximately a half of the patients with vestibular symptoms had exacerbating factors of PPPD in the early stages of the disease. Patients who develop PPPD are likely to have its exacerbating factors in the initial stages after presentation.

Level of Evidence: 3.

KEYWORDS
exacerbating factors, persistent postural-perceptual dizziness, spectrum, the Niigata PPPD Questionnaire

1 | INTRODUCTION

Persistent postural-perceptual dizziness (PPPD) is persistent chronic vestibular syndrome lasting more than 3 months and classified as a functional disorder, not a structural or psychiatric condition.[1] The symptoms of PPPD are dizziness, unsteadiness or nonspinning vertigo, and are exacerbated by upright posture or walking, active or passive movement, and exposure to moving or complex visual stimuli.[1] The presence of these three exacerbating factors is a prerequisite for PPPD.[1]

The characteristics of the exacerbating factors of PPPD that cause worsening of vestibular symptoms are not specific for PPPD. Powell et al. examined the frequency the symptoms of PPPD in the general population and reported that approximately 10% of the non-clinical population have PPPD symptoms, suggesting that PPPD is a spectrum that preexists in the population, rather than only being a consequence of a vestibular insult.[2] However, it is unknown whether people who have exacerbating factors of PPPD have a tendency to develop PPPD later or not.

PPPD is typically preceded by acute vestibular disorders or symptoms, such as vestibular neuritis, benign paroxysmal positional vertigo, or labyrinthitis.[1] One hypothesis is that PPPD is caused by excessive adaptation of the brain to vestibular disorders, resulting in more reliance on visual information about self-movement. Another hypothesis is that a failure of the postural control system to adapt, leads to conflict in the sensory consequences of self-movement.[3] However, it is still unclear why only a portion of patients develop PPPD after the same vestibular disorders. Here we explore the possibility that patients who develop PPPD have its exacerbating factors in the early period after the acute vestibular insult.

The objectives of this study were to evaluate the frequency of patients with exacerbating factors of PPPD during the early period after vestibular symptoms onset, and to examine possible predictive factors, present from early period after vestibular symptoms onset, for developing PPPD including exacerbating factors as measured with questionnaire, self-reported dizziness handicap, vestibular function test results, and initial diagnosis.

2 | MATERIALS AND METHODS

This study was a retrospective chart review in a tertiary referral center. This study was approved by the Research Ethics Committee, Graduate School of Medicine, Nagoya City University (#60-20-0193) and was conducted according to the tenets of the Declaration of Helsinki. Written informed consent was obtained from all participants.

2.1 | Subjects

We reviewed the medical records of 700 consecutive new patients who visited the Dizziness Clinic of Nagoya City University Hospital and who underwent detailed vestibular function testing for vestibular symptoms from January 2019 to December 2020. Among them, patients who satisfied the following criteria were included: (1) aged 20 years or older, (2) no previous history of vestibular symptoms, and (3) visited the clinic less than 90 days after symptom onset. We excluded patients with (1) severe psychiatric disorders or (2) difficulty in completing the questionnaires because of medical and other factors.

All of these patients received a detailed history-taking and a battery of tests including a physical examination, neurological examination, pure-tone audiometry, and positional/positioning nystagmus testing under infrared CCD goggles. The neurotological examination included electronystagmography, caloric testing, cervical vestibular evoked myogenic potentials (cVEMP), and video head impulse test (vHIT). Neuroimaging studies such as computed tomography and magnetic resonance imaging of the brain were performed when considered necessary. The diagnostic criteria used in this study were: PPPD,[1] benign paroxysmal positional vertigo,[4] Meniere's disease,[5] psychiatric dizziness,[6] vestibular migraine,[7] vestibular neuritis,[8] sudden deafness,[9] delayed endolymphatic hydrops,[10] orthostatic dysregulation,[11] and bilateral vestibulopathy.[12]

All patients filled out two questionnaires at their first visit: the Niigata PPPD Questionnaire (NPQ)[13] and the Dizziness Handicap Inventory (DHI).[14] All patients were followed up for more than 3 months. Patients who have vestibular symptoms presented on most days for 3 months or more were checked whether they were diagnosed as having PPPD according to the diagnostic criteria.[1]

2.2 | The Niigata PPPD Questionnaire

The NPQ was developed to diagnose and assess the severity of PPPD by Yagi et al.[13] The NPQ consists of 12 questions that evaluates the degree of symptom exacerbation by the three exacerbating factors: upright posture or walking, active or passive movement, and visual stimulation (Table S1). The severity of each factor was evaluated using four questions which were scored from 0 (none) to 6 (unbearable). Since it has been reported that a total score of 27 was the cut-off point for diagnosing PPPD with the best sensitivity (70%) and specificity (68%),[13] we judged patients with a NPQ score of 27 or higher as positive for exacerbating factors of PPPD. Additionally, we used the sum of the scores of each factor to assess the severity of exacerbating factors, and judged patients as positive for each factor when the sum of the scores of for that factor was 9 or higher.

2.3 | The Dizziness Handicap Inventory

The DHI is a 25-item self-assessment scale designed to evaluate the self-perceived handicap caused by dizziness.[14] The scale identifies three types of difficulty associated with dizziness: functional difficulties (9 items), emotional difficulties (9 items), and physical difficulties (7 items). The scores on the DHI range from 0 (no handicap) to 100 (significant perceived handicap). The total score can be used to

classify the handicap as mild (0–30), moderate (>30–60), or severe (>60–100).[15]

2.4 | Vestibular function tests

Caloric testing was carried out using air at 24°C and at 50°C for 60 s each. Maximum slow-phase eye velocity was measured using electronystagmography and canal paresis (CP)% was calculated using Jongkee's index formula.[16] A maximum slow phase eye velocity <10°/s bilaterally or CP% >20 were regarded as a significant unilateral or bilateral weakness of responses.[17]

Testing of cVEMP was performed to assess the saccule–inferior vestibular nerve function, using the Neuropack system (Nihon Koden, Japan). Subjects lay in a supine position on a bed with surface electromyographic electrodes placed on the upper half of each sternocleidomastoid muscle (SCM) and a reference electrode over the upper sternum. Subjects were asked to raise their heads off the bed to contract the SCM. Air-conducted short-tone bursts of 500 Hz (rise/fall time = 1 ms, plateau time = 2 ms, 125 dB SPL) were used to induce cVEMP. We calculated the asymmetry ratio (AR) for the amplitude of p13-n23 with the following formula using the amplitude of p13-n23 on the affected side (Aa) and that on the unaffected side (Au): AR (%) = 100 * (Au − Aa)/(Au + Aa). On the basis of results from normal subjects, the upper limit of AR was set to 34.0.[18] When no reproducible p13-n23 was present in 2 consecutive runs, we regarded it as an "absent" response. When a reproducible p13-n23 was present and the AR was greater than the predefined upper limit for normal subjects, we regarded it as a "decreased" response. Both "decreased" and "absent" responses were classified as abnormal responses.

The vHIT was performed to assess the vestibulo-ocular reflex (VOR) in the three semicircular canal planes using an Eye-See-Cam system (Interacoustics, Denmark). Subjects were seated 1 m from a black fixation dot on a wall that served as the visual target. While the subject was asked to stare at the fixation dot, the examiner briefly and unpredictably rotated the subject's head through a 10–20° angle. The head rotations were made in the lateral, the left anterior-right posterior and the right anterior left posterior planes. The head impulses were repeated at least 15 times in each direction, and the eye and head velocities were recorded. A mean VOR gain in vHIT of <0.7 for the vertical canals and <0.8 for the lateral canals was regarded as functionally abnormal.[19]

2.5 | Statistical analysis

The percentage of patients with NPQ scores 27 points or higher was calculated. The patients were divided into two groups: patients who developed with PPPD during the follow-up period and patients who did not develop PPPD. The Shapiro–Wilk test was used to check the normal distribution of the data. The Mann–Whitney *U* test was used to compare the two groups in terms of clinical characteristic and the NPQ and DHI scores. We used the Fisher's exact test to evaluate binary data. All statistical tests were two-sided. A difference of $p < .05$ was considered significant. All statistical analyses were performed

TABLE 1 Demographics and scores of the NPQ and DHI of 155 patients with dizziness

	Total Mean (SD) ($n = 155$)	PPPD Mean (SD) ($n = 8$)	Non-PPPD Mean (SD) ($n = 147$)	*U* value[a]	*p* value[a]
Age	55.4 (16.1)	45.8 (10.1)	55.9 (16.2)	812.5	.070
Gender (male: female)	1: 1.5	1: 1	1: 1.5		.714
Duration from dizziness onset (days)	32.7 (27.0)	31.6 (13.1)	32.8 (27.5)	548.5	.752
Duration of follow-up [range] (days)	543.3 (203.2) [243–945]	451.6 (243.0) [243–854]	548.3 (200.6) [252–945]	790.5	.102
NPQ score					
Total	27.1 (18.3)	40.6 (11.6)	26.4 (18.3)	294.0	.018*
Upright posture/walking	8.9 (6.8)	14.9 (4.5)	8.6 (6.7)	263.5	.009*
Movement	9.4 (6.3)	13.6 (4.7)	9.2 (6.3)	330.0	.037*
Visual stimulation	8.8 (6.8)	12.1 (5.9)	8.6 (6.8)	389.5	.109
DHI score					
Total	41.6 (23.6)	57.5 (23.3)	40.8 (23.4)	355.0	.060
Physical	11.4 (7.3)	15.8 (7.0)	11.1 (7.3)	370.5	.078
Emotional	14.6 (9.3)	20.8 (8.7)	14.3 (9.2)	353.0	.057
Functional	15.6 (10.1)	21.0 (11.9)	15.3 (9.9)	415.5	.163

Abbreviations: DHI, Dizziness Handicap Inventory; NPQ, The Niigata PPPD Questionnaire; SD, standard deviation.
[a]PPPD group versus non-PPPD group.
*$p < .05$.

TABLE 2 Clinical diagnoses of 155 patients with dizziness

Diagnosis	*N* (%)	NPQ Mean (SD)	Positive NPQ *N* (%)
BPPV	51 (32.9)	21.2 (17.1)	21 (41.2)
Meniere's disease	23 (14.8)	32.0 (19.3)	15 (65.2)
Psychiatric dizziness	12 (7.7)	41.8 (18.9)	10 (83.3)
Peripheral vestibular dysfunction	9 (5.8)	18.9 (20.1)	2 (22.2)
Vestibular migraine	8 (5.2)	27.5 (16.5)	4 (50.0)
Vestibular neuritis	6 (3.9)	39.0 (15.3)	5 (83.3)
Sudden deafness with vertigo	5 (3.2)	28.0 (18.4)	2 (40.0)
Delayed endolymphatic hydrops	4 (2.6)	25.0 (17.2)	1 (25.0)
Orthostatic dysregulation	3 (1.9)	16.7 (10.1)	0 (0)
Idiopathic bilateral vestibulopathy	2 (1.3)	21.5 (6.4)	0 (0)
Others	4 (2.6)	31.8(20.0)	3(75.0)
Unknown cause	28 (18.1)	28.6 (16.9)	15 (53.6)

Note: Positive NPQ: NPQ scores ≥27.
Abbreviation: BPPV, benign paroxysmal positional vertigo.

TABLE 3 Clinical characteristics of patients who developed PPPD during the follow up

Case	Sex	Age	Duration	Initial diagnosis	CP (%)	vHIT	cVEMP	NPQ (U/M/V)	DHI
1	M	40	37	Vestibular neuritis	19	Normal	Normal	39 (15/13/11)	40
2	F	28	33	Meniere's disease	10	Normal	Normal	53 (16/19/18)	82
3	M	43	33	Unknown	18	N/A	Normal	32 (9/9/14)	20
4	F	55	22	Unknown	7	N/A	N/A	60 (20/20/20)	72
5	M	42	55	Unknown	2	Normal	Normal	42 (22/14/6)	44
6	F	44	18	Peripheral vestibular dysfunction	8	Normal	No response on the left side	25 (10/6/9)	48
7	F	57	40	Vestibular neuritis	61	Normal	No response on the left side	43 (14/13/16)	88
8	M	57	15	BPPV	24	Normal	Normal	31 (13/15/3)	66

Abbreviations: cVEMP, cervical vestibular evoked myogenic potentials; CP, canal paresis; Duration, duration from dizziness onset (days); F, female; M, male; N/A, not available; U/M/V, upright posture or walking/movement/visual stimulation; vHIT, video head impulse test.

using EZR version 1.37 for Windows (Saitama Medical Center, Jichi Medical University, Saitama, Japan).[20]

3 | RESULTS

Of the 700 consecutive patients with vestibular symptoms, we identified 155 patients (22.1%; 62 men and 93 women; age range 20–91 years, mean age ± SD: 55.4 ± 16.1 years) who were eligible for this study. Five hundred and forty-five patients were excluded because they visited the clinic more than 90 days after the onset. The mean interval from the onset of vestibular symptoms to the first visit to our clinic was 32.7 ± 27.0 days. Table 1 shows the demographics of these patients.

The mean NPQ score of the 155 patients was 27.1 ± 18.3, and 78 patients (50.3%) showed positive NPQ scores (≥27 points). The subscale scores of each exacerbating factor were 8.9 ± 6.8 for upright posture/walking, 9.4 ± 6.3 for movement, and 8.8 ± 6.8 for visual stimulation. The number of the patients who showed a positive subscale score (≥9 points) was 77 (49.7%) for upright posture/walking, 83 (53.5%) for movement, and 74 (47.7%) for visual stimulation.

Table 2 shows the clinical diagnoses of the patients. The total NPQ score was highest in psychiatric dizziness (41.8 ± 18.9), followed by vestibular neuritis (39.0 ± 15.3). The percentage of patients who showed positive NPQ scores (≥27 points) was also high in these diseases (83.3% for psychiatric dizziness and vestibular neuritis).

Patients were followed up for an average of 543.3 ± 203.2 days (range 243–945 days) from the initial presentation and during this time, eight patients (10.3%) developed PPPD (Table 3). Seven of the eight patients (87.6%) showed positive NPQ scores (≥27 points). With respect to the exacerbating factors, all the eight patients had a positive score for upright posture or walking, seven of the patients had a positive score for movement and for visual stimulation. All the patients had at least some symptoms (>0 points) of each of the three exacerbating factors.

We compared the clinical characteristics and the NPQ and DHI scores between patients who developed PPPD (PPPD group) and

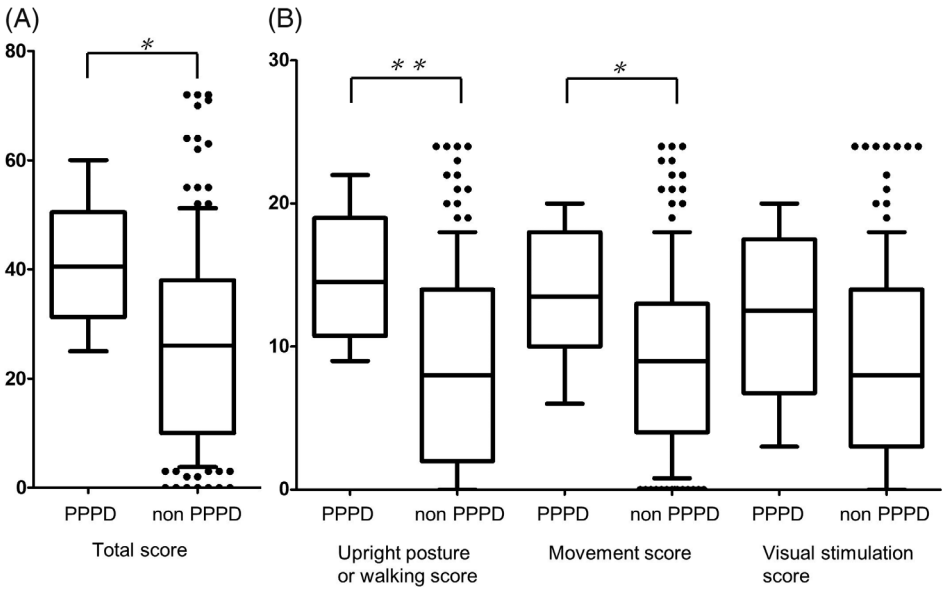

FIGURE 1 Niigata PPPD Questionnaire (NPQ) scores of patients who developed PPPD and those who did not PPPD during the follow up period. (A) The total scores of NPQ of the patients who developed PPPD (PPPD group) and those who did not develop PPPD (non-PPPD group). (B) The scores of three exacerbating factors of PPPD (upright posture or walking, movement, and visual stimulation) of patients in PPPD and non-PPPD groups. $^{**}p < .01$, $^{*}p < .05$

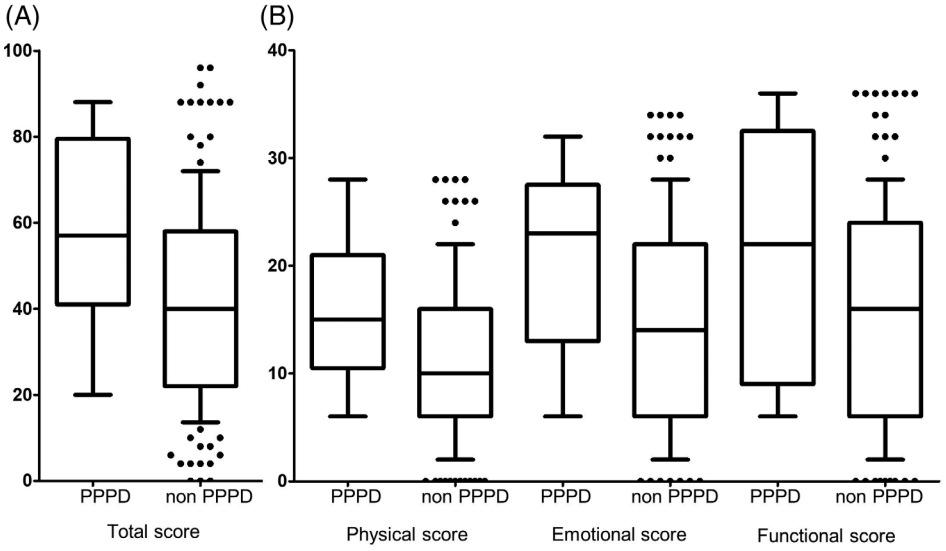

FIGURE 2 Dizziness Handicap Inventory (DHI) scores of patients in PPPD and non-PPPD groups. (A) The total scores of DHI of patients in PPPD and non-PPPD groups. (B) The physical, emotional, and functional score of patients in PPPD and non-PPPD groups

those who did not develop PPPD (non-PPPD group; Table 1). There were no significant differences in the mean age or sex ratio between the two groups (age: $U = 812.5$, $p = .082$; sex ratio: $p = .714$). The total NPQ score of the PPPD group (40.6 ± 11.6) was significantly higher than that of the non-PPPD group (26.4 ± 18.3; $U = 294.0$, $p = .018$; Figure 1A). Among the three exacerbating factors, the upright posture or walking and the movement scores of the PPPD group were significantly higher than those of the non-PPPD group (upright posture/walking: $U = 263.5$, $p = .009$; movement: $U = 330.0$, $p = .037$), while there were no significant differences in the visual stimulation score between the two groups ($U = 389.5$, $p = .109$; Figure 1B).

On the other hand, the total scores of the DHI, and physical, emotional, and functional subscales of the DHI, were not significantly different between the PPPD group and the non-PPPD group (total: $U = 355.0$, $p = .060$; physical: $U = 370.5$, $p = .078$; emotional: $U = 353.0$, $p = .057$; functional: $U = 415.5$, $p = .163$) (Figure 2). There

TABLE 4 Results of vestibular function tests of patients who developed PPPD and those who did not develop PPPD during the follow-up

Examination		PPPD ($N = 8$) N (%)	Non-PPPD ($N = 147$) N (%)	p value
Caloric testing	Normal	6 (75.0)	36 (48.0)	.265
	Abnormal	2 (25.0)	39 (52.0)	
vHIT	Normal	6 (100.0)	42 (76.4)	.326
	Abnormal	0 (0.0)	13 (23.6)	
cVEMP	Normal	5 (71.4)	34 (65.4)	1
	Abnormal	2 (28.6)	18 (34.6)	

Abbreviations: cVEMP, cervical vestibular evoked myogenic potentials; non-PPPD, patients who did not develop PPPD during the follow-up; PPPD, patients who developed PPPD during the follow-up; vHIT, video head impulse test.

were no significant differences in the results of the vestibular function test between the two groups ($p > .2$; Table 4).

4 | DISCUSSION

In the present study, we examined the presence of exacerbating factors of PPPD in patients with vestibular symptoms within 90 days of onset, and revealed that approximately a half of the patients with vestibular symptoms had exacerbating factors from an early stage of the disease. Furthermore, we compared the severity of each exacerbating factor between the patients who went on to develop PPPD and those who did not develop PPPD during the follow-up period, and showed that the patients who developed PPPD had more severe symptoms of the exacerbating factors at the initial stage of their disease in comparison with those patients who did not develop PPPD. Our results suggest that patients who develop PPPD are likely to have its exacerbating factors at the initial stage of their diagnosis.

The pathophysiological mechanisms underlying PPPD are still unclear. Several hypotheses such as alterations in postural control strategies,[21–23] shifts in multi-sensory integration, resulting in more reliance on visual information,[24,25] and reduced cortical integration of spatial orientation and threat assessment networks[26] have been postulated. While anxiety-related personal traits and high levels of anxiety and vigilance about acute vestibular symptoms have been postulated as a possible risk factor, it is unknown why a portion of patients develop PPPD subsequent to vestibular disorders whole others do not.

While the presence of the three exacerbating factors is essential in making a diagnosis of PPPD, the role of these factors in the development of PPPD is still unclear. Powell et al. examined the frequency of people having symptoms of PPPD in the general population using clinical questionnaires, and found that approximately 10% of the non-clinical population have PPPD symptoms.[1] In the present study, we examined the frequency of the exacerbating factors of PPPD in patients with dizziness within 90 days of onset, and found that approximately a half of the patients had the three factors from an early stage of the disease, suggesting that the exacerbating factors of PPPD preexist in many patients with vestibular symptoms as well as in the general population.

Among the clinical diagnoses of patients with vestibular symptoms, psychiatric dizziness showed the highest NPQ scores and approximately 80% of the patients with this disease showed positive NPQ scores. It is suggested that patients with psychogenic dizziness might be misdiagnosed as PPPD if diagnosed solely relying on the NPQ.

In the present study, approximately 10% of the patients with vestibular symptoms developed PPPD during the follow-up period, and most of them had the exacerbating factors of PPPD from the initial presentation. Patients who developed PPPD showed significantly higher NPQ scores compared with those who did not develop PPPD, while there were no differences in the DHI scores or results of vestibular function test between them. These results suggest that the severity of the exacerbating factors of PPPD during the early period of vestibular symptoms might affect the later development of PPPD. It is also possible that other factors than high NPQ scores during the early period can predict development of PPPD. To test this hypothesis, a large prospective study examining the degree of exacerbating factors and development of PPPD is necessary.

This study has several limitations. First of all, this is a retrospective study. There might be the potential for selection bias in the distribution of patients with vestibular symptoms. Second, we included patients who were within 3 months of the onset of vestibular symptoms and defined this as the early period of the disease. It is possible that by restricting the patients included in this study to even earlier stages of their disease (e.g., ≤1 month from onset) the rate of patients with exacerbating factors of PPPD might have been different. Third, the number of patients who developed PPPD during the follow-up period was very small. Therefore, the characteristics of patients who developed PPPD after the follow-up period remain unknown. A large-scale study including multiple dizziness clinic are necessary to elucidate the characteristics of the patients who develop PPPD later.

5 | CONCLUSION

We examined the presence of the exacerbating factors of PPPD in patients with vestibular symptoms during the early period after vestibular symptoms onset and showed that approximately a half of the patients had exacerbating factors. Furthermore, we showed that patients who developed PPPD later had significantly more severe symptoms of the exacerbating factors in comparison with patients who did not develop PPPD, suggesting that severe symptoms of the exacerbating factors of PPPD during the early period after vestibular symptoms onset might affect the later development of PPPD.

CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

ORCID

Kayoko Kabaya https://orcid.org/0000-0002-3815-705X

REFERENCES

1. Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the committee for the Classification of Vestibular Disorders of the Bárány Society. *J Vestib Res*. 2017;27(4):191-208.
2. Powell G, Derry-Sumner H, Rajenderkumar D, Rushton SK, Sumner P. Persistent postural perceptual dizziness is on a spectrum in the general population. *Neurology*. 2020;94(18):e1929-e1938.
3. Brandt T. Phobic postural vertigo. *Neurology*. 1996;46(6):1515-1519.
4. von Brevern M, Bertholon P, Brandt T, et al. Benign paroxysmal positional vertigo: diagnostic criteria. *J Vestib Res*. 2015;25(3–4):105-117.
5. Lopez-Escamez JA, Carey J, Chung WH, et al. Diagnostic criteria for Menière's disease. *J Vestib Res*. 2015;25(1):1-7.
6. Furman JM, Jacob RG. Psychiatric dizziness. *Neurology*. 1997;48(5):1161-1166.
7. Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria. *J Vestib Res*. 2012;22(4):167-172.
8. Jeong SH, Kim HJ, Kim JS. Vestibular neuritis. *Semin Neurol*. 2013;33(3):185-194.

9. Stachler RJ, Chandrasekhar SS, Archer SM, et al. Clinical practice guideline: sudden hearing loss. *Otolaryngol Head Neck Surg*. 2012; 146(3 Suppl):S1-S35.
10. Schuknecht HF. Delayed endolymphatic hydrops. *Ann Otol Rhinol Laryngol*. 1978;87(6 Pt 1):743-748.
11. Kim HA, Bisdorff A, Bronstein AM, et al. Hemodynamic orthostatic dizziness/vertigo: diagnostic criteria. *J Vestib Res*. 2019;29(2–3): 45-56.
12. Strupp M, Kim JS, Murofushi T, et al. Bilateral vestibulopathy: diagnostic criteria consensus document of the Classification Committee of the Bárány Society. *J Vestib Res*. 2017;27(4):177-189.
13. Yagi C, Morita Y, Kitazawa M, et al. A validated questionnaire to assess the severity of persistent postural-perceptual dizziness (PPPD): the Niigata PPPD questionnaire (NPQ). *Otol Neurotol*. 2019; 40(7):e747-e752.
14. Jacobson GP, Newman CW. The development of the dizziness handicap inventory. *Arch Otolaryngol Head Neck Surg*. 1990;116(4): 424-427.
15. Whitney SL, Wrisley DM, Brown KE, Furman JM. Is perception of handicap related to functional performance in persons with vestibular dysfunction? *Otol Neurotol*. 2004;25(2):139-143.
16. Jongkees LB, Maas JP, Philipszoon AJ. Clinical nystagmography. A detailed study of electro-nystagmography in 341 patients with vertigo. *Pract Otorhinolaryngol (Basel)*. 1962;24:65-93.
17. Iwasaki S, Fujimoto C, Kinoshita M, Kamogashira T, Egami N, Yamasoba T. Clinical characteristics of patients with abnormal ocular/cervical vestibular evoked myogenic potentials in the presence of normal caloric responses. *Ann Otol Rhinol Laryngol*. 2015;124(6): 458-465.
18. Murofushi T, Matsuzaki M, Wu CH. Short tone burst-evoked myogenic potentials on the sternocleidomastoid muscle: are these potentials also of vestibular origin? *Arch Otolaryngol Head Neck Surg*. 1999; 125(6):660-664.
19. McGarvie LA, MacDougall HG, Halmagyi GM, et al. The video head impulse test (vHIT) of semicircular canal function—age-dependent normative values of VOR gain in healthy subjects. *Front Neurol*. 2015; 6:154.
20. Kanda Y. Investigation of the freely available easy-to-use software 'EZR' for medical statistics. *Bone Marrow Transplant*. 2013;48(3):452-458.
21. Schniepp R, Wuehr M, Huth S, Pradhan C, Brandt T, Jahn K. Gait characteristics of patients with phobic postural vertigo: effects of fear of falling, attention, and visual input. *J Neurol*. 2014;261(4):738-746.
22. Querner V, Krafczyk S, Dieterich M, Brandt T. Phobic postural vertigo. Body sway during visually induced roll vection. *Exp Brain Res*. 2002;143(3):269-275.
23. Lubetzky AV, Aharoni MMH, Arie L, et al. People with persistent postural-perceptual dizziness demonstrate altered postural strategies in complex visual and cognitive environments. *J Vestib Res*. 2021; 31(6):505-517.
24. Riccelli R, Passamonti L, Toschi N, et al. Altered insular and occipital responses to simulated vertical self-motion in patients with persistent postural-perceptual dizziness. *Front Neurol*. 2017;8:529.
25. Cousins S, Kaski D, Cutfield N, et al. Predictors of clinical recovery from vestibular neuritis: a prospective study. *Ann Clin Transl Neurol*. 2017;4(5):340-346.
26. Indovina I, Riccelli R, Chiarella G, et al. Role of the insula and vestibular system in patients with chronic subjective dizziness: an fMRI study using sound-evoked vestibular stimulation. *Front Behav Neurosci*. 2015;9:334.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

How to cite this article: Kabaya K, Tamai H, Okajima A, et al. Presence of exacerbating factors of persistent perceptual-postural dizziness in patients with vestibular symptoms at initial presentation. *Laryngoscope Investigative Otolaryngology*. 2022;7(2):499-505. doi:10.1002/lio2.735