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Sudden-Onset Vertigo Associated with Persistent Spontaneous Torsional Nystagmus and Imbalance: A Unique Clinical Manifestation of Benign Paroxysmal Positional Vertigo but not Acute Unilateral Peripheral Vestibulopathy (Vestibular Neuritis)

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Abstract

Objective: Benign paroxysmal positional vertigo (BPPV) is the most common peripheral vestibular disease. Acute unilateral peripheral vestibulopathy (AUPVP), previously termed vestibular neuritis (VN), is the 3rd most common peripheral vestibular neuropathy with unknown etiology and pathogenesis. This study aimed to explore the association between BPPV and AUPVP (VN) and identify the potential etiology and pathogenesis of AUPVP (VN).

Methods: The clinical characteristics of 11 patients with sudden-onset vertigo associated with persistent spontaneous torsional nystagmus (PSTN) and imbalance were retrospectively reviewed. The effectiveness of the CCRM treatment was evaluated. The diagnosis of these patients was re.

Results: Balance dysfunction significantly improved after the first CCRM treatment and was completely alleviated after two CCRM treatments. PSTN was transformed to gaze-evoked persistent torsional nystagmus after the first CCRM treatment, and the degree of nystagmus was reduced. PSTN stopped in subsequent CCRM treatments. The severity of the vertigo gradually reduced after each treatment. The clinical manifestations of these patients after one or three CCRM treatments were the same as those of BPPV patients. Vertigo, PSTN, and imbalance were alleviated entirely in all patients after several CCRM treatments, suggesting that these patients had atypical BPPV.

Conclusion: Sudden-onset vertigo associated with PSTN and imbalance is a unique clinical manifestation of BPPV, caused by the canalith jam in the posterior semicircular canal or otolithiasis in multiple semicircular canals. The CCRM treatment is an effective therapeutic method for sudden-onset vertigo associated with PSTN and imbalance when central vestibular vertigo was excluded.

Keywords: Vertigo • Benign paroxysmal positional vertigo • Vestibular neuritis • Otolithiasis • Canalith jam

Introduction

Sudden-onset vertigo with nausea and/or vomiting is the core symptom of an acute vestibular syndrome (AVS), one of the most common symptomatic complaints in the Emergency Department of Neurology [1,2]. AVS is often accompanied by different forms of nystagmus, such as horizontal nystagmus, vertical nystagmus, torsional nystagmus, dissociated nystagmus, spontaneous nystagmus, gaze-evoked nystagmus, and evoked nystagmus (detected by the Dix-Hallpike test and the Roll test), which are associated with the corresponding diseases. Sudden-onset vertigo associated with persistent spontaneous torsional nystagmus (PSTN) and imbalance is a rare peripheral vestibular manifestation commonly diagnosed as vestibular neuritis (VN) based on clinical manifestations and vestibular function examination. Therapeutic options include corticosteroids and vestibular rehabilitation. The etiology and pathogenesis of VN are still unknown, though previous evidence has suggested that it may be caused by inflammation, labyrinthine ischemia, or type 1 herpes zoster infection [3-5]. The term "acute unilateral peripheral vestibulopathy (AUPVP)" has replaced VN because the etiology and pathogenesis of VN are unclear [6]. The present study aimed to explore the etiology and pathogenesis of sudden-onset vertigo associated with PSTN and imbalance and the association between benign paroxysmal positional vertigo (BPPV) and AUPVP (VN). The clinical characteristics, therapeutic methods, and treatment responses of 11 patients with sudden-onset vertigo associated with PSTN and imbalance were retrospectively reviewed.

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Materials and Methods

Patients

In this retrospective study, 11 patients who presented with sudden-onset vertigo associated with PSTN and imbalance were treated in the Emergency Department, Neurological Clinics, and Wards of this hospital between December 2015 and November 2019; five males six females aged between 37 and 66 years. The interval between the onset of vertigo and initiation of treatment was 1–6 days. No patient had prodromal infection. The demographic and clinical characteristics of all patients, including age, gender, previous illness, comorbidity diseases, PSTN, imbalance, brain acute vestibular syndrome (AVS) scan results and/or brain magnetic resonance imaging (MRI) scan results, and neurological examination results were reviewed and summarized in (Table 1).

Treatments

All patients were treated with the combined canalith repositioning maneuver (CCRM), including the Epley maneuver for the posterior semicircular

canal, the Deep head hanging (DHH) maneuver (also called the Yacovino maneuver), or the Kim maneuver for the anterior semicircular canal [7-9] and the Barbecue maneuver for the horizontal semicircular canal [7]. The canalith repositioning procedure was performed simultaneously for bilateral posterior and anterior semicircular canals, combined with the Barbecue maneuver if necessary. During the canalith repositioning procedure, the head of the patient stayed in a specific position until vertigo subsided or stopped. Then, the head was turned to the next position. All patients presented with nausea and/or vomiting. Intramuscular injections of 10 mg of metoclopramide and 25 mg of promethazine were given to alleviate vertigo, nausea, and/or vomiting. The CCRM treatment was performed after relief from vomiting and alleviation or disappearance of nausea. Corticosteroids were not used during treatment. Changes in dizziness, PSTN, and imbalance during each CCRM treatment were recorded. A tandem walking test was performed to assess imbalance.

Table 1. The demographic and clinical characteristics of the patients.

Case	Gender	Age (years)	Past Medical History	Comorb idities	BPPV before	PSTN	Imbalance	Cochlear symptoms	Brain CT scan	Brain MRI scan	Neurological examination		
1a (OP)	M	44	HTN Cerebral infarction	None	Yes	Fast phase to the right	Could not walk independently	None	No related findings	No related findings	+	Normal	Normal
2a (OP)	F	37	Migraine	None	No	Fast phase to the left	Could not walk independently	None	Normal	-	+	Normal	Normal
3a (OP)	F	55	HTN	None	No	Fast phase to the left	Could not walk independently	None	No related findings	No related findings	+	Normal	Normal
4b (OP)	F	44	None	None	No	Fast phase to the left	Could walk independently but unsteadily	None	Normal	Normal	+	Normal	Normal
5b (IP)	F	58	Bell's Palsy	None		Fast phase to the right	Could walk independently but unsteadily	None	No related findings	No related findings	+	Normal	Normal
6a (IP)	M	42	HTN	None	Yes	Fast phase to the right	Could not walk independently	None	Normal	Normal	+	Normal	Normal
7a (IP)	F	66	BPPV	None	Yes	Fast phase to the left	Could not walk independently	None	No related	Then		Then	Then
8 (IP)	M	51	HTN, DM, stumble and trauma	None	No	Fast phase to the right	Could not walk independently	None	Normal	Normal	+	Norma	Normal
9b (OP)	M	53	None	None	No	Fast phase to the right	Could walk independently but unsteadily	None	Normal	Normal	+	Normal	Normal
10b (IP)	M	40	None	None	No	Fast phase to the left	Could walk independently but unsteadily	None	Normal	Normal	+	Normal	Normal
11a (OP)	F	39	None	None	No	Fast phase to the left	Could not walk independently	None	Normal	-	+	Normal	Normal

OP: outpatient; IP: Inpatient; PSTN: Persistent spontaneous torsional nystagmus; FNT: Finger-to-nose test; HKST: Heel-knee-shin test; RS: Romberg sign; HTN: Hypertension; DM: Diabetes mellitus.

a: Could not complete Strait-line walking test or Tandem walking test
b: Could complete Strait-line walking test and Tandem walking test, but unsteadily
c: Neurological examinations were normal except PSTN, strait-line walking, Tandem walking, and Romberg sing.

Results

Patients' characteristics

A total of 11 patients with sudden-onset vertigo associated with PSTN and imbalance were included in this study. The demographic and clinical characteristics of all patients are summarized in (Table 1). They had acute onset and severe vertigo with nausea and/or vomiting. PSTN was present in all patients, with the right gaze being more evident than the left in six patients and the left gaze being more evident than the right in five patients. Seven patients were unable to walk independently due to severe balance dysfunction. No patient presented with tinnitus, hearing change, or other cochlear symptoms.

Changes in dizziness, PSTN, and imbalance after each CCRM treatment

The changes in dizziness, PSTN, and imbalance of each patient after each CCRM treatment are shown in Table 2. The vertigo was aggravated during the first CCRM treatment but then subsided at each head position, with slight residual dizziness. Torsional nystagmus, which occurred at most head positions, was aggravated, and then subsided but did not stop. Vertigo improved after the first CCRM treatment, but continuous dizziness was still present. Balance dysfunction improved significantly. Seven patients who could not independently walk before treatment could walk on their own but unsteadily after the first CCRM treatment. The straight line and Tandem walking tests showed that the imbalance improved significantly (Patient 6: Video 1: Tandem walking test before the first CCRM treatment; Video 2: Tandem walking test after the first CCRM treatment. Patient 9: Video 1: Tandem walking test before CCRM treatment; Video 2: Tandem walking test after the first CCRM treatment. Patient 10: Video 1: Tandem walking test before CCRM treatment). Four patients with unstable walking before treatment showed normal straight line walking test results and improved Tandem walking test results after treatment. PSTN was transformed into gaze-evoked persistent torsional nystagmus, and the degree of nystagmus was reduced.

Table 2. Changes in dizziness, PSTN, and imbalance after each CCRM treatment.

Case	Symptoms and signs	Before treatment	1st CCRM	2nd CCRM	3rd CCRM	4th CCRM	5th CCRM
1(OP)	Dizziness	Persistent Vertigo	Persistent dizziness	Day 5, Episodic dizziness	Day 7, dizziness disappeared	-	-
	PSTN	PSTN, Fast phase to the right	GEPT to the right	Nystagmus disappeared	Evoked nystagmus	-	-
	Imbalance	Could not walk independently; could not complete SLW or TW	Walking on their own but unsteadily; Improved SLW and TW	Waking on their own; Normal SLW and TW	-	-	-
2(OP)a	Dizziness	Persistent vertigo	Persistent dizziness	Day 3, Persistent dizziness	Day 5, little persistent dizziness	Day 9, episodic dizziness	On day 20, episodic dizziness disappeared
	PSTN	PSTN, fast phase to the left	GEPT to the left	Subtle GEPT to the left	Evoked nystagmus	Evoked nystagmus	No nystagmus
	Imbalance	Could not walk independently; Could not complete SLW or TW	Walking on their own, but unsteadily; Improved SLW and TW	Walking normally; Unsteadily in SLW and TW	Walking normally, Normal SLW and TW	-	-
3(OP)a	Dizziness	Persistent vertigo	Persistent dizziness	Day 7, persistent dizziness	Day 10, episodic dizziness	Day 15, episodic dizziness	On day 18, the dizziness disappeared
	PSTN	PSTN, fast phase to the left	GEPT to the left	Subtle GEPT to the left	Evoked nystagmus	Nystagmus disappeared	-
	Imbalance	Could not walk independently; Could not complete SLW or TW	Walking on their own, but unsteadily; Improved SLW and TW	Walking normally; Unsteadily in SLW and TW	Walking normally, Normal SLW and TW	-	-
4(OP)	Dizziness	Persistent vertigo	Persistent dizziness	Day 7 later, episodic dizziness	On day 11, the dizziness disappeared	-	-
	PSTN	PSTN, fast phase to the left	GEPT to the left	Subtle GEPT to the left	Nystagmus disappeared	-	-
	Imbalance	Could not walk independently; Could not complete SLW or TW	Walking on their own, but unsteadily; Improved SLW and TW	Walking normally; Unsteadily in SLW and TW	Walking normally, Normal SLW and TW	-	-
5(IP)	Dizziness	Persistent vertigo	Persistent dizziness	Day 3, episodic dizziness	Day 4, episodic dizziness	On day 5, the dizziness disappeared	
	PSTN	PSTN, fast phase to the right	GEPT to the right	Subtle GEPT to the right	Nystagmus disappeared	-	
	Imbalance	Could walk dependently but unsteadily; Unsteadily in SLW and TW	Walking on their own; Normal SLW and TW	-	-		

6(IP)	Dizziness	Persistent vertigo	Persistent dizziness	Day 2, episodic dizziness	Day 5, episodic dizziness	On day 7, the dizziness disappeared	
	PSTN	PSTN, fast phase to the right	GEPT to the right	Subtle GEPT to the right	Nystagmus disappeared	-	
	Imbalance	Could not walk independently; Could not complete SLW or TW	Walking on their own, but unsteadily; Improved SLW and TW	Walking on their own; Normal SLW and TW	-	-	
7(IP)	Dizziness	Persistent vertigo	Episodic dizziness	On day 3, the dizziness disappeared	-	-	-
	PSTN	PSTN, fast phase to the left	GEPT to the left	Nystagmus disappeared	-	-	-
	Imbalance	Could not walk independently; Could not complete SLW or TW	Walking on their own, but unsteadily; Improved SLW and TW	Walking normally; Normal SLW and TW	-	-	-
8(OP)	Dizziness	Persistent dizziness	Episodic dizziness	On day 2, the dizziness disappeared			
	PSTN	PSTN, fast phase to the right	GEPT to the right	Subtle GEPT to the right			
	Imbalance	Could walk dependently but unsteadily; Unsteadily in SLW and TW	Walking on their own; SLW and TW were normal	-			
9(IP)	Dizziness	Persistent vertigo	Persistent dizziness	Day 2, persistent dizziness	Day 4, episodic dizziness	On day 6, the dizziness disappeared	
	PSTN	PSTN, fast phase to the right	GEPT to the right	Subtle GEPT to the right	Evoked Nystagmus	Nystagmus disappeared	
	Imbalance	Could not walk independently; Could not complete SLW or TW	Walking on their own, but unsteadily; Improved SLW and TW	Walking normally; Unsteadily in SLW and TW	Walking normally, Normal SLW and TW	-	
10(IP)	Dizziness	Persistent vertigo	Persistent dizziness	Day 2, episodic dizziness	Day 4, episodic dizziness	On day 7, the dizziness disappeared	-
	PSTN	PSTN, fast phase to the left	GEPT to the left	Subtle evoked nystagmus	Nystagmus disappeared	-	-
	Imbalance	Could walk dependently but unsteadily; Unsteadily in SLW and TW	Walking on their own; Normal SLW and TW	-	-	-	-
11(OP)	Dizziness	Persistent vertigo	Persistent dizziness	Day 3, episodic dizziness	On day 5, the dizziness disappeared	-	-
	PSTN	PSTN, fast phase to the left	GEPT to the left	Evoked nystagmus	Nystagmus disappeared	-	-
	Imbalance	Could walk dependently but unsteadily; Unsteadily in SLW and TW	Walking on their own; Normal SLW and TW	-	-	-	-

OP: Outpatient; IP: Inpatient; PSTN: Persistent spontaneous torsional nystagmus; SLW: Strait-line walking; TW: Tandem walking; GEPT: Gaze-evoked persistent torsional nystagmus.
a: Barbecue maneuver was given in the 5th CCRM treatment.
Note: The extent and magnitude of nystagmus reduced when PSTN switched to GEPT. The extent of persistent vertigo reduced and switched persistent dizziness and/or episodic dizziness during CCRM treatment.

During the second CCRM treatment, the vertigo was aggravated and relieved briefly at each head position, with no residual dizziness. Transient torsional nystagmus was present at some head positions. Dizziness was alleviated after the treatment. Six patients still had persistent dizziness, but with a lower degree. Five patients presented with transient dizziness episodes, which occurred when the head position changed. All patients could walk normally after treatment, with normal straight line and Tandem walking test results (Patient 6: Video 3: Tandem walking test after the second CCRM treatment; Patient 9: Video 3: Tandem walking test after the second CCRM treatment; Patient 10: Video 2 Tandem walking test after the second CCRM treatment). Five patients presented with no gaze-evoked persistent torsional nystagmus, while six with persistent dizziness had subtler residual gaze-evoked torsional nystagmus.

During the third CCRM treatment, two patients had no dizziness or nystagmus at any head position. Three patients showed transient dizziness without nystagmus at some head positions, relieved after the treatment. In six patients with persistent dizziness, transient dizziness occurred at some head positions, some of which were accompanied by transient slight torsional nystagmus. After the treatment, persistent dizziness was relieved; the six patients presented with mild transient dizziness episodes associated with head position change. Gaze-evoked subtle persistent torsional nystagmus disappeared.

During the following CCRM treatments, two patients achieved a clinical cure. Dizziness was relieved during follow up, and no more CCRM treatment was performed. The symptoms were relieved entirely in three patients after the third CCRM treatment, and no more CCRM was performed. The remaining six patients had the fourth CCRM treatment. Transient dizziness without nystagmus appeared at some head positions. Dizziness was relieved in four patients after the treatment. Two patients still had dizziness episodes after the fourth CCRM treatment and therefore underwent the fifth CCRM treatment. After that, the dizziness was relieved. Among 11 patients, two had the Barbecue maneuver.

Overall treatment responses and follow up

After all CCRM treatments, no changes in speech or limb muscular power in the neurological physical examination were observed. All patients had normal bilateral finger nose test and bilateral heel knee tibia test results. The brain CT and/or MRI scans showed no new cerebellar or brainstem infarction/hemorrhage or other related abnormal findings. The vestibular function of four patients was examined. Three of them showed right horizontal semicircular canal palsy, and one showed left horizontal semicircular canal palsy. Three patients presented with vertigo associated with PSTN and imbalance just after BPPV was diagnosed, and the symptoms were relieved by the canalith repositioning procedure. One patient had a history of stumble and trauma two weeks before treatment.

All patients had complete remission of vertigo, PSTN, and imbalance after the CCRM treatments for bilateral posterior and anterior semicircular canals. The duration of symptoms and signs from initiation of treatment to complete remission was 3–20 days. Eight patients recovered within one week after the treatment, and three recovered within 14–20 days. During follow up, one patient presented with a recurrence of vertigo one month after the treatment and was diagnosed with BPPV. The symptoms were relieved after the canalith repositioning procedure.

Discussion

Sudden-onset vertigo with nausea and/or vomiting is a common clinical symptom of AVS [10], usually first diagnosed and treated in the Emergency Department of Neurology. There are two types of AVS, central vestibular vertigo and peripheral vestibular vertigo, distinguishable by several clinical methods, such as HINTS (Head Impulse, Nystagmus, Test of Skew) and the STANDING (SponTAneous Nystagmus, Direction, head Impulse test, standiNG) algorithm [11-14]. Brain CT and MRI scans are the most reliable auxiliary examinations to distinguish between central and peripheral vestibular vertigo. Cases with sudden-onset vertigo associated with PSTN and imbalance are rare. In this study, Patient 1 had no history of prodromal infection and presented with sudden-onset vertigo associated with PSTN and imbalance after the relief of the BPPV episode, suggesting that the canalith repositioning procedure may be effective. After three CCRM treatments, vertigo, PSTN, and imbalance were alleviated completely, suggesting that this treatment was effective. The following ten patients were also cured after having the same procedure, which confirmed the effectiveness of CCRM for sudden-onset vertigo associated with PSTN and imbalance.

All patients included in this study were excluded for central vestibular vertigo by brain CT and MRI scans. Three of them were diagnosed with BPPV and treated by the canalith repositioning procedure just before sudden-onset vertigo associated with PSTN and imbalance. After several CCRM treatments, all patients presented with the same clinical manifestations as classic BPPV and atypical symptoms of BPPV, such as persistent dizziness, feeling of floating when walking, blurred vision, and head cloudiness [15-18]. The canalith repositioning procedure is an effective therapeutic method for BPPV but not for other types of peripheral vestibular vertigo. The above evidence suggests that sudden-onset vertigo associated with PSTN and imbalance is a unique clinical manifestation of BPPV when central vestibular vertigo is excluded.

The etiology and pathogenesis of sudden-onset vertigo associated with PSTN and imbalance are unclear. Epley et al., initially, observed a sudden conversion of positional nystagmus to a rapid form of spontaneous nystagmus that persists irrespective of the head position. It occurs due to the obstruction of the semicircular canal by free floating otoliths within the semicircular canal. This phenomenon was defined as canalith jam, a rare complication of the canalith repositioning procedure for posterior semicircular canal canalithiasis [19,20]. Canalith jam, caused by the blockage of free floating particles within a canal, is a complication of BPPV after the canalith repositioning procedure or can develop spontaneously. The potential pathogenic mechanisms of canalith jam are as follows: (1) Canalith jam blocks the flow of the endolymph or the movement of the cupula. (2) Canalith jam results in a negative pressure between the cupula and the jamming point and transiently reduce the vestibulocochlear reflex [21]. (3) Canalith jam in a narrow point of the canal blocks the flow of the endolymph and the cupula displacement. The otolithic clump plugs the canal, exerting permanent transcupular pressure that causes persistent spontaneous nystagmus [22]. Only a few studies have reported canalith jam. In 2001, von Brevern et al. examined a patient with persistent vertigo and oscillopsia with spontaneous nystagmus beating to the left, and the vestibular function examination showed right horizontal palsy, suggesting horizontal canal plugging. These findings indicated that transient spontaneous nystagmus with unilateral canal palsy was probably due to the plugging of the horizontal canal [23]. In 2014, Chang et al. and Ko et al. reported cases with canalith jam in the horizontal semicircular canal following the canalith repositioning procedure [21,24]. In 2018, Comacchio et al. reported a case with a canalith jam in the left horizontal semicircular canal [22]. No cases with canalith jam in the posterior semicircular canal were reported after the first report by Epley et al. In this study, 3 out of 11 patients had the same clinical manifestations as described by Epley et al. The otoliths fell off and entered the posterior semicircular canal, resulting in canalith jam in the semicircular canal and abnormal endolymphatic flow, which eventually led to severe vertigo, PSTN, and imbalance. The CCRM treatment enables the reposition of otoliths back to the utricle. The relief of vertigo, nystagmus, and imbalance in patients with sudden-onset vertigo associated with PSTN and imbalance after the CCRM treatment could be explained by the pathogenesis of canalith jam. Trauma can induce secondary BPPV. One patient had a history of stumble and trauma before PSTN and imbalance in this study. The symptoms were relieved after the CCRM treatment, indicating that the onset of symptoms was associated with otoliths. In addition, when performing the Epley maneuver and DHH maneuver for bilateral posterior and anterior semicircular canals during the first CCRM treatment, vertigo and nystagmus presented at head positions on both sides, suggesting that otolithiasis in the unilateral or bilateral multiple semicircular canals may be another pathogenic mechanism of sudden-onset vertigo associated with PSTN and imbalance.

The above reported four patients with horizontal semicircular canalith jam underwent vestibular function test. Three of them presented with horizontal semicircular canal palsy [22-24], and the remaining one showed reduced vestibular function [21]. After the head shaking and canalith repositioning maneuvers, horizontal semicircular canal palsy disappeared, and the vestibular function returned to normal. Four out of 11 patients underwent vestibular function tests. Three patients showed right horizontal semicircular canal palsy, and one showed left horizontal semicircular canal palsy. These

findings suggested that the canalith jam resulted in abnormal vestibular function. After consultation with an otolaryngologist, the four patients with abnormal vestibular function were diagnosed with VN. The CCRM treatment, instead of corticosteroids, was used. All patients recovered after the CCRM treatment. BPPV cannot be excluded when patients with peripheral vestibular vertigo but not cochlear symptoms present with abnormal vestibular function.

VN is the third most common peripheral vestibular neuropathy (after BPPV and Manière's disease). The main clinical manifestations of VN are vertigo, nausea, and gait imbalance. The etiology and pathogenesis of VN are unknown, though possibly caused by inflammation, labyrinthine ischemia, and type 1 herpes zoster infection [3-5]. Patients with atypical BPPV (i.e., sudden-onset vertigo and abnormal vestibular function) may be misdiagnosed as VN; therefore, the term "AUPVP" is used in the international classification of vestibular disorders.3,6 As the clinical manifestations of peripheral AVS without cochlear symptoms do not meet the diagnostic criteria of classic BPPV [25,26], the canalith repositioning procedure was not performed in patients. The diagnosis was made based on clinical manifestations and vestibular function examination. Patients with severe vertigo but not cochlear symptoms are often diagnosed with VN. BPPV originating from the posterior semicircular canal often develops as a sequela in patients with VN [22]. Türk et al. reported 44 patients with BPPV secondary to VN [27]. Mandalà et al. found that the BPPV episode occurred in 5 out of 51 patients with VN during follow up. H-A Kim, et al. found that 51 cases of patients with VN had at least one otolith-related test abnormality in their study; the causality of abnormal otolith function in the pathogenesis of VN was not analyzed further [28].

The above studies suggested that VN and BPPV may share the same etiology and pathogenesis. Moreover, BPPV after VN predominantly affected VN patients who did not fully recover from the disease [29]. It should be further investigated whether BPPV following VN diagnosed before was a sequela of VN, or presented with unique manifestations of BPPV and gradually developed typical BPPV. The clinical manifestations of patients in this study were same as those of VN patients. The symptoms were alleviated entirely after the CCRM treatment. The favorable responses after the CCRM treatment in these patients implied that the etiology and pathogenesis of AUPVP (VN) were related to canalith jam in the semicircular canal or otolithiasis in the unilateral or bilateral multiple semicircular canals, but not inflammation, labyrinthine ischemia, or viral infection. Further clinical studies are needed to verify this hypothesis.

No previous study has reported the effectiveness of the CCRM treatment for sudden-onset vertigo associated with PSTN and imbalance. This study used this method because it could not determine whether a single semicircular canal or multiple semicircular canals were involved and which side was involved. The canalith repositioning procedure for the bilateral posterior and anterior semicircular canals was effective for patients with peripheral vestibular vertigo but not cochlear symptoms. Clinicians need to know when, how, and to whom the canalith repositioning procedure should be performed. Also, patients with peripheral vestibular vertigo but not cochlear symptoms should be treated with the canalith repositioning procedure to exclude BPPV. The canalith repositioning procedure should also be performed before diagnosing bilateral vestibulopathy [30], persistent postural perceptual dizziness [31], and presbyvestibulopathy [32]. Considering the heterogeneous manifestations of BPPV, the term "otolithiasis" instead of BPPV should be used.

Conclusion

Sudden-onset vertigo associated with PSTN and imbalance is a unique clinical manifestation of BPPV, possibly caused by the canalith jam in the posterior semicircular canal or otolithiasis in multiple semicircular canals. The etiology and pathogenesis of AUPVP (VN) may be related to the canalith jam in the semicircular canal or otolithiasis in unilateral or bilateral multiple semicircular canals. The CCRM treatment is effective for patients with sudden-onset associated with PSTN and imbalance when central vestibular vertigo was excluded.

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Conflicts of Interest

The authors declare that they have no conflict of interest.

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Ethical Approval

This study was approved by the medical ethics committee of Qingdao Municipal Hospital. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Authors Contributions

Xuesheng Liu Independently completed the treatment, experimental design and thesis writing of this group of patients.

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