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Autonomic dysfunction as a possible cause of residual dizziness after successful treatment in benign paroxysmal positional vertigo

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HIGHLIGHTS

- We performed autonomic test in patients with benign paroxysmal positional vertigo.
- Sympathoneural dysfunctions were common in patients with residual dizziness.
- Residual dizziness may be associated with sympathoneural autonomic dysfunction.

ABSTRACT

Objective: To investigate whether residual dizziness after successful treatment in patients with benign paroxysmal positional vertigo (BPPV) was associated with autonomic dysfunction.
Methods: Fifty-eight consecutive patients with BPPV who had successful canal repositioning procedures (CRPs) and showed no nystagmus or positional vertigo at the next follow-up visit were enrolled and divided into two groups with and without residual dizziness. We performed a standardized autonomic function test.
Results: Of the 58 patients, 25 (43%) complained of residual dizziness after successful CRPs, in which postural lightheadedness when righting from sitting, or short-lasting nonspecific dizziness that occurred during head movement or walking were common complaints. Orthostatic hypotension (OH) occurred in 11 patients (19%). Incidence of OH was significantly higher in patients with residual dizziness at the next follow-up than those without residual dizziness (40% and 3%, $p = 0.000$). Compared to patients without residual dizziness, patients with residual dizziness had larger falls in systolic BP during the valsalva maneuver and head-up tilt test. However, cardiovagal parasympathetic function was not different between the patients with and without residual dizziness.
Conclusion: In BPPV, residual dizziness after successful treatment may be associated with sympathoneural autonomic dysfunction.
Significance: This investigation could be useful in understanding the mechanism of residual dizziness in patients with BPPV.

1. Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disorder, which is characterized by brief recurrent episodes of vertigo that are triggered by changes in head position (Eggers, 2010; Lee, 2010). Patients with BPPV occasionally nonspecific dizziness with postural lightheadedness, especially when righting from sitting, despite successful removal of detached otolith particles by appropriate canal repositioning procedures (CRPs) (Magliulo et al., 2005; Seok et al., 2008; Faralli et al., 2009; Pezzoli et al., 2010; Teggi et al., 2011; Jung et al., 2012). It is similar to the orthostatic dizziness reported by patients with orthostatic hypotension (OH) (Kim et al., 2013). Although nonspecific residual dizziness with short-lasting postural lightheadedness is a common sensation after successful CRPs in patients with BPPV, its underlying mechanism remains to be elucidated.

Increasing evidence has demonstrated that the vestibular system participates in autonomic regulation, especially in adjusting cardiovascular control during body movement and change in posture (Pezzoli et al., 2010; Yates, 1994, 1998; Mori et al., 2005; Yates, 2009; Jauregui-Renaud et al., 2003; Barman et al., 2011; Sugiyama and Suzuki, 2011). However, most of the results (Yates,

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1994, 1998; Mori et al., 2005; Yates, 2009; Barman et al., 2011; Sugiyama and Suzuki, 2011) were drawn from experimental animal studies and only one prior study (Pezzoli et al., 2010) described autonomic dysfunction in BPPV, but this study was small in number and did not include beat to beat blood pressure (BP) recording, which is an essential for diagnosis of brief episodes of transient OH. To the best of our knowledge, no large, consecutive clinical series has focused on autonomic dysfunction as a possible cause of residual dizziness after successful treatment in BPPV. Thus, we performed this study to investigate whether residual dizziness after successful treatment in patients with BPPV was associated with autonomic dysfunction.

2. Methods and patients

Between January 2011 and December 2011, we initially identified 98 consecutive patients with BPPV from the Dizziness Clinics of Keimyung University School of Medicine. The diagnosis was based on a history of recurrent positional vertigo and the results of the Dix-Hallpike and supine head-turning tests (Eggers, 2010; Lee, 2010). The Dix–Hallpike test was considered to be positive if nystagmus was recorded with appropriate positioning, latency, duration, and fatigue, and reversed when the patient resumed a sitting position. With the affected ear down, geotropic torsional nystagmus (i.e., the upper poles of the eyes beating to the lowermost ear) occurs with an up-beating component for the posterior canal (PC) BPPV. In anterior canal (AC) BPPV, the Dix–Hallpike maneuver on either side may evoke downbeat nystagmus with an ipsitorsional (upper poles of the eyes beating toward the involved ear) component. Horizontal canal (HC) BPPV was diagnosed by horizontal, direction-changing positional nystagmus concurrent with vertigo elicited by the supine head-turning test. The patients were divided into geotropic and ageotropic groups according to the direction of the nystagmus.

The patients were treated with CRPs appropriate for their type of BPPV. Patients with posterior canal (PC) BPPV were treated with the maneuver described by Epley. The reverse Epley maneuver and barbecue rotation were used for the patients with anterior canal (AC) and horizontal canal (HC) BPPV, respectively. After the barbecue rotation, we suggested that the patients with HC BPPV of geotropic type lie on their healthy side during the following night. For patients with HC BPPV of ageotropic type, we recommended a mastoid vibrator or Brand–Daroff exercise to detach debris before applying CRPs. The maneuver was performed several times until repositioning was successful, as defined by the absence of nystagmus and positional vertigo. The presence of residual dizziness was determined at the next follow-up visit in the outpatient department and was based on the result of the positional test. At the next follow-up visit, all patients were independently interviewed by two authors (H.L., H.-A.K.) to determine the presence of nonspecific dizziness after successful treatment. If the positioning test was negative, we collected information on the presence of any residual dizziness and its characteristics at that time. Residual dizziness was defined as the sensation of lightheadedness or unsteadiness without positional vertigo or nystagmus at the time of autonomic function testing (i.e., on the day of the follow-up visit). A simple grading system of residual dizziness was used at follow-up: grade 1, no residual dizziness; grade 2, short-lasting postural lightheadedness or slight unsteadiness (intermittent, often triggered by standing from sitting or supine position, or head motion); grade 3, continuous lightheadedness (always by the same investigators: H.L., H.-A.K.). Additionally, each patient completed a characterized dizziness questionnaire, which included the duration, number, and type (i.e., vertigo or non-vertiginous dizziness) of dizziness, and the conditions under, which dizziness occurred (e.g., standing from sitting or supine position, head movement, or walking). They were also asked to complete a self-rated anxiety scale (SAS) (Zung, 1971).

The inclusion criteria for the present study were based on the following: (1) patients had an absence of both nystagmus and positional vertigo at the next follow-up. (2) Patients had no other medical condition that could potentially affect autonomic function. Patients with conditions such as persistent positional nystagmus and/or vertigo at the next follow-up (i.e., a treatment failure), advanced age (>80 years), NIDDM, secondary causes of BPPV such as head trauma or migraine, concomitant vestibular or central diseases, neurogenic OH due to multiple system atrophy, Parkinson's disease or other neurodegenerative diseases, or other medical illness known to affect autonomic functions were excluded. We excluded patients who were receiving medications that could potentially affect autonomic function, such as beta-blockers and anticholinergic agents. (3) Patients with a flattop Valsalva response (defined as a response in which the BP response increased above baseline by at least 20 mmHg for at least 10 s) or a square-wave variant of the Valsalva maneuver (VM) were also excluded. After 40 patients were excluded, 58 patients (16 men) were finally enrolled for this study. The average age of the patients was 55.8 ± 10.0 years with a range from 32 to 76 years. The PC, HC, and AC were involved in 21, 28, and 7 patients, respectively, and involvement of multiple canals (PC and HC) was observed in the remaining 2 patients. The algorithm used to study patients with BPPV is shown in Fig. 1.

A standardized battery of autonomic tests, including the head-up tilt test, VM, Valsalva ratio (VR), and deep breathing test using Finometer devices (FMS, Amsterdam, The Netherlands) for recording beat-to-beat BP and heart rate (HR) response was performed in all patients according to a previously validated method for the diagnosis of autonomic dysfunction (Low, 1993; Low and Benaroch, 2008). VM was done in the supine position. After a relaxation period of at least 2 min, the patients were instructed to take a deep breath and blow into a syringe through a mouthpiece attached to a manometer for 15 s until expiratory pressure was reached to 40 mmHg. The patients performed the VM at least twice, until a technically adequate tracing was obtained. BP magnitude during VM was determined for four phases, i.e., phase I, early phase II, late phase II, phase III, and phase IV as previously described (Low, 1993; Low and Benaroch, 2008). Example of BP and HR responses during the VM in a healthy control subject is shown in Fig. 2. The change in systolic BP from the peak to the trough value during phase II of the VM (i.e., Valsalva SBP) was calculated. Late phase II was considered to be abnormal if the magnitude of the end of phase II did not exceed the baseline (i.e., a negative value). Phase IV was considered to be abnormal if the magnitude of the phase IV response failed to reach baseline (i.e., a negative value) (Novak, 2011b). Baroreflex–cardiovagal gain, calculated from the slope of the relationship between cardiac interbeat interval and systolic blood pressure during the decline of pressure in phase II of the VM, was also calculated from previously verified method (Schrezenmaier et al., 2007). Deep breathing was done in a supine position. After a relaxation period of at least 2 min, the patients were instructed to breathe at a rate of 6 breaths per minute (5 s of inhalation and 5 s of exhalation). HR response was defined as the average HR difference (maximum–minimum) of the five largest consecutive responses and expiration/inspiration (E/I) ratio during deep breathing were calculated. VR was derived from the maximum HR divided by the lowest HR following the VM. The tilt protocol included at least 10 min in the supine position and 20 min of a tilt at 70 degrees. OH was defined by a decrease in systolic blood pressure of at least 20 mmHg or a decrease in diastolic pressure at least 10 mmHg between supine rest for 10 min and upright posture for 20 min. Persistent OH was defined as an orthostatic