Benign Paroxysmal Positional Vertigo in the Acute Care Setting

Terry D. Fife, MD[a,*], Michael von Brevern, MD[b]

KEYWORDS

• Dizziness • Positional vertigo • Paroxysmal vertigo
• Canalith repositioning maneuver • Liberatory maneuver • Canalolithiasis
• Cupulolithiasis

KEY POINTS

- Benign paroxysmal positional vertigo (BPPV) is a common cause of vertigo caused by calcium carbonate sediment originating from the utricle that dislodges and falls into one of the semicircular canals.
- BPPV usually causes brief attacks of spinning vertigo (10–20 seconds) induced by head position changes.
- Dix Hallpike positioning toward the affected side in posterior canal BPPV is associated with a 1- to 10-second latency and a fast phase.
- Treatment is achieved by "repositioning" calcium carbonate sediment from the posterior semicircular canal to the main vestibule.
- Treatment of posterior canal BPPV using the canalith repositioning maneuvers is effective in more than 90% of patients.

CASE SCENARIO

A 62-year-old woman presents to the emergency department reporting severe dizziness that came on abruptly at 4 AM. She recalls getting up from bed and felt "thrown" back to the bed and could barely stagger to the bathroom. She recalls having intense vertigo and feels unsteady on her feet when she tries to walk. She does not recall any prior similar vertigo and has not had slurred speech, diplopia, limb clumsiness, headache, or hearing loss. On physical examination, she can cautiously get up from bed but feels more "woozy" in doing so. There are no focal examination deficits, but Dix

Disclosures: Dr M. von Brevern has nothing to disclose; Dr T.D. Fife has nothing to disclose.
[a] Department of Neurology, Barrow Neurological Institute, University of Arizona College of Medicine, 240 West Thomas Road, Suite 301, Phoenix, AZ 85013, USA; [b] Department of Neurology, Park-Klinik Weissensee, Schoenstrasse 80, Berlin 13086, Germany
* Corresponding author.
E-mail address: tfife@email.arizona.edu

Neurol Clin 33 (2015) 601–617
http://dx.doi.org/10.1016/j.ncl.2015.04.003 **neurologic.theclinics.com**
0733-8619/15/$ – see front matter © 2015 Elsevier Inc. All rights reserved.

Hallpike positioning to the right side results in pronounced paroxysmal positional nystagmus.

EPIDEMIOLOGY

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo and its incidence increases with advancing age.[1] Unrecognized BPPV can be found in about 10% of certain geriatric populations[2,3] and there is a cumulative incidence of nearly 10% by age 80.[4] BPPV has a lifetime prevalence of 3.2% in women, 1.6% in men, and an overall prevalence of 2.4% in the adult general population.[4]

BPPV may be associated with trauma or viral infection, but the majority of cases are idiopathic.[5] BPPV is the most common vestibular problem, following head trauma.[6] About 13% of traumatic brain injury patients complain of positional vertigo and one-half have BPPV responsive to treatment.[7] BPPV owing to trauma is usually apparent within 1 week of head trauma as long as the patient has been moving sufficiently to provoke symptoms.[8] Adding to the challenge of diagnosis, acute vestibular neuritis can be associated with BPPV, possibly owing to inflammatory effects in the labyrinth or by affecting labyrinthine perfusion.[9] Idiopathic BPPV is about twice as common among women compared with men, whereas BPPV associated with trauma or viral neurolabyrinthitis occur with about equal frequency across genders.[5]

PATHOPHYSIOLOGY

BPPV is caused by calcium carbonate debris that erodes or becomes dislodged from the macula within the utricle. The calcium carbonate material has a density of 2.7 g/mL, which is considerably greater than that of endolymph (about 1 g/mL), so it moves or "sinks" by the effect of gravity and may fall into one of the semicircular canals.[10] The posterior canal is positioned anatomically to be the most likely recipient, which is why close to 80% of BPPV is related to the posterior canal. The term "canalolithiasis" refers to the most common mechanism of BPPV, in which mobile calcium debris triggers abnormal activation of the ampullary nerve. The term "cupulolithiasis" refers to the less common mechanism in which the calcium material is stuck to the cupula itself, causing inappropriate ampullary nerve activation.[11] The cupula is the gel-like structure in the ampulla of each semicircular canal that transduces mechanical endolymph flow into electrochemical nerve signals allowing the central nervous system to sense acceleration in the plane of that canal. This mechanism is supported by the response to canalith repositioning maneuvers, animal models,[12,13] abnormal utricular function in BPPV as determined by ocular vestibular evoked myogenic potential studies,[14] and a mathematical analysis.[15]

The calcium carbonate material seems to originate from the otoconia of the macule of the utricle. It is not well-understood why the material dislodges, but studies in aging rats showed pitting, fissuring, and cracking of otoconia and disruption of linking filaments, all of which correlated with advancing age.[16] Similar age-related changes have been demonstrated by scanning electron microscopy in specimens from the otoconia from the utricles of 5 humans, ranging in age from 47 to 63 years.[17] Osteoporosis and osteopenia may be factors increasing the likelihood of erosion and degeneration of otoconia.[18] Such a process could potentially explain the greater prevalence of idiopathic BPPV in women because of estrogen effects on calcium deposition. The number of otoconia and size was diminished in osteoporotic rats.[19] Postmenopausal women with BPPV had a high prevalence of osteopenia and osteoporosis.[20] Genetic factors leading to predisposition for BPPV may also prove important in some patients; 1 large family suggested linkage to a region on chromosome 15.[21]

Download English Version:

https://daneshyari.com/en/article/3078013

Download Persian Version:

https://daneshyari.com/article/3078013

Daneshyari.com