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Review Article

Revisiting pathophysiology of benign paroxysmal positional vertigo: a review

Santosh Kumar Swain*

Department of Otorhinolaryngology and Head and Neck Surgery, Manipal TATA Medical College, Jamshedpur, Manipal Academy of Higher Education, Manipal, India

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***Correspondence:**
Dr. Santosh Kumar Swain,
E-mail: santoshvoltaire@yahoo.co.in

ABSTRACT

Benign paroxysmal positional vertigo (BPPV) is the most common cause of peripheral vertigo. There are several possible underlying causes of BPPV, although the idiopathic form is the most common. BPPV is characterized by brief recurrent episodes of vertigo that are triggered by changes in head position. Although BPPV is a benign vestibular disorder, it can be a severe and disabling problem for some of patients. The pathophysiology of BPPV is still unclear. The pathophysiology for BPPV is complex and the underlying mechanism is related to free-floating debris/otoliths in the semicircular canal (canalolithiasis) or debris/otoliths attached to the cupula (cupulolithiasis). These otolith/debris are originally accumulated after detachment from the neuroepithelium of the utricular macula secondary to degeneration. BPPV can occur following other vestibular disorders. In the majority of cases, the triggering factors are unknown. Some patients of BPPV have a history of previous inner ear diseases such as Meniere's disease or acute unilateral peripheral vestibulopathy. This clinical entity is well-defined in medical literature and usually effectively treated by certain physical maneuvers. However, the pathophysiology is still obscure and is being critically discussed in this article, which reviews the details pathological mechanism for BPPV. This review article will discuss that aging, trauma, migraine, Meniere's disease, vestibular neuronitis, and vitamin-D deficiency are the most commonly investigated etiopathological factors resulting in BPPV.

Keywords: BPPV, Pathophysiology, Aging, Vitamin-D deficiency

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is defined as transient, positional induced torsional nystagmus with vertigo.[1] The clinical presentations are often provoked with a specific head position.[1] The diagnosis of BPPV is supported by the limited duration of vertigo, brief latency, fatigability, and reversibility of the nystagmus upon returning to an erect position.[2] Although clinical manifestations of BPPV are well-defined and treated effectively with repositioning maneuvers, its pathophysiology is still unclear. The pathophysiology of the BPPV is based on the displacement of the cupula due to either free-floating debris/otoliths in the semicircular canal (canalolithiasis) or attachment of debris/otoliths to the cupula (cupulolithiasis).[3] Any pathology of the inner ear that detaches the otoconia is thought to cause BPPV. Secondary BPPV is found when an associated etiology is identified. However, the exact underlying pathology is still obscure; in such a condition, idiopathic BPPV is diagnosed.[4] This review article is aimed to review the possible pathophysiology of BPPV.

LITERATURE SEARCH

Multiple systematic methods were used to find current research publications on the pathophysiology of BPPV. We started by searching the Scopus, PubMed, Medline, and Google Scholar databases online. A search strategy using PRISMA (Preferred reporting items for systematic

reviews and meta-analysis) guidelines was developed. This search strategy recognized the abstracts of published articles, while other research articles were discovered manually from the citations. Randomized controlled studies, observational studies, comparative studies, case series, and case reports were evaluated for eligibility. There were a total number of 78 articles (26 case reports; 24 cases series; 28 original articles) (Figure 1). This paper focuses only on the Pathophysiology of BPPV. This review article describes the pathophysiology of BPPV and different possible etiological factors. This review article provides a better understanding of the pathophysiology of BPPV. It will also serve as a catalyst for additional study into the pathophysiology of BPPV which will help with its appropriate management.

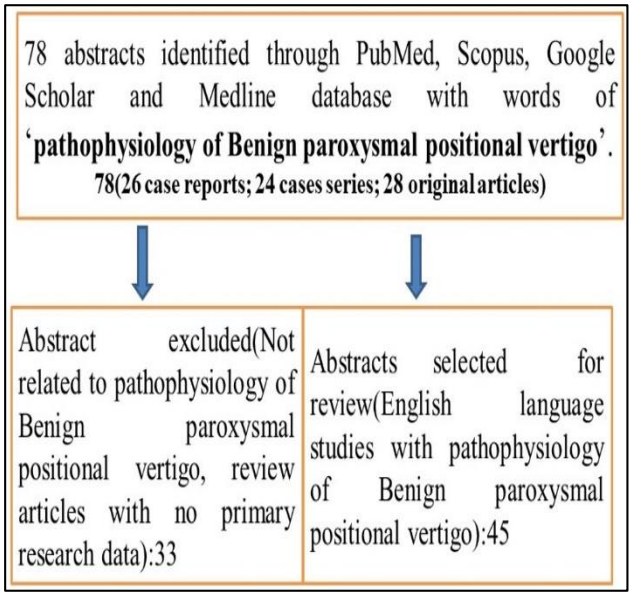

Figure 1: Flow chart of methods of literature search.

BASIC PATHOPHYSIOLOGY FOR BPPV

BPPV is the most common etiology for recurrent attacks of vertigo and is caused by abnormal stimulation of the cupula by free-floating otoliths (canalolithiasis) or otoliths that otoliths that have adhered to the cupula (cupulolithiasis) within any of the three semicircularcanals.[5] Schuknecht and Ruby demonstrated basophilic deposits in the cupula of the downmost posterior semicircular canal crista of the temporal bones from individuals who experienced BPPV, these deposits were thought to represent degenerated otoconia probably originated from the utricular macula.[6] It was hypothesized that these deposits at the cupula converted the sense organ into a gravity receptor that was activated during the Hallpike maneuver. There was the relief of vertigo and nystagmus after selecting the denervation of the posterior semicircular canal sense organ of the downmost labyrinth.[7] Parnes and McClure explained that free-floating particles could be seen in the limb of the posterior semicircular canal in patients undergoing occlusion of this canal for BPPV.[8] The fixed and free-floating types of deposits in the posterior semicircular canal were called by the terms cupulolithiasis and canalolithiasis.

ETIOPATHOLOGICAL FACTORS

Aging

Aging is an important risk factor for utricular dysfunction in idiopathic BPPV.[9] A study revealed that occurrence of otoconial degeneration occurs with aging.[10] Ag age advances, the incidence of BPPV is higher and it is rarely documented in children.[10] One large study showed that 72.6% of patients with BPPV presented with recurrent attacks, and this incidence was higher in older and female patients.[11] The lower incidence of BPPV in children than adult age group suggested that the accumulation of cupular deposits could be due to aging for the vestibular labyrinth. The average age of onset of the first episode in BPPV is more than 50 years.[11] The residual symptoms like dizziness are more common after otolith repositioning in the elderly age and the recurrence rate is very high.[10] This may happen due to otoconia fragmentation secondary to the aging process. However, this process is multifactorial. Although the concept of degenerated otoconia transforming the posterior canal crista into a gravity-sensitive sense organ has received popular support, several series of temporal bone series have shown similar deposits in normal temporal bones, suggesting these are normal changes in the aging labyrinth.

Trauma to the inner ear

Trauma is a common etiology for the development of BPPV. One study showed a clear association between trauma and BPPV where 23.4% of the patients with traumatic events developed BPPV.[12] Another study showed an association of BPPV in patients' dental surgery.[13] One study was on football players who reported BPPV during follow-up in a training program.[14] The common types of traumas causing BPPV to include motor vehicle crashes, falls, stapes or temporal bone surgery, and head trauma.[12] Electrical stimulation or acoustic trauma, pressure intense physical activity, and mechanical injury can result in otoconial dislodgement.[15] BPPV due to trauma may involve multiple semicircular canals and need repeated maneuvers.[15]

Infections and vestibular neuritis

Viral infections of the inner ear or vestibular nerve may play an important role in the pathogenesis of BPPV. The incidence of BPPV after vestibular neuritis is not uncommon.[16] There are higher serological values for herpes virus, Epstein-Barr virus, adenovirus, and cytomegalovirus in patients of BPPV than in thecontrolgroup.[16] The occurrence of BPPV due to vestibular neuronitis is often associated with a lower age at onset, more common involvement of the posterior

semicircular canal, and a higher chance of weakness.[17] The evidence of infection-related otoconial detachment required histopathological confirmation in animal studies.

Migraine

Patients with migraine are prone to the development of BPPV.[18,19] One cohort study reported a higher incidence of BPPV in patients with migraine.[20] One study reviewed residual dizziness and recurrence in patients with BPPV with or without migraine. There was no significant difference in the number of maneuvers required to achieve recovery and stated that a direct etiopathological link between migraine and BPPV is unlikely.[21,22]

Meniere's disease

Several reports are showing an association between Meniere's disease and BPPV. Meniere's disease and BPPV may show common pathological ground because of the high incidence of Meniere's disease and BPPV in the same ear.[23] The incidence of BPPV in Meniere's disease greatly varies from 0.5% to 44.0%.[24]

Osteoporosis and vitamin D deficiency

The association between vitamin D levels and BPPV is evolving. Otoconia contains calcium carbonate crystals, which are connected to saccular and utricular hair cells with protein fibers. The receptors of vitamin D regulate the transportation of calcium through epithelial channels.[25] So, the deficiency of vitamin D may affect the structure of otoconia and its integrity. The prevalence of lowered bone mass density among patients with BPPV is 81%.[26] A low serum vitamin D is an important risk factor for the development of BPPV.[27] The more severe symptoms, longer duration and lower success rate of repositioning maneuvers, and higher recurrence rate in patients of BPPV occur with vitamin D deficiency.[26] The vitamin D levels are low in postmenopausal women with BPPV.[28] A low level of vitamin D is associated with osteoporosis and treatment of osteoporosis can provide protection to BPPV and decrease the incidence of recurrence.[29] The rate of recurrence of BPPV in patients with increased serum vitamin D levels following replacement therapy is quite low in the follow-up period in comparison to the patients with a low serum vitamin D level.[30]

Sleeping style

Sleeping style may be closely associated with the affected side in patients of BPPV. The ear affected by BPPV has been found to be consistent with the head lying side.[31] The debris dislodged from the utricle by the influence of gravity may fall into the posterior or lateral semicircular canals of the undermost ear during sleep.[32] This is particularly found mostly in patients of posterior canal BPPV when the affected ear is down.[33] The recurrence of BPPV is high when the patient continues to sleep on the affected side. Sleeping habits may be associated with hearing impairment. Patients with profound hearing loss often lie on the ear with hearing impairment while sleeping to keep the normal hearing ear open to the environment.

Sudden sensorineural hearing loss

Idiopathic sudden sensorineural hearing loss may accompany BPPV. The incidence of BPPV in patients with sudden sensorineural hearing loss ranges from 5.4% to 12.1%.[34] One study reported that four patients with sudden sensorineural hearing loss and ipsilateral posterior canal BPPV were resolved after the repositioning maneuver.[35] Another study documented five cases of sudden hearing loss and BPPV and proposed a vascular insult as the common pathophysiology for this.[36] BPPV with sudden sensorineural hearing loss requires several sessions of a repositioning maneuver.

Diabetes mellitus and hyperglycemia

Disruption of the capillary vessels results in microvascular degeneration that causes proximal and distal peripheral sensory and motor neuropathy in patients with chronic hyperglycemia and hyperinsulinemia.[37] The incidence of cupular and free-floating deposits in the posterior and lateral semicircular canals was significantly higher in the patients with type 1 diabetes mellitus than in non-diabetes patients and this difference was associated with duration of disease rather than aging.[38] One study reported that BPPV was found in 46% of patients with type 2 diabetes mellitus compared to 37% of patients without diabetes mellitus, and 42% association between type2 diabetes mellitus and BPPV was mediated by hypertension.[39] Hyperglycemia is also an important risk factor for the recurrence of BPPV.[39]

Semicircular canal or vestibule pathology

BPPV is a self-limiting disease that often responds to positioning maneuvers.[40] One study showed the magnetic resonance imaging (MRI) of patients with atypical and intractable BPPV where authors found structural changes such as fractures or filling defects in the semicircular canals.[41] MRI is helpful to detect the micro-abnormalities of the semicircular canals in patients of BPPV with persistent symptoms. Recurrent symptoms of BPPV are associated with volumetric abnormalities of the vestibular aqueduct. The incidence of BPPV in patients of a large vestibular aqueduct is approximately 19%.[42] However, there is no evidence of pathological mechanism was documented.

Deficiency of estrogen

The chance of BPPV increases with age in both genders. However, menopausal women are particularly susceptible to BPPV.[43] Decreased levels of estrogen and progesterone can manifest disturbances in the inner ear

microcirculation in menopausal females.[44] Estrogen receptors are important in otoconia maintenance. Both sides of ovariectomized mice have decreased expression of otoconial components. The density of otoconia decreases whereas the size of otoconial increases in females with removed ovaries in rats. The ectopic debris formation in the ampulla increases under a deficiency of estrogen.[45]

Autoimmune cause

Currently, the disturbances in autoimmunity in patients with BPPV have been a subject of interest. The association of giant cell arteritis and BPPV has been documented in 20% of patients, showing an ischemic pattern of otoconia degeneration.[46] One study showed successful intratympanic methylprednisolone treatment in seven of nine patients with persistent posterior semicircular canal BPPV.[47] Another study revealed anti-thyroid antibodies in 27% of patients with BPPV and proposed the existence of immune complex-mediated inner ear disease.[48]

Neurological diseases

BPPV has been seen in patients with Parkinson's disease and multiple sclerosis.[49] However, the association between BPPV and neurological disorders is not strong enough to prove the pathological similarities. The occurrence of BPPV in patients with neurological disorders seems to be coincidental.

Cochlear implant and BPPV

BPPV also occurs in patients following cochlear implantation. There are several mechanisms have been suggested: the entry of bone dust particles into the cochlea during cochleostomy, the vibration caused when boring of the cochlea is sufficient to dislodge the otoconia into the labyrinth,and the displacement of an otolith because of the electrical stimulation.[50]

FUTURE RESEARCH

Future research will be helpful to understand the exact pathophysiology of the BPPV. The research on humans is confined to clinical observation of symptomatic patients and dissection of temporal bones, so it limits access to the inner ear in real-time. Animal model experiments are still scarce and difficult to replicate the human vestibule. One study confirmed the otoconia role in BPPV in bullfrogs, whose posterior semicircular canal or their utricle was removed. However, better models are desirable. A space traveler's brain usually no longer relies on the gravity sensation of the inner ear structures for getting information about position and motion, as they are more dependent on the visual system. Currently, no data are available for the possible occurrence of BPPV in a zero-gravity environment.

CONCLUSION

The pathophysiology of BPPV is not clearly understood. Although the idiopathic form is considered the most common type of BPPV, inner ear disorders like Meniere's disease, sudden hearing loss, and vestibular neuronitis are often associated with BPPV. Otoconial degeneration due to the aging process seems to be an important factor. However, BPPV cannot be solely explained by aging. Certain inner ear disorders are also important etiological factors for the development of BPPV. Vitamin D and estrogen deficiency should be considered when managing a postmenopausal lady with BPPV. BPPV following trauma is often clear in cases of head trauma, surgery, or road traffic accidents. BPPV may occur in combination with many pathological conditions.

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