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CASE REPORT

Benign Paroxysmal Positional Vertigo After Nonotologic Surgery: Case Series

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Abstract Benign paroxysmal positional vertigo is one of the most common types of vertigo caused by peripheral vestibular dysfunction. Although head trauma, migraine, long-term bed rest, Ménière disease, viral labyrinthitis, and upper respiratory tract infections are believed to be predisposing factors, most cases of benign paroxysmal positional vertigo are idiopathic. Ear surgery is another cause, but after non-otologic surgery, attacks of benign paroxysmal positional vertigo are rare. We describe three cases of benign paroxysmal positional vertigo attacks after non-otologic surgery (one patient after a nasal septoplasty and two patients after dental endodontic treatment) and discuss the pathophysiological mechanism of benign paroxysmal positional vertigo seen after non-otologic surgery, its diagnosis and treatment.

Keywords Benign paroxysmal positional vertigo · Dental surgery · Nasal septoplasty

Introduction

Benign paroxysmal positional vertigo (BPPV) is one of the most common types of vertigo caused by peripheral vestibular dysfunction. It is characterized by short, intense vertigo episodes associated with predominantly horizontal-rotational nystagmus. They are provoked by a quick change in head position such as lying down, rolling over in bed, bending over, or looking up [1–3]. Although head trauma, migraine disease, long-term bed rest, extended travel, Ménière disease, viral labyrinthitis, vestibular neuronitis, and upper respiratory infection are believed to predisposing factors, most cases of BPPV (50 to 70%) are idiopathic [4, 5]. Ear surgery is another causative factor in BPPV [6].

The pathophysiologic mechanism of BPPV can be caused either by canalithiasis or cupulolithiasis. Canalithiasis is most commonly accepted; the endolymph system of posterior or lateral semicircular canals is disturbed by free-floating otoliths, which detach from the utricle or saccule and accumulate in the long arm of the posterior semicircular canal. It moves with gravity. This concept was first described in 1979 by Hall, Ruby, and McClure, and the phenomenon was first demonstrated *in vivo* by Parnes and McClure in 1992 [4, 7].

Conversely, Schuknecht explained the pathophysiology of the cupulolithiasis by saying that particles detached from otoconial membrane are deposited in the cupula of the posterior semicircular canal. The detached otoconial material remains free in the utricle until it enters the semicircular canal [2, 8].

We describe three cases of BPPV after non-otologic surgery (one patient after septoplasty, two patients after dental endodontic treatment) and discuss the pathophysiologic mechanism of BPPV, its diagnosis and treatment.

Clinical Cases

Case 1

A 38-year-old man came to the endodontics clinic for tooth pain. The results of his clinical and radiologic evaluations

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established tooth decay in his left upper first molar tooth. No other systemic disorder was found that could affect his balance. Treatment of root canals was begun. We began treatment with a low-powered surgical drill. After pulp extirpation, the canals were expanded and the tooth was filled. The surgery lasted about 40 min.

While sitting up after surgery, the patients experienced intense vertigo with nausea, especially when he changed the position of his head. He sat in the dental chair and rested for 30 min, but the vertigo remained. The dentist referred the patient to the emergency department. The results of standard laboratory analyses, and his neurologic examination were within normal limits. His oculomotor examination was normal. No spontaneous nystagmus was observed. The Dix–Hallpike test was performed and during the left-sided swing, the patient experienced vertigo and rotatory nystagmus was observed. The nystagmus had a 4- to 7-second latency and lasted $\sim$25 s. The Epley maneuver with mastoid oscillation was performed on the left side, and he was sent home. After the procedure, the patient was advised to avoid moving his head abruptly, to sleep in a slightly elevated position, and to avoid turning during sleep toward the affected ear for 48 h. A cervical collar or medication for BPPV was not used. After the first Epley maneuver, the patient was asked to revisit our clinic in 3 days. The results of a control Dix–Hallpike test were negative, and the patient was symptom-free.

Case 2

A 44-year-old healthy woman came to the endodontics clinic with tooth pain. After clinical and radiographic examination, tooth decay was established in right upper first and second molar tooth. Like first case, a low-powered surgical drill was used to preserve healthy tooth tissue. The surgery lasted about 35 to 40 min. After the procedure, the patient was sent home. Her husband called us after 2 or 3 h and he said that his wife had intense vertigo and nausea. The patient was referred to otolaryngology department for diagnosis and treatment of vertigo. The history of central nervous system or otological disease was negative in her past medical history. There was no systemic disease like hypertension, anemia, increased cholesterol level, coronary artery disease or diabetes mellitus. The otological symptoms as hearing loss, tinnitus or aural pressure were established. A Dix–Hallpike test was done. During the right swing, the patient had severe vertigo, and rotatory nystagmus started 4 to 6 s after and was observed for 25 to 30 s. An Epley maneuver was performed on the right side. After the procedure, the patient was advised to avoid moving her head abruptly, to sleep in a slightly elevated position, and to avoid turning during sleep toward the affected ear for 48 h. A cervical collar or medication for BPPV was not used. The results of a control Dix–Hallpike test were negative.

In both cases, a low-powered drill was used for endodontic treatment. This drill is used to keep the tooth tissue healthy, but when it touches the tooth, high vibration would be caused in the tooth and the maxilla.

Case 3

A 30-year-old female nurse came to the otolaryngology clinic with a nose obstruction since 2 to 3 years. On examination, we found she had a deviated septum and bilateral inferior turbinate hyperplasia. The patient was in good physical health. She was taking no medications. With the patient under general anesthesia, she underwent a nasal septoplasty, and radiofrequency was applied bilaterally to the inferior turbinate. The day after the operation, she had vertigo and nausea. There was no hearing loss, tinnitus or vomiting. The physical and neurological examinations were normal. No spontaneous nystagmus was noted. The results of a Dix–Hallpike test demonstrated vertigo, and torsional nystagmus was noted when she hung her head to the right. The canalith repositioning maneuver with mastoid oscillation was done on the right side. After the procedure, the patient was advised to avoid moving her head abruptly, to sleep in a slightly elevated position, and to avoid turning during sleep toward the affected ear for 48 h. A cervical collar or medication for BPPV was not used. On follow-up 3 days later, she felt better. The results of the Dix–Hallpike maneuver were normal. None of the patients re-experienced BPPV during 1-year follow-up.

Discussion

Although BPPV is usually idiopathic, cases have been discovered after traffic accidents, head trauma, otologic surgery, or other surgical interventions with prolonged bed rest. More recently, BPPV has been reported as a complication of surgical procedures involving the cochlea, such as a stapedectomy, a stapedotomy, and a cochlear implant. Atacan and associates found a 6.3%, and Magliulo and associates found an 8.5% incidence of BPPV after stapedectomy [6, 9]. During these surgical procedures, the occurence of BPPV could be explained in 2 different ways by the pathophysiological mechanisms in the literature—direct trauma or indirect trauma (vibration induced by the drill) [5]. The tip of the piston could be affected by direct trauma. The vibration of the drill on the cochlea would be sufficient to dislodge several otoconia into the labyrinth, where they could cause canalolithiasis.

The vibratory trauma affecting the cochlea during use of the drill plays a fundamental role in developing paroxysmal

vertigo in patients with dental surgery. The vibrations involving the cochlea are sufficient to dislodge otoconia, as reported in the case of a dental implant, performed with the use of osteotomes [5]. In our two patients with endodontic treatment, we used the low-powered drill.

Indirect trauma on the posterior labyrinth is linked with use of either a drill or a hammer and a chisel on the maxilla. Vibrations are propagated throughout the bone structures, eventually reaching the posterior labyrinth. At this level, mechanical energy would travel through endolymphatic fluids or bone eventually leading to macular trauma. The membranous structures of the inner ear, which are contained in bony chambers, are particularly vulnerable to traumatic lesions owing to the traveling of a mechanical wave. Even mild trauma, when caused by rotating structures whose vibrations are prolonged, can damage semicircular canals. The vibrations dislodge otoliths, which then enter canal causing BPPV [10, 11].

Dieago and associates think that use of the bone expansion technique with osteotomes in dental surgery can increase the incidence of BPPV. To prevent this, they recommended use of a surgical fraise in combination with osteotomes [2]. We used a dental drill for the surgery on our patients, but BPPV was seen.

Su and associates defended a theory that percussive force may detach otoliths from the utricle or saccule of the vestibular system in the inner ear. The posteriorly displaced otoliths may then induce BPPV. During surgical positioning of the patient face up with his head hyper-extended may facilitate displacement of the detached otoliths into the posterior semicircular canal [7, 11].

Kaplan and associates thought that the use of an osteotome during rhinoplasty was sufficient to dislodge otoconia and produce BPPV [8]. The presumed cause of BPPV in our third case was blunt head trauma caused by the osteotomy. Also, tilting the head, particularly in patients having a nasal septoplasty or in those who need to be intubated during general anesthesia, may cause BPPV [10, 11].

The diagnosis of BPPV is easily made by the Dix–Hallpike test, which produces vertigo and nystagmus after the patient is rapidly moved from a sitting to head-hanging position [11]. Patients with BPPV experience vertigo when moved rapidly into a supine position with the head turned, so that the affected ear is 30 to 45° below the horizontal plane. Vertigo occurs 1 to 40 s after the patient has been placed in such a position. The patient also develops a characteristic nystagmus, with the eyes directed toward the affected side. The vertigo and nystagmus disappear in 30 to 60 s [11].

Although BPPV is a self-limiting disorder and commonly resolves within a couple of months, the symptoms are unpleasant. Treatment consists fundamentally of maneuvers to restore the calcium carbonate crystals from the anomalous location in the semicircular canal to their correct place in the utricle. Advocated treatments are maneuvers of canalith repositioning. The Epley maneuver is the most common. Here, the patient is seated with the operator behind. The head is placed over the end of the table and turned to affected ear 45°. While the head is tilted downward, it is rotated 45° to the unaffected ear. The head and body are rotated until they face downward 135° from a supine position. While the head is turned to the unaffected side, the patient is brought to a sitting position. The head is turned forward, with the chin down 20°. This maneuver is repeated as necessary at weekly intervals until the vertigo symptoms have cleared and Dix–Hallpike maneuver is negative [12].

Dentists and surgeons must bear in mind that after dental treatment and surgical procedure, an episode of BPPV may occur.

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