

만성중이염 수술 후 발생한 방향변환성두위안진을 보이는 체위성어지럼증: 양성발작성두위현훈인가?

최성준, 신정은, 김창희

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Positional Vertigo Showing Direction-Changing Positional Nystagmus after Chronic Otitis Media Surgery: Is It Benign Paroxysmal Positional Vertigo?

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This case report describes a patient who developed positional vertigo after surgery for chronic otitis media on the right side. Canal wall up mastoidectomy was performed, and the stapes was moderately mobilized during removal of the inflammatory granulation tissues that were attached to it. Immediately after the surgery, positional vertigo developed. The patient showed weakly left-beating spontaneous nystagmus in a seated position. Examination of positional nystagmus revealed geotropic direction-changing positional nystagmus with a prolonged duration and weak intensity in a supine head-roll test, which may be caused by a change in inner ear fluids due to a disruption of inner ear membrane around the oval window or penetration of toxic materials into the labyrinth during surgery.

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Keywords: Postoperative vertigo; Ear surgery; Direction-changing nystagmus; Benign paroxysmal positional vertigo

INTRODUCTION

Dizziness after otologic or maxillofacial surgery is not a rare postoperative complication [1,2]. Among them, benign paroxysmal positional vertigo (BPPV) has been often reported to develop after tympanomastoidectomy [3], stapes surgery [4],

or surgical repair of superior canal dehiscence [5]. Head trauma is known as the most common cause of secondary BPPV, and surgical drilling of bone during otologic surgery may impose traumatic injury to the head. Detachment of otolith particles from the utricular otoconia and gravity-dependent movement of otolith particles within the semicircular canal is known as

a possible mechanism of BPPV.

The canalolithiasis-type of horizontal semicircular canal (hSCC) BPPV typically exhibits geotropic direction-changing positional nystagmus (DCPN) upon head turning to either side (head-roll test) in a supine position. Geotropic DCPN of hSCC canalolithiasis lasts transiently, has latency of a few seconds, and shows fatigability. On the other hand, when geotropic DCPN on a head-roll test is persistent without latency, light cupula phenomenon, which indicates lower specific gravity of

the cupula than surrounding endolymph, of hSCC has been proposed as a possible mechanism [6].

Here, we report a patient with posttympanomastoidectomy positional vertigo showing persistent geotropic DCPN on a head-roll test, and discuss the cause of this characteristic positional nystagmus.

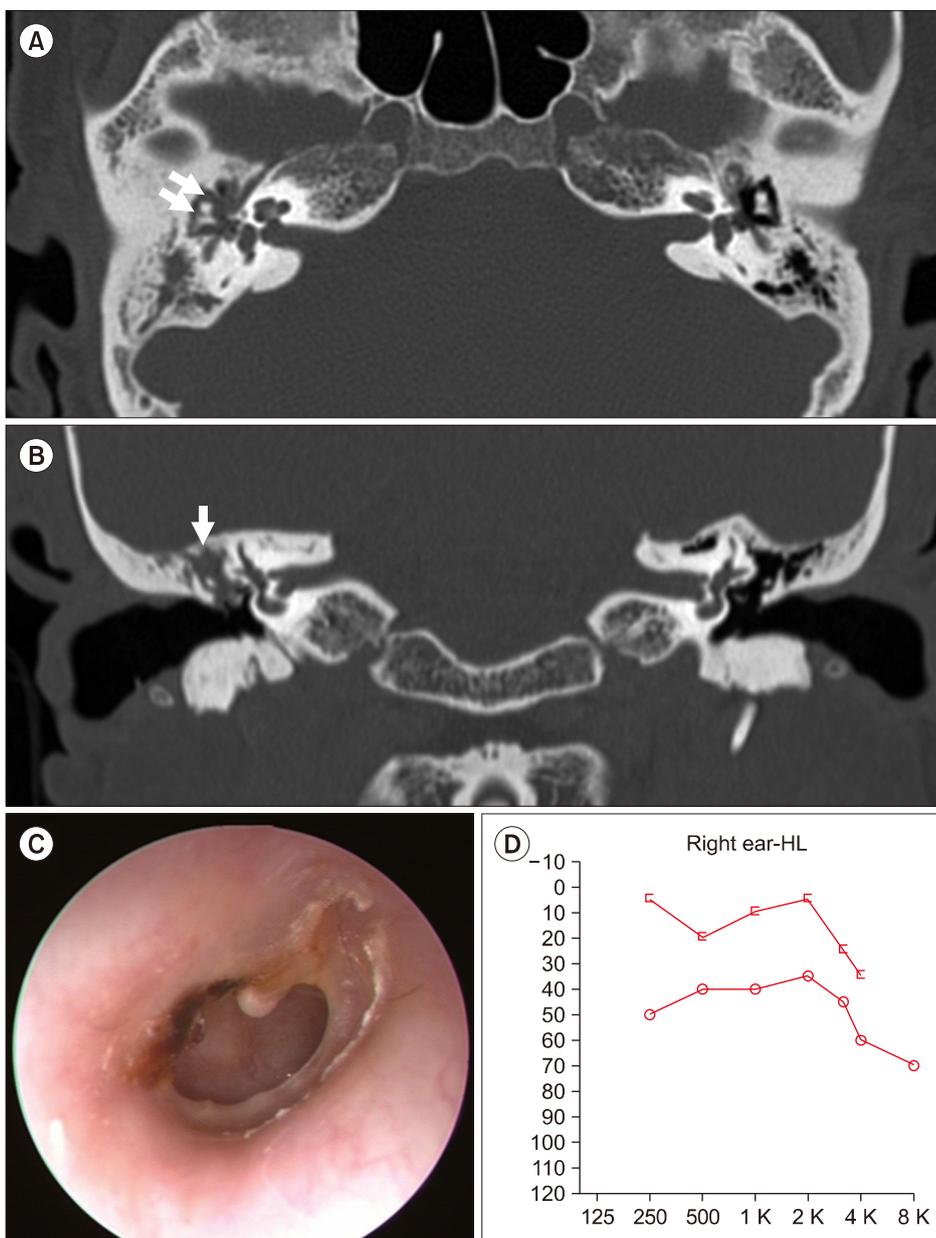


Fig. 1. Temporal bone computed tomography shows soft tissue density (arrows) in the middle ear cavity. (A) Axial view. (B) Coronal view. (C) Otoendoscopic examination reveals a large perforation in the right tympanic membrane. (D) Pure tone audiometry shows conductive hearing loss (HL) with air-conduction threshold of 40 dB (average threshold at 500, 1,000, 2,000, and 3,000 Hz).

CASE REPORT

A 55-year-old woman presented to our clinic complaining of persistent otalgia on the right side, hearing loss on both sides, and intermittent bilateral otorrhea. She reported no history of vertigo or otologic surgery. The otoscopic examination revealed a large perforation (Fig. 1C) and a moderate perforation on the right and left tympanic membrane, respectively. Air-conducted pure tone audiometry (PTA) revealed an average threshold (at 500, 1,000, 2,000, and 3,000 Hz) of 40 dB on the right side (Fig. 1D) and 40 dB on the left side. Bone-conducted PTA showed an average threshold of 15 dB on the right side and 19 dB on the left side. Temporal bone computed tomography showed a sclerotic change in both the mastoid bone and soft tissue density in the right mastoid and middle ear cavity (Fig. 1A, B). The patient was diagnosed with chronic otitis media, and intact canal wall tympanomastoidectomy was performed on the right side under general anesthesia. The mastoid antrum was filled with inflammatory granulation tissue and cholesteatoma which were attached to the ossicles was observed in the epitympanic area. Posterior tympanotomy was

performed, and a long process of the incus and a suprastructure of the stapes were found to be partially eroded. The incus was removed after dislocating the malleo-incudal joint, and the inflammatory granulation tissue and cholesteatoma was carefully extracted. The stapes was moderately mobilized during an effort for removal of the inflammatory granulation tissue that were attached to the stapes. Ossiculoplasty with a titanium prosthesis was carried out following complete removal of the inflammatory tissues.

Immediately after surgery, the patient complained of vertigo, which was aggravated by a position change. The Weber test showed lateralization to the right side, and bone-conducted PTA revealed no change in the hearing threshold compared to the preoperative threshold. She did not complain of headache or other neurological symptoms, and the head impulse test did not reveal catch-up saccade. Neurological examination exhibited no focal neurologic sign. A fistula test could not be performed because the patient's ear was packed with gauze. Weak spontaneous nystagmus beating toward the left side was observed in the sitting position. When the patient bent her head forward at 90° (bowing) in a sitting position, weak right-beating nystagmus was persistently observed (Fig. 2A;

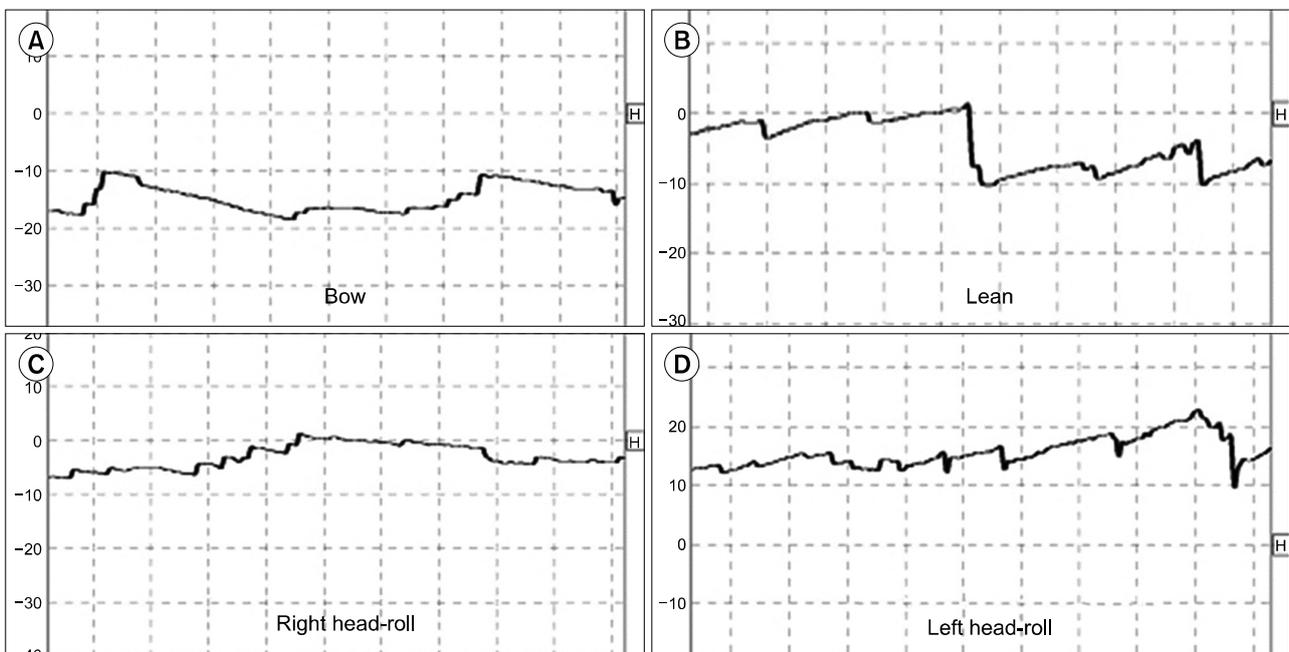


Fig. 2. Change in the direction of horizontal nystagmus at different head positions. Right-beating horizontal nystagmus is observed in bowing position (A), and right head-rolling in a supine position (C). Left-beating spontaneous nystagmus is observed in leaning position (B), and left head-rolling in a supine position (D).

Supplementary video clip 1). Weak left-beating nystagmus was persistently observed in the leaning or supine position (Fig. 2B; Supplementary video clip 1). When the patient's head was turned to the right or to the left at 90° in a supine position, persistent geotropic DCPN with low intensity was observed (Fig. 2C, D; Supplementary video clip 1). A null plane, at which nystagmus ceases and the direction of nystagmus changes by further rotation [6,7], was identified when the patient turned her head slightly to the right in the supine position (Supplementary video clip 2), which led to a possible diagnosis of 'light cupula' on the right (operated) side. Canalith repositioning procedure was not performed and, the patient was prescribed vestibular suppressants for symptomatic relief. Thereafter, positional vertigo gradually decreased and persistent geotropic DCPN on a head-roll test disappeared at the fifth postoperative day.

DISCUSSION

Postoperative dizziness after middle ear surgery is not a rare complication. Development of BPPV, which is the most common cause of positional vertigo, has been reported after stapedotomy [4], stapedectomy [8], or other ear surgery using an electronic drill such as tympanomastoidectomy, cochlear implantation, canaloplasty, transmastoid facial nerve decompression and the repair of superior canal dehiscence [3,5]. The incidence of BPPV after electronic drilling of the temporal bone during ear surgery was reported as 1.03% [3], which is lower than the incidence of secondary BPPV after head trauma [9]. Positional vertigo developed within 3 days after surgery, and canalolithiasis-type of BPPV involving the contralateral hSCC was the most common type in the previous study [3]. The interesting findings were hSCC, although BPPV involving multiple canals was common, was most commonly affected (9 out of 10 patients) while posterior SCC BPPV has been reported to most commonly develop after head trauma [9], and BPPV mainly occurred on the opposite side of the operated ear (8 out of 10 patients). It was proposed that skull vibration from surgical drilling may detach otolith particles from the contralateral utricle during mastoid surgery, and the dislodged particles may easily enter the most gravity-dependent contralateral

hSCC due to the prolonged contralateral ear-down position during surgery and postoperative bed-rest period [3].

In the present study, our patient complained of positional vertigo which developed immediately after middle ear surgery, and a supine head-roll test showed geotropic DCPN. Although the amplitude of nystagmus was low, geotropic DCPN lasted persistently (longer than 2 minutes). To explain persistent geotropic DCPN in a head-roll test, the concept of 'light cupula' has been introduced [6,10-12]. However, the pathophysiology of light cupula still remains unclear. The attachment of light debris onto the hSCC cupula has been suggested as a cause of light cupula [11], but the light debris has not been identified yet. Increased specific gravity of the endolymph was proposed as another possible mechanism for light cupula [6], which has been criticized that horizontal nystagmus is mainly observed in patients with light cupula without vertical and torsional components.

It has been pointed out that determination of the duration of DCPN and identification of a null plane are important in a differential diagnosis between hSCC canalolithiasis and light cupula [6,7]. Because our patient showed persistent geotropic DCPN with a null plane on the operated side, the authors think that the condition of light cupula is responsible for the positional vertigo in this patient. The cause of light cupula after middle ear surgery is ambiguous. Penetration of toxic materials such as antibiotic solutions or bleeding into the inner ear during middle ear surgery may cause alteration within the inner ear fluids. Moreover, given that stapes mobilization may disrupt the inner ear membrane and interrupt the inner ear fluid homeostasis, stapes mobilization during the removal of inflammatory granulation tissue might have caused ipsilateral light cupula phenomenon. It was reported that persistent geotropic DCPN can be observed in patients with sudden sensorineural hearing loss with vertigo or acute otitis media complicated by serous labyrinthitis. It was hypothesized that penetration of toxic and inflammatory mediators into the inner ear might have caused the change in relative specific gravity between the hSCC cupula and the endolymph resulting in persistent geotropic DCPN in acute otitis media [13]. An animal study demonstrated that intratympanic injection of solutions with various specific gravity elicited persistent

DCPN, which might be caused by a change of specific gravity in the hSCC due to the infiltration of the solutions into the inner ear fluids from the middle ear cavity [14]. In sudden sensorineural hearing loss with vertigo, increased concentration of water-soluble macromolecules such as blood plasma proteins within the endolymph was suggested as a cause of positional vertigo showing persistent geotropic DCPN, which was supported by the findings of 3-dimensional, fluid-attenuated inversion recovery magnetic resonance imaging [15]. In the present case, we speculate that the stapes mobilization might have caused influx of toxic materials or blood contents into the inner ear fluids through the disrupted oval window membrane resulting in the condition of 'light cupula' even though the possibility of hSCC canalolithiasis cannot be completely ruled out.

In summary, we report a patient who showed the persistent geotropic DCPN in a head-roll test immediately after tympanomastoidectomy, which is caused by the condition of light cupula in the operated ear. Although the mechanism of the light cupula after tympanomastoidectomy remains to be elucidated, light cupula can be one of the causes of postoperative positional vertigo after tympanomastoidectomy.

중심 단어: 체위성어지럼, 방향변환성 안진, 만성중이염 수술, 양성발작성두위현훈

SUPPLEMENTARY MATERIALS

Supplementary video clips 1-2 can be found via <https://doi.org/10.21790/rvs.2018.17.1.23>.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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