Vertigo Associated with Otosclerosis and Stapes Surgery

Subjects: Otorhinolaryngology

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Otosclerosis is a pathological condition affecting the temporal bone, and is characterized by remodelling of the labyrinthine bone tissue through a dynamic process of osteolysis and osteogenesis. This condition progressively leads to hearing loss, tinnitus, and vertigo.

Keywords: otosclerosis ; vertigo ; dizziness

1. Introduction

The otosclerotic process starts in the anterior part of the oval window, near the fissula ante fenestram resulting in fenestral otosclerosis. The disease can progress beyond the fissula ante fenestram and extend to the pericochlear otic capsule in cochlear otosclerosis or retrofenestral otosclerosis. The advanced stages can affect structures such as round window, semicircular canals, labyrinth, or vestibular nerve endings [1].

2. Preoperative Vertigo

The incidence of preoperative vestibular symptoms in otosclerosis patients varies greatly from one study to another. Different studies have reported incidence ranging from 8.6 to 30% [2][3][4]. While the exact cause of vertigo in patients with otosclerosis is not fully understood, several factors have been proposed.

One factor is the presence of otosclerotic foci, which can affect the endolymphatic duct and sac, leading to hydrops [5]. This abnormal accumulation of fluid in the inner ear can contribute to vertigo. Temporal bone studies have shown the presence of endolymphatic hydrops (EH) in specimens with extensive otosclerotic lesions in the cochlear endosteum or in the vestibular aqueduct, obstructing the flow of endolymph and disrupting labyrinthine fluid homeostasis [5][6]. The presence of endolimphatic hydrops can be visualized on delayed three-dimensional (3D) fluid-attenuated inversion recovery (FLAIR) MRI images obtained after intravenous administration of gadolinium. Sone et al. [7] suggested that the presence of preoperative asymptomatic vestibular EH could serve as a predictive factor for postoperative complications following stapes surgery. EH located adjacent to the oval window could be a contraindication for stapes surgery. The proximity of EH to the surgical site may increase the risk of vestibular complications after the procedure, including vertigo.

Detachment of the otoconia from the macula of the utricle is another factor that could contribute to positional vertigo, and may explain anomalies observed in the ocular and cervical vestibular-evoked myogenic potentials (oVEMP and cVEMP), which are used to assess the status of the utricle and saccule, respectively [8][9][10].

The third factor is vestibular end organ and/or neural degeneration due to otosclerotic foci involving the utricular or ampullary nerve. These changes could be related to the utricular deficit and oVEMP anomalies in patients with vertigo and otosclerosis [11].

Additionally, hydrolytic enzymes originating in the otosclerotic foci have been identified in the perilymph of otosclerosis patients; these can produce vascular and neuroepithelial lesions. Moreover, the cytokines produced in these foci can cause changes in labyrinthine fluids' chemical composition and homeostasis [12].

Degeneration of receptor cells in the vestibule and changes in the nonsensory epithelium have been observed in temporal bone studies of otosclerosis patients. Kaya et al. [13] studied temporal bones harvested from patients diagnosed with otosclerosis and found a decrease in the population of vestibular dark cells and vestibular transitional cells in temporal bone specimens with endosteal involvement. The role of these cells is to maintain the homeostasis of the labyrinthine fluid

by controlling the transport of ions and water in order to prevent vestibular dysfunction. Another study by Hizli et al. [14] found that the mean density of type I hair cells in the saccule was significantly reduced in cases with endosteal involvement, suggesting that the extension of the otosclerotic foci towards the endosteum may be an important factor in the occurrence of vestibular symptoms in patients with otosclerosis. They suggested that this might explain the abnormal oVEMP and cVEMP response in patients with otosclerosis and vestibular symptoms.

Saka et al. [15] studied the vestibular-evoked myogenic potential in response to bone-conducted sound (BC-VEMP) in a group of 25 patients and showed that 9 of 10 patients with vestibular dysfunction presented abnormal BC-VEMPs. This suggests that saccular dysfunction may be involved in these patients, possibly due to saccular hydrops or the extension of otosclerotic foci to the saccular macula or saccular afferent, considering the anatomical proximity of the saccule to the oval window.

On the other hand, another study involving 27 patients with otosclerosis and vertigo reported abnormal oVEMPs in response to impulsive stimulation, suggesting pathological abnormalities related to the utricle [11]. Hayasi et al. [8] studied 35 temporal bones with otosclerosis, reporting a higher incidence of cupular deposits compared to temporal bone without otosclerosis. They suggested that the origin of these deposits was probably the otoconia from the utricle, from where they detached and migrated to the cupula of semicircular canals.

The specific manifestation of vestibular symptoms can vary from person to person, and may be influenced by factors such as the extent of otosclerotic involvement and individual differences in anatomy and physiology. A study conducted by Eza-Nuñez et al. [16] highlighted the diversity of vestibular symptoms experienced by patients with otosclerosis. Patients with otosclerosis mention vertigo or imbalance, which can manifest in different ways, including a single episode or recurrent attacks either triggered by positional changes or occurring spontaneously. In their study, positional vertigo was associated with otosclerosis in 32.5% of patients and Ménière syndrome was reported in 30% of patients. Around 27.5% of patients experienced spontaneous recurrent vertigo, approximately 7.5% of patients presented with chronic unrelapsing imbalance, and a small percentage of patients (2.5%) had acute unilateral vestibulopathy [16].

These findings suggest that multiple factors, including endolymphatic hydrops, detachment of otoconia, degeneration of receptor cells, and cupular deposits, may lead to vertigo in otosclerosis patients. Further research is needed in order to fully understand the underlying mechanisms and develop targeted interventions for vestibular symptoms in this population.

3. Immediate Postoperative Vertigo

Treatment of otosclerosis is mainly surgical, generally with good results. The most common technique is stapedotomy, a minimally invasive technique that has largely replaced stapedectomy due to having fewer complications, including vestibular disorder [2]. The surgical approach can be either classical, using a microscope, or endoscopic, and the stapedotomy can be carried out by a conventional or laser-assisted technique.

Vertigo is reported to occur intraoperatively in 2.1% of patients, mainly due to the manipulation of the footplate. It may occur due to frequent suctioning in the middle ear, and less often to a floating footplate. This is treated by reassurance of the patient and vertiginous drugs [17].

After surgical treatment, immediate postoperative vertigo can occur in a significant percentage of patients. The reported incidence of vestibular symptoms varies among studies, ranging from 3.4% to 70% [18][19][20].

Early postoperative vertigo is usually temporary, and authors report remission of symptoms in most cases after 5 to 7 days with conservative management, including medication and bed rest [21][22].

The use of a CO_2 Laser in footplate perforation has been suggested to reduce the prevalence of postoperative vertigo due to minimal mechanical trauma to the inner ear through lesser footplate manipulation [18][23].

Several factors contribute to the occurrence of immediate postoperative vertigo. A possible cause of premature vertigo could be serous and chemical labyrinthitis, which involves irritation of the membranous labyrinth, particularly the macula of the utricle located near the oval window [24]. Suction of the perilymph from the vestibule or contact of the instrument with the membranous labyrinth can trigger vertigo [25]. According to measurements taken Pauw et al., penetration of instruments or the prosthesis into the vestibule is considered less risky in the centre and lower third of the oval window [26].

Nystagmus is observed postoperatively in approximately 65.7% of patients, and may persist for over one month, as shown by Fukuda et al. [27] in a study conducted in 2021.

Singh et al. [23] used posturography to evaluate patients, and found that patients experienced vestibular deficits and increased subjective symptom scores at one week after surgery, with remission occurring after four weeks.

Assessment of cVEMP with air conducted stimuli before surgery and three months after stapedotomy showed a significant reduction in the amplitude of P1/N2 waves in patients who complained of dizziness and vertigo, suggesting a saccular lesion in these patients [28]. The reduction of air conduction (AC) and bone conduction (BC) VEMPs in patients with otosclerosis was reported by Trivelli et al. [29], with the observation that although the air conduction thresholds improved after surgery in all patients, AC-VEMP and BC-VEMP did not significantly improve in operated patients. Akazawa et al. [30] evaluated the cervical and ocular VEMPs through bone-conducted vibration before and after surgery, finding no significant changes in VEMPs in the operated ear after stapes surgery.

Postoperative vertigo following stapedotomy may be attributed to traumatization of the utricle, release of proteolytic enzymes, antigen–antibody reactions, pressure changes in labyrinthine fluids, and reduction of blood supply to the labyrinth caused by a floating footplate during the operation [24][31].

Among the three semicircular canals (SCC), the lateral SCC appears to be the most affected in both otosclerosis and after stapes surgery. SCC function can be evaluated by vHIT. Postoperative vHIT results have indicated subclinical damage to the lateral and posterior SCC. This is further supported by studies on temporal bones which revealed degeneration of the sensory epithelium in the cristae of the SCC [32][33]. Kujala et al. [34] evaluated patients after stapes surgery and found latent spontaneus horizontal-torsional nystagmus in 33% of patients on the day of surgery. The presence of this nystagmus suggests minimal impairment of the SCC.

Overall, immediate postoperative vertigo is a common occurrence following surgery for otosclerosis, though it is usually temporary and resolves with conservative management. Monitoring vestibular function through VEMPs and other tests can provide insights into the underlaying mechanisms and help to evaluate the impact of surgery on vestibular function.

4. Late Postoperative Vertigo

Late postoperative vertigo can occur following stapes surgery for otosclerosis, with a reported incidence ranging from 0.5% to 17% [25]. The persistence of vertigo beyond four weeks is observed in approximately 4% of patients, as shown by Birch et al. [35] in a study of 722 patients, while in Albera's study 17% (58/347) [25] showed changes in the caloric test even up to 15 years after surgery. A small percentage of patients (2.6%) may experience vertigo lasting over 12 months, indicating permanent postoperative vestibular hypofunction [36].

One potential cause of late postoperative vertigo is the perilymphatic fistula, which occurs due to inadequate sealing around the prosthesis at the oval window. Its incidence is variable from one study to another, ranging from 1.3% to 10% [37][38][39][40]. Pedersen et al. [37] suggested that the cause may be inadequate sealing around the prosthesis in the oval window. A systematic review of the results and complications of stapes surgery confirms that perilymphatic fistula is a rare complication of stapes surgery and represents approximately one third of surgical revision cases [41]. Although its existence has been highlighted intraoperatively in only a few cases, the correlation between filling with tissue or fibrin glue and remission of symptoms suggests that the perilymphatic fistula is often underestimated [42].

The perilymphatic fistula can persist postoperatively if the hole around the prosthesis has not been closed, or may appear later if the graft or prosthesis moves as a consequence of increased pressure due to coughing or sneezing. Usually, complaints involve fluctuating hearing loss and vertigo, and the audiogram indicates deterioration of cochlear function [43]. Incidence can be reduced in the case of stapedectomy by placing a graft over the oval window [44].

Due to the risk of meningitis and hearing loss, the presence of perilymphatic fistula represents a serious complication. If a perilymphatic fistula is suspected and the symptoms do not improve with treatment, surgical exploration of the ear is necessary in order to close the fistula with a soft tissue graft. Persistence of the fistula can lead to irreversible hearing loss and persistence of vertigo [45].

According to Nakashima et al., the incidence of perilymphatic fistula can reach 22% in patients in whom the obliteration was performed with gelfoam and 4% in those in whom the obliteration was performed with tissue [46]. An older study comparing gelfoam, fat tissue, and fascia showed an incidence of perilymphatic fistula of 3.5% in case of gelfoam, 1.9% in

case of fat tissue, and 0.6% in the case of fascia [47]. According to Lim et al. [48], from an auditory point of view fatty tissue is to be preferred in stapedotomy and fascia in stapedectomies.

Other causes of postoperative vertigo include irritation produced by a protracted prosthesis or a displaced one. Symptoms intensify when moving the head or during the Valsalva manoeuvre. The patient may experience dizziness related to hiccupping, burping, yawning, popping of the ears, and specific acoustic stimuli [49].

Reparative granuloma, which is a pyogenic inflammatory reaction, autoimmune or allergic reaction, or exuberant healing response, can occur in approximately 0.1% to 18% of cases after stapedectomy or stapedotomy [50][51][52][53]. Reparative granulomas occur more frequently after stapedectomy, and are characterized by sensorineural hearing loss along with vertigo. Typically, reparative granuloma manifests 7 to 15 days after surgery [54].

Persistent late vertigo can be due to bone fragments entering the vestibule during surgery; additional causes include direct compression of the saccule due to adhesion between the prosthesis and the tympanic membrane, Eustachian tube dysfunction, and Tullio phenomenon [55][56][57]. Stapes surgery can damage the inner ear and eventually leads to endolymphatic hydrops without decreasing the hearing threshold at low frequencies [58]. Endolymphatic hydrops (EH) can be associated with otosclerosis as a secondary condition following stapes surgery, when EH can occur immediately after the surgery or at a later time. Clinical manifestations include low-frequency fluctuating sensorineural hearing loss, episodic vertigo, tinnitus, and aural fullness. However, according to Halpin et al. [58] these symptoms are much more rare compared to the presence of histopathological findings on TB specimens of patients who underwent otosclerosis surgery.

Rarely, late-onset vertigo can be associated with pneumolabyrinth or barotrauma, a condition presented by Mandala et al. in a patient who started to have vertigo years after surgery [59]. Additionally, Gomes et al. [60] published a case report about a patient who came back 4 weeks after stapedectomy for displacement of the prosthesis and the graft. In most cases, this complication occurred a few weeks or months after the surgery. The diagnosis is based on HRCT scan showing the presence of air bubbles in the vestibule in patients with vertigo and a positive fistula test.

Several factors can contribute to prolonged vertigo, including age, sex, stapes surgery in the opposite ear, the seal around the prosthesis in the footplate, and postoperative hearing outcomes. A history of stapes surgery in the opposite ear has been identified as a significant predictive factor for prolonged nystagmus and subjective vestibular symptoms [27].

Intractable vertigo may be an indicator for revision surgery in otosclerosis cases [25]. Prompt diagnosis and appropriate management are crucial in addressing the underlying causes of late postoperative vertigo and improving patient outcomes.

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