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CASE REPORT

Dead Ear Following Stapedotomy: Case Report and Literature Review

George Psillas, Iosiz Vital, Elena Beretouli, Konstantinos Markou, Jiannis Constantinidis, Victor Vital

1st Academic ENT Department, Aristotle University, AHEPA Hospital, Thessaloniki, Greece (GP, IV, JC, VV)
Pathology, Aristotle University, AHEPA Hospital, Thessaloniki, Greece (EB)

Our aim was to report a case of dead ear after stapedotomy due to reparative granuloma and to determine the incidence of other causes of dead ear following stapes surgery. A 41-year-old woman underwent stapedotomy on the right ear for otosclerosis. During the postoperative 5 weeks, the patient developed a progressive sensorineural hearing loss and dead ear on the operated side associated with dizziness. The revision surgery revealed reparative granuloma filling the oval window and a large part of the tympanic cavity. The pathological tissue was carefully dissected and the piston was removed, but without hearing improvement. According to the literature, there was a higher prevalence of dead ear after revision surgery (0.2 to 14.2%-mean:2.7%) then after primary stapes surgery (0.2 to 2.9%-mean:1%). In almost half of cases the dead ear was idiopathic. Otosurgeons should bear in mind that factors such as preoperative inner ear symptoms, obliterative otosclerosis, reparative granuloma, long stapes prosthesis, labyrinthitis, and intraoperative bleeding after surgical trauma may result in dead ear.

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Introduction

Dead ear is the most dreaded complication of stapes surgery. We present the case of a woman who developed a progressive sensorineural hearing loss following an uneventful stapedotomy, which gradually deteriorated in dead ear. The revision surgery revealed a reparative granuloma in the operated area. Other causes of dead ear have also been reported, with often unclear pathophysiology.

Case Report

A 41-year-old previously healthy Caucasian woman was admitted to our department with a history of hearing loss predominant at right. Otoscopic examination was normal. Rinne was negative in both sides, and the Weber lateralized to the right. A pure tone audiogram disclosed a 40-dB conductive hearing loss on the right ear and 30-dB on the left ear. Tympanometry, performed using a 226-Hz probe tone, showed a type A tympanogram result bilaterally. The stapedial reflex was absent in both sides. The most probable diagnosis was otosclerosis and the patient was prepared for surgery on the right side.

At surgery, the usual steps were followed as in routine way. Thus, following an endaural incision, the tympanomeatal flap was elevated, and the bone covering the oval window was removed with a sharp curette. The palpation of incus and stapes verified the presence of otosclerosis. The incudostapedial joint was disrupted and the stapedial tendon was cut. Then, the stapes supra structure was removed and a small opening was created with perforators in the centre of the posterior part of the footplate. A Schuknecht Teflon piston prosthesis (4.5 X 0.6 mm) was placed in the fenestra and crimped to the incus. No gelfoam or fat was put in the oval window around the prosthesis..

Corresponding address:
George Psillas
1st Academic ENT Department, AHEPA (The American Hellenic Educational Progressive Association) Hospital,
Aristotle University of Thessaloniki, 1, Stilponos Kyriakidi St., 546 36 Thessaloniki, Greece
Phone: +302310994762 • Fax: +302310994916
E-mail: psill@otenet.gr

The first post-operative day, although the Weber test was deviated to the right, the patient was suffering by a continuous sensation of dizziness and unsteadiness; a nystagmus beating to the operated side was observed, which changed direction beating to the healthy side on the following days. Ten days later, the ear canal package was removed. The otoscopic examination revealed an erythematous tympanic membrane and antibiotic drops were used for local treatment; the tonal audiogram showed mixed hearing loss on the operated ear at 40 dB level. The following five weeks the patient developed a progressive hearing loss on the same side accompanied with dizziness, despite oral antibiotic and steroid treatment; the tonal audiogram showed a conversion of the mixed to purely sensorineural hearing loss from 40 dB level to progressively profound hearing loss (dead ear). The patient was readmitted to the department and a revision of the stapedotomy revealed abundant granulation tissue filling the entire oval window enclosing the stapes piston and extended up to the incus and around on the mucosa of the tympanic cavity. The pathological tissue was carefully dissected and the piston was removed. Histology of the tissue was compatible with granuloma including inflammatory cells and fibroblasts (Figure 1).

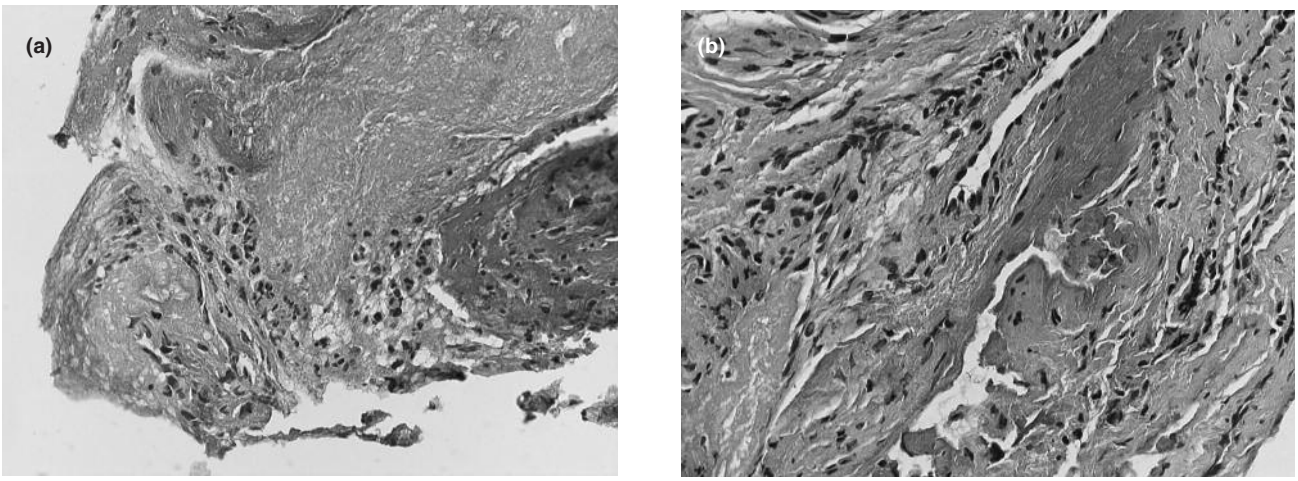

Figure 1. (A-B). Histopathologic specimens of the reparative granuloma from the oval window showing a) inflammatory cells and b) fibroblasts.

Three months later, the patient was free from dizziness, and the dead ear did not recover.

Discussion

Reparative granuloma was first described by Harris and Weiss in 1962 as a complication following stapes surgery. It constituted an excessive inflammatory reaction around the prosthesis and the oval window, such as shown in our specimen composed by inflammatory cells and fibroblasts. The incidence of reparative granuloma after stapedotomy varies from 0.07 to 5%.[1] In case of reparative granuloma the hearing after stapes surgery is improved, but gradually or suddenly is worsening in the 1st to 6th postoperative week.[2] Hearing loss is often associated with vertigo, although is some reports vertigo was reported to be the predominant symptom.[3]

The pathogenesis of reparative granuloma remains obscure. The hypothesis of a foreign body reaction to fat or gelfoam has been mainly suspected when these materials were used as an oval window seal.[4] Teflon[5] or gold piston[1] prosthesis has been reported to be an etiologic factor of reparative granuloma formation. Recently, reparative granuloma was interestingly attributed to occur as the result of persistent perilymphatic leak (perilymphatic fistula) irritating the middle ear mucosa after stapes surgery.[6] It has also

been mentioned that the inner ear symptoms appear when the reparative granuloma extend into the vestibule and not when they limited in the oval window region or in the middle ear cavity.[7] Finally, local autoimmunologic process has also been reported to be implicated in the pathogenesis of the reparative granuloma.[2,3]

The management of reparative granuloma is still controversial. According to a survey for reparative granuloma,[3] over half of the members of the American Otological and Neurotology Societies (53%) recommended immediate revision surgery and 42% preferred delayed revision surgery; 58% of surgeons removed and replaced the prosthesis, and 36% of them only removed the reparative granuloma. The immediate revision surgery has been supported to prevent sensorineural hearing loss[3] although this is contraindicated to cause permanent hearing loss in a fragile ear.[9] Although the conservative therapy including corticosteroids and antibiotics has been supported,[3] in our case it did not prevent the hearing deterioration into dead ear.

A review of the literature was attempted in order to determine the incidence and possible causes, including granuloma, which may result in dead ear after stapes surgery. Dead ear as a complication may occur after primary stapes surgery (Table 1) or after revision surgery (Table 2). According to the literature, the incidence of dead ear after primary surgery ranged from 0.2 to 2.9% (mean 1%)[10-14] and after revision surgery from 0.2 to 14.2% (mean 2.7%).[15-30] The higher prevalence of dead ear after revision surgery reflects the greater risk of damage to the inner ear compared to the primary operations.[10] The reported causes of dead ear after stapes surgery included preoperative inner ear symptoms such as vestibular symptoms or sensorineural hearing loss (13 cases), obliterative otosclerosis (8 cases), reparative granuloma (7 cases), long stapes prosthesis (2 cases), infection (2 cases), intraoperative bleeding (2 cases), delayed sudden dead ear one year after revision surgery (1 case) and idiopathic in 26 (42%) cases. The majority of these reports did not mention details about time onset of dead ear (immediate or delayed), preoperative symptoms or surgical findings.

Our patient presented inner ear symptoms after the primary stapedotomy that would be the expression of perilymphatic leakage or fistula following the stapes surgery. During stapedotomy (or stapedectomy) a perilymph fistula is by definition created; perilymph leakage persists until the oval window mucoperiosteum results in the production of an inflammatory repair envelop around the prosthesis sealing the opening into the oval window.[34] If the fistula does not close spontaneously or during revision surgery there is a great risk of progressive hearing loss and possibly total deafness;[35] in almost half of 49 fistula cases after stapes surgery a sensorineural hearing loss was detected.[34] It has also been supported that a small-fenestra stapedotomy was less likely to result in fistula formation than stapedectomy.[28] However, there were cases operated for otosclerosis, in which although the oval window was sealed with vein

Table 1. Incidence and possible causes of dead ear after primary stapes surgery

Author	Deaf ear n (%)	Possible causes (n cases)
Schmid & Häusler[10]	6 (2.9)	Granuloma (3)
		Idiopathic (3)
Fisch[11]	1 (0.3)	Idiopathic (1)
Van Drie[12]	NR (0.6)	Idiopathic (NR)
Szymański[13]	1 (0.2)	Long prosthesis (1)
Vital[14]	3 (1.1)	Idiopathic (3)

NR: not reported

Table 2. Incidence and possible causes of dead ear after revision stapes surgery

Author	Dead ear n (%)	Possible causes (n cases)
Haberkamp[15]	1 (3.3)	Granuloma (1)
Babighian & Albu[16]	1 (1.2)	Delayed sudden cophosis (1)
Crabtree[17]	5 (14.2)	Idiopathic (5)
Sheehy[18]	7 (3)	Repeat drill of obliterative otosclerosis (3)
		Preoperative inner ear symptoms (2)
		Idiopathic (2)
Lippy[19]	1 (0.2)	Idiopathic (1)
Cokkeser[20]	3 (5.3)	Preoperative vestibular symptoms (2)
		Long prosthesis (1)
Gros[21]	1 (1.6)	Idiopathic (1)
Bhardwaj & Kacker[22]	2 (2.2)	Obliterative otosclerosis (1)
		Idiopathic (1)
Van Drie[12]	NR (2)	Idiopathic (NR)
Schmid & Häusler[10]	2 (1.2)	Preoperative inner ear symptoms (2)
Magliulo[23]	2 (3.1)	Idiopathic (2)
Han[24]	1 (1.3)	Delayed infection (1)
De La Cruz & Fayad[25]	3 (1.4)	Preoperative moderate hearing loss (2)
		Preoperative cophosis (1)
Palva & Ramsay[26]	1 (1.3)	Idiopathic (1)
Derlacki[27]	3 (1.3)	Preoperative inner ear symptoms (3)
Glasscock[28]	4 (2.7)	Idiopathic (4)
Vartiainen[29]	1 (2.2)	Obliterative otosclerosis (1)
Silverstein[30]	1 (2.5)	Repeat drill of obliterative otosclerosis (1)
Mann[31] a	12 (0.9)	Repeat drill of obliterative otosclerosis (2)
		Granuloma (2)
Kisilevsky[32] a	1 (0.1)	Idiopathic (1)
Leighton[33] a	6 (3.2)	Intraoperative bleeding (2)
		Granuloma (1)
		Preoperative inner ear symptoms (1)
		Infection (1)
		Idiopathic (1)

a : It was not clear whether dead ear was resulted from primary or revision surgery

NR: not reported

graft,[14,36] a postoperative sensorineural hearing loss even a dead ear was manifested.[14] Moreover, Lippy and Schuring[36] recommended the use of tissue seal across the oval window when a stapedectomy / stapedotomy was performed as they found that the rate of oval window fistulas was greater when tissue seal was not used (50% of cases versus 4%).

When perilymphatic fistula is present after stapes surgery, vestibular symptoms are also commonly

described in association with sensorineural hearing loss. Harrison et al[37] mentioned that 35% of their patients with perilymphatic fistula suffered from vertigo and 39% of their cases reported sensation of imbalance. A constant electronystagmographic finding is spontaneous nystagmus towards the non-operated side.[36] In case of persistent gusher, a more serious stapes surgery complication, a marked postoperative vertigo was also encountered.[38]

Extensive involvement of the oval window and footplate may lead to obliterative otosclerosis. When the obliterative otosclerosis is found during the primary stapes surgery, partial or complete re-closure of the oval window may occur, requiring revision surgery.[39] The repeat use of the drill in primary or revised cases of obliterative otosclerosis is a potentially dangerous procedure, which may result in severe sensorineural hearing loss or dead ear.[18] In the presence of obliterative otosclerosis, the use of hearing aid should be considered before a revision surgery could result in severe inner ear damage.[26,39]

Direct injury of the inner ear by a long piston could be a potential cause of sensorineural hearing loss and deaf ear.[13] On the other hand, during a revision stapes surgery the removal of long prosthesis also carries a significant risk of severe hearing loss or dead ear due to existing adhesions between otolithic membrane and prosthesis.[15,20] Therefore correct adjustment of the length of the prosthesis is necessary in order to avoid unfavorable movements.

Serous or suppurative labyrinthitis following stapedotomy/stapedectomy may result in dead ear although the routinely perioperative use of antibiotics. Suppurative labyrinthitis may occur within days after surgery or even after a long delay. The labyrinth is usually contaminated with bacteria coming from the tympanic cavity through the space between the footplate and the rim of the oval window, or through a subluxated footplate.[8] Belal and Ylikoski[40] demonstrated, in a temporal bone, a case of suppurative labyrinthitis with bilateral profound hearing loss and dizziness which occurred 9 years after stapedectomy in both sides; the left and right cochlea showed ossification in the basal turn and diffuse perilymphatic fibrosis.[40] Labyrinthitis is better diagnosed with magnetic resonance than CT-examination by illustrating high signal intensity of the labyrinth on non contrasted T1-weighted images.[8]

Conclusion

Dead ear, although rare, may happen especially after stapes revision surgery. Its aetiology in almost half of cases (42%) remains obscure. The otosurgeon should keep in mind that factors such as preoperative inner ear symptoms, obliterative otosclerosis, reparative granuloma, long stapes prosthesis, labyrinthitis, and intraoperative bleeding after surgical trauma have a greater risk for dead ear following stapes surgery.

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