Bilateral Hearing Loss Following Unilateral Stapes Surgery

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ABSTRACT

Otosclerosis is a common cause of conductive hearing loss. It is surgically treated with stapedectomy or stapedotomy. Sensory-neural hearing loss is an uncommon but known complication of this surgery. A 25-year-old Iranian female with bilateral otosclerosis underwent right stapedotomy and developed bilateral sensorineural hearing loss a few days after the surgery, which recovered with appropriate treatment. The possible causes and their clinical courses are discussed in this study. Sensorineural hearing loss after stapedotomy has many different etiologies. Generally, the causes which are not directly related to the surgery should also be considered.

Keywords: Otosclerosis, Hearing loss, Trauma.


INTRODUCTION

Otosclerosis is one of the most common causes of conductive hearing loss in people 15-50 years of age. The disease now refers to the bony ear capsule and may result in progressive and conductive hearing loss, mixed hearing loss and also absolute sensorineural hearing loss in some rare cases. Otosclerosis is a hereditary disease which is transmitted, in an autosomal dominant form with incomplete penetrance (1). Bilateral otosclerosis has been observed in 60% of patients. Otosclerosis occurs most commonly among Caucasians with an incidence of 1% followed by Asians at 0.5% (1). Progressive and conductive hearing loss particularly in low frequencies (500-2000 Hz) which may sometimes occur with sensorineural hearing loss has been identified as the main clinical finding of otosclerosis (1). The diagnosis is made based on the clinical history, the physical examination, and tests such as pure tone audiometry, voice audiometry and immittance testing. Imaging may also provide relevant diagnostic information. Computed tomography (CT) is the method of choice (1). The treatment may be medical (oral anti bone remodeling drugs) or surgical (stapedotomy or stapedectomy). Personal sound amplification devices (PSAD) are a further option, particularly in patients with surgical contraindications (2). The most bothersome complication of stapes surgery is sensorineural hearing loss that occurs in less than 1% of cases and the pathogeneses are varied. Usually, the pathogenesis of sensory-neural hearing loss (SNHL) can be evaluated by clinical examinations and imaging (3-5). The aim of this study is to report a rare case of bilateral hearing loss following unilateral stapedotomy and to review the literature on this theme.

Case Report

A white Iranian female, aged 25 years, presented to otolaryngology department of Loghman Hakim hospital with complaining of bilateral progressive hearing loss for 2 years; there were no vestibular or tinnitus symptoms. The patient reported no episodes of otitis during the past years. The patient had no family history of otosclerosis. The otologic exam revealed a normal tympanic membrane at both sides. In pure tone audiometry, air-bone gap was 40 dB on the right side, and 30 dB on the left. Average hearing level of the right ear was 46 dB and the left ear was 43dB. Imittance testing revealed decreased bilateral tympanic membrane complacency and bilateral absence of the stapedial reflex. CT scan before the surgery showed no significant abnormalities (Fig1, 2). Stapedotomy of the right ear was performed and Teflon prosthesis inserted. Surgical findings included a restricted movement of

Figure 1. Post-operative high resolution CT scan. Top: Coronal, bottom: Axial. Note the normal
which revealed no significant findings (Figures 1 and 2).

The patient underwent treatment with intravenous Methyl Prednisolone (500mg/daily) for 3 days and intra tympanic injection of Dexamethasone in both ears every other day for three times. This treatment was successful and the hearing thresholds returned to the initial levels one week after the initiation of the treatment (Figure 3).

**DISCUSSION**

Sensorineural hearing loss (SNHL) occurs in less than 1% of stapedectomized ears. It may arise because of many reasons. This low percentage remains as an irreducible minimum even among the most experienced and competent surgeons. The etiology has been hypothesized; however, the actual cause remains unknown. The pathogeneses are varied. Bulging of the prosthesis into the
vestibule, trauma to the membranous labyrinth, postoperative granuloma, oval window fistula, labyrinthitis, and endolymphatic hydrops are the primary known etiopathogenic factors (2, 8). Systematic surgical revision is the method of choice for identifying some of these underlying causes of SNHL. High resolution CT can depict a malpositioned prosthesis, postoperative middle ear granuloma, perilymphatic fistula, and inner ear malformations. Nonsuppurative labyrinthitis is relatively common in the immediate postoperative period, and may cause transient SNHL, (7). As mentioned before, HRCT and MRI were performed in this case and no abnormality was detected. Endolymphatic hydrops may occur due to otosclerosis or as two separate disease entities. Stapedectomy in the presence of uncontrolled Ménière’s disease can potentially result in a dead ear and should be avoided (9). Our patient had no history of Ménière’s disease and there were no signs or symptoms in favor of the disease in the past few years. Perilymphatic fistula is a rare complication after stapes surgery with an incidence ranging from 3 to 10% (7). Patients complain of a mixed conductive sensorineural hearing loss with some vague unsteadiness and, rarely vertigo. It may occur between weeks to years following surgery and persist until an endosteal membrane forms at the oval window. CT scan studies may show fluid in the middle ear, depending on the pressure of the vestibule. This finding may suggest perilymphatic fistula if associated with a pneumolabyrinth which, is the presence of air bubbles on the tip of the prosthesis (2, 7). Our patient had no vestibular complaint and her normal CT excluded perilymphatic fistula. Serous labyrinthitis is common after stapedectomy due to inner ear inflammation. Patients may exhibit mild unsteadiness, positional vertigo, and/or a slight decrease in high-frequency hearing. The above symptoms typically fade within several days to weeks (9). Our patient had neither the clinical findings nor the clinical course compatible with a serous labyrinthitis. Postoperative reparative granuloma has been recognized as a cause of sensorineural hearing loss after stapedectomy (2). Patients present with initial improvement in hearing followed by a gradual or sudden deterioration 1 to 6 weeks postoperatively. Vertigo can also occur and a reddish discoloration in the postero-superior quadrant of the tympanic membrane is sometimes evident (2, 9). None of these signs and symptoms matches the present reported case. Suppurative labyrinthitis is a severe complication of stapes surgery. It occurs within days after the surgery or even after a long delay (2, 7). The clinical manifestation of our patient was not consistent with the ones of suppurative labyrinthitis. There are several possibilities to explain the contralateral hearing loss after stapedectomy. It could be secondary to idiopathic sudden sensorineural hearing loss. This disease has an annual incidence of approximately 0.001% (10). Progressive cochlear otosclerosis in the contralateral ear is also a possibility (10). Willis studied 300 stapedectomy patients and found that over a minimum of 10 years, sensorineural hearing loss developed in the operated ear in 19%. In 5 of the 300 patients (1.6%), sensorineural hearing loss developed in the contralateral ear in the same time period (11). Another study demonstrated a similar decline in bone conduction in the non-operative ear after stapedectomy with at least a 5-year follow up. The contralateral ear showed a significantly greater annual decline in bone conduction thresholds than the ear that underwent stapedectomy (10). Sympathetic cochleolabyrinthitis (SC) is a very uncommon occurrence after stapedectomy. SC develops from an activation of immune response to exposed inner ear antigens as a result of trauma or surgery (10, 12, 13). Richards ML et al., have proposed that revision stapedectomy rarely is associated it hearing loss in the contralateral ear and that may be due to sympathetic cochleolabyrinthitis (10). There is no report of SE happened after primary non-complicated stapedectomy. Furthermore, the reported course of this potential immunologic process is not compatible with one of our patient. Any asymmetric progression of a sensorineural loss after stapes surgery should provoke suspicion of a pathologic process. These include perilymphatic fistula, labyrinthine otosclerosis or ischemia and acoustic neurinoma. In coexistence of an acoustic neurinoma with otosclerosis, the diagnosis of acoustic neuroma is usually delayed because more common causes of sensorineural hearing loss associated with otosclerosis or its surgery are usually considered (3, 13).
Our patient did not have the characteristics of any previous mentioned complications. The only matched scenario is occurrence of a bilateral idiopathic sensorineural hearing loss. The absences of the concomitant clinical and imaging finding as well as the dramatic response to steroid are in favor of this diagnosis.

CONCLUSION
Sensorineural hearing loss after stapedectomy may have many potential causes. Nonetheless, in rare cases, which cannot be matched with the first line possible etiologies, the other causes of SNHL, which may not necessarily be directly related to the surgery, must be taken into account. This is important because a prompt and urgent treatment is often necessary to achieve a better clinical outcome.

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REFERENCES


