Acute Hearing Loss in the Contralateral Ear after Vestibular Schwannoma Removal

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Hearing loss in the contralateral functioning ear is a rare and distressing complication after vestibular schwannoma removal. Various possible mechanisms have been proposed, however, the etiology of hearing loss is not clear. Fortunately, this is an extremely rare occurrence, sporadic case reports have appeared in the literatures. We report two cases of acute contralateral hearing loss after vestibular schwannoma removal and discuss the possible mechanisms of the phenomenon. Although permanent deafness may result, in our cases, the hearing loss was temporary, returning to near preoperative level within one month. The etiology of hearing loss in one case is thought to be cerebrospinal fluid leakage. However, in the other case, the cause of hearing loss is not clear. A better understanding of these events may lead to preventive measures to avoid contralateral hearing loss after vestibular schwannoma removal.

**KEY WORDS**: Acute hearing loss · Contralateral ear · Vestibular schwannoma.

Introduction

Hearing loss in a contralateral functioning ear is a rare but distressing complication after unilateral vestibular schwannoma removal. This is an extremely rare occurrence, and sporadic case reports documenting the sudden loss of hearing in the contralateral ear after tumor removal have been reported [8, 10, 15, 17]. Various mechanisms have been proposed for this phenomenon including meningitis, autoimmune reaction, vascular compromise, drill noise acoustic trauma, perilymph loss due to CSF drainage, and imbalance in neural control [1, 8, 15, 17]. However, to date, none of these theories has been convincingly proved.

The authors present two rare cases of hearing loss in a contralateral ear after suboccipital removal of unilateral vestibular schwannoma and briefly review the pertinent literatures.

Case Report

Case 1

A 52-year-old woman presented with a 2-year history of decreased hearing in the left ear. Before six months, the hearing was decreased to difficulty with word comprehension and tinnitus in left ear began. On examination, there were decreased auditory acuity and mild trigeminal nerve hypesthesia in left side. A preoperative audiogram showed profound sensorineural hearing loss on the left and normal hearing on the right (Fig. 1). The magnetic resonance imaging on admission revealed a left cerebellopontine angle (CPA) mass approximately 3.5cm in diameter (Fig. 2). The patient underwent a left suboccipital craniotomy and total removal of the tumor on supine position. No movement of the brain stem was observed intraoperatively and no change of blood pressure was detected. Postoperative course was uneventful until the tenth postoperative day. On postoperative day ten, the patients began to report decreased hearing in the right ear. No evidence of a CSF wound leak was found. The audiogram taken on postoperative day 11 demonstrated no measurable hearing on the operated side and a moderate to severe sensory neuronal hearing loss (SNHL) on right side. The audiograms showed 50dB Speech awareness threshold and 16% discrimination rate at 80dB (Fig. 1). Follow up brain CT was checked and there was no findings indicating brain stem shift or pneumocephalus. However, her hearing was improved progressively and nearly completely recovered at the one month after operation. The follow up audiogram was taken on one month later and it showed a 25
to 30 dB improvement from the initial postoperative audiogram. It showed 30 dB speech reception threshold and 100% discrimination rate at 70 dB (Fig. 1).

Case 2
A 52-year-old woman with a left side vestibular schwannoma presented with a 3-year history of decreased hearing on the left.

There were no associated neurological signs, symptoms, or laboratory abnormalities except decreased hearing.

The audiogram showed a moderate SNHL on the left and normal hearing on the right (Fig. 3). MR imaging demonstrated a left cerebellopontine angle mass approximately 3.5 cm in diameter, with erosion and extension of tumor into the porus acusticus.

Fig. 2. The T2-weighted axial image on admission revealed a left cerebellopontine angle mass approximately 3.5 cm in diameter (Fig. 4). The patient underwent total excision of tumor through a left suboccipital craniotomy with left hearing preservation on supine position. There were no specific signs or symptoms except a mild hearing impairment of operated ear. However, the postoperative course was complicated by a paradoxical CSF rhinorrhea on seven days after operation. The CSF leakage was managed with placement of a lumbar subarachnoid drain. One day after lumbar drain, the patient complained fulling sensation with fluctuating hearing on the contralateral ear without difficulty with communication. An audiogram demonstrated a mild decrease in the pure-tone thresholds in left ear and moderate hearing loss in right ear (Fig. 4). Follow up brain CT was checked and there was no abnormalities. After four weeks, the audiogram demonstrated no change in the operated ear. However, it showed progressive improvement and nearly normalized to the preoperative levels in the non-operated ear.

Discussion
It is well recognized that the ear contralateral to the vestibular schwannoma appears to be significantly worse than the hearing of the normal-hearing population especially in larger tumors. These abnormalities may be reversible after resection of the tumor. However, a number of contralateral hearing losses after unilateral removal of vestibular schwannoma have been reported.

Loss of hearing in the contralateral nonoperated ear is one of the most distressing complications after removal of vestibular schwannoma. A variety of theories trying explaining the cause of contralateral hearing loss have been proposed; drill noise, brain stem shift, autoimmunity, vascular compromise, imbalance in neural control of outer hair cell, perilymph loss due...
to CSF drainage. It is generally thought that drill-generated noise is transmitted via the bone, and this might be responsible for the development of contralateral SNHL after vestibular schwannoma surgery, especially using translabyrinthine approach. However, Tos et al. reported that no case of SNHL could be demonstrated postoperatively after evaluating 50 consecutive cases and concluded that the drill noise does not play a role in postoperative development of contralateral SNHL after the surgery of vestibular schwannoma.

It has been generally accepted that contralateral complications of vestibular schwannomas were associated mainly with large tumors, presumably due to brain stem shift. Combination of preoperative compression and postoperative stretching to the contralateral cochlear nerve is possibly involved in the pathogenesis of hearing loss. However, this does not appear to apply to the patients in the present series since they had relatively small tumors with a size of 3.5cm and 1.5cm. Follow up brain CT showed no findings indicating brain stem shift in our cases.

Harris et al. explained the hearing losses by proposing a condition similar to sympathetic ophthalmia. They proposed that inner ear antigens can be exposed to immune system during manipulating the inner ear and then contralateral ear dysfunction can develop. However, their analysis was performed in patients consisted of a translabyrinthine approach and a middle fossa approach. In cases using suboccipital approach, there are no manipulations and injuries to the inner ear. So we were not able to explain the hearing loss in present patients by this proposal.

Vascular compromise has been another explanation for postoperative contralateral hearing loss. Keyser et al. reported that occlusion or perfusion insufficiency of the internal auditory artery caused by brain stem shift, postoperative edema, or vasospasm are a possible explanations for sudden hearing loss.

The imbalance in neural control of outer hair cell is another explanation for postoperative contralateral hearing loss. Indeed, it is well recognized that the ear contralateral to the vestibular schwannoma appears to be significantly worse than the hearing of the normal-hearing population. Perhaps it represents the compensatory inhibition of central nervous system for a partial loss of function of the organ of Corti, afferent nerve fibers, and effferent nerve fibers. In most cases, this compensatory phenomenon is reversible after tumor removal. And a paradoxical effect, that is an increasing inhibition effect of CNS, has been thought to be another possible explanation for postoperative contralateral hearing loss.

Fig. 4. The T1-weighted axial image with contrast shows the heterogeneous enhanced cerebellopontine angle mass approximately 1.5cm in diameter.

Fig. 3. Case 2. Case 1. A: Preoperative pure tone and speech audiogram. B: Comparative audiograms a day after a sudden hearing loss. C: End stage recovery 1 month later.
Several experimental works demonstrated that the perilymphatic pressure changes synchronously with the CSF pressure via cochlear aqueduct. Thus, the lowering of perilymphatic pressure could lead to a relative endolymphatic hypertension similar to that of endolymphatic hydrops. Apparently, a series of procedure involving loss of CSF can produce a similar hearing trouble.

In case 2, the hearing loss of operated side developed immediately after tumor removal, and the loss of contralateral side developed after the lumbar CSF drain. So, we think that a persistent CSF loss via paradoxical rhinorrhea and continuous lumbar drain is the possible cause of hearing loss of contralateral side. Unfortunately, in case 1, we could not explain the hearing loss exactly by any proposal.

Conclusion

The etiology of contralateral hearing loss in one case (case 1) is thought to be CSF leakage, but in the other case, the cause of hearing loss is not clear. We think that the better understandings of exact pathophysiologic mechanism might lead to preventive measures to avoid contralateral hearing loss after vestibular schwannoma removal.

References