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Pneumolabyrinth, intracochlear and vestibular fluid loss after cochlear implantation

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ABSTRACT

The present case was a 38-year-old male who presented with progressive hearing loss, resulting in profound bilateral hearing loss. He had a past history of childhood medulloblastoma, which was treated with posterior fossa craniotomy and radiotherapy. A ventriculoperitoneal (VP) shunt was put in place to manage the hydrocephalus. Cochlear implantation (CI) was carried out on his right ear by a standard procedure. At CI activation, the electric impedance of the electrode was very high, and computed tomography revealed that there was no area of liquid density, suggesting depletion of the perilymph in the cochlea and vestibule. Eight months later, the impedance improved gradually, and the cochlea was filled with perilymph. Consequently, one of the causes of the pneumolabyrinth in the present case was that a scarred stenotic cochlear canaliculus secondary to surgery or radiation therapy might have prevented the CSF from filling the scala. In addition, it is also possible that the VP shunt might have altered the CSF pressure, leading to depletion of the perilymph.

1. Introduction

Cochlear implantation (CI) is indicated for patients with profound hearing loss who do not experience sufficient improvement in hearing with hearing aids, and is widely recognized as a safe procedure. Prior to CI surgery, it is necessary to consider the presence of any pre-existing complications, such as cochlear malformations, which might result in cerebrospinal fluid (CSF) leakage [1]. Once the CI electrode is inserted correctly into the cochlea, hearing can be improved even in patients with cochlear malformations in most cases. However, there are cases in which it is not possible to accurately predict CSF leakage using static assessment techniques such as computed tomography (CT) imaging.

Herein, we report an adult patient who experienced severe pneumolabyrinth, intracochlear and vestibular fluid loss, and depletion of perilymph after cochlear implantation resulting in a marked impediment to multiple electrode contacts in the cochlea.

2. Case report

A 38-year-old male with progressive hearing loss was referred to Shinshu University hospital for counseling on the possibility of CI. He had a past history of childhood medulloblastoma, and underwent posterior fossa craniotomy and resection of the tumor as an initial treatment at the age of 7. Tumor recurrence was later detected, and he underwent reoperation and radiotherapy at 10 years old. A ventriculoperitoneal (VP) shunt was also inserted into the right lateral ventricle to manage the hydrocephalus. At 27 years, the patient experienced trouble with the right lateral ventricle VP shunt, leading to impaired consciousness, and hydrocephalus was

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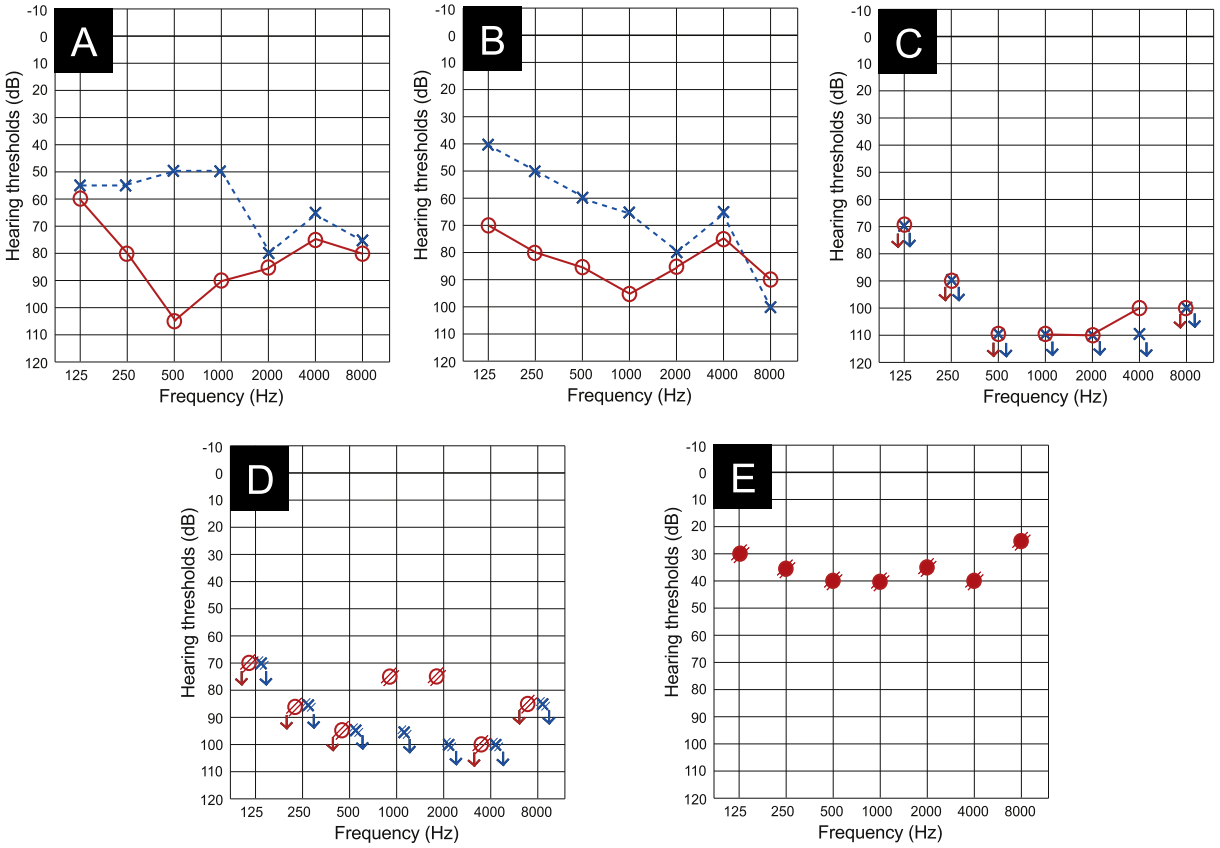

Fig. 1. Audiograms of the present case. (A) At the time of the VP shunt trouble on the right side (27 years old). Hearing loss in the right ear had already existed from childhood, and he became aware of hearing loss in the left ear after the right side VP shunt trouble. (B) At the age 30. His hearing level gradually deteriorated. (C) At the age of 37. Preoperative hearing levels. (D) Preoperative hearing levels with hearing aids. (E) Postoperative hearing levels with cochlear implant at day 257 after cochlear implantation in the right ear.

observed on MR images. Immediately after, a new VP shunt was inserted into the left lateral ventricle. He had severe hearing loss in the right ear, which appeared to have existed from childhood, and he became aware of hearing loss in the left ear after the right side VP shunt trouble (Fig. 1A). His hearing level continued to deteriorate, and he started to use a hearing aid for his left ear at age 30 (Fig. 1B); however, his hearing level further deteriorated, and the hearing aid became useless by 37 years old (Fig. 1C).

Upon visiting our hospital, his audiogram showed profound bilateral hearing loss, with the threshold for the left ear even with a hearing aid found to be inadequate for communication (Fig. 1D). The caloric test with cool water showed bilateral canal paralysis; however, he did not have any subjective symptoms of vertigo. He demonstrated slight mental retardation, gait disturbance and postural instability due to sequelae associated with the previous treatment for childhood medulloblastoma. CT imaging of the head and temporal bone demonstrated the presence of bilateral VP shunts, and soft tissue density was observed in the middle ear and mastoid cavities, which was thought to be due to the previous radiotherapy for his tumor. No abnormalities were observed in the cochlea or vestibule bilaterally (Fig. 2A). The hearing loss of shorter duration in the left ear was thought to be better with regard to CI outcome; however, if the VP shunt on the left side lay adjacent to the CI it could lead to a breakdown due to CI site infection or some other complication, perhaps resulting in hydrocephalus, which could prove potentially fatal. Additionally, the skin of his head was quite thin and stiff due to the previous craniotomy and radiotherapy. We, therefore, considered that the implant should be placed in the right rather than in the left ear for safety reasons.

CI surgery for the right ear was performed using a conventional procedure consisting of mastoidectomy and posterior tympanotomy. We implanted a MED-EL CONCERTO FLEX28 electrode through the round window membrane. Minor leakage of perilymph was observed as a small incision was made in the membrane, and the electrode was inserted carefully. The round window was completely filled with the electrode together with packing consisting of small pieces of fascia and fibrin glue. Complete electrode insertion was confirmed by X-ray images taken immediately after implantation. Electrode impedance and field telemetry measurement were within the normal range and appropriate for electrical stimulation (Fig. 3). No complications, such as fever or skin deficit, were observed after surgery.

Twenty-seven days after implantation, the CI was activated. However, the electric impedance over most of the electrode contact area was very high (Fig. 3), and we were not able to activate this CI. CT scans revealed that although the electrode was inserted in the correct position in the cochlea, there was no area of water density, suggesting depletion of the perilymph in

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