

Successful Cochlear Implantation in a Patient With Bilateral Progressive Sensorineural Hearing Loss After Traumatic Subarachnoid Hemorrhage and Brain Contusion

Chisato Fujimoto, MD; Ken Ito, MD; Shingo Takano, MD;
Shotaro Karino, MD; Shinichi Iwasaki, MD

Objectives: We address the proper indications for cochlear implantation for profound deafness with possible retrocochlear involvement by reporting successful implantation in a patient with traumatic subarachnoid hemorrhage and brain contusion.

Methods: We present a patient (55-year-old man) who had bilateral progressive sensorineural hearing loss after traumatic subarachnoid hemorrhage and brain contusion. Preoperative imaging and functional studies were done, as well as routine tests, to evaluate the possible performance of the cochlear implant.

Results: Sensorineural hearing loss developed promptly after head trauma with progressive deterioration. The cause of progressive sensorineural hearing loss remained unknown. Distortion product otoacoustic emissions demonstrated bilateral inner ear (outer hair cell) damage. Highly impaired speech discrimination despite less marked pure tone average elevation and a focal lesion in the left middle temporal gyrus suggested the possibility of coexisting retrocochlear lesions. After thorough discussion of the possible outcomes, cochlear implantation was successfully performed 25 months after the trauma. The patient became able to use a telephone.

Conclusions: We advocate that profound bilateral sensorineural hearing loss caused by traumatic head injury, even with possible involvement of central auditory pathways, should not be regarded as a contraindication to cochlear implantation, as long as bilateral inner ear dysfunction is clearly demonstrated and there is no obvious evidence of central deafness.

Key Words: bilateral progressive sensorineural hearing loss, brain contusion, cochlear implantation, head trauma, subarachnoid hemorrhage.

INTRODUCTION

Identification of the lesion site is sometimes difficult in hearing impairment after traumatic brain injury. There have been reports of cochlear implantation in patients with bilateral severe sensorineural hearing loss after head trauma.¹⁻⁴ However, it is difficult to determine the indication for cochlear implantation when the lesion site of hearing impairment remains unclear and possible involvement of retrocochlear auditory pathways cannot be excluded.

Here we report a rare and informative case of bilateral progressive sensorineural hearing loss after traumatic subarachnoid hemorrhage and brain contusion, in which cochlear implantation was very successful. The cause of progressive hearing loss is also discussed.

CASE REPORT

A 55-year-old man fell downstairs and suffered a head injury. He was admitted to a nearby hospital,

and computed tomography (CT) scans demonstrated subarachnoid hemorrhage, skull fracture sparing the temporal bones, and brain contusion. The patient's consciousness was disturbed. Two days after the trauma, when he recovered consciousness, he was aware of hearing impairment in the right ear. He also noted hearing loss in the left ear 4 days after the trauma. The bilateral hearing impairment worsened. Tinnitus was absent. He was referred to our hospital 1 month after the trauma. There was no history of administration of ototoxic agents, including aminoglycosides. On examination, he had no apparent cognitive, behavioral, or communicative deficits, including aphasia. Both eardrums were normal with type A tympanograms. There was no nystagmus, and the fistula sign was negative. Pure tone audiometry showed bilateral sensorineural hearing loss with a pure tone average (PTA) of 68 dB hearing level (HL) on the right and 73 dB HL on the left. Contralateral stapedial reflexes were demonstrated on stimulation of the right ear (100 dB HL or higher at 500 Hz, 110 dB

From the Department of Otolaryngology, Faculty of Medicine, University of Tokyo, Tokyo, Japan.

Correspondence: Ken Ito, MD, Dept of Otolaryngology, Faculty of Medicine, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655, Japan.

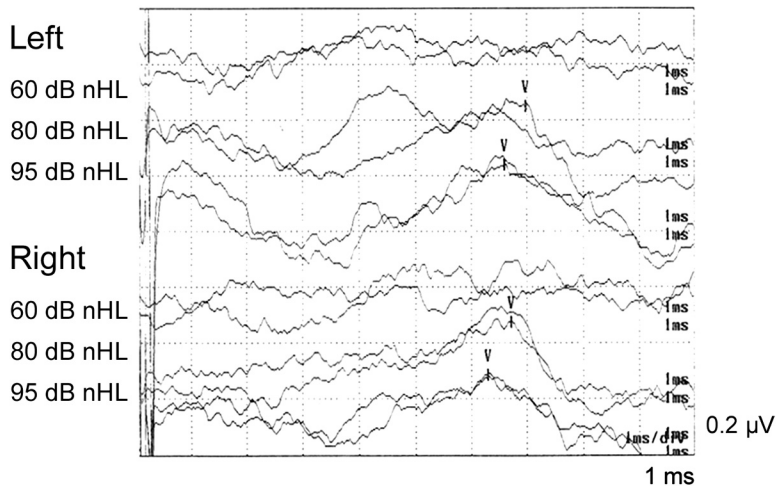


Fig 1. Click-evoked auditory brain stem responses.

HL at 1,000 Hz, and 110 dB HL at 2,000 Hz), demonstrating recruitment, but contralateral reflexes were absent on stimulation of the left ear. Self-recording audiometry (Beke's test) showed type I findings on Jerger's classification⁵ on both sides, indicating neither cochlear nor retrocochlear lesion. On auditory brain stem response (ABR) tests, only wave V was observed on both sides at 80 dB nHL (normal hearing level) and higher (Fig 1). Distortion product otoacoustic emission responses were very poor and mostly below noise level on both sides (Fig 2), indicating severe inner ear (outer hair cell) damage. The maximal speech discrimination score was 10% at 90 dB HL on the right and 15% at 90 dB HL on the left. Caloric tests with ice water stimuli showed normal responses on both sides. The results are summarized in the Table. On imaging studies, high-intensity focal lesions were found in bilateral frontal lobes and the left middle temporal gyrus on T2-weighted mag-

netic resonance imaging (MRI) and fluid-attenuated inversion recovery (FLAIR) images (Fig 3). There were no remarkable findings in the inner ear, internal auditory canal, or cerebellopontine angle. On high-resolution CT, a temporal bone fracture was absent and no abnormal findings were observed in either inner ear, including the vestibular aqueducts.

Because the patient understood short sentences fairly well (77%) under audiovisual conditions with hearing aids, he started wearing a hearing aid, which was effective to a certain extent. Thereafter, he noticed worsening of hearing in the right ear 11 months after the trauma. The PTA was 88 dB HL on the right and 80 dB HL on the left. Administration of oral corticosteroids was started (prednisolone 40 mg/d for 3 days, 30 mg/d for 2 days, and 20 mg/d for 2 days), without success. Isosorbide was then administered at a daily dose of 90 mL. However, the

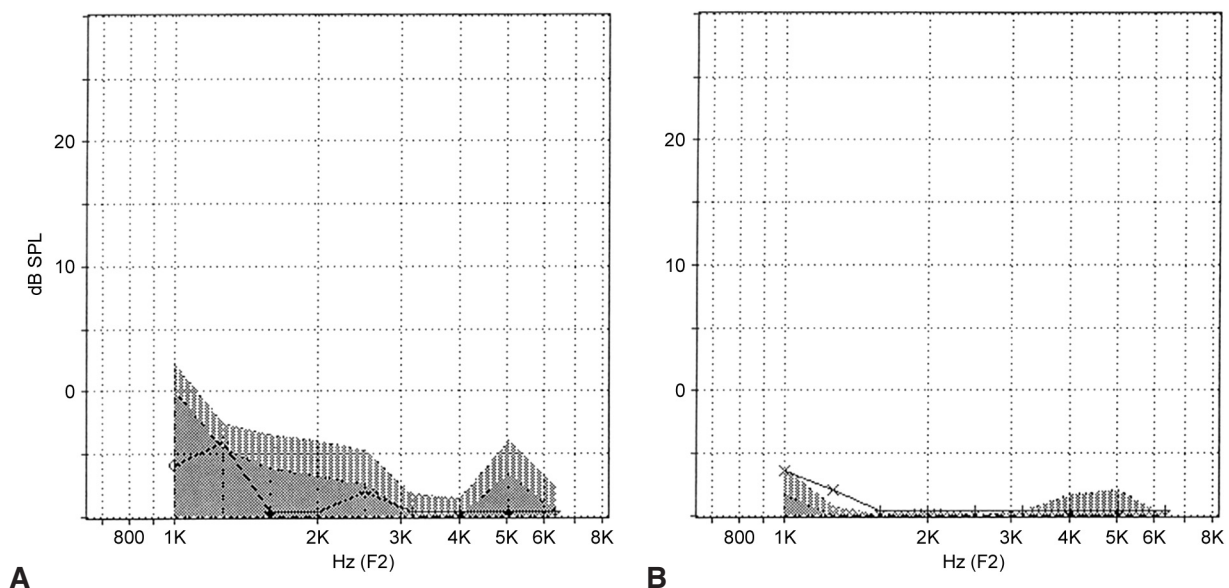


Fig 2. Distortion product otoacoustic emissions. SPL — sound pressure level. **A)** Right side. **B)** Left side.

NEURO-OTOLOGIC TEST RESULTS

| | Right | Left |
|--|-------------------------------|-----------------------|
| Pure tone average | 68 dB | 73 dB |
| Speech discrimination | 10% (90 dB) | 15% (90 dB) |
| Tympanogram | Type A | Type A |
| Stapedial reflex | Positive, with recruitment | Negative |
| Bekesy's audiometry | Jerger type I | Jerger type I |
| Auditory brain stem reponse | Wave V (80 dB nHL) | Wave V (80 dB nHL) |
| Distortion product otoacoustic emissions | Negative | Negative |
| Caloric test | Normal | Normal |

bilateral hearing loss progressed with fluctuation of the hearing threshold. The PTA was more than 105 dB HL (unmeasurably high) on the right and 88 dB HL on the left 13 months after the trauma, and was 97 dB HL on the right and 92 dB HL on the left 15 months after the trauma. Twenty-three months after the trauma, the patient's sentence comprehension score had degraded to 11% under the audio-visual condition with a hearing aid, and indeed the hearing aid was no longer effective. The PTA was 100 dB HL on the right and 92 dB HL on the left. The course of the PTA is shown in Fig 4. The maximal speech discrimination score was 5% at 100 dB HL on the right and 10% at 100 dB HL on the left. Auditory brain stem responses were absent on both sides. Caloric tests showed 31% canal paresis on the

right side 23 months after trauma. Vestibular evoked myogenic potentials were normal on both sides.

At this stage, we considered cochlear implantation and performed promontory stimulation tests, which demonstrated stable dynamic ranges throughout the test frequencies of 50 to 1,600 Hz (wider on the right side than on the left). Because the sensorineural hearing loss developed promptly after traumatic brain contusion, and because the speech discrimination score was very low, coexisting retrocochlear lesions could not be excluded. The efficacy of cochlear implantation was questioned because of the possibility of retrocochlear involvement. After obtaining adequate informed consent from the patient and his family, we performed cochlear implantation on the right ear 25 months after the trauma (Cochlear Nucleus 24 device). The device was switched on 2 weeks after implantation, and all electrodes were available for electrical stimulation. The patient became able to have conversations without lipreading. Speech recognition tests confirmed good performance (in the audio-alone condition: 50% for single syllables, 68% for words, and 98% for short sentences) 3 months after implantation, and the patient became able to use a telephone.

DISCUSSION

Causes of Progressive Hearing Loss After Head Trauma. It has been reported that among cases of

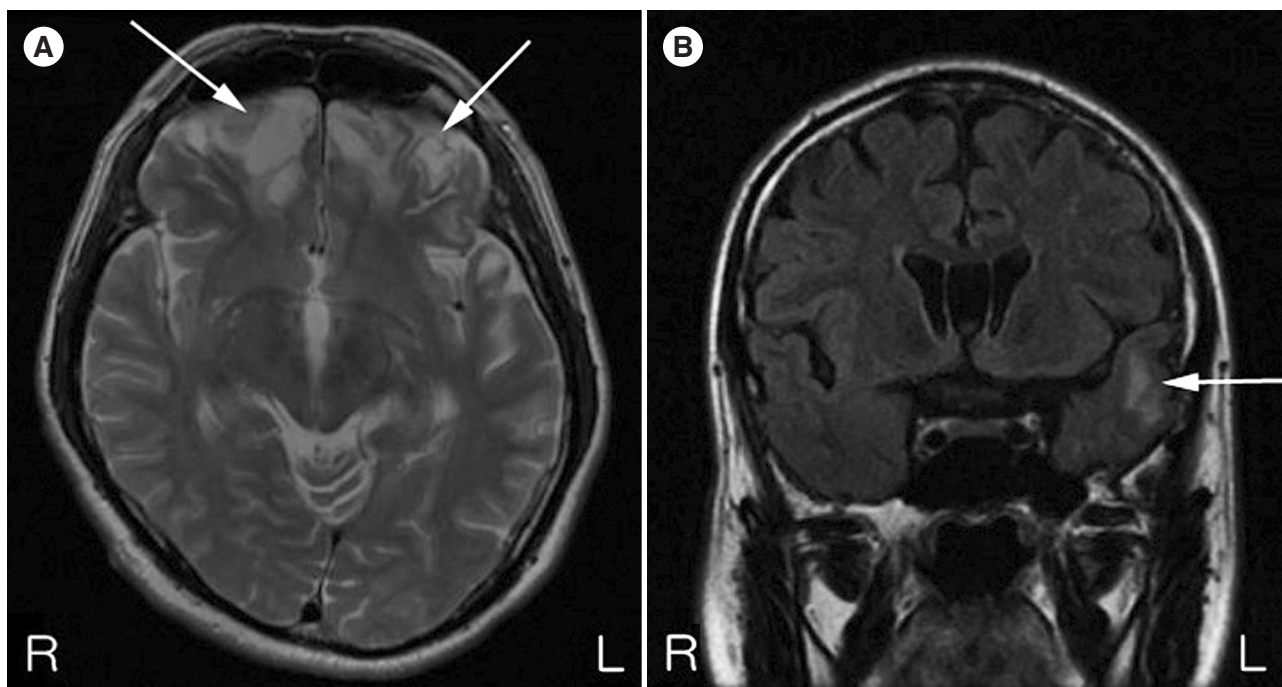


Fig 3. Magnetic resonance imaging. **A)** T2-weighted axial image demonstrates high-intensity focal lesions in bilateral frontal lobes (arrows). **B)** Fluid-attenuated inversion recovery (FLAIR) image demonstrates high-intensity focal lesions in left middle temporal gyrus (arrow).

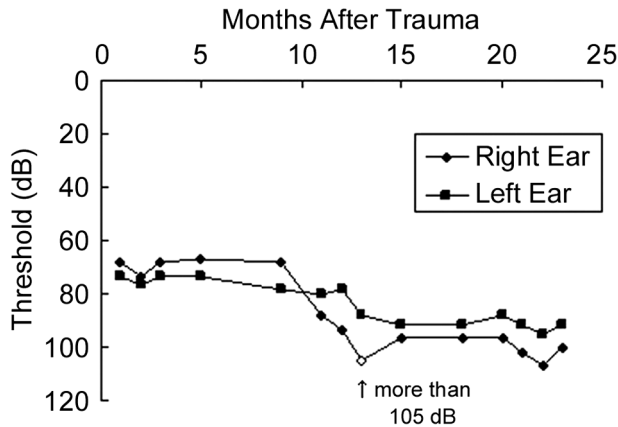


Fig 4. Course of pure tone averages after head trauma.

closed head injury, there was a higher incidence of progressive hearing loss in those with temporal bone fracture than in those with brain contusion only, and that there was no significant difference in the incidence between the contusion-only group and a control group with hearing loss from other causes.⁶ Our patient exhibited progressive sensorineural hearing loss despite the absence of temporal bone fracture. Perilymphatic fistula due to traumatic brain injury can cause progressive sensorineural hearing loss. However, the probability of perilymphatic fistula in our case was considered low, because of the absence of complaints of tinnitus or balance disorder and the absence of a fistula sign. Intracranial hypotension, which is occasionally induced by traumatic head injury, can cause cochlear symptoms such as hearing loss.^{7,8} It can also cause fluctuating progressive sensorineural hearing loss, but this disorder was not considered the cause of hearing loss in our case, because of the absence of typical symptoms of intracranial hypotension, eg, the exacerbation of headache or hearing loss in the standing or sitting position and its improvement in the lying position. Superficial siderosis of the central nervous system, which may follow subarachnoid hemorrhage, can generate bilateral progressive hearing loss.⁹⁻¹³ However, superficial siderosis was also excluded, because MRI did not show any specific findings such as rims of hypointensity surrounding the brain stem or typical linear hypointensities coating the cerebellar folia. Bergemalm⁶ reported that higher age at the time of trauma increased the risk of progressive hearing loss. The present patient (55 years old) belongs to the high-risk group according to their criterion, but this epidemiological fact fails to suggest the cause of hearing impairment. Thus, the cause of hearing loss in the present case remains unknown. One speculative hypothesis is that certain structural damage to the cochlea, eg, tear of the basal membrane, progressed with scar formation.

Cochlear Implantation in Cases With Possible Retrocochlear Involvement. Indications for cochlear implantation for a patient with possible central deafness have been controversial. The Otorhinolaryngological Society of Japan provides guidelines according to which cochlear implantation is contraindicated for adult patients with disorders in retrocochlear auditory pathways. There have been few reports on cochlear implantation in patients with bilateral severe sensorineural hearing loss after head trauma.¹⁻³ Moore and Cheshire³ reported an unsuccessful case of cochlear implantation in a patient with cognitive, behavioral, and communicative deficits including aphasia after head trauma. However, implantation in patients without cognitive, behavioral, or communicative deficits has tended to be relatively successful.¹⁻³

In the present case, the maximal speech discrimination score was very low (10% to 15%) despite less marked PTA elevation (around 70 dB, boundary between moderate and severe hearing impairment) 1 month after the trauma. Magnetic resonance imaging showed a focus of brain contusion in the left middle temporal gyrus, which is related to processing below the word level (phonological processing) and processing of sentences (semantic processing).¹⁴ These findings suggested possible disturbance of the retrocochlear auditory pathways or the area involved in language processing. As this case was also atypical of progressive sensorineural hearing loss after head trauma without temporal bone fractures, with the cause being unknown, the indication for cochlear implantation was determined very carefully.

The promontory stimulation tests showed good dynamic ranges in both ears. However, this finding should not be overestimated, because perception of sound with promontory stimulation tests could only show that the "primary" auditory cortex was probably spared. In cases of bilateral cochlear disturbance like the present case, there are no methods to verify the integrity of central auditory processing, including retrocochlear pathways and association cortices. Taking all of these factors into account, we finally concluded that the possible retrocochlear lesion would not critically involve the central auditory pathway, as no symptoms indicating aphasia were observed and the lesion on MRI seemed to spare the central areas of the auditory cortices (Heschl's gyrus, superior temporal gyrus) and the auditory radiation (radiatio acustica). Bilateral inner ear dysfunction, confirmed by absent distortion product otoacoustic emissions, could also justify cochlear implantation. Therefore, we decided that cochlear

implantation was not contraindicated.

After implantation, communication by telephone became possible — a result that we never expected. The lesion in the left middle temporal gyrus might have spared the portion important for language processing, or the nearby cortices might have already compensated for the loss. It is also possible that the middle temporal gyrus lesions had already resolved to a certain extent. From the present experience, the authors advocate that profound bilateral sensorineu-

ral hearing loss caused by traumatic head injury, even with possible involvement of central auditory pathways, should not be regarded as a contraindication to cochlear implantation, as long as bilateral inner ear dysfunction is clearly demonstrated and there is no obvious evidence of central deafness. However, as there have been few reports of similar cases, further accumulation of experience in bilateral deafness following head trauma is needed to draw conclusions on the precise handling of this rare disorder.

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