Hearing loss after microvascular decompression for hemifacial spasm

Risk factors

The cochlear nerve function is at risk during microvascular decompression for hemifacial spasm.

Cause and risk factors are highly variable.

A study strongly suggested a correlation between the cerebellar retraction factors, especially retraction depth and duration, and possibility of hearing loss following MVD for HFS [1].

Intradural compression due to overinfusion of saline may lead to postoperative hearing loss, although the incidence is low, and immediate decompression by drainage may be required [2].

Prevention

Intraoperative monitoring of brainstem auditory evoked potentials (BAEPs) can be a useful tool to decrease the danger of hearing loss [3] [4] [5].

It is important to emphasize the need for clean exposure of the lower cranial nerves (except for cranial nerve VIII) to obtain enough working space, sharp arachnoid dissection, minimal cerebellar retraction, and proper responses to changes identified during intraoperative monitoring [6].

Diagnosis

Prolongation of the inter-peak latency of waves I-III seems to be associated with the occurrence of delayed hearing loss. It is possible that BAEP changes may predict delayed hearing loss, but confirmatory evidence is not available as yet. Analysis of more cases is necessary to determine the utility of BAEP monitoring to predict delayed hearing loss after MVD and to identify its exact cause [7].

Case series

Lee et al., from the Samsung Medical Center in a study aimed to analyze cases of delayed hearing

loss after microvascular decompression (MVD) for hemifacial spasm and identify the characteristic features of these patients.

They retrospectively reviewed the medical records of 3462 patients who underwent MVD for hemifacial spasm between January 1998 and August 2017.

Among these, there were 5 cases in which hearing was normal immediately postoperatively but delayed hearing loss occurred. None of the 5 patients reported any hearing disturbance immediately after the operation. However, they developed hearing problems suddenly after some time (median, 22 days; range 10-45 days). On examination, sensorineural hearing loss was confirmed. High-dose corticosteroid treatment was prescribed. Preoperative hearing levels were restored after several months (median duration from the time of the operation, 45 days; range 22-118 days). Interestingly, the inter-peak latency of waves I-III in the brainstem auditory evoked potentials (BAEP) was prolonged during the surgery, but recovered within a short time.

Delayed hearing loss may occur after MVD for HFS. Prolongation of the inter-peak latency of waves I-III seems to be associated with the occurrence of delayed hearing loss. It is possible that BAEP changes may predict delayed hearing loss, but confirmatory evidence is not available as yet. Analysis of more cases is necessary to determine the utility of BAEP monitoring to predict delayed hearing loss after MVD and to identify its exact cause [8].

Nine hundred and thirty-two patients with HFS who underwent MVD with intraoperative monitoring (IOM) of BAEP were analyzed. Park et al., used a 43.9 Hz/s stimulation rate and 400 averaging trials to obtain BAEP. To evaluate HL, pure-tone audiometry and speech discrimination scoring were performed before and one week after surgery. We analyzed the incidence for postoperative HL according to BAEP changes and calculated the diagnostic accuracy of significant warning criteria.

Only 11 (1.2%) patients experienced postoperative HL. The group showing permanent loss of wave V showed the largest percentage of postoperative HL ($p < 0.001$). No patient who experienced only latency prolongation (≥1 ms) had postoperative HL. Loss of wave V and latency prolongation (≥1 ms) with amplitude decrement (≥50%) were highly associated with postoperative HL.

Loss of wave V and latency prolongation of 1 ms with amplitude decrement ≥50% were the critical warning signs of BAEP for predicting postoperative HL [9].

Jung et al., retrospectively analyzed the medical records of patients with HFS who underwent MVD with the same surgeon from March 2003 to October 2016, and reviewed the pertinent literature. Patients who were followed up for more than 6 months were selected, resulting in the analysis of 1434 total patients. Postoperative hearing complications were evaluated audiometrically and subjectively (patient-reported symptoms). Clinical factors such as the intraoperative findings were reviewed to identify their correlation with auditory function.

Symptoms in 1333/1434 patients (93.0%) resolved more than 90% from their preoperative state. Among them, 16 patients (1.1%) complained of hearing impairment after surgery. Most impairment was transient, although 6/1333 patients (0.4%) required additional interventions for persistent hearing deficits (one surgical intervention and five hearing aids). A >50% decrease in the amplitude of brainstem auditory evoked potentials during the operation was significantly associated with

postoperative hearing deficits.

Few auditory complications, mostly transient, result from MVD. Although MVD is a commonplace surgical technique, to reduce complications it is important to emphasize the need for clean exposure of the lower cranial nerves (except for cranial nerve VIII) to obtain enough working space, sharp arachnoid dissection, minimal cerebellar retraction, and proper responses to changes identified during intraoperative monitoring [10)]

Three hundred thirty-one patients with HFS underwent MVD from March 2009 to October 2010.

Brain stem auditory evoked potential (BAEP) was monitored during the surgery. Before completion of the dural closure, the surgical field was routinely filled with warm saline to avoid postoperative pneumocephalus and epidural hematoma.

Seven patients experienced a change in wave V amplitude and latency after the dural closure. In 2 patients, the amplitudes decreased by less than 50%, and latencies were delayed by less than 1.0 ms, ipsilaterally in 1 patient and contralaterally in the other. In 1 patient, decreased amplitude and delayed latency appeared bilaterally with more severity on the operated side, accompanied by delayed ipsilateral permanent hearing loss. In 4 of the 7 patients, an ipsilateral response of BAEP was completely absent. Of these 4 patients, 2 experienced permanent hearing loss, and another 2 patients who underwent dural reopening and saline drainage had restoration of their normal hearing.

Intradural compression due to overinfusion of saline may lead to postoperative hearing loss, although the incidence is low, and immediate decompression by drainage may be required [11)].

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668 patients (95.7%) had no hearing loss immediately after surgery (group 1). 17 patients (2.4%) had a postoperative decrease in PTA exceeding 15 dB and a decrease in SDS which was proportional to the postoperative PTA thresholds (group 2). Eight patients (1.2%) had poor SDS that appeared to be out of proportion to the degree of hearing loss depicted by the postoperative PTA thresholds, suggesting retrocochlear or cochlear nerve pathology (group 3). Five patients (0.7%) had total deafness after surgery (group 4). In group 2, 12 patients (70.6%) returned to their preoperative hearing capacity. However, among the eight patients in group 3 and five in group 4, only two (25%) and none (0%) have returned to their preoperative hearing status, respectively.

In this large study, permanent hearing loss occurred in 16 patients (2.2%). Patients with a mild hearing loss with a good SDS (cochlear type) demonstrated much better prognosis than those with poor SDS (retrocochlear type) or total deafness. In addition, total deafness after surgery had no chance of recovery to preoperative hearing capacity [12)].

Auditory function was studied before and after surgery in 143 consecutive patients who were operated on for hemifacial spasm by microvascular decompression of the intracranial portion of the facial nerve. The acoustic reflex was abnormal preoperatively in 41% of the patients, indicating that the vascular abnormalities that caused the hemifacial spasm also affected the auditory nerve. Three patients suffered a profound hearing loss in the ear on the operated side, and one lost hearing function totally. In addition, 24 patients had a moderate elevation in the pure-tone threshold at one or more octave frequencies. Of these, 16 patients experienced a hearing loss at only one frequency

(8000 Hz), while eight had a threshold evaluation of no more than 20 dB in the speech frequency range (500, 1000, and 2000 Hz). Two patients were deaf on the side of the spasm before the operation. Three patients were not tested postoperatively, and one patient was tested only after surgery. Thus, in this series of 143 patients, only 2.8% suffered a significant hearing loss as a complication of facial nerve decompression to relieve hemifacial spasm [13].

Case reports

Onoda et al., reported two unusual cases of delayed hearing loss after microvascular decompression (MVD) for hemifacial spasm. In the first case, A 59-year-old female noted left hearing loss one week after receiving MVD for left hemifacial spasm. In the second case, A 39-year-old male also noticed ipsilateral hearing loss on the 7th day after MVD for right hemifacial spasm. Both cases were treated by steroid. Two months after the onset, their hearing function improved dramatically. These cases indicated that the delayed hearing loss after MVD for hemifacial spasm can occur, even when gentle microsurgical technique is used, but the prognosis for this condition is fairly good [14].

Unclassified

Delayed hearing loss after microvascular decompression for hemifacial spasm-an unsolved conspiracy of the cochlear apparatus [15].

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