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MAIN ARTICLE

Effect of surgical intervention on middle-ear cholesteatoma with associated facial paralysis

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Abstract

Objectives: To investigate the presenting symptoms, intra-operative findings and long-term facial nerve function in patients treated for cholesteatoma with associated facial paralysis.

Methods: Fifteen patients with facial paralysis due to middle-ear cholesteatoma who underwent tympanomastoidectomy surgery from February 2000 to February 2015 were retrospectively reviewed. After removal of the cholesteatoma, a limited area of the fallopian canal, in which facial nerve oedema or redness was evident, was opened. Incision of the epineural sheath for nerve decompression was not performed.

Results: Pre-operative House–Brackmann grade was grade II in two patients, grade III in four, grade IV in seven, grade V in one and grade VI in one. Facial nerve perineurium damage was observed in two patients with poor prognoses. All patients treated within the first 15 days after paralysis onset showed normal facial function at long-term follow up. Post-operative House–Brackmann grade was grade I in 11 patients, grade II in 1, grade III in 2 and grade VI in 1.

Conclusion: Early surgical treatment is more likely to give good results, and poor outcomes are observed in patients with facial nerve perineurium damage.

Key words: Facial Paralysis; Cholesteatoma, Middle Ear; Prognosis

Introduction

Facial nerve paralysis is an uncommon complication of chronic otitis media, especially chronic otitis media with cholesteatoma. Although its incidence has decreased with the use of antibiotics, the reported frequency of facial nerve paralysis in chronic otitis media ranges from 0.16 to 5.1 per cent.[1,2] Facial nerve paralysis markedly affects a patient's social life, and causes serious physiological and psychological damage. Appropriate management of this complication is also necessary to ensure healing without sequelae.

Although the mechanism of facial nerve paralysis that occurs as a result of chronic otitis media is not fully understood, treatment recommendations have focused on both antibiotic treatment and surgery, including myringotomy, mastoidectomy and nerve re-decompression, in order to re-establish the physiological state of the facial nerve.[3,4] Surgical eradication of the disease is the most viable way to overcome facial nerve paralysis; however, there are a lack of data from which to draw significant conclusions on surgical management.[5] The variance in application of surgical treatment for these patients stems from the lack of predictors for facial nerve paralysis recovery.[6,7]

Facial nerve paralysis associated with middle-ear cholesteatoma is well known; however, its clinical features and management have only been evaluated in a few studies. The present study aimed to investigate the presenting symptoms, the intra-operative findings and the long-term facial nerve function in patients treated for cholesteatoma with associated facial paralysis within a period of 15 years.

Materials and methods

Study design

A retrospective study was performed of patients with facial nerve paralysis due to middle-ear cholesteatoma who underwent tympanoplasty surgery from February 2000 to February 2015. A total of 2350 patients underwent tympanomastoidectomy during this period in our institution. The present study included a homogeneous group of patients; because of this, only tympanomastoidectomy cases with facial paralysis and cholesteatoma were included in the study. Patients younger than 15 years of age, and patients with syndromes that may affect the middle ear (e.g. Down's syndrome, Kartagener's syndrome, cleft palate) were excluded

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from the study. Fifteen patients were finally enrolled in the study.

Outcome parameters

Patients' charts, clinical notes and operation reports were reviewed. The following data were analysed: otoscopic findings, facial paralysis characteristics, surgery type, intra-operative findings related to the facial nerve (such as nerve appearance and facial canal defect type), pre- and post-operative House–Brackmann grades, and follow-up duration (months).

Surgical procedures

All operations were performed under general anaesthesia with endotracheal intubation, using a post-auricular approach under the supervision of the senior author. After removal of the cholesteatoma lesion, a limited area of the fallopian canal, in which facial nerve oedema or redness was evident, was opened. Incision of the epineural sheath for nerve decompression was not performed.

Results

The study group consisted of 15 patients (7 females, 8 males) with a mean age of 58.46 ± 8.32 years (range, 48–64 years). All of the patients had at least a one-year follow up after surgical intervention. The mean follow-up period was 80.2 ± 41.4 months (range, 12–174 months).

Symptoms associated with facial paralysis were: vertigo, in three patients; high fever, in two patients; tinnitus, in one patient; and headache, in one patient. Seven patients had no associated symptoms. In addition to conductive hearing loss, facial paralysis was the sole complication in 11 patients. Two patients had multiple complications that included: labyrinthitis ($n = 2$), sudden total hearing loss ($n = 1$), lateral sinus thrombosis ($n = 1$) and epidural abscess ($n = 1$). Pre-operative House–Brackmann grade was grade II in two patients, grade III in four patients, grade IV in seven patients, grade V in one patient and grade VI in one patient.

Table I presents data on pre-operative facial nerve function, surgical technique, facial nerve injury type and site of facial nerve involvement.

Mean (±standard deviation) facial nerve paralysis duration was 8.73 ± 7.64 days (range, 3–28 days). Intra-operatively, cholesteatoma was confirmed in every case. Radical mastoidectomy was performed in eight patients (53.3 per cent), canal wall down mastoidectomy was performed in six patients (40 per cent) and intact canal wall mastoidectomy was performed in one patient (6.7 per cent). All patients had decompression of the fallopian canal from the geniculate ganglion to the stylomastoid foramen without opening the epineural sheath. The tympanic segment was the most common site of involvement in 14 patients (93.3 per cent). Of these 14 patients, 7 also had exposure on the mastoid segment. The mastoid segment was the only site of involvement in one patient. The facial nerve was compressed in seven patients, oedematous and congested in four patients, and intact and normal in two patients. The perineural sheath was damaged in two patients. The medical therapy given (in combination with surgery) to reduce the oedema included antibiotics and steroids in all patients.

Eleven patients (73.3 per cent) demonstrated dramatic recovery within three months after surgery. Post-operative House–Brackmann grade was grade I in 11 patients, grade II in 1 patient, grade III in 2 patients and grade VI in 1 patient. All patients treated within the first 15 days after paralysis onset showed normal facial function at long-term follow up.

Discussion

Facial nerve paralysis secondary to chronic otitis media has several possible causes. The proposed aetiological factors include: osteitis, bony erosion, compression resulting from oedema, and direct inflammation of the nerve by bacteria or by neurotoxic substances, which may be secreted from the cholesteatoma matrix.[8,9] It has been demonstrated that the facial nerve fills 35–65 per cent of its canal.[10] The remaining portion is filled with extraneural blood vessels and connective tissue, without leaving any space. Thus, oedema secondary to infection can easily affect neural transmission.

Nevertheless, a dehiscent facial canal can cause otogenic facial paralysis in the presence of chronic suppurative otitis media. It has been reported that in some instances, dehiscence of the vestibular surface of the facial canal is out of the surgeon's view, and, frequently, it is the site of the portal of infection.[11] As reported by other authors, in the present series, the second portion of the nerve was the most common involved site (in 93.3 per cent of the cases).[11–13] Savic and Djeric proposed that the tympanic portion of the nerve is most commonly involved because the nerve is congenitally exposed or it is covered by a very thin layer of bone.[12] In addition, the involvement of the tympanic portion of the nerve reflects the most frequent way of spread of an acquired cholesteatoma from the posterior epitympanum to the aditus ad antrum and the mastoid.

There has been no consensus in the English literature regarding the area of surgical opening of the fallopian canal or whether the nerve sheath should be incised for facial nerve decompression.[13,14] Cawthorne postulated that nerve sheath incision is not necessary for cases of incomplete paralysis, but should be performed for cases of complete paralysis.[15] Yetiser *et al.* performed decompression of the fallopian canal from the geniculate ganglion to the stylomastoid foramen, but did not open the epineural sheath for decompression.[11] We opened the fallopian canal in a limited area, in which there was redness or oedema of the facial nerve, because exposure of neural tissue should be minimised, given the possible presence of local

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TABLE I
FACIAL NERVE FUNCTION, SURGICAL TECHNIQUE, FACIAL NERVE INJURY TYPE AND SITE OF INVOLVEMENT

Pt no.	Facial nerve involvement	Surgical technique	Time of surgery (days after onset)	Pre-operative House–Brackmann grade	Facial nerve injury type	Follow-up duration (months)	Post-operative House–Brackmann grade
1	T	Canal wall down	4	II	Compressed	44	I
2	T	Radical mastoidectomy	5	III	Oedematous & congested	38	I
3	T & M	Radical mastoidectomy	12	IV	Oedematous & congested	98	II
4	T & M	Canal wall up	7	IV	Oedematous & congested	102	I
5	T & M	Radical mastoidectomy	3	IV	Compressed	78	I
6	T & M	Radical mastoidectomy	4	IV	Compressed	23	I
7	T	Canal wall down	20	VI	Perineural damage	93	VI
8	T & M	Radical mastoidectomy	5	III	Compressed	94	I
9	T	Radical mastoidectomy	3	IV	Normal	88	I
10	T	Canal wall down	3	II	Normal	174	I
11	M	Canal wall down	28	IV	Perineural damage	84	III
12	T	Canal wall down	19	V	Oedematous & congested	93	III
13	T & M	Canal wall down	7	III	Compressed	52	I
14	T	Radical mastoidectomy	4	III	Compressed	128	I
15	T & M	Radical mastoidectomy	7	IV	Compressed	14	I

Pt no. = patient number; T = tympanic portion of facial nerve; M = mastoid portion of facial nerve

infection. For similar reasons, we did not perform nerve sheath incision. Based on the results of Savic and Djeric,[12] our decision may have been appropriate.

In the present series, cholesteatoma treatment and nerve decompression allowed for the recovery to House–Brackmann grade I in 73.3 per cent of cases. Ikeda *et al*. reported a recovery to House–Brackmann grade I–II in 82 per cent of patients, whereas Yetiser *et al*. reported complete recovery in 57.2 per cent of patients with cholesteatoma.[11,16] The surgical treatment of facial nerve paralysis due to chronic otitis media without cholesteatoma is associated with a similar facial nerve recovery, confirming that the spread of infection is the main factor in otitic facial palsy.[11,12]

Early surgical intervention can lead to more favourable improvements in facial function. Longer durations can cause more severe deterioration of the facial nerve and lead to poor surgical outcomes. Patients with facial paralysis due to chronic otitis media should be operated on as early as possible, regardless of the facial dysfunction severity, cholesteatoma presence, onset type, age and any previous otological surgical history. In the present series, all the patients operated on within 15 days from palsy onset attained House–Brackmann grade I or II, whereas patients operated on 15 days or more after palsy onset showed a variable outcome. These findings suggest that a prolonged infection of the nerve fibres leads to irreversible damage of these fibres, as shown by Savic and Djeric, and that the recovery of the facial function depends on prompt removal of the infection.[12] One study reported that patients who underwent surgery more than two months after the onset of paralysis displayed a poorer outcome.[16]

All of the patients who had surgery were followed up at regular intervals. A total of 11 patients (73.3 per cent) showed complete recovery of facial functions. The recovery rate of our investigation is similar to that reported by Savic and Djeric, and Altuntas *et al*.[12,17]

- **Intra-operative findings and post-operative facial nerve function were evaluated in cholesteatoma patients with facial paralysis**
- **All patients treated within 15 days after paralysis onset showed normal facial function at long-term follow up**
- **Surgical findings indicated that early treatment is more likely to give good results**
- **Poor outcomes were observed in patients with facial nerve perineurium damage**

The limitations of our study include the retrospective design and the relatively small number of patients in our series. In addition, some details of patient history and factors that may influence the outcome may not have been completely documented. Given these

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restrictions, associations should be interpreted with caution.

Facial nerve paralysis is one of the important complications of chronic otitis media and should be treated in a semi-emergent manner. In the present series, destruction of the facial canal was evident in all patients. The defect was mostly located in the tympanic segment of the canal. The complete recovery rate was 73.3 per cent in our study. Facial nerve paralysis in chronic otitis media can be prevented with surgery in patients with chronic suppurative otitis media.

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