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Management of facial nerve paralysis in noncholesteatomatous chronic otitis media

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Abstract. *Management of facial nerve paralysis in noncholesteatomatous chronic otitis media. Objective*: The objective of this study was to analyse the clinical presentations, treatment course, and outcomes for patients treated for chronic otitis media associated with facial paralysis in noncholesteatomatous ears.
Methodology: The present study looked at 13 patients (10 men, 3 women; ranging in age from 15 to 59 years) treated for facial paralysis due to chronic otitis media without cholesteatoma.
Results: Six patients had dehiscence of the fallopian canal whereas the bony canal was intact in the remaining patients. Decompression of the facial nerve was not performed in 5 of the 7 ears with an intact fallopian canal. Four ears underwent total decompression from the geniculate ganglion to the stylomastoid foramen, while the remaining 4 ears underwent partial nerve decompression. However, statistical analysis did not show any difference in recovery between the patients with surgical decompression and those without decompression ($p = 0.171$). All the patients not receiving decompression had successful outcomes (80% classified as Grade I and 20% as Grade II).
Conclusion: All patients not receiving decompression had successful outcomes. Intravenous antibiotic treatment in conjunction with steroid therapy is the mainstay management of facial paralysis due to chronic otitis media without cholesteatoma. It is therefore not necessary to decompress the facial nerve in cases of facial paralysis in noncholesteatomatous chronic otitis media.

Introduction

Facial nerve paralysis is one of the most feared complications of chronic otitis media (COM), though it is not a life-threatening condition. Nerve paralysis may result in a severe cosmetic deformity that may alter a patient's social life. In the pre-antibiotic era, this complication was seen more frequently as a result of acute otitis media, although the introduction of antibiotics has reduced prevalence. Currently, otogenic facial paralysis (FP) due to COM is still a frequent problem, especially in developing countries.[1] Facial nerve paralysis has been discussed in only a few studies with a limited number of patients, suggesting that more data is needed on this subject.[2-6]

Although the precise mechanism of FP development in COM is not known, it seems most likely to be the result of 1) an infection process resulting in osteitis, bone erosion and neuritis with subsequent nerve entrapment; 2) bacterial toxins that impair nerve conduction by demyelination; and 3) direct inflammation of the nerve.[2,3]

The vast majority of clinical studies in the literature have focused on facial paralysis associated with either acute otitis media or COM with cholesteatoma. There is only one report documenting the results of therapy in a series of patients in whom facial nerve dysfunction is associated with noncholesteatomatous COM.[4]

Although the pathophysiology of FP has not yet been clearly elucidated, decompression of the nerve as soon as possible is generally recommended for the treatment of FP with or without cholesteatoma.[5]

The present study looked at 13 patients treated for COM associated with FP in noncholesteatomatous ears over the past 12 years. Despite being limited in size, this series represents the largest published relating to FP in ears without cholesteatoma. Our objective was to review the clinical presentations, treatment course, and outcomes of the patients, and to discuss evolving strategies to manage the involved nerve over the past 12 years.

Materials and methods

We conducted a retrospective chart review to analyse all patients admitted to our hospital between January 1995 and June 2007 with a diagnosis of FP. Patients with facial nerve dysfunction due to

cholesteatoma (secondary, congenital or petrous bone cholesteatoma), and those with iatrogenic and idiopathic facial palsies were excluded. Thirteen patients with facial nerve paralysis secondary to noncholesteatomatous COM were identified during this period. Patients' charts and clinical and operative records were analysed. Facial palsy that occurred within the first 24 hours was defined as sudden, and as more gradual when occurring after 24 hours. Evaluation took place of the degree of the facial nerve dysfunction, otoscopic findings, intra-operative records, type of surgery, whether or not facial nerve decompression was performed, and post-operative results. Pre- and post-operative facial nerve functions were assessed using the House-Brackmann (HB) classification.[7] Grades of II-IV indicate incomplete nerve function and Grades V-VI indicate complete paralysis. Post-operative grades I and II were considered to indicate a successful outcome. Surgery was performed as soon as possible in all patients and all received pre-operative, intra-operative and post-operative intravenous antibiotics. All patients also received an oral antibiotic for an additional 10-day period after intravenous treatment. A bolus dose of 250 mg methylprednisolone was given intravenously during the operation. Five days after the operation, oral prednisolone was administered for 15 days, beginning with 1 mg/kg and gradually lowering the dose.

Statistical analysis was performed with the statistics program SPSS for Windows (version 11.5; SPSS, Inc., Chicago, IL). Mann-Whitney U test was used to compare the nerve function outcome between the groups and Wilcoxon-Signed Rank Test for comparisons within the groups. A p-value $< .05$ was considered statistically significant.

Results

Table 1 lists the clinical findings of the 13 patients (10 males and 3 females). The patients' ages ranged from 15 to 59 years with a median of 34 years. The duration of FP from the onset to surgery ranged between 4 days to 154 days (median 24 days). The onset of FP was sudden in 9 (69.2%) and gradual in 4 (30.8%) patients. Using the House-Brackmann (HB) grading system, 10 patients were classified as incomplete paralysis (1 patient Grade II, 2 patients Grade III, 7 patients with Grade IV) and 3 patients were classified as complete paralysis (2 patients Grade V and 1 patient with Grade VI). All of the patients had a central tympanic membrane perforation without cholesteatoma, 3 patients had dry ears and 10 patients had suppurative middle ears. Infected mucosa of the middle ear cavity and granulation tissue in the mastoid was present in 10 patients, 6 of whom had dehiscence of the fallopian canal. The facial nerve was evaluated for dehiscence in all segments of the nerve at surgery by visual inspection under operating microscope with high magnification and also by palpation with a blunt pic. In all of the patients with dehiscence, the infected and/or granulation tissue was dissected non-traumatically off the bony defect on the fallopian canal. Three patients had dry ears and there was tympanosclerosis in 2 patients. The long process of the incus was eroded in 5 of the 13 ears.

The intact canal wall tympanomastoidectomy was performed in all 13 patients, either with or without decompression of the facial nerve. Fallopian canal dehiscence was present in 6 patients and all of the dehiscent regions were located in the tympanic segment and 2nd genu. Decompression of the fallopian canal was performed from the geniculate ganglion to the stylomastoid foramen in 4 patients. In an additional 4 patients, the fallopian canal was opened in a limited area 2-3 mm proximal and distal to the area of dehiscence. A total of 8 patients therefore underwent decompression with either the total or partial technique. The epineural sheath was never incised during decompression in either case because the epineural sheath is a natural barrier for the spread of infection. Five of the 7 patients without fallopian canal dehiscence did not undergo nerve decompression and only an intact canal wall tympanomastoidectomy was performed in this group. Two out of 7 patients without dehiscence underwent total decompression from the geniculate to the stylomastoid foramen.

In 4 out of 5 patients who did not undergo facial nerve decompression, a complete recovery to HB Grade I was achieved and, in one patient, post-operative facial dysfunction improved to HB Grade II. Results for the patients without dehiscence are also presented in Table 1. In the 4 patients who had facial nerve decompression from the geniculate ganglion to the stylomastoid foramen, the outcomes were variable (Table 1). However, two of these patients had a protracted facial nerve

Table 1

Clinical and intra-operative findings and surgical technique used in 13 patients with chronic otitis media complicated by facial nerve paralysis

Patient	Sex	Age	Onset of paralysis	Days until surgery	Dehiscence	Decompression	Preop grade	Postop grade
1	M	21	Gradual	154	+	Total	IV	IV
2	F	25	Sudden	98	+	Total	V	III
3	M	45	Sudden	56	+	Partial	IV	II
4	M	37	Gradual	37	+	Partial	VI	II
5	F	27	Sudden	25	–	Total	IV	II
6	M	15	Gradual	15	–	Total	III	I
7	M	47	Sudden	5	+	Partial	II	I
8	M	59	Sudden	11	+	Partial	IV	I
9	M	17	Sudden	24	–	None	IV	I
10	M	48	Gradual	32	–	None	IV	II
11	M	52	Sudden	16	–	None	III	I
12	F	34	Sudden	4	–	None	V	I
13	M	34	Sudden	18	–	None	IV	I

paralysis (154 days and 98 days, respectively). Figure 1 shows the pre- and post-operative HB Grades of the patients with or without decompression. However, statistical analysis did not show any difference in recovery between the patients with surgical decompression and those without decompression ($p = 0.171$). Recovery was significant for both of the groups receiving treatment, either with or without decompression ($p = 0.016$ vs $p = 0.041$). Statistical analysis did not show any difference in recovery between the patients with complete (HB V-VI) and those with partial (HB II-IV) paralysis ($p = 0.469$).

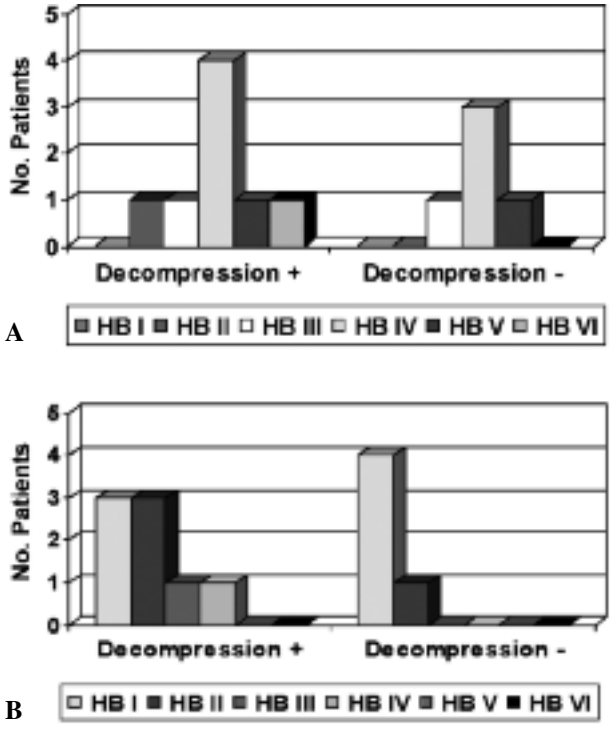

Figure 1

Pre-operative (A) and post-operative (B) HB grades of the 13 patients with or without decompression.

Discussion

Facial nerve paralysis secondary to COM without cholesteatoma is a rare and challenging situation. Management of FP resulting from cholesteatomatous COM has always received more attention than FP resulting from noncholesteatomatous COM. Most of the authors recommend decompression for the management of FP due to COM with cholesteatoma.[2,4,5] However, even on this less divisive issue, different surgical approaches have been proposed for the decompression method, including total or partial decompression with or without sheath incision. Management of FP in noncholesteatomatous ears is more controversial, as there is only one published study specifically discussing the treatment in these patients.[4] Because there has been little discussion of this subject, various treatment strategies have been put forward, with particular divergence regarding

the necessity of decompression. The extent to which the dehiscent canal should be widened in order to relieve compression or oedema, and whether the nerve sheath should be incised or not, are questions that need to be addressed.

In 1992, Harker and Pignatari[4] reviewed 6 cases of FP secondary to noncholesteatomatous COM and reported complete recovery of facial nerve functions (HB Grade I) in 5 patients and Grade II recovery in one patient. In 3 patients, decompression and neurolysis were not performed. All of these 3 cases eventually healed to Grade I. Half of these patients did not therefore undergo decompression and all recovered. In a study by Yetiser *et al*.,[5] patients with FP due to COM were reviewed and 7 patients (in a series of 24) with FP had noncholesteatomatous COM. All patients underwent decompression from the geniculate ganglion to the stylomastoid foramen without nerve sheath incision. Five of the 7 patients without cholesteatoma achieved complete recovery (Grade I), whereas 2 of the 7 recovered to Grade II. In the group without decompression (n = 5 patients), our study revealed complete recovery to Grade I in 4 patients and Grade II in one patient, although the patient with Grade II recovery received treatment after 32 days from onset. Nevertheless, the results for our patients without decompression were also good and satisfactory and did not differ significantly from those for the patients with either partial or total decompression. Even though the patients undergoing decompression also improved, it is clear that there is always a possible risk of iatrogenic injury to the facial nerve during decompression surgery. In our opinion the patients who improved with decompression did not in fact improve because of the decompression process, but mainly due to antibiotics and steroids. Since the results are satisfactory whether the nerve is decompressed or not, decompression is unnecessary.

In COM, the onset of FP is either sudden or gradual. Gradual onset of FP is generally thought to be the result of a compressive effect of cholesteatoma. Yetiser *et al*.[5] reported that 75% of their patients had gradual onset, whereas Ikeda *et al*.[8] and Quaranta *et al*.[3] found sudden onset to be more frequent. However, these reports primarily evaluated the results of FP associated with cholesteatoma. Our findings in COM without cholesteatoma showed gradual onset occurring in 30.8%, and acute onset in 69.2% of patients. Most sudden cases were thought to result from an acute infectious exacerbation superimposed on the chronic process. Pathophysiologically, therefore, direct nerve involvement with infection and accompanying inflammation seems more likely to be the cause of FN paralysis, especially in noncholesteatomatous COM.[4]

Currently, there is no consensus regarding the area for the surgical opening of the fallopian canal or whether the nerve sheath should be incised for nerve decompression. Cawthorne[9] reported that nerve sheath incision should be performed only for cases of complete paralysis. Altuntas *et al*.[2] performed decompression of the nerve and opened the epineural sheath, leaving the perineurium intact. In order to protect the nerve from a possible infection, Yetiser *et al*.[5] and Ikeda *et al*.[8] did not perform nerve sheath incision. We opened the fallopian canal from both ends in a limited area in 4 patients and decompressed from the geniculate ganglion to the stylomastoid foramen in another 4 patients. All of the 4 patients who underwent total decompression had this operation before 2001. Since 2001, however, a more conservative treatment modality (opening the canal in a limited area or without performing decompression) was preferred in COM without cholesteatoma. However, nerve sheath incision was not performed in any of the patients before or after 2001 in order to protect the nerve from a possible infection.

It has been reported that direct nerve involvement with infection along the nerve tissue rather than compression atrophy is the primary reason for the vast majority of FP cases due to COM.[4,5,10] It has also been stated that a dehiscent fallopian canal is not necessary for the entry of microorganisms. The infection may enter the canal through the canaliculi of stapedial and chorda tympani nerves.[5] Our results corroborate the above findings. In our study, all 5 patients who did not receive nerve decompression had successful outcomes.

Conclusion

In our opinion, therefore, intravenous antibiotic treatment in conjunction with steroid therapy should be the mainstay in managing FP resulting from COM without cholesteatoma. A tympanomastoidectomy should also be performed in order to close the tympanic membrane perforation and to eradicate the infection from the mastoid and middle ear cavity

permanently, improving the healing process and preventing the recurrence of infections. We always discourage nerve sheath incision since the sheath is a natural barrier to the spread of infection. Because our results are satisfactory, we do not have to decompress the facial nerve in noncholesteatomatous chronic otitis media.

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