e-ISSN: 0975-5160, p-ISSN: 2820-2651

Available online on www.ijtpr.com

International Journal of Toxicological and Pharmacological Research 2023; 13(12); 343-348

Original Research Article

Total Facial Nerve Decompression for Severe Traumatic Facial Nerve Paralysis

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Received: 11-09-2023 / Revised: 12-10-2023 / Accepted: 23-11-2023
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Conflict of interest: Nil

Abstract

Background: There has been debate over the years over the appropriateness and timing of facial nerve decompression for facial paralysis, as well as the amount of the anatomical decompression. Research shows that over the years, less surgical procedures have been performed. According to Chang and Cass' analysis of a substantial body of published data from 1966 to 1999 about the management of facial nerve injury caused by temporal bone trauma, patients who have normal facial nerve function following injury, regardless of how their condition progresses, those who present with incomplete paralysis but do not progress to complete paralysis, and those who have less than 95% degeneration on ENoG at initial admission typically do not need surgical intervention.

Aim: The aim of this study was to evaluate the facial nerve motor function and audiological results of the trans-mastoid facial nerve decompression surgery in the late period after traumatic FNP.

Material and Method: The Department of ENT has carried out this retrospective investigation. Computerized tomography was used as soon as possible for every patient. Every patient experienced facial paralysis with instant onset. The function of the facial nerve was assessed using the House-Brackmann (HB) grading system. Patients with facial paralysis underwent electromyography or, if feasible, electroneurography. During electroneurography, the excitation threshold, latency, and amplitude of the orbicularis oculi muscle were used to compare the paralyzed and normal sides. In accordance with the guidelines set forth by the institutional ethics committee, each research subject signed an informed consent form. Informed written consent was taken from the patients or their guardians willing to participate in the study. Those diagnosed within 3 days of onset also received antiviral therapy.

Results: When comparing the clinical improvement scores of patients who had surgery between 21 and 60 days (group I) to those who had surgery after 60 days, no discernible difference was seen. On the other hand, we observed that, for both simple functional gain and weighted functional gain, patients who had surgical decompression within 60 to 89 days (group IIa) had a statistically significant greater clinical improvement score than patients who had surgery within 90 days (group IIb). Using final HB grade and weighted functional gain as our metrics of facial nerve improvement, we found a weak connection between the duration of follow-up and improvement in facial nerve function.

Conclusion: The current investigation provides more proof that could bolster the idea that delayed decompression surgery can lower blood pressure. When appropriate, we advise facial nerve decompression for blood pressure within the first two weeks. However, our findings imply that, in the absence of a chance to proceed with surgery sooner, patients with excessive blood pressure may benefit from decompression surgery up to ninety-nine days following the onset of symptoms. More research is necessary to validate our results and clarify the function of postponed surgery in the BP therapy protocol, particularly for those who remain paralyzed for more than three months.

Keywords: Facial Nerve, Decompression, Facial Paralysis and Surgical Decompression.

Introduction

The most common symptom of facial nerve palsy (FNP) is unilateral facial muscle paresis or paralysis. Of cases of acute FNP, idiopathic FNP, or Bell's palsy, accounts for about 75%. [1] Although most patients with Bell's palsy recover from the functional nerve dysfunction following the initial insult [2], 30% of complete facial palsy patients exhibited an incomplete recovery that was

closely linked to aesthetic, psychological, and social problems. [3] Complete facial palsy is also known to be related with a bad prognosis when it is discovered during early examination. [4] To achieve the best possible therapeutic outcomes, therefore, knowledge of the prognosis and prompt management are essential, particularly in cases with Bell's palsy.

There are numerous potential causes of facial nerve paralysis (FNP), including tumors, infections, and trauma. Head traumas, temporal bone traumas, and idiopathic (Bell's palsy) are the most common causes of FNP. [5] FNP may also result from iatrogenic damage to the facial nerve sustained during otologic surgery. There is a chance of facial nerve damage with surgery for cerebellopontine angle lesions, congenital aural atresia, and chronic otitis media with or without cholesteatoma. [6] Ten percent of temporal bone fractures (TBF), which are usually caused by car accidents, might result in FNP. [7] They are classified as otic capsule-violating and otic capsule-sparing fractures. [8] Depending on the relationship between the fracture line and the long axis of the petrous bone, there is also a categorization that shows the linear, transverse, and mixed kinds. Twenty percent of TBFs result in facial paralysis, and eighty percent of TBFs are longitudinal. Twenty percent of TBFs are transverse fractures, and fifty percent of them involve FNP. [9] After examining 58 cases of facial nerve injury, Brodie and Thompson found that all patients with initial incomplete paralysis recovered, as did 8 out of 9 patients with delayed and 3 out of 5 patients with abrupt onset facial paralysis following surgical decompression. [10] After comparing the outcomes of patients with delayed-onset traumatic facial paralysis to those with immediate-onset facial paralysis, Mc Kennan and Chole discovered that 94% of patients with delayed-onset facial paralysis are likely to recover without the need for surgery. [11]

While there is strong evidence for initial conservative medical treatment in patients with BP [12-13], surgical decompression of the facial nerve remains a more controversial subject. Regarding the best surgical technique, degree of nerve decompression, and surgical scheduling, differing views exist. There are supporters of both the middle fossa and trans-mastoid techniques. According to several writers, the trans-mastoid technique is a low-complication therapeutic method that provides enough access to the facial nerve. [14] They have provided methods for decompressing the distal labyrinthine segment and the geniculate ganglion without compressing the temporal lobe. It has a tolerable risk profile for complications and, in their opinion, provides better access to the facial nerve medial to the geniculate ganglion as well. [15] The dispute about the degree of nerve decompression is intimately linked to the argument around the surgical method selection. Certain authors have suggested decompressing the facial nerve, particularly its meatal and labyrinthine parts. Nevertheless, the Cochrane review noted earlier relied solely on two trials that employed a trans-mastoid technique for FND. Nevertheless, a recent meta-analysis revealed that, when compared to medical treatment based on the House-Brackmann grading system (HBGS), FND does not result in a statistically significant improvement in facial nerve function. [16] Nevertheless, a noticeably greater degree of heterogeneity among the trials constrained the results. Therefore, it is necessary to reestablish the therapeutic significance of FND in the management of total Bell's palsy. This study aims to offer retrospectives of trauma patients who underwent total facial nerve decompression and developed complete facial paralysis.

Material and Methods

This Retrospective study has been conducted in the Department of ENT. All patients had computerized tomography at the earliest. All patients had immediate-onset facial paralysis. House-Brackmann (HB) grading system was used to evaluate the function of the facial nerve. Electromyography or electroneurography, if possible, was taken from the patients with facial paralysis. Excitation threshold, latency, and amplitude of orbicularis oculi muscle were used to compare normal and paralytic sides during electroneurography. Under the rule and regulation of the institutional ethical committee, signed informed consent was taken from every study subject. Informed written consent was taken from the patients or their guardians willing to participate in the study. Patients presenting early, within 2 weeks of the onset of BP, were treated medically with oral corticosteroids. Those diagnosed within 3 days of onset also received antiviral therapy. Surgical decompression was systematically offered to patients with persistent facial paralysis HB grade 5 or 6 who showed more than 90% denervation on electroneuronography. A total of 30 patients were selected based on the following Inclusion Criteria.

Inclusion Criteria: We studied the records of patients who met the following criteria:

- ✓ House–Brackman(HB) grade V and VI FNP one month after trauma
- ✓ Patients with traumatic FNP who were administered systemic corticosteroid treatment, with no observable recovery
- ✓ Patients with traumatic FNP who did not have any middle ear disease or cerebellopontine angle tumors
- ✓ Patients with traumatic FNP undergoing trans-mastoid approach (TMA) for FND.

Exclusion Criteria

- ✓ Patients recovering spontaneously or after medical treatment;
- ✓ Patients with HB grades I through IV
- ✓ Patients who needed another advanced surgical approach other than FND

Surgical Indications

- ✓ Presence of fibrillation, spike potentials, and total axonal degeneration on EMG (electromyography);
- ✓ Absence of volunteer potentials and facial movement;
- ✓ Fracture line apparently the crossing fallopian canal as seen on high-resolution computerized tomography (HRCT);
- ✓ Severe facial nerve paralysis (H–B grade V-VI).

Electromyographical Examinations were performed during the first examination. The HRCTs of temporal bone were obtained from all patients preoperatively. Fracture types and localizations were classified and noted. Audiometric evaluations were performed preoperatively and postoperatively in the 3rd and 12th months. Air–bone gap (ABG) values were calculated by subtracting bone PTA values from air PTA values. Hearing gains were calculated by subtracting the postoperative (first year after decompression) ABG values from the preoperative ones. Auditory brainstem response was tested in 1 patient who was a 3-year-old boy. Facial nerve function was graded according to the HB system both preoperatively and 12 months postoperatively. HB scale results of grade III or better were evaluated as good results

Surgical Technique:

Facial Nerve Decompression: Under general anesthesia, a complete mastoidectomy was performed through a retro auricular incision. The facial nerve was found in the posterior edge of the posterior tympanostomy. For most cases where the main insult was at the peri-geniculate region, if bony spurs could be eliminated effectively and the nerve could be decompressed, then the incus was not removed, especially in patients with a wide epitympanic region. However, the incudostapedial joint was separated and the incus was removed to access the peri-geniculate region if the traumatized area could not be achieved. The facial nerve bony canal was examined and removed 180 degrees, from the labyrinthine segment and the geniculate ganglion to the stylomastoid foramen, under the operating microscope. Bony spicules embedding in the nerve were removed during surgery. In the case of bilateral FNP, there was a complete nerve cutting on the left side.

Statistical Analysis: All statistical analyses were performed using SPSS version 19.0 (SPSS, Chicago, IL, USA). Data presented as ratios was analyzed using the Pearson $\chi 2$ test or Fisher exact 2-tailed test if there were fewer than 10 patients in any cell of a 2 × 2 grid. Parametric demographic and clinical data were analyzed using the ANOVA and t-test. Simple and weighted functional gains were evaluated using the Mann-Whitney test as these represented non-parametric data.

Result

Table 1: Demographic data and clinical characteristics of patients

Characteristic	Total (N = 30)	Decompression, 21–60 days (N = 14)	Decompression, 60–89 days (N = 10)	Decompression, ≥ 90 days (N = 6)
Age (Mean ± SD)	45.0 ± 12.2	44.0 ± 10.5	45.4 ± 15.1	48.3 ± 13.2
Male/female ratio	15/13	5/5	5/3	5/4
Median months of follow-up (range)	10.1 (1.0–97.3)	6.5 (1.0–24.3)	14.3 (1.0–97.3)	12.0 (2.0–84.0)
Affected side (Right/Left)	13/15	5/5	4/7	5/3

Our studied population included 15 male (50.0%) and 15 (50.0%) female patients. The mean age at diagnosis was 45.0 ± 12.2 years. Three (8.6%) patients had a recurrent condition at the time of assessment. Among our group of patients, 22 (72.2%) patients presented total facial paralysis (HB VI). ENoG studies were available for 8 (36.1%) patients, all of whom had 0% residual nerve function on testing. Follow-up visits of patients were between 30 days and 8 years postoperatively. A mild correlation was noted between the length of follow-up and improvement in facial nerve function when using final HB grade and weighted functional gain as our measures of facial nerve improvement. No correlation was noted between the length of follow-up and simple functional gain.

Table 2: Pre and Postoperative clinical parameters

Parameters	Total (n = 30)	Decompression21–60 days (n = 15) (Group I)	Decompression 60–89 days (n = 9) (Group IIa)	Decompression ≥ 90 days (n = 6) (Group IIb)
Patients treated with preoperative steroids (No %)	15 (52.8)	10(61.1)	4 (54.5)	1(28.6)
Preoperative EMG (No %)	10 (30.6)	5 (27.8)	4 (36.4)	1 (28.6)
Preoperative ENoG (No %)	12 (36.1)	7 (38.9)	4 (36.3)	1 (28.6)
Delay before surgery (Mean days, range)	60 (21–205)	35(21–57)	64 (62–79)	107 (90–205)
Initial Facial Function (HB Mean, ± SD)	4.3± 0.3	4.3 ± 0.3	4.4 ± 0.5	4.5± 0.4
Final Facial Function (HB Mean ± SD)	2.1 ± 0.6	2.1 ± 0.5	1.6 ± 1.0	3.0 ± 0.5
Simple Functional Gain (Mean ± SD)	1.2 ± 1.0	1.3 ± 1.0	1.6 ± 0.7	1.5 ± 1.1
Weighted Functional Gain (Mean ± SD)	1.4 ± 0.6	1.5 ± 0.6	1.1 ± 1.0	1.0 ± 0.4
Final HB score 3 or better (No %)	20 (61.1)	10 (61.1)	9 (81.8)	1 (28.6)
Final HB score 2 or better (No%)	5 (16.7)	3 (16.7)	2 (27.3)	0 (0)

A comparison of clinical improvement scores revealed no significant difference between surgical decompression in patients who underwent surgery 21 to 60 days (group I) compared to patients operated on after 60 days. However, we noted that patients who underwent surgical decompression between 60 and 89 days (group IIa) showed a statistically significant higher clinical improvement score in comparison to those operated on the past 90 days (group IIb) for simple functional gain and weighted functional gain.

Discussion

As far as we are aware, this study is one of the biggest evaluations of patients who had delayed surgical decompression for excessive blood pressure that was unresponsive to medication. The subgroup analysis indicates that patients with severe BP may benefit from decompression surgery up to 90 days after the beginning of symptoms, even if no significant differences were found between the two major comparison groups. This is especially true if there is no chance to continue with surgery earlier. Patients with refractory BP have shown inconsistent outcomes from delayed facial nerve decompression, according to prior research. Similar to our findings, Bodenez et al.2010 [17] have reported favorable outcomes with delayed facial nerve decompression in 13 patients with advanced facial paralysis who were operated on between 1 to 4 months from the onset of BP.

Li et al.2016 [18] examined the outcome of trans-mastoid decompression 2 months after the onset of symptoms in refractory BP patients who were initially treated with corticosteroids. At the three-month follow-up, patients who received surgery between two and three months after the beginning of symptoms showed a higher initial rate of improvement in facial function when compared to the control group. That difference, though, was absent from the follow-up visit after a year. Similarly, Kim et al.2016 [19] evaluated the effectiveness of delayed trans-mastoid facial nerve decompression between 3 weeks and 2 months in 12 patients with BP. In recent decades, as traffic and population have grown, so too has the prevalence of temporal bone damage and related facial nerve injury. [20] Traumatic facial nerve diseases are difficult to treat. The primary factors that determine the prognosis are the type of damage, whether it is abrupt or delayed in onset, whether it results in complete or partial paralysis, where the injury is located, and how severe the conduction block is based on the results of the electrophysiological testing. It is difficult to determine which form of fracture has the most chance of interfering with the nerve's path when there is cranial damage. It may or may not be associated with a temporal bone fracture. According to Coker et al., 14 out of 18 patients who required facial nerve exploration due to a temporal bone fracture also had a longitudinal fracture. [21]

When compared to delayed intervention, early intervention (within two weeks of beginning) produced better results, according to several studies. [22] A recent meta-analysis demonstrated that middle fossa decompression within 14 days of

symptom onset improved the HBGS compared to medical management. [23] Early intervention has been shown to ameliorate the risk of ischemia in the fallopian canal, which is correlated with prognosis. [24] Given that Wallerian degeneration originates from long-standing compression of the facial nerve in the fallopian canal [25] delayed intervention is likely to be associated with poor prognosis. Nonetheless, evidence of the benefits of delayed decompression surgery which occurs within 90 days after onset has increased for patients who cannot afford early surgery. [26]

The early use of oral steroids in the BP treatment algorithm is highly recommended by the most recent clinical practice standards. High-dose oral steroids, with or without antiviral medicine, have been found in several cases to result in a better recovery of nerve function when compared to placebo. In a recent double-blind randomized controlled study, Sullivan et al.2007 [27] reported that early treatment with prednisolone significantly improved the potential of complete recovery, up to 94.4% within 9 months compared to 81.6% for patients not taking steroids. These findings have been further supported by a recent Cochrane review. [28] There was no control group in our study where participants received conservative care solely from medication. However, a portion of our data is comparable to the results of cautious management found in existing literature. It would be most suitable to include in this comparison the subset of our patients who had oral steroids prior to surgery and who had at least a 6-month follow-up. Of our thirty subjects, eleven meet these requirements. Of these, 100% received HB 3 or above, while 45.5% received HB grade 2 or better. Previous studies' control groups recovered to HB 2 or better in 41.7–65% of cases, and improved to HB 3 or better in 81.1–94.4% of cases. [29]

The retrospective character of our study, the low percentage of patients who had pre-operative ENoG and EMG examinations, and the lack of a control group receiving just medicinal care are some of its shortcomings. Some patients were not able to receive an electrophysiological evaluation of their facial nerve function because of resource constraints in primary and secondary care facilities and delayed referrals to our tertiary care facility. We were unable to verify that, before surgery, >90% of our patients had lost their nerves. As a result, there's a chance that the effects of delayed decompression will be overstated. Furthermore, although being the most widely utilized instrument for the clinical evaluation of facial nerve function, the HB grading scale has built-in limitations with regard to its subjectivity, dependability, and longevity. [30]

Conclusion

The current investigation provides more proof that could bolster the idea that delayed decompression surgery can lower blood pressure. When appropriate, we advise facial nerve decompression for blood pressure within the first two weeks. However, our findings imply that, in the absence of a chance to proceed with surgery sooner, patients with excessive blood pressure may benefit from decompression surgery up to ninety-nine days following the onset of symptoms. More research is necessary to validate our results and clarify the function of postponed surgery in the BP treatment plan, particularly for patients who have persistent paralysis longer than three months. Even though early decompression is preferred, late decompression performed one to three months later can still produce satisfactory results for facial nerve function, particularly in patients who spent a significant amount of time in the intensive care unit. When treating traumatic FNP, the trans-mastoid approach is a useful technique that permits ossicular chain rebuilding in situations of trauma-induced CHL.

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