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ORIGINAL ARTICLE

Surgical management of facial paralysis resulting from temporal bone fractures

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Abstract

Conclusion: To achieve good facial reanimation in cases with facial paralysis resulting from temporal bone fractures, the ideal timing for surgical intervention is at least within 1 month of injury and an appropriate surgical approach should be selected depending on the site of facial nerve injury. *Objective:* This paper aimed to address the ideal time for surgical intervention and the appropriate surgical approach for patients with facial paralysis resulting from temporal bone fractures. *Methods:* We retrospectively investigated 60 patients with facial paralysis due to temporal bone fractures who underwent facial nerve decompression via different operative approaches within 1 month after trauma, of which 48 were surgically treated by the middle cranial fossa approach (80%), 8 by a pure transmastoid approach (13.3%), and four by a combined transmastoid and middle cranial fossa approach (6.7%). The House–Brackmann (H-B) grading system was used to evaluate the recovery of facial nerve function. *Results:* The follow-up period for all the patients was 1 year. Among 60 patients who were surgically treated, 39 achieved grade I of facial nerve function, 18 achieved grade II, two achieved grade III, and one achieved grade IV according to the H-B grading system.

Keywords: *Surgical approach, facial nerve injury, middle cranial fossa approach, transmastoid approach*

Introduction

Traumatic facial nerve paralysis accounts for about one-third of peripheral facial nerve paralysis 80% of which is caused by temporal bone fractures, and the remainder are associated with all kind of iatrogenic injuries, such as otologic and neurotologic surgery. A facial nerve function deficit was observed in 50% of these temporal bone fractures and was usually incomplete, delayed, and transitory [1]. Presently, the incidence of post-traumatic facial paralysis is decreasing due to the use of airbags and seat belts in motor vehicles [2]. In general, traumatic injury to the facial nerve requires exploration and repair. However, the detailed treatment of traumatic facial nerve paralysis is viewed as a Gordian knot all over the world, and different methods for dealing with facial nerve paralysis are still controversial. Surgical decompression of all nerve segments affected may be a good choice for facial paralysis. Appropriate routes need to be selected depending on the site of facial nerve injury and the timing for performing surgery is a problem that requires attention. That is the key for rehabilitating a paralyzed face. We conducted a retrospective study of 60 cases of peripheral post-traumatic facial paralysis resulting from temporal bone fractures. A total of 60 patients underwent facial nerve decompression via different surgical approaches and the postoperative outcome was assessed at our institution.

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Material and methods

Clinical material

Our sample included a total of 60 patients with facial paralysis following temporal bone fractures; 50 were male and 10 cases were female (Table I). The patients' ages ranged from 4 to 58 years, with a mean age of 29. Facial paralysis involved the right side in 38 patients and the left side in 22 cases. Facial paralysis occurred immediately in 50 cases, while in the other cases facial paralysis appeared 2 days after bone fractures. Spiral CT was employed to examine all the cases, and showed longitudinal fractures in 49 cases, transverse fractures in 8 cases, and mixed fracture in 3 cases. High-resolution CT (HRCT) demonstrated that the geniculate ganglion and the labyrinthine segment of the facial nerve were injured in 48 cases, the tympanic segment was identified in 8 cases, while the region from the labyrinthine segment to the pyramid segment was involved in 4 cases. Electromyography was used to assess the spontaneous activity of facial muscles. Facial nerve electrograms exhibited 90% or greater neural degeneration in all the patients, and a nerve excitability experiment showed that the deviation between the healthy side and the affected side was ≥3.5 mV. The facial nerve paralysis was all House-Brackmmann (H-B) grade V or VI. The Schirmer test was carried out in all the cases; 52 cases were positive and 8 cases were normal. None of the cases had a stapedial reflex according to tympanometry. Chemical gustometry showed that the taste sense of the anterior two-thirds of the tongue was reduced or was lost. As regards audio acuity, 10 cases were documented as having conductive hearing loss, 18 cases had sensorineural hearin loss, 30 cases had mixed hearing loss, and 2 cases had normal hearing. Fifty cases underwent facial nerve decompression within 1 month of onset of facial paralysis, and the other 10 cases underwent the treatment between 1 and 3 months.

Table I. Demographic and clinical information for the 60 patients with facial paralysis resulting from temporal bone fractures.

Feature	Value
Total no. of patients	60
Age (years)	
Mean	29
Range	4–58
Sex (F:M)	10:50
Timing of the onset of paralysis (days)	
Immediate	50
2 days after injury	10
Affected side (L:R)	22:38
Site of bone fracture	
Longitudinal	49
Transverse	8
Mixed	3
Segment of facial nerve injury	
Tympanic segment	8
Geniculate ganglion and labyrinthine segment	48
Labyrinthine segment to pyramid segment	4
Follow-up period (years)	1

Surgical procedure

The facial nerve decompression surgery via different approaches was carried out under general anesthesia. The surgical approach was determined by the portion of facial nerve affected. Forty-eight cases with geniculate ganglion and the labyrinthine segment injury according to HRCT were treated by the middle cranial fossa approach. A longitudinal cutaneous incision 2 cm before the tragus was made. A 3 × 4 cm section of bone flap was cut above the zygomatic arch. Then the brain meninges were separated and elevated from the middle cranial fossa floor, and the spinous foramen and arcuate eminence were exposed. About 0.5–1 cm behind the foramina spinosum was the greater superficial petrosal nerve. The bone was removed by electrical drill along with the greater superficial petrosal nerve to expose the geniculate ganglion. The labyrinthine segment and the tympanic segment were exposed and decompressed. Normally, the brain meninges do not adhere to the basis cranii, so if they are found, that is where the fracture line has occurred. Sometimes, the brain meninges impact in the sutura, so it should be peeled carefully to avoid avulsion. Generally speaking, the roof of the bony canal of the geniculate ganglion, labyrinthine segment, and tympanic segment should be removed so as to completely decompress the nerve. It was necessary to incise the nerve sheath to clear the hematoma. Then, rerouting, end-to-end suture, and covering of the fibers with biocolloid or gelatin sponge were carried out.

Eight of the 60 cases involved the tympanic section below the geniculate ganglion according to HRCT, and were decompressed via a pure transmastoid approach. In this approach, the mastoid cells were removed using an electrical drill, the tympanic cavity was exposed, and then the fallopian canal of the vertical segment of facial nerve was opened along with the fracture line. The incus and the head of the malleus were removed through the posterior tympanum, and the geniculate ganglion was reached. Then we cut the

nerve sheath to release the compression, and agglutinated the nerve fibers end-to-end if the fibers were broken. Finally, the incus was replaced.

A combined transmastoid and middle cranial fossa approach was performed in the four patients with injury of the geniculate ganglion and tympanic segment. Generally speaking, decompression was carried out first via the middle cranial fossa approach, then via the transmastoid route.

Results

In the 48 cases that were treated via a middle cranial fossa approach, the greater superficial petrosal nerve could not be found in three cases, and was completely detached in four cases. The labyrinthine segment exhibited facial nerve congestion and edema in 15 cases, the nerve sheath was broken in 4 cases, and bone fragments constricted the nerves in the bottom of the internal acoustic meatus in 3 cases. Congestion and edema occurred at the tympanic segments of facial nerves in 10 cases, and the bone fractures constricted this segment in 3 cases. The geniculate ganglion was injured in different situations, which showed congestion and edema in 21 cases, bone fragments constricting the segments in 9 cases, and hyperosteogeny and compression in 1 case. The nerve sheath was injured in seven cases, there was a partial tear in nine cases, and it was completely ruptured in one case that underwent neuroanastomosis. The sensorineural deafness was exacerbated after operation in one case. Cerebrospinal fluid leaked in two cases, but after the use of a strengthening pack in the operative region for 5 days, the leakage stopped; no other treatments were given.

In the eight cases where the transmastoid approach was employed, we found that the tympanic segment of the facial nerves exhibited edema to varying degree. In four cases, granulation tissue was found around the facial nerve. Bone fragments surrounded the tympanic segment in three cases. No nerve fracture was found. One patient with normal hearing showed mild conductive hearing loss after operation. Mild conductive hearing loss arose in the other seven cases before operation, but the deafness appeared more serious in various degrees following the surgery.

In the four cases that underwent decompression of the entire section of the facial nerve by the transmastoid and middle cranial fossa approach, we found that edema emerged in the geniculate ganglion and the tympanic segment; bone fragments constricted the geniculate ganglion in two cases. After incising the neurilemma, effusion, hematoma or the facial nerve bulge was seen, but no nerve fracture was found. The audition test demonstrated all mixed deafness before operation. After operation, the air-conduction threshold improved but no change was seen in bone conduction.

The H-B grading system was employed to evaluate the function of facial muscles. The function started to recover 2 weeks after surgery. The results of the 60 cases were as follows. After 2 months, 3, 10, 35, and 12 cases achieved H-B grade I, II, III, or IV, respectively; and after 1 year, 39, 18, 2, and 1 cases achieved grades I, II, III, or IV, respectively (Figure 1). The single case that was evaluated as grade IV was the one that had a completely detached facial nerve.

Discussion

Temporal bone fractures are a part of head trauma, which accounts for about 15–48% of skull fractures, and often happens in traffic accidents. Usually, the

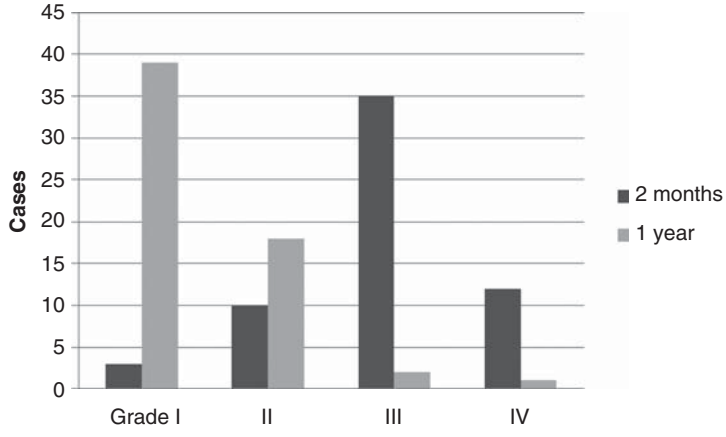

Figure 1. Facial nerve function 2 months and 1 year after surgical management in a total of 60 cases, assessed by the House-Brackmann (H-B) grading system.

fractures can be classified as either longitudinal, transverse or mixed fracture, according to the relationship between the fracture line and the long axis of the petrous pyramid. In fact, temporal bone fractures are rarely unilateral [3]. Transverse fractures are more likely to cause facial nerve damage, occurring in about 40–50% of fractures, but facial paralysis only occurs in 10–20% of longitudinal fractures. However, regardless of the kind of fracture, there is some dispute in the literature concerning the role, timing, and type of surgery for the management of traumatic facial paralysis [4]. Up to now, athough there are still issues regarding the timing of surgical intervention [5], the mainstream view is that the earlier the surgical decompression is carried out, the greater the efficacy. In Fisch's opinion [5,6], surgical operation should be performed immediately in patients with neural degeneration of the facial nerve >90% by facial nerve electrogram (the timing for performing electroneurography after temporal bone fracture should be defined) [7]. Naohito Hato et al. indicated that the ideal time for facial nerve decompression surgery was within the first 2 weeks after the trauma. The reported rate of complete recovery was 85.7% [8]. But in the abstract, motor neuron cell bodies recover 21 days after damage; at that time, constructive metabolism is at its most vigorous. So if facial nerve decompression is performed at that time, the curative effect would be most beneficial. On the other hand, it is easier to locate the damaged position of the nerve at the early stage of onset, the nerve fibers are easier to exposed and have no tension. Too long a time after injury, the nerves are prone to shrink, or scars occur at the broken end, so neuromas are formed and the peripheral organs atrophy, which is not beneficial for functional recovery of the facial nerve. Ylikoski [9] reported two cases of facial paralysis resulting from temporal bone fractures that underwent exploratory operation 1 year after injury. The pathology showed considerable endoneural fibrosis, with only little nerve fiber surviving. Ylikoski believed that endoneural fibrosis is the main reason for nerve dysfunction, and it is endoneural hematoma that induces endoneural fibrosis. Early operation may remove compression of hematoma, prevent anabatic neurodegeneration, decrease inogenesis in the nerve, and stimulate nerve fiber regeneration and directional control. In addition, judging from the facial nerve edema, the earlier the surgery is given, the milder the nerve edema, and the facial nerve can obtain better recovery. Our study documented that 57 cases (95%) reached above H-B grade II functional recovery when the surgery was performed within 1 month after trauma, compared with 78% H-B grade I or II recovery reported by Quaranta et al., where patients were operated in 1–3 months [10]. In our opinion and considering the views mentioned above, to achieve satisfactory efficacy, the timing for undergoing surgical intervention is at least within 1 month of onset of paralysis.

For the patients who suffer from facial paralysis resulting from temporal bone fractures and need surgical treatment, it is very important to determine the site of injury, which is the key to determine the surgical approach, i.e. incranial or extracranial [11]. Presently, there are several types of approaches, such as the middle cranial fossa approach, pure transmastoid approach, and transcanal approach. Fisch [6] believes that in most transverse temporal bone fractures the area of the injury is in the labyrinthine segment (about 80%); on the other hand, in the majority of longitudinal temporal bone fractures the area of injury is the geniculate ganglion [12]. Clinically, HRCT scan is the main method for identifying the damaged sites. In our study of 60 cases, the results of HRCT scans are basically the same as for exploratory operation, which proves that HRCT is very important in the diagnosis of facial paralysis due to temporal bone fractures and determination of the damaged portion of the facial nerve. Additionally, we can also identify the damaged position by functional lesion of the facial nerve branches, for instance: the Schirmer test, stapedius reflex, and chemical gustometry. Panda et al. [13] believe that the Schirmer test can be regarded as the main diagnostic criterion to identify the damaged position. However, clinical inspection showed that the tests listed above could only have diagnostic value in patients with complete paralysis, and were not absolutely reliable. In our research, the Schirmer test was positive in 45 cases and normal in 15 cases. Although it can be seen as an important criterion, the affected position can only be accurately diagnosed by combining it with medical history, HRCT scan, examination of the nervous system and ear; the HRCT scan is especially valuable in the diagnosis. Our data indicate that injuries at the labyrinthine segment of the facial nerve and geniculate ganglion were found in 48 cases, tympanic segment injuries in 8 cases, and both geniculate ganglion and tympanic segment in 4 cases, which is in general accord with Fisch's report [6]. However, other studies [14] have discovered that the sites of injury are mostly the tympanic segments in patients with paralysis resulting from temporal bone fractures. According to preoperative judgments, in the present study the damaged portion was located before the geniculate ganglion in 52 cases, and this generally agreed with surgical exploration. The reason for the disagreement with results quoted above may be related to the differences in cases that were collected,

and the range of the decompression surgery was not enough to reach the damaged position. As a result, for the facial paralysis resulting from temporal bone fractures, the operative approach should be determined by the site of facial nerve injury as defined preoperatively. For patients whose facial nerve injuries are located before the geniculate ganglion, the middle cranial fossa approach is essential. The transmastoid approach has a limited surgical field and is inconvenient to perform, leading to an insufficient decompression range to the geniculate ganglion, even seriously influencing the aural comprehension. Of the 60 cases in our study, 48 cases were treated via the middle fossa approach, 8 cases by the transmastoid approach, and 4 cases by the transmastoid and middle cranial fossa approach. A 1-year follow-up period showed that 57 cases reached above H-B grade II, which suggests a satisfactory curative effect. A combined approach should be used in those patients whose temporal bone fractures are complicated, so that the best decompression effect can be achieved.

Conclusion

To sum up, for cases with facial paralysis resulting from temporal bone fractures, in order to achieve the best facial nerve functional recovery, the ideal time is at least within 1 month and the appropriate surgical approach should be selected based on the differing sites of facial nerve injury according to preoperative evaluation.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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