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Case Report

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A case of facial nerve palsy caused by severe head injury treated by translabyrinthine approach

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ABSTRACT

Background: Several treatments for traumatic facial paralysis have been reported, but the role of surgery is still controversial.

Case Description: A 57-year-old man was admitted to our hospital with head trauma due to a fall injury. A total body computed tomography (CT) scan showed a left frontal acute epidural hematoma associated with a left optic canal and petrous bone fractures with the disappearance of the light reflex. Hematoma removal and optic nerve decompression were performed immediately. The initial treatment was successful with complete recovery of consciousness and vision. The facial nerve paralysis (House and Brackmann scale grade 6) did not improve after medical therapy, and thus, surgical reconstruction was performed 3 months after the injury. The left hearing was lost entirely, and the facial nerve was surgically exposed from the internal auditory canal to the stylomastoid foramen through the translabyrinthine approach. The facial nerve's fracture line and damaged portion were recognized intraoperatively near the geniculate ganglion. The facial nerve was reconstructed using a greater auricular nerve graft. Functional recovery was observed at the 6-months follow-up (House and Brackmann grade 4), with significant recovery in the orbicularis oris muscle.

Conclusion: Interventions tend to be delayed, but it is possible to select a treatment method of the translabyrinthine approach.

Keywords: Facial nerve reconstruction, Greater auricular nerve graft, Mastoidectomy, Severe head trauma, Translabyrinthine approach

INTRODUCTION

Several treatments for traumatic facial paralysis have been reported, but the role of surgery is still controversial.^[11] Surgical treatment in the acute phase is recommended for severe paralysis at the onset.^[4,8] In contrast, surgical treatment of facial nerve paralysis is usually delayed for severe head trauma cases until the patient's condition stabilizes. Importantly, facial nerve paralysis due to

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severe head injury is often associated with inner ear disorder, allowing the surgeon to use the translabyrinthine approach to decompress the petrous portion of the facial nerve from the internal acoustic meatus to the stylomastoid foramen. This wide decompression allows the surgeon to identify the injured portion of the nerve for reconstruction.

Here, the authors report a case of facial nerve paralysis due to infratemporal blunt trauma, which was treated by surgical decompression and reconstruction of the facial nerve using a greater auricular nerve graft through the translabyrinthine approach. We analyze and discuss intraoperative anatomy, surgical techniques, and postoperative outcome of the case.

CASE REPORT

A 57-year-old man fell from a height of 1.5 m during his work shift and was transported to our hospital's emergency room. He had minor consciousness impairment at arrival (GCS; E4V4M6) but no remarkable facial palsy. A total body CT scan showed a thick left frontal acute epidural hematoma associated with skull fractures of the left frontal, petrous and occipital bone, the clivus, the lateral wall of the left orbit, and the maxilla. The petrous bone fracture was a mixed pattern [Figure 1]. Neurological examination revealed left eye blindness with anisocoria (left pupil larger). Thus, emergency surgery was immediately performed for hematoma removal and left optic nerve decompression. A postoperative CT scan revealed the formation of another acute extradural hematoma at the left posterior fossa, and therefore, reoperation was immediately performed. This hematoma was caused by a laceration of the sigmoid sinus caused by a left occipital bone fracture; the sigmoid sinus was sutured to stop the bleeding, and the hematoma was evacuated. Left facial nerve damage was also suspected due to a mixed longitudinal and horizontal petrous bone fracture [Figure 2]. Lifesaving procedures were prioritized at that point, and no surgical treatment for the left facial nerve injury was made. On the 2nd postoperative day, a neurological examination revealed a left facial nerve paralysis (House and Brackmann grade 6) associated with ipsilateral ear deafness. Visual acuity gradually improved postoperatively, allowing the patient to perform daily activities at the 3-month follow-up. However, facial nerve palsy did not improve and remained stable with an H-B grade VI [Figures 3a and b].

Tear production was preserved, but the sense of taste at the anterior two-thirds of the tongue was reduced. The authors hypothesized that the facial nerve injury was between the geniculate ganglion and the origin of the chorda tympani and since the patient's facial palsy is an immediate type, no improvement was observed by conservative treatment. Thus, we judged that reconstruction was indicated for the patient.

The translabyrinthine approach was utilized to expose the facial nerve from the internal auditory meatus to the stylomastoid foramen.

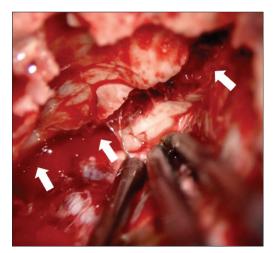


Figure 2: The temporal bone fracture was confirmed when the left suboccipital hematoma was removed (white arrow).

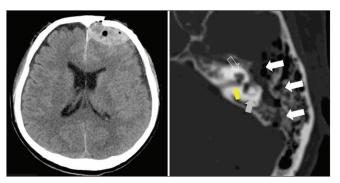


Figure 1: Preoperative CT showed left frontal epidural hematoma and temporal bone fracture (white arrow). The yellow, open white, and gray arrows indicate the internal auditory canal, cochlear, and lateral semicircular canal, respectively.



Figure 3: Follow-up at 6 months postoperative showing recovery of both orbicularis oris function and lower lip depressors. (a and b) before reconstruction surgery.

The facial nerve injury was intraoperatively confirmed along the fracture line near the distal side of the geniculate ganglion, and granulated tissues covered it. After dissecting the facial nerve, a greater auricular nerve graft was harvested, and the facial nerve was reconstructed using the greater auricular nerve as an interposition graft [Figure 4]. Functional recovery was confirmed mainly in the left orbicularis oris muscle about half-year after the reconstruction surgery (House and Brackman grade 4) [Figures 5a and b].

DISCUSSION

Facial nerve paralysis associated with temporal bone fractures occurs in 7–10% of temporal bone fractures and is accountable

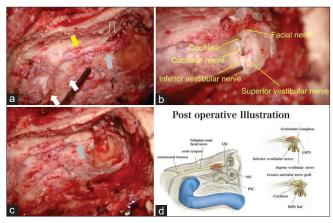


Figure 4: Intraoperative image of the facial nerve decompressed in the mastoid (yellow arrow) and tympanic (open white arrow) segments; (left ear, mastoidectomy, and posterior tympanotomy were performed, luxated incus and head of the malleus were removed). White arrows point to the fracture line. The gray arrow indicates a granulated injured facial nerve in (a) and replaced by a greater auricular nerve in (c). Normal facial nerve was confirmed by translabyrinthine approach (b). (d) A schematic image of recontraction surgery.



Figure 5: Follow-up at 6 months postoperative showing recovery of both orbicularis oris function and lower lip depressors. (a and b) after reconstruction surgery.

for most traumatic facial nerve paralysis.^[7] According to the literature, blunt head trauma can lead to facial nerve injury in approximately 1.5%. Facial nerve injury mechanism includes nerve rupture and extension, bleeding and edema in the bone canals, and direct nerve compression and laceration by the fractured bone fragments.^[11] For this variety of reasons, there is no unanimous consent on the timing of surgery, the site of the facial nerve injury, the most appropriate surgical approach, and the outcome of the surgical procedures.^[7] Xu *et al.* reported that most injuries lead to contusion, stretching, intraneural hematoma, and fibrosis formation around the nerve, with few cases of nerve continuity interruption.^[19]

There are many reports of facial nerve decompression to treat traumatic facial nerve paralysis. When the facial nerve is transected, direct cooptation is the best choice of treatment, and a nerve interposition autograft should be employed if the motor end-plates are still intact and direct end-to-end anastomosis is not feasible.^[10,16]

Facial paralysis is classified as immediate paralysis if the onset is within 24 h after trauma and delayed paralysis is when the onset is after 24 h. Immediate paralysis is likely to occur when direct damage such as traction or compression by bone fragments occurs. Delayed paralysis is more likely to happen when the nerve is indirectly injured by hematoma, edema, or compressed by surrounding swollen tissues.^[11]

Spontaneous recovery is unlikely to occur for immediate or severe paralysis, and Fisch reported that complete facial nerve palsy within 6 days of onset needs immediate decompression with other institutions adopting this surgical intervention strategy.^[2,4,12-14,17,20] There are many reports that decompression was effective, especially when interventions were performed in the acute phase.^[3,5,6,9,18,19]

However, most patients with temporal bone fractures due to severe head injury suffer from intracranial or systemic complications and present with impaired consciousness. Facial nerve function assessment is challenging for patients with impaired consciousness,^[1] and poor clinical conditions delay intervention.

On the other hand, facial nerve paralysis due to severe head injury is often accompanied by inner ear disorder, which allows a translabyrinthine approach that can offer wide facial nerve decompression. Wide facial nerve decompression exposes the damaged part of the nerve, allowing nerve reconstruction. Several techniques have been described for facial nerve reconstruction.^[7,11] Ricciardi *et al.* reported no difference in facial paralysis recovery between the direct coaptation and the interposition graft technique.^[15] Since there are reports that surgical intervention can improve facial paralysis even up to 2 years after injury, it is paramount to tailor the treatments of these traumatized patients depending on their clinical condition.

CONCLUSION

Interventions tend to be delayed for facial paralysis associated with severe head trauma. On the other hand, it is possible to select a treatment method that exposes the facial nerve from the brain stem to the stylomastoid foramen using the translabyrinthine approach since inner ear injuries are likely to be present for these patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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