Med & Health Jul 2026; 21(2): 1160-1165 https://doi.org/10.17576/MH.2026.2102.38

CASE REPORT

Traumatic Facial Nerve Palsy: A Case of Delayed-Onset High-Grade Paralysis Managed with Surgical Decompression

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Received: 11 September 2025 / Accepted: 12 February 2026

ABSTRAK

Kelumpuhan saraf muka traumatik (FNP) menyumbang kepada kira-kira 17% daripada semua kes FNP periferi dan sering berlaku dalam kalangan pesakit yang mengalami retakan tulang temporal. Pengurusan FNP traumatik masih menjadi isu yang kontroversial, terutamanya berkaitan peranan dan masa yang sesuai untuk intervensi pembedahan. Kami melaporkan kes seorang lelaki berusia 24 tahun yang mengalami FNP sebelah kanan dengan permulaan lewat (House–Brackmann Gred V) selepas kemalangan jalan raya. Setelah dirujuk ke pusat kami, penilaian elektrodiagnostik menunjukkan degenerasi saraf yang teruk. Walaupun telah menerima rawatan konservatif yang mencukupi, tiada penambahbaikan klinikal diperhatikan. Oleh itu, dekompresi saraf muka telah dilakukan sebagai keputusan klinikal yang disesuaikan dengan keadaan pesakit. Selepas pembedahan, pesakit menunjukkan pemulihan fungsi yang ketara. Kes ini menekankan kepentingan pemilihan pesakit yang teliti serta pendekatan membuat keputusan secara individual apabila mempertimbangkan intervensi pembedahan dalam kes FNP traumatik yang bermula lewat, teruk dan berterusan.

Kata kunci: *Lumpuh saraf muka; pembedahan dekompresi; pembebasan saraf; traumatik*

ABSTRACT

Traumatic facial nerve paralysis (FNP) accounts for approximately 17% of all peripheral FNP cases and is frequently observed in temporal bone fractures. Its management is still controversial and remains a subject of debate, particularly concerning the optimal timing of surgical intervention. We report a case of a 24-year-old male who developed delayed onset right-sided FNP (House-Brackmann Grade V) following a road traffic accident, On referral to our centre, electrodiagnostic studies demonstrated severe nerve degeneration. Despite adequate conservative management, there was no clinical improvement. Consequently, facial nerve decompression was undertaken as an individualised clinical decision. Postoperatively, the patient demonstrated marked functional recovery. This case underscores the importance of careful patient selection and individualised decision-making when considering surgical intervention in delayed-onset, severe, and persistent traumatic facial nerve palsy.

Keywords: Decompressive surgery; facial nerve palsy; neurolysis; traumatic

INTRODUCTION

Traumatic facial nerve paralysis (FNP) accounts for approximately 17% of all peripheral facial paralysis cases, with 7-10% of temporal bone fractures being associated with facial nerve dysfunction (Junior et al. 2009; Odebode & Ologe 2006; Ragaban et al. 2024). Depending on its severity, traumatic FNP can result in significant morbidity and negatively affect quality of life. Despite advances in imaging and electrophysiological assessment, the management of traumatic FNP, particularly delayed-onset paralysis remains clinically challenging.

Electrophysiological studies, particularly electroneurography (ENoG), are commonly employed to assess the extent of axonal degeneration and to guide management decisions. Established surgical indications, such as greater than 90-95% degeneration within the first two weeks, are primarily derived from studies involving immediate-onset complete facial paralysis (Shah et al. 2020; Walker et al. 2023). The optimal timing for surgical exploration of traumatic FNP remains controversial, with differing opinions on whether early or delayed intervention yields better outcomes (Hato et al. 2011). In contrast, clear evidence-based criteria for surgical intervention in delayed-onset traumatic FNP are lacking, and management strategies remain controversial. Consequently, most delayed-onset cases are initially managed conservatively with corticosteroids and close observation (Turel et al. 2005).

Nevertheless, a subset of patients with delayed-onset traumatic FNP may exhibit persistent high-grade paralysis, plateaued recovery or significant functional impairment despite appropriate conservative therapy. In such situations, surgical decompression may be considered on an individualised basis, although supporting evidence remains limited.

Herein, we reported a case of delayed-onset, high-grade traumatic facial nerve palsy in a young adult following a motor vehicle accident, who failed conservative management and subsequently underwent facial nerve decompression with favourable functional recovery. This case aimed to contribute to the ongoing discussion regarding individualised decision-making in the management of delayed-onset traumatic FNP.

CASE REPORT

A 24-year-old Malay male with no prior comorbidities was involved in a motor vehicle accident, sustaining trauma to the right side of his head. Post-trauma, he experienced self-limiting right ear bleeding without clear watery otorrhea, vertigo or hearing loss. Four days later, while at home, he developed right-sided facial weakness, initially able to close the eye, which progressed over two days to complete inability to close the eye. He sought medical attention at a nearby clinic, where he was prescribed oral prednisolone 60 mg for 14 days and referred to our center due to worsening facial asymmetry. Despite the completion of steroids, there was no clinical improvement.

On day 18 post-trauma, the patient was fully conscious. Neurological examination revealed a right lower motor neuron facial nerve palsy, House-Brackmann (HB) (Grade V, with Bell phenomenon present (Figure 1). Otoscopy showed minimal blood clots at the floor of the external auditory meatus, an intact tympanic membrane and no hemotympanum. Tuning fork examination demonstrated a positive Rinne bilaterally and Weber lateralised to the right, indicating mild right-sided conductive hearing loss with air-bone gap <15 dB. Nasoendoscopy and other examinations were unremarkable. Pure tone audiometry confirmed normal hearing bilaterally.

Given persistent high-grade facial palsy, lack of improvement after conservative therapy, and functional concerns, ENoG was performed on day 19, revealing 64% degeneration. Temporal bone computed tomography (CT) did not show bony spicules or direct impingement on the facial nerve, only an irregular bony wall near the first genu and tympanic segment of the right facial canal, which was suspicious of fracture (Figure 2). Considering the plateaued recovery and

FIGURE 1: Pre-operative photograph taken 19 days post-trauma of right longitudinal temporal bone fracture, showing right facial nerve palsy House-Brackmann Grade V

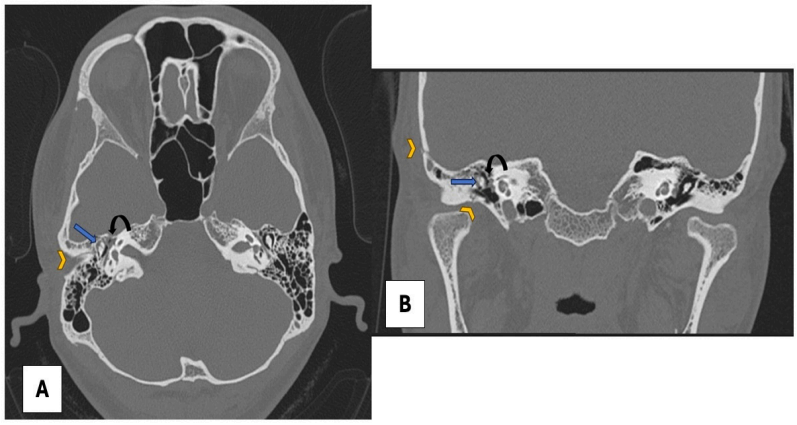

FIGURE 2: High-resolution computed tomography temporal in (A) axial and (B) coronal views showed an undisplaced longitudinal fracture of the right temporal bone extending to the petrous apex (yellow arrowhead) with diasthesis of the right incudomalleolar joint (blue arrow). The first genu and the tympanic segment of the right facial canal wall appearred irregular, suspicious of fracture, with fluid within the middle ear cavity (curved black arrow)

progressive deficits, transmastoid facial nerve decompression and neurolysis were performed (Figure 3).

Postoperatively, the patient demonstrated significant improvement, achieving HB Grade II function (Figure 4). Early structured facial rehabilitation, including neuromuscular retraining and synkinesis prevention was initiated, contributing to optimised functional recovery.

DISCUSSION

Traumatic FNP occurs when the facial nerve is injured following trauma, most commonly from temporal bone fractures secondary to blunt or penetrating head injury. Traumatic FNP accounts for approximately 17% of peripheral facial paralysis, with temporal bone fractures contributing 4-10% of cases (Junior et al. 2009; Odebode & Ologe 2006; Ragaban et al. 2024). Road traffic accidents are the predominant

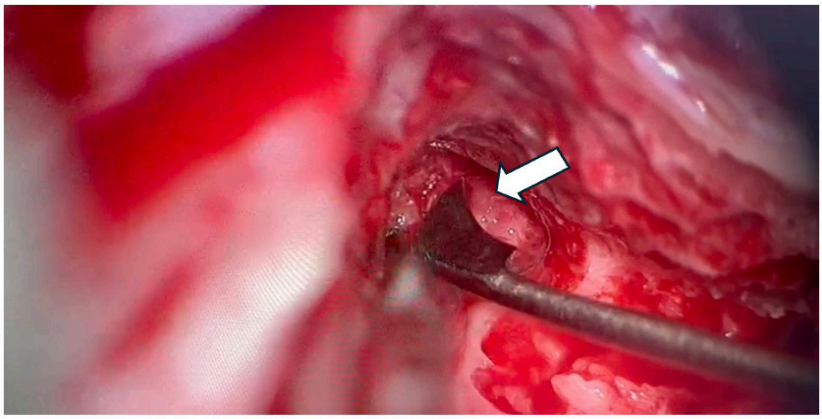

FIGURE 3: Neurolysis was performed using a sickle knife (white arrow)

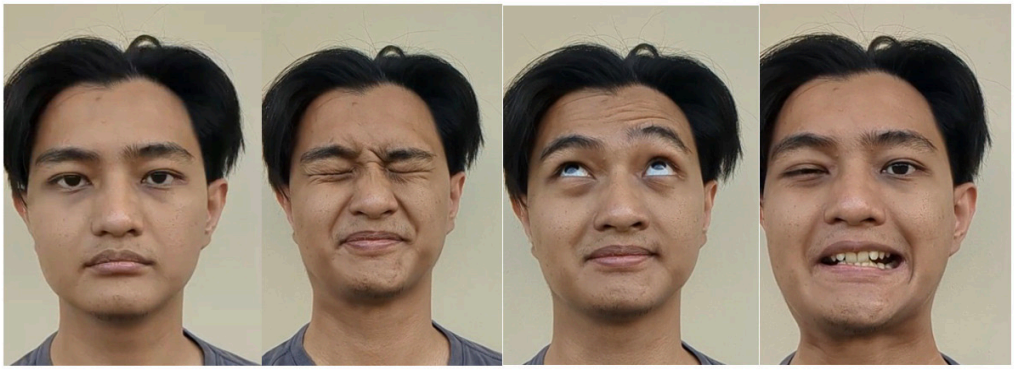

FIGURE 4: Post-operative photographs, 5 months after right facial nerve decompressive surgery, showed recovery of the facial nerve to HB II from HB V (HB: House-Brackmann)

cause. Epidemiological studies show a male predominance and increasing incidence with age (Odebode & Ologe 2006; Ragaban et al. 2024).

The facial nerve is particularly vulnerable due to its long, tortuous course within the temporal bone. Mechanisms of injury include compression, contusion, transection, or ischemia secondary to edema or displaced bone fragments (Walker et al. 2023). Fracture type influences severity: transverse fractures often cause immediate and severe FNP, whereas longitudinal fractures more commonly produce delayed-onset, milder deficits (Shah et al. 2020; Walker et al. 2023). Our patient sustained a longitudinal fracture with delayed-onset HB Grade V facial palsy.

Delayed-onset traumatic FNP is generally attributed to secondary mechanisms such as progressive neural edema, ischemia or intraneural hematoma rather than direct nerve transection. Consequently, most cases demonstrate favourable spontaneous recovery with conservative management, including corticosteroid therapy and close observation.

ENoG is a valuable tool in assessing axonal degeneration, but it should only be performed after 72 hours post-injury to allow for Wallerian degeneration (Lee 2016). Surgical exploration is generally recommended in cases of immediate-onset complete FNP when ENoG demonstrates >90-95% degeneration within the first two

weeks. (Shu et al. 2023; Ulug & Ulubil 2005; Xu et al. 2017). However, evidence guiding surgical intervention in delayed-onset facial nerve palsy remains limited and controversial, and no universally accepted electrophysiological thresholds currently exist for this subgroup.

In the present case, despite ENoG showing 64% degeneration, which is below conventional surgical thresholds, facial nerve decompression was undertaken as an individualised clinical decision, based on persistent HB Grade V paralysis, failure of conservative management, plateaued recovery and significant functional impairment. Although computed tomography did not demonstrate overt bony impingement, subtle intraneural edema or compression cannot be reliably excluded on imaging.

The transmastoid approach was selected due to the anatomical involvement of the tympanic and mastoid segments and for the preservation of normal hearing, avoiding more invasive approaches like those in the middle cranial fossa or translabyrinthine approaches. Alternative minimally invasive approaches, such as endoscopic transcanal decompression, are limited to the tympanic segment and require precise technique (Aboud et al. 2013; Shu et al. 2023).

Postoperatively, the patient demonstrated significant functional improvement, achieving HB Grade II. Early initiation of structured facial rehabilitation likely contributed to recovery by promoting neuromuscular retraining and reducing the risk of synkinesis. This outcome suggests that, in carefully selected cases, surgical decompression may still offer benefit despite subthreshold ENoG values.

Limitation

This report describes a single patient, and the findings cannot be generalised to all cases of delayed-onset traumatic facial nerve palsy. The absence of robust, high-level evidence supporting surgical intervention in delayed-onset cases with less than 90% ENoG degeneration must be acknowledged. Additionally, spontaneous recovery cannot be entirely excluded as a contributing factor to postoperative improvement. Nevertheless, this case highlights the complexity of decision-making in delayed traumatic FNP and underscores the importance of individualised, patient-centered management when recovery plateaus despite conservative treatment.

CONCLUSION

Delayed-onset traumatic FNP often recovers spontaneously, but persistent high-grade deficits may warrant surgical intervention. In this case, despite 64% ENoG degeneration and no detectable bony impingement on CT, transmastoid decompression was justified due to plateaued recovery, progressive asymmetry and functional impairment. Postoperative improvement to HB Grade II underscores the potential benefit of individualised surgical decision-making in select delayed-onset cases. Early structured facial rehabilitation is essential to maximise recovery and prevent synkinesis. This case demonstrates the importance of integrating clinical progression, electrophysiology, imaging and patient-centered priorities in managing delayed-onset traumatic FNP.

Author contributions: All authors were involved in the conception and design of the manuscript, drafting the paper, revising it critically for intellectual content and final approval of the version to be published.

Funding: The authors receive no external funding.

Conflict of interest: The authors declare no conflicts of interest.

Informed consent statement: Consent was obtained from the patients for participation in the study, publication and any accompanying images.

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