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Delayed Post-traumatic Bilateral Facial Nerve Paralysis: A Case Report

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Abstract

Bilateral facial nerve palsy is a rare complication of craniofacial trauma, particularly following temporal bone fractures. Its delayed onset may complicate early recognition, especially when facial movements are initially preserved. Clinical progression, fracture characteristics, and electrophysiological findings are important considerations in diagnostic assessment and management.

We report the case of a man in his early 40s who sustained cranioencephalic and thoracoabdominal trauma following a fall from approximately 5 m. Computed tomography revealed bilateral temporal bone fractures, including a transverse fracture involving the left perigeniculate region and a longitudinal fracture involving the right mastoid segment of the facial nerve canal, together with additional facial fractures. Facial movements were initially preserved. On the fourth day after trauma, the patient developed left-sided peripheral facial weakness, followed by contralateral involvement within hours, progressing to severe bilateral facial nerve palsy (House-Brackmann grade V) by the fifth day. Early electroneurography demonstrated markedly reduced left-sided motor responses compared with relatively preserved right-sided responses, although the examination preceded the development of clinically evident right-sided paralysis. Audiological assessment revealed asymmetric bilateral hearing loss, more pronounced on the left. Conservative management included systemic corticosteroids, ocular protection, and facial physiotherapy. No significant improvement in facial function was observed at discharge on day 10. At one month, right-sided facial function had recovered completely, while partial improvement was observed on the left. At the final seven-month follow-up, only mild residual left-sided weakness remained (House-Brackmann grade II).

This case illustrates the importance of serial clinical assessment in patients with temporal bone fractures, as facial nerve palsy may develop despite initially preserved facial movements. It also highlights the limitations of early electrophysiological findings in predicting long-term functional recovery. Although substantial improvement was observed following conservative management, the contributions of treatment and spontaneous nerve recovery cannot be distinguished in a single case. Therapeutic decisions should therefore be individualized according to clinical progression, imaging findings, and the patient's overall condition.

Categories: Trauma, Other, Physical Medicine & Rehabilitation
Keywords: bilateral facial nerve palsy, craniofacial trauma, delayed-onset facial palsy, facial nerve rehabilitation, temporal bone fracture

Introduction

Facial nerve palsy is a potentially debilitating complication of temporal bone fractures, resulting in impaired facial movement and potentially affecting eye protection, speech, and facial expression. Although unilateral facial nerve palsy is more frequently encountered following trauma, bilateral involvement is exceptionally rare and may be particularly challenging to recognize when facial weakness is symmetric.

The facial nerve (cranial nerve VII) controls the muscles of facial expression and follows a complex course through the temporal bone within a narrow bony canal before exiting the skull through the stylomastoid foramen. This anatomical relationship makes the nerve vulnerable to injury following temporal bone trauma, particularly near the geniculate ganglion, where the nerve changes direction [1-4]. Facial nerve injury may result from direct disruption or compression by displaced bone fragments, as well as indirect mechanisms such as edema, hematoma formation, or ischemia. Paralysis may develop immediately after trauma, potentially indicating severe structural nerve injury, or appear hours to days later (delayed-onset palsy), more commonly reflecting progressive swelling or compression within the facial canal.

Temporal bone fractures have traditionally been classified as longitudinal, transverse, or mixed according to their orientation relative to the long axis of the petrous temporal bone. However, classification based on otic capsule involvement - otic capsule-sparing versus otic capsule-violating fractures - provides additional clinical information regarding the risk of associated complications, including facial nerve injury [1-3]. The

location and extent of the fracture, particularly its relationship to the facial canal, are therefore important considerations when assessing the likelihood and severity of nerve damage.

Clinical evaluation requires a detailed history and comprehensive physical and neurological examination, supported by high-resolution computed tomography (CT) and electrophysiological testing. CT allows identification of fracture patterns, facial canal involvement, and potential nerve compression by displaced bone fragments. Electroneuronography (ENoG) assesses facial nerve function by measuring the electrically evoked muscle response and comparing the affected and unaffected sides, providing an estimate of nerve degeneration and helping to inform prognosis and management decisions. In bilateral facial palsy, however, interpretation may be more challenging because a clinically unaffected side is unavailable for comparison. The timing of paralysis onset, severity of dysfunction, imaging findings, and electrophysiological results are important factors when considering conservative treatment or surgical exploration and decompression. Although delayed-onset paralysis is generally associated with a more favorable prognosis than immediate complete paralysis, recovery depends on the underlying mechanism and extent of nerve injury [5].

Despite the available literature on traumatic facial nerve palsy, bilateral involvement following temporal bone fractures remains poorly documented, particularly regarding the integration of clinical, radiological, and electrophysiological findings into therapeutic decision-making. This report describes a case of severe bilateral facial nerve palsy following bilateral temporal bone fractures, highlighting the diagnostic challenges associated with symmetric facial weakness and the role of multimodal assessment in guiding individualized management and rehabilitation.

Case Presentation

A man in his early 40s with an unremarkable medical history was admitted to the Emergency Department following a 5 m fall resulting in cranioencephalic and thoracoabdominal trauma. On admission, the patient was conscious and alert, with a Glasgow Coma Scale score of 15. He reported left-sided hearing impairment and right-sided thoracic pain. Initial clinical examination revealed restricted left ocular movements during levoversion and levoelevation, associated with binocular diplopia during supraversion, dextroversion, and levoversion. Mild limitation of mouth opening (30 mm) was also observed. Facial movements were preserved bilaterally, with no clinically apparent facial nerve dysfunction, and initial otoscopic examination revealed no apparent abnormalities. Cranial CT demonstrated bilateral temporal bone fractures and associated craniofacial injuries, without evidence of additional intracranial traumatic lesions. High-resolution CT of the temporal bones revealed distinct fracture patterns: a longitudinal fracture of the right petrous temporal bone involving the mastoid segment of the facial nerve canal (Figures *1A*-*1B*) and a transverse fracture of the left petrous temporal bone extending through the perigeniculate region (Figures *1C*-*1D*). Additional injuries included fractures of the anterior and posterior walls of the frontal sinus (Figure *2A*), the left medial orbital wall (Figure *2B*), and the left zygomatic bone and arch (Figure *2C*). No evidence of inferior rectus muscle entrapment was identified. The ocular motility abnormalities and diplopia were attributed to divergent incomitant strabismus, likely related to post-traumatic edema or hematoma. Thoracic CT also revealed a right-sided pneumothorax, which was promptly treated with chest drainage. The patient was subsequently hospitalized for close clinical monitoring.

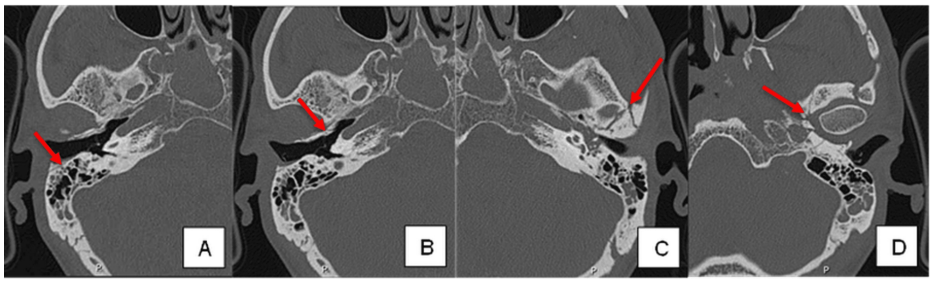


FIGURE 1: (A)-(B): CT scan, axial cuts, showing a longitudinal fracture of the right temporal bone (red arrow); (C)-(D): transverse fracture of the left temporal bone (red arrow).

CT, computed tomography

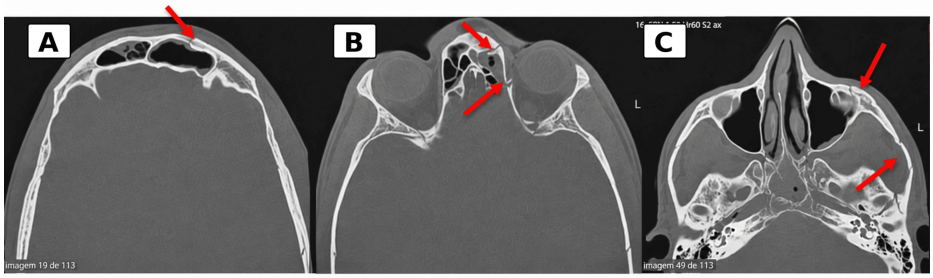


FIGURE 2: (A-C): Axial CT scans showing (A) aligned fractures of the frontal sinus walls, (B) a fracture of the medial orbital wall, and (C) fractures of the zygomatic bone and arch.

CT, computed tomography

On the fourth day after trauma, the patient developed left-sided peripheral facial nerve palsy, followed within hours by progressive weakness involving the upper third of the right hemiface. By the fifth day, bilateral facial nerve paralysis was evident, classified as House-Brackmann grade V on both sides [6]. The patient exhibited marked facial weakness, with an expressionless appearance, inability to wrinkle the forehead (Figure *3A*), incomplete bilateral eye closure despite maximal effort (Figure *3B*), and deviation of the oral commissure toward the right (Figure *3C*). These deficits were accompanied by difficulties with eating and speech articulation. Facial sensation was documented as preserved on clinical examination, while mild left ocular motility restriction and binocular diplopia persisted. The progression from initially preserved bilateral facial movements to sequential unilateral and subsequently bilateral paralysis supported the diagnosis of delayed-onset post-traumatic bilateral peripheral facial nerve palsy.



FIGURE 3: (A) The patient is unable to wrinkle the forehead; (B) despite maximal effort, he is unable to close both eyes; (C) deviation of the mouth corner to the right is also noticeable.

The patient provided written and signed consent, allowing publication of this identifiable facial image in an open-access journal.

Facial nerve electroneurography (ENoG) was performed on the fourth day after trauma, coinciding with the clinical onset of left-sided facial weakness. Motor responses were recorded bilaterally from the orbicularis oculi, nasalis, and orbicularis oris muscles. The examination demonstrated a marked reduction in compound muscle action potential (CMAP) amplitude and area on the left compared with the right, indicating substantial asymmetry in facial nerve motor responses (Figure *4*). Right-sided responses were relatively preserved at the time of examination, despite the subsequent progression to clinically evident bilateral facial weakness. These findings suggested more pronounced facial nerve dysfunction on the left at the time of testing. However, ENoG alone cannot reliably distinguish between different pathological grades of nerve injury, particularly axonotmesis and neurotmesis, nor can preserved early motor responses definitively establish neuropraxia. Moreover, the interpretation of ENoG findings depends on the interval between nerve injury and electrophysiological assessment, as Wallerian degeneration may require at least 72 hours to become detectable in distal facial nerve segments [7]. Accordingly, the electrophysiological findings were interpreted in conjunction with the clinical evolution and radiological assessment rather than as definitive evidence of a specific mechanism of nerve injury.

Motor Nerve Conduction					
Nerve / Sites	Lat1 (ms)	Dur. (ms)	AMP (mV)	Area (mVms)	Diff. (ms)
Facial - 3-Ch Rec Orb oculi, Nasalis, Orb oris					
Right Postauricular	2.7	5.8	2.9	9.5	2.7
Left Postauricular	3.1	7.2	0.2	0.5	3.1

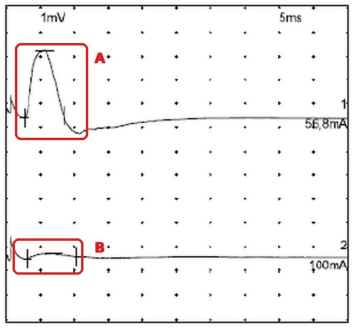


FIGURE 4: Facial nerve electroneurography (ENoG) performed on the fourth day after trauma.

Motor nerve conduction study of the facial nerve, with bilateral recordings from the orbicularis oculi, nasalis, and orbicularis oris muscles. Panel A shows the right facial nerve responses, while panel B shows the left facial nerve responses.

Red boxes highlight the recorded CMAPs, demonstrating a marked reduction in amplitude and area on the left (B) compared with the relatively preserved responses on the right (A). The examination was performed on the day of clinical onset of left-sided facial weakness. These findings indicate more pronounced facial nerve dysfunction on the left at the time of testing but do not, in isolation, establish the specific type or severity of nerve injury.

Lat1 (ms), onset latency; Dur. (ms), CMAP duration; AMP (mV), CMAP amplitude; Area (mV·ms), CMAP area; Dist. (mm), distance between stimulation sites; Diff. (ms), latency difference between stimulation sites; CV (m/s), conduction velocity

Audiological assessment revealed asymmetric bilateral hearing loss, more pronounced on the left (Figure *5A*). Pure-tone audiometry demonstrated mild-to-moderate high-frequency sensorineural hearing loss in the right ear and severe-to-profound hearing loss across the tested frequencies in the left ear, consistent with post-traumatic auditory impairment. Immittance testing showed a type A tympanogram on the right, indicating normal middle ear compliance, and a flat type B tympanogram on the left, indicating reduced middle ear compliance (Figure *5B*). Acoustic reflexes were absent bilaterally under all tested conditions. Although absent acoustic reflexes may be compatible with facial nerve dysfunction, these findings are not specific and must be interpreted alongside hearing thresholds and middle ear status, particularly on the left. Overall, the audiological findings demonstrated bilateral post-traumatic auditory impairment, more severe on the left, in association with bilateral facial nerve dysfunction.

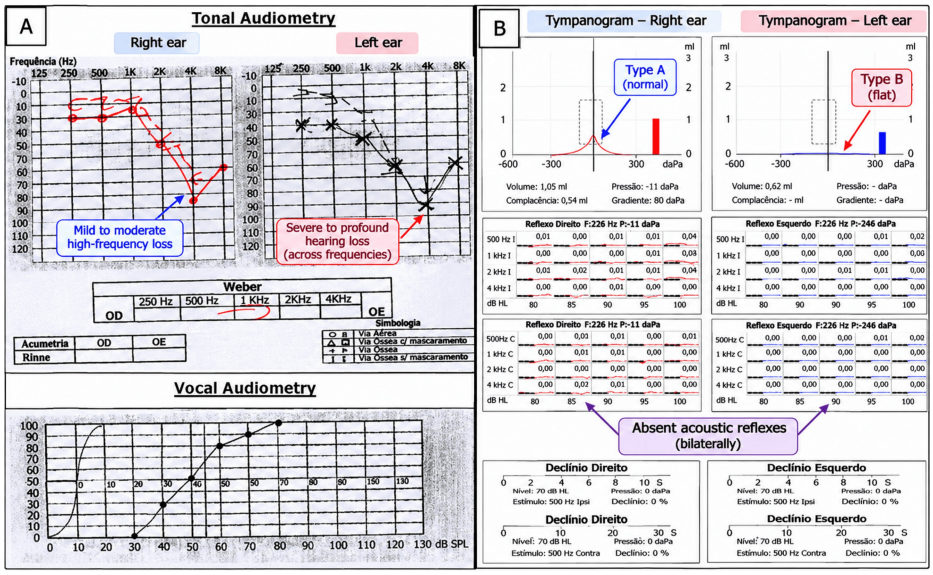


FIGURE 5: Audiological assessment following bilateral temporal bone fractures.

(A) Pure-tone audiometry: Asymmetric bilateral hearing loss, with mild-to-moderate high-frequency sensorineural hearing loss in the right ear and severe-to-profound hearing loss across the tested frequencies in the left ear. These findings indicate post-traumatic auditory impairment, more pronounced on the left.

(B) Tympanometry and acoustic reflexes: A type A tympanogram was recorded in the right ear, consistent with normal middle ear compliance, whereas a flat type B tympanogram was observed in the left ear, indicating reduced middle ear compliance. Acoustic reflexes were absent bilaterally under the tested conditions. These findings should be interpreted in conjunction with the audiometric results and clinical presentation, as neither a type B tympanogram nor absent acoustic reflexes independently establishes the underlying mechanism of middle ear or facial nerve dysfunction.

A conservative treatment strategy was adopted, considering the delayed onset of facial paralysis, the absence of direct radiological evidence of facial nerve compression by displaced bone fragments, and the potential morbidity associated with bilateral surgical exploration. The patient received intravenous methylprednisolone at a dose of 80 mg twice daily for five days. Ocular protection measures included topical oxytetracycline and a left eye patch to reduce the risk of exposure keratopathy. The patient also initiated physiotherapy focusing on facial expression exercises, performed three times weekly. By the eighth day after trauma, ocular motility restrictions and diplopia had completely resolved. However, no significant improvement in facial nerve function was observed by day 10. The patient was subsequently discharged with instructions to continue physiotherapy and oral prednisolone at a dose of 20 mg three times daily, gradually tapered over six days. Surgical decompression of the facial nerve was not performed, given the delayed onset of paralysis, the absence of identifiable compression by displaced bone fragments within the facial canal, and the assessment that the potential risks of bilateral surgical exploration outweighed the anticipated benefits.

At the one-month follow-up, complete clinical resolution of right-sided facial weakness was observed. On the left, substantial improvement was noted, although residual weakness persisted, particularly during forehead wrinkling and complete eye closure. Facial rehabilitation was continued during follow-up, with progressive improvement in facial nerve function. At the final seven-month assessment, the patient demonstrated near-complete functional recovery, with only mild residual left-sided facial weakness, classified as House-Brackmann grade II. Clinical examination revealed preserved forehead wrinkling with mild left-sided asymmetry during eyebrow elevation (Figure *6A*), slight asymmetry during forced eye closure (Figure *6B*), and facial symmetry during smiling (Figure *6C*). Overall, the patient achieved a favorable functional outcome following conservative management. The timeline of clinical presentation, management, and follow-up is presented in Table *1*.

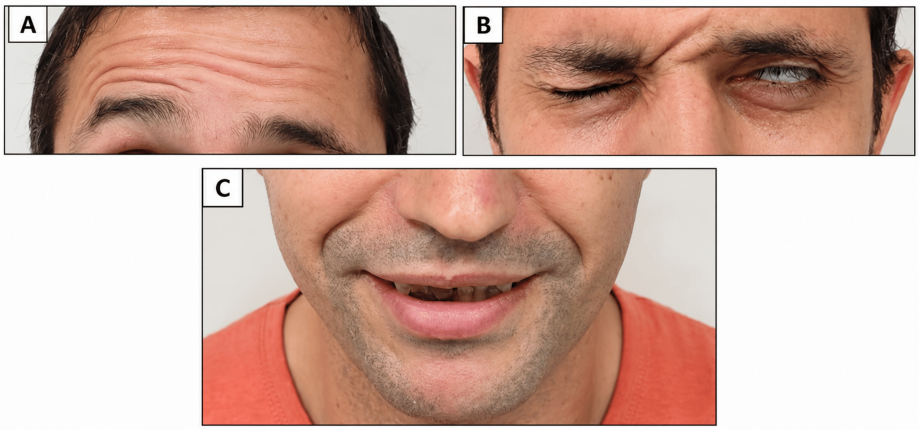


FIGURE 6: The patient exhibits a residual House-Brackmann grade II left facial nerve palsy.

(A) Preserved forehead wrinkling during eyebrow elevation with mild asymmetry on the left; (B) mild asymmetry during forced eye closure; (C) symmetry during smiling.

The patient provided written and signed consent, allowing publication of this identifiable facial image in an open-access journal.

Time after trauma	Key clinical findings and management
Day 0	Admission following a five-meter fall. GCS 15. Bilateral temporal bone fractures. Facial movements preserved.
Day 4	Onset of left facial palsy, followed by right-sided weakness. ENoG performed.
Day 5	Bilateral facial nerve palsy (House-Brackmann grade V).
Days 5-10	Conservative treatment with corticosteroids, ocular protection, and facial rehabilitation.
Day 8	Resolution of diplopia and ocular motility restrictions.
Day 10	Discharge without significant improvement in facial paralysis.
1 month	Complete recovery on the right; partial improvement on the left.
7 months	Mild residual left facial weakness (House-Brackmann grade II).

TABLE 1: Timeline of clinical presentation, management, and follow-up.

GCS, Glasgow Coma Scale; ENoG, electroneurography

Discussion

The temporal bone, characterized by its pyramidal shape, is highly resistant to mechanical forces, and its fracture generally results from high-energy trauma [3]. The diagnosis and management of temporal bone fractures require a multidisciplinary approach because of their potential neurological, otological, and functional complications [2,4]. Clinical manifestations may include otorrhagia, hemotympanum, vertigo, conductive or sensorineural hearing loss, tympanic membrane perforation, cerebrospinal fluid leakage, and facial nerve paralysis. Facial nerve injury has been reported in approximately 7%-10% of temporal bone fractures [4]. Temporal bone fractures may be classified according to otic capsule involvement as otic capsule-sparing or otic capsule-violating, with the latter generally associated with a higher risk of facial nerve injury [2,3].

Although unilateral facial nerve paralysis is a recognized complication of temporal bone fractures, bilateral involvement is uncommon and represents a diagnostic challenge. Post-traumatic facial paralysis may occur immediately or develop after a symptom-free interval, typically within 2-21 days following trauma, with an average onset of approximately four to five days [7,8]. Immediate paralysis is generally associated with direct nerve injury, including laceration, contusion, compression by displaced bone fragments, or traction. In contrast, delayed-onset paralysis may result from progressive edema, intraneural or perineural

hemorrhage, vascular compromise, or increased pressure within the facial nerve canal, potentially leading to secondary nerve dysfunction [7,8]. Delayed-onset facial paralysis has been described in several previous reports [7,9-12].

Most reported cases of post-traumatic bilateral facial nerve paralysis are associated with bilateral temporal bone fractures, although bilateral facial nerve dysfunction following unilateral temporal bone fractures has also been described [13]. De Villiers proposed that a longitudinal fracture of the petrous temporal bone could produce contralateral injury through displacement of the petrous apex and associated sphenoid fractures [13,14].

In the present case, CT demonstrated two distinct temporal bone fractures: a transverse fracture on the left involving the perigeniculate region and a longitudinal fracture on the right involving the mastoid segment of the facial nerve canal. These differences in fracture orientation and anatomical involvement may have contributed to the asymmetric facial nerve dysfunction and subsequent recovery observed on each side. However, the precise pathological grade of nerve injury could not be established from the available electrophysiological findings.

The clinical evolution was particularly relevant to the diagnostic assessment. Facial movements were preserved bilaterally on admission, followed by the onset of left-sided weakness on the fourth day after trauma and progression to bilateral House-Brackmann grade V paralysis by the fifth day. This delayed and sequential presentation suggests that secondary post-traumatic mechanisms, such as edema or hemorrhage within the facial nerve canal, may have contributed to the development of facial nerve dysfunction. However, the precise mechanism could not be established from the available clinical and imaging findings.

ENoG performed on the fourth day after trauma demonstrated markedly reduced left-sided CMAP amplitude and area, whereas right-sided responses were relatively preserved. These findings indicated greater electrophysiological dysfunction on the left at the time of testing but did not establish the anatomical severity of nerve injury or reliably predict long-term recovery. Importantly, the examination was performed on the day of clinical onset of left-sided weakness and before the development of clinically evident right-sided paralysis. Given the time required for Wallerian degeneration to become detectable in distal facial nerve segments, particularly in relation to the onset of paralysis, the findings of a single early ENoG examination should be interpreted cautiously. The subsequent improvement to House-Brackmann grade II on the left and complete recovery on the right further illustrate the limitations of using early electrophysiological findings in isolation to determine prognosis.

The audiological findings demonstrated asymmetric bilateral hearing loss, more pronounced on the left. The left-sided type B tympanogram indicated reduced middle ear compliance but did not establish a specific structural abnormality, such as ossicular chain disruption. Similarly, the absence of acoustic reflexes could not be attributed exclusively to facial nerve dysfunction, particularly in the presence of severe-to-profound hearing loss and abnormal tympanometry on the left. These findings highlight the importance of interpreting audiological investigations alongside clinical and radiological findings rather than as independent evidence of the underlying mechanism of facial nerve injury.

Management of traumatic facial nerve paralysis remains individualized and depends on the timing and severity of paralysis, radiological findings, electrophysiological assessment, and overall clinical condition. In selected patients with complete traumatic facial paralysis (House-Brackmann grade VI), greater than 90% facial nerve degeneration on ENoG has traditionally been considered a criterion for surgical decompression [3,15]. Early decompression, preferably within approximately 14 days of paralysis onset, has been advocated in some studies; however, the optimal timing remains controversial, and later intervention may be beneficial in selected patients [3,15]. The available recommendations are based predominantly on observational evidence, and high-quality randomized controlled trials directly comparing surgical and conservative management are lacking [15,16]. Furthermore, the interpretation of ENoG depends on the timing of testing and the availability of an unaffected contralateral nerve for comparison [3]. In the present case, bilateral House-Brackmann grade V paralysis developed after an initially symptom-free interval. ENoG was performed at the onset of left-sided weakness, before clinically evident right-sided paralysis, and a reliable percentage of degeneration could not be established from the available data. Therefore, the conventional electrophysiological threshold could not be directly applied. The delayed onset of paralysis, absence of displaced bone fragments compressing the facial nerve, and potential risks associated with bilateral surgical exploration favored a conservative approach. Nevertheless, the favorable outcome observed in this patient cannot establish the superiority of conservative management over surgical intervention.

The patient received corticosteroid therapy, ocular protection, and facial physiotherapy focusing on facial expression exercises, performed three times weekly. Complete clinical recovery of right-sided facial function was observed at one month, while the left side showed progressive improvement, reaching House-Brackmann grade II at seven months. Although this favorable outcome occurred following conservative management, the relative contribution of corticosteroids, rehabilitation, and spontaneous nerve recovery cannot be determined from a single case. The observed improvement should therefore not be interpreted as evidence of treatment efficacy.

This report has several limitations. As a single case report, its findings cannot be generalized to all patients with post-traumatic bilateral facial nerve paralysis. Electrophysiological assessment was limited to an early ENoG examination, without documented serial ENoG or electromyographic evaluation to characterize the subsequent evolution of nerve function. Additional magnetic resonance imaging was not available to further assess the facial nerves and surrounding soft tissues. Functional recovery was assessed primarily using the House-Brackmann grading system, without complementary standardized measures such as the Sunnybrook Facial Grading System or eFACE. Furthermore, synkinesis was not systematically assessed during follow-up, limiting the characterization of residual facial nerve dysfunction. The absence of detailed longitudinal audiological and vestibular assessments also limits characterization of associated otological recovery. Finally, although corticosteroids were administered as part of conservative management, their specific contribution to recovery and their risk-benefit profile in the setting of recent head trauma cannot be established from this report.

Conclusions

Delayed-onset bilateral facial nerve paralysis is a rare complication of temporal bone fractures that requires careful clinical assessment and follow-up. This case illustrates the importance of recognizing delayed facial nerve dysfunction despite initially preserved facial movements and highlights the limitations of early electrophysiological findings in predicting long-term recovery. Substantial functional improvement was observed following conservative management, with complete recovery on the right and mild residual weakness on the left at seven months. However, the contribution of treatment cannot be distinguished from spontaneous recovery. Management decisions should therefore be individualized according to clinical progression, imaging findings, and the overall condition of the patient.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Anabela Quitério, Inês Santos, Maria Pereira, Joana Magalhães

Acquisition, analysis, or interpretation of data: Anabela Quitério, Mariana De Miranda Pinto

Drafting of the manuscript: Anabela Quitério, Joana Magalhães

Critical review of the manuscript for important intellectual content: Anabela Quitério, Inês Santos, Maria Pereira, Mariana De Miranda Pinto

Supervision: Inês Santos, Maria Pereira, Joana Magalhães, Mariana De Miranda Pinto

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors have declared that no AI tools were used during the work or in the drafting of this article. The authors take full responsibility for the work. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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