Main Article

Surgery or Watchful Waiting? Dilemma in Managing Facial Palsy Following Temporal Bone Fracture

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ABSTRACT

Introduction

Post-traumatic facial nerve paralysis (FNP) is challenging, often leaving surgeons in a dilemma between conservative management and surgical intervention. This study was done to evaluate the clinical outcome of FNP resulting from temporal bone fracture (TBF).

Materials and Methods

A prospective observational study was conducted on patients with radiologically confirmed TBF. FNP was identified in 51 cases and graded using House-Brackmann grading system.

Results

Of the 69 cases included in the study, 51 patients exhibited FNP. 34 patients were included in immediate onset (21/34 had indeterminate onset) group and 17 patients were included in delayed onset group. All patients received oral corticosteroids initially. Follow-ups were conducted at 15, 30, 90 and 180 days. Seven patients who showed no signs of reinnervation on electromyography at day 30 were advised to undergo surgical intervention. Only three patients opted for facial nerve (FN) decompression, while four declined surgery. At six-month follow-up, 49 patients (including two post-surgical cases) demonstrated following outcomes: 26 patients improved to HB grade I, 15 to grade II, five to grade III, two to grade IV, and one to grade V. Among the three patients who underwent FN decompression, two improved from grade V-IV to grade III. Of four patients who refused surgery, three remained at HB grades IV-V. Over extended follow-up, one patient deteriorated to grade VI by 54 weeks, while the other two improved to grade II by 64 and 70 weeks, respectively.

Conclusion

Immediate onset post-traumatic FNP has the potential for spontaneous recovery without surgical decompression.

Keywords

Facial Nerve; Temporal Bone; Skull Fracture; Facial Paralysis; Decompression

The temporal bones (TB) form part of the skull base, situated between the sphenoid and occipital bones. Despite their thickness and durability, these bones contain several foramina that create areas of relative weakness, making them susceptible to traumatic injury. The incidence of such fractures has increased significantly in recent years, largely due to high-impact incidents such as road traffic accidents (RTAs), assaults, and sports-related injuries. Temporal bone fractures can cause substantial morbidity due to damage to structures such as the external auditory canal (EAC), ossicles, cochlea, vestibule, semicircular canals, auditory-vestibular nerve, and facial nerve (FN). Severe trauma may also involve the carotid artery or jugular vein, resulting in life-threatening haemorrhage.

In cases of temporal bone fractures, the management of life-threatening injuries often takes precedence, potentially leading to delayed recognition of facial paralysis. This delay complicates treatment and rehabilitation, resulting in long-term functional impairment and diminished quality of life. This study explores the management options for facial nerve paralysis (FNP)

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following temporal bone fractures, focusing on clinical outcomes and treatment strategies.

Materials and Methods

This prospective observational study was conducted at a tertiary care hospital over three years. It involved a consecutive cohort of patients with TB fractures. Ethical approval was obtained from the institutional ethics committee (Approval no. 2847A/2022-23), and the study itself was conducted from January 2021 to December 2023, with patient recruitment and follow-up occurring over this 3-year period. The study was conducted in accordance with the Declaration of Helsinki.

Patients were referred to the Department of Ear, Nose, and Throat (ENT) either directly from the trauma center or from the departments of neurosurgery or maxillofacial surgery. Inclusion criteria were patients with documented TB fractures on high-resolution computed tomography (HRCT) and evidence of FNP.

The onset of paralysis was categorized as immediate onset if FNP was documented within 24 hours after sustaining the injury and delayed onset if later than that. Patients with indeterminate onset were considered in immediate onset group.

Exclusion criteria: (i) Any pre-existing FN condition such as Bell's palsy, or prior mastoid/facial surgery with injury to FN. (ii) Any maxillofacial injury that could preclude evaluation of FN function. (iii) Patients unwilling to provide informed consent.

Data Collection: Detailed case histories were recorded, including the date and nature of the trauma, initial treatment, and the onset and progression of otologic symptoms. A thorough ENT examination, including a neurotologic assessment, was performed. HRCT of the temporal bone was obtained to document fracture lines, determine the extent of trauma, and correlate radiologic findings with clinical symptoms.

Treatment Protocol: Patients with FNP were initially treated with oral prednisolone (1 mg/kg/day) for five days. None of the patients had received steroids before presentation in the ENT department. Recovery was categorized using the House-Brackmann (HB) scale:

(i) Complete recovery: Patients who achieved House-Brackmann (HB) grade I.

(ii) Partial recovery: Patients who achieved HB grades II-IV.

(iii) No recovery: Patients who achieved HB grades V-VI.

Treatment was reassessed on the sixth day. If improvement was observed, the dose was tapered over five days. Based on our treatment protocol, in cases with no improvement or worsening, the treatment was continued for an additional five days, followed by tapering. Electromyography (EMG) was performed on 12 patients who had no observable improvement in FN function after one month of conservative treatment irrespective of the grade of FNP at the time of presentation. Seven patients with no regenerative potential on EMG were considered for FN decompression. Three patients underwent surgery on the 33rd, 37th, and 101st day respectively, while four patients declined surgery. Operated patients were followed up for more than six months, while one patient was lost to follow-up.

Surgical Technique: In all three patients the fracture line was undisplaced and involved the second genu and tympanic segment, second genu and mastoid segment, mastoid segment alone respectively. A transmastoid approach was used, involving mastoidectomy, exposure of the short process of the incus, transmastoid atticotomy, and posterior tympanotomy which was extended superiorly to visualise the tympanic segment and first genu of FN. Incus and malleus head were removed in all three cases irrespective of hearing status to achieve good exposure. The FN was decompressed from the first genu to the stylomastoid foramen by removing bone 180 degree around the nerve's circumference, and small bony fragments impinging on the nerve were removed. The nerve sheath was slit using an ophthalmic knife. The ossicular chain was reconstructed by interposing the incus between the stapes and malleus.

Complete nerve transection was not seen in any of the three patients. In two patients, small bony fragments were seen impinging on the FN in the mastoid segment which were not identified radiologically. These fragments were removed atraumatically. In one patient who had

involvement of tympanic segment and second genu, though no bony fragments were impinging on the nerve, a small intraneural haematoma was seen underneath the intact epineurium which was evacuated once the nerve sheath was slit open.

Results

The study included 69 patients, with 61 (88%) males and 8 (12%) females. The mean age was 33.1 years (SD ± 9.5), with most patients in the 21-40 age group. Road traffic accidents accounted for 57 (83%) of the cases, with other causes including falls, sports injuries, and assaults.

Otological Features: Patients commonly presented with ear bleeding/discharge, hearing loss, tinnitus, vertigo, and facial asymmetry [Table I]. Other clinical signs included Battle's sign, mastoid tenderness, lacerations of the EAC, tympanic membrane perforation, hemotympanum, FNP, and cerebrospinal fluid (CSF) otorrhea.

Table I: Otological manifestations of patients with fracture temporal bone

SYMPTOMS	NUMBER (%)
Ear bleed	29 (42%)
Hearing loss	38 (55%)
Tinnitus	12 (17%)
Vertigo	10 (14%)
Facial asymmetry	51 (74%)
SIGNS	**NUMBER (%)**
Battle sign	12 (17%)
Mastoid tenderness	20 (29%)
External canal laceration	31 (45%)
Tympanic membrane perforation	5 (7%)
Haemotympanum	23 (33%)
Hearing loss	38 (55%)
Nystagmus	2 (3%)
Facial nerve palsy	51 (74%)
CSF otorrhea	4 (6%)

Fracture Patterns: HRCT revealed that most fractures were longitudinal (67%) [Fig 1 (A)], with a higher incidence of oblique fractures compared to transverse fractures [Fig 1 (B)]. The tympanic, mastoid, and first genu segments of facial canal were most commonly affected [Table II].

In most cases, fractures were undisplaced, with only two documented cases of displaced fractures [Fig 1 (C)]. The facial canal was involved in 34 (49.3%) cases [Fig 1 (D)].

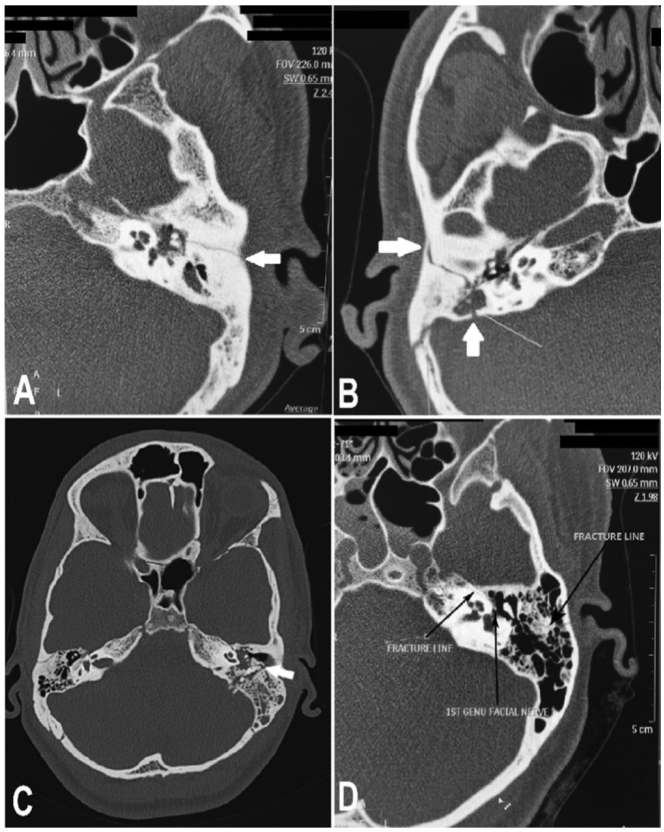

Fig. 1. High resolution CT scan of temporal bone. (A) Axial image shows longitudinal fracture line involving the left temporal bone (white arrow). (B) Axial image shows an oblique fracture line involving the right temporal bone (white arrow). (C) Axial image shows displaced bony fragments along the fracture line (white arrow). (D) Axial image shows a fracture line in the left temporal bone involving the first genu of the facial nerve (black arrow)

Table II: Pattern of temporal bone fracture line in patients with post-traumatic facial nerve paralysis

PATTERN OF TEMPORAL BONE FRACTURE (N= 69)		NUMBER (%)
Type of fracture according to the plane of fracture line	Longitudinal	46 (66.7%)
	Transverse	8 (11.6%)
	Oblique/ mixed	15 (21.7%)
Type of fracture according to involvement of otic capsule	Otic capsule involving	16 (23.18%)
	Otic capsule sparing	53 (76.8%)
Fracture line involving Facial canal	First genu	6 (17.6%)
	Second genu	5 (14.7%)
	Labyrinthine segment	1 (2.9%)
	Tympanic segment	9 (26.5%)
	Mastoid segment	6 (17.6%)
	1st genu & tympanic segment	2 (5.9%)
	2nd genu & tympanic segment	3 (8.8%)
	2nd genu & mastoid segment	2 (5.9%)

Facial Palsy Outcomes: Lower motor neuron FNP was observed in 51 (74%) patients. Overall, 13 cases were immediate onset of which 7/13 presented to the ENT specialist on the same day; cases detected between Day 2-5 had indeterminate onset, of these 15/21 patients presented for treatment during that timeframe [Fig 2]. 17 cases were delayed onset and presented later, typically in the 2nd or 3rd week following the onset of symptoms. The severity of facial palsy was classified using the House-Brackmann (HB) scale, with 19 patients in grade V, 9 in grade IV, 7 in grade III, and 16 in grade II. [Fig 3].

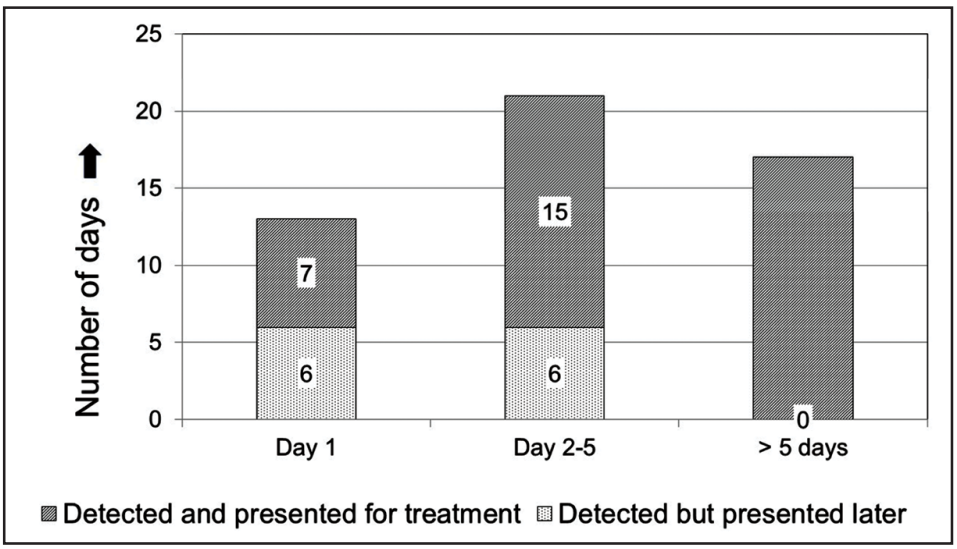

Fig. 2. Day of detection and day of presentation for ENT consultation in the immediate onset post-traumatic facial nerve paralysis group (n=34)

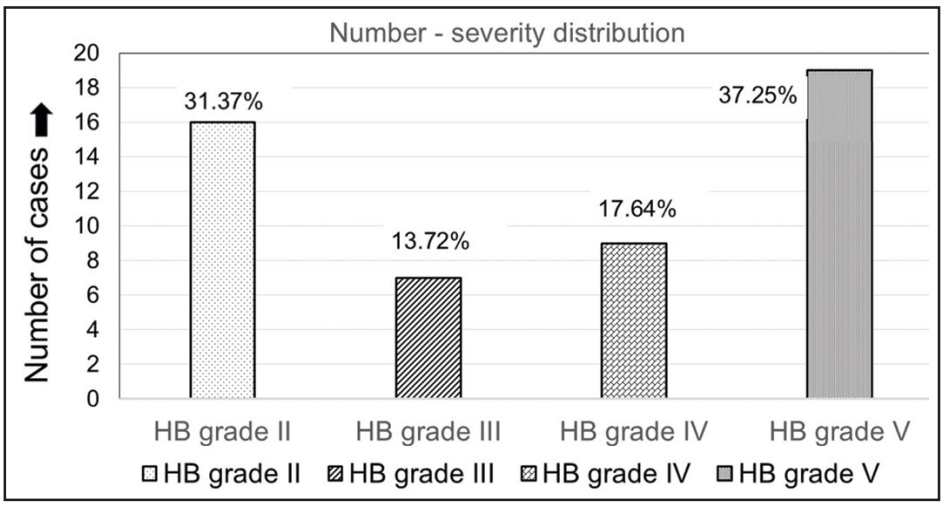

Fig. 3. **Distribution of post-traumatic facial nerve paralysis as per House Brackmann**

FN recovery was assessed at days 15, 30, and 90 [Fig 4]. On day 15, clinically significant recovery was noted, with two patients achieving HB grade I (two cases), and others improving to grades II (16 cases), III (11 cases), and IV (8 cases). However, 14 patients (grade V) showed no improvement by day 15. By day 30, improvements were more pronounced, with 11 patients achieving grade I recovery, 19 improving to grade II, eight cases to grade III. Twelve patients (who belonged to the immediate onset group) did not show satisfactory recovery (grade IV-V) by day 30. Of these, seven showed no reinnervation potential on EMG and were planned for surgical decompression. Three patients underwent surgery on the 33rd, 37th, and 101st day, respectively while four declined surgical intervention. One patient with unsatisfactory recovery who had declined surgical intervention succumbed to neurosurgical complications. The outcomes of facial palsy cases, excluding those who underwent surgical decompression (47 cases), were evaluated at day 90 [Table III]. At the 90-day follow-up, 55.31% of patients showed complete recovery (HB grade I), 36.17% had partial recovery (grades II-IV), and 8.51% had no recovery (grade V).

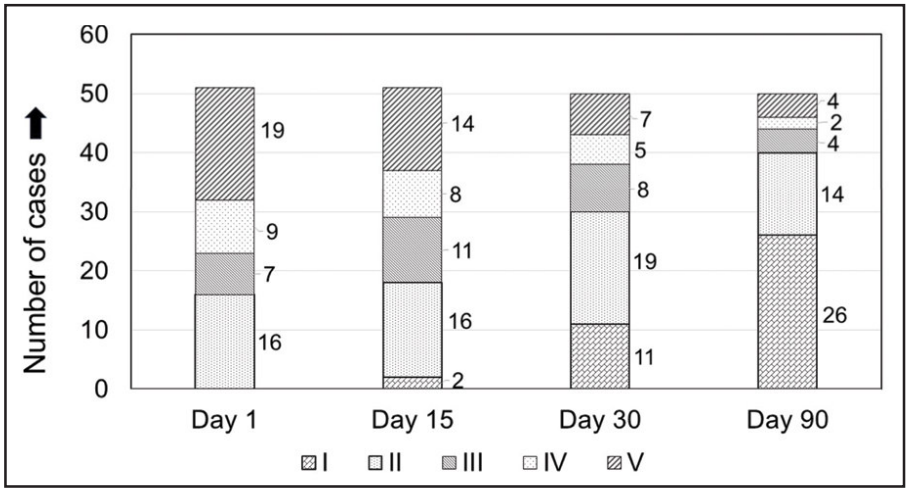

Fig. 4. Pattern of House Brackmann grade of recovery in cases of post-traumatic facial nerve paralysis over 90 days

Table III: Outcome of facial palsy at 90 days of follow-up in 47 unoperated patients of post-traumatic facial nerve paralysis

LEVEL OF RECOVERY FROM FACIAL PALSY	NO. OF CASES(N=47)	PERCENTAGE (95% CI)
Complete recovery (HBG I)	26	55.31% (41-69%)
Partial recovery (HBG II-IV)	17	36.17 % (23-50%)
No recovery (HBG V-VI)	04	8.51 % (3-20%)

HB Grade- House Brackmann grade

Long-term Follow-Up (upto or more than six months): Overall, 49 patients, which also includes two cases who underwent surgical management were analysed at six months - 26 patients were classified as HB grade I, 15 as grade II, five as grade III, two as grade IV (both had declined surgical intervention), and grade V-one case (had declined surgical intervention) [Table IV]. Of the three patients who underwent surgery, two improved to HB grade III from HB grade IV and V respectively while one was lost to follow up.

Three patients with HB grade IV and V, all of whom had declined surgical intervention, were regularly monitored. Two of these (one with grade IV and one with grade V) had displaced fracture segments on HRCT. Two patients (one of them with displaced fracture segment on HRCT) recovered to HB grade II by 64 and 70 weeks, respectively, over time, one patient progressed to HB grade VI by 54 weeks (had displaced fracture segment on HRCT). EMG was repeated for this patient at 54 weeks. It showed presence of sharp positive waves and fibrillation potentials in the mentalis muscle indicative of active denervation. This patient was advised surgical intervention at 54 weeks which he again declined. After 54 weeks this patient could not be followed up.

Table IV: House Brackman Facial nerve grading of patients (n=51) over more than six-month period of observation

GRADE	D 1	D 15	D 30	D 90	D 180	>50 WEEKS
I	0	2	11	26	26	26
II	16	16	19	14	15	17
III	7	11	8	4	5	5
IV	9	8	5	2	2	0
V	19	14	7	4	1	0
VI	0	0	0	0	0	1
Total	51	51	50	50	49	49

*One patient succumbed to his neurosurgical morbidity before day 30; another patient who underwent facial nerve decompression was lost to follow-up post-surgery hence only 49 out of 51 patients were available for long-term follow-up

Discussion

FNP can be classified into two broad categories: iatrogenic and accidental. Iatrogenic cases, typically due to neurotologic surgeries, account for approximately 20% of all FNP cases. The remaining 80% result from trauma, particularly fractures of the temporal bone (TB).[1] In our study, RTA's were the leading cause of TB fractures, which is consistent with findings from Mishra et al, but differs from studies in Western countries where falls are often cited as the predominant cause of TB fractures.[2-5]

The complex anatomy of the FN as it traverses the TB makes it vulnerable to injury in multiple locations, depending on the nature of the fracture. Our study found a notably high incidence of FNP (74%) following TB fractures, which is significantly higher than the 1.6% and 7% reported by Schuble and Brodie, respectively, in Western studies.[4,5] Yetiser's study, however, reported a similarly high incidence (68.57%).[6] This disparity may reflect differences in trauma patterns, driving behaviors, and safety measures between regions, which could contribute to the increased frequency of TB fractures and subsequent FNP in our cohort.

Post-traumatic FNP can be either immediate onset or delayed onset. The aetiology of delayed-onset facial palsy following temporal bone fracture is debatable. Unlike immediate facial palsy, which is usually attributed to direct nerve transection or severe mechanical disruption, delayed palsy is conjectured to result from either progressive neural oedema within the bony fallopian canal, intraneural or perineural hematoma formation, which results in nerve compression, vascular compromise leading to ischemic damage, stretching of the nerve or inflammatory demyelination. The narrow labyrinthine segment is most vulnerable due to its limited capacity to accommodate nerve oedema. As there are more chances of preservation of axonal continuity, a favourable prognosis compared to immediate complete palsy is expected.

Mechanisms of FN Injury and HRCT Findings: High-resolution CT (HRCT) scans play a crucial role in identifying FN injury in TB fractures, particularly fractures of the bony facial canal, displacement of bone fragments, or impalement of the nerve by bone fragments, which can help guide treatment decisions.[7] In our cohort, the tympanic segment of the FN was the most frequently injured (26.5%), a finding that contrasts with the geniculate ganglion being the most affected site in other studies (30-80%).[8] This highlights the importance of a comprehensive evaluation of the FN, considering various potential sites of injury.

The management of FNP, particularly in cases with immediate complete onset, often poses a clinical dilemma: to proceed with conservative management or opt for surgical decompression. The decision is influenced by factors such as the timing of onset, the degree of nerve damage, and the presence of radiological signs of severe injury as mentioned above. The extent of facial nerve injury is a key factor in determining functional recovery and long-term prognosis. Mild injuries corresponding to neuropraxia demonstrate early and near-complete recovery due to preserved axonal integrity. In contrast, axonotmesis is followed by Wallerian degeneration and requires the axon to regenerate, resulting in delayed and incomplete recovery. Neurotmesis, which is associated with the disruption of nerve is associated with poor prognosis. Electrophysiological parameters such as CMAP amplitude and increased latency are objective indicators of the degree of injury. Greater reductions in CMAP amplitude correlate with extensive axonal loss and are associated with slower and incomplete recovery. Early evaluation of damage severity aids in prognosis and directs treatment choices.

In cases of delayed or incomplete paralysis, medical management with corticosteroids is typically the first-line treatment, as it can help reduce inflammation and promote nerve recovery.[5] However, for patients with immediate onset complete facial paralysis, the need for surgical intervention remains controversial. While some studies advocate early surgical decompression (within 8 weeks of injury) to minimize long-term damage to the nerve, others suggest that conservative management with steroids may suffice, even in cases where electroneuronography (ENoG) shows severe degeneration of the nerve.[9-11] House's observations suggest that surgery often results in only modest

improvement in FN function, indicating that a cautious, wait-and-see approach may be appropriate in many cases.[12]

Our study supports the idea that conservative management, particularly when combined with corticosteroid therapy, leads to good outcomes for many patients. Only 4 out of 47 conservatively treated patients failed to show improvement by 90 days, and even these cases except one patient showed recovery beyond the 90-day follow-up period. In contrast, both patients who underwent early FN decompression surgery (performed at day 33 and day 37) only achieved a recovery up to HB grade III. Although 2 cases underwent surgical decompression, the comparison with 47 non-surgical cases does not provide statistical significance and should be interpreted as a descriptive observation rather than a definitive conclusion. For patients who did not show improvement within the initial protocol timeframe, extending steroid treatment may be considered on a case-by-case basis. Further research is needed to assess the efficacy of prolonged steroid therapy for non-responders.

Electrodiagnostic tests, such as ENoG and electromyography (EMG), play a pivotal role in predicting the recovery potential of the FN. ENoG can assess the extent of nerve degeneration and provide critical information regarding the likelihood of spontaneous recovery. The chances of spontaneous recovery drop to 50 % if greater than 95% degeneration is documented by ENoG at the end of 2-3 weeks.[13] ENoG may, however, overestimate degeneration because a dyssynchronous response by the regenerating nerve fibres may not contribute to the compound muscle action potential (CMAP). Overall, the role of various electrodiagnostic tests in predicting acute facial nerve paralysis recovery is controversial. Esslen's theory, which proposed that the amplitude of CMAP is directly proportional to the number of degenerated fibres has been challenged by various authors. Sittel et al and Grosheva et al have reported that EMG is a more reliable tool for predicting unfavourable outcomes.[14- 15] In a study by Grosheva et al, ENoG prognosis was correct only in 73% patients compared to EMG, which correctly predicted recovery in 97% patients. In the same study, sensitivity, specificity, positive predictive value, and negative predictive value of EMG was reported to be higher than ENoG.[16] Due to the unavailability of ENoG in our institution, we relied on EMG to guide our treatment decisions. EMG, while useful in detecting denervation and reinnervation, has limitations, particularly in the early phase of injury.

Considering surgical decompression in patients who do not show recovery by 4–6 weeks, particularly if ED tests suggest either significant degeneration (ENoG) or lack of reinnervation (EMG) may appear a prudent clinical decision. Alternatively, if radiological evidence suggests mechanical compression or transection of the FN it can be done earlier.[13, 17] The optimal timing for surgical intervention remains a subject of debate, with Fisch advocating for decompression within the first 3 days, while others suggest waiting for 20-21 days to allow for optimal axoplasmic flow; or Thakar et al, who advocate super-selection of cases to operate only after 16-18 weeks.[11, 18-19] Our study found that despite surgical decompression (average timing of decompression 5-14 weeks) in three patients with unfavourable clinical and electrophysiologic response, patients did not have a dramatic recovery at the end of six-month follow-up. On the contrary 98% cases had good clinical recovery with conservative management (46 patients out of 47 improved to grade I-III) including one with displaced fracture segment at six months. This reinforces the view that conservative management for a period of 16-18 weeks as suggested by Thakar et al[11] may be equally effective in many cases of complete FNP.

The literature on the outcomes of surgical decompression versus conservative management is mixed. Several studies, such as those by Liu et al., Quaranta et al, and Gür et al, report good recovery (HB grade I-III) in a significant proportion of patients undergoing surgery.[1, 14, 20] However, studies like that of Darrouzet et al equivocal recovery patterns have been reported with conservative and surgical management in traumatic FNP.[21] Thakar et al treated 28 cases of complete facial palsy following TB fracture with oral steroids and

reported recovery to HB grade I/II in 96% cases, albeit after 24 weeks and 100% had recovered to HB grade I/II/III by 40 weeks.[11] They argue that early surgical intervention for undisplaced longitudinal fractures is 'unjustified' and advocate for clinical judgment regarding surgical treatment in significantly displaced fractured segments with suspicion of nerve transection. Similarly, Nash et al suggest that conservative management leads to a higher rate of full recovery (36% with observation vs. 16% with surgery.[22]

The ongoing debate about the timing and outcome of surgical intervention remains inconclusive. In our study, the majority of patients managed conservatively recovered to HB grades I-III. Surgical intervention was required in a small proportion and did not contribute to a dramatic recovery. The lack of use of ENoG results in surgical decision-making and the small number of patients who received surgical decompression are the limitations of our study.

Ultimately, our findings support a selective approach to surgery, recommending conservative management as the first-line treatment for most cases of traumatic FNP. Surgery should be reserved for cases with substantial nerve damage or those showing no signs of recovery after 3–6 months of observation. Further studies documenting the role of conservative management in patients with poor ENoG and EMG response, though ethically challenging to conduct, are required to validate our findings. Also, studies with larger sample sizes and longer follow-up periods are needed to refine treatment protocols and better understand the long-term outcomes of different management strategies for FNP following temporal bone fractures. In managing post-traumatic FN palsy, our study primarily employed corticosteroid therapy, with surgical intervention reserved for cases where EMG results indicated poor chances of spontaneous recovery. We did not explore rehabilitation strategies or the role of hyperbaric oxygen therapy, both of which may hold promise for improving outcomes in cases of partial or no recovery. Further research should examine these adjunct therapies.

Conclusion

In conclusion, traumatic FN paralysis following temporal bone fractures is a complex condition that requires careful evaluation and management. Our study suggests that most patients with FNP following TB fractures can achieve good recovery through conservative management. Electrodiagnostic tests like ENoG and EMG should be incorporated into the decision-making process when available, as they provide valuable prognostic information and help in selection of cases requiring surgical intervention. Surgical decompression should be reserved for cases where HRCT shows displaced fracture segments indicative of severe nerve damage or nerve transection, or fractured segments are seen along the course of Fallopian canal leading to mechanical compression, or when there is a lack of any sign of recovery after 3-6 months of observation. The utility of HRCT in decision-making should be validated in future studies.

References

1. Liu Y, Han J, Zhou X, Gao K, Luan D, Xie F et al. Surgical management of facial palsy resulting from temporal bone fractures. Acta Oto-Laryngologica. 2014; 134: 656–60
2. Shankar A, George S, Somaraj S. Evaluation of Clinical Outcome in Traumatic Facial Nerve Paralysis. Int Arch Otorhinolaryngol 2021; 26(1): e010-e019. doi: 10.1055/s-0040-1718962
3. Vajpayee D, Mallick A, Mishra AK. Post Temporal Bone Fracture Facial Paralysis: Strategies in Decision Making and Analysis of Efficacy of Surgical Treatment. Indian J Otolaryngol Head Neck urg 2018; 70(4): 566-71. doi: 10.1007/s12070-018-1371-y
4. Shuble SD, Klein TR. Robitsek RJ et al. Temporal bone fracture: evaluation in the era of modern CT. Imaging 2016; 47(9): 1893–97
5. Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. Am J Otol 1997; 18(2): 188–97

6. Yetiser S, Hidir Y, Gonul E. Facial nerve problems and hearing loss in patients with temporal bone fractures: demographic data. J Trauma 2008; 65(6): 1314–20
7. Ulug T, Arif Ulubil S. Management of facial paralysis in temporal bone fractures: a prospective study analyzing 11 operated fractures. Am J Otolaryngol 2005; 26(4): 230–8
8. Chen Y, Zhang K, Xu Y, Che Y, Guan L, Li Y. Reliability of temporal bone high-resolution CT in patients with facial paralysis in temporal bone fracture. Am J Otolaryngol 2018; 39(2): 150-2. doi: 10.1016/j.amjoto.2017.12.003
9. Kasbi AA, Kouhi A, Ashtiani MTK, Anari MR, Yazdi AK, Emami H. Conservative versus Surgical Therapy in Managing Patients with Facial Nerve Palsy Due to the Temporal Bone Fracture. Craniomaxillofac Trauma Reconstr 2019; 12(1): 20-6. doi: 10.1055/s-0038-1625966
10. Xie S, Wu X, Zhang Y, Xu Z, Yang T, Sun H. The timing of surgical treatment of traumatic facial paralysis: a systematic review. Acta Otolaryngol 2016; 136(12): 1197-1200. doi: 10.1080/00016489.2016.1201862
11. Thakar A, Gupta MP, Srivastava A, Agrawal D, Kumar A. Nonsurgical Treatment for Posttraumatic Complete Facial Nerve Paralysis JAMA. Otolaryngol Head Neck Surg 2018; 144(4): 315–21
12. House JW. Facial nerve grading system. Laryngoscope. 1983; 93: 1059-69
13. Fisch U. Surgery for Bell's palsy. Arch Otolaryngol. 1981; 107:1-11
14. Sittel C, Stennert E. Prognostic value of electromyography in acute peripheral facial nerve palsy. Otol Neurotol. 2001; 22(1): 100-4. doi: 10.1097/00129492-200101000-00019
15. Grosheva M, Guntinas-Lichius O. Significance of electromyography to predict and evaluate facial function outcome after acute peripheral facial palsy. Eur Arch Otorhinolaryngol. 2007 ;264(12):1491-5. doi: 10.1007/s00405-007-0376-z
16. Grosheva M, Wittekindt C, Guntinas-Lichius O. Prognostic value of electroneurography and electromyography in facial palsy. Laryngoscope. 2008 ;118(3): 394-7. doi: 10.1097/MLG.0b013e31815d8e68
17. Quaranta A, Campobasso G, Piazza F, Quaranta N, Salonna I. Facial nerve paralysis in temporal bone fractures: outcomes after late decompression surgery. Acta Otolaryngol 2001; 121(5): 652-5
18. Fisch U. Facial paralysis in fractures of the petrous bone. Laryngoscope. 1974; 84(12):2141-54
19. McCabe BF. Injuries to the facial nerve. Laryngoscope. 1972; 82(10): 1891-96
20. Gür H, Görür K, Ýsmi O, Vayýsoðlu Y, Bal KK, Özcan C. Surgical Outcomes of Transmastoid Facial Nerve Decompression for Patients with Traumatic Facial Nerve Paralysis. J Int Adv Otol 2021; 17(4): 294-300. doi: 10.5152/iao.2021.8506
21. Darrouzet V, Duclos JY, Liguoro D, Truilhe Y, De Bonfils C, Bebear JP. Management of facial paralysis resulting from temporal bone fractures: our experience in 115 cases. Otolaryngol Head Neck Surg 2001; 125(1): 77–84
22. Nash JJ, Friedland DR, Boorsma KJ, Rhee JS. Management and outcomes of facial paralysis from intratemporal blunt trauma: a systematic review. Laryngoscope 2010; 120(7): 1397-1404.