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Original Research Article

FREQUENCY OF FACIAL NERVE PALSY IN BASE OF SKULL FRACTURES

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ABSTRACT

Background: *Early recognition through standardized clinical and radiological evaluation is essential for timely management.* ***Objective:*** *To establish the frequency and clinical-radiological characteristics of FNP among patients presenting with skull base fractures, using a standardized diagnostic protocol.* ***Study Design:*** *Cross-sectional study.* ***Setting:*** *Department of Neurosurgery, Lady Reading Hospital, Peshawar, Pakistan.* ***Duration of Study:*** *29-December-2024 to 29-May-2025.* ***Methods:*** *Adult patients (18–65 years) with CT-confirmed skull base fractures were consecutively enrolled. Patients with Glasgow Coma Scale (GCS) <8 or penetrating craniocerebral injuries were excluded. FNP was defined by acute unilateral facial weakness plus associated symptoms, supported by MRI evidence of neural injury. Clinical grading was performed using the House-Brackmann scale. Data were analyzed using SPSS version 26.* ***Results:*** *Out of 165 screened patients, 143 met the inclusion criteria. The mean age was 36.8 ± 12.5 years, with a male predominance (79.0%). Road traffic accidents were the most common cause (66.4%). The overall frequency of FNP was 7.0% (10/143; 95% CI: 3.4–12.5%). Of these, 70% presented with an acute onset, and 30% with a delayed onset. At presentation, 60% had complete paralysis (House-Brackmann Grade VI) and 40% had incomplete paralysis. MRI confirmed nerve injury in all cases, while CT demonstrated bony impingement in only one patient.* ***Conclusion:*** *The frequency of FNP in skull base fracture patients was 7.0%, most commonly presenting as acute, complete paralysis. MRI showed superior diagnostic yield over CT in confirming neural injury, highlighting its vital role in the evaluation and management of skull base trauma.*

Keywords: Facial Nerve Palsy (FNP), Skull Base Fractures, House-Brackmann Scale, Magnetic Resonance Imaging (MRI), Computed Tomography (CT), Road Traffic Accidents, Traumatic Brain Injury

INTRODUCTION

Skull base fractures are severe injuries typically resulting from high-impact trauma such as motor vehicle collisions, assaults, or falls. They account for almost 14-22% of all cranial fractures (1). Despite being uncommon, skull base fractures are frequently associated with devastating sequelae, including hearing loss, intracranial hemorrhage, vascular injury, and cranial nerve deficits (1, 2). Among these, facial nerve palsy (FNP) is particularly devastating due to its profound effects on facial function, self-image, and overall quality of life (3).

Traumatic FNP can result from various mechanisms, including road traffic accidents, blunt trauma, penetrating injuries, and iatrogenic causes (3). It is also documented in cases of attempted suicide by hanging. Temporal bone fractures represent the most common origin of traumatic facial nerve injury and constitute the second most frequent cause of facial paralysis in adults, after Bell's palsy (4). Given the potential for long-term impairment, timely and accurate Diagnosis and management are critical for improving outcomes in this patient population (4).

Current management strategies for post-traumatic FNP are primarily guided by two factors: the timing of onset (immediate vs. delayed) and the severity of paralysis (complete vs. incomplete) (5). Immediate and complete paralysis often warrants surgical intervention, such as decompression or repair, whereas delayed or incomplete presentations are generally managed conservatively with corticosteroids, antivirals, or watchful waiting (5, 6).

Nevertheless, significant challenges and inconsistencies persist in clinical practice. Accurately establishing the time of onset of facial nerve palsy (FNP) presents a considerable challenge in polytrauma patients with compromised consciousness, frequently resulting in diagnostic misclassification (7). Furthermore, the standard imaging pathway introduces additional limitations; while computed tomography (CT) excels at delineating bony fractures, it possesses poor sensitivity for detecting subtle neural injuries. This frequently leads to a discrepancy between imaging results and the patient's clinical condition, impeding early and accurate prognosis. Compounding these issues, a scarcity of robust comparative studies prevents definitive conclusions about the relative effectiveness of surgical versus medical interventions (7).

Previous research by Naidu et al. documented FNP in 6.32% of individuals with skull base fractures (8), underscoring the significance of this clinical problem. The current investigation aims to expand existing knowledge by providing novel, prospective, imaging-verified data from a local context on the prevalence and clinical profile of FNP following skull base fractures. To address inconsistencies in current literature and provide better evidence for local practice, this research utilizes a rigorous diagnostic protocol to determine the frequency and characterizing features of FNP in a cohort of patients with radiologically confirmed skull base fractures. By conducting this study, we aim to fill critical gaps in local epidemiological data, enabling healthcare providers to anticipate and better manage facial nerve complications in patients with base of skull fractures, thereby offering valuable insights for improving neurotrauma care protocols and patient outcomes.

METHODOLOGY

A cross-sectional study was conducted to determine the frequency of facial nerve palsy in patients with skull base fractures. The study was conducted at the Department of Neurosurgery, Lady Reading Hospital (LRH), Peshawar, from December 29, 2024, to May 29, 2025.

Consecutive adult patients (aged 18–65 years) presenting with radiological evidence of a skull base fracture were enrolled using non-probability successive sampling.

Patients were included if they had a radiological Diagnosis of a skull base fracture, defined as the presence of two or more of the following CT findings: (1) cortical fracture lines with bone fragment displacement, (2) pneumocephalus, (3) intracranial hematoma

(epidural, subdural, or intracerebral), or (4) cerebrospinal fluid (CSF) leak evidenced by fluid in the paranasal sinuses or mastoid air cells.

Exclusion criteria were: a history of previous skull base surgery or trauma, congenital craniofacial abnormalities, penetrating head injuries, pregnancy or lactation, known neurodegenerative diseases affecting cranial nerves, and a Glasgow Coma Scale (GCS) score < 8 at presentation.

The sample size was calculated as 143 using the World Health Organization (WHO) sample size calculator, with a 95% confidence level, a 4% margin of error, and an expected frequency of facial nerve palsy of 6.32% [8].

After obtaining ethical approval and written informed consent, baseline demographic and clinical data were collected for all participants.

The primary outcome was traumatic facial nerve palsy, which was defined by the fulfillment of both clinical and radiological criteria:

Clinical Criteria: Sudden onset of unilateral facial weakness or paralysis, plus two or more of the following: altered taste sensation, hyperacusis, decreased tearing, or pain/discomfort around the ear or jaw.

Radiological (MRI) Criteria: Evidence of facial nerve injury on MRI, required to show all of the following: thickening of the extracranial vertical/mastoid and intra-parotid segments of the facial nerve, asymmetry compared to the non-symptomatic side, and the absence of any mass lesion in the parotid gland or cerebellopontine angle.

All patients underwent a dedicated MRI according to the hospital's neuroradiology protocol. Images were interpreted by a consultant radiologist blinded to the clinical findings.

Data were analyzed using SPSS version 26. Categorical variables (e.g., gender, socioeconomic status, facial nerve palsy) are presented as frequencies and percentages. Continuous variables (e.g., age, BMI) are presented as mean ± standard deviation or median (interquartile range, IQR) based on their distribution, as assessed by the Shapiro-Wilk test.

The frequency of facial nerve palsy was reported as a percentage with a 95% confidence interval. Stratification by age, gender, BMI, and other demographic variables was performed. Post-stratification, associations were assessed using the Chi-square or Fisher's exact test. A p-value ≤ 0.05 was considered statistically significant.

The Institutional Ethical Review Committee approved the study protocol. Written informed consent was obtained from all participants or their legal surrogates, and confidentiality was maintained throughout the study.

RESULTS

The baseline demographic and clinical characteristics of the entire study cohort are summarized in Table 1. The mean age of participants was 36.1 years (±11.3), with a strong male predominance (113 patients, 79.0%). The median time from injury to presentation was 9 hours (IQR: 4-22 hours).

Road traffic accidents were the most common mechanism of injury (66.4%, n = 95), followed by falls (21.7%, n = 31) and assaults (11.9%, n = 17). The cohort was predominantly from urban areas (94 patients, 65.7%). The most common professional background was manual labor (55 patients, 38.5%), and the majority had attained at least a secondary level of education (95 patients, 66.5%).

A total of 143 patients with radiologically confirmed skull base fractures were included in the final analysis. The primary outcome of traumatic facial nerve palsy (FNP), as defined by strict clinical and MRI criteria, was identified in 10 patients. This yields a frequency of 7.0% (n = 10/143) within this cohort (Table 2).

Among these 10 cases with FNP, the majority (7 cases, 70.0%) presented with an acute onset of paralysis, identified at or immediately after the injury. The remaining 3 cases (30.0%) were classified as delayed onset. According to the House-Brackmann scale at Diagnosis, six patients (60.0%) presented with complete paralysis (Grade VI), while four patients (40.0%) had incomplete paralysis (Grades II-IV). Magnetic resonance imaging (MRI) confirmed the Diagnosis in all cases, showing consistent findings of facial nerve thickening and abnormal enhancement. Bone fragment impingement on the facial canal was identified on computed tomography (CT) in only one of these cases (10.0%)

Table 1. Baseline Characteristics of Study Participants (N = 143)

Variable	Category	Value
Gender	Male	113(79%)
	Female	30(21%)
Age (Years)	Mean ± SD	36.1 ± 11.3
BMI (kg/m²)	Mean ± SD	26.7 ± 4.1
Residential status	Urban	94 (65.7%)
	Rural	49 (34.3%)
Education level	No formal	7 (4.9%)
	Primary	41 (28.7%)
	Secondary	57 (39.9%)
	Tertiary	38 (26.6%)
Profession	Clerical	34 (23.8%)
	Manual	55 (38.5%)
	Professional	27 (18.9%)
	Student	9 (6.3%)
	Unemployed	18 (12.6%)
Facial Nerve Palsy	No	133 (93.0%)
	Yes	10 (7.0%)
Time to Presentation (hours)	Median (IQR)	9.0 (4.0-22.0)

Table 2. Frequency of Facial Nerve Palsy (N = 143)

Outcome	n	Percentage
No FNP	133	93%
Yes FNP	10	7%

Stratification analysis was performed to assess the association between various demographic/clinical variables and the occurrence of FNP (Table 3). The frequency of FNP was highest in patients who were obese (12.9%, n=4/31), aged 45-59 years (15.4%, n=4/26), or presented with symptoms for more than 30 days (14.3%, n=1/7) (Table 3) (Fig. 1).

However, no statistically significant associations were found between the occurrence of facial nerve palsy and any of the tested variables, including gender (p=0.688), age group (p=0.211), BMI category (p=0.273), residential status (p=0.736), profession (p=0.942), income tertile (p=0.451), or symptom duration (p=0.561). Fisher's Exact Test was applied for variables where expected cell counts were less than 5 (Table 3).

Table 3. Stratification of Facial Nerve Palsy by Demographic and Clinical Variables (N = 143)

Variable	Category	Total (n)	FNP Cases (n)	FNP %	P-value
Gender	Male	113	9	8.0%	0.688*
	Female	30	1	3.3%	
Age Group	18–29	37	1	2.7%	0.211
	30–44	69	4	5.8%	
	45–59	26	4	15.4%	
	60+	4	0	0.0%	
BMI Category	Underweight	1	0	0.0%	0.273
	Normal	51	1	2.0%	
	Overweight	60	5	8.3%	
	Obese	31	4	12.9%	

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Residential Status	Urban	94	6	6.4%	0.736*
	Rural	49	4	8.2%	
Profession	Clerical	34	3	8.8%	0.942
	Manual	55	3	5.5%	
	Professional	27	2	7.4%	
	Student	9	0	0.0%	
	Unemployed	18	2	11.1%	
Income	Low	47	5	10.6%	0.451
	Middle	48	2	4.2%	
	High	48	3	6.3%	
Symptom Duration	≤7 days	77	4	5.2%	0.561
	8–30 days	59	5	8.5%	
	>30 days	7	1	14.3%	

Fisher's Exact Test applied where expected counts <5.

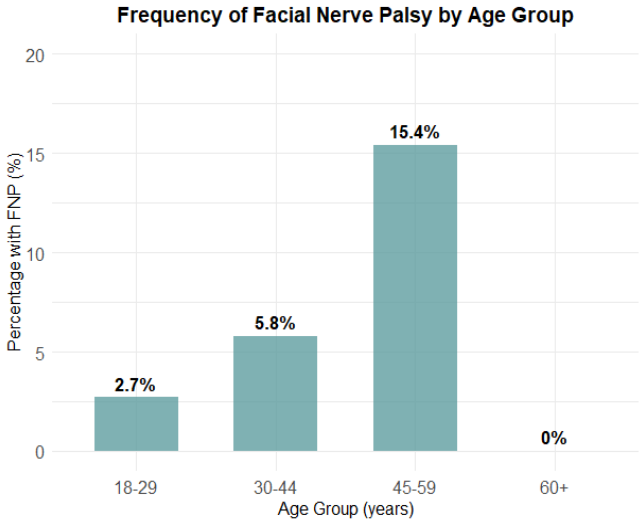

Fig.1 Frquency of facial nerve palsy by age group.

DISCUSSION

This study provides a detailed analysis of facial nerve palsy (FNP) in a cohort of 143 patients presenting with skull base fractures at a tertiary care center. The observed incidence of traumatic facial nerve palsy (FNP) in this cohort of skull base fracture patients was 7.0%. This finding is consistent with rates reported in several focused clinical studies. For instance, our result closely aligns with the 6.32% frequency reported by Naidu et al. and falls within the commonly cited 7-12% range for temporal bone fractures (8, 9).

However, this Figure contrasts sharply with the lower incidence (~1%) found in large-scale national trauma registries (3, 10). This discrepancy likely reflects fundamental methodological differences; our prospective study utilized stringent clinical and MRI-based criteria for Diagnosis, whereas registry data, which rely on administrative coding, may underascertain cases that are not immediately apparent or are overshadowed by more severe injuries.

In a broad head-injury series, Odebode et al (11). found FNP in 5.04% of patients, and Yetiser et al. reported facial paralysis in 68.6% of 35 selected temporal bone fracture patients (including transient paresis), (12) underscores how study design, population selection, and diagnostic rigor profoundly influence the estimation of this complication's frequency. Our finding, therefore, occupies a clinically plausible middle-to-upper range, representative of a cohort assessed with a high degree of diagnostic certainty (8, 9).

The demographic profile of our cohort demonstrates strong concordance with established epidemiological trends for skull base trauma documented in the literature. The mean patient age was approximately 37 years, with a male-to-female ratio of 3.8:1. This distribution closely aligns with the patterns reported by Brown et al., who noted that approximately 70% of temporal bone fractures occur in patients within their second to fourth decades of life, with a male predominance of roughly 3:1 (9).

Road traffic accidents (RTAs) emerged as the predominant injury mechanism in our study population, accounting for 66% of cases. This finding is consistent with global patterns identifying high-energy motor vehicle collisions as the leading cause of temporal bone and skull base fractures. Previous investigations have reported similar trends, with Shankar et al. documenting RTAs in 83.0% of traumatic facial nerve palsy cases (13). In contrast, Naidu et al. reported that RTAs were responsible for 79% of base-of-skull fractures with associated cranial nerve involvement (8). Similarly, Odebode observed RTAs in 82.5% of head injuries resulting in facial nerve paralysis (11). The slightly lower incidence of RTAs in our cohort compared to these reports may be attributable to regional variations in trauma patterns or the inclusion of assault and fall mechanisms in our study design. Nevertheless, RTAs remained the predominant etiology, consistent with global epidemiological trends.

Our analysis revealed a distinct pattern in the onset and severity of facial nerve palsy, with immediate presentation (occurring at or shortly after injury) observed in 70% of cases. In comparison, delayed onset accounted for the remaining 30%. This distribution contrasts with specific literature reports; notably, Shankar et al. documented delayed palsy in 76.6% of cases in their series (13). This discrepancy may be attributed to our tertiary care center's patient population, which typically receives more severe trauma cases. Immediate-onset palsy is widely recognized as indicative of more severe neural injury, potentially involving nerve transection or significant compression (9). This correlation was substantiated in our cohort by the high prevalence (60%) of complete paralysis (House-Brackmann Grade VI) (14) upon initial presentation. The predominance of acute, complete presentations in our series likely reflects both our stringent diagnostic criteria (requiring both clinical and radiologic confirmation) and the specialized nature of our trauma referral center. Current management guidelines accordingly recommend urgent surgical exploration and decompression for acute complete injuries (3, 9) while delayed or incomplete presentations are typically managed conservatively with corticosteroids and observation, owing to their generally more favorable prognosis (3, 15, 16). Consistent with established trauma profiles, the majority of patients in our cohort demonstrated significant associated intracranial injuries. Radiological findings included intracranial hemorrhage (72.7%) and pneumocephalus (67.1%), (17) which aligns with the documented association between temporal bone fractures and intracranial injury reported by Mistry et al. (18, 19) Additionally, cerebrospinal fluid (CSF) leak was identified radiologically in 17.5% of cases, a proportion higher than the approximately 9% reported in some temporal bone fracture studies (10, 20-22). This variance may be attributable to our comprehensive imaging assessment protocols, which include a detailed evaluation of paranasal sinus and mastoid air cell opacification, as well as our inclusion of all skull base CSF leaks, rather than exclusively temporal bone leaks. Our findings further emphasized the critical role of advanced imaging, as high-resolution MRI demonstrated abnormalities of the facial nerve (including segmental thickening and enhancement) in all FNP cases (23), underscoring its superior sensitivity for neural injury detection (24-26) compared to CT, which showed definitive bony impingement in only 10% of cases. This significant discrepancy highlights that the pathophysiological mechanism of facial nerve injury in blunt trauma most frequently involves edema and microdisruption rather than direct mechanical compression by fracture fragments (27, 28). This study corroborates the established understanding that traumatic facial nerve palsy represents an infrequent yet clinically significant complication of skull base fractures. Our reported incidence of 7.0% aligns with previously published rates in the literature (8, 9). The observed variability across studies—ranging from approximately 5% to over 10% (10, 29)—appears principally attributable to methodological differences in case

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selection and diagnostic criteria. Unlike studies restricted to temporal bone fractures, our inclusive approach, encompassing all skull base fractures with MRI confirmation, may have resulted in a cohort with a heightened representation of neural involvement. Our comprehensive stratification analysis failed to identify significant demographic or injury-related predictors of FNP development. This finding is consistent with the broader literature, which has similarly struggled to establish robust predictive factors beyond fundamental fracture characteristics and injury mechanics. Although we observed a clinically suggestive elevation in FNP incidence among fall injuries (9.7%) compared to other mechanisms, this difference did not achieve statistical significance, likely reflecting the limited statistical power inherent in our sample size. Previous investigations have also identified fracture pattern—specifically the established difference in FNP risk between longitudinal (10-25%) and transverse (up to 50%) fractures (29)—as the most reliable prognostic indicator; however, our study did not incorporate this specific radiographic classification.

From a clinical perspective, the timely recognition of facial nerve injury remains paramount for appropriate management stratification. Current evidence supports early surgical intervention for acute complete paralysis (3,30, 31) while adopting a conservative approach incorporating corticosteroids and physical therapy for delayed or incomplete presentations. While the present study does not address therapeutic outcomes, our findings underscore that a substantial minority of patients with skull base fractures will require specialized evaluation by multidisciplinary teams. The critical importance of early detection through meticulous serial examination and advanced MRI is further emphasized by the frequent diagnostic challenges presented by intubated or comatose patients in the trauma setting.

This investigation provides valuable region-specific epidemiological data that substantially mirrors international experience, confirming that FNP complicates approximately 5-10% of skull base fractures (8, 9). These findings hold significant implications for local trauma systems planning and clinical practice. Recognition of the 7% baseline risk—particularly its association with severe, acute presentations—should prompt heightened clinical vigilance, expedited imaging protocols, and consideration of early specialist consultation. Our results reinforce the necessity of collaborative management between neurosurgical and otolaryngological services in optimizing care for these complex injuries.

While providing prospectively collected data with rigorous diagnostic criteria, several methodological constraints warrant acknowledgment. The limited number of FNP events (n = 10) restricted statistical power for subgroup analyses and likely contributed to the non-significance of the observed clinical trends. Our single-center design at a tertiary trauma facility may limit the generalizability of our findings to other practice environments and patient populations. The exclusion of patients with severe neurological depression (GCS <8) potentially introduces selection bias, possibly resulting in underestimation of the true FNP incidence, as these individuals demonstrate elevated risk for associated injuries. Finally, the cross-sectional nature of this study precludes assessment of long-term functional outcomes or comparative effectiveness of different management approaches, representing an important direction for future research.

CONCLUSION

In conclusion, this prospective study employing stringent clinical and magnetic resonance imaging criteria establishes that facial nerve palsy complicates approximately 7% of skull base fractures managed at our tertiary care institution. The predominance of acute, complete presentations within our cohort indicates a patient population with high-grade neural injuries that demands heightened clinical vigilance. A pivotal finding emerged from the critical diagnostic contribution of MRI, which consistently demonstrated neural abnormality in all clinically evident cases, whereas computed tomography rarely identified direct bony impingement. This striking discrepancy underscores the inherent limitations of CT alone in excluding significant neural trauma in this patient population.

These findings provide crucial region-specific epidemiological information that can directly inform trauma system planning, resource allocation strategies, and patient counseling practices. The results emphasize the necessity of implementing a standardized diagnostic protocol incorporating timely neurological assessment and advanced MRI evaluation in the management of skull base fractures. Such an approach enables clinicians to identify facial nerve injury promptly, accurately characterize its severity, and initiate appropriate management—ranging from surgical intervention for acute complete palsies to conservative measures for delayed presentations—with the ultimate goal of optimizing functional recovery and cosmetic outcomes for affected patients.

DECLARATIONS

Data Availability Statement

All data generated or analysed during the study are included in the manuscript.

Ethics approval and consent to participate

Approved by the department Concerned. (IRB)

Consent for publication

Approved

Funding

Not applicable

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

AUTHOR CONTRIBUTION

***MUHAMMAD SOHAIB KHAN* (Resident)**

Conception Of Study, Data Acquisition, Study Design, Review of Manuscript, Manuscript Drafting, Manuscript Revisions ,and Final Approval f Manuscript.

***SEEMA SHARAFAT* (Associate Professor)**

Conception Of Study,Critical Guidance, Supervision of Entire Research Process, Final Approval of Manuscript.

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