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Evaluation of Clinical Outcome in Traumatic Facial Nerve Paralysis

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Abstract

Introduction The facial nerve is the most commonly paralyzed nerve in the human body, resulting in far-reaching functional, aesthetic and emotional concerns to the patient.

Objective Evaluation of the clinical outcome of 47 patients with traumatic facial nerve paralyses, with respect to clinical recovery and audiological sequelae.

Methods A descriptive longitudinal study was conducted over 24 months between January 2017 and December 2018 at a tertiary center with detailed clinical, topodiagnostic, audiometric and radiological evaluation and regular follow-up after discharge.

Results Road traffic accidents constituted 82.98% of the trauma cases, out of which 76.60% were found to be under the influence of alcohol.

Delayed facial paralysis was observed in 76.60% cases. Temporal bone fracture was reported in 89.36%, with otic capsule (OC) sparing fractures forming 91.49% of the cases. Topologically, the injury was mostly at the suprachordal region around the second genu. The majority of the patients (65%) attained full recovery of facial nerve function with conservative medical management. Audiometrically, 77.27% of the patients had hearing loss at the time of presentation, of which 64.71% were conductive in nature; 51.22% attained normal hearing at follow-up visits.

Conclusion Early initiation of steroid therapy, concurrent eye care and physiotherapy are the cornerstones in the management of traumatic facial nerve paralysis.

Keywords
- facial nerve
- facial paralysis
- temporal bone
- logistic regression

Introduction

The facial nerve is the most frequently affected cranial nerve in the body following trauma, resulting in functional, cosmetic and emotional issues for the victim.[1] The main indicative symptoms of lower motor neuron (LMN) facial paralysis causing distress to the patients includes loss of wrinkling, inability to close the eyes and deviation of the angle of the mouth (►**Fig. 1 A**, **B** and **C**). This is particularly true in a country like India where road traffic accidents (RTAs) and assault related injuries are widely prevalent. Apart from RTAs, traumatic facial nerve paralysis may result from several other causative factors such as blunt trauma, fall, stab wounds, gun shots or iatrogenic injury.[2–5] Acute facial nerve paralysis can also occur due to suicide attempt by hanging.[6] Temporal bone

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Fig. 1 Indicative symptoms like: (A). loss of wrinkling. (B). inability to close the eyes and (C) deviation of the angle of mouth, respectively.

fractures constitute the most common cause of traumatic injury to the facial nerve,[2,4,7,8] and are the second most common cause of facial paralysis in adults after Bell palsy.[9] It is against this background that the present study attempted to evaluate the clinical outcome of cases of traumatic facial nerve paralysis who presented to our tertiary referral center.

Method

A descriptive longitudinal study was conducted on patients diagnosed as having traumatic facial nerve paralysis at the tertiary care center during the period from January 2017 to December 2018, after obtaining clearance from the institutional ethics committee. A written informed consent was obtained from all respondents. All cases of facial nerve paralysis following a history of road traffic accidents, fall, assault or sports-related injuries with clinical features suggestive of trauma presenting to the study center were included, while cases of facial paralysis with etiology other than trauma were excluded. A total of 47 cases were reported during this period for the study. Follow-up was conducted up to a period of 3 months, during which 43 cases turned up while 4 patients were lost to follow-up.

The relevant information was collected using a pre-tested structured proforma. Clinicoradiological features of the respondents were recorded. Detailed clinical, topodiagnostic, audiometric and radiological evaluation were performed according to the institutional protocol. The hearing assessment was done by the tuning fork (TF) tests and pure tone audiometry (PTA), while the facial nerve function was evaluated using the House-Brackmann (HB) grading. There are different TF tests described in the literature; but the Rinne and Weber tests are complementary to each other and form the most widely used tuning fork tests for screening. The Rinne and Weber tests were performed using a tuning fork of 512 Hz frequency. The rationale for using the 512 Hz tuning fork were optimum decay time, minimal number of overtones and relation to speech frequency. The radiological evaluation was done using high resolution computed tomography (HRCT) of the temporal bone. The option of conservative medical or surgical management was documented. The Follow-up was conducted after 3 months to assess the recovery parameters with respect to facial nerve function and audiological deficits. The House-Brackmann grade was re-evaluated during the follow-up visits to assess the recovery along with a reassessment of hearing deficits using TF and PTA.

Data collected were coded and entered into a Microsoft Excel (Microsoft Corporation, Redmond, WA, USA) worksheet and analyzed using the statistical software SPSS for Windows, version 16.0 (SPSS Inc, Chicago IL, USA). Quantitative variables were summarized on mean and standard deviation (SD). The categorical variables were summarized as proportions and percentages based on the frequency of occurrence. The Pearson chi-squared (χ^2) test was employed to assess whether the proportions of observations falling in different categories were the same as the expected. A prerequisite for the χ^2 test is to have the expected frequencies in each cell > 5. The Fisher exact test was relied upon to overcome the problem when such situation was encountered.[10] A probability value $(p) < 0.05$ was considered statistically significant.

Two conditions are to be satisfied for a paired *t*-test. First, the paired observations must be related, and second the difference of paired samples are normally distributed. The paired *t*-test could not be conducted as the differences of the paired observations were not found to be normally distributed based on the Shapiro-Wilk test. The Wilcoxon matched-paired test, a nonparametric equivalent of the paired *t*-test, was therefore employed to assess whether the clinical intervention resulted in improvement over the respective benchmark observations for facial nerve functioning and hearing status. The effect size (ES) was estimated to assess the magnitude of the observed effect independent of the sample size.[10]

The ES for the chi squared test was computed as: $\frac{\chi 2}{(N)*(K-1)}$ where, N = total sample size across all categories, and K= number of categories. The ES for Wilcoxon matched paired test was worked out as: $\frac{Z}{\sqrt{N}}$. The suggested threshold levels for ES are: 0 to 0.2 representing small ES; 0.3 to 0.5 representing medium ES; and > 0.5 representing large ES.[10]

An attempt was made to identify the factors influencing the clinical outcome of good and bad prognosis using a binary logic regression model. The outcome variable was prognosis: (0: for poor prognosis and 1: for good prognosis) and the parameters of the model were estimated by the method of Maximum Likelihood Estimation (MLE), which ensured that the estimators are consistent and efficient asymptotically. The logistic coefficients are interpreted as the change in the log odds ratio (OR) associated with one unit change in the independent variable. As the logistic regression is sensitive to multicollinearity (i.e., high correlations among the predictor variables) and outlier values, a diagnostic test was performed to rule out both problems.[10] The goodness of fit of the model was tested by the Pearson χ^2 statistic and the independence of explanatory variables was tested by the Hosmer and Lemeshow χ^2 test. It is analogous to the global F test, estimated by dividing the - 2 times the log of the likelihood (- 2 LL) of the model by the original -2 LL. It has a chi squared distribution with N–M degrees of freedom. The significance of the explanatory variables were tested using the WALD statistic. It tells us whether the β coefficient for the predictor is significantly different from zero.

Results

Road traffic accident was the major cause of trauma accounting for nearly 83% of cases reported in the study. This was followed by fall from height (12.76%). There was one case each of assault and sports-related injury. Of the 39 RTAs, 32 cases were involving two-wheelers (82.05%). Only 3 out of 32 two-wheeler drivers were using protective devices like helmet at the time of trauma. The influence of alcohol was detected in 36 out of 47 trauma (76.60%) cases presented.

Table 1 Onset of traumatic facial paralysis at presentation

Onset of Facial Paralysis	Frequency	Per cent	χ^2 values
Immediate	11	23.40	$\chi^2 = 0.308$ df = 1 $p = 0.579$ ES = 0.007
Delayed	36	76.60	
Total	47	100.00	

Abbreviations: df, degrees of freedom; ES, effect size.

Among the 47 cases presented, 40 (85%) were males and 7 (15%) were females, with a male-female ratio of 6:1. The age of the patients varied from 5 to 53 years old (►**Fig. 2 A** and **B**). The most common age group was between 16 and 30 years old (36.17%) and between 31 and 45 years old (36.17%), followed by between 46 and 60 years old (19.15%). The mean age of the patients was 32.72 years old ± 12.74 standard deviation (SD).

Nearly 81% of cases had ear bleeding at the time of presentation, constituting the major presenting complaint. This was followed by other complaints such as hard of hearing (72.34%), loss of consciousness (70.21%), altered sensorium (65.96%) and headache (61.70%), in that order. Around 45% of the cases had vomiting and 40% had nose bleeding following trauma at the time of presentation. Less frequent presenting complaints included tinnitus (25.53%), vertigo (23.40%), oral bleeding (19.15%), seizure (8.51%) and clear otorrhoea suggestive of cerebrospinal fluid (CSF) leak in 3 (6.38%) cases.

Out of the 47 cases, 11 cases (23.40%) exhibited immediate paralysis, while 36 cases (76.60%) had delayed onset of facial paralysis (►**Table 1**). The χ^2 analysis was statistically nonsignificant at $\alpha = 0.05$. Therefore, the null hypothesis that the observed proportions falling in the immediate and

Fig. 2 Facial asymmetry: (A) Pediatric case (B). Adult case.

Table 2 Hearing assessment based on tuning fork tests at presentation

Hearing Status	Frequency	Per cent
Normal	10	21.28
Hearing Loss	37	78.72
Total	47	100.00

Table 3 Hearing assessment based on pure tone audiometry at presentation

Hearing Status	Frequency	Per cent
Normal	10	22.73
Hearing Loss	34	77.27
Total	44	100.00

Table 4 Type of hearing loss based on pure tone audiometry at presentation

Type of Hearing Loss	Frequency	Per cent	Fisher Exact Test
CHL	22	64.71	$\chi^2 = 5.020$ df = 2 Exact $p = 0.081$ ES = 0.074
SNHL	2	5.88	
Mixed HL	10	29.41	

Abbreviations: df, degrees of freedom; CHL, conductive hearing loss; ES, effect size; HL, hearing loss; SNHL, sensorineural hearing loss.

Table 5 Topological Location of Facial Nerve Injury

Location	Frequency	Percent
Suprachordal	27	57.45
Infrachordal	9	19.15
Transgeniculate	11	23.40
Total	47	100.00

Table 6 House-Brackmann grades at the time of presentation

HB Grade	Frequency	Per cent
Grade I	0	0.00
Grade II	3	6.38
Grade III	13	27.66
Grade IV	21	44.68
Grade V	10	21.28
Grade VI	0	0.00

Table 7 Conventional Classification of Temporal Bone Fractures

Type of Temporal Bone Fracture	Number	Per cent
Longitudinal Fracture	28	59.57
Transverse Fracture	6	12.77
Mixed Fracture	13	27.66
Total	47	100.00

delayed categories are the same as expected is accepted. The effect size (ES = 0.007) was also small, confirming that the deviations between the observed and expected frequencies were minimal and insignificant.

The hearing assessment based on TF tests at presentation revealed that 37 out of 47 cases (78.72%) had post-traumatic hearing loss (►**Table 2**). As PTA is a standardized and sensitive tool for hearing assessment in clinical research, the same has been relied upon for classification of hearing loss. The hearing assessment based on PTA revealed hearing loss in 77%, leaving just 23% with normal hearing at the time of presentation (►**Table 3**). Nearly two-thirds of the patients suffered from conductive hearing loss (CHL), while ~ 6% had sensorineural hearing loss (SNHL), leaving 29% of cases to the category of mixed hearing loss (►**Table 4**). The Fisher exact test was performed instead of the classical chi-squared test because one cell (SNHL) was having a frequency < 5. The exact probability value was > 0.05 and, hence, the test was nonsignificant. The null hypothesis that there is no difference between the observed and expected frequencies of hearing loss was accepted. The small effect size also highlighted the minimal deviations between the observed and expected frequencies.

The location of the probable site of injury on the facial nerve is of utmost importance. The possible site of nerve injury was hypothesized utilizing three topodiagnostic tests, that is, Schirmer test, stapedial reflex and taste sensation over anterior tongue. Subsequently, the cases were categorized into injury at the suprachordal, infrachordal and transgeniculate region based on the classification by Singh et al.[11] The most common topological location of injury in the present study was at the suprachordal region (►**Table 5**) around the second genu (57.45%), followed by the transgeniculate region (23.40%).

Clinical evaluation of the facial nerve dysfunction based on the HB grading revealed that Grade IV (moderately severe dysfunction) constituted the majority of the cases (44.68%) at the time of presentation. This was followed by 27.66% Grade III injuries (moderate dysfunction) and 21.28% Grade V injuries (severe dysfunction) (►**Table 6**). No cases of Grade VI injury (total dysfunction) were encountered.

High resolution computed tomography of the temporal bone was relied upon to detect temporal bone fracture, course of the fracture line, presence of compressive bony spicule(s), to assess ossicular status and involvement of the OC and of the carotid canal. Temporal bone fractures (TBFs) were recorded in 42 patients (89.36%), out of which 37 (79%) were unilateral fractures, while 5 (11%) were bilateral fractures. The traditional classification of TBF based on fracture line with respect to the long axis of the petrous ridge showed that out of 47 temporal bone fractures encountered, 28 (60%) were longitudinal fractures and 6 (13%) were transverse fractures (►**Table 7**). Mixed fractures were 13 (27%) in number.

Table 8 Temporal Bone Fractures based on Otic-capsule disruption

Type of Temporal Bone Fracture	Number	Per cent	Fisher's Exact Test
Otic Capsule Sparing Fractures	43	91.49	$\chi^2 = 0.246$ df = 1 Exact $p = 0.769$ ES = 0.005
Otic Capsule Violating Fractures	4	8.51	
Total	47	100.00	

Abbreviation: df, degrees of freedom.

There is a widespread view that the traditional classification do not correlate well with the clinical findings and prognosis. Therefore, Kelly and Tami proposed a new categorization of "otic capsule sparing" (OCS) and "otic capsule violating" (OCV) system of temporal bone fracture classification in 1994. As the new classification system emphasized the functional outcome without losing the descriptive ability of the traditional system of classification, it was adopted at the Meeting of Facial Nerve Study Group of the Academy of Otolaryngology (1997). According to the new system of classification, 43 fractures (92%) were OCS while only 4 (8%) were otic capsule violating (OCV) (►**Table 8**). The Fisher exact test was performed in view of one cell (OCV fractures) having a frequency < 5. The exact probability value (p) was > 0.05 and, hence, the null hypothesis of no difference between the observed and expected frequency of OCS and OCV fractures was accepted. The small ES also indicated minimal deviations between the observed and expected frequencies.

All the cases were initially managed conservatively. The management consisted of administration of intravenous steroids such as methylprednisolone or dexamethasone; multivitamins, facial physiotherapy and eye care. Different physical rehabilitation regimen like faciomotor exercises (34 cases), electrical nerve stimulation (9 cases) and facial splint (1 case) were provided at the Department of Physical Medicine and Rehabilitation based on the individual requirement of the patients.

Surgical intervention is warranted in cases of immediate facial paralysis, where the HRCT scan indicates compressive bony spicule(s)/transection involving the fallopian canal; and where electroneuronography (ENoG) points toward Wallerian degeneration > 90%. In case of delayed onset of facial paralysis, surgical intervention is suggested if the paralysis does not improve clinically, and if the ENoG degeneration is > 90%. No cases of delayed onset of facial palsy conforming to the above criteria cropped up in the study reported.

There were 4 cases with immediate facial paralysis and supportive radiological and ENoG findings, but two patients were not willing to undergo surgical intervention for their own reasons in spite of proper counseling. Out of the other two cases who were willing to undergo facial nerve exploration and decompression, one patient had a fracture involving the tympanic segment of the facial nerve at the level of the lateral semicircular canal with bony spicules and surrounding hematoma. The other case had an injury involving the facial canal just distal to the second genu with compressive hematoma. In both instances, the compressive elements were cleared, the facial nerve sheath was excised, and nerve fiber continuity was confirmed. In addition, proximal-distal decompression of the bony nerve canal was achieved. There were no intra or postoperative complications and the recovery from general anaesthesia (GA) was uneventful. Among the 47 cases considered in the present study, there were 2 cases with immediate facial paralysis with supportive radiological and ENoG findings. However, these 2 patients refused surgical intervention for their own reasons in spite of proper counseling. But there were 2 cases that were managed surgically and not included in the present sample size ($n = 47$). The exclusion of the 2 cases above was done to ensure uniformity with regards to facial nerve function grading, because their preoperative facial nerve function was assessed by HB grading while the postoperative facial nerve function was evaluated by the Repaired Facial Nerve Recovery Scale (RFNRS).

The hearing status was reassessed with tuning fork tests and PTA during the follow-up visits after 3 months. Hearing evaluation was confirmed with PTA at the time of follow-up for all cases ($n = 47$) except for 4 particular cases that were lost to follow-up. Thus, 43 cases were reviewed in all at the time of follow-up ($n = 47 - 4 = 43$). Pure tone audiometry could not be performed in 2 further cases that came for follow-up, ear infection being the cause in one case. Another specific case refused to undergo PTA citing financial problems, for whom the TF test was performed instead. As a result, PTA could be done in only 41 cases at the time of follow-up ($n = 43–2 = 41$). Accordingly, the hearing status during presentation was reworked out for 41 cases for normalization, so that the comparison is more meaningful to undertake the matched paired Wilcoxon test (►**Table 9**). It is evident from the comparison that post-traumatic hearing loss reduced considerably from 31 to just 20 cases in a period of 3 months, representing a 27% reduction. The Wilcoxon matched paired test was highly significant at $\alpha = 0.01$, lending statistical support to the impact of clinical interventions made, pointing toward the fact that the reduction in the number of patients with hearing loss was not due to chance. The ES was also large (ES > 0.5).

The 4 cases lost to follow-up originally were excluded while comparing the HB grades during presentation and also during

Table 9 Hearing Status based on pure tone audiometry at presentation and follow-up

Hearing Status	At presentation		At follow-up	
	Frequency	Per cent	Frequency	Per cent
Normal	10	24.39	21	51.22
Hearing Loss	31	75.61	20	48.78
Total	41	100.00	41	100.00
Wilcoxon Test	Z = - 4.093; $N = 41$; 2-tailed significance ($p = 0.000$) ES = 0.639			

Abbreviation: ES, effect size.

Table 10 Comparison of Facial Nerve injury at presentation and follow-up

HB Grade	At presentation		At follow up	
	Frequency	Per cent	Frequency	Per cent
Grade I	0	0.00	28	65.12
Grade II	2	4.65	8	18.60
Grade III	12	27.91	5	11.63
Grade IV	19	44.19	2	4.65
Grade V	10	23.25	0	0.00
Grade VI	0	0.00	0	0.00
Total	43	100.00	43	100.00
Wilcoxon test	$Z = -5.846$; $N = 43$; 2-tailed significance ($p = 0.000$) ES = 0.892			

Abbreviation: ES, effect size.
* 2 surgical cases were exempted of the HB scale as these cases were measured on Repaired facial nerve recovery scale (RFNRS) after surgery.
Case 1 was reported with HB grade V at presentation which improved to grade E post surgery.
Case 2 was reported with HB grade IV at presentation which improved to grade A post surgery.

follow-up. It may be noted that during follow-up, all Grade V (severe dysfunction) injuries had recovered partially or fully (►**Table 10**). The number of Grade IV injuries reduced drastically from 19 to 2. Similarly, Grade III injuries (moderate dysfunction) had a recovery from 12 to 5. The number of patients with Grade I facial function (no dysfunction) became 28 after 3 months. More than two thirds of the reviewed cases were normal (Grade I) or near normal (Grade II) within a period of 3 months (►**Fig. 3 A** and **B**). The Wilcoxon matched paired test conducted between the HB grade at presentation and follow-up was statistically significant at $\alpha = 0.01$, leading to the conclusion that the sample values differed significantly after the clinical interventions. The large value for the ES also highlighted a large and significant impact of the interventions made. The HB grades were Grade IV and V for the two surgical cases. They improved significantly and became Grade A and E on the RFNRS, respectively, at the time of follow-up.

The logistic regression model used for studying the clinical outcome of good and bad prognosis included age, alcoholic status and habits and HB grade at presentation as the most useful predictor variables (►**Table 11**). The Pearson χ^2 was highly significant at $p < 0.01$, indicating the goodness of fit of the model. Diagnostic test was performed to rule out the problems of multicollinearity and outlier values. The variance inflation factor (VIF) values were very close to 1, indicating that there was no problem of multicollinearity in the model. Similarly, the Cook Distance was < 1, highlighting that the model was free from outlier values. The Nagelkerke R^2 is analogous to the R^2 value in any classic regression equation. It showed that 37.2% of the variation in the outcome variable was explained by the explanatory variables included in the model with an overall accuracy of 79.1%. The null hypothesis of the Hosmer and Lemeshow χ^2 test assumes independence of variables. A nonsignificant Hosmer and Lemeshow χ^2 value implied that the expected frequencies considered by the model of independence were close to those observed in the table. The signs of all the estimated βs were negative, indicating the inverse relationship between the outcome variable (prognosis) and predictor variables like age, alcoholic status and habits and HB grade at presentation. However, only the HB grade at presentation was statistically significant at $\alpha = 0.01$. It meant that a higher HB grade at presentation generally carried a low likelihood of early, good prognosis within 3 months. The

A B

Fig. 3 (A) Deviation of the angle of mouth with facial asymmetry at presentation and (B). Complete facial symmetry at follow-up.

Table 11 Logistic regression for clinical outcome of good and bad prognosis

Variables included in the model	β	Wald Statistic	Significance	Exp(B)
Constant	9.065 (3.048)	8.846	0.003	0.954
Age	- 0.047 (0.032)	2.125	0.145	0.196
HB Grade at presentation	- 1.629 (0.584)	7.781	0.005	0.643
Alcoholic status	- 0.442 (0.949)	0.217	0.642	8.647E3
Pearson χ^2	13.707 ($p = 0.003$)			
Hosmer and Lemeshow χ^2	12.265 ($p = 0.140$)			
Nagelkerke R^2	0.372			
% correct prediction				
Good prognosis	85.7%			
Bad prognosis	66.7%			
Overall	79.1%			

Figures in parentheses indicate the Standard Error (SE) of the respective estimates.

OR of each predictor variable is indicated by Exp (B). All the values are < 1, which indicated that as the predictor increases by 1 unit, the odds of outcome variable decrease by the estimated OR. For example, when the HB grade at presentation increases to a higher grade by 1 unit, the odds of good prognosis decrease by a factor of 0.643 when all other factors remain constant.

Discussion

Road traffic accident (RTA) was the major cause of trauma in the present study. Road accidents are increasingly being recognized as a major public health problem. Temporal bone injury with subsequent facial nerve paralysis is one of the most common injuries occurring from RTAs. High intensity trauma to the skull can cause more serious intracranial injuries with associated facial nerve dysfunction.

The significant male preponderance of cases reported could be attributed to more male drivers involved in road traffic accidents, especially involving two-wheelers, as is the case in the present study. As most of the RTA cases were two-wheeler riders than pedestrians, it may safely be inferred that the driving habits in males and females are different. Female drivers are, by and large, less rash and more cautious than the risk-taking behavior by male drivers while driving.[12,13] There are other reports that highlighted that males are three to four times more prone to trauma than females while driving.[14–17]

Different age groups of people have different exposure risk. Age is a predisposing as well as a prognostic factor in trauma. For instance, younger people, especially males, are at increased risk of injury because they are generally more active than older persons. Though youngsters have better road awareness and reflexes, they also turn out to be rash and negligent in their driving habits, contributing to more accidents. Elderly people with poor vision, weak lower extremities and impaired or decreased balance are also at greater risk of injury. In the present study, more cases (72.34%) were reported from the young age groups of between 16 and 30 years old and between 31 and 45 years old age groups. In other words, adolescents and young adults are at higher risk of traffic accident-related injury and post-traumatic facial paralysis. The main causes of RTAs in developing countries have been reported as over speed, failure to use protective devices like helmets in two wheeler driving and seatbelts in four wheeler driving.[16] The pattern is not different in the present study, with nearly 83% of the RTAs involving two-wheelers with 90% of two-wheeler drivers not using a helmet at the time of trauma.

Impairment of alertness by alcohol is well known.[12,15,18] As many as 77% of trauma victims were detected to be under the influence of alcohol at the time of trauma. The pattern can be summed up as intoxicated driving by young rash drivers with less driving experience devoid of adequate protection like a helmet, culminating in trauma and resultant facial nerve injury.

Around two-thirds of the trauma victims had developed delayed onset of facial paralysis, while the rest suffered from immediate onset. Studies report immediate onset of facial paralysis in 27% of cases while the remaining 73% had delayed onset of facial paralysis among patients with temporal bone fractures.[13,19] The chi-squared test also indicated that there was no statistical difference between the observed frequencies in the present study with the expected frequency reported by the earlier studies. The onset of immediate facial paralysis could be due to nerve stretching/compression by bone spicule(s) or fracture fragment/crushing/division.[20] Crush or stretch injuries result in interruption of nerve conduction similar to axonotmesis, which usually recovers in a period of 3 to 6 months. Delayed onset facial paralysis is often seen within 1 to 10 days of the trauma, with nerve edema/expanding hematoma within the fallopian canal and nerve entrapment within the fibrous tissues contiguous with the fracture being the cause.[20,21]

All the cases had varying presenting symptoms like ear bleeding, hard of hearing, loss of consciousness or altered sensorium, vertigo and tinnitus with ear bleeding. Identical post-traumatic symptoms were reported by many past studies.[5,13,22–24] The probable causes of ear bleeding include injury to the external auditory canal (EAC) or to the tympanic membrane (TM) and skull base fracture.[24–26] Traumatic involvement of the carotid canal or jugular bulb can cause profuse hemorrhage.[27] The different causes of hearing loss following temporal bone injuries include blood clots in the EAC, traumatic TM perforation, CSF or blood within the middle ear, ossicular disruption, perilymph fistula, acoustic trauma, fracture of the OC, labyrinthine or brainstem concussion.[25–28] The most common etiology of post-traumatic vertigo is Benign

Positional Paroxysmal Vertigo (BPPV).[26,27] Other causes implicated are perilymph fistula, fractures of the OC, labyrinthine or vestibular concussion, injury to the brainstem or cervical spine (that is, whiplash injury) and post-traumatic endolymphatic hydrops.[5,25,27] Post-traumatic tinnitus could be attributed to direct cochlear trauma, injury to the auditory nerve or brain parenchyma.[26]

The hearing assessment using TF and PTA highlighted widespread post-traumatic hearing loss in a vast majority (more than two-thirds) of cases. The degree of hearing loss varied from slight hearing loss to profound hearing loss. The majority of the cases suffered either from mild (27.28%) or moderate (22.73%) hearing loss. One case each suffered from severe and profound hearing loss. Conductive hearing loss (CHL) was reported in two thirds of the cases, while nearly one third of the cases had mixed hearing loss. Sensorineural hearing loss (SNHL) was recorded in two cases. The Fisher exact test showed that the observed frequencies were in accordance with the expected frequencies reported in the past.[29] The ES also indicated that the deviations between the observed and expected frequencies were minimal. Conductive hearing loss may be due to blood clots within the EAC, TM perforation, hemotympanum or ossicular disruption. Disruption of the membranous labyrinth, interruption of cochlear blood supply, cochlear concussion/hemorrhage, acoustic trauma, perilymph fistula, endolymphatic hydrops due to endolymphatic duct obstruction by the fracture may lead to SNHL.[13,23] Mixed hearing loss can occur with combined injuries to the external, middle and inner ears.[13]

Many past works consider the perigeniculate region as the most common site of facial nerve injury[24,25,30,31]. In another study, 66% of the fractures were located at the geniculate ganglion, followed by 20% at the second genu and 8% at the tympanic segment.[26] However, different views also prevailed in studies whereby the tympanic segment of the second genu and labyrinthine segment have all been implicated as the most common site of traumatic facial nerve injury.[32] The relatively higher involvement of the tympanic and vertical segment of the facial nerve was attributed to RTAs involving two wheelers, where injury is often sustained to the temporal bone while falling to the side. The tympanic segment of the nerve is susceptible to traction along its axis in longitudinal fractures (which is the most common type of temporal bone fractures encountered).[33] The most common site of injury in the present study is in the suprachordal region around the second genu.

High resolution computed tomography scan revealed that 89% of the trauma victims presented had temporal bone fractures (TBFs), with $\sim$ 79% being unilateral TBFs, while 11% were bilateral TBFs. Out of these, 60% were longitudinal fractures, whereas 13% were transverse fractures and 28% were mixed in nature. Another study[34] reported 64% longitudinal fractures, 23% transverse fractures and 13% mixed fractures. In another study, longitudinal fractures constituted between 70 and 80%, transverse fractures between 10 and 30%, and mixed fractures up to 20% in yet another study.[19] In the absence of a broad consensus on the frequency of occurrence of fractures based on the conventional classification, a goodness of fit using the chi-squared test was not attempted.

According to the new classification of TBFs, $\sim$ 91% of the fractures were OC sparing (OCS), while $\sim$ 9% accounted for OCV type of fractures. Out of 234 temporal bone fractures studied by Little et al,[35] 80% were reported as OCS and 20% as OCV. A more recent study by Kutz et al[36] reported that OCS fractures occur far more frequently ($>$ 94%) as compared with fractures that violate the OC ($<$ 6%). The exact probability value of the Fisher test and the small ES were supportive of the second finding.

There were cases of facial paralysis with no demonstrable temporal bone fracture on imaging in 5 out of 47 patients (10.64% of cases). Such an eventuality was reported in earlier studies too.[31,37] The cases of facial paralysis without temporal bone fractures were attributed to micro-trauma of the temporal bone with microtears and nerve edema, which are often missed on a plain HRCT scan.

The clinical evaluation of the facial nerve dysfunction using HB grades revealed that HB Grade IV was the most common grade of facial paralysis (44% of cases) while 23% had suffered high grade (HB V) paralysis. No case of post-traumatic total paralysis (Grade VI) was encountered in the study. A total of 33% of the cases were low grade (Grade II and III). Past works have reported Grade IV facial paralysis as the most common grade of facial dysfunction was with no case of grade VI injuries.[38,39]

All the presented cases were managed with intravenous steroid administration, eye care and physical rehabilitation. A total of 65% of the cases treated with conservative management had full recovery to HB Grade I, while 19% attained near normal facial function of HB Grade II within 3 months at the time of follow-up. Complete recovery of facial function with conservative management was reported earlier also.[2,36] Although it is difficult to attribute the efficacy of one type of rehabilitation over the other, the feedbacks of the patients were positive in that physical rehabilitation has helped them in recovery. The effectiveness of neuromuscular facial retraining techniques in combination with electromyography for improving facial function is well documented.[40]

Grade I is defined as full recovery, while a return to grade III or higher grade is indicated as poorer recovery.[41,42] All the cases who were followed-up subsequently had improvement of facial function by at least one grade. The follow-up clinics revealed that all Grade V dysfunction improved with conservative medical management and physical rehabilitation regime in 3 months, to either full (HB Grade I) or near full recovery (HB Grade II). The number of Grade IV dysfunction also reduced drastically from 19 cases to just 2 cases. All HB Grade III and Grade II injuries resolved in 3 months at the time of follow-up. The Wilcoxon matched paired test between the HB Grade at presentation and follow-up yielded statistical evidence regarding significant improvement in facial nerve function at $\alpha = 0.01$. The ES was also large. The logistic regression model suggested that the HB grade at presentation turned out to be the most decisive prognosis factor over age or alcoholic habits of the patients. It also indicated that higher HB grade at presentation was associated with lower likelihood of good prognosis within 3 months.

After comparing medical and surgical management and outcomes of temporal bone fractures, Darrouzet et al.[43] concluded that 98% of medically managed fractures associated with facial nerve paralysis resulted in recovery of function to normal or near-normal (HB Grade I to II/VI). There should be full resolution of post-traumatic edema and hematoma with steroid therapy. This indirectly indicates that the possible benefit of surgery should be only to that population of patients with radiological and/or electrophysiological indications.

The TF tests at follow-up after 3 months revealed that the hearing loss reduced from 77% cases to 44% of cases, recording a drastic fall. The PTA also revealed an identical recovery rate with a reduction of post-traumatic hearing loss from 76% of cases to 49% of cases. The Wilcoxon signed ranks matched paired test based on PTA at presentation and follow up revealed that there was a statistically significant improvement in hearing status at $\alpha = 0.01$. Normalcy returned to 51.22% cases in terms of degree of hearing loss. There was a fall in CHL from 20 cases to 15, but the number of SNHL cases increased from 2 to 5. This is due to mixed hearing loss resolving to CHL and SNHL. There were 9 cases of mixed hearing loss, out of which 3 shifted to SNHL at follow-up, while the rest resolved to CHL. Almost universally in patients with head trauma, CHL is caused by initial blood clots in the EAC, hemotympanum or effusion, which resolves after the resolution of the cause over a few days to few weeks postinjury.[2] Conductive hearing loss may persist only due to the presence of TM perforation or ossicular disruption, which occurs in 20% of the patients.[13] Resolution of post traumatic SNHL is possibly due to the efficacy of steroids on inner ear inflammation and ischemia following injury. Surgery is contraindicated if the SNHL is present in the only hearing ear.

Conclusion

The following conclusions emerge from the present study after detailed clinical, topodiagnostic, audiometric and radiological evaluation:

Road traffic accidents were the major cause of trauma, with two-wheelers involved in a large number of cases. The study subjects were predominantly young male patients in the age group of between 16 and 45 years old. Most of the victims were drivers without adequate protective devices like helmet and driving under the influence of alcohol. Facial paralysis was not immediately evident in the majority of cases, with more than three fourths of the patients developing delayed onset of facial nerve paralysis. Temporal bone fractures were present in the majority of trauma cases, with a major share being unilateral fractures. Longitudinal fractures were the major type of temporal bone fractures. The majority of the temporal bone fractures were OCS by the new classification. The most common injury according to the HB grading was of Grade IV (moderately severe dysfunction). The most common topological location of injury was at the suprachordal region around the second genu. Post-traumatic hearing loss was very common, CHL being the most common type.

Conservative management with intravenous steroids and physical rehabilitation had a good clinical outcome in terms of full recovery in nearly two thirds of the patients. More than half of the cases attained normal hearing during the follow-up at the 3rd month. The logistic regression model for predicting the clinical outcome of prognosis included age, alcoholic status and HB grade at presentation as the most useful predictor variables. House-Brackmann grade at presentation turned out to be the most decisive prognosis factor over age or alcoholic habits of the patients in indicating prognosis.

Conflict of Interests
The authors have no conflict of interests to declare.

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