

Hearing Loss in Post Traumatic Injuries

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ABSTRACT

Background: Traumatic ear and head injuries can damage the tympanic membrane, middle ear, cochlea, auditory nerve, or central auditory pathways, producing conductive, sensorineural, or mixed hearing loss. **Objective:** To determine the frequency, degree, and type of hearing loss among patients with post-traumatic ear and head injuries and describe associated otoscopic and tympanometric findings. **Methods:** This hospital-based cross-sectional study included 93 patients aged 5–45 years recruited through purposive sampling at Services Hospital Lahore and Jinnah Hospital Lahore from February to July 2025. Participants underwent otoscopy, tympanometry, and pure-tone audiometry. Categorical findings were summarized as frequencies and percentages. **Results:** Males constituted 60.2% of participants, and 78.5% were aged 16–45 years. Central tympanic membrane perforation was observed in 53.8%, while 74.2% demonstrated Type B tympanograms. Moderate hearing loss was the most frequent severity category (37.6%), followed by severe (31.2%) and mild hearing loss (26.9%). Conductive hearing loss was predominant (43.0%), followed by mixed (26.9%) and sensorineural hearing loss (25.8%). Overall, 95.7% had hearing impairment, and 69.9% had conductive or mixed loss. **Conclusion:** Post-traumatic auditory dysfunction was characterized predominantly by tympanic membrane and middle-ear abnormalities, although sensorineural involvement was also common. Comprehensive audiological assessment should form part of the evaluation of traumatic ear and head injuries. **Keywords:** Hearing loss; post-traumatic injury; tympanic membrane perforation; conductive hearing loss; sensorineural hearing loss; mixed hearing loss; hemotympanum; tympanometry; pure-tone audiometry.

INTRODUCTION

Hearing loss is a major sensory-health problem that can adversely affect communication, educational attainment, employment, social participation, psychological well-being, and overall quality of life. The World Health Organization estimates that hearing impairment affects a substantial proportion of the global population and that its burden will continue to increase without effective prevention, early identification, treatment, and rehabilitation strategies (1). Although ageing, congenital conditions, infections, ototoxic exposure, and environmental noise are well-recognized causes of auditory dysfunction, traumatic injury remains an important and potentially preventable cause, particularly among children, adolescents, and economically productive adults.

Trauma may affect the external ear, tympanic membrane, middle ear, inner ear, vestibulocochlear nerve, or central auditory pathways. Direct ear trauma, road traffic accidents, head injuries, blast exposure, barotrauma, occupational incidents, slap injuries, and insertion of foreign objects or cleaning instruments may produce temporary or permanent auditory impairment. Depending on the mechanism, anatomical site, and severity of injury, patients may develop tympanic membrane perforation, hemotympanum, ossicular-chain disruption, temporal-bone injury, labyrinthine concussion,

pneumolabyrinth, cochlear damage, or neural dysfunction (2,3). These abnormalities may result in conductive, sensorineural, or mixed hearing loss and may be accompanied by tinnitus, vertigo, dizziness, otalgia, ear fullness, or disequilibrium.

Traumatic tympanic membrane perforation is among the most frequently encountered otological consequences of direct ear trauma. It may occur following blast exposure, slap injuries, sudden pressure changes, road traffic accidents, foreign-body insertion, or instrumentation of the external auditory canal. The resulting hearing deficit is influenced by the size and location of the perforation, the degree of middle-ear involvement, and the presence of associated ossicular or inner-ear injury. Although uncomplicated perforations commonly produce conductive hearing loss, more severe trauma may damage both sound-conduction and sensorineural structures, resulting in mixed or persistent sensorineural impairment (4). A Type B tympanometric pattern may accompany tympanic membrane perforation, hemotympanum, or other middle-ear abnormalities and can therefore provide clinically useful information when interpreted alongside otoscopic and audiometric findings.

Head trauma can also impair auditory function in the absence of an obvious tympanic membrane lesion. Temporal-bone fracture, temporal-bone concussion, cochlear injury, and damage to central auditory pathways may produce hearing loss, tinnitus, dizziness, and balance disturbance. Auditory dysfunction may occur even when radiological evidence of temporal-bone fracture is absent, indicating that subtle cochlear, neural, or central injury can contribute to post-traumatic symptoms (5). Population-level evidence has also demonstrated an association between head injury and subsequent hearing-related impairment, supporting the need to incorporate hearing assessment into the clinical evaluation of trauma patients (6).

Previous studies have reported considerable variation in the type and severity of post-traumatic hearing loss. In a retrospective review of patients with post-traumatic auditory dysfunction, Alpsoy et al. identified a substantial burden of sensorineural hearing loss and documented frequent temporal-bone fractures and associated neurological injuries (7). Blast-related trauma may simultaneously affect the tympanic membrane, ossicular system, and cochlea, producing conductive, sensorineural, or mixed deficits (8). Long-term follow-up studies have further shown that although auditory thresholds may improve in some patients, persistent sensorineural hearing loss can occur following severe blast exposure, cochlear injury, or disruption of middle-ear structures (9). These findings demonstrate that trauma mechanism alone does not fully determine the resulting audiological profile and that comprehensive assessment is required to establish the type, degree, and likely anatomical basis of impairment.

The clinical characteristics and outcomes of traumatic tympanic membrane perforation have been investigated in several settings. Young adult males frequently represent the largest affected demographic, reflecting greater exposure to road traffic incidents, occupational hazards, interpersonal violence, and high-risk activities. Hearing loss and tinnitus are among the most common presenting symptoms, while perforation size, anatomical location, and penetrating mechanisms may influence healing and subsequent auditory function (4). Nevertheless, published findings vary according to patient population, referral setting, trauma mechanism, diagnostic procedures, and the timing of audiological evaluation.

Evidence describing post-traumatic hearing loss in Pakistan remains limited. Available studies and clinical reports have generally focused on selected mechanisms, such as blast injury, tympanic membrane perforation, or temporal-bone trauma, rather than simultaneously evaluating traumatic aetiology, otoscopic findings, tympanometric patterns, hearing-loss severity, and hearing-loss type within a single clinical cohort. Regional differences in road safety, occupational exposure, healthcare access, injury patterns, and referral practices may influence the presentation and distribution of trauma-related auditory dysfunction. Locally generated evidence is therefore required to guide early screening, specialist referral, diagnostic assessment, and rehabilitation planning.

A clearer understanding of the relationship between trauma mechanism, tympanic membrane pathology, middle-ear function, and hearing status may assist clinicians in identifying patients at increased risk of clinically important auditory impairment. Accordingly, this study aimed to determine the frequency, degree, and type of hearing loss among patients presenting with post-traumatic ear and head injuries at tertiary-care hospitals in Lahore and to describe the associated otoscopic and tympanometric findings. The study additionally examined the distribution of hearing-loss patterns across reported trauma mechanisms. It was hypothesized that conductive hearing loss would be the predominant audiological pattern because of the expected contribution of traumatic tympanic membrane and middle-ear pathology.

MATERIALS AND METHODS

A hospital-based observational cross-sectional study was conducted to evaluate the clinical and audiological characteristics of patients presenting with hearing loss following traumatic ear or head injury. The cross-sectional design was selected because the study was intended to describe trauma mechanisms, tympanic membrane status, tympanometric findings, and hearing-loss profiles at the time of clinical assessment rather than to evaluate recovery, prognosis, or treatment effects over time. Data were collected at Services Hospital Lahore and Jinnah Hospital Lahore, Pakistan, from February to July 2025 following approval from the relevant departmental research committee.

The target population comprised patients presenting to the participating hospitals with a history of traumatic ear or head injury and suspected or confirmed auditory dysfunction. Participants were recruited through non-probability purposive sampling during the study period. Patients of either sex between 5 and 45 years of age were eligible when they presented with traumatic ear injury, head injury associated with hearing complaints, tympanic membrane perforation, hemotympanum, or post-traumatic conductive, sensorineural, or mixed hearing loss. Patients were excluded when they had an active ear infection, pre-existing chronic ear disease, a non-traumatic auditory disorder, or another medical condition considered likely to independently influence hearing status. Written informed consent was obtained from adult participants and from a parent or legal guardian for participants who could not independently provide consent.

The required sample was estimated as 93 participants using an online sample-size calculator for frequency estimation. The calculation was based on the anticipated frequency of post-traumatic hearing loss, a 95% confidence level, and an acceptable margin of error. Eligible patients encountered during the study period were assessed according to a structured clinical and audiological protocol until the required sample was achieved.

Data collection included demographic characteristics, trauma history, reported mechanism of injury, side of ear involvement, otoscopic findings, tympanometric findings, and pure-tone audiometric results. Age was grouped as 5–15, 16–25, and 26–45 years. Trauma mechanisms were recorded using predefined categories, including slap injury, blast injury, suction-related injury, stick or cleaning-tool injury, road traffic accident, occupational accident, traffic accident occurring inside a vehicle, traffic accident occurring outside a vehicle, and other reported causes. Where clinically applicable, the reported cause of hemotympanum and the cause of traumatic tympanic membrane perforation were recorded separately.

Each participant underwent otoscopic examination to evaluate the external auditory canal and tympanic membrane. Tympanic membrane status was classified as intact, centrally perforated, or marginally perforated. Hemotympanum and the affected side were also documented. Ear involvement was categorized as right-sided, left-sided, or bilateral. Tympanometry was subsequently performed to assess middle-ear function, and findings were categorized as Type A or Type B according to the recorded tympanometric pattern. Otoscopic and tympanometric findings were interpreted together to characterize the presence and likely extent of traumatic middle-ear involvement.

Pure-tone audiometry was performed according to standard principles of manual pure-tone threshold assessment (10). Audiometric findings were used to classify hearing sensitivity by both degree and type. The degree of hearing loss was categorized as normal, mild, moderate, or severe according to the audiometric classification applied during the clinical assessment. Hearing-loss type was categorized as normal hearing, conductive hearing loss, sensorineural hearing loss, or mixed hearing loss. Conductive hearing loss was defined as auditory impairment predominantly attributable to disruption of sound transmission through the external or middle ear, including pathology associated with tympanic membrane perforation or hemotympanum. Sensorineural hearing loss represented an audiometric pattern compatible with cochlear or neural involvement following trauma. Mixed hearing loss was defined as the coexistence of conductive and sensorineural components.

The primary outcome was the type of hearing loss, classified as normal, conductive, sensorineural, or mixed. The secondary outcome was the degree of hearing loss, classified as normal, mild, moderate, or severe. Additional variables included age group, sex, trauma mechanism, cause of hemotympanum, cause and type of tympanic membrane perforation, side of ear involvement, and tympanometric pattern. The distribution of hearing-loss type and severity was also examined across the predefined trauma categories.

Measurement and classification bias were reduced by applying a consistent sequence of assessment comprising trauma-history documentation, otoscopy, tympanometry, and pure-tone audiometry. Predefined categories were used for demographic variables, trauma mechanisms, tympanic membrane findings, tympanometric patterns, and audiological outcomes. Data forms were reviewed for completeness and internal consistency, and coded entries were checked before statistical analysis. Age, sex, and trauma mechanism were considered clinically relevant factors when examining variations in hearing-loss type and severity.

Data were analysed using descriptive and inferential statistical methods. Categorical variables were summarized as frequencies and percentages. These included age group, sex, trauma mechanism, hemotympanum cause, tympanic membrane status, side of involvement, tympanometric pattern, hearing-loss degree, and hearing-loss type. Associations between trauma-related risk factors and hearing-loss type or degree were assessed using the chi-square test. Fisher's exact test was considered where expected cell frequencies were insufficient for valid chi-square testing. Statistical significance was defined as a two-sided p-value of less than 0.05. Missing or incomplete entries were reviewed before analysis, and observations without an available value for a particular outcome were omitted only from the corresponding variable-specific analysis.

Ethical approval was obtained from the relevant departmental research committee before commencement of data collection. Participation was voluntary, and informed consent was obtained before clinical and audiological assessment. Participant confidentiality was maintained by recording study variables in a structured format and analysing the information in aggregate form. No directly identifying patient information was included in the analysis or presentation of findings.

RESULTS

A total of 93 patients with post-traumatic ear or head injuries were included in the analysis. All reported demographic, clinical, otoscopic, tympanometric, and overall audiological variables contained observations for the full sample.

The largest age group comprised participants aged 26–45 years, accounting for 37 of 93 patients (39.8%), followed closely by those aged 16–25 years (38.7%). Overall, 73 participants (78.5%) were between 16 and 45 years of age. Males constituted 56 cases (60.2%), producing a male-to-female ratio of approximately 1.5:1.

Traffic accidents occurring inside vehicles were the most frequently reported cause of hemotympanum, affecting 31 patients (33.3%). Blast injuries accounted for 21 cases (22.6%), while occupational and traffic accidents outside vehicles accounted for 10.8% and 8.6%, respectively. Regarding the reported cause of tympanic membrane perforation, road traffic accidents were the most frequent category, involving 23 patients (24.7%), followed by blast injuries in 20 (21.5%) and suction-related injuries in 17 (18.3%).

Table 1. Demographic Characteristics of Participants (n = 93)

Characteristic	Category	n (%)
Age group, years	5–15	20 (21.5)
	16–25	36 (38.7)
	26–45	37 (39.8)
Sex	Male	56 (60.2)
	Female	37 (39.8)

Percentages were calculated using 93 as the denominator and may not total exactly 100.0% because of rounding.

Table 2. Clinical and Otoscopic Characteristics of Participants (n = 93)

Characteristic	Category	n (%)
Reported cause of hemotympanum	Traffic accident inside a vehicle	31 (33.3)
	Traffic accident outside a vehicle	8 (8.6)
	Occupational accident	10 (10.8)
	Blast injury	21 (22.6)
	Other causes	23 (24.7)
Reported cause of tympanic membrane perforation	No specified traumatic cause	12 (12.9)
	Slap injury	8 (8.6)
	Blast injury	20 (21.5)
	Suction-related injury	17 (18.3)
	Stick or cleaning-tool injury	5 (5.4)
	Road traffic accident	23 (24.7)
	Other causes	8 (8.6)
Tympanic membrane status	Intact	37 (39.8)
	Central perforation	50 (53.8)
	Marginal perforation	6 (6.5)
Side of ear involvement	Right	36 (38.7)
	Left	31 (33.3)
	Bilateral	26 (28.0)

Percentages were calculated using 93 as the denominator. Percentages may not total exactly 100.0% because of rounding.

Table 3. Tympanometric and Audiological Findings (n = 93)

Characteristic	Category	n (%)
Tympanometric pattern	Type A	24 (25.8)
	Type B	69 (74.2)
Degree of hearing loss	Normal hearing	4 (4.3)
	Mild	25 (26.9)
	Moderate	35 (37.6)
	Severe	29 (31.2)
Type of hearing loss	Normal hearing	4 (4.3)
	Conductive	40 (43.0)
	Mixed	25 (26.9)
	Sensorineural	24 (25.8)

Percentages were calculated using 93 as the denominator and may not total exactly 100.0% because of rounding.

Central tympanic membrane perforation was identified in 50 patients (53.8%), whereas marginal perforation was present in 6 (6.5%). Collectively, 56 of 93 participants (60.2%) had either a central or marginal perforation, while 37 (39.8%) had an intact tympanic membrane. Unilateral involvement

occurred in 67 patients (72.0%), including 36 right-sided cases (38.7%) and 31 left-sided cases (33.3%); bilateral involvement was recorded in 26 patients (28.0%).

Type B tympanograms were observed in 69 patients (74.2%), approximately three times the number with Type A tympanograms, which were identified in 24 patients (25.8%). Moderate hearing loss was the most common degree of impairment, affecting 35 participants (37.6%), followed by severe hearing loss in 29 (31.2%) and mild hearing loss in 25 (26.9%). Moderate-to-severe hearing impairment was therefore present in 64 of 93 participants (68.8%), while only 4 participants (4.3%) had hearing thresholds categorized as normal.

Conductive hearing loss was the predominant audiological type and was identified in 40 patients (43.0%). Mixed hearing loss was present in 25 participants (26.9%), and sensorineural hearing loss was present in 24 (25.8%). Overall, 89 of 93 patients (95.7%) demonstrated some degree of hearing impairment. Conductive or mixed hearing loss, both of which include a conductive component, accounted for 65 cases (69.9%), consistent with the high frequencies of tympanic membrane perforation and Type B tympanometric findings in the study population.

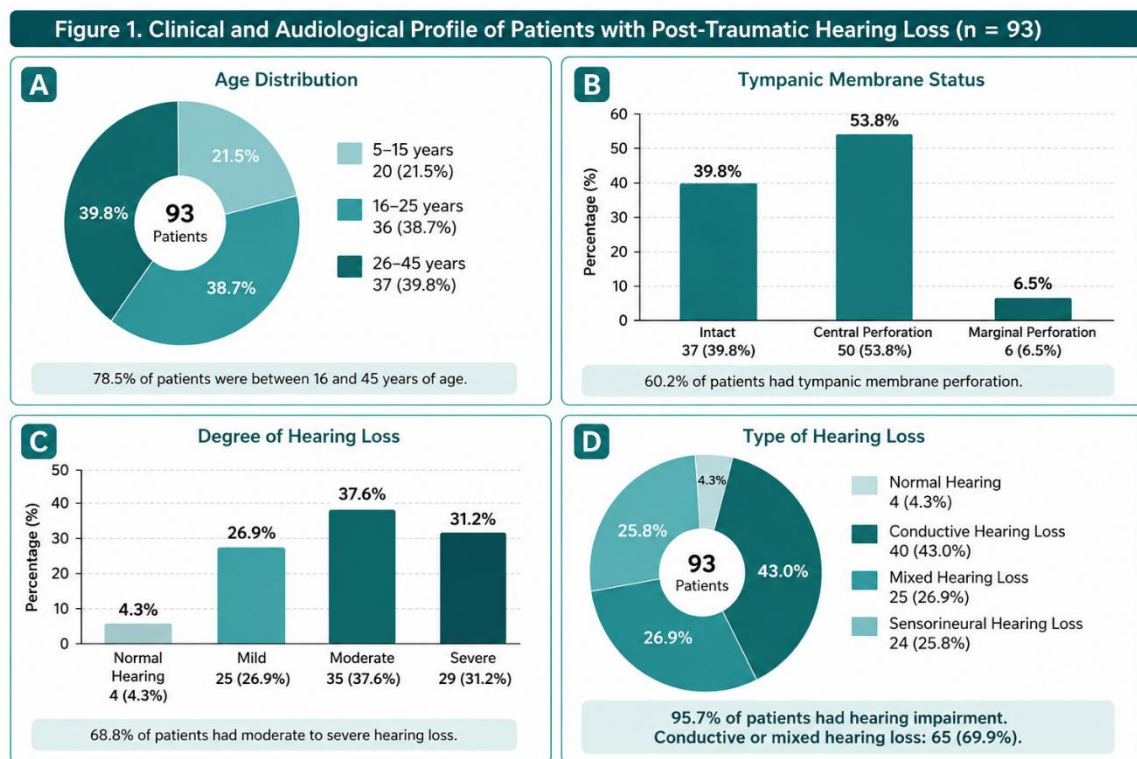


Figure 1. Clinical and audiological profile of patients with post-traumatic hearing loss. Panel A presents the age distribution, showing that 39.8% of participants were aged 26–45 years, 38.7% were aged 16–25 years, and 21.5% were aged 5–15 years. Panel B demonstrates that central tympanic membrane perforation was the predominant otoscopic finding (53.8%), followed by an intact tympanic membrane (39.8%) and marginal perforation (6.5%). Panel C shows that moderate hearing loss was the most frequent severity category (37.6%), followed by severe (31.2%) and mild hearing loss (26.9%), while 4.3% had normal hearing. Panel D demonstrates that conductive hearing loss was the predominant audiological type (43.0%), followed by mixed (26.9%) and sensorineural hearing loss (25.8%); collectively, conductive or mixed impairment affected 69.9% of participants, consistent with the substantial burden of traumatic middle-ear pathology.

DISCUSSION

This hospital-based cross-sectional study characterized the demographic, otoscopic, tympanometric, and audiological profiles of 93 patients presenting with post-traumatic ear or head injuries at two tertiary-care hospitals in Lahore. The principal findings were a predominance of patients aged 16–45 years and of male participants, a high frequency of central tympanic membrane perforation and Type B tympanograms, and a substantial burden of clinically important hearing impairment. Conductive

hearing loss was the most frequent audiological type, while moderate hearing loss was the most common severity category. Collectively, conductive or mixed hearing loss affected more than two-thirds of the participants, indicating that traumatic disruption of external- or middle-ear sound transmission contributed substantially to the observed auditory dysfunction.

Nearly four-fifths of the participants were between 16 and 45 years of age, and approximately three-fifths were male. This demographic pattern is consistent with the greater exposure of adolescents and working-age adults to road traffic incidents, occupational hazards, interpersonal trauma, blast exposure, and other high-risk activities. Otological manifestations following head injury have similarly been reported most frequently among young and middle-aged males in tertiary-care settings (11). The concentration of trauma-related auditory impairment within economically productive age groups is clinically important because even non-profound hearing loss may adversely affect communication, employment, family participation, and quality of life.

Traffic-related mechanisms represented a prominent component of the reported trauma history. Accidents occurring inside vehicles were the most frequently recorded cause of hemotympanum, whereas road traffic accidents were the leading reported cause of traumatic tympanic membrane perforation. High-energy vehicular trauma can transmit force through the temporal bone and auditory apparatus, producing hemotympanum, tympanic membrane rupture, ossicular disruption, labyrinthine concussion, cochlear injury, or neural dysfunction. The resulting audiological pattern may therefore range from a predominantly conductive deficit to mixed or sensorineural hearing loss depending on the structures involved and the magnitude of injury (5,7). These findings support the inclusion of otoscopic and audiological assessment in the evaluation of patients with ear symptoms or hearing complaints following road traffic and head trauma.

Tympanic membrane perforation was identified in 60.2% of the study population, with central perforation accounting for most abnormalities. Traumatic perforations commonly involve the pars tensa and may occur after blast exposure, slap injury, instrumentation, sudden pressure change, or direct trauma. Previous research has shown that the extent and location of a perforation, the traumatic mechanism, and associated ossicular damage can influence the resulting hearing deficit and subsequent recovery (4). In the present cohort, the high frequency of central perforation provides a clinically plausible explanation for the predominance of hearing-loss patterns containing a conductive component. Similar work in children and young adults has demonstrated that traumatic tympanic membrane perforation frequently produces conductive impairment, although the magnitude of hearing loss varies with the characteristics of the lesion and associated middle-ear injury (12).

Type B tympanograms were present in 74.2% of participants, approximately three times the frequency of Type A patterns. A flat tympanogram may occur with tympanic membrane perforation, hemotympanum, middle-ear fluid, or other abnormalities that substantially alter tympanic membrane mobility and acoustic admittance. Its predominance was therefore concordant with the high frequencies of perforation and traumatic middle-ear pathology observed during otoscopy. Tympanometry should nevertheless be interpreted together with ear-canal volume, otoscopic findings, and audiometric thresholds, because a Type B configuration alone does not identify a single pathological condition. The agreement among otoscopic, tympanometric, and audiometric patterns in this cohort supports the clinical utility of combining these assessments rather than relying on an isolated diagnostic test.

Moderate hearing loss was the most common severity category, while almost one-third of participants had severe impairment. Overall, 68.8% of the cohort had moderate-to-severe hearing loss, demonstrating that the sample comprised patients with a substantial burden of clinically meaningful auditory dysfunction. This finding may partly reflect the tertiary-care setting, where patients with more severe injury or persistent symptoms are more likely to be referred. It may also reflect delayed presentation, coexisting middle- and inner-ear damage, or the inclusion of patients following high-energy trauma.

Because the study did not evaluate time from injury to testing or longitudinal recovery, the reported thresholds represent hearing status at assessment and should not be interpreted as permanent outcomes.

Conductive hearing loss was the most frequent audiological type, affecting 43.0% of participants. When conductive and mixed hearing loss were considered together, 69.9% of patients had an impairment involving a conductive component. This distribution is biologically consistent with the observed frequencies of tympanic membrane perforation, hemotympanum, and Type B tympanometric patterns. Disruption of tympanic membrane integrity reduces efficient sound transmission, while blood or fluid within the middle-ear cleft increases acoustic impedance. More extensive trauma may additionally interrupt the ossicular chain, further increasing the air–bone gap and the severity of conductive dysfunction (14).

Mixed and sensorineural hearing loss were also common, affecting 26.9% and 25.8% of participants, respectively. These findings indicate that traumatic auditory damage was not confined to the tympanic membrane or middle ear. Blunt head trauma, blast exposure, temporal-bone concussion, rapid pressure changes, and high-energy acceleration–deceleration forces may damage cochlear hair cells, the labyrinth, the auditory nerve, or central auditory pathways. Blast-related ear injury can involve both conductive and sensorineural mechanisms, particularly when tympanic membrane rupture coexists with cochlear or ossicular injury (8,13). Persistent sensorineural threshold shifts have also been described following blast-induced perforation, emphasizing that visible middle-ear injury does not exclude deeper auditory damage (9).

The descriptive trauma cross-tabulations initially supplied with the study suggested variation in hearing-loss pattern according to injury mechanism. However, those tables contained totals that did not reconcile with the overall sample or the validated hearing-loss distributions. Consequently, trauma-specific differences should not be presented as statistically established associations until the original participant-level dataset has been verified and the analyses repeated. The present interpretation therefore focuses on the overall distributions, which were internally consistent. A reanalysis using corrected data could subsequently examine whether trauma mechanism independently predicts hearing-loss type or severity after accounting for age, sex, laterality, and tympanic membrane status.

The findings have practical implications for trauma services, otorhinolaryngology departments, and audiology units. Patients with traumatic ear injury, head trauma, hemotympanum, tympanic membrane perforation, tinnitus, vertigo, or subjective hearing difficulty should receive timely ear examination and appropriate audiological evaluation. Otoscopy can identify visible structural abnormalities, tympanometry can characterize middle-ear function, and pure-tone audiometry can determine the degree and type of impairment. Early differentiation between conductive, sensorineural, and mixed hearing loss may guide medical or surgical referral, monitoring, hearing rehabilitation, and counselling. Patients with sensorineural or mixed deficits may require particularly prompt specialist assessment because cochlear or neural injury may not be evident on routine otoscopy.

This study contributes regional evidence by evaluating multiple trauma mechanisms and combining otoscopic, tympanometric, and pure-tone audiometric findings within the same clinical cohort. The use of a standardized assessment sequence and complete reporting of the principal categorical outcomes strengthened internal descriptive consistency. Nevertheless, several limitations should be considered. The cross-sectional design did not permit evaluation of hearing recovery, persistence, or treatment response. Purposive sampling and recruitment from two tertiary-care hospitals may have produced referral and selection bias and may limit generalizability to community populations or patients with minor injuries. The sample size restricted detailed subgroup analysis, and advanced radiological findings were not available to correlate auditory impairment with temporal-bone, ossicular, labyrinthine, or intracranial injury.

Additional limitations include the absence of reported time intervals between trauma and audiological testing, detailed audiometric frequency thresholds, ear-specific air- and bone-conduction findings, acoustic-reflex results, speech audiometry, and longitudinal follow-up. The supplied aggregate data also did not permit valid multivariable analysis or reliable inferential comparison across trauma mechanisms. Future prospective multicentre studies should use consecutive recruitment, clearly defined audiometric threshold classifications, calibrated testing protocols, ear-specific reporting, verified radiological findings where indicated, and repeated follow-up assessments. Such studies could identify prognostic factors for persistent hearing loss and determine how trauma mechanism, tympanic membrane status, injury severity, and time to evaluation influence auditory recovery.

CONCLUSION

Post-traumatic ear and head injuries in this tertiary-care cohort were associated with a high burden of hearing impairment, particularly among young and middle-aged male patients. Central tympanic membrane perforation, Type B tympanograms, moderate hearing loss, and conductive hearing loss were the most frequent findings, while mixed and sensorineural deficits also affected a substantial proportion of participants. These results support the routine use of otoscopy, tympanometry, and pure-tone audiometry for patients presenting with traumatic ear injury, head trauma, or post-traumatic auditory symptoms to facilitate timely characterization and management of hearing impairment.

REFERENCES

1. World Health Organization. World report on hearing. Geneva: World Health Organization; 2021.
2. Comacchio F, Guidetti G, Guidetti R, Mion M. Pneumolabyrinth and recurrent paroxysmal positional vertigo after traumatic stapes fracture. *Ann Otol Rhinol Laryngol*. 2019;128(4):352–356.
3. Ren Y, Landegger LD, Stankovic KM. Gene therapy for human sensorineural hearing loss. *Front Cell Neurosci*. 2019;13:323.
4. Sogebi OA, Oyewole EA, Mabifah TO. Traumatic tympanic membrane perforations: characteristics and factors affecting outcome. *Ghana Med J*. 2018;52(1):34–40.
5. Kong TH, Lee JW, Park YA, Seo YJ. Clinical features of fracture versus concussion of the temporal bone after head trauma. *J Audiol Otol*. 2019;23(2):96–101.
6. Kozin ED, Knoll RM, Bhattacharyya N. Association of pediatric hearing loss and head injury in a population-based study. *Otolaryngol Head Neck Surg*. 2021;165(3):455–457.
7. Alpsoy MY, Sönmez S, Orhan Z, Orhan EK, Aslıyüksek H, Orhan KS. Evaluation of patients with post-traumatic hearing loss: a retrospective review of 506 cases. *J Int Adv Otol*. 2021;17(3):239–245.
8. Pusz MD, Robitschek J. Traumatic hearing loss in the context of blast-related tympanic membrane perforation. *Mil Med*. 2017;182(1–2):e1645–e1648.
9. Littlefield PD, Brungart DS. Long-term sensorineural hearing loss in patients with blast-induced tympanic membrane perforations. *Ear Hear*. 2020;41(1):165–172.
10. American Speech-Language-Hearing Association. Guidelines for manual pure-tone threshold audiometry. *ASHA*. 2005;20:1–16.
11. Sakthignanavel A, Poduval J, Kurien M. Otological manifestations in head injury: experience from a tertiary academic centre. *Indian J Otolaryngol Head Neck Surg*. 2022;74(Suppl 1):495–500.
12. Jabłońska J, Pietraś A, Partycka-Pietrzyk K, Mielnik-Niedzielska G. Hearing loss in children after tympanic membrane perforation: cluster analysis of 27 cases. *J Hear Sci*. 2022;12(4):1–8.

13. de Régloix SB, Crambert A, Maurin O, Lisan Q, Marty S, Pons Y. Blast injury of the ear by massive explosion: a review of 41 cases. *BMJ Mil Health*. 2017;163(5):333–338.
14. Merchant SN, Nadol JB. *Schuknecht's pathology of the ear*. 3rd ed. Shelton (CT): People's Medical Publishing House; 2010.