The Journal of International Advanced Otology

J Int Adv Otol 2024; 20(3): 210–215 • DOI: 10.5152/iao.2024.22989

Original Article

Contralateral Hearing Loss in Temporal Bone Fractures: A Potential Association with Combined Intracranial Injury

Bum-Joon Kim[1], Insik Song[2], June Choi[2], Yoon Chan Rah[2,3]

[1]Department of Neurosurgery, Korea University College of Medicine, Republic of Korea
[2]Department of Otorhinolaryngology - Head and Neck Surgery, Korea University College of Medicine, Republic of Korea
[3]Jenks Vestibular Physiology Laboratory, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts

B.J.K. 0000-0002-3701-4030, I.S. 0000-0003-3798-4413, J.C. 0000-0002-6330-279X, Y.C.R. 0000-0003-1559-5396

Cite this article as: Kim B, Song I, Choi J, Chan Rah Y. Contralateral hearing loss in temporal bone fractures: A potential association with combined intracranial injury. *J Int Adv Otol.* 2024;20(3):210-215.

BACKGROUND: Temporal bone (TB) fractures are frequently accompanied by intracranial injury. This study aimed to analyze combined intracranial injuries in relation to functional changes in the inner ear, including those of the contralateral ear, in patients with TB fractures.

METHODS: Ninety-four patients (mean age: 35.6 ± 18.7 years, M : F = 67 : 27) diagnosed with unilateral TB fracture were included. Bone conduction (BC) threshold, word recognition score (WRS), and changes in vestibular function were compared based on intracranial injuries, focusing on the contralateral side.

RESULTS: Various types of intracranial injuries were observed (67.9%). Among these, a significant association between traumatic brain injury (TBI) and otic capsule-violating fractures was noted. The BC threshold on the fractured side significantly deteriorated in patients with TBI. Additionally, a significantly worse BC threshold was confirmed on the contralateral side in patients with TBI, intracranial hemorrhage (ICH), and contrecoup injury. The follow-up BC threshold did not improve or differ, regardless of high-dose steroid administration. The initial WRS and canal paresis in the bithermal caloric test were not significantly different in the presence of each intracranial injury. Concurrent fluctuations in the pressure of the cerebrospinal fluid space and perilymphatic space were speculated to be the potential underlying mechanisms.

CONCLUSION: A significantly worse BC threshold was confirmed on the contralateral side of patients with TBI, contrecoup injury, ICH, and on fracture sides of patients with TBI.

KEYWORDS: Temporal bone fracture, contrecoup labyrinthine concussion, traumatic brain injury, hearing loss

INTRODUCTION

Temporal bone fracture is a common head injury, which accompanies 14%-70% of patients with traumatic cranial fractures.[1-3] It is frequently associated with sensory deficits, such as hearing loss, occurring in up to 57% of reported cases owing to damage to the middle and inner ear structures.[4-6] Dizziness, with an incidence of up to 83%, is also recognized as one of the most frequently associated consequences.[7,8] Therefore, temporal bone fractures have attracted attention in various clinical fields. Nevertheless, disruption of the microstructures inside the inner ear has been regarded as the main cause of auditory and vestibular functional deterioration.[4-8] Considering the microanatomy and physiology of the inner ear, this seems indisputably reasonable. However, there are patients complaining of unexplained hearing loss without apparent inner ear disruption after head trauma and these cases are often overlooked as post-concussive syndromes. Traumatic brain injury (TBI) is a highly prevalent critical health problem with 2.87 million newly developed cases and 50000 deaths per year in the United States. Hearing loss is reported in up to 67% of these cases.[9-12] A careful investigation is warranted for these patients with hearing loss after TBI. Specifically, the authors focused on hearing changes in the contralateral ear based on the observation of so-called contrecoup brain injury in a proportion of patients. Previous studies suggested that the generated intracranial pressure waves delivered to the contralateral side of the cerebrospinal fluid space can subsequently affect the contralateral inner ear. [13,14]

Corresponding author: Yoon Chan Rah, e-mail: rah_yoonchan@korea.ac.kr
Received: November 30, 2022 • **Revision requested:** September 23, 2023 • **Last revision received:** November 22, 2023 • **Accepted:** January 19, 2024 • **Publication Date:** May 23, 2024
Available online at www.advancedotology.org

This study aimed to analyze the impact of intracranial injuries on the functional changes of the inner ear, and thereby, to focus on the changes not only on the fractured side but also on the contralateral ear.

MATERIAL AND METHODS

A retrospective review of medical records was carried out for 94 patients with otic capsule sparing (OCS) temporal bone fractures who first visited our hospital from June 2007 to August 2020 [15-17]. Cases were selected based on the following inclusion criteria: (1) radiologically confirmed unilateral temporal fracture and (2) existence of validated initial pure-tone audiometry with adequate auditory masking, measured within 1 month of the causative accident. Radiologic images of high-resolution temporal bone computed tomography were reviewed and classified by 2 otologists and a neurosurgeon, blinded to each other. Conflicting cases were reviewed again and the determination was based on the opinion of the majority. In patients with intracranial injury, the radiological parameters were defined as follows: (1) TBI, when any brain injury was identified on neuroimaging; (2) contrecoup brain injury, when focal brain injury was identified contralaterally to the temporal bone fracture; (3) intracranial hemorrhage (ICH); and (4) parenchymal injury of the temporal lobe.

Sixty-three patients completed follow-up audiometry more than 2 months after the initial audiometry and were included in the comparison of hearing changes after treatment. Twenty-seven patients with confirmed bithermal caloric test results were included in the analysis of vestibular functional changes.

A standard protocol for the pure-tone audiometric test was applied with adequate auditory masking.[18] The bone-conduction and air-conduction threshold of pure-tone average (PTA) was obtained by averaging the thresholds of 0.5, 1, 2, and 3 kHz.[19] To avoid contralateral hearing, adequate auditory masking was applied based on the plateau method. It was applied for all bone conduction tests and the air conduction tests with unmasked threshold difference of both ears greater than interaural attenuation values. [20] The speech recognition threshold (SRT) and word recognition score (WRS) were obtained simultaneously. The SRT was the lowest level at which the participants could identify 50% of the suggested bi-syllabic words. The WRS was conducted using 50-monosyllabic words 30-40 dB above the SRT in each ear. [18]

MAIN POINTS

- Intracranial injury was observed in 67.9% of patients with temporal bone fracture.
- On contralateral side of temporal bone fracture, significantly worse bone conduction pure tone thresholds were identified in patients with contrecoup injury, intracranial hemorrhage, and traumatic brain injury.
- The bone conduction threshold on the fractured side also significantly deteriorated in patients with traumatic brain injury.
- Concurrent fluctuations of the pressure in cerebrospinal fluid space and perilymphatic space were speculated to be a potential underlying mechanism.

This study was approved by the institutional review board of ethics committee of Korea University Ansan Hospital (Approval No: 2020AS0260; Date: September 15, 2020). The committee waived the requirement for informed consent from patients because this was a retrospective review of medical records. The studies were performed in accordance with the approved guidelines and the Declaration of Helsinki.

Statistical Analyses

The hearing thresholds were compared primarily based on the bone conduction (BC) thresholds of pure tone audiometry instead of the air-conduction threshold because the BC threshold principally reflects auditory neural function. The initial BC thresholds of the PTA were compared based on the presence of each type of intracranial injury. Changes in BC thresholds were analyzed between the initial and final hearing thresholds, which were obtained at least 2 months after the initial audiometry. Hearing changes were analyzed according to whether or not high-dose steroids were administered. Changes in word recognition score (WRS) was also analyzed.

The association between subjective dizziness and intracranial injury was assessed. Changes in the canal paresis CP (%) of the bithermal caloric test were analyzed. The time constant (Tc) of the step velocity acceleration in the rotary chair test and the inter-aural amplitude difference (IAD) of the cervical vestibular-evoked myogenic potential (cVEMP) were also analyzed.

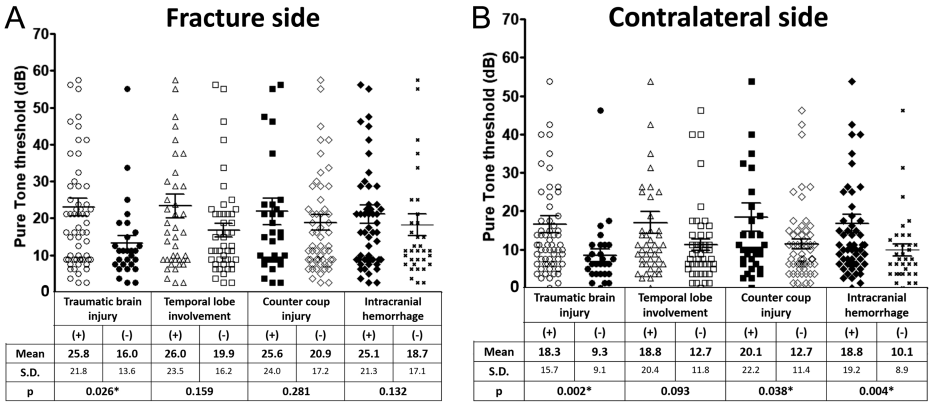
All data are expressed as mean ± standard deviation (SD). Student's *t*-test, paired *t*-test, or 1-way analysis of variance (ANOVA) were applied to compare the mean values of the data. For non-parametric cases, the Mann–Whitney *U* test, Wilcoxon signed-rank test, or Kruskal–Wallis test were applied. The chi-square test or Fisher's exact test was applied to calculate the statistical significance of the difference between the expected and observed frequencies. Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) software version 15.0 (SPSS Inc.; Chicago, IL, USA).

RESULTS

Hearing Thresholds

The average age of the 94 patients with OCS fracture who were included in the hearing analyses was 35.6 ± 18.7 years (range: 4.5–80.6 years) with a predominance of male patients (M : F = 67 : 27). Both sides were similarly affected (right : left = 41 : 53). Among them, 64 (68.1%) had various types of TBI (Supplementary Table 1).

All patients got initial PTA measured at 15.1 ± 10.3 days after the causative accident. The BC thresholds of the PTA were significantly deteriorated in patients with TBI on the fracture side (25.8 ± 21.8 dB, n = 63) compared with that in those without the injury (16.0 ± 13.6 dB, n = 30) (P = .026*, Figure 1A). The contralteral BC thresholds were also significantly deteriorated (18.3 ± 18.4 dB vs. 9.3 ± 9.1 dB, P = .002*) in patients with TBI (Figure 1A). The patients with a contrecoup brain injury and ICH had significantly worse BC thresholds (20.1 ± 22.2 dB, n = 34 for contrecoup brain injury; 18.8 ± 19.2 dB, n = 57 for ICH) on the contralateral side of the fracture than that in those without injury (12.7 ± 11.4 dB, n = 59, P = .038* for contrecoup brain injury; 10.1 ± 8.9 dB, n = 36, P = .004* for ICH) (Figure 1B).



	Traumatic brain injury		Temporal lobe involvement		Counter coup injury		Intracranial hemorrhage	
	(+)	(-)	(+)	(-)	(+)	(-)	(+)	(-)
Mean	25.8	16.0	26.0	19.9	25.6	20.9	25.1	18.7
S.D.	21.8	13.6	23.5	16.2	24.0	17.2	21.3	17.1
p	0.026*		0.159		0.281		0.132	

	Traumatic brain injury		Temporal lobe involvement		Counter coup injury		Intracranial hemorrhage	
	(+)	(-)	(+)	(-)	(+)	(-)	(+)	(-)
Mean	18.3	9.3	18.8	12.7	20.1	12.7	18.8	10.1
S.D.	15.7	9.1	20.4	11.8	22.2	11.4	19.2	8.9
p	0.002*		0.093		0.038*		0.004*	

Figure 1. Bone conduction thresholds in otic capsule sparing temporal bone fracture cases. Bone conduction thresholds according to the intracranial injuries were calculated for 93 cases with otic capsule sparing temporal bone fracture. Significantly worse (higher) bone conduction threshold was confirmed in patients with traumatic brain injury ($P = .026$) on the fracture side of temporal bone (A). Significantly worse bone conduction threshold was also confirmed in patients with traumatic brain injury ($P = .002$), counter-coup injury ($P = .038$), and intracranial hemorrhage ($P = .004$) on the other side (B).

The patients who underwent craniotomy had significantly worse BC thresholds (39.5 ± 22.2 dB, n = 5) on the fracture side compared to those who did not (21.4 ± 39.5 dB, n = 87) ($P = .032$*). However, the hearing thresholds did not differ according to the presence of temporal lobe injury (n = 42).

The changes in BC thresholds were analyzed by comparing the initial and the final PTA for patients who had a follow-up audiometry following more than 2 months from the initial test (average 8.6 ± 11.4 months, range: 2.1–58.2 months). In the analysis of the fractured and contralateral sides, the BC thresholds were not significantly changed in the final PTA regardless of the type of injury as well as when analyzed for all OCS patients (Table 1). Among the 63 patients with OCS fractures who had follow-up PTA data, 39 received high-dose steroid therapy (mostly 48–64 mg of prednisolone or an equivalent dose of another type of glucocorticoid). However, the average BC threshold was not changed despite steroid administration; the fracture side (1.14 ± 16.9 dB improvement for steroid group, 1.00 ± 9.97 dB improvement for the non-steroid-treated group, $P = .890$), the contralateral side (0.65 ± 19.73 dB worsening for steroid group, 0.82 ± 8.88 dB improvement for the non-steroid-treated group, $P = .671$).

The SDS score did not differ according to the type of intracranial injury (Table 2). Three patients had less than 50% WRS on the fracture side with a BC threshold of the same side greater than 60 dB. Two patients had severe ICH, and the third patient had a major contusion in the ipsilateral temporal lobe.

The most frequent mode of injury was slip (fall) down (n = 40, 42.5%), followed by traffic accidents (n = 34, 36.2%), and assault (n = 4, 4.3%). Patients injured in a traffic accident more frequently had temporal lobe injuries ($P = .011$) than those injured in a fall (Supplementary Table 2A). The initial bone conduction threshold did not differ between 2 types of causative injury (Supplementary Table 2B).

Vestibular Functional Changes

Although the CP in the bithermal caloric test was generally higher (worse) in patients with intracranial injury, it was not statistically different based on the type of intracranial injury. (Supplementary Table 3A). Subjective dizziness was significantly associated with TBI ($P = .037$; odds ratio, 4.800). Detailed results of the vestibular function tests are described in 'Supplementary Table 3B' for patients with OCS fracture who had an abnormal CP (≥ 25%) in the bithermal caloric test. Nine of the 10 patients had intracranial injuries. Three patients (cases 2, 8, and 10) were almost non-responsive on the fracture side in terms of CP, although the bony labyrinth was intact. In these 3 cases, the Tc of the rotary chair test decreased on the same side. Three patients (cases 3, 4, and 9) had abnormal CP on the contralateral side of the injury. In these cases, the decreased amount was relatively small (33.33 ± 9.23%), and all had TBI with 2 cases of ICH and 1 case of contrecoup injury.

The IAD of cVEMP could not be calculated in 5 of the 7 cases since the myogenic potential was unmeasurable on the fracture side, probably owing to accompanying injuries and hemorrhage in the tympanum.

Table 1. Hearing Changes Between Initial and Last Bone Conduction Threshold According to the Type of Traumatic Brain Injury

		Fracture Side			Contralateral Side		
	n	Initial†	Last†	*P*	Initial†	Last†	*P*
All OCS cases	63	22.6 ± 17.6	22.0 ± 19.2	.691	13.8 ± 13.2	14.7 ± 16.6	.559
Traumatic brain injury	47	24.5 ± 18.6	23.2 ± 19.9	.475	16.1 ± 14.5	14.6 ± 11.7	.439
Intracranial hemorrhage	41	21.9 ± 16.2	20.7 ± 17.4	.524	15.9 ± 15.1	13.8 ± 11.0	.315
Counter-coup injury	25	21.7±15.9	20.9±17.2	.601	16.6 ± 18.1	14.0 ± 12.6	.489
Temporal lobe injury	34	23.1 ± 18.6	21.6 ± 18.9	.465	15.7 ± 15.9	13.2 ± 11.7	.431

OCS, otic capsule sparing.
†Bone conduction thresholds of pure tone audiometry were compared for the cases with both pre- and post-treatment data.

Table 2. Speech Discrimination Scores According to Each Kind of Intracranial Injury

	Fracture Side			Contralateral Side		
	(+)	(–)	*P*	(+)	(–)	*P*
Traumatic brain injury	91.9 ± 15.4	93.7 ± 17.6	.778	93.1 ± 9.6	94.7 ± 11.0	.573
Intracranial hemorrhage	91.8 ± 15.7	93.3 ± 16.1	.794	94.9 ± 10.9	95.8 ± 12.6	.626
Counter-coup injury	92.5 ± 13.3	92.2 ± 17.3	.951	97.5 ± 11.5	97.2 ± 12.9	.725
Temporal lobe injury	89.2 ± 18.7	95.5 ± 11.3	.209	96.7 ± 11.1	97.3 ± 12.9	.538

Multivariate Analysis

Multiple linear regression analysis was performed for the principal variables, including ossicular injury and each type of intracranial injury (TBI, ICH, contrecoup injury, and parenchymal injury of the temporal lobe). For fracture sides, the presence of TBI (P = .003*, $\beta = 0.584$) and ICH ($P < .026$*, $\beta = 0.428$) were revealed to be significant determinants for BC threshold ($R^2 = 0.099$, $P = .008$*). On the contralateral side, the presence of TBI ($P = .030$*, $\beta = 0.224$) was a single significant determinant for BC threshold ($R^2 = 0.050$, $P = .030$*).

DISCUSSION

The integrity of the otic capsule has been considered to be the most important radiological indicator for estimating functional changes in the inner ear after a temporal bone fracture since it encloses and protects the sensory neuroepithelium of the inner ear.[4,12,20,21] Since the otic capsule is a harder bony structure compared to the surrounding trabecular mastoid bones, the otic capsule violating (OCV) fracture is relatively rare, accounting for only 2.5%-7% of all temporal bone fractures.[5,22,23] Therefore, sensorineural hearing loss comprising 27% of the cases with temporal bone fracture, mostly occurs in OCS cases.[4,6] However, relatively few studies have focused on the causative pathophysiological changes associated with sensorineural hearing loss in OCS cases.[23,24] Additionally, intracranial injuries have rarely been analyzed considering their potential association with or without hearing changes.

In our case series, a large number of patients experienced hearing loss on the fractured side, although there was no radiological evidence of direct inner ear injury (OCS cases). Previously reported determining factors mostly described changes inside the middle ear or mastoid cavity, such as the length of the fracture line, dislocation of ossicles, and volume of the mastoid cavity, to explain the cause of sensorineural hearing loss of the fractured side in OCS cases.[23,24,25] It seems reasonable for the fractured side considering that the impact of trauma could be more easily delivered to the inner ear by physiologic air or bone conduction sound transmission pathways through the ossicles or skull than a relatively indirect intracranial path.[23-26]

Instead, we focused on hearing changes on the contralateral side to minimize the direct impact of the fracture and determine the potential causative contribution of intracranial changes. Prior to the analysis, the integrity of the otic capsule and the structures of the tympanic cavity and mastoid were carefully investigated. Fairly consistent hearing loss on the contralateral side was observed in patients with intracranial injury due to TBI ($P = .002$), contrecoup injury ($P = .038$), or ICH ($P = .004$). These patterns of hearing loss in the contralateral ear could correspond to "contrecoup inner ear concussion" or "contrecoup labyrinthine concussion" considering that hearing loss was confirmed without apparent anatomical changes.[27-30]

Both possibilities could be considered for the association between contrecoup labyrinthine concussion and intracranial injuries. First, both injuries could occur separately at the same time from the impact of the causative fracture.[27-30] Second, there could be a more direct anatomical or physiological association between both injuries.[31,32] Considering the significant associations between both injuries identified in this study, the latter could be supplemented in detail as follows. As the brain floats in the cerebrospinal fluid (CSF), it could create an abrupt pressure change in the CSF during acceleration–deceleration of the brain during the head trauma.[13] Considering that the perilymphatic pressure is closely related to the CSF pressure, it is estimated that this impact can cause a sudden increase in the perilymphatic pressure of the contralateral inner ear (Figure 2A).[33] Another hypothesis is that patients with TBI could have elevated intracranial pressure due to contusion or hemorrhage during the early period of injury. Therefore, it is expected that increased CSF pressure over a considerable period could induce sustained pressure increase in the perilymph, negatively affecting the inner ear function. As reported in previous studies, pressure changes in the perilymph can subsequently damage the organ of Corti, sensory neuroepithelium, basilar membrane, and membranous labyrinth (Figure 2A).[27,28,30,33-36] On the other hand, accompanied CSF leak could also cause hearing loss. Previous studies reported that CSF leak during brain surgery was associated with hearing loss including the hearing of contralateral ear.[37-39] In these cases, the hearing loss preferentially affected low frequencies and was frequently transient.[37-39]

Direct injury to the central auditory pathway could be associated with TBI. Electrophysiological tools have frequently been employed to investigate these injuries.[40-45] In ABR, which mainly reflects the early responses in the auditory brainstem, delays in absolute latency, inter-wave latency of wave III or wave V, or changes in the amplitude of waves were reported.[43-45] In middle latency auditory-evoked potential recorded from the thalamus to the primary auditory cortex, delays or decreased amplitudes of waves were also reported.[43,46-48] However, reported data were very limited to reliably support a definite association between brain parenchymal injuries and hearing loss, while a few papers described several cases with complete hearing loss in bilateral traumatic midbrain injury.[41] In our case series, we did not find any case suspected to have so-called cortical deafness, although a decrease in speech discrimination was observed in several cases. The WRS of our patients was mostly within the normal range or showed values that could explain the poor pure tone thresholds. Only 3 patients showed asymmetrically decreased WRS. Two of 3 patients experienced severe ICH. However, in contrast, 5 patients with severe or multiple ICH with pneumocephalus or who required surgical decompression had excellent SDS (≥95%), which made it difficult to confirm a clear association between brain injury and speech recognition.[49] Research for hearing loss with temporal bone fracture has inevitable limitations since conventional hearing tests are not feasible in most cases of severe intracranial injury.

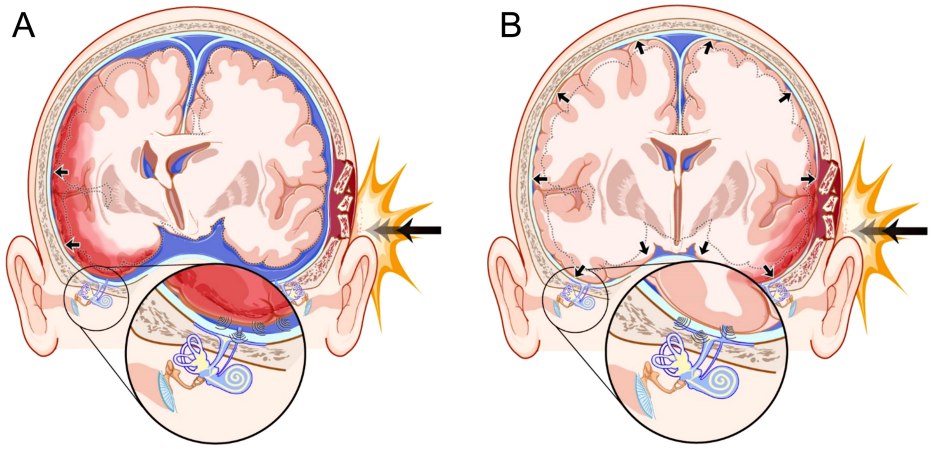


Figure 2. Presumed mechanism of contralateral labyrinthine concussion based on the pressure changes in the CSF space. A sudden acceleration-deceleration process of the brain at the moment of head trauma could cause abrupt pressure changes in the CSF space, which in turn could generate a strong pressure wave inside the perilymphatic space of the inner ear (A). An elevated intracranial pressure due to contusion or hemorrhage during the early period of injury could also result in a sustained increase of perilymphatic pressure over a considerable time periods (B). The pressure change can affect both inner ears, but on the fracture side, the effect of direct inner ear damage is much greater, so it is difficult to accurately measure the effect of the delivered pressure. *The figures are schematic diagrams that effectively show presumed effect on the contralateral ear, and may differ from actual anatomical details. CSF, cerebrospinal fluid.

This limitation becomes even more pronounced in assessing speech recognition problems because such cases frequently have generalized cognitive disorders, which obscure the exact assessment of decreased speech recognition. This limitation must be considered when interpreting hearing changes associated with temporal bone fractures.

Similarly, the true functional changes from causative accidents could be biased to some extent if data from severely injured cases are not comprehensive. As the current study was based on a retrospective collection of medical data, only patients who underwent a hearing test were included. Therefore, most slip (fall) down cases were those injured by rolling on the stairs or falling within a 2 m height (82.5%, 33 of 40 cases). Since there were fewer fall-down cases from considerable heights, it is presumed that the impact of head trauma could be underestimated. Relatively few patients underwent vestibular function tests because these tests were only recommended for patients complaining of subjective dizziness.

In the multivariate analysis, intracranial injuries were significant determinants of BC changes on both sides, whereas ossicular injury was not included. These findings suggest that the impact of intracranial injuries need to be considered as one of the major cause of hearing loss next to the obvious microanatomical damages in otic capsule.[50]

Despite the above-mentioned limitations, our study confirmed an unexpectedly high chance of concurrent intracranial injuries in patients with temporal bone fractures, which were frequently overlooked in the course of treatment. This finding suggests that close interdisciplinary cooperation is required, considering the mutual association between peripheral inner ear function and intracranial injuries. Changes in hearing on the contralateral side and their association with intracranial injuries were also proved. Although hearing changes were small on the contralateral side, those could have a considerable impact on patients because the contralateral side usually has better hearing thresholds, predominantly determining overall auditory and speech perception.

In conclusion, our data confirmed that 68.1% of the patients had intracranial injuries along with temporal bone fractures. In patients with OCS temporal bone fractures, a significantly worse BC threshold was associated with TBI on the fractured side. The impact of intracranial injuries on hearing changes was more prominent contralaterally, showing a significantly worse BC threshold in patients with TBI, contrecoup injury, and ICH.

Ethics Committee Approval: This study was approved by Ethics committee of Korea University Ansan Hospital (Approval No: 2020AS0260; Date: September 15, 2020).

Informed Consent: The ethics committee waived the requirement for informed consent from patients because this was a retrospective review of medical records.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – Y.C.R., B.K.; Design – Y.C.R., B.K.; Supervision – Y.C.R., J.C.; Resources – Y.C.R., B.K.; Materials – Y.C.R., B.K.; Data Collection and/or Processing – Y.C.R., B.K., I.S.; Analysis and/or Interpretation – Y.C.R., B.K.; Literature Search – Y.C.R., B.K.; Writing – Y.C.R., B.K.; Critical Review – Y.C.R., B.K.

Declaration of Interests: The authors have no conflict of interest to declare.

Funding: This work was supported by ICT Creative Consilience Program through the Institute of Information & Communications Technology Planning & Evaluation(IITP) grant funded by the Korea government (MSIT) (IITP-2024-2020-0-01819).

REFERENCES

1. Saraiya PV, Aygun N. Temporal bone fractures. *Emerg Radiol*. 2009;16(4):255-265. [CrossRef]
2. Nageris B, Hansen MC, Lavelle WG, Van Pelt FA. Temporal bone fractures. *Am J Emerg Med*. 1995;13(2):211-214. [CrossRef]
3. Hasso AN, Ledington JA. Traumatic injuries of the temporal bone. *Otolaryngol Clin North Am*. 1988;21(2):295-316. [CrossRef]
4. Honeybrook A, Patki A, Chapurin N, Woodard C. Hearing and Mortality Outcomes following Temporal Bone Fractures. *Craniomaxillofac Trauma Reconstr*. 2017;10(4):281-285. [CrossRef]

5. Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. *Am J Otol*. 1997;18(2):188-197.
6. Yalçıner G, Kutluhan A, Bozdemir K, Cetin H, Tarlak B, Bilgen AS. Temporal bone fractures: evaluation of 77 patients and a management algorithm. *Ulus Travma Acil Cerrahi Derg*. 2012;18(5):424-428. [CrossRef]
7. Gottshall KR, Gray NL, Drake AI, Tejidor R, Hoffer ME, McDonald EC. To investigate the influence of acute vestibular impairment following mild traumatic brain injury on subsequent ability to remain on activity duty 12 months later. *Mil Med*. 2007;172(8):852-857. [CrossRef]
8. Jury MA, Flynn MC. Auditory and vestibular sequelae to traumatic brain injury: a pilot study. *N Z Med J*. 2001;114(1134):286-288.
9. Centers for Disease Control and Prevention. *Percent distributions of TBI-related emergency department visits by age group and injury mechanism—United States, 2006–2010*. 2014. http://www.cdc.gov/traumaticbraininjury/data/dist_ed.html. Accessed 11 Oct 2014.
10. Lew HL, Garvert DW, Pogoda TK, et al. Auditory and visual impairments in patients with blast-related traumatic brain injury: effect of dual sensory impairment on Functional Independence Measure. *J Rehabil Res Dev*. 2009;46(6):819-826. [CrossRef]
11. Lew HL, Jerger JF, Guillory SB, Henry JA. Auditory dysfunction in traumatic brain injury. *J Rehabil Res Dev*. 2007;44(7):921-928. [CrossRef]
12. Lew HL, Pogoda TK, Baker E, et al. Prevalence of dual sensory impairment and its association with traumatic brain injury and blast exposure in OEF/OIF veterans. *J Head Trauma Rehabil*. 2011;26(6):489-496. [CrossRef]
13. Drew LB, Drew WE. The contrecoup-coup phenomenon: a new understanding of the mechanism of closed head injury. *Neurocrit Care*. 2004;1(3):385-390. [CrossRef]
14. Besenski N. Traumatic injuries: imaging of head injuries. *Eur Radiol*. 2002;12(6):1237-1252. [CrossRef]
15. Katz J. *Handbook of Clinical Audiology*. 5th ed. Lippincott, Williams and Wilkins; 2002.
16. Monsell EM. New and revised reporting guidelines from the Committee on HEARing and Equilibrium. American Academy of Otolaryngology-Head and Neck Surgery Foundation, Inc. *Otolaryngol Head Neck Surg*. 1995;113(3):176-178. [CrossRef]
17. Smith BL, Markides A. Interaural attenuation for pure tones and speech. *Br J Audiol*. 1981;15(1):49-54. [CrossRef]
18. Nicol JW, Johnstone AJ. Temporal bone fractures in children: a review of 34 cases. *J Accid Emerg Med*. 1994;11(4):218-222. [CrossRef]
19. Ishman SL, Friedland DR. Temporal bone fractures: traditional classification and clinical relevance. *Laryngoscope*. 2004;114(10):1734-1741. [CrossRef]
20. Dahiya R, Keller JD, Litofsky NS, Bankey PE, Bonassar LJ, Megerian CA. Temporal bone fractures: otic capsule sparing versus otic capsule violating clinical and radiographic considerations. *J Trauma*. 1999;47(6):1079-1083. [CrossRef]
21. Kim SY, Kim YJ, Kim YH, Park MH. Audiologic patterns of otic capsule preserving temporal bone fracture: effects of the affected subsites. *Clin Exp Otorhinolaryngol*. 2016;9(3):206-211. [CrossRef]
22. Vrabec JT. Otic capsule fracture with preservation of hearing and delayed-onset facial paralysis. *Int J Pediatr Otorhinolaryngol*. 2001;58(2):173-177. [CrossRef]
23. Mun SK, Oh KH, Hong YH, et al. Using temporal bone computed tomography to predict sensorineural hearing loss in otic capsule-sparing temporal bone fracture. *Injury*. 2017;48(12):2879-2883. [CrossRef]
24. Song SW, Jun BC, Kim H. Clinical features and radiological evaluation of otic capsule sparing temporal bone fractures. *J Laryngol Otol*. 2017;131(3):209-214. [CrossRef]
25. Dommerby H, Tos M. Sensorineural hearing loss in posttraumatic incus dislocation. *Arch Otolaryngol*. 1983;109(4):257-261. [CrossRef]
26. Kang TK, Ha R, Oh JH, Sunwoo W. The potential protective effects of temporal bone pneumatization: A shock absorber in temporal bone fracture. *PLoS One*. 2019;14(5):e0217682. [CrossRef]
27. Chiaramonte R, Bonfiglio M, D'Amore A, Viglianesi A, Cavallaro T, Chiaramonte I. Traumatic labyrinthine concussion in a patient with sensorineural hearing loss. *Neuroradiol J*. 2013;26(1):52-55. [CrossRef]
28. Ulug T, Ulubil SA. Contralateral labyrinthine concussion in temporal bone fractures. *J Otolaryngol*. 2006;35(6):380-383. [CrossRef]
29. Ishai R, Knoll RM, Chen JX, et al. Otopathologic Changes in the Cochlea following Head Injury without Temporal Bone Fracture. *Otolaryngol Head Neck Surg*. 2018;159(3):526-534. [CrossRef]
30. 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):96-102. [CrossRef]
31. Marchbanks RJ, Reid A. Cochlear and cerebrospinal fluid pressure: their inter-relationship and control mechanisms. *Br J Audiol*. 1990;24(3):179-187. [CrossRef]
32. Wit HP, Feijen RA, Albers FW. Cochlear aqueduct flow resistance is not constant during evoked inner ear pressure change in the guinea pig. *Hear Res*. 2003;175(1-2):190-199. [CrossRef]
33. Fitzgerald DC. Head trauma: hearing loss and dizziness. *J Trauma*. 1996;40(3):488-496. [CrossRef]
34. Toh A, Ho EC, Turner N. Contralateral deafness post head injury without temporal bone fractures. *Am J Otolaryngol*. 2010;31(1):54-56. [CrossRef]
35. Brusis T. Sensorineural hearing loss after dull head injury or concussion trauma. *Laryngorhinootologie*. 2011;90(2):73-80. [CrossRef]
36. Paparella MM, Mancini F. Trauma and Meniere's syndrome. *Laryngoscope*. 1983;93(8):1004-1012. [CrossRef]
37. Walsted A. Effects of cerebrospinal fluid loss on hearing. *Acta Otolaryngol Suppl*. 2000;543:95-98. [CrossRef]
38. Bliss MR, Jackler RK, Gurgel RK. Recurrent contralateral hearing loss after 2 craniotomies for vestibular schwannoma: etiologic implications. *Otol Neurotol*. 2013;34(7):1237-1240. [CrossRef]
39. Pogodzinski MS, Shallop JK, Sprung J, Weingarten TN, Wong GY, McDonald TJ. Hearing loss and cerebrospinal fluid pressure: case report and review of the literature. *Ear Nose Throat J*. 2008;87(3):144-147. [CrossRef]
40. Washnik NJ, Anjum J, Lundgren K, Phillips S. A review of the role of auditory evoked potentials in mild traumatic brain injury assessment. *Trends Hear*. 2019;23:2331216519840094. [CrossRef]
41. Howe JR, Miller CA. Midbrain deafness following head injury. *Neurology*. 1975;25(3):286-289. [CrossRef]
42. Nölle C, Todt I, Seidl RO, Ernst A. Pathophysiological changes of the central auditory pathway after blunt trauma of the head. *J Neurotrauma*. 2004;21(3):251-258. [CrossRef]
43. Munjal SK, Panda NK, Pathak A. Relationship between severity of traumatic brain injury (TBI) and extent of auditory dysfunction. *Brain Inj*. 2010;24(3):525-532. [CrossRef]
44. Podoshin L, Ben-David Y, Fradis M, Pratt H, Sharf B, Schwartz M. Brainstem auditory evoked potential with increased stimulus rate in minor head trauma. *J Laryngol Otol*. 1990;104(3):191-194. [CrossRef]
45. Gallun FJ, Diedesch AC, Kubli LR, et al. Performance on tests of central auditory processing by individuals exposed to high-intensity blasts. *J Rehabil Res Dev*. 2012;49(7):1005-1025. [CrossRef]
46. Musiek FE, Geurkink NA, Weider DJ, Donnelly K. Past, present, and future applications of the auditory middle latency response. *Laryngoscope*. 1984;94(12 Pt 1):1545-1553. [CrossRef]
47. Kraus N, McGee T. Clinical applications of the middle latency response. *J Am Acad Audiol*. 1990;1(3):130-133.
48. Soustiel JF, Hafner H, Chistyakov AV, Barzilai A, Feinsod M. Trigeminal and auditory evoked responses in minor head injuries and post-concussion syndrome. *Brain Inj*. 1995;9(8):805-813. [CrossRef]
49. Garde MM, Cowey A. "Deaf hearing": unacknowledged detection of auditory stimuli in a patient with cerebral deafness. *Cortex*. 2000;36(1):71-80. [CrossRef]
50. Park E, Chang YS, Kim BJ, et al. Improved prediction of hearing loss after temporal bone fracture by applying a detailed classification for otic capsule-violating fracture: A wide scope analysis with large case series. *Otol Neurotol*. 2023;44(2):153-160. [CrossRef]

Supplementary Table 1. Distribution of Accompanied Traumatic Brain Injuries

		No. of patients
Traumatic brain injury	+	64 (68.1%)
	–	30 (31.9%)
Counter-coup injury†	+	36 (38.3%)
	–	58 (61.7%)
Intracranial hemorrhage	+	57 (60.6%)
	–	37 (39.4%)
Temporal lobe injury	+	46 (48.9%)
	–	48 (51.1%)

Supplementary Table 2. Intracranial Injury and Hearing Threshold According to the Cause of Injury

A. Association between intracranial injury and cause of injury (fall down and traffic accident)

		Total	Fall †	Traffic accident†	*P*	Odds
Traumatic brain injury	+	46	21	25	.068	2.513
	-	28	19	9		
Counter-coup injury	+	23	10	13	.220	1.857
	-	51	30	21		
Intracranial hemorrhage	+	42	20	22	.203	1.833
	-	32	20	12		
Temporal lobe injury	+	34	13	21	.011*	3.355
	–	40	27	13		

†Associations were calculated for total cases with both otic capsule sparing and otic capsule violating fracture

Supplementary Table 2a. Intracranial Injury and Hearing Threshold According to the Cause of Injury

B. Initial bone conduction threshold (BCT) according to the cause of injury (fall and traffic accident)

	Fracture side			Contralateral side	
	n	BCT†	*P*	BCT†	*P*
Fall	40	18.84±17.08	.874	12.77±12.15	.729
Traffic accident	34	18.37±16.56		11.85±10.09	

†Bone conduction thresholds were compared between cases with an otic capsule-sparing fracture
Abbreviations: n, number; BCT, bone conduction threshold

Supplementary Table 3. Vestibular Function and Subjective Dizziness According to the Type of Traumatic Brain Injury

A. Canal paresis of the bithermal caloric test

		n	Canal paresis (%)	*P*
Traumatic brain injury	+	24	49.71±34.35	.151
	-	3	19.67±11.93	
Counter coup injury	+	14	47.88±37.99	.817
	-	13	44.77±30.28	
Intracranial hemorrhage	+	21	49.24±34.86	.403
	-	6	36.33±30.82	
Temporal lobe injury	+	14	48.64±34.98	.725
	-	13	43.92±33.87	

Supplementary Table 3a. Vestibular Function and Subjective Dizziness According to the Type of Traumatic Brain Injury

B. Cases with abnormal response on bithermal caloric test (CP>25%) among temporal fractures with otic capsule preservation

Case No.	Sex/age	Fracture side	Bithermal caloric test		Rotation test		cVEMP		TBI	ICH	CC
			CP	Side	Tc Rt (s)	Tc Lt. (s)	IAD	Side			
1	M/27	Rt.	48%	Rt	8	10	N/A[1]	Both	+	+	-
2	M/50	Rt.	88%	Rt	4	8	N/A[1]	Both	+	+	-
3	F/53	Rt.	44%	Lt	9	11	N/A[1]	Both	+	+	-
4	F/62	Lt.	28%	Rt	-	-	-	-	+	-	-
5	M/48	Lt.	45%	Lt	14	11	N/A[1]	Both	+	+	+
6	M/16	Rt.	57%	Rt	-	-	-	-	+	+	+
7	M/14	Lt.	28%	Lt	13	12	11%	Lt	-	-	-
8	M/37	Rt.	96%	Rt	4	11	-	-	+	-	-
9	F/46	Lt.	28%	Rt	-	-	75%	Lt	+	+	+
10	M/27	Lt.	90%	Lt	12	8	N/A[1]	Both	+	+	-–

[1]IAD cannot be calculated because the myogenic potential was not measurable on the fracture side.
Abbreviations: No, Number; M, male; F, female; Rt, Right; Lt, Left; CP, canal paresis; cVEMP, cervical vestibular-evoked myogenic potential; N/A, not applicable; TBI, traumatic brain injury; ICH, intracranial hemorrhage; IAD, inter-aural amplitude difference; CC, counter coup brain injury; Tc, time constant.