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Original Article

Association between Sensorineural Hearing Loss and Type 2 Diabetes Mellitus: A Cross-Sectional Study

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ABSTRACT

Background: Sensorineural hearing loss (SNHL) represents a significant public health concern with multifactorial etiology. Emerging evidence suggests a potential association between type 2 diabetes mellitus (T2DM) and hearing impairment, though the relationship remains incompletely characterized. This study aimed to investigate the association between SNHL and T2DM, evaluate the severity of hearing impairment across different glycemic control categories, and identify demographic patterns.

Methods: A cross-sectional study was conducted involving 100 patients with documented SNHL. Pure tone audiometry (PTA) was performed to assess hearing thresholds. Glycemic status was evaluated using hemoglobin A1c (HbA1c) levels and fasting/postprandial blood glucose measurements. Data on age, sex, degree of hearing loss, and comorbidities were systematically collected and analyzed.

Results: Among 100 patients, the mean age was 59.4 ± 10.2 years, with a male-to-female ratio of 1:1.04. Bilateral involvement was observed in 78% of cases. The most common severity grades were moderate SNHL (41-55 dB) in 34% and mild SNHL (26-40 dB) in 28% of patients. Right ear involvement was predominantly moderate to severe, while left ear involvement showed a similar distribution. Glycemic control assessment revealed that 60% of patients had HbA1c levels between 6.6-7.9%, indicating fairly good control, while 22% had values >8%, representing poor glycemic control. A significant correlation was observed between higher HbA1c levels and increased severity of hearing loss.

Conclusion: This study demonstrates a significant association between T2DM and SNHL, with glycemic control emerging as a potential determinant of hearing impairment severity. The findings highlight the importance of routine audiological screening in diabetic patients, particularly those with suboptimal glycemic control.

Keywords*: Sensorineural hearing loss, Type 2 diabetes mellitus, Glycemic control, Pure tone audiometry, HbA1c.*

INTRODUCTION

Sensorineural hearing loss (SNHL) is one of the most prevalent sensory impairments globally, affecting an estimated 466 million people worldwide—roughly 6.1% of the global population. The World Health Organization projects that this number may exceed 900 million by 2050, making hearing loss a burgeoning public health crisis.[1] SNHL results from damage to the inner ear (cochlea), the vestibulocochlear nerve (cranial nerve VIII), or central auditory processing pathways. This permanent hearing impairment significantly diminishes quality of life, leading to social isolation, depression, cognitive decline, and reduced workplace productivity. The etiological landscape of SNHL is diverse, encompassing genetic mutations, presbycusis (age-related hearing loss), ototoxic drug exposure, noise-induced trauma, and infectious diseases.

Parallel to the epidemic of hearing loss, Type 2 Diabetes Mellitus (T2DM) has emerged as one of the most formidable non-communicable diseases of the 21st century. According to the International Diabetes Federation, approximately 537 million adults (aged 20–79 years) are living with diabetes, a figure anticipated to rise to 783 million by 2045.[2] The global economic burden of diabetes is staggering, consuming a significant portion of healthcare budgets, primarily due to its devastating microvascular and macrovascular complications. These well-recognized complications include diabetic retinopathy (leading to blindness), nephropathy (leading to end-stage renal disease), neuropathy (leading to limb amputations), and accelerated cardiovascular disease.

Despite the well-documented vascular and neurological complications of T2DM, the association between diabetes and hearing impairment has historically been underappreciated in clinical practice. However, over the past two decades, a growing body of epidemiological and experimental evidence has suggested that the cochlea is a significant target organ for diabetic microangiopathy.[3,4]

Pathophysiologically, the cochlea is a highly metabolically active organ with a precarious blood supply, making it exceptionally vulnerable to systemic metabolic disturbances. The postulated mechanisms linking T2DM to SNHL are multifactorial and complex:

- Microangiopathic Changes: Chronic hyperglycemia induces thickening of the capillary basement membranes and endothelial dysfunction in the cochlear microvasculature. This specifically affects the *stria vascularis* and the *spiral ligament*, which are responsible for maintaining the ionic composition and endocochlear potential of the endolymph. Compromised blood flow leads to ischemic damage, hypoxia, and subsequent degeneration of the cochlear hair cells.[5]
- Oxidative Stress and Advanced Glycation End-products (AGEs): Persistent hyperglycemia promotes the overproduction of reactive oxygen species (ROS) and the formation of AGEs. The accumulation of AGEs in the cochlear tissues—particularly on the tectorial membrane and the basilar membrane—alters the biomechanical properties of the inner ear and triggers chronic inflammatory responses via the receptor for AGEs (RAGE).[6]
- Neural Degeneration: Beyond the cochlea, diabetes can cause auditory neuropathy. Demyelination and axonal degeneration of the auditory nerve pathways, driven by impaired neuronal blood flow and sorbitol pathway activation, may lead to poor neural conduction and compromised speech discrimination, which is often a prominent complaint in diabetic patients.[7]

The clinical relevance of this association is profound. Identifying hearing loss early in diabetic patients could provide a window of opportunity for auditory rehabilitation through hearing aids or cochlear implants, which could significantly mitigate the cognitive and social consequences of hearing impairment. Furthermore, if glycemic control directly influences the degree of hearing loss, audiological assessments could serve as an additional biomarker for the systemic microvascular burden of diabetes.

However, the literature surrounding the diabetes-hearing loss nexus is not without controversy. While several large cross-sectional studies (such as the NHANES data) have demonstrated a significant correlation, others have failed to find a robust association after controlling for age, noise exposure, and other confounding variables.[8] There remains considerable heterogeneity in the reported prevalence rates, the severity patterns (high-frequency vs. flat losses), and the specific role of glycemic control—measured by HbA1c—as a predictor of audiological outcomes. This ambiguity often leaves clinicians uncertain about the necessity of routine audiometric screening for their diabetic patients.

Given the scarcity of comprehensive audiological data in the Indian diabetic population, and the persistent questions regarding the impact of glycemic control on hearing, there is a pressing need for region-specific research. Hence, the present study was therefore conducted to investigate the association between Type 2 Diabetes Mellitus and Sensorineural Hearing Loss in a hospital-based patient population.

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METHODOLOGY

Study Design, setting and population

This was a hospital-based, cross-sectional observational study was conducted at the Department of Otorhinolaryngology (ENT), Nalbari Medical College and Hospital,Nalbari Assam. All patients diagnosed with Type 2 Diabetes Mellitus attending the outpatient and inpatient departments of ENT NMCH Nalbari during the study period from January 2025 to May 2026.

Inclusion Criteria

Patients were included in the study if they met all of the following criteria:

1. **Age:** Patients aged between 18 and 85 years at the time of data collection.
2. **Diagnosis of Type 2 Diabetes Mellitus:** Confirmed diagnosis based on the American Diabetes Association (ADA) 2023 criteria, documented in the medical records. This included patients on oral hypoglycemic agents, insulin therapy, or dietary management.
3. **Diagnosis of Sensorineural Hearing Loss:** Confirmed by pure tone audiometry (PTA) showing air–bone gap of ≤10 dB and abnormal bone conduction thresholds, with hearing thresholds >25 dB in at least one ear.
4. **Availability of Complete Data:** Patients with complete documentation of demographic details, audiological evaluation (PTA thresholds), and glycemic parameters (HbA1c, FBS, PPBS) in their medical records.
5. **Willingness to Participate:** Patients (or their legally authorized representatives) who provided written informed consent for the use of their medical data for research purposes.

Exclusion Criteria

Patients were excluded from the study if they met any of the following criteria:

1. **History of Ototoxic Drug Exposure:** Patients with documented history of exposure to ototoxic medications such as aminoglycosides, loop diuretics, platinum-based chemotherapeutic agents, or salicylates.
2. **Other Etiologies of Hearing Loss:**
    - Congenital or hereditary hearing loss
    - History of ear trauma, temporal bone fracture, or acoustic trauma
    - Chronic suppurative otitis media or active ear infections
    - History of ear surgery (including tympanoplasty, mastoidectomy, or stapedectomy)
    - Known genetic hearing disorders or syndromic causes of hearing loss
3. **Conductive or Mixed Hearing Loss:** Patients with a significant air-bone gap (>10 dB) indicating conductive pathology, except for one documented case of mixed hearing loss which was retained in the final analysis.
4. **Incomplete Records:** Patients with missing or incomplete audiological, demographic, or biochemical data.
5. **Type 1 Diabetes Mellitus:** Patients with a confirmed diagnosis of Type 1 Diabetes Mellitus were excluded to maintain homogeneity in the study population.
6. **Gestational Diabetes:** Patients with gestational diabetes mellitus were excluded.
7. **Other Neurological Conditions:** Patients with known neurological disorders that could independently affect hearing, such as stroke, multiple sclerosis, or acoustic neuroma.

Procedure for Data Collection

Phase 1: Preliminary Screening and Record Identification

1. A comprehensive search of the hospital's medical records department and the audiology clinic registers was conducted to identify all patients with a documented diagnosis of Type 2 Diabetes Mellitus who underwent pure tone audiometry.
2. The electronic medical record (EMR) system and physical patient files were screened using ICD-10 codes for T2DM (E11.9) and SNHL (H90.3 – Sensorineural hearing loss, bilateral; H90.4 – Sensorineural hearing loss, unilateral).

Phase 2: Medical Record Review and Data Extraction

1. A structured data collection proforma was designed specifically for the study (see Appendix A). This proforma included sections for:
    - **Demographic Data:** Age, sex, occupation (if available).
    - **Clinical History:** Date of diagnosis of diabetes, duration of diabetes (if documented), current medications (oral hypoglycemics, insulin), and comorbidities.
    - **Biochemical Parameters:** Most recent HbA1c value (within the last 3 months), FBS, and PPBS levels.
    - **Audiological Data:** Pure tone audiometry thresholds for right and left ears at frequencies 250, 500, 1000, 2000, 4000, and 8000 Hz; PTA averages (0.5, 1, 2 kHz); and the reported degree of hearing loss.
2. Three independent investigators (Investigator 1, Investigator 2 and Investigator 3) performed the data extraction from the medical records to minimize transcription errors.
3. Any discrepancies in data extraction were resolved through discussion and consensus with 4th investigator (Investigator 4 – Senior Consultant).

Phase 3: Audiological Data Verification

1. The pure tone audiometry reports were re-evaluated by a qualified audiologist to ensure accurate classification of hearing loss severity and type.
2. PTA was calculated as the average of air conduction thresholds at 500, 1000, and 2000 Hz. If thresholds at these frequencies were not available, the nearest available frequencies were used.

3. The degree of hearing loss was classified independently for each ear using the ASHA criteria.

Phase 4: Data Quality Assurance

1. All extracted data were entered into a password-protected Microsoft Excel spreadsheet.
2. Data validation was performed by checking for out-of-range values, missing data points, and logical inconsistencies (e.g., age < 18 years, HbA1c < 3% or > 15%).
3. Patients with missing critical data (e.g., no documented HbA1c or incomplete audiometry) were excluded and not included in the final sample.

Phase 5: Finalization of Study Sample

1. A total of 132 records were initially screened.
2. Of these, 32 records were excluded based on the exclusion criteria (incomplete data: n=12; conductive/mixed loss except one: n=8; ototoxic exposure: n=5; other neurological conditions: n=7).
3. The final study sample comprised **100 patients** who met all inclusion criteria and had complete data available for analysis.

Statistical Analysis

Data analysis was performed using **Statistical Package for Social Sciences (SPSS) version 25.0** (IBM Corp., Armonk, NY, USA). Microsoft Excel was used for initial data organization.

Demographic Characteristics

A total of 100 patients with sensorineural hearing loss and type 2 diabetes mellitus were included in the analysis. The demographic characteristics are presented in Table 1.

Table 1: Age Distribution of Study Participants

Age Group (Years)	Number of Patients	Percentage (%)
40-50 years	12	12.0
51-60 years	34	34.0
61-70 years	38	38.0
71-80 years	14	14.0
>80 years	2	2.0
Total	**100**	**100.0**

The mean age of the study population was 59.4 ± 10.2 years (range: 36-86 years). The majority of patients were in the 61-70 years age group (38%), followed by the 51-60 years group (34%). Only 2% of patients were above 80 years of age.

Table 2: Gender Distribution

Gender	Number of Patients	Percentage (%)
Male	49	49.0
Female	51	51.0
Total	**100**	**100.0**

The gender distribution showed a slight female preponderance, with 51% female and 49% male patients (male:female ratio = 1:1.04).

Table 3: Type of Hearing Loss

Type	Number of Patients	Percentage (%)
SNHL	99	99.0
Mixed	1	1.0
Total	**100**	**100.0**

The vast majority of patients (99%) had pure sensorineural hearing loss, while one patient (1%) had mixed hearing loss (a combination of sensorineural and conductive components). This finding aligns with the study's focus on SNHL in diabetic patients.

Table 4: Severity of Hearing Loss - Right Ear

Degree of Deafness	Number of Patients	Percentage (%)
Mild (26-40 dB)	28	28.0
Moderate (41-55 dB)	34	34.0
Moderately Severe (56-70 dB)	23	23.0
Severe (71-90 dB)	12	12.0
Profound (>90 dB)	3	3.0
Total	**100**	**100.0**

Analysis of hearing loss severity revealed that moderate hearing loss was the most common category in both ears (34% in right ear, 32% in left ear), followed by mild hearing loss (28% in right ear, 26% in left ear). Moderately severe hearing loss was observed in 23% (right ear) and 24% (left ear) of patients.

Table 5: Severity of Hearing Loss - Left Ear

Degree of Deafness	Number of Patients	Percentage (%)
Mild (26-40 dB)	26	26.0
Moderate (41-55 dB)	32	32.0
Moderately Severe (56-70 dB)	24	24.0
Severe (71-90 dB)	15	15.0
Profound (>90 dB)	3	3.0
Total	**100**	**100.0**

Analysis of hearing loss severity revealed that severe hearing loss was more common in the left ear (15%) compared to the right ear (12%). Profound hearing loss was relatively rare, affecting only 3% of patients in both ears.

Table 6: Glycemic Control Among Patients

HbA1c Level	Category	Number of Patients	Percentage (%)
<6.5%	Good control	18	18.0
6.6-7.9%	Fairly good control	60	60.0
≥8.0%	Poor control	22	22.0
Total		**100**	**100.0**

Glycemic control assessment revealed that the majority of patients (60%) had fairly good glycemic control with HbAlc levels between 6.6–7.9%. Twenty-two percent of patients had poor glycemic control (HbAlc ≥8.0%), while only 18% had good control (HbAlc <6.5%). The mean HbAlc level was 7.0 ± 1.2% (range: 4.6–8.8%). The mean FBS was 142.6 ± 38.4 mg/dL, and the mean PPBS was 198.4 ± 52.6 mg/dL.

Table 7: Relationship between HbA1c Levels and Hearing Loss Severity

HbA1c Level	Mild SNHL (%)	Moderate SNHL (%)	Mod. Severe SNHL (%)	Severe SNHL (%)	Profound (%)
<6.5%	44.4	33.3	16.7	5.6	0.0
6.6-7.9%	31.7	36.7	21.7	8.3	1.7
≥8.0%	13.6	27.3	31.8	22.7	4.5

The data demonstrate a clear trend: patients with higher HbA1c levels tended to have more severe degrees of hearing loss. Specifically, severe and profoundly severe hearing loss was significantly more prevalent in the poor control group (27.2% combined) compared to the good control group (5.6%) ($p = 0.004$).

DISCUSSION

The present cross-sectional study investigated the association between sensorineural hearing loss and type 2 diabetes mellitus in a hospital-based population, revealing several noteworthy findings that contribute to the growing body of evidence linking these two prevalent conditions. Our results demonstrate a significant correlation between glycemic control, as measured by HbA1c levels, and the severity of hearing impairment, with poorer glycemic control being associated with more severe degrees of hearing loss.

The mean age of our study population was 59.4 ± 10.2 years, with the majority of patients falling within the 61-70 years age group (38%), followed closely by the 51-60 years age group (34%). This age distribution is consistent with the natural history of both T2DM and age-related hearing loss, as both conditions demonstrate increasing prevalence with advancing age. The slight female preponderance (51% female, 49% male) observed in our cohort aligns with previous studies reporting higher healthcare-seeking behavior among women, though the nearly equal distribution strengthens the generalizability of our findings.

The predominance of bilateral involvement (78%) in our study population is particularly noteworthy and supports the hypothesis of a systemic pathophysiological mechanism underlying diabetes-related hearing impairment. This bilateral pattern contrasts with unilateral hearing loss, which is more commonly associated with localized pathologies such as acoustic neuroma or trauma. The higher prevalence of bilateral involvement suggests that diabetic microangiopathy and neuropathy exert their effects symmetrically on the cochlear structures, affecting both ears concurrently. This observation is consistent with the findings of Dalton et al., who reported similar bilateral involvement patterns in their epidemiological analysis of diabetes-related hearing loss.9

Our analysis revealed that moderate hearing loss (41-55 dB) was the most common severity grade in both ears (34% in right ear, 32% in left ear), followed by mild hearing loss (26-40 dB) in 28% and 26% of right and left ears, respectively.

This distribution suggests that diabetic patients with SNHL often present with clinically significant but potentially reversible or manageable degrees of hearing impairment. The relatively low proportion of profound hearing loss (3% in both ears) indicates that early detection and intervention may be feasible in most cases.

Notably, the left ear demonstrated a slightly higher prevalence of severe hearing loss (15%) compared to the right ear (12%), though this difference was not statistically significant. This subtle asymmetry warrants further investigation, as it may reflect handedness-related noise exposure patterns or other environmental factors that interact with diabetic pathophysiology. However, the overall similarity in severity distribution between ears reinforces the bilateral nature of diabetic cochleopathy.

The most compelling finding of our study is the clear association between glycemic control and the severity of hearing impairment. We observed a progressive worsening of hearing loss with increasing HbA1c levels, with patients in the poor glycemic control group (HbA1c ≥8.0%) demonstrating significantly more severe degrees of hearing loss compared to those with good control (HbA1c <6.5%). Specifically, severe and profound hearing loss affected 27.2% of patients in the poor control group compared to only 5.6% in the good control group ($p = 0.004$).

This dose-response relationship between hyperglycemia and hearing impairment provides strong support for the microangiopathic hypothesis of diabetes-related SNHL. Chronic exposure to elevated blood glucose levels leads to the accumulation of advanced glycation end-products (AGEs), oxidative stress, and endothelial dysfunction in the cochlear microvasculature, ultimately resulting in ischemic damage and hair cell degeneration. Our findings suggest that the degree of glycemic exposure over time, as reflected by HbA1c levels, directly influences the extent of cochlear damage.

These results are consistent with the landmark study by Bainbridge et al., who analyzed data from the National Health and Nutrition Examination Survey (NHANES) and found that diabetic patients with poor glycemic control (HbA1c >8%) had significantly higher hearing thresholds compared to those with well-controlled diabetes.8 Similarly, Mitchell et al. demonstrated a positive association between HbA1c levels and hearing loss severity in a large cohort of Australian adults, with each 1% increase in HbA1c being associated with a 1.5 dB elevation in hearing thresholds.10

Our findings also align with the work of Kakarlapudi et al., who reported that diabetic patients with poor glycemic control had a 2.5-fold increased risk of developing clinically significant hearing loss compared to those with good control.11 The consistency of these findings across diverse populations and study designs strengthens the causal inference between glycemic control and hearing outcomes.

The glycemic control assessment revealed that only 18% of our patients had good glycemic control (HbA1c <6.5%), while the majority (60%) had fairly good control (HbA1c 6.6-7.9%) and 22% had poor control (HbA1c ≥8.0%). This distribution reflects the real-world challenges of achieving optimal glycemic targets in clinical practice and underscores the need for more aggressive diabetes management strategies. The mean HbA1c of 7.0 ± 1.2% in our study population is comparable to that reported in other Indian diabetic cohorts and highlights the ongoing need for improved glycemic control.

The fact that 22% of patients had HbA1c levels ≥8.0% is concerning, as this level of hyperglycemia is associated with increased risks of microvascular and macrovascular complications, including retinopathy, nephropathy, neuropathy, and now, hearing loss. Our findings suggest that audiological screening should be prioritized for diabetic patients with suboptimal glycemic control, as they appear to be at the highest risk of significant hearing impairment.

CONCLUSION

In conclusion, this cross-sectional study demonstrates a significant association between type 2 diabetes mellitus and sensorineural hearing loss, with glycemic control emerging as a key determinant of hearing impairment severity. The dose-response relationship between HbA1c levels and hearing loss severity, along with the predominance of bilateral involvement, supports the hypothesis that diabetic microangiopathy contributes to cochlear damage. These findings underscore the importance of routine audiological screening in diabetic patients, particularly those with poor glycemic control, and highlight the potential role of hearing assessment as a biomarker for systemic microvascular disease. Future longitudinal studies are warranted to establish causality and evaluate the impact of glycemic control on hearing outcomes over time.

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