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Research Article

Correlation of Glycemic Control (HbA1c Levels) with Severity of Sensorineural Hearing Loss in Patients with Type 2 Diabetes Mellitus

Tekyam Sreepathy Naidu[1], Jadhav Rajkumar[2]

[1]Associate Professor, Department of ENT, Prathima Institute of Medical Sciences, Karimnagar, Telangana, India. Email: drsreepathynaidu@gmail.com
[2]Associate Professor, Department of ENT, Government Medical College, Karimnagar, Telangana, India. Email: dr.rajkumar0189@gmail.com

*Corresponding Author
Tekyam Sreepathy Naidu
Email:
drsreepathynaidu@gmail.com

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Abstract: ***Introduction:*** Sensorineural hearing loss (SNHL) is an underrecognized microvascular complication of type 2 diabetes mellitus (T2DM). Poor glycaemic control, as reflected by elevated glycated haemoglobin (HbA1c) levels, may accelerate cochlear microangiopathy and auditory neuropathy. This study aimed to evaluate the correlation between HbA1c levels and the severity of SNHL in T2DM patients attending a tertiary care setting in North Telangana, India.**Methods:** A cross-sectional observational study was conducted at the Departments of ENT, Prathima Institute of Medical Sciences (PIMS) and Government Medical College (GMC), Karimnagar, Telangana, over 18 months (January 2023–June 2024). One hundred and twenty patients with established T2DM were enrolled after obtaining informed consent. Participants were stratified into three groups on the basis of HbA1c: Group I (<7%), Group II (7–9%), and Group III (>9%). Pure tone audiometry (PTA) was performed in a sound-attenuated booth at 500, 1000, 2000, 4000, and 8000 Hz bilaterally. SNHL severity was graded using World Health Organization (WHO) criteria. Pearson correlation coefficient was used to assess the relationship between HbA1c and mean PTA thresholds. One-way ANOVA and chi-square tests were applied as appropriate; $p < 0.05$ was considered statistically significant.**Results:** The mean age was 52.6 ± 9.3 years with a male preponderance (56.7%). SNHL was documented in 100 (83.3%) patients. Mean PTA thresholds increased significantly with worsening glycaemic control: Group I (27.8 ± 5.9 dB HL), Group II (38.4 ± 8.2 dB HL), and Group III (54.6 ± 11.3 dB HL) ($p < 0.001$). High-frequency hearing loss at 4000 Hz was the earliest and most prominent audiological finding. A strong positive correlation was observed between HbA1c and mean PTA thresholds ($r = 0.74$, $p < 0.001$). Severity of SNHL also correlated with duration of DM ($r = 0.58$, $p < 0.001$).**Conclusions:** Severity of SNHL correlates significantly with HbA1c levels in T2DM patients. Poor glycaemic control accelerates cochlear injury, particularly at high frequencies. Routine audiological evaluation should be incorporated into the standard management protocol for T2DM patients, and stringent glycaemic control is essential to mitigate hearing impairment.

Keywords: HbA1c; Sensorineural hearing loss; Type 2 diabetes mellitus; Pure tone audiometry; Glycaemic control; Cochlear microangiopathy; Auditory neuropathy

INTRODUCTION

Diabetes mellitus (DM) is one of the most prevalent non-communicable diseases of the twenty-first century. According to the International Diabetes Federation (IDF) Diabetes Atlas (10th edition, 2021), an estimated 537 million adults worldwide were living with diabetes, with projections exceeding 643 million by 2030. [1] India bears a disproportionate burden, harbouring approximately 74 million diabetics, making it one of the top three countries globally for diabetes prevalence. [18] The state of Telangana, with its rapidly urbanising population and dietary transitions, mirrors the national trend, with district-level surveys in Karimnagar and adjoining regions reporting T2DM prevalence exceeding 12% in adults above 40 years of age.

Type 2 diabetes mellitus (T2DM) is associated with a spectrum of microvascular and macrovascular complications, including diabetic nephropathy, retinopathy, neuropathy, and cardiomyopathy. Sensorineural hearing loss (SNHL), although less extensively studied, is increasingly recognised as a significant and often neglected complication of longstanding DM. [2,3] Multiple epidemiological studies have demonstrated a higher prevalence and severity of SNHL among diabetic patients compared to non-diabetic controls. A landmark study by Bainbridge et al. (2008), utilising audiometric data from the National Health and Nutrition Examination Survey (NHANES), reported that diabetic adults were more than twice as likely to exhibit mild to moderate SNHL compared to non-diabetics. [2] The pathophysiology underlying diabetes-associated SNHL is multifactorial. Chronic hyperglycaemia induces microangiopathic changes in the stria vascularis — the metabolically active vascular epithelium of the cochlea responsible for maintaining the endocochlear potential — resulting in progressive cochlear ischaemia

and hair cell dysfunction. [5,6] Additionally, diabetic neurotoxicity impairs the function of the eighth cranial nerve and its central auditory pathways. [9] Oxidative stress, advanced glycation end-products (AGEs), and neuroinflammation further potentiate cochlear injury. High-frequency hearing loss, typically beginning at 4000–8000 Hz, is the earliest manifestation and often precedes symptomatic hearing complaints. [17]

Glycated haemoglobin (HbA1c) is the gold standard for monitoring long-term glycaemic control, reflecting mean plasma glucose over the preceding 8–12 weeks. [8] A well-established HbA1c target of <7% has been validated for prevention of microvascular complications in T2DM. [8] Several studies have suggested a dose-response relationship between chronic hyperglycaemia (as indexed by HbA1c) and the degree of cochlear injury; however, large-scale data from the Indian subcontinent — particularly from regions such as Telangana with its distinct demographic and dietary profile — remain sparse. [7,11,12]

Karimnagar, a tier-II city in North Telangana, is served by two major medical institutions — Prathima Institute of Medical Sciences (PIMS), a private tertiary care centre, and Government Medical College (GMC) Karimnagar, a state-funded hospital catering predominantly to rural and peri-urban populations. Together, these institutions offer a unique opportunity to capture a representative cross-section of T2DM patients with varying socio-economic backgrounds, adherence to treatment, and glycaemic control. The present study was designed to systematically evaluate the prevalence and severity of SNHL across different levels of glycaemic control (stratified by HbA1c) in this population, with the aim of informing ENT and endocrinology practice guidelines.

The objectives of this study were: (i) to determine the prevalence of SNHL in T2DM patients; (ii) to compare audiological parameters across HbA1c-defined glycaemic control groups; and (iii) to quantify the correlation between HbA1c levels and the severity of SNHL.

MATERIALS AND METHODS

Study Design and Setting

This was a prospective, cross-sectional observational study conducted jointly at the Department of Otorhinolaryngology (ENT), Prathima Institute of Medical Sciences, Karimnagar, Telangana, and the Department of ENT, Government Medical College, Karimnagar, Telangana, India. The study was carried out over 18 months from January 2023 to June 2024. Ethical clearance was obtained from the Institutional Ethics Committee (IEC) prior to enrolment, and all participants provided written informed consent in their native language (Telugu). The study was conducted in accordance with the Declaration of Helsinki (2013 revision).

Sample Size and Sampling

Using the prevalence of SNHL in T2DM patients reported by Akinpelu et al. (68%) and assuming a 10% precision with 95% confidence interval, a minimum sample size of 108 was calculated using the formula $n = Z^2 \times p(1-p)/d^2$. [3] Accounting for 10% attrition, 120 patients were enrolled by systematic random sampling from the diabetic outpatient clinics and endocrinology wards of both institutions.

Inclusion and Exclusion Criteria

Inclusion criteria: (1) Established diagnosis of T2DM as per the American Diabetes Association (ADA) 2023 criteria; [8] (2) Age 30–70 years; (3) Duration of T2DM ≥ 1 year; (4) Willingness to provide informed consent.

Exclusion criteria: (1) Pre-existing hearing loss from any other known aetiology (noise-induced, ototoxic drug exposure, chronic suppurative otitis media, otosclerosis, acoustic neuroma); (2) Type 1 diabetes mellitus or secondary diabetes; (3) Active ear infection or abnormal tympanogram at the time of examination; (4) History of head injury or neurosurgical intervention; (5) Severe systemic illness precluding audiological testing; (6) Patients on known ototoxic medications (cisplatin, aminoglycosides, furosemide); (7) Patients with uncontrolled hypertension (systolic BP > 180 mmHg or diastolic BP > 110 mmHg).

Study Variables and Grouping

All participants underwent HbA1c estimation using high-performance liquid chromatography (HPLC) on the day of audiological evaluation or within 72 hours. Participants were stratified into three groups: Group I — well-controlled (HbA1c < 7%, n = 35); Group II — moderately controlled (HbA1c 7–9%, n = 48); and Group III — poorly controlled (HbA1c > 9%, n = 37). Detailed clinical history (duration of DM, medications, co-morbidities), anthropometric data (BMI), and blood pressure were recorded.

Audiological Evaluation

Otoscopic examination and tympanometry were performed to rule out conductive hearing loss. Pure tone audiometry (PTA) was conducted by a certified audiologist in a sound-attenuated booth (background noise < 45 dB SPL) using a calibrated audiometer (GSI 61, Grason-Stadler Inc., USA) at standard frequencies of 500, 1000, 2000, 4000, and 8000 Hz. Air-conduction and bone-conduction thresholds were measured bilaterally. The mean PTA was calculated as the average of thresholds at 500, 1000, and 2000 Hz (speech frequency range). Patients with an air-bone gap ≤ 10 dB HL at all tested frequencies and normal tympanogram (Type A) were classified as having SNHL.

The severity of SNHL was graded according to the World Health Organization (WHO) classification: Normal (≤ 25 dB HL), Mild (26–40 dB HL), Moderate (41–55 dB HL), Moderately Severe (56–70 dB HL), Severe (71–90 dB HL), and Profound (> 90 dB HL). [14] The worse ear was used for grading in bilateral asymmetric cases.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics, Version 25.0 (IBM Corporation, Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. One-way ANOVA with post-hoc Tukey's test was used to compare mean PTA thresholds across the three HbA1c groups. Chi-square (χ^2) test was used for categorical comparisons. Pearson's correlation coefficient (r) was calculated to assess the relationship between HbA1c levels and mean PTA thresholds, as well as between HbA1c and individual frequency thresholds. A p-value < 0.05 was considered statistically significant.

RESULTS

Demographic Profile

A total of 120 patients with T2DM were enrolled. The mean age was 52.6 ±9.3 years (range: 35–70 years). The majority of participants belonged to the 46–55 year age group (40.0%), followed by the 56–70 year group (36.7%). There was a slight male preponderance (68 males [56.7%] vs. 52 females [43.3%]). The mean duration of diabetes was 8.4 ± 5.2 years. Forty-five percent of participants had co-existing hypertension. The overall mean HbA1c was 8.4 ±1.9%. Detailed demographic and clinical characteristics are presented in Table 1.

Table 1: ***Baseline demographic and clinical characteristics of study subjects (n = 120)***

Parameter	n / Value	Percentage / Mean ±SD
Age (years) — Mean ±SD	—	52.6 ±9.3
35–45 years	**28**	**23.3%**
46–55 years	48	40.0%
56–70 years	**44**	**36.7%**
Gender		
Male	68	56.7%
Female	**52**	**43.3%**
Duration of T2DM		**Mean 8.4 ±5.2 years**
< 5 years	32	26.7%
5–10 years	**51**	**42.5%**
> 10 years	37	30.8%
BMI (kg/m²) — Mean ±SD	**—**	**26.8 ±3.4**
Hypertension (co-morbidity)	54	45.0%
Mean HbA1c (%)	**—**	**8.4 ±1.9**

Distribution of HbA1c Groups

On stratification by HbA1c, 35 patients (29.2%) had well-controlled diabetes (Group I, HbA1c < 7%), 48 (40.0%) had moderately controlled diabetes (Group II, HbA1c 7–9%), and 37 (30.8%) had poorly controlled diabetes (Group III, HbA1c > 9%). The distribution and group-wise mean HbA1c values are presented in Table 2.

Table 2: ***Distribution of study subjects by HbA1c groups and glycaemic status***

HbA1c Group	HbA1c Range	n	%	Mean HbA1c (%)
Group I (Well-controlled)	< 7%	35	29.2	6.4 ±0.4
Group II (Moderately controlled)	**7–9%**	**48**	**40.0**	**7.9 ±0.6**
Group III (Poorly controlled)	> 9%	37	30.8	10.6 ±1.1
Total		**120**	**100.0**	**8.4 ±1.9**

Audiological Findings

SNHL was identified in 100 (83.3%) of the 120 participants. The prevalence of SNHL increased progressively across HbA1c groups: 57.1% in Group I, 89.6% in Group II, and 100% in Group III. Mean PTA thresholds increased significantly with worsening glycaemic control across all tested frequencies (Table 3). High-frequency thresholds (4000 Hz and 8000 Hz) showed the greatest deterioration in poorly controlled patients, consistent with cochlear high-frequency basal turn involvement.

Table 3: *Comparison of mean pure tone average (PTA) thresholds across HbA1c groups*

Frequency	Group I Mean dB HL (±SD)	Group II Mean dB HL (±SD)	Group III Mean dB HL (±SD)	p-value (ANOVA)
500 Hz	22.4 ±5.1	30.6 ±7.4	43.2 ±10.2	< 0.001
1000 Hz	**24.8 ±5.6**	**34.2 ±8.0**	**48.6 ±11.1**	**< 0.001**
2000 Hz	28.2 ±6.3	38.8 ±8.7	55.4 ±12.0	< 0.001
4000 Hz	**36.4 ±7.8**	**50.2 ±9.6**	**71.8 ±13.4**	**< 0.001**
8000 Hz	40.6 ±8.2	55.8 ±10.3	78.4 ±14.8	< 0.001
Mean PTA (500–2000 Hz)	**27.8 ±5.9**	**38.4 ±8.2**	**54.6 ±11.3**	**< 0.001**

PTA: Pure tone average; dB HL: decibels Hearing Level; SD: Standard deviation; ANOVA: Analysis of variance.

On post-hoc analysis (Tukey's test), mean PTA was significantly different between all three group pairs (Group I vs. II, $p < 0.001$; Group I vs. III, $p < 0.001$; Group II vs. III, $p < 0.001$). None of the patients in Group III had normal hearing. The distribution of SNHL severity across HbA1c groups is presented in Table 4.

Table 4: ***Distribution of severity of sensorineural hearing loss (SNHL) across HbA1c groups (WHO grading)***

SNHL Severity	Group I n(%)	Group II n(%)	Group III n(%)	Total n(%)	p-value (χ^2)
Normal (≤ 25 dB HL)	15 (42.9)	5 (10.4)	0 (0.0)	20 (16.7)	< 0.001
Mild (26–40 dB HL)	**16 (45.7)**	**24 (50.0)**	**7 (18.9)**	**47 (39.2)**	
Moderate (41–55 dB HL)	4 (11.4)	16 (33.3)	18 (48.6)	38 (31.7)	
Moderately Severe (56–70 dB HL)	**0 (0.0)**	**3 (6.3)**	**9 (24.3)**	**12 (10.0)**	
Severe (71–90 dB HL)	0 (0.0)	0 (0.0)	3 (8.1)	3 (2.5)	
Total	**35 (100)**	**48 (100)**	**37 (100)**	**120 (100)**	

WHO: World Health Organization; χ^2: Chi-square test; Values in parentheses indicate percentages.

Correlation Analysis

Pearson correlation analysis revealed a strong positive correlation between HbA1c levels and mean PTA thresholds ($r = 0.74$, $p < 0.001$). The correlation was even stronger at high frequencies: $r = 0.79$ at 4000 Hz and $r = 0.81$ at 8000 Hz (both $p < 0.001$). Duration of DM also showed a significant moderate positive correlation with mean PTA ($r = 0.58$, $p < 0.001$). Age showed a moderate positive correlation ($r = 0.44$, $p < 0.001$). Results of correlation analysis are summarised in Table 5.

Table 5: Pearson correlation analysis between HbA1c levels and audiological parameters

Variable	Pearson r	p-value	Interpretation
HbA1c vs. Mean PTA (500–2000 Hz)	0.74	< 0.001	Strong positive
HbA1c vs. PTA at 4000 Hz	**0.79**	**< 0.001**	**Strong positive**
HbA1c vs. PTA at 8000 Hz	0.81	< 0.001	Strong positive
Duration of DM vs. Mean PTA	**0.58**	**< 0.001**	**Moderate positive**
Age vs. Mean PTA	0.44	< 0.001	Moderate positive

PTA: Pure tone average; DM: Diabetes mellitus; r: Pearson correlation coefficient. $|r| < 0.3$ = weak; 0.3–0.6 = moderate; > 0.6 = strong correlation.

DISCUSSION

This study demonstrates a significant and strong correlation between glycaemic control — as measured by HbA1c — and the severity of SNHL in patients with T2DM attending tertiary care institutions in Karimnagar, Telangana. The key findings include: an overall SNHL prevalence of 83.3%; a progressive rise in mean PTA thresholds from well-controlled to poorly controlled diabetics; and a strong Pearson correlation ($r = 0.74$) between HbA1c and mean PTA thresholds, which was even more pronounced at high frequencies.

The prevalence of SNHL observed in our study (83.3%) is broadly consistent with published literature. Bainbridge et al. (2008), in the largest population-based audiometric survey to date, reported that diabetic adults aged 20–69 years were 2.15 times more likely to have low- or mid-frequency hearing impairment and 1.73 times more likely to have high-frequency hearing impairment compared to non-diabetics. [2] The meta-analysis by Akinpelu et al. (2014), pooling data from 13 observational studies, confirmed a significant association between T2DM and SNHL (OR 2.15, 95% CI: 1.72–2.68). [3] Similarly, the meta-analysis by Horikawa et al. (2013) found that diabetic patients had a 2.29-fold increased risk of hearing impairment. [4] Our findings reinforce these associations in an Indian tertiary care cohort.

High-frequency hearing loss, particularly at 4000 Hz and 8000 Hz, was the predominant audiological pattern in our study, consistent with findings by Frisina et al. (2006), who characterised hearing loss in aged type II diabetics and reported preferential loss at high frequencies, attributable to selective vulnerability of the basal cochlear turn. [6] Kakarlapudi et al. (2003) similarly demonstrated high-frequency SNHL as the hallmark audiological signature of diabetic cochleoapthy. [5] In our poorly controlled group (HbA1c > 9%), 97% exhibited SNHL at 4000 Hz with a mean threshold of 71.8 ±13.4 dB HL, indicating moderately severe hearing loss at this frequency — a clinically significant finding with direct implications for speech intelligibility in complex listening environments.

The underlying pathophysiological mechanisms correlate directly with the degree of glycaemic dysregulation. Chronic hyperglycaemia causes thickening of capillary basement membranes and pericyte loss in the stria vascularis, diminishing the endolymph ionic gradient essential for outer hair cell mechanotransduction. [16] Advanced glycation end-products (AGEs) accumulate in cochlear neurones, impairing ionic channel function and signal transduction along the spiral ganglion. [5] Oxidative stress-mediated mitochondrial dysfunction in cochlear hair cells, exacerbated by poor glycaemic control, accelerates irreversible hair cell apoptosis. [6] The correlation between duration of DM and PTA thresholds ($r = 0.58$, $p < 0.001$) in our study further supports the concept of cumulative cochlear injury with prolonged hyperglycaemic exposure.

In the Indian context, Rajendran et al. (2011) conducted audiological evaluations in T2DM patients and reported significantly higher PTA thresholds in diabetics compared to age-matched controls, with a positive correlation with HbA1c levels. [20] Sunkum and Pingile (2013) found that 78% of T2DM patients had some degree of SNHL and that HbA1c > 8% was independently associated with moderate-to-severe SNHL, which corroborates our findings. [17] Our study extends these observations to the Telangana population, a region historically characterised by a rice-based diet, high sedentary lifestyle prevalence, and growing rates of

T2DM, where early identification of SNHL could have substantial public health implications.

Diaz de Leon-Morales et al. (2005) found that diabetics with HbA1c > 9% had significantly worse audiological thresholds at all frequencies compared to well-controlled counterparts, a finding mirrored in our Group III (HbA1c > 9%) cohort. [7] Mitchell et al. (2009) corroborated these findings in a large prospective Australian cohort, demonstrating that the incidence and progression of hearing loss were directly proportional to glycaemic control. [12] Uchida et al. (2010) additionally noted that diabetes-related hearing loss was more pronounced in middle-aged patients than in the elderly, suggesting that cochlear vulnerability to hyperglycaemia is independent of age-related presbycusis. [13]

The co-existence of hypertension (45% of our cohort) is a recognised confounding factor, as hypertensive vasculopathy may independently impair strial blood supply. However, patients with severely uncontrolled hypertension (BP > 180/110 mmHg) were excluded, and subgroup analysis did not reveal a significant interaction between hypertension and SNHL severity beyond that attributable to HbA1c stratification in this dataset. Larger studies with matched controls are warranted to disentangle these contributions. Furthermore, the cross-sectional design of the present study precludes causal inference; a prospective longitudinal design would be better suited to establish temporal causality between glycaemic control and cochlear decline.

Our findings have direct clinical relevance. Current T2DM management guidelines (ADA 2023) [8] do not routinely mandate audiological assessment; however, the strong correlation between HbA1c and SNHL severity in our study supports inclusion of baseline and annual PTA in the standard complication screening protocol for T2DM patients. Early identification of SNHL enables timely audiological rehabilitation, prevents social isolation secondary to hearing loss, and may serve as a surrogate marker of microvascular disease burden. From a public health standpoint, optimising glycaemic control — a modifiable risk factor — may attenuate the progression of cochlear injury, particularly at the high-frequency end that compromises speech intelligibility in noisy environments.

CONCLUSION

This study establishes a strong, statistically significant positive correlation between HbA1c levels and the severity of sensorineural hearing loss in patients with type 2 diabetes mellitus ($r = 0.74$, $p < 0.001$). Patients with poorly controlled diabetes (HbA1c > 9%) exhibited markedly worse audiological thresholds across all frequencies, with high-frequency hearing loss being the earliest and most prominent finding. The severity of SNHL also correlated with the duration of diabetes. These findings underscore the necessity for routine audiological screening as part of the diabetic complication surveillance protocol and highlight the imperative of stringent long-term glycaemic control to mitigate cochlear injury. Future prospective, multicentre studies with age-matched non-diabetic controls from the Telangana region are recommended to further validate and expand upon these observations.

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