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Original Research Article

Prevalence and Association of Sensorineural Hearing Loss Among Diabetic Patients: A Community-Based Cross-Sectional Study in Jamuhar, Sasaram, Bihar

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Abstract:

Background: Type 2 diabetes mellitus (DM) is a chronic metabolic disorder affecting multiple systems, and growing evidence links it to sensorineural hearing loss (SNHL) through mechanisms such as cochlear microangiopathy and auditory neuropathy. Data from rural India remain scarce.

Objective: To estimate the prevalence of SNHL among diabetic patients in Jamuhar, Sasaram, Bihar, and assess its association with disease characteristics using audiological tests.

Methodology: A cross-sectional community-based study was performed at Narayan Medical College and Hospital, Jamuhar, Sasaram, Bihar on 100 subjects; 50 having type 2 DM and 50 matched on age and gender having non-diabetes. All participants underwent biochemical analysis and audiological tests: Pure Tone Audiometry (PTA), Distortion Product Otoacoustic Emissions (DPOAE), and Brainstem Evoked Response Audiometry (BERA). Statistical significance was set at $p < 0.05$.

Results: SNHL was more common in diabetics (84% vs. 62%) and was more severe as indicated by moderate to profound loss in 52% of the diabetic subjects versus 28% in non-diabetic subjects; DPOAE indicated abnormal cochlear function for the diabetic subjects in 80% of cases versus 56% for the controls, indicating clinically unevaluated cochlear function. BERA showed prolongation in the wave V and I–V interpeak latencies across all test intensities ($P < 0.001$), consistent with delay conduction along the auditory pathway. The diabetic subjects were older (mean age 52.3 years vs. 34.6 years) and had poorer glycemic control (mean HbA1c 8.1%).

Conclusion: Diabetes markedly increases the prevalence and degree of SNHL by disturbing the cochlear and retrocochlear pathways. Routine audiological screening and strict control of glycemic levels are recommended in order to mitigate the occurrence of auditory problems.

Keywords: Type 2 Diabetes Mellitus, Sensorineural Hearing Loss, Pure Tone Audiometry, DPOAE, BERA, Rural Health.

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Introduction

Type 2 diabetes mellitus (DM) is a chronic and metabolic disorder identifiable by persistent hyperglycemia (state of higher-than-normal blood glucose) due to relative insufficiency of insulin secretion, insulin resistance to action or both. Common antecedents of type 2 DM consist of lifestyle antecedents of obesity, physical inactivity and poor dietary intake and genetic susceptibility increases the risk [1]. Over the past few decades, type 2 DM has been important in public health because we estimated there were 392 million people living with type 2 diabetes in 2015 and they would have comprised almost 90% of diabetes and almost 6% of the global population [2]. The prevalence of disease is not homogenous - women and some ethnic groups including South Asians and Pacific Islanders, Latinos and Native Americans are disproportionately affected for a multifactorial reason of soco-cultural and biological [3].

The systemic manifestations of diabetes are well understood while the cardiovascular, renal, neurological, and ocular manifestations are well reviewed. The auditory system has increasingly become a focal area as one more location of diabetic microvascular and neuropathic associations. Diabetes is recognized to cause eighth cranial nerve (vestibulocochlear nerve) neuropathy and hence the microangiopathy of the cochlear vasculature is responsible for development of gradual hearing loss [5]. These pathological processes usually present as sensorineural hearing loss (SNHL) which makes up nearly 90% of diabetic auditory loss. SNHL is hearing loss secondary to cochlear or retroocular vestibulocochlear nerve auditory system dysfunction [6].

Studies have indicated that compared to non-diabetic patients, diabetics are more likely to experience sensor neural hearing loss (SNHL) and the severity of hearing loss is proportional to the severity and duration of diabetes. These considerations make the SNHL associated with diabetes classified as a low-profile complication of diabetes, of high clinical importance due to the expected significant burden of hearing loss on the patient's communicative and social function and quality of life. This burden is especially concerning in older people where both diabetes and hearing loss are common and contribute to double disability.

Appropriate diagnosis of SNHL is absolutely essential for early detection and management. Pure-tone audiometry (PTA) remains the state-of-the-art measurement for threshold of hearing as it allows not only to quantify the severity of the hearing, the type of loss, and pattern of impairment is [7]. Aural audiometry and tympanometry provide supplementary diagnostic support. Recently, with oto-acoustic emission (DPOAE) and brain-evoked response audiometry (BERA), there are valuable tools to diagnose subclinic hearing impairment. DPOAE is a non-invasive and reliable tool and is helpful for revealing early cochlear damage while BERA supports the differential use of the central and retro cochlear auditory pathways.

Results of the electrophysiological tests have also solidified the association of diabetes with hearing impairment. Delayed wave V latencies on BERA tracings are indicative of retro cochlear and central nervous system disorders, and higher interpeak latencies (wave I–V) point out latency of auditory conduction at the midbrain and brainstem levels [8]. This evidence highlights the requirement of complete auditory assessment—with PTA, DPOAE, and BERA—as a part of the complete management of diabetes patients.

Despite the growing global evidence, research on type 2 diabetes mellitus and its association with hearing loss is limited in low resource situations, particularly in rural India. Bihar is one of the most populous Indian states and has the double burden of communicable and non-communicable diseases where diabetes is a very concerning one. In rural areas such as Jamuhar of Sasaram, there is limited access to facilities and there is a lack of awareness and late diagnosis of chronic disorders such as diabetes and its sequelae. In this context, prevalence and pattern of noise-induced hearing loss (SNHL) in patients with diabetes is crucial for the right planning of preventive and screening actions.

The cross-sectional community survey of Jamuhar, Sasaram, was hence designed to compare the prevalence of SNHL between type 2 DM patients and matched non-diabetic patients by sex and age, evaluate the relationship of hearing threshold and diabetes duration, and correlate the findings of DPOAE and BERA. By these objectives, the investigation attempts to provide integrated perceptions of the burden, relationships, and diagnostic characteristics of SNHL among diabetic patients, and therefore contribute toward improved clinical practice and programmed planning at the community level among analogous rural communities.

Methodology

Study Design: This was a community-based analytical cross-sectional study conducted to assess the prevalence of sensorineural hearing loss (SNHL) among diabetic patients and its association with diabetes mellitus.

Study Area: The study was carried out in the Department of Otorhinolaryngology, Narayan Medical College and Hospital, Jamuhar, Sasaram, Bihar, India.

Study Duration: The study was conducted over a period of one year.

Study Population: The study population consisted of two groups: patients diagnosed with type 2 diabetes mellitus attending the diabetic clinic in the Endocrinology outpatient department and a control group of age- and sex-matched non-diabetic individuals from the community.

Sample Size: A total of 100 participants were recruited, comprising 50 patients with biochemically confirmed diabetes mellitus and 50 non-diabetic controls. The sample size was determined to ensure adequate statistical power to compare the prevalence of sensorineural hearing loss between the two groups.

Inclusion Criteria

- Patients aged ≤ 50 years of either gender.
- Biochemically confirmed diabetic patients (for the study group).
- Non-diabetic, apparently healthy individuals (for the control group).

Exclusion Criteria

- Patients with any other systemic illness or metabolic disorder.
- Patients with gestational diabetes.
- History of use of ototoxic drugs.
- Previous history of ear surgery or occupational noise exposure.

- Patients with chronic suppurative otitis media (CSOM), conductive hearing loss (CHL), or noise-induced hearing loss.
- Patients with congenital sensorineural hearing loss.

Data Collection: The data was collected by a systematic method involving demographic profiling, clinical history, biochemical test and audiological testing which involved otorhinolaryngological examination to exclude the profusion of any trouble with middle or external ear pathology. The demographic particulars of all subjects included age and sex and occupation, for diabetic patients the additional particulars included duration of diabetes, whether on treatment or not and glycemic control. The biochemical tests performed included fasting blood sugar, post-prandial blood sugar and HbA1c to confirm diabetic status and degree of control over diabetes. The non-diabetic subjects had fasting blood glucose and post-prandial blood glucose estimated to confirm their diabetic control status. Otorhinolaryngological examination was carried out on all subjects as part of clinical history.

Procedure: Participants who agreed to participation after providing written informed consent were assessed for inclusion in the study following the protocol. A full medical history was obtained to exclude potential confounds such as systemic disease, ototoxic medication, a previous history of ear surgery, or occupational noise exposure. Following this, general and otorhinolaryngological examinations were performed. Audiological evaluation was then performed in all participants by a certified audiologist, using pure tone audiometry (PTA) in a soundproof booth, followed by bilateral brainstem evoked response audiometry (BERA) to evaluate the auditory pathway, and distortion product otoacoustic emissions (DPOAE) to evaluate cochlear outer hair cell. In the diabetic participants, audiological results were then correlated with biochemical parameters inclusive of diabetes duration and glycemic control. All data was recorded in a systematic manner in a predesigned proforma.

Statistical Analysis: The data collected were enter and managed with Microsoft Excel. Continuous variables (age, hearing thresholds) were summarized as mean ± standard deviation, while categorical variables (presence/absence of hearing loss) were reported as frequencies and percentages. Comparisons between diabetic and control groups were assessed using student's t-test for continuous variables and Chi-square test for categorical variables. A p-value of less than 0.05 was considered statistically significant.

Result

Table 1 illustrates the gender breakdown of the participants in this study. Out of the sample of 100 individuals, there were 31 (31.0%) males and 69 (69.0%) females. Within the diabetic group (n=50), males made up 20 (40.0%) and females 30 (60.0%). In the non-diabetic group (n=50), 11 (22.0%) were male and 39 (78.0%) were female. This shows that within the study, females made up the majority in both the diabetic and non-diabetic groups, but there were a higher percentage of males represented in the diabetic group compared with the non-diabetic group.

Table 1: Gender distribution

Gender	Overall N (%)	Diabetic N (%)	Non-diabetic N (%)
Male	31 (31.0%)	20 (40.0%)	11 (22.0%)
Female	69 (69.0%)	30 (60.0%)	39 (78.0%)
Total	100 (100.0%)	50 (100.0%)	50 (100.0%)

Table 2 shows the demographic information for the subjects in this study. The diabetic group exhibited a significantly higher average age of 52.3 ± 8.2 years (ranging from 32 to 74 years) compared to 34.6 ± 8.9 years (ranging from 18 to 56 years) in the non-diabetic group, which signifies the higher incidence of diabetes among older people. The average fasting blood sugar levels were elevated in diabetics at 145.2 ± 30.4 mg/dL (ranging from 90 to a maximum of 260 mg/dL) relative to non-diabetics, who had average glucose levels of 95.4 ± 8.1 mg/dL (range from 70 to 110 mg/dL). The mean blood sugar level post-prandial was also significantly higher for diabetics at 190.6 ± 45.1 mg/dL (range from 110 to a maximum of 360 mg/dL) than that of the non-diabetic participants, which were 115.2 ± 12.3 mg/dL (range from 90 to 140 mg/dl). According to the glycemic control marker of HbA1c, diabetic subjects had elevated HbA1c levels at 8.10 ± 1.20% (range from 6.0 to 12.0%) compared to the reported HbA1c levels of non-diabetic participants at 5.40 ± 0.30% (range from 4.8 to 5.9%) which suggest that long-term glycemic control was poor for the diabetic cohort in this study.

Table 2: Demographic details

Parameter	Diabetic group	Non-diabetic group
Mean age (years)	52.3 ± 8.2 (Range 32–74)	34.6 ± 8.9 (Range 18–56)
Mean fasting blood sugar (mg/dL)	145.2 ± 30.4 (Range 90–260)	95.4 ± 8.1 (Range 70–110)
Mean post-prandial blood sugar (mg/dL)	190.6 ± 45.1 (Range 110–360)	115.2 ± 12.3 (Range 90–140)
Mean HbA1c (%)	8.10 ± 1.20 (Range 6.0–12.0)	5.40 ± 0.30 (Range 4.8–5.9)

Table 3 shows how hearing loss was distributed among the study population according to PTA findings. The diabetic group had 8 (16.0%) individuals with normal hearing, while the non-diabetic group had 19 (38.0%) individuals with normal hearing. These findings show that hearing loss is more common in diabetics than non-diabetics. A total of 6 (12.0%) in the diabetic group and 9 (18.0%) in the non-diabetic group had minimal SNHL. A total of 10 (20.0%) in the diabetic group and 8 (16.0%) in the non-diabetic group had mild SNHL. The number of individuals with moderate SNHL was a substantially different number, 14 (28.0%) in the diabetic group, and 9 (18.0%) in the non-diabetic group. Regarding severe hearing loss, there were 6 (12.0%) with severe hearing loss in diabetic individuals and 3 (6.0%) in non-diabetic individuals. A total of 6 (12.0%) had profound SNHL in diabetic individuals compared to only 2 (4.0%) in non-diabetic individuals. Overall, the 100 participants in this study 27 (27.0%) had normal hearing, and 73 (73.0%) had some degree of sensorineural hearing loss, and there is a disproportionate amount of diabetics with moderate to profound hearing impairment.

Table 3: Grade of hearing loss in study participants

PTA grade	Diabetic N (%)	Non-diabetic N (%)	Overall N (%)
Normal	8 (16.0%)	19 (38.0%)	27 (27.0%)
Minimal SNHL	6 (12.0%)	9 (18.0%)	15 (15.0%)
Mild SNHL	10 (20.0%)	8 (16.0%)	18 (18.0%)
Moderate SNHL	14 (28.0%)	9 (18.0%)	23 (23.0%)
Severe SNHL	6 (12.0%)	3 (6.0%)	9 (9.0%)
Profound SNHL	6 (12.0%)	2 (4.0%)	8 (8.0%)
Total	**50 (100.0%)**	**50 (100.0%)**	**100 (100.0%)**

Table 4 shows the distribution of participants according to DPOAE results, highlighting a clear difference between diabetic and non-diabetic groups. Among the diabetics, a large majority, 40 (80.0%), fell into the “Refer” category, suggesting abnormal cochlear function, while only 10 (20.0%) achieved a “Pass.” In contrast, the non-diabetic group showed better outcomes, with 28 (56.0%) in the “Refer” category and 22 (44.0%) in the “Pass” group. Overall, out of 100 participants, 68 (68.0%) were categorized as “Refer” and 32 (32.0%) as “Pass.” These findings indicate that diabetic patients are more likely to exhibit impaired outer hair cell function compared to non-diabetic individuals, reflecting a higher risk of cochlear involvement in this group.

Table 4: Distribution of study patients according to DPOAE

DPOAE	Diabetic N (%)	Non-diabetic N (%)	Overall N (%)
Refer	40 (80.0%)	28 (56.0%)	68 (68.0%)
Pass	10 (20.0%)	22 (44.0%)	32 (32.0%)
Total	**50 (100.0%)**	**50 (100.0%)**	**100 (100.0%)**

Table 5 presents the comparative BERA results between diabetic and non-diabetic groups, showing significantly prolonged wave latencies in diabetics at all tested intensities. At 70 dBnHL, wave V latency was 6.56 ± 0.52 ms in diabetics compared to 6.01 ± 0.27 ms in non-diabetics ($p < 0.001$), and I–V interpeak latency was 3.81 ± 0.26 ms versus 3.54 ± 0.08 ms, respectively ($p < 0.001$). At 80 dBnHL, wave V latency in diabetics was 6.51 ± 0.46 ms compared to 5.67 ± 0.22 ms in non-diabetics ($p = 0.004$), with I–V latency also longer in diabetics (4.78 ± 0.43 ms) than non-diabetics (4.11 ± 0.13 ms, $p = 0.035$). Similarly, at 90 dBnHL, wave V latency was 6.57 ± 0.48 ms in diabetics compared to 5.32 ± 0.20 ms in non-diabetics ($p < 0.001$), while I–V latency was 5.58 ± 0.61 ms versus 4.87 ± 0.25 ms ($p < 0.001$). These results indicate that diabetic patients exhibit delayed neural conduction in the auditory pathway, suggesting both peripheral and central auditory dysfunction compared to non-diabetic individuals.

Table 5: Comparative BERA results of diabetic and non-diabetic groups

Wave latency	Intensity (dBnHL)	Non-diabetic Mean ± SD (ms)	Diabetic Mean ± SD (ms)	P value
V	70	6.01 ± 0.27	6.56 ± 0.52	<0.001
I–V	70	3.54 ± 0.08	3.81 ± 0.26	<0.001
V	80	5.67 ± 0.22	6.51 ± 0.46	0.004
I–V	80	4.11 ± 0.13	4.78 ± 0.43	0.035
V	90	5.32 ± 0.20	6.57 ± 0.48	<0.001
I–V	90	4.87 ± 0.25	5.58 ± 0.61	<0.001

Discussion

The study population was comprised of 100 patients, of which only 16% of the diabetic patients had normal hearing, and the majority presented with varying levels of SNHL ranging from minimal (12.0%) to profound (12.0%). This trend is indicative of a high burden of hearing impairment within the diabetic population. Similar results were shown by Rajendran et al. (2011) [9], who observed that 73.3% of diabetes patients had SNHL when contrasted with only 6.7% of non-diabetic controls and therefore significantly supported diabetes-induced auditory impairment. Likewise, Tiwari and Mudhol (2018) [10] recorded that 76.8% of type 2 diabetes patients had SNHL and therefore added weight to the high prevalence recorded within our group. Dadhich et al. (2018) [11] further indicated that 73% of diabetics had hearing impairment and therefore was comparable with our observations of higher SNHL burden within diabetics. These are collectively additives and strengthen the evidence that diabetes has a high likelihood of inducing hearing loss.

Despite that, the distribution of severity of our study was slightly different from some previous reports. Although moderate hearing loss was the most prevalent finding in our diabetic patients (28.0%), Ferreira et al. (2016) [12] found that minimal hearing loss was the majority, followed by mild impairment of hearing, with 30% of patients preserving normal hearing. This may be due to differences in the age profiles of the study populations, glycemic control or diabetes duration. Our diabetics' mean age was 52.3 years and significantly higher when compared with the non-diabetic control group, whereas Ferreira only studied patients younger than 50 years. Therefore, our older group may have greater susceptibility to cumulative metabolic and vascular insults and hence higher severity of SNHL.

The contribution of poor glycemic control is also underscored by our results, where the average HbA1c of diabetics was 8.10%, significantly higher than the non-diabetic norm of 5.40%. Poor glycemic control has been found reliably related to auditory impairment in earlier work. Lerman-Garber et al. (2012) [13] found patients with early type 2 diabetes mellitus more likely to have SNHL, with higher HbA1c related to poorer thresholds. Likewise, Sunkum and Pingile (2013) [14] found 82% prevalence of SNHL in diabetics and blamed long-standing hyperglycemia inducing cochlear vascular changes. This is similar to our results where significant cochlear impairment was shown on Distortion Product Otoacoustic Emissions (DPOAE) test in 80% of diabetics.

Our DPOAE results revealed a higher proportion of “refer” outcomes in diabetics (80.0%) compared with non-diabetics (56.0%). This indicates subclinical cochlear dysfunction even in patients without clinically significant hearing loss. Similar patterns were observed by Ferreira et al. (2016) [12], who reported absent otoacoustic emissions in 78.9% of diabetic patients, and by Prabhu and Shanthala (2016) [15], who found absent OAEs in 68% of type 2 diabetics. These parallels strongly suggest that outer hair cell dysfunction is an early manifestation of diabetic auditory impairment, detectable even before overt SNHL develops on pure tone audiometry. Thus, our findings contribute to the growing body of evidence advocating for the inclusion of DPOAE testing in routine audiological evaluation of diabetics.

In addition to cochlear impairment, our study demonstrated significant abnormalities in retro cochlear auditory processing, as evidenced by delayed Brainstem Evoked Response Audiometry (BERA) latencies across all tested intensities. At 70 dBnHL, wave V latency was prolonged to 6.56 ms in diabetics compared with 6.01 ms in non-diabetics, while the I–V interpeak latency was significantly longer at 3.81 ms versus 3.54 ms. The delays became even more pronounced at higher intensities (80 and 90 dBnHL). These findings reflect impaired neural conduction and brainstem transmission, likely resulting from diabetic neuropathy. Comparable results were reported by Joshi et al. (2017) [16], who observed significantly prolonged BERA latencies for waves II and V, along with interpeak delays in diabetic patients. Batham et al. (2017) [17] and Suresh et al. (2018) [18] also found statistically significant differences in wave latencies and interpeak intervals between diabetics and non-diabetics, with abnormalities increasing with disease duration and severity of hyperglycemia. Singh et al. (2019) [19] further confirmed a progressive rise in wave V latency among diabetics, consistent with our observations of delayed auditory neural transmission.

Despite the prevalence of the majority of the studies, including ours, indicating a strong positive association of diabetes and SNHL, variations do exist at prevalence and severity levels. For instance, Ashish et al.'s study of Srinagar found lower prevalence of moderate-to-profound SNHL when they were compared with our population and proposed that geographical differences, genetic preponderances, and lifestyle factors may contribute to the manifestation of the disease. Variability may also arise because of differing diagnostic work-up, e.g., inclusion of younger-age-group patients or exclusion of long-standing diabetics. Nevertheless, the overall trend holds steady: diabetes significantly predisposes patients toward cochlear and retro cochlear hearing loss.

From both our findings and those of previous research, it is clear that diabetes mellitus appears to exert deleterious impacts on both central and peripheral auditory pathways. The strong agreement with Indian and international work points to the need for heightened vigilance related to hearing complications in the diabetes patient. Standard audiological assessments, including objective measures such as DPOAE and BERA, should be incorporated into the general practice of all diabetes care, particularly amongst those with poor glycemic control and/or long duration of the disease. We also need large, longitudinal studies that are specifically designed to help better clarify causal pathways, prevention strategies, and the potential role of glycemic control as a prevention measure for hearing loss.

Conclusion

The current cross-sectional community-based study from Jamuhar, Sasaram, Bihar, is remarkable in demonstrating a definitive link between diabetes and sensorineural hearing loss. Results suggest that diabetics are significantly more likely to have hearing impairment of varying degrees when compared with non-diabetic controls, and the majority of diabetics have moderate to profound sensorineural hearing loss. Objective measures such as DPOAE and BERA support this association and show cochlear damage and prolonged conduction times along the auditory pathways of diabetics, which indicate auditory function compromise at both the peripheral and central levels. Participant demographics highlight the older age and poorer glycemic controls of diabetics and suggest possible additional effects of age and glycemic control on hearing loss. Overall, the study establishes diabetes as a strong risk factor for hearing loss, underscores the important role of early screening, repeat audiology assessments, and strict glycemic control in the complete management of diabetes to assist in the preservation of auditory function and the improved quality of life of diabetic populations.

Refreences

1. Anjana RM, Ali MK, Pradeepa R, Deepa M, Datta M, Unnikrishnan R, et al. The need for obtaining accurate nationwide estimates of diabetes prevalence in India Rationale for a national study on diabetes. Indian J Med Res. 2011;133:369 80.
2. Diabetes blue circle symbol. International Diabetes Federation. Available at: https://www.idf.org/whowe-are/about-idf/logo.html. Accessed on 20 October 2021.
3. Melmed S, Polonsky KS, Larsen P. Williams textbook of endocrinology. 12th ed. Philadelphia: Elsevier/Saunders; 2005:1371-35.
4. Roeser RJ. Roeser's audiology desk reference. 2nd ed. New York: Thieme; 2005;415-9.
5. Jorgensen MB. The inner ear in diabetes mellitus. Arch Otolaryngol. 1961;74:373-81.
6. Newman CW, Weinstein BE, Jacobson GP, Hug GA. Test-retest reliability of the hearing handicap inventory for adults. Ear Hearing. 1991;12 (5):355-7.
7. Sindhusake D, Mitchell P, Smith W, Golding M, Newall P, Hartley D, et al. Validation of self-reported hearing loss. Int J Epidemiol. 2001;30:1371-78.
8. Virtanierni J, Laakso M, Nuutinen J, Karjalainens. Scott Brown's otolaryngology. 6th ed. London: Butterworths; 1997:3;145-9.
9. Rajendran S, Anandhalakshmi, Mythili B, Viswanatha R. Evaluation of the incidence of sensorineural hearing loss in patients with type 2 diabetes mellitus. Int J Biol Med Res. 2011;2(4):982- 7.
10. Tiwari A, Mudhol RS. Prevalence of sensorineural hearing loss among type-II diabetes mellitus patients attending KLES Dr. Prabhakar Kore Hospital and MRC: A cross-sectional study. Indian J Health Sci Biomed Res. 2018;11:165-9.
11. Dadhich S, Jha SG, Sinha V, Samanth TU. A prospective, observational study of incidence of sensory neural hearing loss in diabetes mellitus patients. Indian J Otol. 2018;24(2):80-2.
12. Ferreira JM, Fontenele-e-Silva Câmara M, César de Almeida P, Brandão Neto J, Antonio Bruno da Silva C. Audiologic characteristics of patients with diabetes mellitus type 2. Rev. 2016;18:5.
13. Lerman-Garber I, Cuevas-Ramos D, Valdés S, Enríquez L, Lobato M, Osornio M. Sensorineural hearing loss a common finding in early-onset type 2 diabetes mellitus. Endocr Pract. 2012;18(4):549-57.
14. Sunkum AJK, Pingile S. A clinical study of audiological profile in diabetes mellitus patients. Eur Arch Otorhinolaryngol. 2013;270(3):875-9.
15. Prabhu P, Shanthala SP. Efferent Auditory System Functioning and Speech Perception in Noise in Individuals with Type II Diabetes Mellitus. J Phonet Audiol. 2016;2:1-5.
16. Joshi KD, Galagali JR, Kanzhuly MK, Singh ID. A comparative study on effects of diabetes on auditory functions as measured by BERA and DPOAE. Int J Otorhinolaryngol Head Neck Surg. 2017;3:347-53.
17. Batham C, Choudhary AK, Yousuf PS. Brainstem auditory evoked responses with duration of type-II diabetes mellitus. Ann Med Health Sci Res. 2017;7:40-5.
18. Suresh S, Ramlan S, Somayaji G, Sequeira N. Brainstem auditory responses in type-2 diabetes mellitus. Int J Oto Head Neck Surg. 2018;4(2):45-9.
19. Singh V, Agrawal U, Chaudhary AK, Ranjan M. Study of variation and latency of wave v of brain stem evoked response audiometry in north central India. Indian J Otolaryngol Head Neck Surg. 2019; 71(2): 1408-11.