# Auditory dysfunction in patients with type 2 diabetes mellitus with poor versus good glycemic control

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**Background**
Auditory functions among patients with type 2 diabetes mellitus (DM) are controversial regarding cochlear or neural changes and the relationship between these changes and serum level of glycosylated hemoglobin ($HbA_{1c}$%).

**Aim of the study**
The aim of the study was to investigate auditory dysfunctions in type 2 DM patients with poor versus good glycemic control.

**Materials and methods**
The present study was conducted on three groups: two diabetic groups with poor and good glycemic control ($n$ = 18 and 14, respectively) based on serum $HbA_{1c}$% and one healthy control group ($n$ = 30) matched with age, sex, and BMI. All participants were subjected to clinical assessment, audiometry, brainstem auditory evoked potential (BAEP), and evoked acoustic emissions transient evoked otoacoustic emissions and distortion product otoacoustic emissions (TEOAEs and DPOAEs).

**Results**
Diabetic patients with poor glycemic control had significantly elevated hearing thresholds compared with other groups at low and high frequencies in audiometry ($P$ < 0.01 and $P$ < 0.001). They showed significantly prolonged absolute latency in wave I and interpeak latency (III–V) in the BAEP test compared with other groups ($P$ < 0.001). DM patients with poor glycemic control had significantly low amplitudes at all frequencies in the TEOAE test, as well as at high frequencies (4 and 6 kHz) on the DPOAE test, compared with other groups ($P$ < 0.001 and $P$ < 0.05, respectively). There were significant correlations between $HbA_{1c}$% and interpeak latency III–V ($r$ = 0.340, $P$ = 0.004) on the one hand and overall response of TEOAE amplitude ($r$ = –0.471; $P$ = 0.000) on the other.

**Conclusion**
Diabetic patients with poor glycemic control had worse auditory dysfunctions on both cochlear and neural findings.

**Keywords:**
auditory dysfunction, brainstem auditory evoked potential, glycemic control, hearing loss, otoacoustic emissions

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## Introduction

Diabetes mellitus (DM) is a metabolic disorder that can cause a variety of metabolic, neurological, and vascular complications [1]. Despite a correlation between DM and hearing loss being shown in previous studies [2,3], there is still no consensus about the exact etiopathogenesis of hearing loss and the site of auditory system involvement [3]. Thus, some authors have mentioned DM as a possible cause for sudden hearing loss. Despite other causative factors such as atherosclerosis and viral infection, DM is seen as the main cause of deafness due to diabetic microangiopathy [4].

However, the exact etiopathogensis is controversial in the literature, because most of the time DM involves older patients, thus making it even harder to connect hearing loss to DM or due to presbycusis [5]. Alternatively, others suggested that it could be related to injuries at various points of the auditory pathways, possibly due to neural or cochlear defects, as reported in a previous study [6]. However, Friedman *et al.* [6] indicate that the hearing loss patterns found in patients do not comply with senility-related hearing loss in terms of frequency distribution. Also in a recent study, Ferreira *et al.* [7] found a high frequency of sensorineural hearing loss (SNHL) in 37.5% of the patient population. However, these results were not compared against a control group [7].

Although auditory alterations are not a typical symptom, they can occur in patients with type 1 DM. These alterations are normally related to structures of the inner ear (cochlea), including the organ of Corti and structures of the central auditory pathway, from the nerve until the auditory cortex [8]. The audiogram is only capable of detecting alterations occurring from injury to the inner hair cells and/or alterations in the central auditory pathway. However, auditory alterations resulting from a dysfunction — that is, subclinical alterations – can be detected only through electrophysiological auditory tests including the otoacoustic emissions (OAEs) and auditory brainstem response (ABR) tests. These two tests are also capable of determining the location of the injury, with OAE distinguishing the sensorial alterations and ABR distinguishing the neural alterations [9].

### Aim

This comparative study aimed to investigate the auditory system functions of these type 2 DM patients with poor versus good glycemic control and compare them with an age-matched and sex-matched healthy control group. The study also aimed to investigate the relationship between serum $HbA_1c$% and auditory function parameters [brainstem auditory evoked potential (BAEP) and OAE].

## Materials and methods

Thirty-two type 2 DM patients (64 ears) (14 men and 18 women) were recruited from the outpatient clinic of the general internal Medicine Department in Assuit University Hospital. Their ages ranged from 32 to 70 years and the duration of illness was 2–7 years. They received oral hypoglycemic agents for DM control. On the basis of their serum $HbA_{1c}$% 14 patients had good glycemic control (serum $HbA_{1c}$% was ≤6.8%) and 18 patients had poor glycemic control (serum $HbA_{1c}$% was >6.8%).

In addition the study included a control group of 30 healthy volunteers (12 men and 18 women) matched in age, sex, and BMI. Their ages ranged from 32 to 68 years.

The exclusion criteria of the study were as follows: past history of otologic disease or exposure to ear trauma or surgery; history of noise exposure or ototoxic drug intake or head trauma; history of systemic or neurological diseases; manifestation of diabetic complications in the eyes, kidneys, or heart; presence of motor deficit or positive ECG or neuroimaging findings (computerized tomography of the brain).

All participants underwent the following: history taking, physical examination (BMI), systemic, cardiac, ophthalmic, and neurological examination, electrophysiological auditory function tests, and laboratory examinations of fasting serum glucose, serum $HbA_{1c}$%, and serum urea and creatinine, ECG, and brain computerized tomography.

### Auditory function assessments

Full history taking and otological examinations were carried out to evaluate any ear disorders and for identifying the presence of wax, which might impede the exams.

### Basic audiological evaluation

Pure tone and speech audiometry were carried out for each ear using a diagnostic audiometer (Madsen OB 822, Madsen electronics, Copenhagen, Denmark) with sound delivered through headphones (TDH 39-Telephonics).

In the present study, hearing loss was considered if the hearing threshold at one or more frequencies exceeded the normal intensity limits (0–25 dB HL for adults) [10].

Tympanometry and acoustic reflex threshold testing were carried out on each ear using a middle ear analyzer (interacoustic Az26) to exclude middle ear disease that may interfere with the accuracy of OAEs.

### Brainstem auditory evoked potential or auditory brainstem response

It was performed using the Nihon Kohden model MEB-7102 (Nihon Kohden Corp., Tokyo, Japan). BAEP was recorded using headphones; the type of sound used was clicks, with the duration of stimulus 0.1 ms, rate of stimulus 10 Hz, averaging 2000, and intensity of stimulus 90 dB. An active electrode was attached in the zone of the scalp (Cz) at the vertex; the reference electrode was placed on the ear lobule of the tested ear, and the ground electrode was placed at the midline of the forehead. The waves routinely analyzed in BAEP were numbered I–V. The absolute latency (stimulus to peak) of each peak (I, II, III, IV, and V) and interpeak latencies (IPL: I–III, I–V, and III–V) were measured.

Evoked OAEs: TEOAEs and DPOAEs were recorded using ILO 92, Otodynamic Analyser (Otodynamic Ltd, Hartfield, Hertz, UK), with participants sitting in a comfortable chair and as quiet as possible. The three-port probe (one microphone and two transducers) was positioned in the participants' outer ear canal with a good probe fit using a foam ear tip so that the emission would be properly recorded.

### TEOAE measurement

A series of 260 stimuli were presented in blocks of eight clicks in each testing situation, according to the nonlinear technique proposed by Kemp *et al.* [11]. Clicks were evoked with an intensity of 80 ± 2 dB SPL, and the collection of TEOAEs was terminated after 1024 accepted sweeps. The emission and noise amplitudes were calculated by the software in half-octave frequency bands centered at 1.0, 1.5, 2.0, 3.0, and 4.0 kHz, as well as the total emission and noise amplitude across frequencies. TEOAEs were present if emission amplitude exceeded the noise amplitude with at least 6 dB signal-to-noise ratio (SNR ≥6 dB) in at least three tested frequencies.

### DPOAEs measurement

2f1–f2 DPOAE levels were measured using the DP-grams procedure, in response to pure tones of intensity L1 = L2 = 70 dB SPL [12] with a frequency ratio f2/f1 of 1.22 and plotted with respect to the f2 primary tones. Amplitudes of three points per octave were assessed in the frequency range 0.5–6 kHz. DPOAE was considered present when the emission amplitude at all individual frequencies was at least 6 dB higher than its associated noise amplitude (SNR ≥6 dB) [13].

### Ethics

This study was conducted after obtaining approval from the Ethical Committee of the Faculty of Medicine, Assiut University. All participants gave written consent for participation in the study before assessment.

### Statistical analysis

The statistical analyses were performed using SPSS 16.0 for Windows (SPSS Inc., Chicago, Illinois, USA). Three separate statistical analyses were performed. First, the demographic data between diabetic patients and the control group were compared. Categorical variables were compared using the $\chi^2$-test. Continuous variables within two groups were compared using the independent *t*-test for parametric data and the Mann–Whitney *U*-test for nonparametric data.

The reference limits from the control group were derived from the mean ± 2 SD. The data exceeding the reference limits were considered to be 'outside reference data'. Second, continuous variables were compared among three groups using one-way analysis of variance and the Kruskal–Wallis test for parametric and nonparametric data, respectively. Also, we gathered data of auditory variables including BAEP between the right and left ears of the studied diabetic groups as there was no significant side-related difference on using the Wilcoxon test. Spearman's correlation was performed between serum $HbA_{1c}$% and BAEP and TEOAE responses. *P* less than 0.05 (two-tailed) was regarded as statistically significant.

## Results

No statistically significant differences were found between the mean age ± SD of the diabetic groups with poor versus good glycemic control and that of the healthy control group (51.3 ± 3.8 vs. 50.9 ± 12.4 and 45.2 ± 15 years, respectively), nor in BMI (32.3 ± 4.3 vs. 30.9 ± 5 and 30.6 ± 5 $m^2$) and systolic (133.6 ± 6.8; 130.9 ± 3.4 vs. 129.8 ± 5.8 mmHg) and diastolic (84.9 ± 6.1; 80.4 ± 6.9 vs. 80.9 ± 10.9 mmHg) blood pressure. The duration of illness in diabetic patients with poor glycemic control and those with good glycemic control was 6.3 ± 3.5 and 4.9 ± 2.3 years ($P = 0.291$). However, there was a significant difference between the mean values of serum fasting blood glucose and $HbA_{1c}$% in diabetic patients with poor and good glycemic and the control group (15.4 ± 3.8; 6.12 ± 1.9 vs. 5.6 ± 1.8 mmol/l; and 8.7 ± 0.8, 5.2 ± 1.1 vs. 4.7 ± 0.6%, respectively) ($P = 0.0001$ for all).

All studied patients had been treated for at least 6 months with various oral hypoglycemic drugs such as Glibenclamide (eight patients), Gliclazide modified release (MR) (seven patients), Glimepiride (14 patients), and combination of Glimepiride and Metforamin (three patients).

In the audiometry study, the percentage of hearing loss ranged from 8.8 to 70.6% among diabetic patients with poor glycemic control at different frequencies, with significant tendency to increase at higher frequency on trends analysis ($P = 0.0001$). However, there was no case with hearing loss either among diabetic patients with good glycemic or in the control group (Fig. 1a).

Patients with poor glycemic control had significantly high mean hearing thresholds at low and high frequencies when compared with other groups or compared with the good glycemic group ($P < 0.1$ and $P < 0.001$) (see Table 1 and Fig. 1b).

Speech discrimination scores were excellent in both study and control groups. Normal middle ear functions and normal acoustic reflex thresholds were evident in all participants.

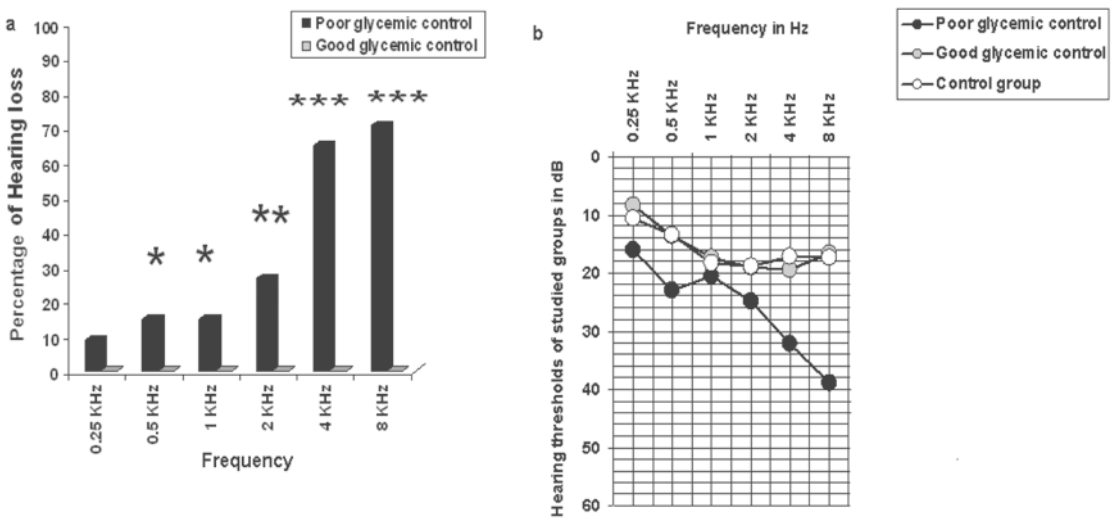
We found that 12 out of 18 patients (66.7%) with poor glycemic control had hearing loss. These patients were receiving oral hypoglycemic drugs as follows; two patients in each group received Glibenclamide (2/8; 25%) and Gliclazide MR (2/7; 28.5%), six patients (6/14; 42.8%) received Glimepiride, and the remaining

two patients (2/3; 66.7%) received a combination of Glimepiride and Metforamin. However, no significant association was found between hearing loss and various oral hypoglycemic drugs, whether Glibenclamide, Gliclazide MR, or Glimepiride, as measured by the $\chi^2$-test, among 29 patients ($P = 0.100$). The remaining three patients who received a combination of Glimepiride and Metforamin were excluded from estimation of the effect of oral hypoglycemic drugs on hearing loss in the statistical analysis because of their small number.

Both diabetic groups recorded prolonged absolute latencies of wave I with consecutive delay in wave latencies III and V and IPLs III–V in BAEP, which exceeded mean ± 2 SD of control values.

The diabetic group with poor glycemic control had significant prolongation of absolute latencies of waves I, III, and V and IPL III–V compared with the other groups ($P < 0.01$ and $P < 0.001$) (details are illustrated in Table 2).

**Figure 1**




(a) A statistically significant difference in the percentage of hearing loss between diabetic groups (poor vs. good glycemic control) at different frequencies (0.25–8 kHz). *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$. (b) Hearing thresholds in the audiogram at different frequencies between the studied groups (diabetic groups with poor vs. good glycemic control and the healthy control group).

**Table 1 Hearing thresholds in the studied groups at different frequencies**

| Frequency | Poor glycemic control ($n = 36$) (mean ± SD) | Good glycemic control ($n = 28$) (mean ± SD) | Control group ($N = 30$) (mean ± SD) | *P* value between three groups using the Kruskal–Wallis test | *P* value between diabetic groups using the Mann–Whitney *U*-test |
|---|---|---|---|---|---|
| 0.25 kHz | 16.1 ± 10.9 | 8.5 ± 5.3 | 10.7 ± 2.4 | 0.010 | 0.002 |
| 0.5 kHz | 22.9 ± 10.8 | 13.7 ± 4.1 | 13.5 ± 4.2 | 0.000 | 0.000 |
| 1 kHz | 20.6 ± 13.4 | 17.4 ± 5.1 | 18.4 ± 2.8 | 0.879 | 0.633 |
| 2 kHz | 24.8 ± 14.6 | 19.1 ± 6.5 | 18.9 ± 2.6 | 0.377 | 0.230 |
| 4 kHz | 32.1 ± 16.1 | 19.4 ± 3.2 | 17.2 ± 2.8 | 0.000 | 0.001 |
| 8 kHz | 38.9 ± 19.9 | 16.7 ± 6.4 | 17.3 ± 3.3 | 0.000 | 0.000 |

**Table 2 Auditory brainstem evoked potentials in the studied groups**

| Wave latency/IPL | Poor good glycemic control ($n = 36$) (mean ± SD) | Good glycemic control ($n = 28$) (mean ± SD) | Control group ($n = 30$) | *P* value between three groups using the Kruskal–Wallis test | *P* value between diabetic groups using the Mann–Whitney *U*-test |
|---|---|---|---|---|---|
| ABR-wave I | 1.7 ± 0.2 | 1.5 ± 0.2 | 1.2 ± 0.1 | 0.000 | 0.073 |
| ABR-wave III | 3.8 ± 0.2 | 3.7 ± 0.1 | 3.2 ± 0.1 | 0.000 | 0.321 |
| ABR-wave V | 5.7 ± 0.3 | 5.5 ± 0.2 | 5.1 ± 0.1 | 0.000 | 0.008 |
| IPL I–III | 2.1 ± 0.2 | 2.1 ± 0.2 | 2 ± 0.1 | 0.143 | 0.004 |
| IPL III–V | 2 ± 0.3 | 1.7 ± 0.1 | 1.9 ± 0.2 | 0.000 | 0.000 |
| IPL I–V | 4 ± 0.3 | 3.9 ± 0.2 | 3.8 ± 0.4 | 0.130 | 0.088 |

ABR, auditory brainstem response; IPL, interpeak latency.

Concerning cochlear function assessment, poor glycemic control had significantly low amplitudes at all frequencies, as well as low overall response, in the poor glycemic control group compared with other groups ($P < 0.01$ and $P < 0.001$) in the TEOAE test. Moreover, the mean values of DPOAE amplitudes were significantly reduced in diabetic patients with poor glycemic control compared with other groups at high frequencies of 4 and 6 kHz ($P = 0.043$ and 0.016, respectively; Tables 3 and 4, respectively).

The effect of demographic data – age, sex, and duration of disease — on auditory functions either on sensory or neural parameters was estimated using Spearman's correlation. There was a significant correlation between age and IPL I–V as well as overall response of TEOAE amplitude ($r = 0.308$, $P = 0.035$; $r = -0.501$, $P = 0.011$, respectively). However, there was no significant correlation between these auditory parameters and sex ($P = 0.922$ and 0.433) or duration of disease ($P = 0.243$ and 0.604).

However, the effect of glycemic control on auditory functions either on sensory or neural parameters was estimated using Spearman's correlation and illustrated in Fig. 2a and b, which revealed that $HbA_{1c}$% was

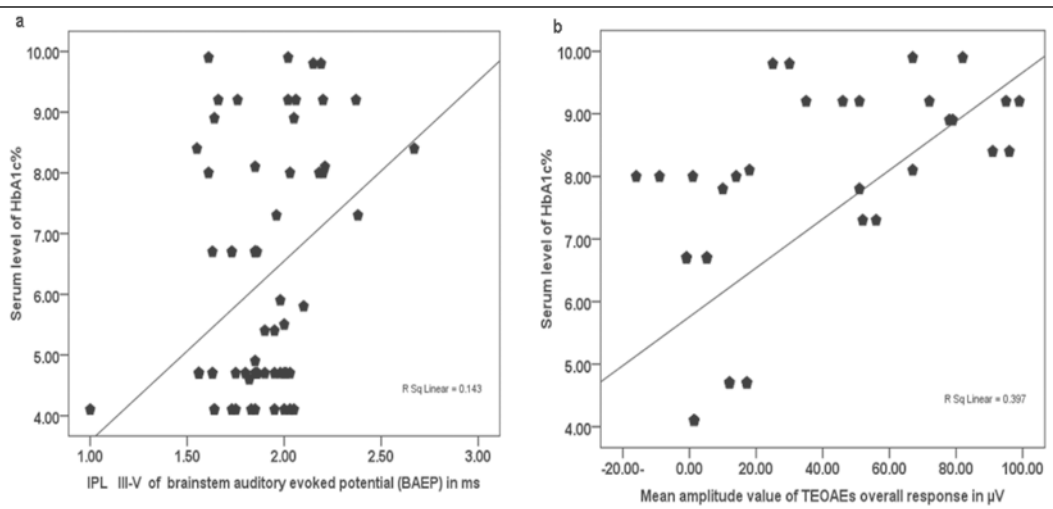
**Table 3 Mean values of TEOAE amplitude at different frequencies in the studied groups**

| S/N ratio frequency | Poor glycemic control ($n = 36$) (mean ± SD) | Good glycemic control ($n = 28$) (mean ± SD) | Control group ($n = 30$) (mean ± SD) | *P* value between three groups using the Kruskal–Wallis test | *P* value between diabetic groups using the Mann–Whitney *U*-test |
|---|---|---|---|---|---|
| Overall response | 2.8 ± 5.4 | 7.9 ± 6.7 | 14 ± 2.3 | 0.000 | 0.001 |
| 1 kHz | 2 ± 5.7 | 8.3 ± 7 | 14.4 ± 2.6 | 0.000 | 0.000 |
| 1.5 kHz | 5.4 ± 7.8 | 11.7 ± 6.4 | 17.4 ± 2.3 | 0.000 | 0.000 |
| 2 kHz | 5.4 ± 8.1 | 13.2 ± 8.1 | 14.1 ± 1.3 | 0.000 | 0.000 |
| 3 kHz | 3.2 ± 6.1 | 7.1 ± 4.4 | 10.5 ± 1.7 | 0.000 | 0.001 |
| 4 kHz | 2 ± 5.1 | 3.2 ± 5.1 | 11.7 ± 2.2 | 0.000 | 0.160 |

**Table 4 Mean values of DPOAE amplitude at different frequencies in the studied groups**

| Frequency | Poor glycemic control ($n = 36$) (mean ± SD) | Good glycemic control ($n = 28$) (mean ± SD) | Control group ($n = 30$) (mean ± SD) | *P* value between three groups using the Kruskal–Wallis test | *P* value between diabetic groups using the Mann–Whitney *U*-test |
|---|---|---|---|---|---|
| 1 kHz | 24 ± 11.1 | 21.7 ± 7.4 | 22.5 ± 6.6 | 0.229 | 0.147 |
| 1.5 kHz | 27.5 ± 13.6 | 28.5 ± 5.3 | 25.7 ± 4.5 | 0.075 | 0.472 |
| 2 kHz | 28.5 ± 12.2 | 27.7 ± 7.7 | 27.6 ± 5.5 | 0.292 | 0.255 |
| 3 kHz | 31.6 ± 13.8 | 31.6 ± 7.7 | 32.8 ± 6.1 | 0.444 | 0.224 |
| 4 kHz | 31 ± 15.1 | 32.1 ± 2.5 | 36 ± 4 | 0.043 | 0.446 |
| 6 kHz | 20.6 ± 15.4 | 23.7 ± 14.4 | 31.44 ± 6.2 | 0.016 | 0.386 |

**Figure 2**




The effect of glycemic control on auditory functional parameters (IPL III–V in brainstem auditory evoked potential and overall response to TEOAE amplitude). (a) Significant positive correlation between serum level of $HbA_{1c}$% and IPL III–V ($r = 0.340$, $P = 0.004$), but (b) significant negative correlation between serum level of $HbA_{1c}$% and overall response to TEOAE amplitude ($r = -0.471$; $P = 0.000$) among all participants. $HbA_{1c}$, glycosylated hemoglobin; IPL, interpeak latency.

significantly correlated with IPL III–V ($r$ = 0.340; $P$ = 0.004) and with overall response of TEOAE amplitude ($r$ = –0.471; $P$ = 0.000).

## Discussion

In the present comparative study, auditory functions were evaluated in detail among patients with poor versus good glycemic control and the healthy control group to detect subclinical alteration.

According to audiometric results, SNHL ranged from 8.8% (at 0.250 kHz) to 70.6% (at 8 kHz frequency) among diabetic patients with poor glycemic control. It is consistent with studies by Wu *et al.* [2] and Austin *et al.* [3], who found an association between hearing loss and DM. Moreover, the poor glycemic control group had significantly higher hearing thresholds compared with the good glycemic control group and the control group (at all frequencies except 1 and 2 kHz). Our data are partially consistent with the data reported by Karabulut *et al.* [14], who found SNHL at high frequencies in both patients and controls. Our findings could be attributed to the fact that aging and DM together accelerate hearing loss, or presbycusis, by synergistic action as reported previously by Cullen and Cinnamond [15].

Although metaformin has been claimed to increase the loss of vitamin B12 because of malabsorption of vitamin B12 in diabetic patients, which exacerbates the DM neuropathy and cranial DM neuropathy [16], in our study the small number (three patients) of patients who used a combination of Glimepiride and metaformin were excluded from the statistical analysis of drug effect on hearing loss. However, no significant association was found between other oral hypoglycemic drugs, whether Glibenclamide, Gliclazide, or Glimepiride, and hearing loss among diabetic patients.

In the present study, patients with poor glycemic control had significant prolongation of absolute latency of wave I with consecutive prolongation latency of waves III and V as well as IPL III–V compared with other groups, which may suggest both peripheral and central auditory (mainly retrocholear) pathway affection related to glycemic control as previously reported in other studies [17,18]. Our findings are consistent with common findings reported in ABEP studies [19,20], whereas the latency of waves III and V were prolonged. However, other studies [21,22] reported no relation between levels of $HbA_{1c}$% and results of BAEP.

Prolonged IPL I–III in the diabetic groups is partially consistent with the findings reported by Huang *et al.* [23], but no significant prolonged IPL I–V was found in the studied groups in their study [23].

TEOAE was used for early detection of cochlear dysfunctions in the studied groups. Patients with poor glycemic control showed significantly reduced amplitude at all frequencies when compared with other groups. Our findings are consistent with those of other studies [24–26]. However, our results are inconsistent with those of Ugur *et al.* [27], who found no difference in amplitudes of TEOAEs. Our findings suggest that the metabolic changes caused by DM can modify the micromechanics of the inner ear, generating an early subclinical finding. This was previously confirmed through histopathological studies [28,29] of the inner ear, which demonstrated the following:

(1) A thickening of the capillary walls of the vascular stria epithelial stratification that forms the endolymphatic edge of cochlear sac, which is important in the production of ionic gradients and of the endocochlear potential;
(2) Peri and endolymphatic hemorrhaging;
(3) Reduction in the number of fibers of the spiral plate; and
(4) Degenerative changes in the organ of Corti and reduction in the number of outer hair cells [28,29].

The DPOAE test is more specific for confirming cochlear function affection at high frequencies. Measurement of DPOAEs corresponds closely to the physiological state of the cochlear outer hair cells and determines the site of pathology associated with SNHL [26]. The results of our study showed a significantly low amplitude of DPOAEs at high frequencies (4–6 kHz) among patients with poor glycemic control, which suggests that the peripheral auditory alterations affect the high frequencies earlier in these patients.

Our findings are consistent with those of Erdem *et al.* [30], who reported low amplitude among diabetic patients at high frequencies on DPOAE, but it is partially inconsistent with the data of Karabulut *et al.* [14], who found a statistically significant difference between patients and control at all frequencies ($P < 0.05$) except 1 kHz on the DPOAE test.

In the study groups, significant association was found between age and auditory functions, either neural or sensory parameters, as age was significantly correlated with IPL I–V ($r$ = 0.308, $P$ = 0.035) as well as with overall response of TEOAE amplitude ($r$ = –0.501; $P$ = 0.011), without significant correlation with sex or duration of disease. Our findings were consistent with those of Durmus *et al.* [31] and Dabrowski *et al.* [25]. In contrast, Durmus *et al.* [31]. reported a correlation

between age (>30 years) and IPLs (I–V and III–V) in the ABR study, but no correlation to duration of disease or sex, although Dabrowski *et al.* [25] found a negative correlation between amplitude TOAE and age, without correlation to duration of disease. The significant association found by us could be attributed to the fact that age is an important factor leading to hearing loss, and the presence of DM accelerates age-related hearing loss, or presbycusis, by synergistic action, as reported by Cullen and Cinnamond [15]. However, Cullen and Cinnamond [15] found that hearing loss is more predominant among men than among women, which may be secondary to noise exposure. This difference in reported findings could be attributed to the selected inclusion and exclusion criteria of the studied sample in different studies.

In the present study, the effect of glycemic control on auditory functional parameters was estimated and revealed significant correlation mainly between $HbA_{1c}$% and IPL III–V in the BAEP test ($r = 0.340$ and $P = 0.004$) on the one hand and between $HbA_{1c}$% and overall response of TEOAE amplitude ($r = -0.471$; $P = 0.000$) on the other. These data indicate glycemic control effect and its enhanced related metabolic changes in DM on the auditory nerve pathway on the one hand and outer hair cell functions at the cochlea on the other.

The TEOAE correlation findings are consistent with the reported findings of Ugur *et al.* [27], who recorded a negative correlation between the TEOAE amplitude and the values of $HbA_{1c}$% in type 1 DM. These data demonstrate the importance of metabolic control in mediating cochlear functions through the following effects: glucose metabolism has great influence on the inner ear and subsequently both hypoglycemia and hyperglycemia may affect its normal functioning. In addition, vascular striae depend on a constant concentration of blood glucose [32].

The significant positive correlation between serum $HbA_{1c}$% and IPL III–V in the BAEP test confirms the importance of metabolic control for brainstem function among diabetic patients. This result supports the study by Seidl *et al.* [33]. In contrast, Díaz de León-Morales *et al.* and Talebi *et al.* [21,22] did not find a relationship between serum $HbA_{1c}$% and BAEP results.

## Conclusion and recommendation

Patients with type 2 DM had clinical and subclinical auditory dysfunctions, either cochlear or neural. These auditory dysfunctions are influenced by glycemic control (serum $HbA_{1c}$%). Early glycemic control is mandatory for preventing auditory comorbidities among diabetic patients. Further studies are needed on a large number of patients with large investigatory tools to study the predictors of auditory dysfunctions.

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**Conflicts of interest**

There are no conflicts of interest.

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