ORIGINAL ARTICLE

Is there a direct correlation between the duration and the treatment of type 2 diabetes mellitus and hearing loss?

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Abstract

Aim: The aim of the study was to determine the impact of the duration of diabetes and the control of glycemia on the auditory function of patients with type 2 diabetes mellitus (T2DM).

Materials and Methods: This prospective study included 80 patients with T2DM (divided depending on when T2DM was diagnosed, and also according to the control of glycemia), and 50 healthy subjects.

Results: The hearing threshold in T2DM patients was statistically significantly higher for 1,000 Hz, 2,000 Hz, 4,000 Hz and 8,000 Hz. Absolute latencies of brainstem auditory evoked potentials (BAEP) revealed significant differences between average absolute latencies for waves I, III and V, as well as inter-wave latencies I–V and I–III ($p < 0.001$). A statistically significant difference was noted in the presence of transitory otoacoustic emissions (TEOAE) ($p < 0.001$). In T2DM patients with poor glycemic control, where the glycated hemoglobin (HbA1c) is above 7%, the hearing threshold levels were statistically significantly higher in both ears at 8,000 Hz and at 2,000 Hz in the right ear, and the absolute latency of wave V was prolonged in the right ear. There was no evidence that the duration of diabetes significantly affected the auditory threshold, absolute and inter-wave BAEP latencies.

Conclusion: The patients with T2DM displayed an increased hearing threshold, qualitative changes in BAEP and the absence of TEOAE. The duration of poorly-controlled glycemia had a greater effect on the patients' auditory function than the duration of T2DM. Hippokratia 2016, 20(1): 32-37

Keywords: Type 2 diabetes mellitus,pure tone audiometry, transitory otoacoustic emissions, brainstem auditory evoked potentials

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Introduction

Type 2 diabetes mellitus (T2DM) is a chronic, progressive metabolic disease whose incidence and prevalence is on the increase[1]. A large number of studies indicated a correlation between diabetes and sensorineural hearing impairment[2-5]. Examination with pure tone audiometry of patients with T2DM suggested an incidence of sensorineural hearing impairment, ranging from 44% to 69.7%, which is 1.5 to 2.5 times higher when compared to an age-matched control group[2,4]. A variety of diagnostic audiological tests have been employed in an attempt to determine the pathophysiology of these changes.

Otoacoustic emissions (OAE) can demonstrate the cochlear dysfunction, i.e. they indicate changes in the cochlear micromechanics caused by the functional damage of outer hair cells[6]. Some studies reported their absence, as well as a reduction in their amplitude in T2DM patients[6,7].

Brain stem auditory evoked potential (BAEP) testing is used to diagnose initial functional damage of the peripheral and central parts of the auditory pathway in its initial phase[8]. Several authors reported prolonged absolute and inter-wave latencies in T2DM patients[9-11], while others did not observe any significant changes[12]. The results in the literature are divergent regarding the impact of the duration of diabetes, the control of glycemia and the presence of chronic complications of diabetes on sensorineural hearing impairment, BAEP, and OAE[3,5,11,13]. The objectives of this paper were to determine the auditory function of T2DM patients in relation to the control group, using pure tone audiometry, transient evoked OAE (TEOAE) and BAEP, as well as to determine the impact of the duration of diabetes and the control of glycaemia on the auditory function of patients with T2DM.

Materials and Methods

This prospective study included 80 patients with T2DM, aged between 40 and 60 years, of both genders (34 men and 46 women) that constituted the study group and a control group consisting of 50 healthy subjects, also

Table 1: The comparison of the hearing thresholds between the 80 patients with T2DM patients and the 50 healthy controls.

Frequency (Hz)	Control group (n =50) Median (min-max)(dB)	Diabetes mellitus (n =80) Median (min-max)(dB)	p value
125 R	20 (10-30)	20 (10-40)	0.100
125 L	20 (10-30)	20 (10-30)	0.078
250 R	20 (10-40)	20 (10-40)	0.424
250 L	20 (10-30)	20 (10-40)	0.588
500 R	20 (10-40)	20 (10-40)	0.107
500 L	20 (10-30)	20 (10-40)	0.356
1,000 R	20 (10-40)	30 (20-40)	0.008
1,000 L	20 (10-30)	25 (20-40)	0.004
2,000 R	30 (10-40)	30 (20-70)	<0.001
2,000 L	25 (10-40)	30 (20-60)	<0.001
4,000 R	30 (10-50)	40 (20-80)	<0.001
4,000 L	30 (20-40)	40 (20-70)	<0.001
8,000 R	30 (10-50)	40 (20-90)	<0.001
8,000 L	40 (10-50)	60 (20-80)	<0.001

T2DM: Type 2 diabetes mellitus, n: number of subjects, Hz: Hertz, dB: Decibel, R: right ear, L: left ear, p: Mann-Whitney U test value (p <0.05, bolded if significant).

Table 2: TEOAE in the control (50 healthy subjects) and the diabetic (80 patients with T2DM) groups.

TEOAE	Control group (n =50)		Diabetes mellitus group (n =80)		p value
	present	absent	present	absent	
Right ear	90.0%	10.0%	61.2%	38.8%	<0.001
Left ear	88.0%	12.0 %	57.5 %	42.5 %	<0.001

TEOAE: Transitory otoacoustic emissions, n: number of subjects, p: $\chi 2$- test value (p <0.05, bolded if significant).

aged between 40 and 60 years (21 men and 29 women).

We excluded from the study patients with chronic middle ear disease, head injuries with labyrinth commotion, hereditary deafness, and patients who used ototoxic medications or had been exposed to high levels of noise and vibration in their workplace. Bone conduction did not interfere with our protocol since we excluded patients with conductive and combined hearing loss from this study. The research was approved by the Ethical Committee of the University Clinical Center of Nis, Nis, Serbia (No 15941/14, 6/2/2014). After the diagnosis and treatment at the Endocrinology Clinic, the patients underwent auditory testing at the Ear, Nose and Throat Clinic, at the University Clinical Center of Nis and were enrolled in the study. The research was conducted from March 2014 to February 2015. The impact of the duration of diabetes and the control of glycemia on the auditory function was analyzed in these patients with T2DM.

Pure tone audiometry was performed in a soundproof environment using a Madsen OB 822 audiometer (Madsen electronics, Copenhagen, Denmark). Hearing thresholds were determined by measuring air conduction of sound at tone frequencies of 125 Hz, 250 Hz, 500 Hz, 1,000 Hz, 2,000 Hz, 4,000 Hz, and 8,000 Hz.

TEOAE were performed using the Eclipse Platform TEOAE 25, software version 3.03 (Interacoustics, Middelfart, Denmark). This device was used to register click-evoked OAE in each ear, following the standard

Table 3: The comparison of absolute and inter-wave latencies of BAEP between the 80 T2DM patients and the 50 non-diabetic healthy controls.

BAEP latencies	Control group (n =50) Mean ± SD (ms)	Diabetes mellitus group(n =80) Mean ± SD (ms)	p value
I R	1.32 ± 0.04	1.34 ± 0.04	0.006
I L	1.31 ± 0.04	1.34 ± 0.04	0.001
III R	3.48 ± 0.08	3.61 ± 0.08	<0.001
III L	3.48 ± 0,04	3.61 ± 0.08	<0.001
V R	5.35 ± 0.11	5.49 ± 0.10	<0.001
V L	5.35 ± 0.10	5.48 ± 0.09	<0.001
I-III R	2.17 ± 0.07	2.27 ± 0.08	<0.001
I-III L	2.17 ± 0.07	2.26 ± 0.07	<0.001
III-V R	1.86 ± 0.08	1.87 ± 0.06	0.645
III-V L	1.86 ± 0.07	1.87 ± 0.06	0.328
I-V R	4.03 ± 0.10	4.14 ± 0.09	<0.001
I-V L	4.03 ± 0.10	4.14 ± 0.07	<0.001

T2DM: Type 2 diabetes mellitus, n: number of subjects, BAEP: brainstem auditory evoked potentials, SD: standard deviation, ms: millisecond, R: right ear, L: left ear, p: Student's t-test value (p <0.05, bolded if significant).

Table 4: The correlation between the duration of T2DM and the hearing threshold.

	Duration of diabetes		
Frequency (Hz)	**≤ 10 years (n =51) Median (min-max)(dB)**	**>10 years (n =29) Median (min-max)(dB)**	**p value**
125 R	20 (10-40)	20 (10-40)	0.662
125 L	20 (10-30)	20 (10-20)	0.772
250 R	20 (10-40)	20 (10-40)	0.755
250 L	20 (10-40)	20 (10-40)	0.820
500 R	20 (10-40)	20 (20-40)	0.623
500 L	20 (10-40)	20 (20-40)	0.583
1,000 R	30 (20-40)	30 (20-40)	0.931
1,000 L	20 (20-40)	30 (20-40)	0.482
2,000 R	30 (20-70)	40 (20-60)	0.285
2,000 L	30 (20-60)	30 (20-60)	0.885
4,000 R	40 (20-80)	40 (20-60)	0.264
4,000 L	40 (20-60)	40 (20-70)	0.395
8,000 R	40 (20-90)	40 (20-70)	0.455
8,000 L	40 (20-80)	40 (20-80)	0.138

T2DM: Type 2 diabetes mellitus, n: number of subjects,Hz: Hertz, dB: Decibel, R: right ear, L: left ear, p: Mann-Whitney U test value ($p < 0.05$, bolded if significant).

protocol[14]. The stimuli consisted of the standard number (1,000) of non-linear clicks at 83 dB sound pressure level (SPL). The 83 dB SPL click stimuli were delivered in a closed ear canal at regular intervals of 20 ms. According to the obtained results, the study and the control group were each divided into two subgroups, with one subgroup consisting of patients with TEOAE and the other consisting of patients without TEOAE.

BAEP were tested using the Eclipse Platform EP 25, software version 3.03 (Interacoustics, Middelfart, Denmark). Acoustic stimulation was performed by delivering 100 dB monaural clicks of alternating polarities and with repetition rates of 27.4. The total number of stimuli for each intensity was 2,000[12]. Absolute latencies for waves I, II, III and V, as well as inter-wave latencies for waves I-III, III-V, I-V, were recorded.

The glycated hemoglobin (HbA1c), the most significant indicator of glycemia control, was measured in T2DM patients. We divided the patients into two categories: those with well-controlled diabetes (HbA1c ≤7%) and those with poorly-controlled diabetes (HbA1c >7%). Then we selected 40 patients from each of these two categories, and thus created two subgroups that constituted our study group.

The patients were also divided into two different subgroups, depending on when T2DM was diagnosed; the first subgroup consisted of patients who had had the disease for less than ten years (51 patients), whereas the second subgroup consisted of patients who had had the disease for more than ten years (29 patients). During our study of the impact of the duration of T2DM on audiological function, glycemia was well-controlled in all patients. Auditory test results obtained for each subgroup of T2DM patients were compared.

The sample size was calculated using STATISTICA software for Windows, Version 8.0 (StatSoft Inc, Tulsa, OK, USA). We determined the approximate average values and standard deviations of the observed variables from a pilot study and, based on these values as well as the significance threshold ($\alpha = 0.05$) and the power test ($1-\beta = 0.8$),

Table 5: The correlation between the duration ofT2DM and absolute and inter-wave latencies of BAEP.

	Duration of diabetes		
BAEP latencies	**≤ 10 years (n =51) Mean ± SD (ms)**	**>10 years (n =29) Mean ± SD (ms)**	**p value**
I R	1.34 ± 0.04	1.34 ± 0.05	0.995
I L	1.33 ± 0.04	1.35 ± 0.04	0.261
III R	3.60 ± 0.08	3.62 ± 0.08	0.300
III L	3.61 ± 0.08	3.61 ± 0.09	0.686
V R	5.47 ± 0.10	5.51 ± 0.08	0.091
V L	5.47 ± 0.10	5.51 ± 0.08	0.119
I-III R	2.26 ± 0.07	2.27 ± 0.08	0.462
I-III L	2.26 ± 0.07	2.26 ± 0.07	0.844
III-V R	1.86 ± 0.07	1.89 ± 0.04	0.127
III-V L	1.86 ± 0.06	1.88 ± 0.05	0.087
I-V R	4.13 ± 0.98	4.17 ± 0.07	0.066
I-V L	4.13 ± 0.08	4.16 ± 0.06	0.167

T2DM: Type 2 diabetes mellitus, n: number of subjects, BAEP: brainstem auditory evoked potentials, SD: standard deviation, ms: millisecond, R: right ear, L: left ear, p: Student's t-test value ($p < 0.05$, bolded if significant).

Table 6: The correlation between glycemic control and hearing thresholdin T2DM patients.

	Diabetes mellitus		
Frequency (Hz)	**HbA1c ≤ 7% (n =40) Median (min-max)(dB)**	**HbA1c >7% (n =40) Median (min-max)(dB)**	**p value**
125 R	20 (10-40)	20 (10-40)	0.264
125 L	20 (10-30)	20 (10-30)	0.556
250 R	20 (10-40)	20 (10-40)	0.143
250 L	20 (10-40)	20 (10-40)	0.292
500 R	20 (20-40)	20 (10-40)	0.212
500 L	20 (10-40)	20 (10-40)	0.602
1,000 R	20 (20-40)	30 (20-40)	0.090
1,000 L	20 (20-40)	30 (20-40)	0.113
2,000 R	30 (20-60)	40 (20-70)	0.021
2,000 L	30 (20-60)	30 (20-60)	0.136
4,000 R	40 (20-60)	40 (20-80)	0.520
4,000 L	40 (20-70)	40 (20-60)	0.075
8,000 R	40 (20-70)	40 (20-90)	0.041
8,000 L	40 (20-70)	40 (20-90)	0.030

HbA1c: Glycated hemoglobin, n: number of subjects, Hz: Hertz, dB: Decibel, R: right ear, L: left ear, p: Mann-Whitney U test value (p <0.05, bolded if significant).

we obtained the minimum sample size (n =25).

Normality of distribution was tested using the Kolmogorov-Smirnov test. The mean values of variables with normal distribution were compared using Student's t-test for independent samples and variables with a lack of normality were compared using the non-parametric Mann-Whitney U test for two independent samples. Categorical variables were compared using a chi-squared test. The data were analyzed with SPSS 16.0 for Windows (SPSS Inc., Chicago, USA). The probability level of p <0.05 was considered to be statistically significant.

Results

The mean age of T2DM subjects was 52.60 ± 6.36 years, whereas the age of the subjects in the control group was 52.48 ± 6.21 years. In the study group, the mean duration of T2DM was 11.2 years.

The hearing threshold in T2DM patients was higher than in the subjects from the control group at all frequencies. A statistically significant difference was noted in both ears in T2DM patients at 1,000 Hz, 2,000 Hz, 4,000 Hz and 8,000 Hz (Table 1).

The analysis of the results, i.e. the presence of TEOAE in both ears in T2DM patients and in the healthy subjects from the control group, which was performed using a chi-squared test for p <0.001 (Table 2), revealed a statistically significant difference.

The analysis of absolute latencies in the study and control groups using Student's t-test for independent samples revealed significant differences in the mean values, more precisely, the mean values for waves III and V were p <0.001 in both ears, and the mean values for wave I were p =0.006 in the right ear and p =0.001 in the left ear (Table 3). No significant differences were observed ininter-wave latencies III-V. On the other hand, a statistically significant difference was seen in inter-wave latencies I-III for the right and the left ears. The obtained inter-wave latencies I-V for both ears in the study group were statistically significantly different from those obtained in the control group (Table 3). The differences in inter-wave la-

Table 7: The correlation between glycaemic controlin T2DM patients and absolute and inter-wave latencies of BAEP.

	Diabetes mellitus		
BAEP latencies	**HbA1c ≤ 7% (n =40) Mean ± SD (ms)**	**HbA1c> 7% (n =40) Mean ± SD (ms)**	**p value**
I R	1.34 ± 0.04	1.34 ± 0.04	0.466
I L	1.34 ± 0.04	1.34 ± 0.04	0.781
III R	3.60 ± 0.09	3.63 ± 0.07	0.156
III L	3.61 ± 0.08	3.61 ± 0.08	0.775
V R	5.46 ± 0.10	5.51 ± 0.09	0.043
V L	5.47 ± 0.09	5.50 ± 0.09	0.115
I-III R	2.26 ± 0.09	2.27 ± 0.07	0.440
I-III L	2.26 ± 0.07	2.26 ± 0.07	0.914
III-V R	1.86 ± 0.06	1.88 ± 0.06	0.138
III-V L	1.86 ± 0.05	1.88 ± 0.06	0.106
I-V R	4.12 ± 0.09	4.16 ± 0.08	0.055
I-V L	4.12 ± 0.07	4.16 ± 0.07	0.056

T2DM: Type 2 diabetes mellitus, HbA1c: glycated hemoglobin, n: number of subjects, BAEP: brainstem auditory evoked potentials, SD: standard deviation, ms: millisecond, R: right ear, L: left ear, p: Student's t-test value (p <0.05, bolded if significant).

tencies I-V were caused by the alteration of inter-wave latencies I–III ($p < 0.001$).

No statistically significant difference was observed in the hearing threshold in T2DM patients at all the tested frequencies, or in the absolute and inter-wave latencies of BAEP between the subgroups that the patients were divided into according to the duration of the disease (Table 4, Table 5). Depending on the control of glycemia in T2DM patients, there was a statistically significant difference in the hearing threshold between the subgroup with well-controlled and that with poorly-controlled glycemia at 8,000 Hz in the right ($p = 0.041$) and the left ear ($p = 0.030$) and at 2,000 Hz in the right ear ($p = 0.021$) (Table 6). The analysis of BAEP results revealed a significant difference between these subgroups in the absolute latency of wave V for the right ear ($p = 0.043$). The difference in inter-wave latencies I-V for the right and the left ears between the subgroup with well-controlled glycemia and the subgroup with poorly-controlled glycemia was of borderline significance (Table 7).

Discussion

Our results indicate that the hearing threshold was higher at any frequency in T2DM patients in comparison with the controls, with a statistical significance at 500 Hz, 1,000 Hz, 2,000 Hz, 4,000 Hz and 8,000 Hz, which is in agreement with the results reported by other authors[3,10]. By testing the hearing threshold using pure tone audiometry, several authors reported that the hearing threshold in T2DM patients is higher than that in the control group at all frequencies[3,10,15]. Some authors noted a gradual, progressive bilateral sensorineural hearing loss in T2DM patients, particularly at the high frequencies[3,7,16,17], while others observed a sensorineural hearing loss at the low frequencies[18]. The meta-analysis conducted by Akinpelu et al[4] showed that the hearing threshold in T2DM patients was higher at all frequencies but statistically significantly higher only at 6,000 Hz and 8,000 Hz.

The pathological changes described in the literature that may cause sensorineural hearing loss in T2DM patients include cochlear microangiopathy, hyperglycemia in the cerebrospinal fluid and perilymph, auditory neuropathy and diabetic encephalopathy[5]. Temporal bone histopathological findings in T2DM patients with symptoms of sensorineural hearing loss suggested a significant thickening of the walls of the blood vessels in the basal membrane and the capillaries in the striae vascularis, and atrophy of the striae vascularis. In addition to this, a significant reduction in the number of outer hair cells in the basal part of the cochlea was reported[19].

We noted a statistically significant difference between absolute latencies for waves I, III and V, and inter-wave latencies for waves I-V, caused by the impact of prolonged I-III inter-wavelatencies in T2DM patients as opposed to the controls. Moghaddam et al[12] did not report differences in any BAEP parameter in T2DM patients in comparison with healthy subjects. Toth et al[8] and Durmus et al[20], noted a statistically significant prolongation of absolute latencies for waves I, III and V in T2DM patients. Other authors noted prolonged absolute latencies for waves III and V[8,9,21].

Most studies observed a statistically significant prolongation of absolute latencies for wave V and of inter-wave latencies for waves I-V in T2DM patients in relation to the controls[10,11]. Prolonged I-V inter-wave latencies in T2DM patients may be caused by prolonged I-III inter-wave latencies[8,11], or III-V inter-wave latencies[9,22,23], or both[13,21]. Prolonged absolute latencies for waves III and V, in addition to prolonged I-III, III-V, and I-V inter-wave latencies in T2DM patients indicate brainstem dysfunction even in subclinical hearing impairment.

Some studies reported prolonged latencies of BAEP responses in T2DM patients with microvascular complications and peripheral neuropathy[20,24], while other studies did not note any such correlation[13]. Díaz de León-Morales et al[23] observed a subclinical hearing loss at high frequencies and prolonged absolute latencies for wave V, as well as I−V inter-wave latencies, caused by prolonged III−V inter-wave latencies in T2DM patients, regardless of other complications. BAEP analyses in T2DM patients give divergent results, which may be explained by the differences in individual patient reactions to T2DM but also by the impact of comorbidity on the results.

Our study revealed a statistically significant difference in the presence of TEOAE in both ears in T2DM patients as compared to the control group. Erdem et al[6] identified a subclinical auditory dysfunction in T2DM patients in the form of a reduction in the distortion-product otoacoustic emissions (DOAE) amplitude at 4 kHz and the presence of hyperlipoproteinemia; however, they did not observe any statistically significant changes in the presence of TEOAE at all frequencies. Reduced DOAE and TEOAE amplitudes in T2DM patients were documented in some studies[6,7]. Diabetes can cause a reduction in the function of outer hair cells, and the presence of TEOAE is in correlation with the degree of hearing impairment[6].

Some studies noted that patients who had had diabetes for over ten years were at risk for hearing loss[2], whereas other studies did not report this correlation[5,15,25]. The results of our study did not indicate that the duration of diabetes had a statistically significant impact on the hearing threshold in T2DM patients or on their BAEP results.This may be explained either by comorbidity or by the fact that T2DM is sometimes diagnosed late, i.e. it is diagnosed after the onset of disease complications. Many authors noted that the duration of diabetes had no effect on BAEP results[11,24], although there are studies showing that the duration of diabetes of over ten years did affect BAEP results[13].

We demonstrated that in T2DM patients with poorly-controlled glycemia (HbA1c >7%) the hearing threshold was statistically significantly higher in both ears at 8,000 Hz and at 2,000 Hz in the right ear, in comparison to the patients with well-controlled glycemia. Our study confirmed the results obtained by some other authors[2,15,17]. Considering the fact that HbA1c indicates the average glucose control over the previous 2-3 months, these findings may be the result of a cumulative effect of the end prod-

ucts of glucose on the inner ear[15].

Experimental studies noted changes in outer hair cells due to hyperglycemia[26]. Some authors did not establish any correlation between the control of glycemia in T2DM patients on the one hand and hearing loss[3,7,25,27], and BAEP results on the other[9]. We observed a statistically significant difference in absolute latencies for wave V for the right ear between the subgroup with well-controlled glycemia and that with poorly-controlled glycemia. Sharma et al[13] reported that the control of glycemia in T2DM patients affected BAEP results.

Conclusion

T2DM patients displayed a statistically significantly higher hearing threshold in both ears at 1,000 Hz, 2,000 Hz, 4,000 Hz, and 8,000 Hz. A statistically significant difference was observed in the absolute latencies in both ears for waves I, III and V, as well as in I–V inter-wave latencies, caused by prolonged I–III inter-wave latencies. There was also a statistically significant difference in the presence of TEOAE in both ears.

In T2DM patients with poorly-controlled glycemia (HbA1c >7%), the hearing threshold was significantly higher at 8,000 Hz in both ears, and at 2,000 Hz in the right ear only. A prolonged absolute latency for wave V was also noted in the right ear in this subgroup of patients.

Our study did not establish that the duration of diabetes had a significant impact on hearing thresholds, absolute and inter-wave BAEP latencies.

Conflict of Interest

The authors declare no conflict of interest.

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