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Distortion Product Otoacoustic Emissions (DPOAE) patterns in individuals with Type 1 and Type 2 Diabetes Mellitus - A comparative study

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Abstract

Diabetes mellitus (DM) is a chronic and potentially life-threatening condition, incidence of which is increasing rapidly in the present era. Various studies showed conflicting relationship between DM and Hearing Loss (HL). The current study was carried out to find the effect of Type 1 and Type 2 DM on hearing. The study group consisted of 90 adults in the age range of 20 to 40 years from various hospitals in and around Calicut, Kerala. These participants were divided into three groups: Group1 (Experimental group 1), included 30 individuals with Type 1 DM; Group 2 (Experimental group 2), included 30 individuals with Type 2 DM and Group 3 (Control group) included 30 age-matched non-diabetic individuals with normal hearing sensitivity. Results of the study revealed that there was significant difference in the DPOAE amplitude between Type 1 and Type 2 DM with control group and there was no significant difference in the DPOAE amplitude between Type 1 and Type 2 DM group. From the results it can be concluded that DPOAE amplitude were reduced in both Type 1 and Type 2 DM when compared to control group. This could be attributed to damage of the cochlear Outer Hair Cells (OHCs). Further, it could be assumed that damage to the OHCs due to DM in both Type 1 and Type 2 groups are relatively equal.

Keywords: Hearing Loss; Diabetes Mellitus; Otoacoustic Emission; Signal to Noise Ratio

1. Introduction

DM is a group of metabolic diseases of middle age and older adults worldwide (about 7% and rising) in whom a person has high blood sugar, either because the pancreas does not produce enough insulin or because cells do not respond to the insulin that is produced [1]. It is the most common non-communicable disease globally. Estimated number of adults with diabetes in 2007 was 246 million. Of these, 80% live in developing countries, the largest numbers in the Indian subcontinent and China. Approximately 85–95% of all diagnosed cases of diabetes are Type 2 diabetes (non-insulin dependent). India has 41 million diabetics and this number is expected to increase to 70 million by 2025 [2].

The key differences between Type 1 and Type 2 are that the former affects 5% - 10% of individuals and is usually manifested at a younger age and lasts a lifetime. While Type 2 diabetes affects about 90% - 95% of individuals and the disease generally occurs at adulthood. In Type 1 diabetes, the body attacks cells in pancreas which leads to non-production of insulin. Reasons for Type 2 diabetes can be inherited genetics or environmental factors. The symptoms for Type 1 arise more quickly. Management of type 1 is by using insulin to control blood sugar levels. In contrast, in Type 2 diabetes, the body is unable to make enough insulin or the insulin the body makes does not work correctly. Type

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2 symptoms are easier to miss as they appear gradually. Management of Type 2 diabetes can be in more ways than Type 1. This can be via medication, exercise and proper diet.

Relationship between diabetes and Hearing Impairment (HI) was first suggested in 1857 in a person who was in the early stages of diabetic coma. Patients with DM often show dizziness, tinnitus and HI. Diabetes may contribute to HL by damaging blood vessels in the ears. More rarely, HL can result from damage to the auditory part of the brain.

Diabetic influence on Sensorineural Hearing Loss (SNHL) has been a topic of study for over a century. Numerous authors agree upon the fact that DM leads to SNHL [3]. Temporal bone studies on diabetic animal models have revealed thickening of basement membranes of capillaries [4], damage of cochlear Outer Hair Cells (OHCs) [5, 6], loss of Inner Hair Cells (IHCs) [6], neuroganglion cell degeneration [7], edematous variations of the intermediate cells and atrophy in marginal cells in the stria vascularis [6, 8].

However, in an epidemiology of HL study, a community-based investigation conducted in the USA, detected a 40% increased rate of impaired hearing among adults with diabetes compared to people without the condition [11]. The typical HL described is a progressive, bilateral, SN deafness of gradual onset which affects predominantly the higher frequencies and older patients [12].

Studies that have attempted to characterize HL in diabetics showed conflicting results. Specifically, some studies found bilateral HL at high frequencies for children with Type 1 DM [13], correlation was found for metabolic control and duration of DM [13] or found HL in middle age and older Type 2 DM patients [14, 15]. In contrast, another study found no effect on hearing for either type of diabetes and evoked Otoacoustic Emissions (OAEs) were not affected at most frequencies in Type 2 DM subjects. However, other researchers found a significant drop in OAE amplitudes for diabetic subjects [16]. DM can cause mild SNHL which correlated with age and duration of disease [17]. Testing the insulin dependent DM patients by using Transient Evoked (TE) OAE and Distortion Product (DP) OAE found a significant decrease in mean amplitude which suggested the presence of cochlear disorders in diabetic patients, probably produced by impaired functional properties of the OHCs [18]. Another study evaluated the relationship between diabetes and SNHL. The study compared 44 insulin-dependent diabetics with 38 age and gender matched controls. All underwent puretone and speech audiometry, with any diabetics showing SN deafness underwent stapedial reflex (SR) tests. In 14 diabetics, SR tests showed no tone decay in any patient but seven showed evidences of recruitment. Analysis showed the diabetics to be significantly hearing impaired than the control population. The HL affected high frequencies in both genders but also low frequencies in the male. Speech discrimination scores showed no differences. Analysis of the audiograms indicated mostly a high tone loss. They added that duration of diabetes, insulin dosage and family history of diabetes were not found to have significant effect on threshold [19].

Additionally, a variable high frequency HL was noted for those who had poor control of blood sugar levels when compared with well controlled diabetic history [21]. No correlation of hearing threshold and diabetic duration was found. The effects of diabetes on cochlear elements in human beings were evaluated. Twenty-six temporal bones (mean age, 37.5 years) with Type 1 diabetes and 30 age-matched controls were examined by light microscopy. They compared the findings of cochlear vessels, hair cells, spiral ganglion cells, and cochlear lateral walls. According to the results, in diabetics, the walls of vessels of the basilar membrane and vessels of the stria vascularis were significantly thicker in all turns and loss of OHC was significantly greater in the lower basal turn. Atrophy of the stria vascularis in all turns and loss of spiral ligament cells in upper turns were significantly higher than controls. No significant difference was obtained in the number of spiral ganglion cells between groups. The study suggested that Type 1 DM can cause cochlear microangiopathy and subsequently degeneration of cochlear lateral walls and OHCs [22].

In a study to evaluate the cochlear function in Type 1 DM, researchers analyzed OAEs on normal hearing subjects with diabetes and on controls. All patients underwent OAE analysis and brainstem evoked potentials. Fifty-eight normal volunteers were used as controls for the OAE analysis. Seventeen patients (28.3%) had no OAEs in at least one ear and 10% in both ears. The mean intensity of the response was lower in diabetic subjects than in controls. The cochlear impairment was over 5 decibel (dB) for the 1- kilo Hertz (kHz) frequency, which is the critical level for speech understanding. These findings suggested that cochleopathy can be detected in a relatively high proportion of subjects with Type 1 diabetes in spite of a normal audiometric hearing threshold [23].

The functional properties of the cochlea in subjects with Type 1 (insulin dependent) DM were studied. TEOAEs were measured in 21 normal hearing, well controlled, Type 1 diabetic patients, aged 23–42 years and in an age and gender matched healthy, non-diabetic, normally hearing control subjects. Mean TEOAE amplitude was found to be significantly reduced in the diabetic patients compared with those of the control group. It was also found that the most significant reduction in the response was in the group of patients with diabetic microangiopathy but those who had diabetic

neuropathy exhibited similar reduction in the TEOAE response to the group of patients with uncomplicated diabetes [24].

In a study, OAEs were assessed in 50 Type 2 diabetic and non-diabetic patients, 40–50 years of age. Disordered OAEs were recorded in at least one ear in 16% of cases; however, the findings revealed no significant difference in OAEs between type 2 diabetics and nondiabetics [25].

A study investigated auditory dysfunctions in type 2 DM patients with poor versus good glycemic control. TEOAEs and DPOAEs were measured. Patients with poor glycemic control had significantly lower amplitudes at all frequencies in the TEOAE test, as well as at high frequencies (4 and 6 kHz) in the DPOAE test when compared with other two groups [26].

A study to assess impact of duration of Type 2 diabetes and control of glycemia on the auditory function was carried out. A statistically significant difference was noted in TEOAE in Type 2 diabetic patients with poor glycemic control [27].

From the above studies, it is very clear that researchers have studied effect of Type 1 and Type 2 Diabetes on hearing function including OAEs. However, very few studies have compared OAE findings in Type 1 and Type 2 DM patients. This comparison is very much sparse in the Indian context. Considering the huge proportion of patients with DM in India, it is important to compare and understand cochlear function between Type 1 and Type 2 DM patients. Hence this study was conducted.

2. Material and methods

2.1. Subjects

The study group consisted of 90 adults in the age range of 20 to 40 years. These participants were divided into three groups:

- Group1 (Experimental group): This group consisted of 30 individuals with Type 1 DM.
- Group 2 (Experimental group): This group consisted of 30 individuals with Type 2 DM.
- Group 3 (Control group): This group consisted of 30 age matched non-diabetic individuals with normal hearing sensitivity.

2.1.1. Inclusion and exclusion criteria for experimental group

- Should not have history of any other medical, otologic, psychiatric and neurological disorders.
- Should not have exposure to any other source of noise (occupational/other noise).
- Should have puretone threshold within 25 decibel Hearing Level (dB HL) from 250-8000 Hertz (Hz).
- Should have “A” type tympanogram.
- Patients diagnosed as Type 1 or Type 2 DM by general physician.
- Under medication for diabetes about 5-10 years duration.

2.1.2. Inclusion and exclusion criteria for control group

- Should have puretone thresholds within 25 dB HL from 250 to 8000 Hz.
- Should have A type tympanogram and presence of Acoustic Reflexes (ipsilateral and contralateral) in both ears.
- Should not have any history of DM.
- Should not have any medical/otologic/psychiatric/neurological complaints.

2.2. Test procedure

Otoscopic examination was administered to rule out presence of cerumen, middle ear fluid and perforation of Tympanic Membrane.

2.2.1. Puretone audiometry

The air and bone conduction thresholds were recorded for puretones using a 2 channel clinical audiometer (Grason-Stadler Incorporates-GSI-61) for frequencies ranging from 250 Hz to 8000 Hz using supra aural headphone TDH 49 and B-71 bone vibrator. Modified Hughson and Westlake procedure was followed.

2.2.2. Immittance audiometry

Tympanometry and reflexometry were done using Grason-Stadler Incorporates (GSI), Tympstar). The tympanogram pattern, ear canal volume, static compliance and peak pressure were noted. The ipsilateral and contralateral acoustic reflexes were measured for frequencies 500 HZ, 1000 HZ, 2000 Hz, and 4000 Hz.

2.2.3. Otoacoustic emissions (OAE)

Following confirmation of normal hearing thresholds and middle ear status, DPOAEs were recorded using a sensitive microphone. The range of frequencies being tested were set between 500 Hz to 8000 Hz. GSI Audera was used for recording OAEs. Frequency (F)2/F1 ratio of 1.22 was considered. Signal/Noise Ratio (SNR) of minimum +6 dB was considered as normal response. All these measurements were carried out in a sound treated room with ambient noise level within the permissible limit as recommended (American National Standards Institute (ANSI) S3.1 1999).

2.3. Test environment

Prior to testing, a standard consent form was read and signed by each participant. The test room had comfortable lighting and temperature. The time required for all audiological investigations for each subject approximated around 45 minutes. The patients were given sufficient rest periods between tests to facilitate maximum comfort and cooperation. Institutional Ethics Committee approved the protocol for the study.

2.4. Statistical analysis

The obtained data were subjected to statistical analysis; Analysis of Variance (ANOVA) was used to find out the mean and standard deviation of DP SNR across frequencies between Type 1 DM with control group, Type 2 DM with control group and between Type 1 and Type 2 DM. All statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) version 16 software.

3. Results

ANOVA was used to compare the DPOAE SNR in control and experimental group (Type 1 DM) and the mean and Standard Deviation (SD) obtained is shown in Table 1. ANOVA showed that there is significant difference between the scores obtained for the control and Type I DM at frequencies 500 Hz, 1 KHz, 2 KHz, 4 KHz and 8 KHz (Table 2).

Table 1 Mean and standard deviation of DPOAE SNR for Type 1 DM & control group

	Type 1 DM		**Control group**	
	Mean	**SD**	**Mean**	**SD**
500 HZ	10.2143	2.84614	12.5357	2.98741
I KHZ	13.1071	3.97529	15.4643	6.07656
2KHZ	11.1071	3.93784	14.8929	5.94007
4 KHZ	6.7500	3.47078	14.2143	5.56016
8 KHZ	3.3571	2.75162	12.7500	5.81585

Table 2 Significance of difference for mean DPOAE SNR between Type 1 DM and control group

Frequencies	**Control group versus Type 1 DM**		
	Sig	F	Df
500 HZ	0.000	23.179	1
I KHZ	0.038	4.514	1
2KHZ	0.032	4.852	1
4 KHZ	0.007	7.777	1
8 KHZ	0.000	20.182	1

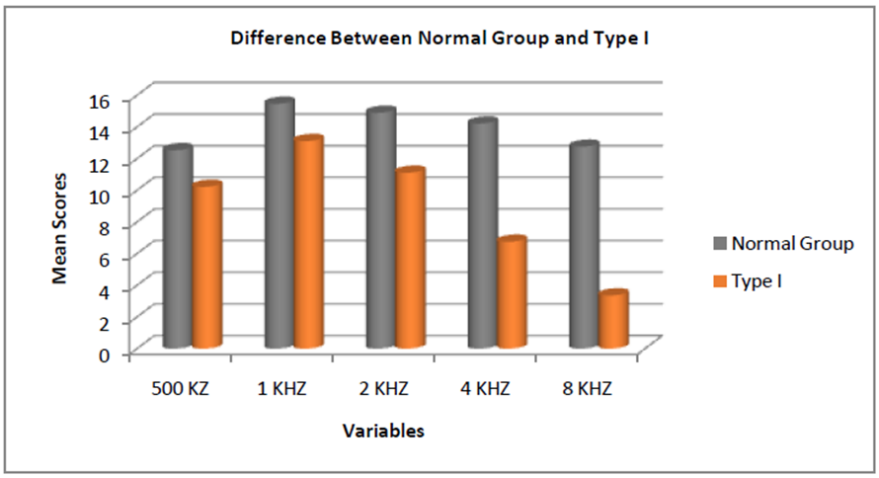

Figure 1 Graphical illustration of DPOAE Signal to Noise Ratio (SNR) mean values for Type 1 DM and control group

To compare the DPOAE SNR in control and experimental group (Type 2 DM), ANOVA was used. The mean and SD of DPOAE SNR obtained are shown in Table 3. ANOVA showed that there is significant difference between the scores obtained for the control and experimental group at 500Hz, 1 KHz, 2 KHz, 4 KHz and 8 KHz frequencies (Table 3).

Table 3 Mean and SD of DPOAE SNR for Type 2 DM & control group

	Type 2 DM		Control Group	
	Mean	**SD**	**Mean**	**SD**
500 HZ	10.6429	5.29300	12.5357	2.98741
I KHZ	14.1786	4.01897	15.4643	6.07656
2KHZ	11.0357	3.13265	14.8929	5.94007
4 KHZ	5.5714	3.51113	14.2143	5.56016
8 KHZ	0.5357	4.15872	12.7500	5.81585

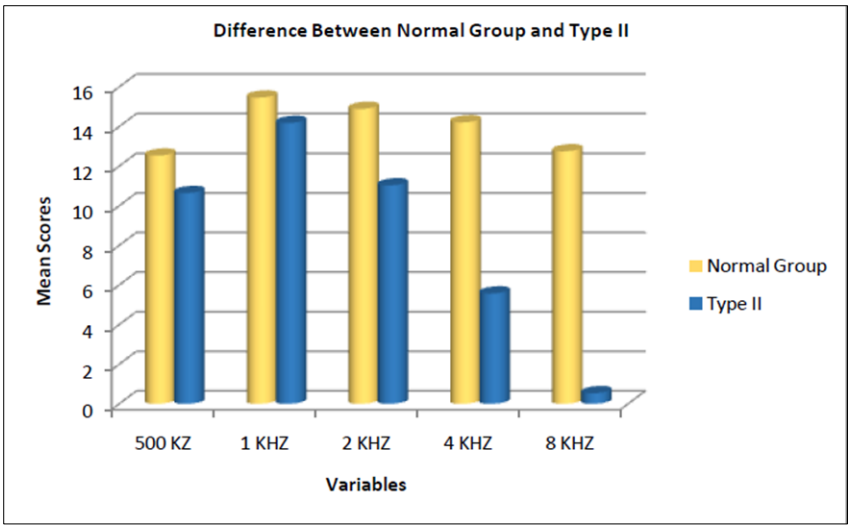

Figure 2 Graphical illustration of mean values of DPOAE SNR for Type 2 DM and control group

Table 4 Significance of difference for mean DPOAE SNR between Type 2 DM and control group

Frequencies	Control group versus Type 2 DM		
	Sig	F	Df
500 HZ	0.000	17.756	1
I KHZ	0.001	13.443	1
2KHZ	0.003	9.322	1
4 KHZ	0.003	9.472	1
8 KHZ	0.000	23.678	1

3.1. Comparison of DPOAE SNR in individuals with Type 1and Type 2 DM

ANOVA was used to compare the DPOAE SNR between Type 1 and Type 2 DM groups. The mean and SD obtained are shown in Table 5. A comparison of Type 1 and Type 2 DM DPOAE SNR with ANOVA revealed no significant difference ($p > 0.05$) between both groups.

Table 5 Mean and SD of DPOAE SNR for Type 1 and Type 2 DM

	Type 1 DM		Type 2 DM	
	Mean	SD	Mean	SD
500 HZ	12.2143	2.84614	13.6429	5.29300
I KHZ	13.1071	3.97529	14.1786	4.01897
2KHZ	11.1071	3.93784	11.0357	3.13265
4 KHZ	6.7500	3.47078	5.5714	3.51113
8 KHZ	3.3571	2.75162	0.5357	4.15872

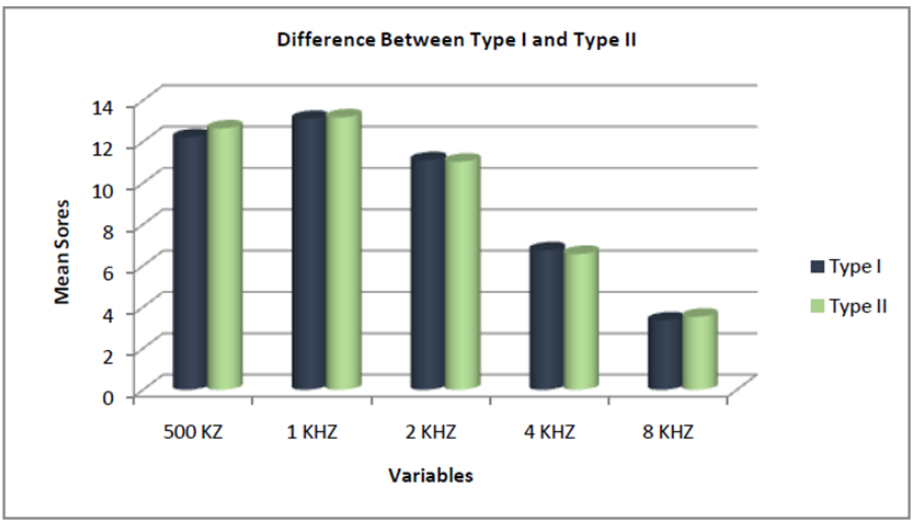

Figure 3 Graphical illustration of mean values of DPOAE SNR for Type 1 and Type 2 DM

Table 6 Significance of difference of DPOAE SNR between Type 1 and Type 2 DM

Frequencies	Type 1 & Type 2 DM		
	Sig	F	Df
500 HZ	0.460	0.552	1
I KHZ	0.068	3.455	1
2KHZ	0.472	0.523	1
4 KHZ	0.433	0.624	1
8 KHZ	0.146	2.170	1

4. Discussion

Diabetes and Hearing Loss was our topic of study and its effects on inner ear mechanisms were of great interest. The present study compared Type 1 and Type 2 diabetes to better understand if any difference exists on its influence on the inner ear structures and was assessed via OAE's. Results of the present study revealed that there was a significant difference in the DPOAE SNR for Type 1 DM with control group which denotes the impaired functional properties of the cochlear OHCs. More reduction was seen at high frequencies specifically at 4 KHz and 8 KHz. These results are in agreement with the previous literature, Tay, H. L., Ray, N., Ohri, R.,et.al (1995).

Similar findings were obtained when Type 2 DM and control group were compared. Impaired functional properties of OHCs in Type 2 DM were evident. The study by Park, Park and Choi 2001, is in agreement with the results of the present study [28]. Eren and colleagues 2014, study on forty patients (32 to 76 years) with Type 2 DM showed same results as the current study [30].

From the present study, it is evident that the DPOAE amplitude reduction in both experimental groups (Type 1, Type 2 DM) is comparatively equal. Thus, it could be assumed that damage to the OHCs due to DM in both Type 1 and Type 2 are relatively equal in proportion which probably suggests similar mechanisms and extent of cochlear impairment in both groups.

Considering the social impact of this disease condition, assessment of cochlear Outer Hair Cell function between Type 1 and Type 2 diabetic patients by measurement of DPOAEs was worth investigating. The results were unambiguous and it did not reveal any significant difference of DPOAE between Type 1 and Type 2 DM patients. Considering the dearth of literature on this particular aspect, results of the study could be utilized by researchers for future investigations.

5. Conclusion

Significant difference was observed in amplitude of OAE's obtained from Type 1 and Type 2 DM patients particularly in the higher frequency region when comparted to control group. No noteworthy difference in amplitude values were detected between the two experimental groups. Thus, we can conclude that the use of OAE's are helpful in understanding the changes occurring to OHC's in the inner ear in diabetic patients.

Limitations

- Sample size was relatively small.
- Subject heterogeneity in terms of onset of DM and medications for DM.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest is being reported.

Statement of informed consent

Informed consent was obtained from all participants before the commencement of the study.

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