RMJ Ramathibodi Medical Journal

Rama Med J | Original Article

Comparison of Otoacoustic Emissions in Asymptomatic Diabetes Mellitus and Normal Hearing Control Groups

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Background: Diabetes mellitus (DM) is a chronic metabolic disorder. Previous studies reported the hearing impairment of diabetes patients as a progressive bilateral symmetrical sensorineural hearing loss (SNHL) of gradual onset which predominantly affected the higher frequencies, and found that diabetic patients had unsatisfactory hearing thresholds than nondiabetics in the same age group. For the use of evoked otoacoustic emissions (EOAEs) as a clinical test of hearing, EOAEs are sound which can be recorded in the outer ear canal and give objective information about preneural, mechanical elements of the cochlear function.

Objective: To compare the otoacoustic emissions of asymptomatic DM patients with a normal hearing control group

Methods: All subjects received a routine audiologic evaluation which consisted of pure-tone air-bone conduction tests, tympanograms, and stapedius acoustic reflex tests. OAEs were measured in 30 asymptomatic diabetic patients with a mean age of 55.73 years and an age-matched 30 subjects normal hearing control group with a mean age of 50.87 years.

Results: The pure-tone thresholds at 3, 4, 6, and 8 kHz; transient-evoked otoacoustic emissions (TEOAEs) amplitudes at 1, 1.5, 2, and 3 kHz; and the distortion product otoacoustic emissions (DPOAEs) amplitudes at 1, 1.5, 2, 3, 4, and 6 kHz were significantly different ($P < .05$) between the asymptomatic and control groups.

Conclusions: This study suggested that diabetic patients had a significant hearing impairment. SNHL is gradually progressive and is a common condition in DM patients and their thresholds for hearing are higher at higher frequencies.

Keywords: Diabetes mellitus, Otoacoustic emissions, Sensorineural hearing loss

Rama Med J: doi:10.33165/rmj.2019.42.2.139031

Received: August 21, 2018 **Revised:** May 14, 2019 **Accepted:** May 27, 2019

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Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by hyperglycemia.[1] Many complications are associated with DM, including nephropathy, angiopathy, retinopathy, and peripheral neuropathy.[2] There have been various reports pertaining to the hearing impairment of diabetes patients such as progressive bilateral symmetrical sensorineural hearing loss (SNHL) of gradual onset which predominantly affected their response to higher frequencies.[3-5] Although a decrease in auditory acuity appeared to be similar to that caused by presbycusis, the hearing impairment of an affected diabetic showed a hearing loss of greater degree than could be expected at that age.[4] Axelsson et al[3] concluded that the older the age of diabetes patients, the more likely they will have a hearing impairment.

The relationship between diabetes and hearing thresholds deserves special attention. Despite a number of studies conducted on the hearing function of diabetic patients having well-controlled treatment plans, conflicting data still exist relative to a possible association between bilateral progressive high frequency hearing loss and diabetes. Several researchers reported a higher incidence of hearing loss in diabetic patients in comparison with the general population whereas no differences in pure-tone audiometry (PTA) results between diabetic patients and their controls were reported in other studies.[6] Patients with severe peripheral neuropathy or retinopathy seem to have an increased risk of hearing loss[7] although no association between duration or severity of diabetes and hearing impairment has been reported.[6, 8] In addition, poor metabolic control[6, 8] and hypoglycemic episodes did not show an association with increased hearing thresholds.[9-11]

Evoked otoacoustic emissions (EOAEs) are sounds which can be recorded in the outer ear canal and provide objective information about preneural, mechanical elements of the cochlear function. EOAEs are believed to originate from a healthy cochlea and appear to be a general property of the human peripheral auditory system. The presence of EOAEs or distortion product otoacoustic emissions (DPOAEs) indicate a healthy cochlear function. On the other hand, their absence may indicate by outer or middle ear problems or cochlear hearing loss. Elevated hearing thresholds higher than 45 dB hearing level (HL) have been reported to be associated with the absence of OAEs. Simoncelli et al[12] reported on the cochlear function of 20 diabetic patients with normal hearing by using EOAEs. These data were compared with the data obtained from a group of nondiabetic control subjects with normal hearing. The mean EOAE intensity and amplitude in the 100 Hz frequency band were significantly lower for diabetic patients than subjects in the control group. Also, mean EOAE amplitude was reported to be significantly reduced in diabetic patients with a lower nerve conduction velocity (NCV), but not for those without neuropathy when compared with a control group.[13]

However, there are other disorders which also have been reported as having some degree of abnormal effect on the hearing function such as hyperlipidemia, smoking, and hypertension.[14, 15] The hearing thresholds, tympanograms, and OAEs of these diabetic patients were evaluated in order to compare their audiometric responses with those of a normal hearing control group.

Methods

Subjects

In the present study, diabetic patients were selected who did not have other coexistent diseases, especially smoking, hypertension, and hyperlipidemia, in order to form an uncontaminated diabetic group. The control group of 30 subjects, with ages between 15 - 75 years, had normal hearing thresholds based on audiometric measurement. The other group of 30 DM patients did not have complaints about hearing-related symptoms. These asymptomatic patients were selected from the outpatient Endocrine Clinic at Ramathibodi Hospital. All subjects provided written informed consent. The

DM patients had been evaluated by a physician based on: fasting (overnight), a venous plasma glucose concentration ≥ 7.8 mmol/L (140 mg/dL) on at least 2 separate occasions, or a venous plasma glucose concentration of ≥ 11.1 mmol/L (200 mg/dL) at 2 hours following an ingestion of 75 g of glucose.

Exclusion criteria were the following: a history of blood pressure higher than 140/90 mmHg, or a serum cholesterol level higher than 300 mg/dL, or a triglycerides level higher than 172 mg/dL for more than 3 years and needed medical treatment;[16] being treated with an anticonvulsant, methyldopa, nitrofurantoin, reserpine or any medications that might be expected to interfere with central nervous system function; heavy smoking, > 1 pack/day for more than 3 years;[15] head injury and brain surgery; ear disease; temporary hearing loss; occupational exposure to excessive noise; and hereditary deafness in their family, poorly cooperative patients.

Procedure

After an interview to compile their medical history and have a physical examination, all subjects received a routine audiological evaluation which consisted of pure-tone air-bone conduction tests (at the octave frequency range of 0.25 - 8 kHz), tympanograms, and stapedius acoustic reflex testing (ipsilaterally at 0.5 - 2 kHz and contralaterally at 0.5 - 4 kHz).

OAEs Measurement Procedure

Transient-evoked otoacoustic emissions (TEOAEs) were recorded with half-octave spectrum analysis and were analyzed using the method proposed by Bray.[17]

DPOAEs were recorded by means of a calibrated ear canal probe containing 2 ports for the inserted earphones and a sensitive low-noise microphone system. The probe was sealed into the ear with a foam ear tip. The parameters and technique used in recording were set according to Hauser et[18] and Sum et al.[19] Both TEOAEs and DPOAEs were recorded by an ILO 292 II Otodynamic Analyzer (Otodynamics is an ISO9001 and ISO13485 complaint company) with appropriate software and hardware.

The TEOAEs and DPOAEs audiograms from each ear were recorded without removal of the probe. Noise present during all measurements were recorded and monitored to the determine if the noise level was below the satisfactory level of 45 dB sound pressure level (SPL).[20]

Criteria Used to Ensure a Valid Recording of TEOAEs and DPOAEs

The criteria were as follows: TEOAEs, the response should be seen as a clear area above the noise floor and generally 6 dB higher than the noise level.[21] The presence of well-defined, closely superimposed A & B waveforms of more than 70%.[21] A click stimulus waveform without excessive oscillation (depends on proper probe fitting) and a relatively flat click stimulus spectrum from 1 - 5 kHz.[20] DPOAEs, there were considered to be present when the amplitudes were higher than 6 dB above the noise floor.[22]

Statistical Analysis

A statistical package, SPSS version 18 (PASW Statistics for Windows, Version 18.0. Chicago: SPSS Inc; 2009), was used to compute means and standard deviations (SD) for the analysis of the pure-tone thresholds, the TEOAEs amplitudes, and DPOAEs amplitudes. A chi-square test was used to determine if the difference in the percentages of OAEs responses between normal subjects and diabetic patients was significant. A *t* test was used to compare the means of the pure-tone thresholds and, the amplitudes of TEOAEs and DPOAEs, between the normal control group and the diabetic patient group, and to compare the mean ages of both groups. For all statistical analyses, the .05 level of significance was used.

Ethics

This study was approved by the Committee on Human Rights Related to Research Involving Human Subjects, Faculty of Medicine Ramathibodi, Mahidol University on September 14, 2007. The certificate of approval number is MURA2007/341$N_1$$Aug_{18}$.

Results

The mean and SD (range) age of subjects were 50.87 ± 9.77 (32 - 65) years for the control group and 55.73 ± 10.53 (34 - 73) years for the diabetic patient group. The difference in age between the case and control group was not significant (*P* = .06).

The pure-tone air-conduction thresholds of both ears in control group (60 ears) and diabetic (60 ears) group were calculated at a frequency ranging from 0.25 to 8 kHz (Table 1). There were nonsignificant differences in pure-tone thresholds at 0.25, 0.5, 1, and 2 kHz between the control and diabetic groups, although the pure-tone thresholds at these 4 frequencies were higher for DM subjects than control subjects.

This study compared hearing abilities between an asymptomatic DM group and a control group. There was a nonsignificant difference in age between the asymptomatic DM and control groups. The control group ages ranged from 32 to 65 years. Eight subjects showed hearing impairment at high frequencies (12 of 60 ears, 20%). All of these 8 subjects had a mild degree of hearing loss only at the high frequencies of 3, 4 kHz (1 ear), 6 and/or 8 kHz (11 ears). Three subjects had bilateral symmetrical hearing loss and 5 subjects had unilateral hearing loss.

There was nonsignificant difference in TEOAEs amplitudes at 4 kHz between both groups (Table 2).

Table 1. Comparison of Pure-Tone Air-Conduction Thresholds Between the Control and DM Groups

Frequency, kHz	Mean ± SD		*P* Value
	Control Group	DM Group	
0.25	13.92 ± 7.20	15.08 ± 7.51	.38
0.5	15.33 ± 6.24	15.92 ± 6.14	.60
1	14.83 ± 5.60	16.67 ± 6.62	.10
2	12.17 ± 5.78	14.92 ± 9.50	.05
3	15.00 ± 6.77	18.88 ± 10.39	.01
4	14.17 ± 8.74	20.33 ± 12.48	.002
6	20.50 ± 8.22	26.90 ± 13.47	.003
8	15.92 ± 12.30	25.58 ± 15.79	< .001

Abbreviations: DM, diabetes mellitus; SD, standard deviation.

Table 2. Comparison of the TEOAEs Amplitudes (dB SPL) at 1 - 4 kHz of Control and DM Groups

Frequency, kHz	Mean ± SD (No. of Ears)		*P* Value
	Control Group	DM Group	
1	12.31 ± 6.28 (57)	8.25 ± 5.08 (50)	< .001
1.5	17.71 ± 5.68 (60)	12.11 ± 6.35 (59)	< .001
2	16.99 ± 3.77 (59)	12.14 ± 6.53 (56)	< .001
3	13.26 ± 4.90 (58)	10.80 ± 5.84 (53)	.01
4	11.71 ± 6.22 (59)	10.53 ± 6.18 (47)	.33

Abbreviations: DM, diabetes mellitus; SD, standard deviation; SPL, sound pressure level; TEOAEs, transient-evoked otoacoustic emissions.

There were statistically significant differences in all frequencies of DPOAEs amplitudes between the control and diabetic groups (Table 3).

The percentages of TEOAEs, DPOAEs and, overall (TEOAEs and DPOAEs) responses were compared. For the control group, the percentages were 89.33%, 88.89%, and 89.09%, respectively. For the DM group, the percentages were 70.33%, 70.83%, and 70.61%, respectively. The analyses clearly showed that the differences in the percentages of TEOAEs, DPOAEs, and overall (TEOAEs and DPOAEs) responses between the control and diabetic groups were significant (Table 4).

Discussion

The pure-tone air-conduction findings indicated that the control and diabetic groups showed some degree of hearing loss relative to the different types of hearing loss: symmetrical, asymmetrical, or unilateral. However, the DM group showed a more severe degree of hearing loss over a broader range of frequencies than the control group.

Table 3. Comparison of DPOAEs Amplitudes (dB SPL) at 1 - 6 kHz of Control and DM Groups

Frequency, kHz	Mean ± SD (No. of Ears)		*P* Value
	Control Group	DM Group	
1	10.25 ± 4.33 (54)	7.69 ± 5.22 (41)	.01
1.5	15.01 ± 5.75 (60)	11.48 ± 4.93 (58)	.001
2	16.17 ± 6.67 (60)	11.40 ± 4.84 (57)	< .001
3	14.74 ± 5.09 (57)	11.30 ± 5.38 (50)	.001
4	14.63 ± 5.89 (60)	11.60 ± 7.59 (53)	.01
6	16.64 ± 6.59 (55)	12.73 ± 8.43 (47)	.01

Abbreviations: DM, diabetes mellitus; DPOAEs, distortion product otoacoustic emissions; SD, standard deviation; SPL, sound pressure level.

Table 4. Comparison of the Percentages of OAE Responses of Control and DM Groups

OAEs	No./Total No. (%)		*P* Value
	Control Group	DM Group	
TEOAEs			
Response	268/300 (89.33)	211/300 (70.33)	< .001
Absent	32/300 (10.67)	89/300 (29.67)	
DPOAEs			
Response	320/360 (88.89)	255/360 (70.83)	< .001
Absent	40/360 (11.11)	105/360 (29.17)	
Overall (TEOAEs + DPOAEs)			
Response	588/660 (89.09)	466/660 (70.61)	< .001
Absent	72/660 (10.91)	194/660 (29.39)	

Abbreviations: DM, diabetes mellitus; DPOAEs, distortion product otoacoustic emissions; OAE, otoacoustic emission; SD, standard deviation; TEOAEs, transient-evoked otoacoustic emissions.

These findings agreed with those of Edgar[23] in 1915, who was the first researcher to report a high frequency sensorineural hearing loss in a diabetic patient. In addition, Cullen et al[24] in 1993 reported that the hearing loss of diabetic patients was more common at higher frequencies.

In this study, the mean pure-tone thresholds from 3 - 8 kHz of the control subjects, were significantly lower than those of the asymptomatic DM group. These findings agreed with many other studies[5, 10, 24] which reported that DM subjects had higher pure-tone thresholds than control subjects, and hearing loss in the DM group was more pronounced at high frequencies (4 - 8 kHz).[24] Hearing loss in DM patients may be present to a silent or mild degree. Symmetrical or unilateral high frequency hearing loss in asymptomatic DM patients can occur without hearing symptoms prior to the beginning of more progressive hearing impairment in one or both ears. Weng et al[25] in 2005 reported on sudden onset SNHL in 68 diabetic patients. Sudden or progressive changes in hearing thresholds, especially in speech frequency (0.5 - 4 kHz), can cause DM patients to be aware of their hearing acuity.[26]

This study showed that the percentage of overall OAEs in the DM group (70.61%) was significantly lower than in the control group (89.09%) ($P < .001$). Since EOAE recording is a measurement of the active process of cochlear function, especially of the outer hair cells (OHCs), this implies that the active cochlear function, or OHCs activity in DM patients, may be impaired. This impairment has been postulated as being caused by metabolic, structural changes, or microangiopathy involving the inner ear of DM patients.[12, 13, 27]

TEOAEs spectral amplitudes of the DM group were significantly lower than those of the control group from 1 - 3 kHz. These findings agreed with others[12, 13, 27] reporting TEOAEs amplitudes at frequencies from 1 - 4 kHz were significantly reduced in diabetic patients. Also, these findings of the present study suggested that the presence of an alteration in cochlear micromechanics of diabetic patients was possibly caused by damage of the microvasculature of the diabetic cochlea, which might lead to a loss of OHCs or a change in the functioning of hair cells. Reduced hair cell function in the cochlea of DM patients may result from the same disease processes such as metabolic disturbance, or structural changes in the organ of corti, or microangiopathy of the inner ear.[5]

However, a nonsignificant difference in the mean TEOAEs amplitudes between the control and DM groups was found at 4 kHz. The mean TEOAEs amplitude at 4 kHz of the DM group was actually lower than the mean of the control group in according with a lower power spectrum of click stimulus at 4 kHz.

The mean DPOAEs amplitudes at 1 - 6 kHz of the diabetic group of this study were significantly lower than those of the control group, which agreed with Di Nardo et al[27] reporting a significant difference between control subjects and insulin-dependent DM patients with neuropathy at middle and high frequencies. Many researchers agreed that DPOAEs and TEOAEs are generated from the same active process in the cochlea.[12, 13, 27] DPOAEs were diminished or absent when the primary-tone frequencies originated from a damaged cochlear area.[28-30] Thus, lower DPOAEs amplitudes also indicated unsatisfactory hair cell function in DM subjects.

Conclusions

This study suggested that the presence of SNHL is a complication of DM. Diabetic patients had a significant hearing impairment. SNHL is gradually progressive and is condition in DM patients, and their hearing thresholds are higher at higher frequencies. The results of this study might be used as a guideline in using OAEs for hearing evaluation in diabetic patients.

Acknowledgements

This research was supported by grant from Faculty of Medicine Ramathibodi Hospital, Mahidol University, Thailand.

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การศึกษาเปรียบเทียบผลการตรวจวัดเสียงสะท้อนในหูชั้นในของกลุ่มผู้ป่วยเบาหวานที่ไม่มีอาการทางการได้ยิน และกลุ่มควบคุม

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บทนำ: เบาหวานเป็นโรคเรื้อรังเกิดจากความผิดปกติของระบบการเผาผลาญ มีรายงานปัญหาการสูญเสียการได้ยินในผู้ป่วยเบาหวานชนิดประสาทหูเสื่อมเท่ากันทั้งสองหูมีลักษณะค่อยเป็นค่อยไปและกระทบต่อระดับการได้ยินที่ความถี่สูง ซึ่งผู้ป่วยเบาหวานมีการสูญเสียการได้ยินในระดับรุนแรงกว่าผู้ไม่เป็นเบาหวานในกลุ่มอายุเดียวกัน การตรวจวัดเสียงสะท้อนจากหูชั้นใน (Evoked otoacoustic emissions, EOAEs) เป็นการตรวจวัดการได้ยิน เสียงที่ใช้ตรวจสามารถถูกบันทึกในช่องหูชั้นนอก ซึ่งให้ข้อมูลเกี่ยวกับกระแสประสาทและกลไกหน้าที่ของอวัยวะรูปก้นหอย

วัตถุประสงค์: เพื่อศึกษาเปรียบเทียบผลการตรวจวัดเสียงสะท้อนจากหูชั้นในของกลุ่มผู้ป่วยเบาหวานที่ไม่มีอาการทางการได้ยิน และกลุ่มควบคุม

วิธีการศึกษา: กลุ่มตัวอย่างทุกคนได้รับตรวจการได้ยินด้วยเสียงบริสุทธิ์ ตรวจสภาพหูชั้นกลาง และตรวจวัดเสียงสะท้อนจากหูชั้นใน โดยเปรียบเทียบระหว่างผู้ป่วยเบาหวานที่ไม่มีอาการ จำนวน 30 คน อายุเฉลี่ย 55.73 ปี และผู้ที่ไม่เป็นเบาหวาน จำนวน 30 คน อายุเฉลี่ย 50.87 ปี

ผลการศึกษา: การได้ยินเสียงบริสุทธิ์ที่ความถี่ 3 - 8 kHz การตรวจวัดเสียงสะท้อนจากหูชั้นในกระตุ้นด้วยเสียงคลิก (Transient-evoked otoacoustic emissions, TEOAEs) ที่ความถี่ 1, 1.5, 2, 3 kHz การตรวจวัดเสียงสะท้อนจากหูชั้นในด้วยเสียงบริสุทธิ์ 2 เสียงพร้อมกัน (Distortion product otoacoustic emissions, DPOAEs) ที่ความถี่ 1, 1.5, 2, 3, 4, 6 kHz มีความแตกต่างอย่างมีนัยสำคัญ ($P < .05$) ระหว่างผู้ป่วยเบาหวานและผู้ที่ไม่เป็นเบาหวาน

สรุป: การศึกษานี้แสดงให้เห็นว่าผู้ป่วยเบาหวานมีการสูญเสียการได้ยินอย่างมีนัยสำคัญ โดยการสูญเสียการได้ยินแบบประสาทหูเสื่อมที่พบในผู้ป่วยเบาหวานจะมีลักษณะแบบค่อยเป็นค่อยไปและแสดงการเปลี่ยนแปลงของผลตรวจวัดเสียงสะท้อนจากหูชั้นในผิดปกติก่อนจะมีอาการสูญเสียการได้ยินระดับความถี่สูง

คำสำคัญ: เบาหวาน ตรวจวัดเสียงสะท้อนจากหูชั้นใน การสูญเสียการได้ยินแบบประสาทหูเสื่อม

Rama Med J: doi:10.33165/rmj.2019.42.2.139031

Received: August 21, 2018 **Revised:** May 14, 2019 **Accepted:** May 27, 2019

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