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RESEARCH ARTICLE

Evaluation of auditory evoked response among type-II diabetic individuals in central India

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ABSTRACT

Background: The Type II diabetes mellitus (DM) is characterized by hyperglycemia, as a result of damaged beta cell function, along with increase insulin resistance, either at receptor or at postreceptor levels. Neuropathies complications are the most common in diabetes, that's may lead to delayed evoked potentials in the central pathways. **Aims and Objective:** This study was undertaken to know the hearing status of the Type II diabetic patients and to delay development of its related complication like central neuropathy. **Materials and Methods:** This study evaluates central neuropathy by brainstem evoked response audiometry (BERA) in Type II DM patients. **Results:** We observed significant differences in BERA latencies between diabetic patients and healthy controls. There was significantly delayed Wave II, IV latency in left ear (at 80 dB) and Wave II latency in left ear, as well as Wave IV latency in right ear (at 90 dB) as compared to control. Diabetics patients with and without peripheral neuropathy there were absolute delayed latencies of Wave I, III, and V (80 dB). With relation to blood glucose, a significant difference in absolute latencies of Wave II (right ear at 90 dB), Wave IV (in right ear at 90 dB), Wave V (left ear at 80 dB), and interpeak latencies I-V (in right ear at 90 band). **Conclusion:** This study suggests that BERA is a simple, non-invasive procedure to detect early impairment of acoustic nerve, and central nervous system pathways, even in the absence of specific symptoms, if BERA is carried out in diabetic patients, involvement of central neuronal axis can be detected earlier.

KEY WORDS: Brainstem Evoked Response Audiometry; Type II Diabetes Mellitus; Peripheral Neuropathy; Blood Glucose

INTRODUCTION

Diabetes mellitus (DM) is characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both.[1] Among two (Types I and II), broad categories of DM, Type II diabetes is much more common than Type I.[2]

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Type II diabetes, or non-insulin-dependent diabetes or adult-onset diabetes, encompasses individuals who have insulin resistance/insulin deficiency.[3] Although the pathogenesis of morphologic disorder is not yet confirmed in Type II diabetes, but directly related to hyperglycemia, and however there is diffused thickening in the basilar membrane of the cochlea,[4] changes in vascular endothelium along with vasoconstriction of smaller vessels in the inner ear that leads to hypoxia and may leading to hearing loss.[5-8]

Neuropathies complications are the most common in diabetes, that's may lead to delayed evoked potentials in the central pathways.[9] Diabetic patients preserve a normal hearing function until diabetic neuropathy developed; since

then the nerve degeneration, related to decrease in circulating nerve growth factor, leads to a progressive impairment of the auditory pathway with consequent hearing deficit.[10] A simple and non-invasive technique is brainstem evoked response audiometry (BERA) to find out early impairment of acoustic nerve and central nervous system (CNS) pathway, even in the absence of specific symptoms. BERA is a recording of the synchronized response of a large number of neurons in the lower portion of auditory pathway and can evaluate electrophysiologically any lesions from acoustic nerve to the brainstem to show subclinical variances and central neuropathy in diabetics.[11] The involvement of cochlear and eighth nerve has been observed for progressive sensorineural hearing loss in patients with DM.[12]

The previous studies have been demonstrated sensorineural hearing loss at the higher frequencies among DM individuals.[13-15] However, the association between DM and hearing loss has been debated up to now.[16] Hearing impairment related to diabetes has been described as sensorineural in origin, implying that the lesion may be cochlear or of the 8th cranial nerve, but evidence favoring a specific mechanism is unsatisfactory.[17]

Hence, this study was planned to evaluate auditory functions through evoked potentials in Type II DM and to evaluate the role of potentially relevant factors such as blood glucose levels and the presence of complications like peripheral neuropathy.

MATERIALS AND METHODS

Ethnic Statement

The study was approved by the local research advisory committee of L.N. Medical College and Research Centre (LNMC/Dean/2015/2146). The study was performed in accordance with the Declaration of Helsinki. Detailed written consent of all the participants was taken and the purpose of the study was explained to the participants and assurance was given to the participants that test is harmless.

Participants

Inclusion and exclusion criteria

This study was conducted in the Department of Physiology, LN Medical College and Research Center and associated J.K. Hospital, Bhopal, India. Diabetic Patients (n = 50) of both genders (age-30-65 years) attending medical OPD of J.K. Hospital attached to LNMC and RC, and healthy individuals (n = 50) of comparable age were recruited for the study. Participants meeting the following criteria were included; individuals suffering from biochemically proved DM were included in the study. Individuals were referred to the Department of ENT for complete check-up to exclude any ear pathology and were excluded with history of hearing loss before diagnosis of diabetes and had history of ear discharge, associated endocrine disorder such as myxedema, head injury, neurological deficit, cerebrovascular accident or noise exposure in the past as well as acute complication of diabetes such as diabetic ketoacidosis, nonketotic hyperosmolar coma, and hypoglycemia. Individuals with a history of drug intake, known to cause central neuropathy, for example, reserpine, alpha methyldopa, phenytoin and nitrofurantoin and had a history of taking ototoxic drugs, for example, gentamycin, streptomycin, kanamycin, amikacin, and quinine.

All selected participants were divided randomly into following groups. Group I (n = 50) - control group and the study group were diabetic individuals Group II (n = 50). Proper history and anthropometric measurements, as well as complete clinical examinations, including general systemic and audiometric examination, were done for all the included subjects.

Protocol

Estimation of blood glucose

Biochemically random blood glucose was estimated by glucose oxidase peroxidase method to confirm Type II diabetes.[18]

Recording of BERA

Recording of BERA was done on root-mean-square electromyographic Octopus by Clarity Medical Pvt. Ltd. with 2 amplifiers with hardware version 2.5 and software version 4.2 (Figure 1). Before the test, participants were instructed: To wash their hair to be oil free and the procedure was elucidated to the patient and asked to lie down comfortably on bed in a relaxed state in a quiet and dimly lit room. Skin of the forehead and mastoid process was cleaned with acetone soaked swab. Cleaned electrodes (ground electrode: (Fz), reference electrode (Cz): Vertex, and active electrode (Oz): Mastoid process) were properly placed using 10-20 conductive paste applied

Figure 1: Recording of BERA waves

in the recess of electrode and then adhered to cleaned surface of their respective side. Standard silver chloride electrodes of 1 cm diameter were placed according to 10-20 international system.[19] The stimulus was given using head phone. The stimulus rate was set at 11 clicks/s, sweep speed was set at 1 ms/div., low filter was set at 100 Hz, and high filter at 3 KHz. Recording was taken at 80 and 90 dB HL for 3KHz frequency with rare click stimulus. The resistance was kept below 5 Ohm. Auditory stimuli consisting of rarefaction clicks of 100 μs were provided through electrically shielded earphones at a rate of 11.1/s. Contralateral ear was masked with pure white noise of 40 dB. A band pass of 150-3000 Hz was used to filter out undesirable frequencies in the surroundings. Averaging was done for 2000 epochs. Impedance was kept <5 kΩ. At least two recordings were taken to confirm the reproducibility of waveform and the absolute latencies of Wave I, III, and V and interpeak latencies I-III, III-V, and I-V were recorded (Figure 2).

Statistical Analysis

Quantitative data are expressed as the mean ± standard deviation (SD) separately for right and left ear. Comparison among the study groups was done with the help of unpaired *t*-test as per results of normality test. SPSS version 20 was used for statistical analysis. Qualitative data were presented with the help of frequency and percentage table. Association among the study groups was assessed with the help of Chi-square test. $P < 0.05$ was taken as significant.

Sample Size Calculation

Considering a confidence level of 95% and confidence interval of 10 and the number of patients in our study should be 96 to achieve statistical significance. This was calculated by survey system (http://www.surveysystem.com/sscalc.htm#one). The mathematics of probability proves the size of the population is irrelevant unless the size of the sample exceeds a few percent of the total population we are examining. The Survey System ignores the population size when it is "large" or unknown. Population size is only likely to be a factor when we work with a relatively small and known group of people (e.g., the members of an association). Hence, a sample size of 96 was considered adequate for our study (we have studied 100, i.e., 50 diabetic and 50 control subjects).

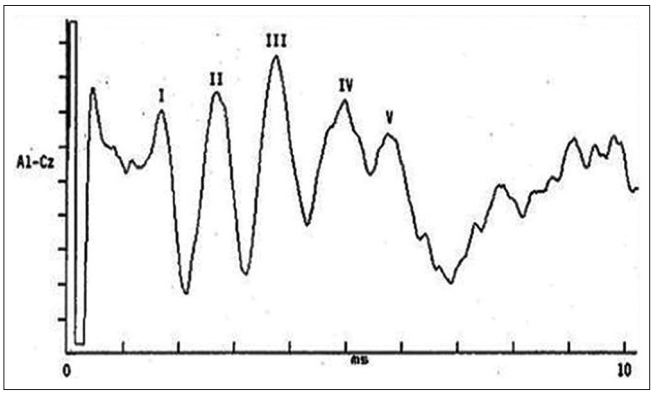

Figure 2: Normal BERA Waveform, where wave I, represent nerve action potential from auditory nerve (VIII nerve), wave II from cochlear nucleus, wave III from superior olivary nucleus, wave IV from lateral lemniscus and wave V from inferior colliculi respectively

RESULT

Blood Glucose Level

Random blood glucose was found to be significantly higher in diabetic patients groups as compared to control group.

Comparison of BERA parameters for 80 dB and 90 dB in absolute latencies (Wave I, II, II, IV, and V) and interpeak latency (I-III: I-V and III-V).

The data are summarized in (Table 1A and B) with mean ± SD. We observed, for 80 dB, there was statistically significant delayed in absolute latencies of Wave II, IV of left ear in diabetic group as compared to control group. Conversely, interpeak latency in both left and the right side was comparable among both diabetic and control groups. Although for 90 dB, there was statistically significant delayed in absolute latencies of Wave II of left ear and Wave IV of right ear in diabetic group as compared to control group. Conversely, interpeak latency in both left and the right side was comparable among both diabetic and control groups.

Comparison of BERA parameters for 80 and 90 dB in absolute latencies (Wave I, II, II, IV, and V) and interpeak latency (I-III: I-V and III-V) among diabetic individuals relation with/without peripheral neuropathy.

The data are summarized in (Table 2A and B) with mean ± SD. Absolute latencies of Waves I and III and interpeak latency difference I-V was more in left and right ear together for 80 dB, however, the difference was statistically significant only in left ear in diabetic's individuals with peripheral neuropathy, as a comparison with diabetic's individuals without peripheral neuropathy. Whereas for 90 dB, there was not any significant alteration in the entire wave of absolute latencies as well as interpeak latency difference.

Comparison of BERA parameters for 80 and 90 dB in absolute latencies (Wave I, II, II, IV, and V) and interpeak latency (I-III: I-V and III-V) among diabetic with relation to blood glucose.

The data are summarized in Table 3A and B. Intended for 80 dB, the difference in the absolute latencies of entire Wave I, II, II, IV, and V and interpeak latency I-III: I-V and

Table 1A: Comparison of BERA parameters for 80 dB between type II diabetics and control

Side	Measurement	Mean±SD		Diabetic versus control	*P* value
		Control	Diabetic	*t* value	
Left	Absolute latencies (ms)				
	I	1.6±0.19	1.54±0.2	1.4	0.138
	II	2.62±0.3	2.47±0.31	2.5	0.01*
	III	3.51±0.2	3.4±0.42	1.6	0.105
	IV	4.58±0.31	4.39±0.41	2.5	0.01*
	V	5.42±0.42	5.43±0.3	0.135	0.89
	Interpeak latencies (ms)				
	I-III	1.92±0.27	1.87±0.51	0.655	0.514
	I-V	3.8±0.46	3.89±0.4	0.874	0.384
	III-V	1.9±0.47	2±0.39	1.47	0.143
Right	Absolute latencies (ms)				
	I	1.49±0.24	1.53±0.21	0.822	0.413
	II	2.6±0.27	2.6±0.32	1.32	0.188
	III	3.4±0.26	3.4±0.31	0.069	0.945
	IV	4.47±0.33	4.48±0.46	0.199	0.843
	V	5.45±0.32	5.48±0.31	0.432	0.667
	Interpeak latencies (ms)				
	I-III	1.98±0.36	1.94±0.47	0.376	0.708
	I-V	3.9±0.46	3.9±0.45	0.239	0.812
	III-V	1.98±0.37	1.98±0.48	0.046	0.963

*Significance change (P<0.05) compared with control, SD: Standard deviation

Table 1B: Comparison of BERA parameters for 90 dB, between type II diabetics and controls

Side	Measurement	Mean±SD		Diabetic versus control	*P* value
		Control	Diabetic	*t* value	
Left	Absolute latencies (ms)				
	I	1.51±0.2	1.51±0.22	0.001	1
	II	2.68±0.26	2.5±0.25	2.7	0.008*
	III	3.61±0.35	3.5±0.41	0.696	0.488
	IV	4.4±0.24	4.5±0.38	1.34	0.18
	V	5.47±0.39	5.27±0.73	1.71	0.089
	Interpeak latencies (ms)				
	I-III	2.09±0.35	2±0.54	0.65	0.517
	I-V	3.9±0.38	3.8±0.41	0.976	0.331
	III-V	1.85±0.45	1.81±0.45	0.375	0.708
Right	Absolute latencies (ms)				
	I	1.55±0.21	1.5±0.28	0.953	0.343
	II	2.6±0.28	2.6±0.31	0.295	0.768
	III	3.58±0.27	3.6±0.27	0.291	0.772
	IV	4.57±0.24	4.33±0.37	3.77	0.0001*
	V	5.37±0.43	5.4±0.38	0.583	0.561
	Interpeak latencies (ms)				
	I-III	2.05±0.35	2.08±0.37	0.433	0.66
	I-V	3.82±0.48	3.9±0.42	1.01	0.314
	III-V	1.8±0.55	1.85±0.48	0.497	0.62

*Significance change (P<0.05), SD: Standard deviation

Table 2A: Comparison of BERA parameters for 80 dB among type II diabetics with peripheral neuropathy and without neuropathy

Side	Measurement	Mean±SD		With versus without peripheral neuropathy	
		Without peripheral neuropathy (16)	With peripheral neuropathy (34)	*t* value	*P* value
Left	Absolute latencies (ms)				
	I	1.45±0.19	1.58±0.19	2.3	0.02*
	II	2.48±0.3	2.46±0.31	0.24	0.81
	III	3.41±0.33	3.4±0.47	0.05	0.96
	IV	4.34±0.5	4.41±0.37	0.58	0.56
	V	5.56±0.26	5.3±0.31	2.01	0.05
	Interpeak latencies (ms)				
	I-III	1.96±0.34	1.82±0.57	0.89	0.375
	I-V	4.1±0.24	3.7±0.41	2.9	0.005*
	III-V	2.1±0.37	1.9±0.38	1.59	0.12
Right	Absolute latencies (ms)				
	I	1.5±0.21	1.55±0.22	0.749	0.45
	II	2.71±0.21	2.57±0.35	1.43	0.15
	III	3.54±0.25	3.45±0.34	0.943	0.35
	IV	4.5±0.39	4.4±0.49	0.211	0.834
	V	5.4±0.39	5.4±0.27	0.418	0.67
	Interpeak latencies (ms)				
	I-III	2.04±0.38	1.9±0.51	0.97	0.33
	I-V	3.9±0.5	3.91±0.43	0.279	0.78
	III-V	1.91±0.44	2±0.49	0.613	0.54

*Significance change ($P<0.05$), SD: Standard deviation

III-V was not statistically significant in diabetics with glucose level less/more than 140 mg/dl, respectively. Whereas 90 dB, among diabetics with glucose level less/more than 140 mg/dl, the difference in the absolute latencies of entire wave and interpeak latency was not statistically significant excluding the difference in the absolute latencies of Wave II as well as IV and inter latency difference I-V, which was statistically significant only in the right ears.

DISCUSSION

The Type II DM is characterized by hyperglycemia, as a result of damaged beta cell function, along with increase insulin resistance, either at receptor or at postreceptor levels.[20] DM has been concerned as an independent contributing factor of sensorineural hearing loss.[16] The electrophysiological testing reflects the bioelectric responses of the nervous system to sensory (somatosensory evoked potentials), auditory (brainstem auditory evoked potentials), or visual stimuli (visual evoked potentials).[21] A study on brainstem auditory evoked potential (BAEP) in overweight and obese individuals indicating CNS conduction delays with brainstem as well as cerebral cortical.[22] BERA comprise five or more waveforms that are recorded within 10 ms of an acoustic stimulus. Wave I originates from peripheral portion of cranial nerve VIII (auditory nerve) near the cochlear nucleus. Wave II originates from cochlear nucleus, Wave III from superior olivary nucleus, Wave IV from lateral lemniscus, and Wave V from inferior colliculi in the midbrain.[23]

In this study, a comparison between the mean values of the various wave latencies and interpeak latency was done separately for both ears, in diabetics as well as control individuals. We observed that at 80 dB, there was a significant delay in latency of Wave II, IV in left ear as compared to control and at 90 dB, there was significantly delayed Wave II latency in left ear and significantly delayed Wave IV latency in right ear as compared to controls. These findings are similar to the some of the previous studies.[24-28] The auditory brainstem response recording revealed that there was delay in neural conductance along the auditory pathway and absolute latencies of Wave I, III, and V were significantly prolonged in the diabetic group when compared with the control group.[23] Bilateral symmetrical hearing loss mainly with the high frequencies (so-called sensory neural hearing loss) was observed in 152 out of 256 diabetes patients.[26] The previous report with BERA on 20 insulin dependent diabetic patients showed that the latency of Wave III to be delayed

Table 2B: Comparison of BERA parameters for 90 dB, among type II diabetics with/without peripheral neuropathy

Side	Measurement	Mean±SD		With versus without peripheral neuropathy	
		Without peripheral neuropathy (16)	With peripheral neuropathy (34)	*t* value	*P* value
Left	Absolute latencies (ms)				
	I	1.48±0.18	1.53±0.24	0.77	0.44
	II	2.54±0.27	2.54±0.25	0.005	0.996
	III	3.66±0.36	3.51±0.43	1.24	0.22
	IV	4.57±0.21	4.47±0.43	0.84	0.401
	V	5.38±0.21	5.22±0.87	0.693	0.491
	Interpeak latencies (ms)				
	I-III	2.1±0.45	1.96±0.58	1.23	0.22
	I-V	3.9±0.34	3.8±0.44	0.497	0.622
	III-V	1.73±0.34	1.85±0.49	0.93	0.35
Right	Absolute latencies (ms)				
	I	1.52±0.26	1.49±0.29	0.39	0.69
	II	2.65±0.37	2.6±0.29	0.21	0.83
	III	3.65±0.26	3.5±0.27	0.96	0.34
	IV	4.37±0.34	4.31±0.4	0.49	0.62
	V	5.38±0.38	5.44±0.39	0.52	0.6
	Interpeak latencies (ms)				
	I-III	2.11±0.32	2.07±0.4	0.39	0.68
	I-V	3.85±0.42	3.94±0.43	0.67	0.5
	III-V	1.74±0.46	1.9±0.49	1.11	0.269

SD: Standard deviation

by 0.30 ms and Wave V by 0.45 ms and interpeak latency Wave III was delayed by 0.24 ms and IV delayed by 0.35 ms on 80, and 90 dB.[29] BERA study revealed that peripheral transmission time (Wave I) and central transmission time (Wave IV) to be delayed in normoacoustic insulin-dependent diabetic subjects.[27] The absolute latency of Wave III representing superior olivary complex, at 80 dB had wave latency of (3.99 ± 0.24) ms and at 90 dB (3.92 ± 0.28) ms. The latency of Wave III was delayed by 0.42, and 0.42 ms at 80, and 90 dB, respectively. However, the absolute latency of Wave V representing inferior colliculus, and it was at 80 dB (5.98 ± 0.27) as well as 90 dB (6.02 ± 0.30) ms as compared with control. The latency of Wave V was delayed by 0.47, and 0.50 ms at 80, and 90 dB, respectively.[27]

At present study, when we compared diabetic patients with and without peripheral neuropathy, we observed that at 80 dB, there was significant delay in Waves I and V latencies in left ear and significant delay in interpeak latencies I-V in left ear, whereas, for 90 dB, there was not any significant alteration in the entire wave of absolute latencies as well as interpeak latency difference. The previous study[30] compared 25 diabetics with peripheral neuropathy and 15 diabetics without peripheral neuropathy and observed delay in absolute latencies of Waves III and V with prolonged interpeak latencies I-III and I-V in diabetic with peripheral neuropathy as compared with diabetic without neuropathy. Symmetrical sensorineural deafness was also observed in 55% of Type II diabetes patients with neuropathy.[11] Abnormal brainstem response with neuropathy was reported in 94% of cases of Type II diabetes patients. When we compared BERA parameters with relation to blood glucose levels <140 mg/dl and blood glucose levels more than 140 mg/dl in Type II DM patients, we observed significant difference in absolute latencies of Wave IV (in right ear at 90 dB), Wave V (left ear at 80 dB) and interpeak latencies I-V (in right ear at 90 dB and in right ear at 70 dB). This differs from the study[31] in which there was a non-significant positive correlation of BAEP latencies with fasting blood glucose levels. The discrepancies between the results can be explained by the fact that the studies used a different methodology, as well as different inclusion and exclusion criteria. BERA abnormalities in diabetes initially seem to appear due to central impairment of the auditory pathway which gradually involves the peripheral parts in due course of time. Therefore, BERA can be of clinical importance intended for the diabetes, as it may reflect the degree of neural affection in the auditory pathway and may alert the patients for adequate glycemic control, which can resist the neuropathic progression any further. From our study, we can say that presences of peripheral neuropathy

Table 3A: Comparison of BERA parameters for 80 dB with relation to blood glucose levels among diabetic

Side	Measurement	Mean±SD		RBS <140 mg/dl versus RBS >=140 mg/dl	
		Blood glucose RBS <140 mg/dl (*n*=9)	Blood glucose RBS >=140 mg/dl (*n*=41)	*t* value	*P* value
Left	Absolute latencies (ms)				
	I	1.51±0.27	1.55±0.19	0.525	0.602
	II	2.4±0.42	2.48±0.28	0.76	0.44
	III	3.21±0.64	3.45±0.36	1.54	0.12
	IV	4.32±0.19	4.4±0.44	0.57	0.57
	V	5.18±0.39	5.49±0.26	2.86	0.006
	Interpeak latencies (ms)				
	I-III	1.7±0.82	1.91±0.41	1.12	0.264
	I-V	3.67±0.56	3.9±0.34	1.8	0.06
	III-V	1.98±0.5	2.03±0.37	0.344	0.73
Right	Absolute latencies (ms)				
	I	1.5±0.21	1.54±0.22	0.51	0.61
	II	2.75±0.19	2.58±0.33	1.42	0.16
	III	3.48±0.27	3.48±0.32	0.07	0.944
	IV	4.36±0.35	4.51±0.48	0.85	0.397
	V	5.4±0.5	5.5±0.26	0.87	0.38
	Interpeak latencies (ms)				
	I-III	1.98±0.39	1.93±0.49	0.281	0.78
	I-V	3.9±0.62	3.93±0.41	0.218	0.82
	III-V	1.91±0.48	1.99±0.48	0.47	0.64

RBS: Random blood sugar, SD: Standard deviation

Table 3B: Comparison of BERA parameters for 90 dB with relation to blood glucose levels among diabetic

Side	Measurement	Mean±SD		RBS <140 mg/dl versus RBS >=140 mg/dl	
		Blood glucose RBS <140 mg/dl (9)	Blood glucose RBS >=140 mg/dl (41)	*t* value	*P* value
Left	Absolute latencies (ms)				
	I	1.48±0.23	1.52±0.22	0.41	0.67
	II	2.6±0.21	2.5±0.26	0.72	0.47
	III	3.44±0.45	3.58±0.41	0.92	0.35
	IV	4.48±0.2	4.51±0.41	0.16	0.87
	V	5.33±0.25	5.26±0.8	0.25	0.79
	Interpeak latencies (ms)				
	I-III	1.95±0.54	2±0.55	0.446	0.65
	I-V	3.85±0.4	3.87±0.41	0.11	0.9
	III-V	1.88±0.41	1.8±0.46	0.51	0.609
Right	Absolute latencies (ms)				
	I	1.44±0.25	1.51±0.29	0.67	0.506
	II	2.4±0.42	2.68±0.28	1.99	0.05*
	III	3.71±0.26	3.57±0.27	1.33	0.18

(*Contd...*)

Table 3B: (*Continued*)

Side	Measurement	Mean±SD		RBS <140 mg/dl versus RBS >=140 mg/dl	
		Blood glucose RBS <140 mg/dl (9)	Blood glucose RBS >=140 mg/dl (41)	*t* value	*P* value
	IV	4.6±0.23	4.27±0.38	2.41	0.02*
	V	5.6±0.27	5.38±0.4	1.51	0.136
	Interpeak latencies (ms)				
	I-III	2.26±0.38	2.04±0.36	1.58	0.11
	I-V	4.16±0.46	3.86±0.4	2.01	0.05*
	III-V	1.9±0.33	1.84±0.51	0.29	0.76

*Significance change (P<0.05), RBS: Random blood sugar, SD: Standard deviation

are definite risk factors for the development of central neuropathy.

CONCLUSION

Due to the high prevalence of DM in the community and its probable adverse effects on the auditory system, this study investigated hearing functions in well-characterized Type II diabetic patients and evaluated the role of potentially relevant factors such as blood glucose levels and presence of complications like peripheral neuropathy. In this study, significant differences in BERA latencies were seen between diabetic patients and healthy controls. These abnormalities were attributed to a diabetic associated central auditory dysfunction. Evaluation of these changes might help to determine early subclinical hearing impairment in these patients. This study suggests that BERA is a simple, non-invasive procedure to detect early impairment of acoustic nerve, and CNS pathways, even in the absence of specific symptoms, if BERA is carried out in diabetic patients, and involvement of central neuronal axis can be detected earlier. Keeping in mind the consistent rise in the global incidence of DM and the detrimental effects that it has on the hearing ability of an individual, it is suggested that BERA testing may also be included in the routine screening procedures that are of vital importance in diabetic patients, wherever it is possible.

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