DOI:10.14744/bej.2018.29491
Beyoglu Eye J 2018; 3(3): 109-115

Original Article

Multifocal Electroretinography in Diabetic Patients without Retinopathy

Ozgur Balta,[1] Gulten Sungur,[2] Mehmet Yakin,[2] Nurten Unlu,[2] Oyku Bezen Balta,[3] Eyup Ozcan,[4] Firdevs Ornek[2]

[1]Department of Ophthalmology, Dr. Nafiz Korez Sincan State Hospital, Ankara, Turkey
[2]Department of Ophthalmology, Ankara Training and Research Hospital, Ankara, Turkey
[3]Department of Family Medicine, Yildirim Beyazit University Faculty of Medicine, Ankara, Turkey
[4]Department of Ophthalmology, Private Goznuru Eye Hospital, Gaziantep, Turkey

Abstract

Objectives: This study aimed to assess the multifocal electroretinography (mfERG) findings in diabetic patients without retinopathy according to the HbA1c levels and diabetes duration.

Methods: A total of 62 eligible patients with Type 2 diabetes mellitus and 30 healthy controls which were matched by age and sex were included in the study. Only the right eye of each patient was analyzed. All of the participants underwent a comprehensive ophthalmic examination, and the mfERG responses which were N1-P1 amplitude, N1 implicit times and P1 implicit times were calculated.

Results: The mfERG N1-P1 amplitude was significantly reduced in diabetic patients compared to controls in inner two rings. There was significant different between controls and stable patients' mfERG P1 implicit times in ring 2. It was found that there were negative correlation trends between diabetes duration and mfERG N1-P1 amplitude, there were negative, positive correlation trends between diabetes duration and N1 implicit times and P1 implicit times in all rings. There was a statistically significant negative correlation between diabetes duration and N1-P1 amplitude only in ring 5. Furthermore, it was found that there were statistically significant positive correlations between diabetes duration and N1 implicit times in ring 1, 2 and 5. There was a significant correlation between diabetes duration and P1 implicit times only in ring 2.

Conclusion: We demonstrated that mfERG N1-P1 amplitude was reduced in inner retinal areas (ring 1 and ring 2), and P1 implicit time was delayed only in ring 2 in Turkish diabetic patients without retinopathy. There was a statistically significant correlation between diabetes duration and N1-P1 amplitude and N1 implicit times in some retinal areas.

Keywords: Diabetes, diabetic retinopathy, multifocal electroretinography.

Introduction

In 2012, there were approximately 93 million people with diabetic retinopathy (DR), 17 million with proliferative DR, 21 million with diabetic macular edema, and 28 million with vision-threatening DR worldwide (1). DR was the fifth most common cause of preventable blindness and fifth most common cause of moderate-severe visual impairment (MSVI) between 1990 and 2010. Furthermore, the age-standardized prevalence of DR causing MSVI had increased slightly from 1990 to 2010 (2).

Electroretinography (ERG) is an objective method of evaluating the retinal function and also demonstrates abnormal results in diabetic patients without any ophthalmoscopic findings (3). The multifocal ERG (mfERG) enables assessment of many retinal areas within roughly 8 min per eye (4).

Address for correspondence: Ozgur Balta, MD. Nafiz Korez Sincan Devlet Hastanesi, Oftalmoloji Anabilim Dalı, Ankara, Turkey
Phone: +90 553 061 95 72 **E-mail:** drozgurbalta@hotmail.com
Submitted Date: March 26, 2018 **Accepted Date:** October 02, 2018 **Available Online Date:** December 19, 2018

Palmowski et al. (5) found that in patients with diabetes mfERG amplitudes were reduced and implicit times were increased compared to healthy peoples. Harrison et al. found that mfERG implicit time is a good predictor of DR onset in patients with diabetes without DR (6). The ability of the mfERG findings to predict the future retinopathy provides clinicians a good tool to screen, follow-up, and even consider early prophylactic treatment of the patients with DR (7).

As shown by many studies, preventing elevated blood glucose significantly reduces the risk of ocular and visual complications of diabetes (8–11). Kahn and Bradley found a strong positive association between retinopathy and duration of diabetes in a random sample of 914 diabetic patients (12). Among younger-onset patients with diabetes, the prevalence of any retinopathy was 8% at first 3 years, 25% at 5 years, 60% at 10 years, and 80% at 15 years. The prevalence of proliferative DR was 0% at first 3 years and increased to 25% at 15 years. The 4-year incidence of developing proliferative retinopathy in the younger-onset group increased from 0% during the first 5 years to 27.9% during years 13–14 of diabetes (13).

The aims of this study were to compare mfERG findings (N1–P1 amplitude and P1-implicit time) in diabetic patients without DR who had different HbA1c levels to healthy control participants without diabetes and to assess the correlation mfERG responses with diabetes duration.

Methods

Study Population and Ethical Considerations

This prospective study was performed in the Departments of Ophthalmology at Ankara Training and Research Hospital, Turkey. This study was approved by the Institutional Review Board. Informed consents were obtained from all patients and tenets of the Declaration of Helsinki were followed.

A total of 62 eligible patients with Type 2 diabetes mellitus and 30 age- and sex-matched healthy controls were included in the study. The diagnosis of Type II diabetes mellitus was based on criteria of the World Health Organization (WHO). Exclusion criteria included best-corrected visual acuity (BCVA) worse than 20/20, high spherical or cylindrical >±1 diopter refractive errors, pseudoexfoliation syndrome, DR, history of uveitis, gluacoma, ocular trauma, previous intraocular surgery, and presence of systemic diseases, such as renal or hepatic dysfunction, obesity, and rheumatological diseases. Furthermore, people who were currently smoking or using alcohol, and/or had prosthetic devices or electromagnetic field generating devices, were excluded in the study.

Age, sex, duration of diabetes, and HbA1c levels were recorded. Diabetic patients were classified into two groups by HbA1c levels. Patients with 7% or less HbA1c levels were grouped as a stable patient group, patients with >7% were grouped unstable patient group. Furthermore, only the right eye of each patient was analyzed.

All of the patients underwent a comprehensive ophthalmic examination, including medical history review, refraction, BCVA, and intraocular pressure measured by Goldmann applanation tonometer, anterior, and fundus segment examinations.

Multifocal ERG

Retiscan Retipor 32 TM (Roland Consult, Wiesbaden, Germany) was used for recording according to the International Society for Clinical Electrophysiology of Vision standards (2011 edition) (14). After the anesthetizing with topical 0.5% proparacaine hydrochloride, the active electrodes (ERG-Jet Universo, Switzerland) were placed on the cornea following pulling up the upper eye limb. The neuter of the reference electrodes was placed at the frontal region; the other one was positioned to 2 cm lateral of the external canthal region, with plaster after the concave inner surfaces were filled with 2–3% methylcellulose.

The local retinal sensitivity changes were observed in the 61 points from the totally 60° field which were 30° on either side of the fixation point after the case was positioned 24 cm away from the screen. The test was applied after the pupils were dilated with 1% tropicamide and 2.5% phenylephrine, and the participants were standing in room light for 15 min. The stimuli were presented on a 20-inch monitor (Sony Multiscan G520 TM, Japan), driven at a 60-Hz frame rate and consisted of an array of 61 hexagonal elements, according to binary m-sequence with a base interval of 13.3 ms. White hexagons had a luminance of 120 cd/m^2, and dark hexagons were 1cd/m^2 in local contrasts of 99%. Signals were amplified 100.000 times and filtered with a range of 10–100 Hz and recorded with a sampling interval of 83 ms (16 times per video frame) under fixation control. In the presence of the conditions that caused the degradation of the signals such as blinking of patients and loss of fixation, the recording was repeated. The averages of the first-order kernel mfERG amplitudes and latancies were calculated for each five concentric rings. The retinal areas of rings were ring 1 (central-2.1°), ring 2 (1.4°–6.7°), ring 3 (5.7°–12.0°) ring 4 (9.5°–19.8°), and ring 5 (15.1°–28.5°).

Statistical Analysis

Statistical analyzes were performed with the Statistica version 10 (StatSoft Inc.). Descriptive statistics (median, minimum, maximum, and frequencies) were used to describe the baseline characteristics of the study groups. Kruskal–Wallis test was used to compare non-normally distributed quantitative variables between the study groups. Multiple com-

parisons of mean ranks for all groups were carried out as post hoc test, and Bonferroni adjustment was used for p values. To compare qualitative variables, the Pearson Chi-square test was used. The association of diabetes duration with mfERG N1-P1 amplitude, N1 implicit times and P1 implicit times were analyzed by Spearman correlation test in diabetes patients.

Results

Out of 30 control participants 18 (60.0%), out of 30 stable patients 18 (60.0%) and out of 32 unstable (62.5) were male. The mean age of controls was 49.4±4.7, stable patient group was 51.9±4.8, and unstable patient group was 49.7±7.6. The gender (p=0.987) and age (p=0.314) were not statistically different between study groups (Table 1).

In-ring 1, the mfERG N1-P1 amplitude was significantly reduced in stable and unstable patients compared to controls (0.0003). In post hoc analysis, it was found that there was a statistically significant difference between controls and stable patients (p=0.0051), and unstable patients (p=0.0004). In-ring 2, there were also statistically significant differences between controls and patient groups (p=0.0267). In post hoc test, while there was no significantly significant difference between controls and stable patients (p=0.1683), and there was a statistically significant difference between controls and unstable patients (p=0.0265) in ring 2. The mfERG N1-P1 amplitude findings of controls and patient groups were statistically similar in rings 3, 4, and 5. In post hoc analysis, there was no statistically significant difference between two patient groups in all rings (post hoc test results were not shown) (Table 2).

It was found that there were no statistically significant differences in the mfERG P1 implicit times findings between controls and patient groups in rings 1, 3, 4, and 5. However, there was a significantly significant difference between groups in ring 2 (p=0.0138). In post hoc analysis, while there was significantly significant difference between controls and stable patients (p=0.0169), and there was no statistically significant difference between controls and unstable patients (p=0.1913) in ring 2 (post hoc test results were not shown) (Table 3).

In an analysis of the mfERG N1 implicit times, it was found that there were no statistically significant differences between controls and patient groups in all rings 1, 2, 3, 4, and 5 (Table 4).

Although it was found that there were negative correlation trends between diabetes duration and mfERG N1-P1 amplitude, there were positive correlation trends between

Table 1. Baseline characteristics of the study group

	Controls (n=30)	Stable patients (n=30)	Unstable patients (n=32)	p
Gender				
Male (%)	18 (60.0)	18 (60.0)	20 (62.5)	*0.987
Female (%)	12 (40.0)	12 (40.0)	12 (37.5)	
Age (years)				
Mean±SD	49.4±4.7	51.9±4.8	49.7±7.6	**0.314
Median	50.0	50.0	51.5	
Minimum	42.0	42.0	40.0	
Maximum	60.0	59.0	60.0	

*Chi-square test was used; **Kruskal–Wallis test was used; SD: Standard deviation.

Table 2. Median, minimum and maximum N1-P1 amplitude (nV/deg^2) for 5 rings of retina

Area of retina	Controls (n=30)			Stable patients (n=30)			Unstable patients (n=32)			*p
	Median	Min	Max	Median	Min	Max	Median	Min	Max	
Ring 1	123.45	84.90	157.90	103.45	59.70	144.10	96.90	61.80	155.60	0.0003
Ring 2	54.30	40.80	75.40	53.00	1.50	71.60	49.30	7.90	76.10	0.0267
Ring 3	27.90	20.40	41.30	28.60	12.00	35.10	25.40	17.90	37.90	0.1033
Ring 4	19.00	15.40	26.70	19.85	10.20	26.20	17.95	11.50	25.60	0.0936
Ring 5	12.25	7.84	121.10	11.45	7.12	16.60	11.50	5.63	17.70	0.1148

*Kruskal-Wallis test was used.

Table 3. Median, minimum and maximum P1 implicit times (ms) for 5 rings of retina

Area of retina	Controls (n=30)			Stable patients (n=30)			Unstable patients (n=32)			*p
	Median	Min	Max	Median	Min	Max	Median	Min	Max	
Ring 1	35.80	33.80	38.80	35.80	33.90	39.80	35.80	31.90	41.80	0.1161
Ring 2	31.90	3.90	32.90	32.40	30.90	37.80	31.90	9.00	35.80	**0.0138**
Ring 3	31.40	29.90	33.90	31.90	30.90	34.80	31.90	29.90	35.80	0.0521
Ring 4	31.90	29.90	33.90	32.90	30.90	35.80	31.90	3.90	36.80	0.1551
Ring 5	32.90	30.90	35.80	33.90	31.90	36.80	32.90	31.90	39.80	0.2809

*Kruskal-Wallis test was used.

Table 4. Median, minimum and maximum N1 implicit times (ms) for 5 rings of retina

Area of retina	Controls (n=30)			Stable patients (n=30)			Unstable patients (n=32)			*p
	Median	Min	Max	Median	Min	Max	Median	Min	Max	
Ring 1	16.90	12.90	149.00	16.90	14.90	20.90	16.90	13.90	22.90	0.3446
Ring 2	14.90	12.90	16.90	14.90	13.90	16.90	14.90	12.90	17.90	0.1203
Ring 3	14.90	12.90	16.90	14.90	13.90	17.90	14.90	12.90	17.90	0.0895
Ring 4	15.40	12.90	17.90	15.40	14.90	18.90	15.90	12.90	19.90	0.4047
Ring 5	16.90	13.90	18.90	16.90	15.90	18.90	16.40	14.90	19.90	0.1123

*Kruskal-Wallis test was used.

Table 5. The correlation of diabetes duration with mfERG N1-P1 amplitude, N1 implicit times and P1 implicit times

Area of retina	Diabetes duration with N1-P1 amplitude		Diabetes duration with N1 implicit times		Diabetes duration with P1 implicit times	
	Correlation Coefficient (R)	*p	Correlation Coefficient (R)	*p	Correlation Coefficient (R)	*p
Ring 1	-0.127	0.342	0.287	0.029	0.249	0.059
Ring 2	-0.207	0.118	0.438	0.001	0.342	0.009
Ring 3	-0.099	0.461	0.225	0.089	0.170	0.201
Ring 4	-0.196	0.140	0.221	0.096	0.186	0.162
Ring 5	-0.317	0.015	0.270	0.040	0.172	0.197

*Spearman Correlation test was used; mfERG: Multifocal electroretinography.

diabetes duration and N1 implicit times and P1 implicit times in all rings (Fig. 1). There was a statistically significant negative correlation between diabetes duration and N1-P1 amplitude in ring 5 ($r=-0.317$, $p=0.015$), but not significant correlation in other rings. Furthermore, it was found that there were statistically significant positive correlations between diabetes duration and N1 implicit times in ring 1, 2, and 5, but not in ring 3 and 4.

While there were not statistically significant correlations between diabetes duration and P1 implicit times in ring 1, 3, 4, and 5, there was significant correlation between diabetes duration and P1 implicit times only in ring 2 (Table 5).

Discussion

The WHO estimates that, globally, 422 million people aged over 18 years were living with diabetes in 2014. In 2012, there were 1.5 million deaths worldwide directly caused by diabetes, and it was the eighth leading cause of death (15). DR was the cause of MSVI in 1.9% and of blindness in 2.6% globally in 2010 (2). Good metabolic control significantly re-

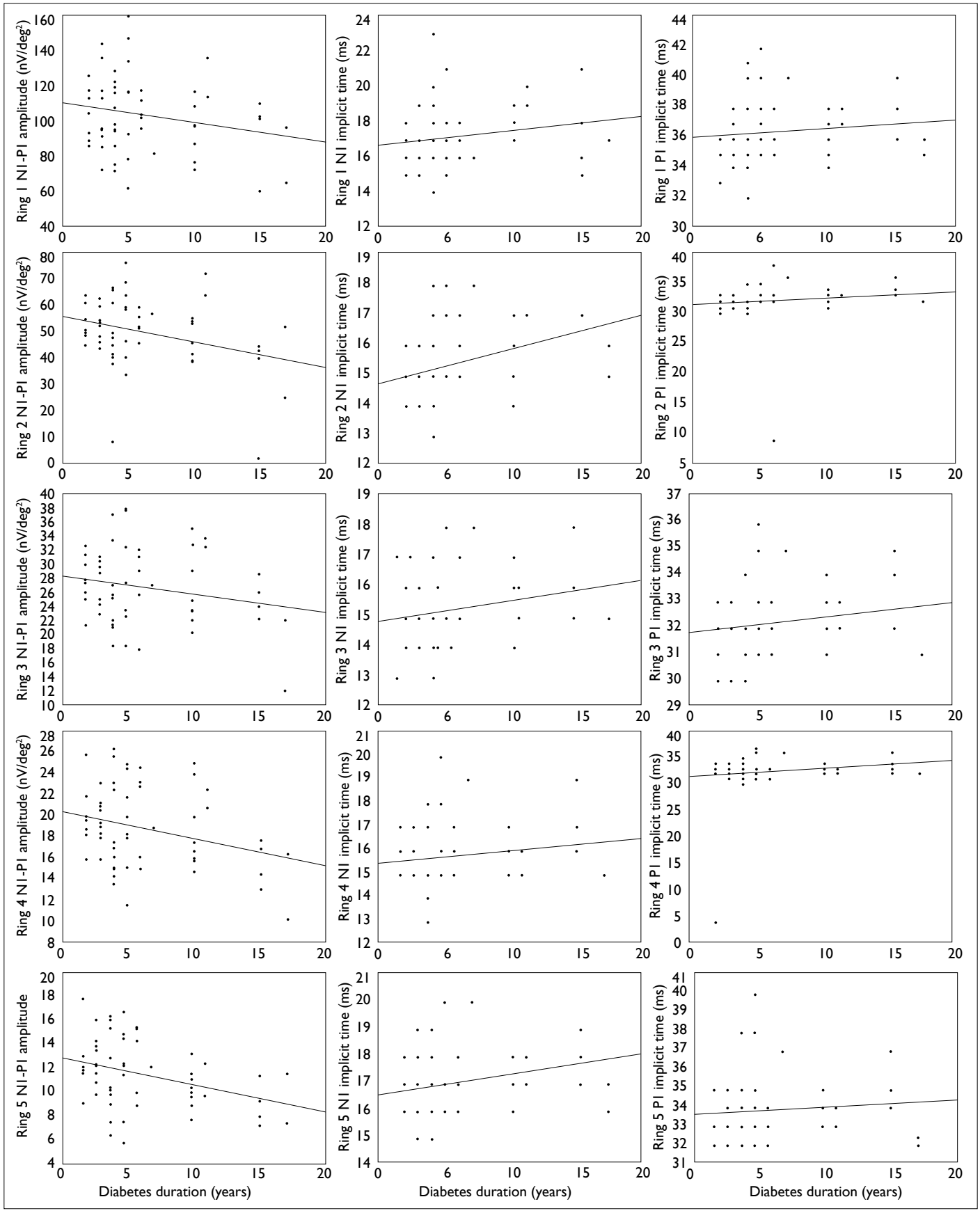

Figure 1. Correlation of N1-P1 amplitude, N1 implicit time, P1 implicit time and diabetes duration.

duces the risk of development and progression of ocular and visual complications of both Type 1 and Type 2 diabetes (9).

Bearse and Sutter reported that abnormal mfERG implicit times are locally predictive of the development of new DR over 1 and 2 years, and these functional abnormalities are spatially associated with retinopathy once it is present. They said that retinal dysfunction in early DR is primarily due to neuropathy or neuro vasculopathy rather than microvascular pathology alone (7).

It is complicated whether sex/gender itself or other related risk factors cause the male and female differences in diabetes and DR. Therewithal, sex/gender is a variable that must be considered inaccurate viewpoints of diabetes and DR in any case of etiology (16, 17). There is a huge literature about age's effect on diabetes. Namperumalsamy et al. reported that older age (>50 years) was a significant risk factor for the prevalence of DR in a South Indian community (18). The Wisconsin epidemiological study of DR reported that severity of retinopathy was related to younger age at diagnosis, and the 10-year incidence of retinopathy and progression of retinopathy were highest in the group disease onset before 30 years (19, 20). In this study, sex and age were statistically similar between study groups. This situation made the evaluation of the study results convenient.

We found that mfERG N1-P1 amplitude was reduced in inner retinal areas (ring 1 and ring 2) in diabetic patients compared to controls. Furthermore, we found that P1 implicit time was delayed only in ring 2, and N1 implicit time was similar in the diabetic patient group which had different HbA1c levels compared to the control group. There was a statistically significant negative correlation between diabetes duration and N1-P1 amplitude only in ring 5. Furthermore, it was found that there were statistically significant positive correlations between diabetes duration and N1 implicit times in ring 1, 2, and 5, and there was significant correlation between diabetes duration and P1 implicit times only in ring 2.

Adhikari et al. (21) demonstrated reduced mfERG N1–P1 amplitude and delayed P1-implicit time in Nepalese diabetic patients without retinopathy. They found that diabetes duration and fasting blood glucose have a significant influence on implicit time, but not on amplitude.

Han et al. (22) studied mfERG responses to predict the development of DR with 22 patients which 11 are diabetic patients with nonproliferative DR (NPDR), and 11 are diabetic patients without retinopathy. They retested patients 12 months after the first examination. They found that new retinopathy developed in 7 of the eyes with NPDR, and the eyes without retinopathy did not develop new retinopathy after 1 year. Furthermore, they reported that mfERG implicit times were more delayed in NPDR eyes, but not in eyes without retinopathy and control eyes, and mfERG amplitudes had no predictive power.

Dhamdhere et al. (23) found that Type 2 diabetes patients had reduced mfERG amplitude and longer P1 implicit time than the controls and Type 1 diabetes patients in their trial which was studied 45 diabetic patients without retinopathy (10 with Type 1 and 35 with Type 2 diabetes).

In a 78 eyes included study to predict local formation of DR in diabetic patients without retinopathy, it was reported that multivariate analysis (mfERG implicit time Z-score, sex, diabetes duration, blood glucose, HbA1c, age, and diabetes type) showed mfERG implicit time to be predictive for DR development in a zone after adjusting for diabetes type, with a sensitivity of 80% and a specificity of 74% (6).

Laron et al. (24) reported that mean mfERG implicit time was significantly longer in the patients compared with controls, but amplitude was similar, in their study which they evaluate associations between mfERG responses in 115 Type 1 diabetic adolescents without retinopathy compared to 30 controls. They found that implicit time was positively correlatedwith HbA1c but not correlated with diabetes duration, BG, or age.

In a study conducted with 14 Type 1 diabetic patients without retinopathy and 14 healthy controls, authors found that, during acute normoglycemia, patients demonstrated an overall 1.36-ms delay of the P1 first-order implicit times (p=0.0013) and a 0.72-ms delay of the second-order P1 (p=0.0049) compared with healthy subjects. They reported that, during acute hyperglycemia, the P1 first-order delay was only 0.81 ms (p=0.02), and the P1 second-order implicit time was comparable to that of healthy subjects (p>0.05). The magnitude of the diabetes-associated implicit time delay, at both levels of glycemia, was proportional to the level of chronic hyperglycemia at study entry, as expressed by the patients' HbA1c. They said that the results show that chronic hyperglycemia induces an adaptational response that tends to normalize retinal implicit times at a higher level of habitual glycemia (25).

Conclusion

We demonstrated that mfERG N1-P1 amplitude was reduced in inner retinal areas (ring 1 and ring 2), and P1 implicit time was delayed only in ring 2 in Turkish diabetic patients without retinopathy. There was a statistically significant negative correlation between diabetes duration and N1-P1 amplitude only in ring 5, and statistically significant positive correlations between diabetes duration and N1 implicit times in some retinal areas. These results may be specific to the study group, so further multicenter and longitudinal studies are required to evaluate different manifestation forms of diabetes.

Disclosures

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship Contributions: Involved in design and conduct of the study (OB, GS, NU, OBB, FO); preparation and review of the study (OB); data collection (OB, GS); and statistical analysis (OB, EO).

References

1. Yau JW, Rogers SL, Kawasaki R, Lamoureux EL, Kowalski JW, Bek T, et al; Meta-Analysis for Eye Disease (META-EYE) Study Group. Global prevalence and major risk factors of diabetic retinopathy. Diabetes Care 2012;35:556–64.
2. Bourne RR, Stevens GA, White RA, Smith JL, Flaxman SR, Price H, et al; Vision Loss Expert Group. Causes of vision loss worldwide, 1990-2010: a systematic analysis. Lancet Glob Health 2013;1:e339–49.
3. Tzekov R, Arden GB. The electroretinogram in diabetic retinopathy. Surv Ophthalmol 1999;44:53–60.
4. Bearse MA, Sutter EE. Imaging localized retinal dysfunction with the multifocal electroretinogram. J Opt Soc Am A Opt Image Sci Vis 1996;13:634–40.
5. Palmowski AM, Sutter EE, Bearse MA, Fung W. Mapping of retinal function in diabetic retinopathy using the multifocal electroretinogram. Invest Ophthalmol Vis Sci 1997;38:2586–96.
6. Harrison WW, Bearse MA Jr, Ng JS, Jewell NP, Barez S, Burger D, et al. Multifocal electroretinograms predict onset of diabetic retinopathy in adult patients with diabetes. Invest Ophthalmol Vis Sci 2011;52:772–7.
7. Bearse MA Jr, Adams AJ, Han Y, Schneck ME, Ng J, Bronson-Castain K, et al. A multifocal electroretinogram model predicting the development of diabetic retinopathy. Prog Retin Eye Res 2006;25:425–48.
8. Effect of intensive therapy on the development and progression of diabetic nephropathy in the Diabetes Control and Complications Trial. The Diabetes Control and Complications (DCCT) Research Group. Kidney Int 1995;47:1703–20.
9. Stratton IM, Kohner EM, Aldington SJ, Turner RC, Holman RR, Manley SE, et al. UKPDS 50: risk factors for incidence and progression of retinopathy in Type II diabetes over 6 years from diagnosis. Diabetologia 2001;44:156–63.
10. The relationship of glycemic exposure (HbA1c) to the risk of development and progression of retinopathy in the diabetes control and complications trial. Diabetes 1995;44:968–83.
11. Rodriguez-Fontal M, Kerrison JB, Alfaro DV, Jablon EP. Metabolic control and diabetic retinopathy. Curr Diabetes Rev 2009;5:3–7.
12. Kahn HA, Bradley RF. Prevalence of diabetic retinopathy. Age, sex, and duration of diabetes. Br J Ophthalmol 1975;59:345–9.
13. Klein R, Klein BE, Moss SE, Davis MD, DeMets DL. The Wisconsin epidemiologic study of diabetic retinopathy. II. Prevalence and risk of diabetic retinopathy when age at diagnosis is less than 30 years. Arch Ophthalmol 1984;102:520–6.
14. Hood DC, Bach M, Brigell M, Keating D, Kondo M, Lyons JS, Marmor MF, et al; International Society For Clinical Electrophysiology of Vision. ISCEV standard for clinical multifocal electroretinography (mfERG) (2011 edition). Doc Ophthalmol 2012;124:1–13.
15. WHO. Global Report on Diabetes. Geneva: World Health Organization, 2016. Available at: http://apps.who.int/iris/bitstream/handle/10665/204871/9789241565257_eng.pdf?sequence=1. Accessed Dec 10, 2018.
16. Ozawa GY, Bearse MA Jr, Adams AJ. Male-female differences in diabetic retinopathy? Curr Eye Res 2015;40:234–46.
17. Ozawa GY, Bearse MA Jr, Bronson-Castain KW, Harrison WW, Schneck ME, Barez S, et al. Neurodegenerative differences in the retinas of male and female patients with type 2 diabetes. Invest Ophthalmol Vis Sci 2012;53:3040–6.
18. Namperumalsamy P, Kim R, Vignesh TP, Nithya N, Royes J, Gijo T, et al. Prevalence and risk factors for diabetic retinopathy: a population-based assessment from Theni District, south India. Br J Ophthalmol 2009;93:429–34.
19. Klein R, Klein BE, Moss SE, Davis MD, DeMets DL. The Wisconsin epidemiologic study of diabetic retinopathy. III. Prevalence and risk of diabetic retinopathy when age at diagnosis is 30 or more years. Arch Ophthalmol 1984;102:527–32.
20. Klein R, Klein BE, Moss SE, Cruickshanks KJ. The Wisconsin Epidemiologic Study of diabetic retinopathy. XIV. Ten-year incidence and progression of diabetic retinopathy. Arch Ophthalmol 1994;112:1217–28.
21. Adhikari P, Marasini S, Sah RP, Joshi SN, Shrestha JK. Multifocal electroretinogram responses in Nepalese diabetic patients without retinopathy. Doc Ophthalmol 2014;129:39–46.
22. Han Y, Bearse MA Jr, Schneck ME, Barez S, Jacobsen CH, Adams AJ. Multifocal electroretinogram delays predict sites of subsequent diabetic retinopathy. Invest Ophthalmol Vis Sci 2004;45:948–54.
23. Dhamdhere KP, Bearse MA Jr, Harrison W, Barez S, Schneck ME, Adams AJ. Associations between local retinal thickness and function in early diabetes. Invest Ophthalmol Vis Sci 2012;53:6122–8.
24. Laron M, Bearse MA Jr, Bronson-Castain K, Jonasdottir S, King-Hooper B, Barez S, et al. Association between local neuroretinal function and control of adolescent type 1 diabetes. Invest Ophthalmol Vis Sci 2012;53:7071–6.
25. Klemp K, Sander B, Brockhoff PB, Vaag A, Lund-Andersen H, Larsen M. The multifocal ERG in diabetic patients without retinopathy during euglycemic clamping. Invest Ophthalmol Vis Sci 2005;46:2620–6.