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Original Research Article

An Observational Evaluation of Visual Evoked Potential in Diabetics: a Case Control Study

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Abstract

Aim: The aim of the present study was to analyse visual evoked potential in diabetes and age matched controls.

Methods: The study was conducted in the Department of Physiology, Netaji Subhas Medical College and Hospital, Bihta, Patna, Bihar, India. In this study, diabetics (total 50) between 25 to 55 years attending medical outpatient department of Netaji Subhas Medical College and Hospital, Bihta, Patna, Bihar, India were selected and 50 normal age matched subjects were selected randomly from the general population.

Results: The patients with diabetes mellitus have subclinical visual impairment as revealed by impaired visual evoked potential. Diabetics showed delayed latencies and reduced amplitude of various parameters of VEP. There was a positive correlation between prolongation of latencies and duration of diabetes and FBS levels.

Conclusion: VEP abnormalities in diabetes initially seem to appear due to central impairment of visual pathway. Thus, VEP can be of clinical importance for diabetes, as it reflects the degree of neural affection and may alert patients for adequate glycemic control, which can resist neuropathic progression any further. Although from our study we can say that duration of illness and poor glycemic control are definitive risk factors for the development of central neuropathy, a larger sample size would have had a significant outcome. It is recommended to perform VEP initially on all diabetic patients and to keep this as an "initial record of visual examination of patients".

Keywords: Diabetes mellitus; vision; visual evoked potential (VEP); VEP & duration of diabetes mellitus; VEP & FBS levels; waves N70 & P100

Introduction

Type 2 diabetes mellitus (T2DM) is a metabolic disorder that is characterized by abnormal glucose homeostasis, in the context of insulin resistance and relative insulin deficiency. [1] T2DM is a global epidemic its prevalence is rapidly increasing all over the globe. [2] The International Diabetes Federation estimates the total number of diabetic patients to rise to 69.9 million by the year 2025. [3] With an increasing trend in the incidence of diabetes reported, there is also an increase in complications of T2DM due to damage and dysfunction of the organs such as the eye. [4]

Diabetes is a major cause of blindness. [5] Diabetic retinopathy (DR) is a complication of T2DM, which is the sixth common cause of blindness in India [6] the overall prevalence being 17.6% in the Indian population. [7] There is enough evidence to show that at least 90% of these new cases could be reduced if there were proper and vigilant treatment and monitoring of the eyes. [8] During the initial stage of DR, most people do not notice any change in their vision. [9] Hence, it is beneficial for the patient to have any changes in the function of the retina identified early enough, to effect early treatment.

A measure of visual function in patients with diabetes can be performed using visually evoked potentials (VEPs), which are electrical potential differences occurring in the visual areas of the occipital cortex, in response to visual stimuli and are recorded from the scalp. Patients with T2DM and with DR have shown abnormalities in VEP recordings, relating to increase in implicit time/latency. [10,11]

According to the International Diabetes Federation report for 2021, approximately 537 million individuals are living with diabetes, and it is projected to reach 783 million by 2045. Diabetic ocular complications are asymptomatic at early stages and cause visual impairment unless diagnosed early and treated. [12] Electrophysiological testing is an objective noninvasive method for evaluating target function. The electroretinogram (ERG), which is an important electrophysiological test in the field of ophthalmology, is capable of evaluating the

functions of various retinal cells and has contributed to deepening our understanding of retinal neuronal damage caused by diabetes.

The aim of the present study was to analyse visual evoked potential in diabetes and age matched controls.

Materials and Methods

The study was conducted in the Department of Physiology, Netaji Subhas Medical College and Hospital, Bihta, Patna, Bihar, India. In this study, diabetics (total 50) between 25 to 55years attending medical outpatient department of Netaji Subhas Medical College and Hospital, Bihta, Patna, Bihar, India for one year were selected and 50 normal age matched subjects were selected randomly from the general population.

Inclusion criteria: Age group between 25 – 55 years, patients who are biochemically proved diabetes mellitus, Patients of type I and type II diabetes mellitus, Normal healthy age matched controls between 25-55 years.

Age group below 25 years and above 55 years, Patients with visual acuity less than 6/18 even with corrected lenses, Patients with acute complication of diabetes like, diabetic ketoacidosis, recurrent ketonuria, non ketotic hyperosmolar coma and hypoglycaemia, Patients with diabetic retinopathy, cataract, glaucoma, vitreous opacities, optic atrophy, maculopathy, Patients taking psychoactive drugs or drug addiction, H/O Hypertension, anaemia, stroke, dementia, Smokers, Alcoholics, H/O cardiovascular or neurological disorders were excluded from the study. Written and informed consent were taken for the study after explaining the procedure and its significance in their vernacular language. The ethical committee clearance was taken. A brief personal history was taken and a clinical examination of all the systems was done to exclude medical problems and to prevent confounding of results. Detailed ophthalmological check up of all patients was done which includes visual acuity, ocular tension and fundus examination.

After selecting the subjects, they were subjected to VEP testing on PC based, 2 channel, RMS EMG. EP MARK II machine manufactured by RMS RECORDERS and MEDICARE SYSTEM, Chandigarh. Procedure in brief: Recording was carried out in a quiet and dimly lit room. Subjects were asked to come without applying oil to scalp and to shampoo hair and make it dry.

VEP RECORDING: VEPs were recorded using the RMS machine and standard silver- silver chloride disc electrodes. A VEP monitor displaying checker board is used to give the pattern reversal stimulus. A montage consisting of one channel is used for the VEP recording. The subject is asked to sit comfortably in front of the checkerboard pattern at an eye screen distance of 100cm. An amplification which ranged between 20,000 and 1,00,000 was used to record the VEPs. The electrode impedance was kept below 5KΩ. The recording was performed in a dark and sound attenuated room. Uniocular stimulation was given to both eyes separately with black and white checks which changed phase (black to white and white to black) abruptly and repeatedly at a specified number of reversals per second, by using a checkerboard.

The usual glasses (if any) were allowed to be put on during the test. The subject is instructed to avoid the usage of meiotic or mydriatic drugs, 12 hours before the test. The electrodes were placed with an electrode paste after cleaning the site with a spirit swab. The scalp electrodes were placed relative to bony landmarks. The anterior/posterior midline measurements were based on the distance between nasion and inion over the vertex. The active electrode was placed in the middle of the variation zone of the calcarine fissure at Oz, which is the highest point on the occiput. The reference electrode was placed at Fz or 12cm above the inion. The ground electrode was placed over the forehead Cz.

Results

Table 1: Comparison of VEP parameters between Diabetics and Healthy controls in left eye

LATENCIES LEFT EYE (ms)	Case		Control		
	Mean	Std. Deviation	Mean	Std. Deviation	P Value
N_{70}	75.05	4.36	66.06	1.48	P<0.001
P_{100}	106.64	6.14	95.90	1.36	P<0.001
N_{155}	138.12	4.86	132.64	3.16	P<0.001
AMPLITUDE LEFTEYE(μv)					
N_{70}-P_{100}	3.56	1.48	6.64	0.86	P<0.001
P_{100}-N_{155}	6.24	2.08	8.92	1.06	P<0.001

Table 2: Comparison of VEP parameters and Duration of Diabetes in left eye

LATENCIES LEFT EYE(ms)	DURATION			P Value
	<10	10-15	>15	
N_{70}	72.88 ± 3.4	75.5 ± 4.16	76.74 ± 4.86	P<0.01
P_{100}	98.82 ± 1.6	105.5 ±4.42	112.88 ± 3.07	P<0.001
N_{155}	138.6 ±4.86	139.35 ± 3.16	140.4 ± 5.95	0.35
AMPLITUDE LEFT EYE(μv)				
N_{70}-P_{100}	5.3 ±0.67	3.47 ±0.82	2.02 ± 0.66	P<0.001
P_{100}-N_{155}	8.42± 0.58	6.48 ±1.12	3.77 ±0.43	P<0.001

Table 3: Comparison of VEP parameters and FBS levels in left eye

LATENCIES LEFT EYE(ms)	FBS			P Value
	< 130	**130-145**	**>145**	
N_{70}	71.6 ± 1.82	72.8 ±5.0	75.5 ±4.6	0.07
P_{100}	98.22 ± 1.2	99.11 ±0.98	106.7 ±5.5	P<0.001
N_{155}	139.6 ± 4.6	139.7 ± 3.16	136.94 ± 5.15	0.42
AMPLITUDE LEFT EYE(μv)				
N_{70}-P_{100}	5.15 ± 0.60	5.07 ± 1.06	3.16 ±1.4	P<0.001
P_{100}-N_{155}	8.92 ±0.42	7.73± 1.43	5.65 ± 1.85	P<0.001

The patients with diabetes mellitus have subclinical visual impairment as revealed by impaired visual evoked potential. Diabetics showed delayed latencies and reduced amplitude of various parameters of VEP. There was a positive correlation between prolongation of latencies and duration of diabetes and FBS levels.

Discussion

Diabetes Mellitus (DM) encompasses a cluster of common metabolic disorders that cause hyperglycemia. The estimated worldwide prevalence of DM has increased substantially over the past three decades, from 30 million cases in 1985 to 425 million in 2017. The International Diabetes Federation has predicted that if the current trend continues more than 629 million people will develop diabetes by 2045. [13] DM and its subsequent pathophysiologic changes affecting multiple organs place a heavy burden not only on patients but also on the entire health care systems. [14]

Worldwide estimates project that, in 2030 the greatest number of individuals with diabetes will be 45-64 years of age. [15] It is a set of clinical syndromes that affect distinct regions of the nervous system, singly or combined, encompasses wide range of abnormalities affecting proximal and distal peripheral sensory and motor nerves and autonomic nervous system. [16] Cranial nerve mononeuropathies are commonly observed in diabetes. The 3rd, 4th and 6th nerves are involved, separately or in varying combination. Optic nerve affection manifested as optic atrophy, as a result of diabetes alone is estimated to occur in 0.6% cases. [17] The patients with diabetes mellitus have subclinical visual impairment as revealed by impaired visual evoked potential. Diabetics showed delayed latencies and reduced amplitude of various parameters of VEP. There was a positive correlation between prolongation of latencies and duration of diabetes and FBS levels.

In our study we found that there was prolongation of latencies of waves N70, P100 and N155 ($p<0.001$) and reduced amplitude of N70-P100 and P100-N155 ($p<0.001$) in diabetics compared to controls in both eyes. The P100 waveform is generated in the striate and peristriate occipital cortex, N70 reflects activity of the fovea and primary visual cortex while N155 reflects activity of visual association areas. [18] The delayed latencies and reduced amplitude which were recorded even in the absence of retinopathy or any ocular pathology is indicative of anterior visual pathway affection.[17] In our study, we found that the latencies of N70 and P100 were significantly prolonged in diabetics with duration of illness between 10-15years and more than 15 years compared to duration of less than 10 years ($p<0.01$, $p<0.001$) respectively. There was also significant reduction in amplitude of N70-P100 and P100-N155 ($p<0.001$) in diabetics of longer duration in both eyes. The present study concurs with findings of V. Gayathri et al., studies have shown that alterations in VEP latency are not present at the onset of diabetes, but occur only after the disease has been present for a mean of at least 3.3 years. Retinal, macular and visual pathway function is differently impaired in diabetes patients with different duration of disease, having no signs of retinopathy. The

impairment starts in the nerve conduction of the visual pathways with an early involvement. It is carried on into the innermost retinal layers and in the macula and ends in the middle and outer retinal layers. [19] Similar findings were reported in Siedl R et al. [20]

According to Pozzessere G et al [21], increasing evidence suggests that the accumulation of glucose susbstrate, as a consequence of relative lack of insulin, increases aldose reductase activity. The increased enzyme activity of alternate polyol pathway at different level, including vessel walls, retina and particularly nerve complex metabolism, may slowly and progressively impair neurologic functions. Ziegler O et al [22], in their study showed that, after 3 days of close blood glucose monitoring the mean latencies were significantly shorter but were still significantly longer than control values

Conclusion

VEP abnormalities in diabetes initially seem to appear due to central impairment of visual pathway. Thus, VEP can be of clinical importance for diabetes, as it reflects the degree of neural affection and may alert patients for adequate glycemic control, which can resist neuropathic progression any further. Although from our study we can say that duration of illness and poor glycemic control are definitive risk factors for the development of central neuropathy, a larger sample size would have had a significant outcome. It is recommended to perform VEP initially on all diabetic patients and to keep this as an "initial record of visual examination of patients".

References

1. Kumar V, Fausto N, Abbas AK, Cotran RS, Robbins SL. Robbins and Cotran Pathologic Basis of Disease. 7th ed. Philadelphia, PA: Saunders; 2005. p. 1194-5.
2. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res. 2007;125 (3):217-30.
3. Kertes PJ, Johnson TM, editors. Evidence Based Eye Care. Philadelphia, PA: Lippincott Williams & Wilkins; 2007.
4. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2009;32 Suppl 1:S62-7.
5. World Health Organization. Global Report on Diabetes. Geneva: WHO; 2016.
6. Sicree R, Shaw J, Zimmet P. Diabetes and impaired glucose tolerance. In: Gan D, editor. Diabetes Atlas. International Diabetes Federation. 3rd ed. Belgium: International Diabetes Federation; 2006. p. 15-103.
7. Rema M, Premkumar S, Anitha B, Deepa R, Pradeepa R, Mohan V. Prevalence of diabetic retinopathy in urban India: The Chennai Urban Rural Epidemiology Study (CURES) ey study, I. Invest Ophthalmol Vis Sci. 2005;46(7): 2328-33.
8. Tapp RJ, Shaw JE, Harper CA, de Courten MP, Balkau B, McCarty DJ, et al. The prevalence of and factors associated with diabetic retinopathy in the Australian population. Diabetes Care. 2003;26(6):1731-7.
9. Bek T, Hammes HP, Porta M, editors. clinical presentations and pathological correlates of retinopathy. Experimental Approaches to Diabetic Retinopathy. Front Diabetes. Vol. 20. Basel: Karger; 2010. p. 1-19.
10. Anastasi M, Lodato G, Cillino S. VECPs and optic disc damage in diabetes. Doc Ophtha lmol. 1987;66:331-6.
11. Collier A, Reid W, McInnes A, Cull RE, Ewing DJ, Clarke BF. Somatosensory and visual evoked potentials in insulin dependent diabetics with mild peripheral neuropathy. Diabetes Res Clin Pract. 1988;5(3):171-5.
12. Wong TY, Sun J, Kawasaki R, Ruamvi boonsuk P, Gupta N, Lansingh VC, Maia M, Mathenge W, Moreker S, Muqit MMK, Resnikoff S, Verdaguer J, Zhao P, Ferris F, Aiello LP, Taylor HR. Guidelines on Diabetic Eye Care: The International Council of Ophthalmology Recommendations for Screening, Follow-up, Referral, and Treatment Based on Resource Settings. Ophthalmology. 2018 Oct;125(10):1608-1622.
13. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, Colagiuri S, Guariguata L, Motala AA, Ogurtsova K, Shaw JE, Bright D, Williams R; IDF Diabetes Atlas Committee. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. Diabetes Res Clin Pract. 2019 Nov;157:107843.
14. Jameson JL, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J. Harrison's principles of internal medicine. (No Title). 2018.
15. Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. Harrison's Principles of Internal Medicine, 18th Ed. New York: McGraw- Hill; 2011: 2968-69.
16. Vinik AI, Mehrabyan A. Diabetic neuropath hies. Medical Clinics. 2004 Jul 1;88(4):947-99.
17. Raman PG, Sodani A, George B. A study of visual evoked potential changes in diabetes mellitus. Int J Diabetes Dev Ctries. 1997;17 :69-73.
18. Chopra D, Gupta M, Manchanda KC, Sharma RS, Sidhu RS. A study of visual evoked potentials in patients of type-2 diabetes mellitus. J Clin Diagn Res. 2011 Jun;5(3):519-22.
19. Gayathri V, Vijayalakshmi B, Chandrasekhar M. Electro physiological assessment of

neuropathy in visual pathway of diabetes mellitus. Journal of diabetology. 2012 Jan 1;3 (1):4.
20. Seidl R, Birnbacher R, Hauser E, Bernert G, Freilinger M, Schober E. Brainstem auditory evoked potentials and visually evoked potentials in young patients with IDDM. Diabetes care 1996 Nov; 19(11): 1220-1224.
21. Pozzessere G, Rizzo PA, Valle E, Mollica MA, Meccia A, Morano S et al. Early detection of neurological involvement in IDDM and NIDDM. Multimodal evoked potential versus metabolic control. Diabetes care 1988 June; 11 (6): 473-480.
22. Ziegler O, Guerci B, Algan M, Lonchamp P, Weber M, Drouin P. Improved visual evoked potential latencies in poorly controlled diabetic patients after short term strict metabolic control. Diabetes care 1994 Oct; 17(10): 1141-1147.