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A Study of Visual Evoked Potential for Functional Assessment of Visual Pathway in Ophthalmologically Normal Diabetes Mellitus Patients

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ABSTRACT

Background and aim: Diabetic retinopathy(DR) is one of the most common complications of long-standing diabetes mellitus(DM). Early detection of this complication in the preclinical stages by Visual Evoked Potential (VEP) may help formulate management strategies to preserve Vision. With this background in mind, we conducted a cross-sectional study to measure the VEP of patients with DM.

Materials and methods: The study was conducted on 30 patients with diabetes mellitus(1 & 11) having no neurological or ophthalmological complications attending the diabetic clinic of RG Kar Medical College, Kolkata. Their PR-VEP(Pattern Reversal VEP) was measured in both eyes. Different VEP parameters(P100 & N75 waves) were compared with 30 age and sex-matched healthy controls and were statistically co-related with nerve conduction velocity, duration of diabetes, and Hb1Ac.

Results: The P100 & N75 wave peaks were significantly prolonged in diabetic patients compared to healthy non-diabetic control. Also, this prolongation was significant with the increasing duration of the disease.

Conclusion: Our study demonstrates that VEP prolongation in ophthalmologically normal DM patients may serve as an important screening tool to pick up visual dysfunction in the early stages.

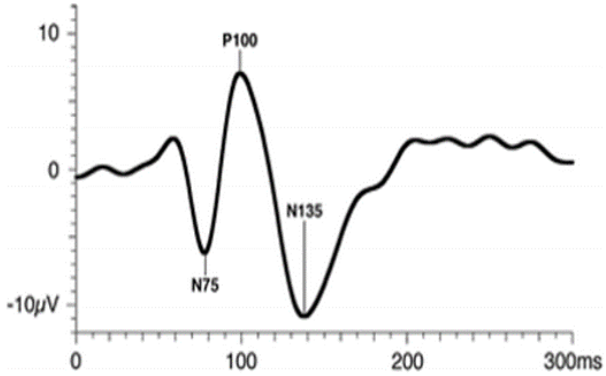
1. Introduction

Diabetes mellitus (DM) is not a single disease; rather, it represents a spectrum of metabolic disorders with the common characteristic feature of hyperglycemia. The metabolic abnormalities underlying these disorders lead to secondary pathophysiological processes in different organ systems, giving rise to myriads of manifestations of diabetes. These complications ultimately impose a huge burden of morbidity and mortality on the individual and the total healthcare system. Early detection and management of its systemic manifestations are very effective for preventing complications as well.[1]

Visual Evoked Potential (VEP) is a non-invasive, sensitive electrophysiologically assesses the functional integrity of the visual pathway and may be used to quantify the affection of the visual pathway in patients of DM. VEP is the visually evoked electrical activity of the striate cortex recorded from the surface of the overlying scalp and extracted from electroencephalographic recordings by averaging.[2] It has been used to assess the functional integrity of the central vision from the cornea up to the striate cortex.[3] VEPs are of different types according to the stimuli used to evoke visual responses. In our study, we have used the 'Pattern Reversal' stimulus through a black and white checkerboard presented by a computer screen at a distance of 76 cm from the test subject's eye.[6]

Pattern reversal (PR) VEP waveforms

The standard PR-VEP waveform consists of two negative (N) and one positive (P) peak. The periods post-stimulus after which the peaks appear are called peak time or peak latencies. Accordingly, the waves are N75, P100, and N135 having peak latencies of 75ms,100ms, and 135ms, respectively.



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There are controversies regarding these nomenclatures, however. The P100 is the most constant wave by consensus opinion. However, the N75 peak is called N70 according to IFCN (International Federation of Clinical Neurophysiology) standards. Again N135 peak is called N140, N145, or N155 in many studies. Average latencies of the negative peaks vary considerably in normal healthy subjects depending upon recording protocols or a wide range of interpersonal variability. We shall stick to this article's ISCEV (International Society for Clinical Electrophysiology of vision) standards to avoid confusion. Although conventionally, the VEP waveform is represented in a reverse manner with negative waves projected upwards from baseline, ISCEV uses usual upright positive notations in its guideline. P100 waveform is generated as a consequence of activation of the primary visual cortex in the striatum and peristriatal occipital cortex as well as discharge from the thalamocortical fibers. The wave most commonly used in the studies is P100. Recent studies, however, have also focused on the N75 and N145 latencies. N75 represents the fovea and primary visual cortex activity, whereas N145 represents the visual, associative area.[11, 12] Research with multichannel scalp recordings, visual fMRI activity, and dipole modelling, suggests that the visual cortex is the source of the early components of the VEP, i.e., N75 before P100. The early phase of the P100 is likely generated in the dorsal extrastriate cortex of the middle occipital gyrus. The later negative component N135 is generated from several areas, including a deep source in the parietal lobe.[11, 12]

Aims and objectives

1. To determine whether VEP parameters (P100& N75peak latencies and peak to peak wave amplitudes of P100-N75) are abnormal in ophthalmologically normal diabetes mellitus patients, compared to nondiabetic controls.
2. To determine if any correlation exists between alterations of VEP parameters with each of the following:

a) Duration of diabetes.
b) HbA1c levels.
c) Treatment received for diabetes.

2. Materials and methods

Study population

Patients with type1/type 2 diabetes mellitus attending the diabetic clinic, fulfilling the inclusion criteria, and willing to be a part of the study were selected as sample populations. The control consisted of age and sex-matched healthy nondiabetic controls.

Inclusion criteria

Patients with type1/type2 diabetes voluntarily consented to be a part of the study.

Exclusion criteria

a) Subjects with chronic illnesses like long-standing hypertension, multiple sclerosis, peripheral neuropathy, seizure disorders, H/O cerebrovascular accident.
b) Patients on long-standing medications like antihypertensives, anti-epileptics, or H/O alcohol consumption>100ml/d>5 years.
c) Patients with visual problems like glaucoma, cataracts, diabetic retinopathy, optic atrophy, vitreous opacities, and low visual acuity, although refraction correction was excluded from the study.

Study period

February 2019 to March 2020.

Sample size

Thirty cases were serially selected from the diabetic clinic and 30 healthy controls.

Study design

Cross-sectional observational study.

Parameters studied

1)VEP-RMS EMG. EP Mark II machine was used to measure VEP.
Mean P100 peak latency of VEP of both eyes in a millisecond.
Mean N75 peak latency of VEP of both eyes in milliseconds.
Mean peak to peak P100-N75 wave amplitude of VEP of both eyes in Microvolts.

2)HbA1c was measured by the colorimeter.

3) Ophthalmological examination:

a) Visual acuity (with glasses on, if any) of both eyes.
b) Inspection of anterior segment.
c)Direct ophthalmoscopy.
d)VEP test procedure.

Active, reference and ground electrodes were placed at OZ, FZ, CZ.respectively according to a 10-20 system of EEG electrodes. The distance of the patient's eyes from the screen displaying checkerboard stimulus was 76 cm.

Pattern reversal VEP was recorded separately for each eye in a uniformly illuminated room using the PC-based two-channel RMS EMG. EP MARK II machine.

Statistical analysis

Statistical analysis has been done using the following statistical tests employing PC based softwares [SPSS Version 20]:

I) two-tailed independent sample t-test.
II) ANOVA test.
III) Spearman's Rank Order Correlation test.

Qualitative data are presented as numbers and percentages, and quantitative data are presented as Mean ± Standard Deviation.

3. Results

Statistical analysis has been done using the following statistical tests employing PC based software:

Table 1. Distribution of male and female subjects among both study and control groups.

	Male	Female	Total
Number	9	21	30
Proportion	30%	70%	100%

Table 2. Age-group-wise distribution of male and female subjects in study and control groups.

Age Group → / Sex ↓	10-30 Years	31-50 Years	51 Years & above	Total
Male	**2**	**3**	**4**	**9**
Female	**2**	**16**	**3**	**21**
Total	**4**	**19**	**7**	**30**

Table 3. Height distribution of study subjects among case and control groups.

	Mean Height (cm)	SD	Range (cm)
Cases (N=30)	153.1	7.25	140-173
Controls (N=30)	155.27	4.74	147-166

Table 4. The weight distribution of study subjects among case and control groups.

	Mean Weight (kg)	SD	Range (kg)
Cases (N=30)	59.0	7.97	47-93
Controls (N=30)	55.4	7.26	46-76

Table 5. Distribution of HbA1c level among case and control groups.

	Mean HbA1c (%)	SD	Range (%)
Cases (N=30)	6.97	1.49	4.92-10.4
Controls (N=30)	4.96	0.62	3.75-6.2

Table 6. Comparison of HbA1c level between case and control groups.

	Cases (N=30)	Controls (N=30)	t value	P-value
HbA1c % [Mean ± SD]	6.970 ± 1.49	4.958 ± 0.62	t(58)=6.82	< 0.0001**

Table 7. Comparison of N75-P100 amplitude of VEP wave latencies between two eyes of cases.

	Right Eye (N=30)	Left Eye (N=30)	t value	P-value
P100 Latency (ms) [mean ± SD]	104.73 ± 5.24	104.77 ± 5.36	t(58)=0.03	0.97 [NS]
N75 Latency (ms) [mean ± SD]	74.32 ± 5.03	75.04 ± 4.57	t(58)=0.58	0.56 [NS]
N75-P100 Amplitude (µs) [mean ± SD]	5.92 ± 7.41	5.90 ± 7.00	t(58)=0.008	0.993 [NS]
N145 Latency (ms) [mean ± SD]	142.64 ± 3.30	142.12 ± 3.34	t(58)=0.60	0.54 [NS]

Table 8. Comparison of VEP wave latencies and N75-P100 amplitude between two eyes of controls.

	Right Eye (N=30)	Left Eye (N=30)	t value	P-value
P100 Latency (ms) [mean ± SD]	101.17 ± 3.90	100.95 ± 3.73	t(58)=0.22	0.82 [NS]
N75 Latency (ms) [mean ± SD]	72.04 ± 4.41	71.63 ± 4.12	t(58)=0.38	0.71 [NS]

N145 Latency (ms) [mean ± SD]	140.56 ± 6.13	141.05± 6.63	t(58)=0.30	0.77 [NS]
N75-P100 Amplitude (µs) [mean ± SD]	6.932 ± 1.50	6.939 ± 1.80	t(58)=0.15	0.98 [NS]

Table 9. Average of Right eye and Left eye values of VEP parameters in cases and controls.

	Cases (N=30)	Controls (N=30)
P100 Latency (ms) [mean ± SD]	104.75 ± 5.21	101.06 ± 3.70
N75 Latency (ms) [mean ± SD]	74.68 ± 4.70	71.84 ± 4.21
N145 Latency (ms) [mean ± SD]	142.38 ± 7.08	141.6 ± 6.33
N75-P100 Amplitude (µs) [mean ± SD]	5.92 ± 3.28	6.93 ± 1.63

Table 10. Comparison of average N75-P100 amplitude and VEP wave latencies of both eyes (in µV) between cases and controls.

	Cases (N=30)	Controls (N=30)	t value	P-value
P100 Latency (ms) [mean ± SD]	104.75 ± 5.21	101.06 ± 3.70	t(58)=3.16	0.002 *
N75 Latency (ms) [mean ± SD]	74.68 ± 4.70	71.84 ± 4.21	t(58)=2.47	0.016 *
N75-P100 Amplitude (µs) [mean ± SD]	5.92 ± 3.28	6.93 ± 1.63	t(58)=1.52	0.133 [NS]
N145 Latency (ms) [mean ± SD]	142.38 ± 7.08	141.6 ± 6.33	t(58)=0.45	0.656 [NS]

Table 11. Correlation between HbA1c level and mean VEP wave latencies and amplitude in diabetics cases.

	Spearman Correlation Coefficient (ρ)	P-value
P100 Latency (ms)	0.37	0.85
N75 Latency (ms)	- 0.07	0.71
N75-P100 Amplitude (µV)	0.07	0.72

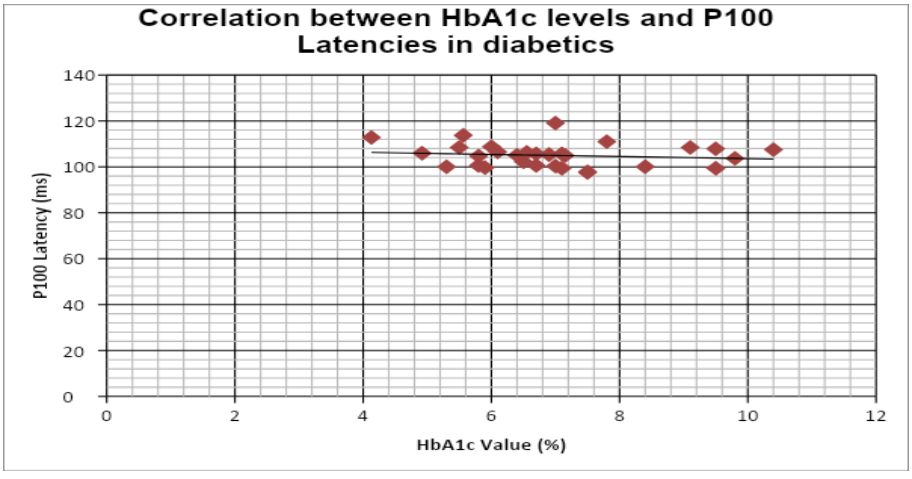

Fig. 1. Correlation between HbA1c levels and P100 Latencies in diabetics.

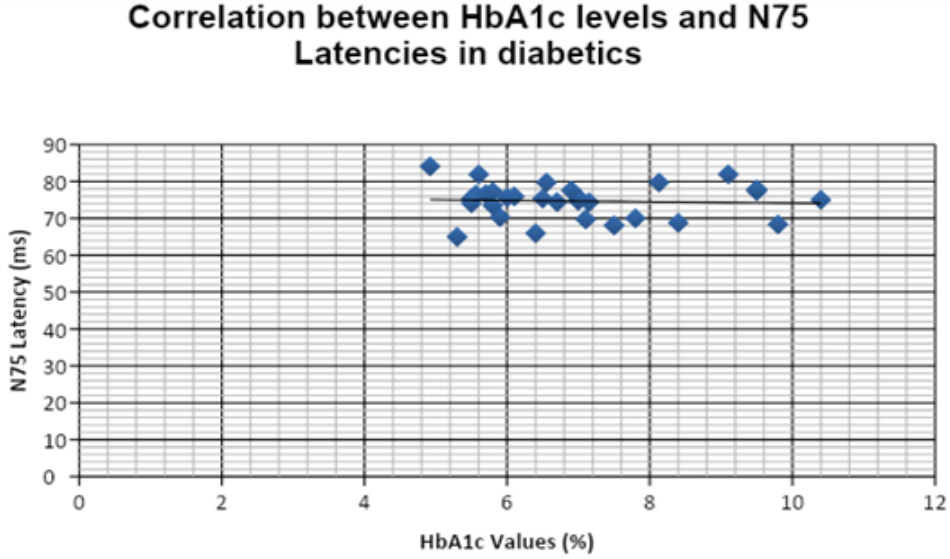

Fig. 2. Correlation between HbA1c levels and N75 Latencies in diabetics.

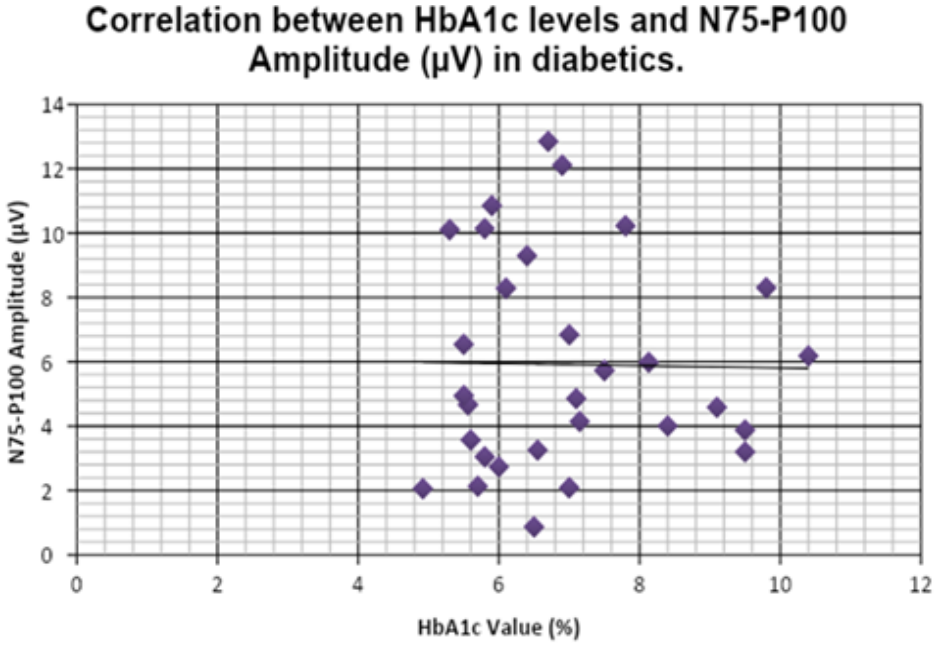

Fig. 3. Correlation between HbA1c levels and N75-P100 Amplitude (µV) in diabetics.

Table 12. Comparison of average VEP wave latencies and the average amplitude of both eyes between group I (having disease duration < 6 years) and group II (having disease duration 6 to 11 years) diabetic patients.

	Group I (N=16)	Group II (N=7)	t value	P-value
P100 Latency (ms) [Mean ± SD]	102.43 ± 4.22	107.69± 2.56	t(21)=3.03	0.006 *
N75 Latency (ms) [Mean ± SD]	72.49± 4.64	76.31 ± 1.78	t(21)=2.09	0.048 *
N75-P100 Amplitude (µV) [Mean ± SD]	6.99± 3.67	5.34 ± 1.65	t(21)=1.12	0.27 [NS]

Table 13. Comparison of average VEP wave latencies and the average amplitude of both eyes between group II (having disease duration 6 to 11 years) and group III (having disease duration ≥ 11 years) diabetic patients.

	Group II (N=7)	Group III (N=7)	t value	P-value
P100 Latency (ms) [Mean ± SD]	107.69± 2.56	108.58 ± 6.52	t(12)=0.33	0.74 [NS]
N75 Latency (ms) [Mean ± SD]	76.31 ± 1.78	78.54± 3.66	t(12)=1.45	0.17 [NS]
N75-P100 Amplitude (μV) [Mean ±SD]	5.34 ± 1.65	4.00± 2.79	t(21)=1.09	0.29 [NS]

Table 14. Comparison of average VEP wave latencies and the average amplitude of both eyes between group I (having disease duration < 6 years) and group III (having disease duration ≥ 11 years) diabetic patients.

	Group I(N=16)	Group III (N=7)	t value	P-value
P100 Latency (ms) [Mean ± SD]	102.43 ± 4.22	108.58 ± 6.52	t(21)=2.72	0.012 *
N75 Latency (ms) [Mean ± SD]	72.49± 4.64	78.54± 3.66	t(21)=3.05	0.006 *
N75-P100 Amplitude (μV) [Mean ± SD]	6.99± 3.67	4.00± 2.79	t(21)=1.91	0.07 [NS]

Table 15. Comparison of average VEP wave latencies and the average amplitude of both eyes between group I (having disease duration < 6 years), group II (having disease duration 6 to 11 years), and group III (having disease duration ≥ 11 years) diabetic patients by ANOVA test.

	Group I(N=16)	Group II (N=7)	Group III (N=7)	P-value
P100 Latency (ms) [Mean ± SD]	102.43 ± 4.22	107.69± 2.56	108.58 ± 6.52	0.0026 *
N75 Latency (ms) [Mean ± SD]	72.49± 4.64	76.31 ± 1.78	78.54± 3.66	0.0042 *
N75-P100 Amplitude (μV) [Mean ±SD]	6.99± 3.67	5.34 ± 1.65	4.00± 2.79	0.16 [NS]

Table 16. Comparison of average VEP wave latencies and the average amplitude of both eyes between only metformin-treated and both metformin and glimepiride treated group of diabetic patients.

	Metformin treated Group (n=9)	Metformin + Glimepiride treated Group (n=14)	t value	P-value
P100 Latency (ms) [Mean ± SD]	104.77 ± 6.52	105.61 ± 4.87	t(21)=0.35	0.73 [NS]
N75 Latency (ms) [Mean ± SD]	74.29 ± 4.30	74.57 ± 5.23	t(21)=0.13	0.89 [NS]
N75-P100 Amplitude (μV) [Mean ± SD]	6.93 ± 3.68	5.66 ± 3.36	t(21)=0.87	0.39 [NS]

Table 17. Comparison of average VEP wave latencies and the average amplitude of both eyes between metformin and glimepiride treated and insulin (alone or along with oral drugs) treated group of diabetic patients.

	Metformin + Glimepiride treated Group (n=14)	Insulin treated Group (n=4)	t value	P-value
P100 Latency (ms) [Mean ± SD]	105.61 ± 4.87	101.07 ± 1.88	t(16)=1.79	0.09 [NS]
N75 Latency (ms) [Mean ± SD]	74.57 ± 5.23	73.19 ± 4.71	t(16)=0.47	0.64 [NS]
N75-P100 Amplitude (μV) [Mean ± SD]	5.67 ± 3.36	6.13 ± 3.77	t(16)=0.24	0.81 [NS]

The study results shown in the tables and figures above are summarized below:

1. Almost 2/3rd (19 patients) belong to 31 to 50 years old with three males and 16 females. Nondiabetic control subjects were selected, keeping the number of males and females in each age group the same as those in diabetic subjects.

2. HbA1c levels of diabetic cases and nondiabetic controls differ significantly with mean values of 6.97% and 4.96%, respectively, the p-value being extremely significant at value < 0.0001.

3. No significant difference in any of the VEP parameters has been observed between the two eyes of diabetic cases. The p-values derived from the independent t-test between right and left eye parameters are 0.97, 0.56, 0.54, and 0.99 respectively for P100 latency, N75 Latency, N145 Latency, and N75-P100 amplitude.

4. There is a significant difference (p=0.002) of P100 latency between diabetic and nondiabetic groups, i.e., the P100 wave peak is significantly delayed in people with diabetes compared to nondiabetic subjects.

Similarly, the N75 wave-peak latency in the diabetic group is significantly prolonged than the nondiabetic group with a p-value =0.006. However, no statistically significant difference in N145 wave latency and N75-P100 amplitude has been found when compared between diabetics and nondiabetics (p=0.656 & p=0,133, respectively).

5. HbA1c percentage values have been considered a marker of the degree of glycaemic control over the past three months. Spearman correlation coefficient (ρ) has been used to find any correlation between glycaemic control (HbA1c level) and VEP parameters. However, the correlations were found to be variable and weak without having any statistical significance.

6. Duration of suffering from diabetes has a significant effect on the prolongation of P100 and N75 latencies. Diabetic patients were divided into three groups according to the duration of suffering in years since the first diagnosis: Group I (< 6 years), Group II (6–11 years), and Group III (≥11 years). When compared between group I and group II, both P100 Latency and N75 Latency were significantly prolonged in group II (p=0.006 & p=0.048 respectively). Similarly, when group I and group III were compared, both P100 and N75 latencies showed a statistically significant delay in group III (p=0.012 & p=0.006, respectively). However, when compared between group II and group III, no significant differences in wave latencies were found. Likewise, when all the three groups were compared by ANOVA test, similar results were obtained with a statistically significant prolongation of P100 and N75 latencies (p=0.0026 & p= 0.0042 respectively) with increasing duration of diabetes across the groups.

7. The effect of the treatment modality was investigated by dividing the diabetic patients according to anti-diabetic medication received and comparing the VEP parameters across the groups. Out of 30 diabetic patients, 9 received only metformin, 14 received glimepiride in addition to metformin, 2 received metformin, glimepiride, and pioglitazone 1 voglibose with metformin and glimepiride, and rest 4 received human insulin injections - regular or 30:70 mixture.

This study demonstrates functional affection of the visual pathway early in diabetes mellitus, and the magnitude of these functional alterations increases with the advancement of the disease. These findings corroborate well with most of those of earlier researchers. However, we could not find any significant association of these changes with intermediate-term glycemic control in the recent past, nor could we find the primary modality of treatment used to control hyperglycemia to impact those results. In the published literature also, there is considerable variability of results in these respects.

4. Discussion

VEP has shown to be a very sensitive and useful tool to investigate even subtle neurophysiological alterations in the visual pathway. P100 latency is the most sensitive as well as consistent VEP parameter across the studies. Our study also demonstrated a significant prolongation of P100 latency in people with diabetes compared to nondiabetic controls. Bhanu R et al., Chopra D et al., Gayathri V et al., [1, 3, 5] have reported similar findings. The correlation was found to be positive between VEP latencies and the duration of diabetes.[5, 6] VEP latencies' prolongation was reported in asymptomatic type 1 diabetic patients without retinopathy in some other studies.[6, 7, 8] Earlier, Yaltkaya et al. suggested that prolongation in the P100 Latency in the diabetic subjects was due to retro chiasmal involvement.[9, 10] N75 Latency in diabetics has also been found to be prolonged in our observation. N75-P100 amplitude also is not affected significantly in metabolic derangements as in DM. We failed to show any significant alterations of N75-P100 amplitude in people with diabetes. Karlica et al. noticed a progressive decrease in P100 wave amplitude values with increasing age among diabetic children, but amplitude also decreases with age in the general population, thus minimising its significance.[7]

As far as the duration of suffering from diabetes is concerned, we have found that both P100 and N75 wave latencies were being delayed progressively with increased duration of DM. This finding is also consistent with the findings of Dolu H et al.[4] Karlica et al. also found that latency values in patients with T1DM tend to increased with time, which might be a direct sign of ganglion cell damage. However, in children with type 1 diabetes, there was no significant statistical correlation between the duration of the disease and N75-P100 wave amplitude. In the preceding paragraphs, while enumerating the pathophysiology of diabetic retinopathy, it has been mentioned that apart from the duration, control of hyperglycemia is also implicated as an important etiopathogenic factor. FBS and PPBS as a marker of glycemic control are not reliable for study as they reflect only transitory glycemic status owing to considerable fluctuations over days. In this regard, HbA1c is a better marker as it reflects the average blood glucose level over the previous 2–3 months. However, we could not find any statistically significant correlation (either positive or negative) between HbA1c level and any VEP parameters in diabetics. That implies that even intermediate-term glycemic control status may not have any statistically significant impact on the functioning of the visual pathway, at least in pre-retinopathic stages. This finding agrees with Dolu H et al., who also found that VEP latency was not associated with HbA1c levels in children with type 1 DM. They have concluded that even three months of stable diabetic control does not favorably alter the natural course of pre retinopathic dysfunctions of the visual pathway. On the contrary, high HbA1c level had been shown to prolong VEP latencies in studies conducted by Gayathri M et al. found that mean N75 latency and P100 latency was prolonged, and the amplitude was reduced in those with HbA1c >7%, and it was statistically significant.[5] Firstly, neuropathogenesis in chronic metabolic derangements like diabetes is a slow and gradual process unlikely to be affected by glycemic dysregulation even for 2 to 3 months. It may be the reason for not observing any significant effect of HbA1c level on VEP parameters. Secondly, studies that did show a considerably strong association of poor glycemic control with VEP latency prolongation, perhaps though unbiased, recruited more people with diabetes having prolonged periods of poorly controlled hyperglycemia, not just for 2/3 months as evidenced by HbA1c level. Both these postulations further underscore the greater role of duration of hyperglycemia in the causation of microvascular complications of DM. Our study also intended to determine whether pharmacological interventions used to treat hyperglycemia affect visual

electrophysiology in the preclinical stage. This aspect has not been investigated widely earlier in published literature. Accordingly, we have analyzed and compared the VEP data among three groups of diabetic patients – one group was treated with metformin alone, the second with glimepiride in addition to metformin, and the third group received some form of insulin with or without other oral antidiabetic agents. However, we did not find a significant difference in any of the VEP parameters across the groups. Hence, it is not possible to find the superiority of any particular drug or drug combination over the others in the primary prevention of visual loss in diabetics.

Limitations of the study

In this study, patients had HBA1C below 7 %. It may explain the lack of association between HBA1C and VEP latencies. Further cohort studies may discover any possible correlation.

5. Conclusion

Optic nerve conduction diminished significantly in DM patients before any ophthalmoscopic change as an early sign of optic nerve or retinal damage.

Conflict of Interest

The authors declared that there is no conflict of interest.

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