

Original article:

Analysis of Electrophysiological parameters of Median nerve in Type 2 Diabetes Mellitus and Its Correlation with Fasting Blood sugar

¹Chethan K, ² Sowjanya T

¹ Assistant Professor, Department of Physiology, ESIC Medical College, Hyderabad, India

² Tutor, Department of Physiology, ESIC Medical College, Hyderabad, India

Corresponding author: Chethan K

Abstract

Introduction Diabetes mellitus is one of the most common metabolic disorder with a multi factorial origin. Diabetic neuropathy depends on both the duration as well as on level of control of blood sugars.

Material and methods: A total of 70 subjects were taken, 35 patients of Type 2 diabetes mellitus (DM) with fasting blood sugar (FBS) level ≥ 126 mg/dL and another 35 patients with FBS level ≤ 126 mg/dL. The entire recording was done using a four channel AD instrument and Compound Muscle Action Potential (CMAP) was recorded by using the LabChart software in both the groups.

Results: We found that there was a significant ($P < 0.05$) decrease in the nerve conduction velocity among the Diabetic population with FBS level ≥ 126 mg/dL (50.73 ± 16.07) compared to the Type 2 DM with FBS level ≤ 126 mg/dL (59.35 ± 14.50). There was a non significant change in the Amplitude ($P > 0.05$) among the Diabetic population with FBS level ≥ 126 mg/dL (455.83 ± 250.94) compared to the Type 2 DM with FBS level ≤ 126 mg/dL (555.22 ± 263.00). There was a non significant change in the Distal latency ($P > 0.05$) among the Diabetic population with FBS level ≥ 126 mg/dL (0.0045 ± 0.0014) compared to the Type 2 DM with FBS level ≤ 126 mg/dL (0.0044 ± 0.0015).

Conclusion: Thus from our study, we would like to conclude that there is a link between the glycemic control of diabetes and nerve conduction velocity.

Key words-Diabetes mellitus, Nerve conduction velocity, Fasting blood sugar

Introduction:

Diabetes mellitus is one of the most common metabolic disorder with a multifactorial origin like genetic factors, altered food habits and sedentary life style. Its incidence is increasing at an alarming rate(1). About 415 million people live with diabetes worldwide, and an estimated 193 million people have undiagnosed diabetes. Type 2 diabetes accounts for more than 90% of patients with diabetes(2).

Due to advent of Oral hypoglycemic agents and insulin, mortality rate has decreased but morbidity is increasing. Diabetic polyneuropathy characterized by structural and functional disturbances in the micro-vascular system since it causes injury to the small vessels supplying nerves (vasa nervorum). It increases oxidative stress and adversely affects glycosylation of axonal, trophic factors and micro-vascular proteins needed for peripheral nerves and their ganglia. This results in damage to the peripheral nerves and development of diabetic neuropathies(3).

The progression of Diabetic neuropathy has a long asymptomatic stage. Diabetic polyneuropathy is under diagnosed most of the times as it is a subjective phenomenon. So it is necessary to detect it in the asymptomatic phase itself as

diabetics are at a high risk of developing foot complications like ulcerations, gangrene and infections which might require limb amputation if not detected early. The diagnosis must be made with very careful clinical examination of the lower back and even if symptoms are absent, it cannot be considered as an absence of signs. Early identification and having a good control over the blood sugar level play important roles in preventing diabetic neuropathy(4).

Nerve Conduction Studies help in detecting diabetic neuropathies at the earlier stages. Nerve conduction velocity is diminished and amplitude potentials are reduced in early diabetic neuropathy(5). There is a correlation between glycemic control and duration of diabetes on development of long term complications(6). In order to quantify the status, nerve conduction studies are useful. So, in our study we used nerve conduction study to see the effect of fasting blood sugars on nerve conduction.

Materials & methods:

This study was undertaken in the Department of Physiology, ESIC Medical College, Hyderabad, after approval from the research and ethical committees. A total of 70 subjects visiting ESIC Hospital were taken, 35 patients of type 2 diabetes mellitus with duration of diabetes 5 years or more, with a fasting blood sugar ≤ 126 mg/dL and another 35 patients suffering from Type 2 diabetes mellitus with fasting blood sugar level ≥ 126 mg/dL. Detailed history regarding symptoms like paraesthesia, tingling sensation, burning feet, hyperaesthesia was taken, history of weakness and gait abnormality was noted. All Type 2 diabetic patients diagnosed in such a manner, were subjected to the nerve conduction study.

On arrival of the patients the details of study were explained and written informed consent was obtained. The height and weight of the patients were noted. The entire recording was done using a four channel AD instrument.

The patient was asked to be seated comfortably and the recording electrodes were placed on the thenar eminence of the palm. The bar stimulus electrode was positioned on the Lateral aspect of the forearm near the wrist joint, and stimulated with the current strength of 8 mA. The Compound Muscle Action Potential (CMAP) was recorded by using the LabChart software. Then the bar electrode was placed on the ventral part of the elbow, near the elbow joint. The difference between the onset latencies was noted.

Distance was measured between the two stimulation points and the velocity was calculated using the formula:

$$\text{Velocity} = \text{Distance travelled} / \text{time taken}$$

Latency, amplitude and nerve conduction velocity were assessed. The data was summarized to test the difference in the mean values between the groups 1, 2 by using the Student's 't' test; p values < 0.05 were taken as the level of significance.

Results:

The study included total of 70 subjects, 35 patients of type 2 DM with duration of diabetes 5 years or more, with a fasting blood sugar level ≤ 126 mg/dL, and another 35 patients suffering from Type 2 diabetes mellitus with fasting blood sugar level ≥ 126 mg/dL. Both the groups were aged between 40 to 70 years [Table 1]. We found that there was a significant ($P < 0.05$) decrease in the nerve conduction velocity among the Diabetic population with fasting blood sugar level ≥ 126 mg/dL (50.73 ± 16.07) compared to the Type 2 DM with fasting blood sugar level ≤ 126 mg/dL (59.35 ± 14.50) [Table 2/Chart 1]. There was a non significant change in the Amplitude ($P > 0.05$) among the Diabetic population with fasting blood sugar level ≥ 126 mg/dL (455.83 ± 250.94) compared to the Type 2 DM with

fasting blood sugar level ≤ 126 mg/dL (555.22 ± 263.00). There was a non significant change in the Distal latency ($P > 0.05$) among the Diabetic population with fasting blood sugar level ≥ 126 mg/dL (0.0045 ± 0.0014) compared to the Type 2 DM with fasting blood sugar level ≤ 126 mg/dL (0.0044 ± 0.0015).

Table 1: Age (in years) comparison between study group and control group

Group	N	Mean	Std. Deviation	
Type 2 DM with fasting blood sugar level ≥ 126 mg/dL	35	51.4	9.48	P = 0.125
Type 2 DM with fasting blood sugar level ≤ 126 mg/dL	35	54.85	9.18	

Unpaired t test, $p > 0.05$

Table 2:

Variable	Type 2 DM with fasting blood sugar level ≥ 126 mg/dL		Type 2 DM with fasting blood sugar level ≤ 126 mg/dL		P value
	Mean	SD	Mean	SD	
1. Amplitude (μ V)	455.83	250.94	555.22	263.00	0.11
2. Distal Latency (s)	0.0045	0.0014	0.0044	0.0015	0.075
3. Nerve conduction velocity (m/s)	50.73	16.07	59.35	14.50	0.021*

*Unpaired t test *significant*

Chart 1:

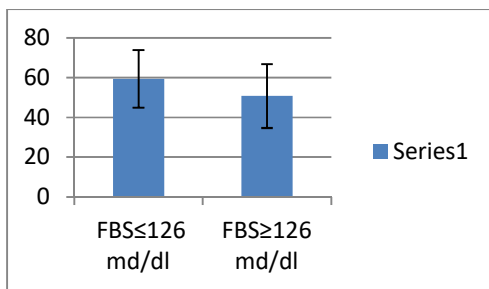


Chart:2

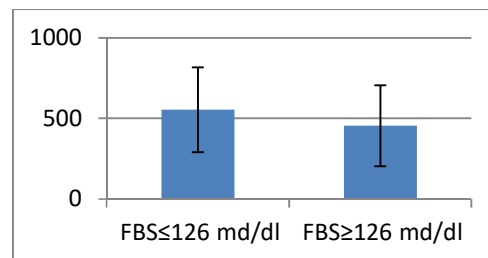
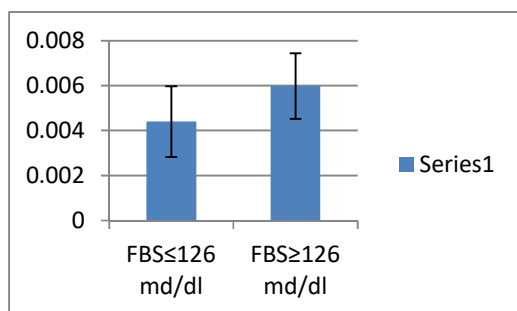


Chart 3:



Discussion:

In this study we compared the nerve conduction velocities of thirty five Type 2 diabetic patients with fasting blood sugar level ≥ 126 mg/dL (mean age 51.4 ± 9.48 years) and of thirty five Type 2 diabetic patients with fasting blood sugar level ≤ 126 mg/dL (mean age 54.85 ± 9.18 years). In our study, we found there was a significant ($P < 0.021$) decrease in Median nerve conduction velocity in Type 2 diabetic patients with patients with fasting blood sugar level ≥ 126 mg/dL.

The decrease in the median nerve conduction velocity found in our study is in accordance with the study done by Farheen A et al(6). In their study they found nerve conduction velocity progressively decreased from the controls to diabetics with good glycaemic control, to the diabetics with poor glycaemic control. Yet, another study also correlates with our findings which implies there will be a decrease in nerve conduction velocity in diabetics with a good glycaemic control to the diabetics with a poor glycaemic control(7). Likewise in a study done by Prasad N et al also showed decreased amplitude and conduction velocities of median nerve of both sides in diabetics and found correlated with blood sugar levels(8).

So, from our study it is clear that there is a link between glycaemic control in Type 2DM with the nerve conduction velocity. The limitation of the present study was that small number of diabetic patients was covered. So, in future studies large number of diabetic population can be included with other parameters to observe the effects on nerve conduction velocity.

Conclusions:

The study was conducted on 70 subjects, among which 35 were Type 2 diabetic with fasting blood sugar level ≥ 126 mg/dL and the rest were Type 2 diabetic with fasting blood sugar level ≤ 126 mg/dL. There was a comparatively significant decrease in nerve conduction velocity among the patients with fasting blood sugar level ≥ 126 mg/dL. Thus from our study, we would like to conclude that there is a link between the glycaemic control of diabetes and nerve conduction velocity. A more detailed study involving more number of patients with Type 2DM of different duration is warranted to come to a definite conclusion.

Acknowledgment:

We would like to thank the first year medical students of ESIC Medical College, Sanathnagar, Hyderabad.

References:

1. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian scenario. *Indian J Med Res.* 2007;
2. Chatterjee S, Khunti K, Davies MJ. Type 2 diabetes. *The Lancet.* 2017.
3. Zaidi AS, Singh P, Gupta M, Siddiqi SS, Masood A. Impact of Long versus Short Duration Diabetes on Sensory Nerve Conduction Velocity in Type 2 Diabetes Mellitus. *J Clin Diagnostic Res [Internet].* 2018;12(8):17–20. Available from: http://jcdr.net/article_fulltext.asp?issn=0973-709x&year=2018&volume=12&issue=8&page=CC17&issn=0973-709x&id=11930
4. Yadav N, Shete A, Yadav P, Yadav N, Khan ST. Study Of Nerve Conduction Velocity In Type II Diabetes Mellitus. *Natl J Integr Res Med.* 2015;6(4):36–43.
5. Sultana MS, Begum N, Ali L, Mosharraf Hossain GM, Bhowmik NB, Parveen S, et al. Electrophysiological Changes of Sensory Nerves In Patients with Type-2 Diabetes Mellitus of Different Duration. *Bsmmu J [Internet].* 2010;3(1):9–12. Available from: <https://www.banglajol.info/index.php/BSMMUJ/article/viewFile/5507/4311>
6. Farheen A, Malipatil BS, Arif G. Original Article Nerve Conduction in Type 2 Diabetics and Its Correlation With. *J Evol Med Dent Sci.* 2015;4(06):1023–34.
7. Shekharappa KR, Srinivas AK, Vedavathi KJ, Venkatesh G. A Study on the Utility of Nerve Conduction Studies in Type 2 Diabetes Mellitus. *J Clin Diag Res.* 2011;5(3):529–31.
8. Prasad N, Pisharody IK, Karandikar MS, Diwanji SA, Raghav PR. Comparative Analysis of Electrophysiological Parameters of Median Nerve in Normal and Diabetic Subjects. 2013;(July):261–4.