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Does Controlled And Uncontrolled Type IIDiabetes Mellitus Affect Median Nerve Conduction Parameters? - An Analytical Study.

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Abstract:Background: *Diabetic neuropathy is one of the most commonly occurring micro vascular complications.Glycatedhaemoglobin (HbA1c) has been established as a marker of glycemic control and it also indicates the risk of developing small vessel complications.Nerve conduction studies are useful in early evaluation in diabetic peripheral neuropathy. Therefore, this study was undertaken to assess the nerve conduction study results among known cases of type II diabetes mellitus with uncontrolled glycemic (HbA1C elevated), diabetics with controlled glycemic (HbA1C at normal level) and to compare it with non-diabetic subjects.*

Methods:*The study was conducted in 60 male subjects in age group of 30-45 years. Subjects were subdivided into 3 groups-Group 1: 20 Type II diabetes mellitus patients with uncontrolled diabetes;Group 2:20 Type II diabetes mellitus patients with controlled diabetes; Group 3: 20 non-diabetic healthy subjects. Nerve conduction velocity, amplitude and latency of right median nerve (motor and sensory component) were recorded by computerized RMSEMG System.*

Results*: Median nerve conduction study showed significant increase in mean latency and significant decrease in amplitude and velocity values in uncontrolled diabetics when compared with both controlled diabetic and non-diabetic group. No significant difference in mean latency, amplitude and velocity values was observed when controlled diabetic group was compared with non-diabetic group*

Conclusions:*Electrophysiological parameters in median nerve conduction (both sensory and motor component) deteriorate both in uncontrolled and controlled type II diabetic mellitus patients as compared to non-diabetics but more significantly in uncontrolled diabetics. Monitoring of HbA1C can be taken as an index for predicting neuropathic changes in type II diabetics.*

Keywords –*Type II diabetes mellitus, Nerve conduction velocity, amplitude, latency.*

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I. Introduction

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia with disturbances of carbohydrate, fat and protein metabolism caused by either lack of insulin secretion or decreased sensitivity of the tissues to insulin.[1] Patients with long standing diabetes are at risk for developing various complications. There are two types of diabetic complications–micro vascular and macro vascular. Micro vascular complications include nephropathy, retinopathy, neuropathy and diabetic foot[2] while macro vascular complications include cerebrovascular, peripheral vascular and cardiovascular diseases. [3] Diabetic neuropathy is one of the most commonly occurring micro vascular complications accounting for 28% of all the complications in diabetics.[4]The prevalence of diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with the maximum increase in India.[5] The prevalence of diabetic peripheral neuropathy among type 2 diabetics within Indian population has been reported as 33.33 %.[6] It is known that poor glycemic control is responsible for micro vascular complications.[7]Glycatedhaemoglobin (HbA1c) has not only been established as a marker of glycemic control but it also indicates the risk of developing small vessel complications.[8] Nerve conduction studies are most sensitive, specific, non-invasive and repeatable, hence useful in early evaluation in diabetic peripheral neuropathy. Mostly nerve conduction studies have been accepted as an essential part of diagnosis for diabetic peripheral neuropathy as it has many benefits. Therefore, this study was undertaken to assess the nerve conduction study results among known cases of diabetes mellitus with uncontrolled glycemic

(HbA1C elevated), diabetics with controlled glycemic (HbA1C at normal level) and to compare it with non-diabetic subjects. Thus aim of the study is to study the nerve conduction parameters in diabetes mellitus patients.

II. Material And Methods

The present study was an analytical study.Permission from the institutional ethics committee was taken before the initiation of the study. Study was carried out in 60 male subjects in the age group of 30-45 years.This study was undertaken in the Department of Physiology in collaboration with Department of Medicine of a government medical college and city based tertiary care hospital in a metropolitan city. Patients attending endocrinology OPD of the hospital were thoroughly interviewed by using a standard proforma.

Male subjects in age group of 30-45 years, with diagnosed type II diabetic mellitus since 5-10 years were included in the present study.

Subjects with family history of peripheral nerve disease; history of any known disease causing neuropathy; subjects consuming alcohol or drugs with potential neurotoxic effects; hypertensive subjects; type I diabetic mellitus patients; subjects with vitamin B12 deficiency or folate deficiency and smokers were excluded from the study.

Finally 40 diagnosed Type II diabetic patients attending endocrinology OPD and 20 healthy age and BMI matched non diabetics were selected for the present study.

On the basis of Glycated hemoglobin level [9] diabetic patients were divided into two groups, while non-diabetic subjects were included in group 3.

Group	Criteria	Number of subjects
Group 1	Type II diabetes mellitus patients with uncontrolled diabetic (HbA1C1c ≥ 7.6)	20
Group 2	Type II diabetes mellitus patients with control diabetic (HbA1C = 5.5 – 6.8).	20
Group 3	Non-diabetic healthy subjects (HbA1C= 4.2 - 6.2).	20

The procedure was explained to all the participants and informed consent was obtained from them. Detailed clinical examination was carried out in all participants and basic parameters (height, weight, blood pressure) were noted with reference to standard proforma. Three parameters i.e. nerve conduction velocity, amplitude and latency of right median nerve (motor and sensory component) were recorded by Computerized RMSEMG System (India), asper standard procedure described by Mishra UK , Kalita J.[10] Calculations and results were interpreted using Graph Pad Prism 5 statistical software.One way Anova test and Bonferroni's Multiple Comparison Test were applied for statistical analysis.

The values were expressed as mean ±SD. p value of < 0.05 was considered to be statistically significant. p value of < 0.001 was considered to be statistically highly significant.

III. Results

Table 1: Baseline characteristics of groups.

Parameters	**Groups**			Anova test p value
	Group 1(G1) n = 20 Mean ± SD	Group 2(G2) n = 20 Mean ± SD	Group 3(G3) n = 20 Mean ± SD	
Age (years)	39.30 ± 4.19	38.90 ± 4.35	38.85 ± 3.30	p>0.05
Body Mass Index (kg/m^2)	23.55 ± 1.25	23.28 ± 1.12	22.90 ± 1.16	p>0.05
HbA1c (%)	8.19 ± 0.83	5.83 ± 0.59	5.38 ± 0.44	p < 0.05

Table 2: Post hoc - Bonferroni's Multiple Comparison Testto show differences in glycaemic status of groups.

.	'p' value		
Parameters	**G1 Vs G2**	**G1 Vs G3**	**G2 Vs G3**
HbA1c (%)	< 0.001 **	< 0.001**	>0.05

Table 3: Comparison of mean values of median (motor) nerve and median (sensory) nerve parameters of groups by ANOVA test.

Right median nerve	**Parameters**	**Groups**			Anova test p value
		Group 1(G1) n = 20 (Mean ± SD)	Group 2(G2) n = 20 (Mean ± SD)	Group 3(G3) n = 20 (Mean ± SD)	
Median (Motor) nerve	Latency (ms)	4.43 ± 0.93	4.03 ± 0.70	3.66 ± 0.65	<0.001**
	Amplitude (μV)	6.19 ± 2.37	8.23 ± 1.80	9.49 ± 1.87	<0.001**
	Velocity(m/s)	47.12 ± 13.44	52.06 ± 10.46	58.45 ± 12.99	<0.001**
Median (Sensory) nerve	Latency (ms)	3.69 ± 0.50	3.30 ± 0.34	3.02 ± 0.34	<0.001**
	Amplitude (μV)	8.25 ± 1.50	11.49 ± 1.46	15.02 ± 2.54	<0.001**
	Velocity (m/s)	43.22 ± 7.46	47.69 ± 6.96	53.19 ± 8.50	<0.001**

Table 4: Post hoc Bonferroni's Multiple Comparison Test to show differences in median (motor) nerve and median (sensory) nerve conduction parameters in groups

Right median nerve	**Para-meters**	**'p' value**		
Median (Motor) nerve		**G1VsG2**	**G1 VsG3**	**G2VsG3**
	Latency (ms)	<0.05*	<0.001**	>0.05
	Amplitude (μV)	<0.001**	<0.001**	>0.05
	Velocity (m/s)	<0.05*	<0.001**	>0.05
Median (Sensory) nerve	Latency (ms)	<0.05*	<0.001**	>0.05
	Amplitude (μV)	<0.001**	<0.001**	>0.05
	Velocity (m/s)	<0.001**	<0.001**	>0.05

*p<0.05 = statistically significant, **p<0.001= statistically highly significant, p >0.05 = not significant

IV. Discussion

In the present study the effect of diabetes of duration 5-10 years on nerve conduction parameter values in 40 diabetic male patients were studied and compared with values of 20 non- diabetic male subjects, in the age group 30-45 years. There was no difference in mean values of age and body mass index (BMI) in all the groups.That means all the groups were comparable with respect to these parameters. However HbA1c (%) was significantly higher in group 1 as compared to group 2 and group 3.(Table 1) In the present study, nerve conduction parameters (mean latency, amplitude and velocity) of both motor and sensory components of right median nerve were found to be significantly altered in uncontrolled diabetic group. We observed significant increase in mean latency, and significant decrease in amplitude and velocity values, in uncontrolled diabetic groupwhen compared with controlled diabetic group. When uncontrolled diabetic group was compared with non-diabetic group, we observed highly significant increase in mean latency and highly significant decrease in amplitude and velocity values in uncontrolled diabetics. However when controlled diabetic group was compared with non-diabetic group, we observed no significant difference in mean latency, amplitude and velocity values. Similar results were observed by Abhijeet AA et al.[11] and W. Hoffman et al.[12] In hyperglycaemia, oxidative stress leads to endothelial dysfunction and decreased capillary blood flow which in turn leads to endoneurial hypoxia causing death of nerve cells and so nerve conduction parameters are altered.[13] Pathological changes in peripheral neuropathy due to diabetes mellitus are characterized by segmental demyelination, axonal degeneration, or a combination of the two.[14]

Nerve conduction reflects several physiological components of peripheral nerve function including nerve size, degree of myelination, internodal distance, diameter of axons and nerve temperature. Nerve conduction velocity reflects integrity of the myelin sheath and indicates transmission time in the large myelinated nerve fibers.[15]

Amplitude reflects the size and number of nerve fibers, and its measurement is important for the evaluation of neuropathy. Both latency and conduction velocity depend on an intact, myelinated nerve as myelin and the saltatory conduction are essential for fast action potential propagation in normal subjects. In contrast, the amplitude of the waveform depends primarily on number of axons functioning within the nerve.[16]Slowing of conduction velocity or prolongation of latency usually implies demyelinating injury, while loss of amplitude usually correlates with axonal loss or dysfunction.[17]

Thus the present study concludes that electrophysiological parameters in median nerve conduction (both sensory and motor component) deteriorate both in uncontrolled and controlled type II diabetic mellitus patients as compared to non-diabetics but more significantly in uncontrolled diabetics. Monitoring of HbA1C can be taken as an index for predicting neuropathic changes in type II diabetics. Early detection and management of diabetic peripheral neuropathy can improve quality of life among patients.

References

[1]. Alvin C Powers. Editors: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, et al. Diabetes Mellitus Harrison's Principles of Internal Medicine, 17th edition. New York: The McGraw-Hill, health professions division; 2008.p. 2275-304.

[2]. Walker BR, Colledge NR, Ralston SH, Penman I (Eds) Davidsons principles and practice of medicine. 22th edition. Churchil Livingstone: pp 797-828, 2014.

[3]. Mundet X, Pou A, Piquer N et al. Prevalence and incidence of chronic complications and mortality in a cohort of type 2 diabetic patients in Spain. *Primary Care Diabetes* 2: 135-140, 2008.

[4]. Tesfaye S, Stevens LK, Stephenson JM, Fuller JH, Plater M, Ionescu-Tirgoviste C et al. Prevalence of diabetic peripheral neuropathy and its relation to glycemic control and potential risk factors. The Euro Diab IDDM complications study. *Diabetologia*1996; 39: 1377-1384.

[5]. Wild S, Roglic G, Green A, Sicree R, King H (2004) Global prevalence of diabetes. Diabetes Care 27:1047–1053.

[6]. Pawde P, Thampi R, Renish RK, Resmi RU, Vivek MR (2013) Prevalence and risk factors of diabetic peripheral neuropathy among type-2 diabetic patients presenting to SMIMS hospital, Kulasekharam, Kanyakumari district, Tamil Nadu, India. Int J Med Sci Public Health 2(1):73

[7]. Mayurasakorn K, Somthip N, Caengow S, Chulkarat N, Wanichsuwan M. Glycemic control and microvascular complications among type 2 diabetes at primary care units. *J Med Assoc Thai* 2009; 92:1094-101.
[8]. Chandalia HB, Krishnaswamy PR. Glycated hemoglobin. *CurrentScience*2002; 83: 1522-1532.
[9]. Nathan, D.M., The Clinical information value of the glycosylated Assay. The New England Journal of Medicine. 1984; 310: 341-346.
[10]. UK Mishra, J Kalita, Nerve Conduction Study. Clinical Neurophysiology, second edition. Elsevier. 2012.
[11]. Abhijeet AA, Anant AD et al. Clinical profile of peripheral neuropathy in diabetes mellitus by nerve conduction study. Ach H App Med Aci. 2014; 2(6A):1973-1977.
[12]. W.Hoffman, Z Hart, R Frank. Correlates of delayed motor nerve conduction and retinopathy in juvenile onset diabetes mellitus. The journol of Pediatrics. 2009; 102 (3): 351-356.
[13]. Abdulsalam A, Al-Sulaiman, Ismail HM, Al- Sultan AI. Electrophysiological findings in newly diagnosed non insulin – dependent diabetics: A prospective study. Annals of Saudi Medicine 1997; 17(4): 399-401.
[14]. Parveenkumar, Michael clark. Diabetes mellitus and other disorders of metabolism, clinical medicine. Sixth edition. Saunders Elsevier; 2005. p. 1101-1134.
[15]. Bansal V, Kalita J, Mishra UK. Diabetic neuropathy. Postgrad Med J. 2006; 82: 95-100.
[16]. Clayton W, Elasy TA. A review of the pathophysiology, classification and treatment of foot ulcers in diabetic patients. Clinical Diabetes.2009; 27(2): 52-58
[17]. Parveenkumar, Michael clark. Diabetes mellitus and other disorders of metabolism, clinical medicine. Sixth edition. Saunders Elsevier; 2005. p. 1101-1134.

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