www.jmscr.igmpublication.org

Impact Factor 3.79
ISSN (e)-2347-176x

IGM Publication

Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Electrophysiological Assessment of Somatic Nerves of Upper Limbs in Diabetics: A Motor Nerve Conduction study

Authors

Neelam B Prasad [1], Indu K Pisharody[2], S A Diwanji[3]

[1]Dept of Physiology, Dr. D. Y. Patil, Medical College, Hospital and Research Center, DPU Pune
[2] Dept of Physiology, Career Institute of Medical Sciences and Hospital, Lucknow
[3]Dept of Physiology, B.K.L. Walawalkar, Rural Medical College, Sawarda, Chiplun

Corresponding Author

Neelam B Prasad

Associate Professor, Dept of Physiology, Dr. D. Y. Patil. Medical College, Hospital and Research Center, DPU, Pune

Email: *neelamprasad8@gmail.com*

ABSTRACT

Assessment of motor functions of somatic nerves of upper limb was carried out in diabetics by nerve conduction studies (NCS). Diabetic neuropathy is one of the major causes of morbidity and disability in the diabetic patients. Early detection is needed to prevent further complications from diabetic neuropathy. The median and ulnar motor nerves were chosen for the study. Conduction study was carried out on 100 male type 2 diabetic patients and 100 healthy male volunteers who served as control. Distal latency, Amplitude and Conduction Velocity of nerves were measured by using computerized EMG/ NCV/EP Mark II and surface electrodes. On comparing the parameters of NCS it was found that distal latency of both the nerves was more in diabetics than controls with statistically significant difference. The amplitude was significantly decreased in diabetics except right ulnar nerve. The conduction velocities of nerves of both sides in diabetics were also found to be decreased which was statistically significant.

Keywords-. *Type 2 diabetes mellitus, peripheral neuropathy, median nerve, ulnar nerve, distal latency, conduction velocity*

INTRODUCTION

Modern medical care suggests various life style changes and pharmaceutical intervention in diabetics which have improved the quality of life and increased the life expectancy thus falling prey to the long term complications in them. Among various long term complications, diabetic peripheral neuropathy (DPN) is one of the major disabling and costly complication of diabetes mellitus. It affects up to 50% of patients and predisposes the patients to severe functional limitations [1]. It is known to be heterogenous by symptoms, pattern of neurologic involvement, course, pathologic alterations and underlying mechanism. Neuropathy may be silent and may go undetected or it can manifest with clinical symptoms and signs that mimic those seen in many other diseases [2],[3].Although there are multiple methods for detecting and monitoring DPN, nerve conduction studies(NCS) are generally considered to be most sensitive and reproducible[4]

NCS has the potential for early diagnosis of neuropathy. To fully realize this potential, increasingly sophisticated technology has been incorporated into devices that perform NCS [5].This study was planned to detect the occurrence of subclinical neuropathy by nerve conduction studies in type 2 diabetic patients. Screening and diagnostic testing for neuropathy in patients with type 2 diabetes will be helpful in order to prevent complications from diabetic neuropathy.

MATERIALS AND METHODS

The study was conducted in the Department of Physiology of Dr. D. Y. Patil Medical College, Hospital and Research Center, Pune (Maharashtra, India) on patients of type 2 diabetes mellitus attending diabetes clinic. Design of the study was cross sectional. In this study motor nerve conduction of median and ulnar nerve was performed on 100 Type 2 diabetic male patients with age ranged 40-65 years. Mean duration of disease was 5.4$\pm$3 years. These patients were compared with 100 apparently healthy controls, age, sex and anthropometrically matched. All the patients with chronic musculoskeletal disorders, retinopathy, nephropathy or chronic disease, alcoholics and smokers were excluded from the study. Detailed socio- demographic data, family history and medical history were taken from all the subjects and their physical and clinical examinations were done on the very first day of visit to OPD. The details of study were explained in the language they understood and an informed consent was taken from each of the subjects. On the day of test, blood samples were collected for blood sugar estimation which was following nerve conduction study.

Anthropometric measurements (height and weight) were taken by using scales on bare foot. Both fasting and post prandial blood glucose levels were estimated by glucose oxidase (GOD/POD) method [6]. Motor nerve conduction study of median and ulnar nerves were performed on both sides of the body in an environment with room temperature ranging from 23^0C to 25^0C using Neuro perfect software on windows based computerized EMG/ NCV/EP Mark II system supplied by Recorders And Medicare Systems, Chandigarh, India and surface electrodes. By standard surface stimulating and recording techniques, peripheral nerve was electrically stimulated and recording was obtained from a muscle supplied by this nerve. Electrodes were coated with electroconductive gel and held in place with adhesive tape.

With the help of stimulating electrodes supramaximal stimulation was given at two different sites (distal site and proximal site) to obtain compound muscle action potential (CMAP). The time it takes for the electrical impulse to travel from the stimulation to the recording site was measured. This value was called the latency and was measured in milliseconds (ms).Both the latencies (distal latency and proximal latency) were obtained for calculating conduction velocity. Amplitude of CMAP was measured in millivolt (mV). For median motor study the distal stimulation (S1) was given at middle of the wrist between tendon of flexor carpi radialis and palmaris longus whereas proximal stimulation (S2) was given at anterior cubital fossa over the brachial artery pulse. Active electrode for recording CMAP was placed over abductor pollicis brevis muscle and reference electrode was placed 3 cm distal to the active electrode at first metacarpophalangeal joint. For ulnar motor study the distal stimulation (S1) was given on medial aspect of wrist adjacent to flexor carpi ulnaris and proximal (S2) stimulation was given at elbow joint, 3-4 cm distal to medial epicondyle. Active electrode for recording CMAP was placed over muscle belly of abductor digiti minimi and reference electrode was placed 3 cm distal to the active electrode at 5th metacarpophalangeal joint. Ground electrode was placed between stimulating electrode and recording electrode for both the nerves. Distance between S1 and S2 was measured in millimeter by measuring tape for both the nerves for calculation of conduction velocity. Distal latency, Amplitude and Conduction Velocity were measured. Conduction velocity of nerve was calculated by dividing distance between S1 & S2 with the difference between two latencies. It was expressed in meter/second(m/s).[7]

Statistical Analysis

All the test results obtained were expressed in Mean ± SD (standard deviation). Statistical analysis of data was done using t test and Microsoft office Excel 2007. For all the analysis probability values (p value) < 0.05 were considered as statistically significant and p value < 0.001 were considered as statistically highly significant. The study was approved by the Ethics Committee of our institution.

RESULTS

In the present study 100 male diabetic subjects with mean duration of disease 5.4±3 years were compared with 100 non diabetic (control) subjects of same age group and sex. The baseline characteristics of subjects are summarized in table 1. On comparing the parameters of motor nerve conduction of nerves between both the groups (summarized in table 2) it was observed that the distal latency of median and ulnar nerves on both sides was significantly higher (p<0.001) in diabetics. The amplitude of CMAP was found to be significantly decreased in diabetics except right ulnar nerve. Further conduction velocities of median and ulnar nerves of diabetics were also found to be significantly (p<0.001) decreased in diabetics.

Table 1: Baseline Characteristics Of Study Population

Characteristics	Diabetics Mean ± SD	Non Diabetics Mean ± SD
Participants(n)	100	100
Age(years)	55.65 ± 7	53.88 ± 6.44
Weight(Kg)	72.15 ± 12.65	69.92 ± 13.18
Height(m)	1.70 ± 0.04	1.71 ± 0.04
Fasting plasma glucose (mg%)	138.11 ± 33.82	96.46 ± 9.53
2-hour plasma glucose (mg%)	230.88 ± 66.23	126.55 ± 12.63
Duration of Disease (Years)	5.4±3	

Table 2: Parameters of motor nerve conduction of Diabetics and Non-Diabetics

Parameters	Subjects	Median		Ulnar	
		Left	Right	Left	Right
Distal Latency (ms)	Diabetics Mean±SD	2.61 ± 0.63	2.60 ± 0.68	1.81 ± 0.50	1.54 ± 0.45
	Non Diabetics Mean±SD	2.21 ± 0.49	2.14 ± 0.49	1.61 ± 0.51	1.35 ± 0.46
	P value	0.000	0.000	0.009	0.004
Amplitude (mV)	Diabetics Mean±SD	16.49 ± 5.15	15.41 ± 4.33	8.06 ± 1.73	8.20 ± 1.13
	Non Diabetics Mean±SD	20.09 ± 4.97	18.60 ± 3.82	8.64 ± 1.63	8.25 ± 1.54
	P value	0.000	0.000	0.01	0.84
Conduction Velocity (m/s)	Diabetics Mean±SD	55.16 ± 5.49	55.87 ± 6.68	57.60 ± 10.93	53.85 ± 8.98
	Non Diabetics Mean±SD	58.62 ± 6.10	59.83 ± 6.18	63.53 ± 8.72	60.55 ± 6.57
	P value	0.000	0.000	0.000	0.000

P< 0.05 statistically significant.

P< 0.001 statistically highly significant.

DISCUSSION

In the present study the motor nerve of diabetic patients has shown significant decrease in electrophysiological functions as compared with non diabetics. This finding is consistent with results of Fagerberg et al. and Gregersen, who reported that motor defects are common in diabetics with neuropathy[8],[9].Our findings support the observation of Sultana S et.al. They also noticed significant reduction in amplitude and conduction velocity in motor nerves of diabetic group with short duration of diabetes [10].Several mechanisms have been suggested by which hyperglycemia results in nerve damage in diabetes. Hyperglycaemia induces rheological changes, which increases endothelial vascular resistance and reduces nerve blood flow. Hyperglycaemia also causes depletion of nerve myoinositol through a competitive uptake mechanism [11]. Moreover, activation of polyol pathway in the nerve through enzyme aldose reductase leads to accumulation of sorbitol and fructose in the nerve and induces non-enzymatic glycosylation of structural nerve proteins. Hyperglycaemia also induces oxidative stress and activation of protein kinase C. Activation

of protein kinase C has been linked to vascular damage in DPN [12]. These changes result in abnormal neuronal, axonal, and Schwann cell metabolism, which result in impaired axonal transport. Endoneural hypoxia is produced by increased vascular resistance and reduced blood flow in the nerve. Hypoxia leads to further capillary damage, which in turn aggravates disturbance in axonal transport and reduced Na-K ATPase activity leading to axonal atrophy and impairment of nerve conduction [11] Thus the cause of diabetic neuropathy though remains unclear, ischemic and metabolic components cannot be ruled.

Many previous studies have also found nerve conduction study alterations suggestive of neuropathy in diabetics. Kimura J et al also found increased latency and decreased conduction velocity in lower limb nerves in diabetics as compared to normal subjects [13]. W. Hoffman et al found significantly slower conduction velocity in diabetics in both upper and lower limb nerves [14].Though not significant, but tendency for reduction of ulnar motor nerve conduction velocity was found in patients of diabetes without neuropathy when compared with non-diabetic healthy controls in a study by Hussain Gauhar et al [15]. Median and ulnar nerves in diabetics are less studied and require further evaluation.

CONCLUSIONS

In diabetics there was significant deterioration in motor nerve conduction parameters as compared to non diabetics. Screening and diagnostic testing for neuropathy in patients with type 2 diabetes is needed in order to prevent complication from diabetic neuropathy.

REFERENCES

1. Vileikyte L, Leventhal H, Gonzalez JS, Peyrot M, Rubin RR, Ulbrecht JS, Garrow A, Waterman C, Cavanagh PR, Boulton AJ. Diabetic peripheral neuropathy and depressive symptoms: the association revisited. Diabetes Care 2005;28(10):2378-83.).
2. Dyck PJ, Kratz KM, Karnes JL, Litchy WJ, Klein R, Pach JM, et al. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. Neurology 1993;43:817–24
3. Llewelyn JG, Tomlinson DR, Thomas PK. Diabetic neuropathies. In: Dyck PJ, Thomas PK, editors. Peripheral neuropathy. 4th ed., Philadelphia: Elsevier; 2005. p. 1951–92.
4. Perkins BA, Olaleye D, Zinman B, Bril V. Simple screening tests for peripheral neuropathy in the diabetes clinic. Diabetes Care.2001;24(2):250-6.
5. Gozani SN, Fisher MA, Kong X, Megerian JT, Rutkove SB.Electrodiagnostic automation: principles and practice. Phys Med Rehabil Clin N Am. 2005;16(4):1015-32)
6. Teitz, N.W. Fundamentals of Clinical Chemistry (243), W.B. Saunders & Co. Philidelphia PA 1972
7. UK Mishra and J Kalita. Clinical Neuophysiology.2nd Ed. B.I.Churchill Livingstone PvtLtd.New Delhi.1999
8. Fagerberg SE, Peterson I, Steg G, Wilhelmsen L. Motor disturbances in diabetes mellitus. A clinical study using electromyography and nerve conduction velocity determination. Acta Medica Scandinavica 1963;174: 711–6
9. Gregersen G. Diabetic neuropathy: influence of age, sex, metabolic control and duration of diabetes on motor conduction velocity. Neurology 1967;17:972–80
10. Sultana S, Begum N,Ali L,Hossain MM,Bhowmik NB, Parveen Z. Electrophysiological changes of motor nerves in patients with type 2 diabetes mellitus. JAFMC Bangladesh.2009;5(5):14-17
11. Bansal V, Kalita J, Misra UK. Diabetic neuropathy. Postgraduate Medical Journal. 2006;82(964):95-100. doi:10.1136/pgmj.2005.036137
12. Kevin L. Farmer, Chengyuan Li, and Rick T. Dobrowsky. Diabetic Peripheral Neuropathy: Should a Chaperone Accompany Our Therapeutic Approach?Pharmacol

Rev. 2012 Oct; 64(4): 880–900.doi: 10.1124/pr.111.005314

13. Kimura J , Yamada T, Stevland NP. Distal slowing of motor nerve conduction velocity in diabetic polyneuropathy. J Neurol Sci.1979 Jul;42(2):291-302.
14. W. Hoffman, Z Hart, R Frank. Correlates of delayed motor nerve conduction and retinopathy in juvenile-onset diabetes mellitus. The journal of Pediatrics. 2009;102(3):351-356.
15. Hussain Gauhar, Rizvi S. Aijaz Abbas, Hasan Abdul Rahman, Mir Abdul Munif, Singhal Sangeeta, Ahmad Jamal. Nerve conduction velocity as an early predictor of diabetic peripheral neuropathy in Type 2 Diabetes Mellitus patients. International Journal of Comprehensive Medical Physiology and Research March 2014 Vol 1 Issue 1.