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ELECTROPHYSIOLOGICAL ASSESSMENT OF ULNAR NERVE IN DIABETICS AND NON-DIABETIC PATIENTS: A NERVE CONDUCTION STUDY

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ABSTRACT

Background: In the diabetic patients, diabetic neuropathy is one of the major causes of morbidity and disability. Therefore early detection of diabetic neuropathy is needed to prevent further complications.

Objective: To assess ulnar nerve conduction parameters in diabetics with respect to non diabetic patients.

Materials and Methods: The cross sectional study was conducted on 50 male type 2 diabetic patients and 50 healthy male volunteers who served as control. Ulnar motor nerves were chosen for the study. Conduction study was carried out on distal latency, amplitude and conduction velocity of nerves were measured by using computerized EMG/ NCV/EP Mark II and surface electrodes.

Results: On comparing the electrophysiological parameters it was found that distal latency of the ulnar nerves was more in diabetics than controls with statistically significant difference. The amplitude was statisatically significant decreased in diabetics. The conduction velocities of nerves of both sides in diabetics were also found to be decreased which was statistically significant.

Conclusion: In diabetics there was significant deterioration in electrophysiological parameters as compared to non diabetics.

KEYWORDS : Type 2 diabetes, nerve conduction study, ulnar nerve

Introduction

Diabetes is a major public health problem globally with an increasing disease trend. 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.[1] There are many 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.[2] It is known to be heterogeneous 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.[3,4] Although there are multiple methods for detecting and monitoring DPN, nerve conduction studies(NCS) are generally considered to be most sensitive and reproducible[5] 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.[6] Screening and diagnostic testing for neuropathy in patients with type 2 diabetes will be helpful in order to prevent complications from diabetic neuropathy.[7] Therefore the present study was planned to detect the occurrence of subclinical neuropathy by nerve conduction studies in type 2 diabetic patients.

Materials and Methods

Design of study was cross sectional. The study protocol was approved by Institutional Ethics Committee of SN Medical College, Bagalkot, Karnataka before the study being started. The details of study were explained and informed consent was taken from each of the subjects. The study was conducted in the Department of Physiology, SN Medical College and HSK Hospital and Research Centre, Bagalkot, Karnataka in collaboration with Tulsigirish Diabetes Hospital and Diabetes Research Foundation Bagalkot, Karnataka and Department of Biophysics, Government Institute of Science, Aurangabad, Maharashtra. The study duration was for 10 months from May 2017 to February 2018. 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 outpatient department. In this study motor nerve conduction of ulnar nerve was performed on 50 male diabetic patients and 50 healthy non-diabetic individuals.

Inclusion criteria

Male individuals suffering from type 2 diabetes mellitus with age ranged 30 to 65 years were included in this study. These patients were compared with non-diabetic apparently healthy controls, age, sex, and anthropometrically matched.

Exclusion criteria

All the patients with chronic musculoskeletal disorders, retinopathy, nephropathy or chronic disease, alcoholics and smokers were excluded from the study.

Routine clinical investigations

All study subjects were examined and physical examination included determination of anthropometric indices (age, height, weight, body mass index and waist circumference) and duration of disease. Anthropometric measurements (height and weight) were taken by using scales on bare foot. Waist circumference was measured such as midway between lateral lower ribs and iliac crest after gentle expiration while patient was standing (in centimeters). Body mass index (BMI) was calculated by using Quetelet's Index.[8]

Biochemical parameters

Both fasting and post prandial blood glucose (2h-plasma glucose) levels were estimated by glucose oxidase (GOD/POD) method.[9]

Electrophysiological parameters of ulnar nerve

Motor nerve conduction study of ulnar nerves was performed on both arm in an environment with room temperature ranging from 23°C to 25°C 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 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 digitiminimi 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 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).[10]

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.

Results

In the present study 50 male diabetic subjects with mean duration of disease 7.40±2.04 years were compared with 50 non-diabetic (control) subjects of same age group and sex. The routine clinical investigations of subjects are summarized in Table 1. Fasting and post prandial blood glucose levels between both the groups (Table 2) showed significantly higher (p<0.001) in diabetics. On comparing the parameters of motor nerve conduction of nerves between both the groups (Table 3) it was observed that the distal latency of ulnar nerve on both sides was significantly higher (p<0.001) in diabetics. The amplitude of compound muscle action potential was significantly less in diabetics. Further conduction velocity of ulnar nerve was also found significantly (p<0.001) decreased in diabetics.

Discussion

In the present study the ulnar nerve of diabetic patients has shown adverse effects in electrophysiological functions as compared with non diabetics. This finding is consistent with results of Kakrani et al[11] and Gregersen G[12], who reported that motor defects are common in diabetics with neuropathy. 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.[13] Our results showed increased prandial blood glucose level in diabetics as compared to non diabetic patients. 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.[14] 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 diabetic polyneuropathy.[15] 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.[14] 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.[16] Hoffman W et al found significantly slower conduction velocity in diabetics in both upper and lower limb nerves.[17] 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 G et al.[18] Ulnar nerves in diabetics are less studied and require further evaluation.

Table 1: Routine clinical investigations across the study groups (n=100)

Clinical investigations	Non-diabetic (n=50)	Diabetic (n=50)	p-value	Significance
Age (years)	51.18±8.12	52.32±7.89	0.4782	Not significant
Height (m)	1.70±0.03	1.69±0.04	0.1605	Not significant
Weight (kg)	65.32±15.77	72.16±10.11	0.0113*	Significant
Waist (cm)	31.12±3.24	33.13±3.22	0.0024*	Significant
BMI (Kg/m^2)	22.49±2.53	25.17±2.78	0.0001*	Highly significant
Duration of disease (years)	-	7.40±2.04	-	-

BMI: Body mass index. All the values quoted as the Mean ± Standard deviation. Paired t-test was used to compare the results across the study groups. The p value of <0.05 was considered statistically significant different and represented by asterisk '*'.

Table 2: Fasting and post prandial blood glucose levels across the study groups (n=100)

Biochemical parameters	Non-diabetic (n=50)	Diabetic (n=50)	p-value	Significance
Fasting blood glucose (mg/dL)	95.56±9.53	139.17±36.23	0.0001*	Highly significant
Prandial blood glucose (mg/dL)	129.43±13.33	231.26±56.22	0.0001*	Highly significant

All the values quoted as the Mean ± Standard deviation. Paired t-test was used to compare the results across the study groups. The p value of <0.05 was considered statistically significant different and represented by asterisk '*'.

Table 3: Electrophysiological parameters of ulnar nerve across the study groups (n=100)

Nerves	Electrophysiological Parametres	Non-diabetic (n=50)	Diabetic (n=50)	p-value	Significance
Right Ulnar	Motor Distal Latency (mSec)	3.36±0.57	3.98±0.84	0.0001*	Highly significant
	Amplitude (mV)	9.63±2.17	5.64±3.16	0.0001*	Highly significant
	Conduction velocity (m/s)	54.84±2.49	46.26±3.84	0.0001*	Highly significant
Left Ulnar	Motor Distal Latency (mSec)	3.33±0.52	4.18±0.76	0.0001*	Highly significant
	Amplitude (mV)	9.94±2.12	6.14±2.42	0.0001*	Highly significant
	Conduction velocity (m/s)	51.24±3.08	42.84±3.12	0.0001*	Highly significant

mSec: Milli second; mV: Milli volt; m/s: Meter/second. All the values quoted as the Mean ± Standard deviation. Paired t-test was used to compare the results between two groups. The p value of <0.05 was considered statistically significant different and represented by asterisk '*'.

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