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Original Article

Nerve Conduction Abnormalities in Patients with Diabetes Mellitus

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Abstract

Introduction: *Diabetic Neuropathy is the most common complication of long-term diabetes mellitus (DM). Neuropathy can be diagnosed by nerve conduction study.*

Methods: *This case control study was conducted over a period of one year with the aim to assess peripheral nerve conduction abnormalities in diabetic patients in the department of neuro-medicine, BIRDEM.*

Results: *A total number of 33 diabetic patients with peripheral neuropathy and 22 diabetic control subjects without peripheral neuropathy were enrolled on basis HbA_1C in this study. DM was diagnosed on basis of World Health Organization criteria (1997) and nerve conduction velocity was measured by electro-physiology. It was observed that the diabetes neuropathy (DN) ones showed higher Ulnar nerve distal latency (UD Latency) (msec, M±SD; 3.18±0.94 Vs 5.28±2.32, p<0.001), lower compound muscular action potential (PCAMP) (μV, 4.28±1.71 Vs 2.20±1.40, p<0.001), lower peripheral nerve conduction velocity (PNCV) (m/sec, 47.54±3.40 Vs 40.96±6.37, p<0.001), higher Sural nerve distal latency (SUD Latency) (msec, 2.11±0.54 Vs 2.40±0.24, p<0.005) and lower sensory ulnar nerve conduction velocity (UNCV) (m/sec, 46.91±4.09 Vs 42.96±5.94, p<0.005).*

Conclusion: *It was concluded that both ulnar (Motor as well as sensory functions) and peroneal nerves (only Motor function) are affected in middle aged type 2 DM patients, was as sural nerve is least affected.*

Key words: *Nerve Conduction Abnormalities, Diabetes Mellitus.*

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Introduction

Diabetes mellitus is a chronic disorder, if not properly treated leads to long-term macrovascular and microvascular complications affecting tissues such as heart, kidney, retina and peripheral nerves. Although defective insulin secretion or insulin resistance are the basic disorders of diabetes but other factors such as genetic susceptibility of certain ethnic groups, environmental and behavioral factors such as sedentary life style, nutrition and obesity are also important.[1]

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But actual reason underlying this disorder is not entirely clear. During the last ten year it has been established that diabetes mellitus is not a single disease but a heterogeneous group of disorders characterized by hyperglycemia with varying degrees of insulin insufficiency and insulin resistance.[2] Diabetic neuropathy is a descriptive term meaning a demonstrative disorder, either clinically evident or subclinical, that occurs in the setting of diabetes mellitus without other causes of peripheral neuropathy. It has been suggested that microvascular disease plays a more important role in development in type 2 than in type 1 diabetes where etiology is metabolic rather than vascular.[3] In the pathogenesis of human diabetic neuropathy no single cause can fully explain its development and progression.[4] A wide range of disturbance may affect the peripheral nervous system.[5] Prevalence rate for diabetic neuropathy are sparse and it is virtually impossible to obtain reliable estimates of the prevalence of neuropathy among the diabetic population. The prevalence of diabetic neuropathy appears to be parallel with duration severity of hyperglycemia in both type-1 and type-2 diabetes.[6,7,8] Studies have confirmed the predominance of men and an association with height and increasing age (irrespective of duration of diabetes). Smoking and microalbuminuria are also seemed to be risk factors for diabetic neuropathy.[9,10]

Materials and methods

This observational case control study was carried out in the department of Neuromedicine, BIRDEM over a period of one year enrolling a total number of 33 diabetic patients with peripheral neuropathy and 22 diabetic control without peripheral neuropathy. Cases and Controls were selected on basis of HbA_{1c} level neuropathic complains & clinical neurological examination. Pregnant subjects, diabetic with acute complication and subjects with chronic illness were excluded. After taking proper informed consent, fasting blood glucose were measured and nerve conduction velocity was measured by standard EMG machine in room with a temperature of 37^{o}C. Data were analyses by SPSS (v. 10) and expressed as Mean (±SD). Comparison between groups was done by independent t-test.

Results

In Diabetic subjects without neuropathy group, Ulnar nerve distal latency (mean±SD, msec) was 3.18±0.94. Mean±SD of compound muscle action potential was 7.40±1.22 µV;

p=0.151 and Ulnar Motor nerve conduction velocity (mean±SD, m/sec) was 48.67±4.69.

In Diabetic subjects with neuropathy group, Ulnar nerve distal latency (mean±SD, msec) was 5.28±2.32. Mean±SD of compound muscle action potential was 6.61±2.72 µV and Ulnar Motor nerve conduction velocity (mean±SD, m/sec) was 49.63±6.56; p=0.529.

In Diabetic subjects without neuropathy group, peroneal nerve distal latency (mean±SD, msec) was 10.37±3.02; p=0.282. Mean±SD of peroneal compound muscle action potential was 4.28±1.71µV; p=<0.001 and peroneal nerve conduction velocity (mean±SD, m/sec) was 47.54±3.40; p=<0.001.

In Diabetic subjects with neuropathy group, peroneal nerve distal latency (mean±SD, msec) was 11.57±4.39; p=<0.282. Mean±SD of peroneal compound muscle action potential was 2.20±1.40µV; p=<0.001 and peroneal nerve conduction velocity (mean±SD, m/sec) was 40.96±6.37; p=<0.001.

In Diabetic subjects without neuropathy group, Sural nerve distal latency (mean±SD, msec) was 3.65±1.10; p=0.184. (Mean±SD) of sural sensory action potential was 16.39±10.53 µV; p=0.085 and Sural nerve conduction velocity (mean±SD, m/sec) was 45.08±7.55; p=0.087.

In Diabetic subjects with neuropathy group Sural nerve distal latency (mean±SD, msec) was 3.99±0.39; p=<0.184. (Mean±SD) of sural sensory action potential was 12.08±4.42µV; p=<0.085 and Sural nerve conduction velocity (mean±SD, m/sec) was 42.08±2.41; p=0.087.

Table I: Latencies of nerves in different groups

Variable	Non-DN(n=22)	DN(n=33)	t/p value
UD latency (msec)	3.18±0.94	5.28±2.32	-4.66/<0.001
UCAMO (µV)	7.40±1.22	6.61±2.72	1.46/0.151
MU CAMP (m/sec)	48.67±4.69	49.63±6.56	-0.63/0.529
PD Latency (msec)	10.37±3.02	11.57±4.39	-1.09/0.282
P CAMP (µV)	4.28±1.71	2.20±1.40	4.25/<0.001
P NCV (m/sec)	47.54±3.40	40.96±6.37	4.55/<0.001
SUD Latency (msec)	2.11±0.54	2.40±0.24	-2.357/0.026
USNAP (µV)	19.66±7.81	17.65±5.63	1.042/0.304
UNCV (m/sec)	46.91±4.09	42.92±5.94	2.948/0.005
SD Latency (msec)	3.65±1.10	3.99±0.39	-1.36/0.184
S SNAP (µV)	16.39±10.53	12.08±4.42	1.78/0.085
S NCV(m/sec)	45.08±7.55	42.08±2.41	1.78/0.087

Note: Non DN= Diabetic subjects without neuropathy, DN= Diabetic subjects without neuropathy, UD Latency=Ulnar nerve distal latency, PCAMP=Lower compound muscular action potential, PNCV=Lower peripheral nerve conduction velocity, SUD Latency=Higher Sural nerve distal latency, UNCV=Lower sensory ulnar nerve conduction velocity.

Discussion

Diabetic neuropathy (DN) is one the least understood complications of diabetes mellitus leading to still inadequate definition, classification and intervention guidelines. It is now regarded as a syndrome comprising of several mono and poly neuropathies involving somatic as well as autonomic nerves.[1,2] It is very often difficult to ascertain to what extent the confounding factors are responsible for the subclinical or clinical forms of the disorder in a particular population. In current study subjects were from a hospital based population and the diagnosis was confirmed by measurement of nerve conduction velocity (NCV) s which is gold standard upto the moment. Analysis of the NCV parameters in the non Diabetic and Diabetic group led to an idea regarding the site and nature of nerve involment in the present type 2 DM subject. It was apparent that both ulnar and Peroneal nerves were highly affected, but the Sural nerves were still protected to a great extent. There was also difference in the details of abnormalities between the ulnar and Peroneal nerves. Decrease in the Compound Muscle Action Potential and Nerve conduction velocity was the main defects in the Peroneal nerve (As PD Latency remaining not different from Non-DM group). The site and pattern of nerve involvement in these patients are, in general, similar to those found in the literature[5,7,8] and in a young diabetic group in the same population.[4] It can thus be inferred that in this middle aged Bangladeshi type 2 diabetic populations a number of electrophysiology abnormalities are present in ulnar (both Motor and Sensory) and Peroneal (only motor) nerves, but Sural nerve is somehow spared.

Conclusion

It was concluded that both Ulnar (motor as well as sensory functions) and Peroneal nerves (only motor function) were affected in middle aged type 2 DM patients where as Sural nerve was least affected.

Contribution of the authors

First author was the principle researcher, second and third authors helped in data collection. Third author also did the statistical analysis of the research.

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