

Frequency and Magnitude of Distal Peripheral Neuropathy among Newly Diagnosed Type 2 Diabetics and its' Correlation with Glycaemic Control and Chronicity of Disease

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ABSTRACT

Aim: To determine the frequency and magnitude of peripheral neuropathy among type 2 diabetics

Method: 42 subjects having type 2 diabetes, recent onset, were included in experimental group by random selection. 25 age and sex matched subjects were taken as control. The nerve conduction velocities of 2 sensory and 2 motor nerves of upper and lower limbs were measured on Electromyograph. Glycemic control was assessed by finding fasting plasma glucose and glycosylated Hb through kit.

Result: %age of peripheral neuropathy was higher in diabetic males 33.33% than in females 16.6%. Fasting plasma glucose level showed significant inverse correlation ($P < 0.05$) with sensory conduction velocities ($r = -0.365$), sural ($r = -0.366$) and motor conduction velocities ($r = -0.366$) and motor conduction velocities ($r = -0.540$). A significant inverse relation ($P < 0.05$) was found between glycosylated Hb and both sensory and motor nerve conduction velocities.

Conclusion: A regular examination of nerve conduction velocities should be made. Also glycosylated Hb levels should be checked on a regular basis and kept under control so that peripheral neuropathy and disability may be prevented in these patients through proper and timely diagnosis and treatment.

Keywords: Distal peripheral neuropathy, diabetes, glycaemic control

INTRODUCTION

The most common aetiology of peripheral polyneuropathy in the Western world is diabetes mellitus¹. 90-95% of diabetics have Type 2 diabetes². Such individuals may have hyperglycaemia for many years without clinical symptoms³ and are more susceptible to develop progressive damage to peripheral nerves⁴. Distal symmetrical peripheral polyneuropathy is the most frequent form⁵. Once it is established is largely irreversible⁶. Severe form of diabetic polyneuropathy results in significant complications including disability, morbidity, severe pain, loss of ambulation and increased risk of non healing ulcers of foot and amputation⁷.

For early diagnosis and measurement of peripheral neuropathy Nerve Conduction Studies (NCS) are the most non-invasive and least subjective single criterion⁸. NCS include measurement of sensory and motor nerve conduction velocities of upper and lower limbs⁹. The criterion for the diagnosis of abnormally slow nerve conduction is reduction in nerve conduction velocity more than 3 SD below the mean for normal. An individual having reduced conduction velocity to such a degree in two or more nerves is labeled as having clinical

neuropathy or positive neuropathic finding¹⁰.

In diabetes due to increased glucose entry and elevated cytosolic glucose in the peripheral nerves certain biochemical changes are induced¹¹ that directly affect Schwann cells (or myelin) and nodes of Ranvier¹². A definite relationship may be discovered between glycaemic level and the decrease in nerve conduction velocities among type 2 diabetic patients¹⁵. As the exact threshold of glycaemic control below which the reduction in microvascular complications is not observed and is not yet found¹³.

SUBJECTS AND METHOD

Forty two patients of uncomplicated type 2 diabetes mellitus of recent onset (<5 years) having age range 40-60 years were recruited in the experimental group by simple random selection (Weerasurya et al 1998) They were having minimal or no symptoms and signs of neuropathy and were not treated on insulin within two years after diabetes onset¹⁶ (Young et al 1993). Twenty five age and sex matched non-diabetic healthy individuals both males and females having no family history of diabetes mellitus were included as control group. The nerve conduction velocities of two sensory and two motor nerves of upper and lower limbs were measured in control and experimental groups. For determination of conduction velocities in right sided upper and lower limbs¹⁷ a written consent was taken and nerve conduction studies were

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conducted at Electromyograph in at Sir Ganga Ram Hospital, Lahore. The skin temperature was maintained within 36-38°C¹⁸. Fasting plasma glucose and glycated haemoglobin were also found to assess the level of glycaemic control in these diabetics. Then correlation of these parameters is found with the decrease in conduction velocity to measure the effect of glycaemia on neuropathy quantitatively.

RESULTS



Numbers of diabetics with reduced nerve conduction velocities are given in parentheses. Total 42 type 2 diabetic patients (12 male, 30 females) had

undergone measurement of conduction velocity in peripheral nerves.

Fig. 1: Comparison of motor and sensory nerve conduction velocities in control and diabetics.

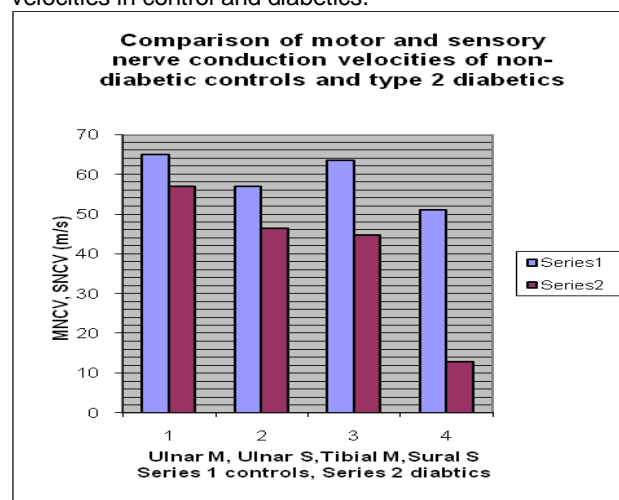


Table 1: Comparison of motor and sensory nerve conduction velocities between non-diabetic controls and type 2 diabetics.

Nerve conduction velocity m/sec	Normal controls (n=25)	Type 2 diabetics (n=42)	P value	Sig.
Ulnar (motor)	65.05±6.56	57.15±7.84	P<0.01	S
Ulnar (sensory)	57.14±5.90	46.60±8.06	P<0.001	HS**
Tibial (motor)	63.57±11.09	44.87±10.35	P<0.001	HS**
Sural (sensory)	51.1±12.03	12.96±19.49	P<0.001	HS**

Table 2: Frequency of peripheral neuropathy in newly diagnosed patients of type 2 diabetes mellitus (Percentage of diabetics with reduced NCV* in two or more nerves)

Type 2 diabetic males (n= 12)	Type 2 diabetic females (n= 30)	Total type 2 diabetics (n= 42)
4(33.33%)	5(16.66%)	9(21.42%)

Table3: %age involvement of motor and sensory nerves of upper and lower limbs in newly diagnosed patients of type 2 DM

Nerves	Type 2 diabetic males (n=12)	Type 2 diabetic females (n=30)	Total type 2 diabetic pts (n=42)
Upper limb			
Ulnar (motor)	0	0	0
Ulnar(sensory)	3(25%)	6(20%)	9(21.42%)
Lower limb			
Tibial (motor)	1(8.3%)	3(10%)	4(9.52%)
Sural (sensory)	8(66.66%)	20(66.66%)	28(66.66%)

Table 4: Correlation between fasting plasma glucose (mg/dl) and sensory motor nerve conduction velocities (m/sec) in type 2 diabetic group

Correlation between Coefficient(r)	Correlation equation	Regression	P value	Sig
FPG and SNCV (ulnar)	-0.365	Y=58.31+-0.07X	P<0.05	S
FPG and MNCV (tibial)	-0.540	Y=67.09 +-0.23X	P<0.05	S
FPG and SNCV (sural)	-0.366	Y=44.51+-0.19X	P<0.05	S

Table5: Correlation between glycated haemoglobin(%) & sensory, motor nerve conduction velocities(m/sec) in type 2 DM

Correlation between Coefficient(r)	Correlation equation	Regression	P value	Sig
HbA _{1c} and SNCV (ulnar)	-0.493	Y=64.36+-1.75X	P<0.05	S
HbA _{1c} and MNCV (tibial)	-0.454	Y=65.86+-2.07X	P<0.05	S

Table 6: Correlation between duration of diabetes (months) & sensory, motor nerve conduction velocities(m/sec) in type 2 DM

Correlation between Coefficient(r)	Correlation equation	Regression	P value	Sig
Duration of diabetes and MNCV (tibial)	-0.472	Y=49.28 +0.25X	P<0.01	S

DISCUSSION

The results of peripheral nerve conduction velocities revealed that mean motor nerve conduction velocities (MNCV) of ulnar and tibial nerves and mean sensory nerve conduction velocities (SNCV) of the ulnar and sural nerves were significantly (P<0.001) reduced among recently diagnosed type 2 diabetics. This is in accordance with the dominant histopathological finding in diabetic neuropathy that is segmental demyelination¹⁹. This result in loss of large, fast conducting fibers and slowing of nerve conduction velocities²¹. Hence nerve conduction measurement is very powerful indicator of neuropathy²².

It was found that nerves of the lower limb were more frequently and intensely affected than the nerves in the arms among type 2 diabetics. This may be on account of greater nerve length in the lower limb. So, peripheral neuropathy seems to be a length dependent phenomenon affecting first the most distal parts of peripheral nerves²⁰.

This study indicated further that peripheral nerve disturbances were more common in the sensory nerves than in the motor nerves. So the sensory nerves are more susceptible to damage than motor nerves as these lack thick myelin sheath²³. The most common cause of distal symmetrical peripheral polyneuropathy is diabetes mellitus¹. According to the criteria for diagnosis and staging of diabetic neuropathy established by Dyck 1988, frequency of clinical peripheral neuropathy among recently diagnosed patients of type 2 diabetes mellitus was 21.42% in this study. Using the same electrophysiological parameters Comi et al 1999 has reported the prevalence of neuropathy 32.3%.

According to the results of the present study, out of 42 type 2 diabetic patients percentage of peripheral neuropathy was higher in diabetic males (33.33%) than in females (16.6%). The possible reason may be the greater length of peripheral nerves in males as peripheral neuropathy seems to be a length related pathology starting distally.

Fasting plasma glucose level showed inverse significant correlation (P<0.05) with sensory conduction velocities of ulnar (r=-0.365), sural (r=-0.366) nerves and motor conduction velocity of tibial (-0.540) nerve in recently diagnosed diabetics. Glycated haemoglobin as well held a significant inverse relation (P<0.05) with both sensory (ulnar nerve=-0.493) and motor (tibial nerve=-0.454) nerve conduction velocities. It was found further by correlation analysis that for each one percent rise in

glycated haemoglobin there was 1.75 meter/second fall in sensory conduction velocity of ulnar nerve and 2.07 meter/second decrease in motor conduction velocity of tibial nerve.

Measurement of these glycaemic parameters in a diabetic patient provides a detailed profile of previous and recent glycaemic control. Hence a prior record of the glycaemic profile during a specified period can help the physician to estimate the degree of nerve damage and to determine the extent of effective glycaemic control essential for such patient to prevent further progression of neuropathy.

This study further suggested a significant inverse correlation (P<0.05, r=0.472) between duration of disease and motor nerve conduction velocity in recently diagnosed type 2 diabetics. This indirect correlation between duration of disease and nerve conduction velocity is also being established by Perkins et al 2001 and Rivner et al 2001. Thus the severity of diabetic neuropathy and slowing of nerve conduction may be related to the total span of the disease²⁶. The metabolic abnormalities induced by hyperglycaemia appear to occur in temporal sequence. These lead to impaired nerve function and decreased blood flow, which initially may be readily reversible. As structural changes emerge and progress, the functional abnormalities become increasingly less responsive to metabolic interventions²⁷. So early diagnosis and control of hyperglycaemia in diabetes may delay the onset and progress of neuropathy and its complications.

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