CODEN [USA]: IAJPBB **ISSN: 2349-7750**

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES
http://doi.org/10.5281/zenodo.3334183

Available online at: http://www.iajps.com ***Research Article***

STUDY TO KNOW THE CORRELATION OF GLYCEMIC CONTROL AND CHRONICITY OF DISEASE WITH MAGNITUDE AND FREQUENCY OF DISTAL PERIPHERAL NEUROPATHY IN PATIENTS NEWLY DIAGNOSED WITH TYPE II DIABETES MELLITUS

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Article Received: May 2019 **Accepted:** June 2019 **Published:** July 2019

Abstract:
Objective: *To determine the frequency and severity of peripheral neuropathy in patients with type 2 diabetes mellitus.*
Study Design: *An Experimental Study.*
Place and Duration: *In the Physiology Department of Services Institute of Medical Sciences in collaboration with Medicine Unit II of Services Hospital Lahore for Six months duration from September 2018 to February 2019.*
Methods: *Forty-two subjects with type 2 diabetes at the beginning were randomly selected into the experimental group. Twenty-five patients with age and sex matched were included as controls. Electromyography showed nerve conduction velocities of 2 sensory nerves and 2 motor nerves of upper and lower extremities. Glycemic control was assessed by the presence of fasting plasma glucose and glycosine Hb with the help of a kit.*
Conclusion: *Peripheral neuropathy was 33.33% higher in diabetic men and 16.6% higher in women. Fasting plasma glucose levels showed a significant inverse correlation with sensory conduction of speeds (r = -0.365), sural (r = -0.366) and motor conduction speeds (r = -0.365) (P <0.05). 0.366) and motor conduction velocities (r = - 0.540). We found a significant inverse correlation (P <0.05) between glycated Hb and sensory and motor nerve conduction velocities.*
Conclusion: *Nerve conduction rates should be performed regularly. In addition, glycosylated Hb levels should be monitored and controlled regularly so that peripheral neuropathy and disability in these patients can be prevented by appropriate and timely diagnosis and treatment.*
Key words: *Distal peripheral neuropathy, diabetes, glycemic control.*

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Please cite this article in press Areeba Khan et al., ***Study To Know The Correlation Of Glycemic Control And Chronicity Of Disease With Magnitude And Frequency Of Distal Peripheral Neuropathy In Patients Newly Diagnosed With Type Ii Diabetes Mellitus.,*** *Indo Am. J. P. Sci, 2019; 06(07).*

INTRODUCTION:

The most common etiology of peripheral polyneuropathy in the Western world is diabetes mellitus [1]. 90-95% of diabetic patients have type 2 diabetes. Such people may have hyperglycemia for years without clinical symptoms and are more likely to develop progressive damage to the peripheral nerves [2]. Distal symmetric peripheral polyneuropathy is the most common form. Once occurred, it is largely irreversible[3]. Severe diabetic polyneuropathy causes major complications, including disability, morbidity, severe pain, loss of circulation, and the risk of foot ulcers that do not heal or enter amputations [3-4]. For early detection and measurement of peripheral neuropathy, nerve conduction studies (NEC) are only non-invasive and most subjective criteria [5-6]. NCS involves sensory and motor nerve conduction velocities of the upper and lower extremities [7]. The criterion for the diagnosis of abnormally slow nerve conduction is that the nerve conduction velocity falls below the normal average of 3 SD [8]. An individual whose conduction rate is reduced to this degree by two or more nerves is labeled as clinical neuropathy or positive neuropathic finding. Due to increased glucose entry and high cytosolic glucose in the peripheral nerves, some biochemical changes occur in diabetes that directly affect Schwann cells (or myelin) and Ranvier nodules. A definitive relationship between glycemic level and decrease in nerve conduction velocity in patients with type 2 diabetes can be discovered.

MATERIALS AND METHODS:

This experimental Study was held in the Physiology Department of Services Institute of Medical Sciences in collaboration with Medicine Unit II of Services Hospital Lahore for Six months duration from September 2018 to February 2019.

Forty-two patients with uncomplicated type 2 diabetes mellitus (<5 years) aged 40-60 years with symptoms or signs were included in the experimental group by simple random selection (Weerasuryia et al. 1998). Neuropathy and diabetes were not treated with insulin within two years of onset (Young et al 1993). Twenty-five healthy non-diabetic subjects were included in the control group, male and female, matched in terms of age and sex and without a family history of diabetes mellitus. Nerve conduction velocities of two sensory nerves and two motors of upper and lower extremities were measured in control and experimental groups. A written consent was obtained for the determination of conduction velocities on the right and upper extremities on the right side, and nerve conduction studies were performed. The temperature of the skin remained between 36-38 ° C. In addition, fasting plasma glucose and glycosylated hemoglobin were found to evaluate the level of glycemic control in these diabetics. The correlation of these parameters is then found by the decrease in driving speed to quantitatively measure the effect of glycemia on neuropathy.

RESULTS:

Diabetic numbers in which nerve conduction velocities have decreased are given in parenthesis. A total of 42 type 2 diabetic patients (12 males and 30 females) were measured for conduction velocity in the peripheral nerves.

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

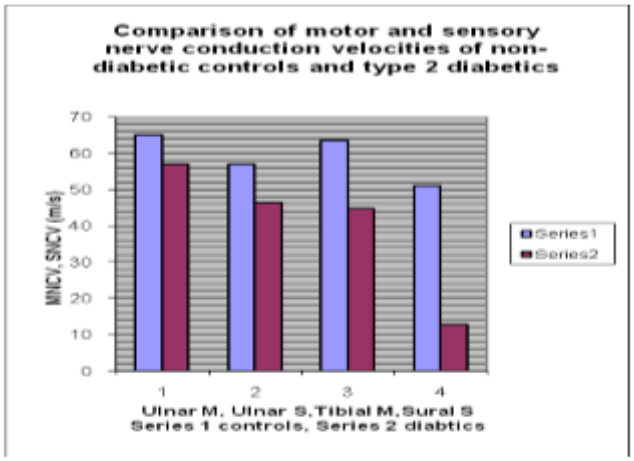
Correlation between Coefficient(r)	Correlation equation	Regression	P value	Sig
FPG and SNCV (ulnar)	-0.365	Y=58.31+-0.07X	P<0.05	S
FPG and MNCV (tibial)	-0.540	Y=67.09 +-0.23X	P<0.05	S
FPG and SNCV (sural)	-0.366	Y=44.51+-0.19X	P<0.05	S

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

Figure 1: Comparison of conduction rates of motor and sensory nerve in control and diabetic patients.

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



DISCUSSION:

The results of the peripheral nerve conduction velocities revealed that the motor nerve conduction velocity (MNCV) of the ulnar and tibial nerves and the average sensory nerve conduction velocities (SNCV) of the ulnar and ulnar nerves were significantly reduced (P <0.05). 0.001) of newly diagnosed diabetic type 2 [9]. This is consistent with the dominant histopathological finding in diabetic neuropathy with segmental demyelination. This leads to the loss of large fibers of fast conductivity and reduced nerve conduction rates. Therefore, measurement of nerve conduction is a very powerful indicator of neuropathy [10]. It was found that the nerves of the lower extremity were affected more frequently and more intensely than the nerves in the arms between type 2 diabetes patients, which may be due to the longer nerve in the lower extremity [11]. Thus, peripheral neuropathy appears to be a length-dependent phenomenon that first affects the more distal portions of the peripheral nerves. This study also showed that peripheral nerve disorders are more common in sensory nerves than motor nerves [12]. Therefore, sensory nerves are more susceptible to damage than motor nerves because they do not have a thick myelin sheath. The most common cause of distal symmetric peripheral polyneuropathy is diabetes mellitus. According to the diagnostic and staging criteria for diabetic neuropathy created by Dyck 1988, the frequency of clinical peripheral neuropathy in newly diagnosed patients with type 2 diabetes mellitus was 21.42%. Using the same electrophysiological parameters, Comi et al. 1999 reported a 32.3% prevalence of neuropathy [13]. According to the results of this study, the percentage of peripheral neuropathy in 42 type 2 diabetes patients was higher in diabetic men (33.33%) than in women (16.6%). The possible cause may be longer peripheral nerves in men, because peripheral neuropathy appears to be a pathology due to distal onset length[14]. Fasting plasma glucose levels were significantly inversely correlated with sensory conduction velocities of ulnar nerves (r = -0.365), sural (r = - 0.366) and nerve conduction velocities (P <0.05). Glycosylated hemoglobin also maintained a significant inverse relationship (P <0.05) with nerve conduction rates of both sensory (ulnar nerve = -0.493) and motor (tibial nerve = -0.454). Furthermore, by correlation analysis, it was discovered that the sensory conduction rate of the ulnar nerve was reduced by 1.75 meters / sec and decreased by 2.07 meters / sec for each percent increase in glycosylated hemoglobin. Measurement of these glycemic parameters in a diabetic patient provides a detailed profile of previous and final glycemic control. Therefore, a previous record of a glycemic profile over a period of time can help the physician estimate the degree of nerve damage and determine the degree of effective glycemic control necessary to prevent the progression of neuropathy for such a patient. This study also suggested a significant inverse correlation (P <0.05, r = 0.472) between disease duration and motor nerve conduction rate in patients with type 2 diabetes mellitus recently diagnosed[15]. This indirect relationship between the duration of the disease and nerve conduction velocity was also reported by Perkins et al. 2001 and Rivner et al. Metabolic abnormalities caused by hyperglycemia occur in a temporal sequence. They cause impaired nerve function and reduced blood flow, which can be easily reversible at the beginning. As structural changes occur and progress, functional anomalies are less and less susceptible to metabolic interventions. Therefore, early diagnosis and control of hyperglycemia in diabetes may delay the onset and progression of neuropathy and its complications.

CONCLUSION:

Nerve conduction rates should be performed regularly. In addition, glycosylated Hb levels should be monitored and controlled regularly so that peripheral neuropathy and disability in these patients can be prevented by appropriate and timely diagnosis and treatment.

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