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EVALUATION OF PREVENTION AND CONTROL OF GLYCACEMIA AS A PROGNOSTICATOR OF DIABETIC NEUROPATHY

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Abstract:

Objective: *Nerve conduction studies (NCS) for measuring peripheral neuropathy are only less invasive and less subjective criteria. In diabetes, due to increased glucose penetration, it directly affects Schwann cells (or myelin) and Ranvier ganglia. In this observational study relationship present between glycemic level and the nerve conduction velocities, among type 2 diabetic patients is found, measuring the velocity of sensory nerve conduction of the ulnar nerves, femoral and motor ulnar and tibial nerves.Distal symmetrical peripheral polyneuropathy is the most common neuropathy in patients with diabetes.*

Duration: *From January 2019 to February 2020.*

Method: *Nerve conduction studies were conducted at the EMG department at the neurology department of Mayo Hospital Lahore. It has been estimated that fasting plasma glucose and glycated hemoglobin levels were estimated to find glycemic control.42 subjects having type 2 diabetes patients with onset of disease ≤ 5 years and an age range of 40-70 years were included by simple random selection.*

Results: *The motor nerve transmission rate of the ulnar nerve is significantly reduced in patients with diabetes ($p < 0.05$). Fasting plasma glucose and glycated hemoglobin are significantly increased in patients with type 2 diabetes ($p < 0.001$), with a significant inverse correlation of these glycemic parameters ($p < 0.05$) with sensory, raw and sensory transmission rates. Engine.The conduction velocity of the motor tibial nerve and the conduction velocity of the sensory nerve of the ulnar and femoral nerve were significantly reduced ($p < 0.001$).*

Conclusion: *This suggests the metabolic basis of the pathogenesis of diabetic neuropathy. Therefore, glycemic parameters have been shown to be important in predicting neuropathy and can therefore be used to assess other microvascular complications of diabetes.*

Keywords: *diabetic neuropathy, motor nerve conduction velocity, glycemic control, fasting plasma glucose, glycated hemoglobin.*

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INTRODUCTION

Diabetic polyneuropathy is largely irreversible once established. The severe form of diabetic polyneuropathy causes significant complications such as disability, morbidity, severe pain, loss of sensation and an increased risk of untreated ulcers [4]. There is no other gold standard for measuring peripheral neuropathy except nerve conduction studies (NCS), which are the most invasive and least subjective criteria [3]. NCS includes measuring the speed of sensory conduction and motor nerve of the upper and lower extremities [5].Distal symmetrical peripheral polyneuropathy is the most common neuropathy in patients with diabetes [1].

Due to increased glucose uptake in the peripheral nerves and an increase in cytosolic glucose, some biochemical changes are induced in diabetes which directly affect Schwann cells (or myelin) and lymph nodes. Ranvier [6-7]. Therefore, there may be a clear relationship between glycemic levels and reduced nerve conduction velocity in patients with type 2 diabetes [8]. A full glucose control threshold at which no reduction in microvascular complications was observed and has not yet been found[9]. In this study, it was found that the nerve conduction velocities of two sensory nerves and the two upper and lower limb motors found a degree of reduction in conduction velocity compared to diabetic controls.

It was also found that fasting plasma glucose and glycated hemoglobin assess the level of glycemic control in these diabetic patients. It was later found that the correlation of these parameters with the decrease in transmission speed quantitatively understands the effect of glycemia on neuropathy.

METHOD:

Nerve conduction velocities were studied in the peripheral nerves on the right side of the ulnar nerve, because there was no significant difference in conduction velocity along the right or left nerve. Skin temperature was maintained between 36-38 degrees. Fasting plasma glucose and glycated hemoglobin levels were estimated using glycemic control finding kits.

Patient selection: Subjects included type 2 diabetes mellitus with onset of disease ≤ 5 years and no advanced symptoms of microvascular complications. Endocrine disorders, nerve damage, hereditary neuropathy, trap traps and stroke patients are not included. Pregnant women, people with chronic alcoholics and vitamin B complexes, inflammatory kidney and liver disease, and nutritional deficiency with advanced liver disease. Diseases were carefully overlooked and overlooked in participants by physical examination.Written consent has been obtained for each subject.Nerve conduction studies were conducted at the neurology department of Mayo Hospital Lahore for one-year duration from January 2019 to February 2020.

RESULTS:

The experimental group consisted of 42 patients with type 2 diabetes, 30 women and 12 men with an average duration of diabetes of 17.76 ± 19.67 months.

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	Significance
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: 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	Regression equation	P value	Significance
FPG and MNCV (ulnar)	0.139	Y=53.75+0.02X	P>0.05	NS*
FPG and SNCV (ulnar)	-0.291	Y=55.19+-0.05X	P<0.05	S**
FPG and MNCV (tibial)	-0.357	Y=60.03 +-0.10X	P<0.05	S**
FPG and SNCV (sural)	-0.332	Y=34.49+-0.13X	P<0.05	S**

Table 3: Correlation between glycated haemoglobin (%) and sensory, motor nerve conduction velocities (m/sec) in type 2 diabetic group

Correlation between Coefficient(r)	Correlation	Regression equation	P value	Significance
HbA_1c and MNCV (ulnar)	-0.125	Y=53.36+0.41X	P>0.05	NS*
HbA_1c and SNCV (ulnar)	-0.493	Y=64.36+-1.75X	P<0.05	S**
HbA_1c and MNCV (tibial)	-0.419	Y=67.24+-2.35X	P<0.05	S**
HbA_1c and SNCV (sural)	-0.092	Y=19.95+-0.74X	P>0.05	NS*

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Table 4: Comparison of glycaemic parameters between controls and type 2 diabetics

Glycaemic Parameters	Normal controls (n=25)	Diabetics (n=42)	P value	Significance
Fasting plasma glucose (mg/dl) (FPG)	76.88 ± 13.73	162.71±40.96	P<0.001	HS*
Glycated haemoglobin (HbA1c) (%)	5.33 ± 0.48	10.12 ± 2.26	P<0.001	HS*

(Mean ± standard deviation), HS* highly significant

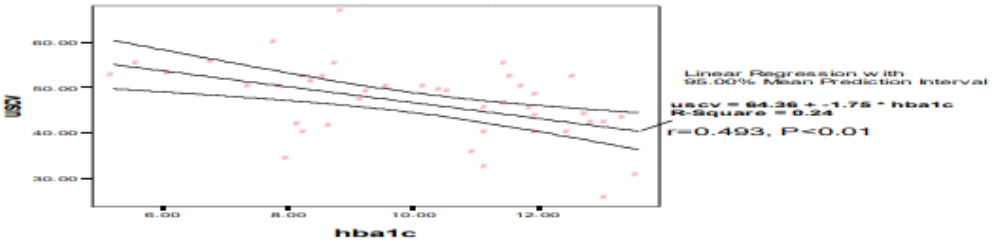
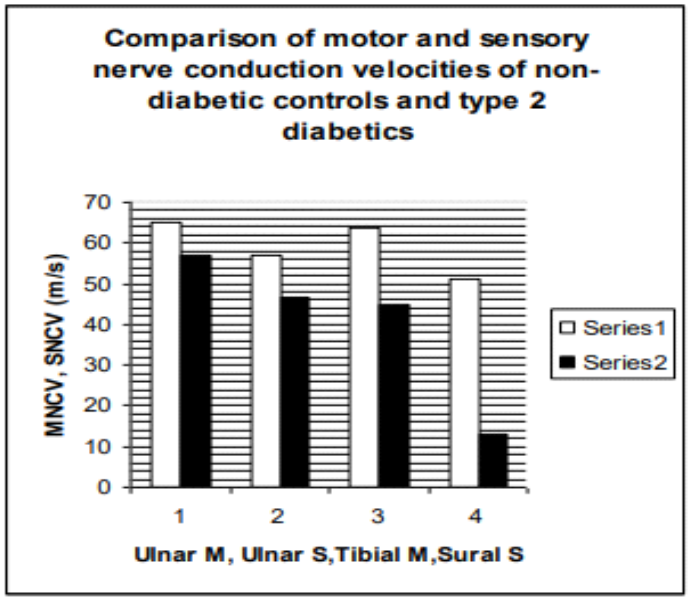


Figure 2: Graph showing correlation between glycated hemoglobin (%) and ulnar sensory nerve conduction velocity . (The line represents the regression li
r= Correlation coefficient)



MNCV Motor nerve conduction velocity
SNCV Sensory nerve conduction velocity
M=Motor, S=Sensory

DISCUSSION:

76.88 + 13.73 mg / dL and 5.33 age corresponded to + 0.48% of healthy control. Fasting plasma glucose indicates current glucose control. Glycan hemoglobin (HbA1c) is a quantitative mean glycemic index 6-10 week's ago [10].Both glycemic indices showed significantly higher values (P <0.001) (mean + SD), including fasting plasma glucose 162.71 + 40.96 mg / dl and glycosylated hemoglobin.

Peripheral nerve conduction velocity results showed that mean motor nerve conduction velocities (MNCV) of the ulnar and tibial nerves and mean sensory nerve conduction velocities (SNCV) association of the ulnar and radial nerves were significant (P <0.001) among novice type 2 diabetics. This is consistent with the dominant histopathological outcome in diabetic neuropathy, which is segmental demyelination [11]. This results in the loss of large, fast-conducting fibers and a slowdown in nerve conduction velocity [12]. It was found that the nerves in the lower limbs were more affected than the nerves in the hands. This may be due to the increased length of the nerves in the lower limb. Therefore, peripheral neuropathy appears to be a length-dependent phenomenon that first affects the most distal parts of the peripheral nerves [13].

This study also showed that peripheral nerve disorders are more common in sensory nerves than motor nerves. Therefore, sensory nerves are more prone to damage than motor nerves, because they lack a thick myelin sheath [14]. A statistically significant inverse correlation with glycemic parameters, including sensory and motor nerve conduction velocities of all nerves tested in type 2 starter type diabetes, and blood sugar and glycated hemoglobin. In peripheral nerves, the rate of nerve conduction significantly decreased with increasing plasma glucose and glycosylated hemoglobin in patients with type 2 diabetes.

Fasting plasma glucose levels show a significantly inverse correlation (P <0.05) with the sensory conduction velocity of the ulnar nerves (r = -0.365), sural (r = -0.366) and the motor nerve conduction velocity in tibial (-0,540) in newly diagnosed diabetics. Glycated hemoglobin also maintains a highly significant inverse relationship (P <0.01) with sensory speeds (ulnar nerve = -0.493) and motor speeds (tibial nerve = -0.445).

Significant correlations between glycemic parameters and peripheral nerve conduction indicators show that metabolic disorders caused by hyperglycemia play an important role in the pathogenesis of diabetic peripheral neuropathy. As indicated in the regression analysis in this study, there is a clear and clear relationship between the degree of hyperglycemia and the degree of nerve conduction, with a decrease of 1.75 for each percent increase in glycine hemoglobin [15]. Velocity of sensory ulnar nerve conduction in meters / second and motor transmission speed of tibial nerve by 2.35 meters / second. Simultaneous measurement of both glycemic parameters in a diabetic patient provides a detailed profile of final and final glycemic control. Therefore, recording a glycemic profile over a period of time can help the physician assess the degree of nerve damage and determine the degree of effective glycemic control required for this patient to prevent neuropathy progression since optimization. Glycemic control is the only medicine available (Diabetes Control and Complications Trial Research Group 1993).

CONCLUSION:

1. The very significant negative correlation between nerve conduction velocity and glycemic control suggests that diabetic neuropathy is the metabolic basis of pathogenesis. Therefore, it has been shown that both fasting plasma glucose and glucose as well as glycated hemoglobin are important in predicting the onset and progression of neuropathy and can be used to assess other microvascular complications of diabetes.
2. In addition, patients should be trained to maintain rigid glycemic control to prevent disability and the onset of progressive neuropathy.
3. The speed of nerve conduction in peripheral sensory and motor nerves has significantly decreased among beginner type 2 diabetics in the upper and lower extremities. Therefore, this test should first be used to diagnose neuropathy.

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