NERVE CONDUCTION IN TYPE 2 DIABETICS AND ITS CORRELATION WITH GLYCOSYLATED HAEMOGLOBIN

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ABSTRACT: INTRODUCTION: Diabetes mellitus is a global problem. It is estimated to increase from 4% in 1995 to 5.4% by the year 2025. Diabetic neuropathy (DN) is most commonly occurring microvascular complication accounting for 28% of all complications in diabetics. There is a progressive neuronal involvement in diabetics which is accelerated by poor glycaemic control. Diabetic neuropathy has a long asymptomatic stage. Nerve conduction study can be employed for screening diabetic patients in subclinical stages. **MATERIAL & METHODS:** Sixty diabetic subjects (male& female) without complications with duration of diabetes 1-10 yrs and 30 normal healthy individuals were included in the study. Nerve conduction study was done using AD-instrument Powerlab/30 series. HbA1c was estimated by Cobas C-111 Autoanalyser. Fasting and Post-prandial blood sugar was measured by GOD-POD method. **RESULTS & DISCUSSION:** Nerve conduction velocity progressively decreased from the controls to diabetics with good glycaemic control, to the diabetics with poor glycaemic control. (HbA1c) **CONCLUSION:** There is progressive slowing of sensory nerves in diabetics which is accelerated by poor glycemic control.

KEYWORDS: Diabetic neuropathy, nerve conduction velocity, Glycosylated haemoglobin.(HbA1c)

INTRODUCTION: Diabetes mellitus is a global problem. The prevalence of diabetes mellitus is growing rapidly worldwide and is reaching epidemic proportions. It is estimated to increase from 4% in 1995 to 5.4% by the year 2025. According to the International Diabetes Federation (IDF) 2013 there are 67.1 million diabetics in India. WHO studies reported total diabetics in India in 2000 was 31.7 million, likely to increase to 79.4 million by 2030.¹

Type 2 Diabetes mellitus is characterized by variable degree of insulin resistance. Both type 1 and type 2 diabetes (T2DM) may occur at any age, but type-2 diabetes is mainly diagnosed after the age of 40 years .² So the present study is done in diabetics aged 40-50 yrs. According to the American Diabetes Association (ADA), Type 2 diabetes accounts for at least 90% of all cases of diabetes.³

Diabetic neuropathy (DN) is one of the most commonly occurring microvascular complications accounting for 28% of all the complications in diabetics.⁴ It is a progressive process that has a long asymptomatic stage. The clinical impact of diabetes is mainly manifest on the peripheral nervous system.

The most common pattern of diabetic neuropathy is a Distal symmetric polyneuropathy (DSP) in which sensory symptoms and deficits predominate sensory symptoms and deficits predominate, weakness is minimal (or) absent until much later.⁵ Distal symmetric polyneuropathy (DSP) is the most common form of nerve injury in diabetes, with an estimated prevalence of 50%.⁶

Diabetic neuropathy is said to be dependent both on degree of control and duration of diabetes, though it is more related to duration of diabetes. Diabetic peripheral neuropathy is more

common in people who have poor control of their blood glucose. Since diabetic neuropathy can lead to serious consequences including foot ulcer leading to gangrene and amputation, it has to be diagnosed at the earliest.

Nerve conduction study (NCS) is the gold standard for measurement of diabetic neuropathy. It is non–invasive, least subjective single criterion.⁷

Glycosylated haemoglobin (HbA1C) is established as a marker of glycemic control for the past 6-8 weeks It is widely accepted and used as most reliable test for assessing chronic glycemia. There is progressive neuronal involvement in diabetics which is accelerated by poor glycemic control.⁸

There is a strong association between polyneuropathy, duration of diabetes, level of HbA1c, thereby indicating that near normal glycemic control should be precautions to delay the beginning or progression of polyneuropathy.⁹

The focus of the present study done in diabetics is to correlate nerve conduction parameters with duration of diabetes, glycosylated haemoglobin and to highlight the role of estimation of both nerve conduction velocity and the HbA1c levels in diabetics to identify neuropathy in the asymptomatic stages as the disease process progresses to the diabetic foot, a highly morbid condition that arises from the infection and the ulceration of the foot, finally leading to amputation.¹⁰

MATERIALS AND METHODS:

SOURCE OF DATA: The present study was undertaken for a period of 8 months on 90 subjects, both males and females aged between 40-50 years after obtaining the permission of the ethical committee of our institution and informed written consent was taken from the subjects.

The study group consisted of 60 diabetic patients, with history of diabetes for 1-10 years attending the medical OPD of Basaveshwar teaching and general hospital, Gulbarga and controls were 30 age and sex matched healthy individuals from the non-teaching staff of M. R. Medical College, Gulbarga.

CRITERIA FOR INCLUSION: Controls included 40-50 years aged 30 normal healthy individuals without diabetes.

Subjects included 40-50 years aged 60 type 2 diabetics under control without complications on oral hypoglycemic drugs with Duration of diabetes 1-10years.

EXCLUSION CRITERIA:

- 1. Duration of diabetes more than 10 years.
- 2. Alcoholics, hypertensives, smokers, pregnant females.
- 3. Diabetic subjects having acute diabetic complications, entrapment neuropathy, nutritional deficiency, endocrinal disorders.
- 4. Muscle weakness, myopathy, neuromuscular diseases inherited neuropathy.
- 5. Neurovascular complications like stroke.
- 6. Any pathology or injury to upper and lower limb.
- 7. Clinical evidence of any other illness like advanced liver disease or renal disease.

NERVE CONDUCTION STUDY (NCS): Nerve conduction study was done using the AD instrument – Power lab/30 series provided by the Department of Physiology, M. R Medical College, Gulbarga. For

motor & sensory nerve study, output range was 20mA,max repeat rate 1Hz,pulse width 0.05ms stimulation with current ranging between 10-20mA was applied with increasing strength until desired response was obtained.

Sensory nerves tested were Median, and Ulnar nerve.

STIMULATION AND RECORDING SITES OF SENSORY NERVES:

Sensory nerve	Method of stimulation	Stimulation site	Recording site
Median	Orthodromic	Index finger	Middle of the wrist
Ulnar	Orthodromic	Little finger	Medial wrist

Sensory nerve conduction study was done:

Parameters Assessed:

- 1. Sensory nerve action potential (SNAP) Amplitude.
- 2. Sensory nerve conduction velocity.

PROCEDURE: Subjects were asked to remove any hearing aids, or other metal objects that may interfere with the procedure and were made to lie down for the test. Room temperature was maintained at 30c.Hand and feet on dominant side were cleaned thoroughly with spirit. The nerve to be tested was located.

- Motor & sensory nerve conduction of nerves of right upper and lower limb was assessed using AD instrument-power lab/30 series provided by the Department of Physiology M.R Medical College Gulbarga.
- The recording electrode and grounding electrode were placed on the skin overlying the muscle supplied by the nerve. The stimulating electrode was placed with a strap along the course of the nerve. These electrodes are connected through bio amp cable to Bio amp console. Stimuli was applied at two different sites along the nerve and the latency of the e.m.g. response at both sites was measured. The difference in response latencies obtained was used as a measure of the time taken for the action potentials to travel along the nerve between the two stimulus points. The distance between the points of stimulation was measured.

Conduction velocity is then calculated m/s by the formula,

Nerve conduction velocity (m/s):=distance (d)/difference in latent periods (t).

This was repeated for each nerve being tested.

- Blood samples will be collected and investigated for HbA1c levels, FBS and PPBS in biochemistry laboratory of Basveshwar teaching and general hospital M.R Medical college, Gulbarga.
- HBA1c is done by Cobas c-111 Autoanalyser. FBS and PPBS was estimated by glucose oxidaseperoxidase method(GOD-POD).For FBS patient is asked to fast for 10-12hrs and blood sample taken and for post prandial blood sample is taken 2hrs after meals.

STATISTICAL ANALYSIS: Statistical analysis was done using SPSS version18 (software statistical package social science). Chi-square test was used to analyse sex-wise distribution of subjects studied, students unpaired t test was used to compare Nerve conduction parameters between the study and

control groups and Pearson's correlation co-efficient were used to find correlation between nerve conduction parameters and glycemic control.

- P Value <0.001 Highly Significant.
- P Value <0.05 Significant.
- P Value >0.05 Not Significant.

RESULTS:

Age	Control group		Study group	
(years)	No	%	No	%
40-45	12	40	33	55
46-50	18	60	27	45
TOTAL	30	100	60	90
MEAN±SD	45.93±3.28 45.34±3.17			
t-Value	t = 0.819			
P-Value	p>0.05, Not significant			
Table 1: Age wise distribution of Subjects Studied				

Table 1 and Figure 1 depicts the age distribution of subjects in both the control group and diabetics. There is no statistical difference of age among control & study group.



Fig. 1: Age wise distribution of subjects studied

Sex	Control group		Study group	
	No	%	No	%
Male	23	76.7	51	85
Female	7	23.3	09	15
Total	30	100	60	10
Table 2: Sex wise distribution of subjects studied				

X2 = 0.584, P >0.05, statistically not significant.



Table-2 and Figure-2depict the sex-wise distribution of subjects in both the control group and diabetics with females being (23.3%) in control group and (15%) in diabetic group

Blood sugar tests	Control group (30) Mean±SD	Study group (60) Mean±SD	t-test	P- value
FBS (mg/dl)	86.53±8.72	109.78±15.77	t =9.01	P<0.001
PPBS (mg/dl)	117± 7.63	188.53±25.16	t =20.32	P<0.001
HbA1c (%)	5.14 ± 0.47	6.49 ± 0.32	t =14.09	P<0.001
Table 3: Comparison of Blood Sugar Levels in Study and Control Group				

Table-3 and Figure-3 depict FBS and PPBS levels in controls and diabetics. FBS, PPBS was significantly higher in diabetics (P<0.001) than controls.





Table-3 and Figure-4 depict the HbA1c % in study and control group. HbA1c levels were significantly higher in study group compared to the control group (P< 0.001.)

Nerve conduction parameters	Control group Mean± S.D	Study group Mean± S.D	t-test	p-value
Median nerve				
AMP(ms)	20.98± 3.68	18.68± 5.16	t =2.46	p<0.05
SNCV(m/s)	57.93 ± 4.05	46.14± 4.96	t =12.82	p<0.001
Ulnar nerve				
AMP(ms)	22.60± 3.88	22.67± 4.76	t =0.075	p>0.05
SNCV(m/s)	57.86± 5.82	45.49± 5.07	t =9.97	p<0.001
Table 4: Comparison of Sensory Nerve conduction parameters in Controls and Diabetics				

AMP -amplitude, SNCV-sensory nerve conduction velocity.



Table-4 & Figure-5 depicts Comparison of sensory nerve parameters in Control Group and diabetics there is significant slowing of sensory nerves median& ulnar (p<0.01) and decrease in amplitude of Median nerve(p<0.05) in diabetics compared to controls.

Sensory nerve conduction parameters				
Madian nama	HBA1c (5-6%)	HBA1c (6-7%)	tualua	n valuo
Meulan hei ve	Mean± S.D (N=12)	Mean± S.D (N=48)	t value	p value
AMP(ms)	16.64± 3.72	17.6 ± 4.87	t =0.75	p>0.05
SNCV(m/s)	48.06 ± 2.86	45.12 ± 4.51	t =2.88	p<0.05
Ulnar nerve				
AMP(ms)	22.64 ± 3.96	22.45 ± 5.24	t =0.43	p>0.05
SNCV(m/s)	42.72± 4.86	45.86 ± 5.12	t =2.17	p<0.05

Table 5: Comparison of Nerve conduction (sensory) parameters with HbA1c Levels in diabetics



with HbA1c Levels in diabetics in Controls & Diabetics

Table-5 and Figure-6 depict comparison of sensory nerve conduction parameters with HBA1c levels in diabetics. In diabetics with HbA1c levels (6-7%) there is significant reduction in conduction velocity of median& ulnar nerve (p<0.05) compared to diabetics with HbA1c (5-6%). There is reduction in amplitude of sensory nerves but values are not statistically significant.

Sensory nerve conduction parameters				
Median nerve	HBA1c (5-6%) (N=12) Correlation co-efficient (r)	p-value		
AMP(ms)	+0.019	p>0.05		
SNCV(m/s)	+0.135	p <0.05		
Ulnar nerve				
AMP(ms)	+0.258	p>0.05		
SNCV(m/s)	+0.162	p>0.05		
Table 6: Correlation between HbA1c Level 5-6 % and Nerve conduction (Sensory) parameters in Diabetics				

Table 6 depicts that there is positive correlation between HbA1c levels 5-6% and velocity of sensory nerves values are significant for median nerve. There is positive correlation between Hba1c levels 5-6% & velocity of sensory nerves and values are not statistically significant.

Sensory nerve conduction parameters				
Median nerve	HBA1c (6-7%) (N=12) Correlation co-efficient (r)	p-value		
AMP(ms)	-0.216	p>0.05		
SNCV(m/s)	-0.315	p<0.05		
Ulnar nerve				
AMP(ms)	-0.216	p>0.05		
SNCV(m/s)	-0.138	p>0.05		
Table 7: Correlation between HbA1c Level 6-7 % and Nerve conduction (Sensory) parameters in Diabetics				

Table 7 depicts that there is negative correlation between HbA1c 6-7% and velocity of median nerve (p<0.05). There is negative correlation between HbA1c levels & amplitude of sensory nerves and values are not significant.

DISCUSSION: Diabetic peripheral neuropathy (DPN) is an important complication and contributes to the morbidity of diabetes mellitus. Evidence indicates early detection of DPN results in fewer foot ulcers and amputations. DPN is a complex disease of progressive nerve fiber loss. A need exists for objective, simple, and reproducible assessment tools like NCS that can be readily used in clinical practice in asymptomatic stage so NCS is employed for screening diabetics for neuropathy in subclinical stage.

There is a progressive neuronal involvement in diabetics which is accelerated by poor glycaemic control. The estimation of both nerve conduction velocity and the HbA1c levels in diabetics is helpful in identifying the risk category for diabetic neuropathy, which is one of the main causes for severe morbidity among the diabetes mellitus patients. The present study is done to study the neuropathy changes in diabetics and correlate it with glycemic control.

As per American Diabetes Association 2014¹¹ the latest recommendations for diagnosis of Diabetes mellitus are as follows A1C \geq 6.5% or Fasting plasma glucose (FPG) \geq 126 mg/dL (7.0 mmol/L) or 2-hour plasma glucose \geq 200 mg/dL (11.1 mmol/L) during an OGTT or.

A random plasma glucose \geq 200 mg/dL (11.1 mmol/L), in patients with classic symptoms of hyperglycemia or hyperglycemic crisis. International Expert Committee added the A1C, (threshold \geq 6.5%) as a third option to diagnose diabetes.¹²

In the present study 60 age matched diabetics with good glycemic control on oral hypoglycemic drugs were included as cases and compared with 30 healthy non-diabetic controls. FBS, PPBS and HbA1C were significantly increased in diabetics compared to controls.

Type 2 DM is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production, and abnormal fat metabolism. In the early stages of the disorder, glucose tolerance remains near-normal, despite insulin resistance, because the pancreatic beta cells compensate by increasing insulin output.

As insulin resistance and compensatory hyperinsulinemia progress, the pancreatic islets in certain individuals are unable to sustain the hyperinsulinemic state. Impaired glucose tolerance, characterized by elevations in postprandial glucose, then develops. A further decline in insulin secretion and an increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia. A1C levels are directly proportional to blood sugar levels and concurrently there is increase in A1C levels in the present study.

The present study is similar to study by Munisekhar¹³ et al, Hussain Gauher et al¹⁴ who found FBS, PPPS & HbA1c levels to be higher in diabetics compared to control and the values were statistically significant.

As insulin resistance and compensatory hyperinsulinemia progress, the pancreatic islets in certain individuals are unable to sustain the hyperinsulinemic state. Impaired glucose tolerance, characterized by elevations in postprandial glucose, then develops. A further decline in insulin secretion and an increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia.A1C levels are directly proportional to blood sugar levels and concurrently there is increase in A1C levels in the present study.

In the present study there was significant decrease in conduction velocity of sensory nerves (Median and ulnar nerve) and amplitude and increase in latency in type 2 diabetics mellitus compared to controls and the values were significant for amplitude of motor nerves and median (Sensory) nerve.

This can be due to the raised blood glucose associated with diabetes causes chemical changes in nerves and damages blood vessels that carry oxygen and nutrients to the nerves. Excessive glucose metabolism causes decrease in the NO in nerves that dilates blood vessels, and low levels of NO may lead to constriction of blood vessels supplying the nerve in diabetic patients. The perfusion deficit is sufficient to cause endoneural hypoxia. These early events occur well before the development of clear pathological alterations to nerve capillaries such as basement membrane thickening, and are accompanied by functional deficits such as reduced NCV. Slowing of NCV indicates ongoing damage to myelin sheath and that the amplitude decreases with rising A1C levels suggesting the onset of axonal degeneration.

In diabetics with A1C levels of 6-7% there is negative correlation between HbA1c level & amplitude of median and ulnar sensory nerve but not significant, and the values are significant for velocity of median sensory nerve (p<0.05).

An extensive study conducted by Pitrat J of nearly 4400 clinical patients, reported a prevalence rate of diabetic neuropathy ranging from 7% of individuals within one year of diagnosis to 50% for those with diabetes for more than 25 years. The risk for complications of neuropathy increases with increasing duration and severity of hyperglycemia.¹⁵

Elevated HbA1c levels indicate improper metabolic control which causes intracellular hyperglycemia. Intracellular hyperglycemia activates the enzyme aldose reductase. This increases the formation of sorbitol leading to its accumulation, which is neurotoxic and this in turn reduces cellular Na-K ATPase. Uncontrolled hyperglycemia results in competitive inhibition of the sodium-dependent transport system responsible for myo-inositol uptake. This decreased uptake is hypothesized to contribute to the decreased concentration of myo-inositol in peripheral nerves as well as decreased sodium potassium ATPase activity which results in decreased nerve conduction.

Our study is in accordance with studies by Grat et al¹⁶ which had shown increased glycemic level was associated with abnormal NCV. A study done by Saboohi saed et al¹⁷ has suggested that A1C levels exhibits an inverse correlation with NCV of ulnar tibial nerves in type 2 diabetics aged 40-70 yrs.

A study done by Kanavi Roopa et al⁸ found negative correlation between HbA1c and NCV. A study done by Vishwanathan et al¹⁸ who observed an inverse correlation between sensory conduction velocity (SNCV) and HbA1c levels.

In a study by Munisekhar et al¹⁹ in similar lines to our study, it was concluded that sensory nerves are affected earlier in diabetic neuropathy. With proper glycaemic control the motor nerve changes and complications of sensory disturbance can be prevented by regular screening for glycosylated hemoglobin levels and nerve conduction studies.

In accordance to our study the study by Neelamba Prasad et al²⁰, revealed link between hyperglycemia and neuropathy and suggested that the aggressive management of hyperglycemia represent an important strategy to prevent the occurrence of neuropathy in patients with type 2 diabetes.

On the contrary, DCCT trial and Sosenko et al²¹found no correlation between A1C levels and peripheral neuropathy.

Nerve conduction study is a powerful test and can help in diagnosing subclinical cases of neuropathy¹⁴ so nerve conduction studies are employed in diabetics,as screening tool to detect neuropathy in subclinical stages so that strict glycemic control (A1C) can prevent onset and progression of neuropathic changes. Lowering of HbA1C is associated with reduction of microvascular and macrovascular complications of diabetes.²² So, strict glycemic control is important in delaying the onset and progression of microvascular complications.

CONCLUSION: The results of the present study done in diabetics on oral hypoglycemic without complications with duration of diabetes 1-10 yrs, indicate that there is a significant decline in the sensory conduction velocity of diabetics compared to normal healthy controls.

We observed progressive decline in sensory conduction velocity with the duration of the disease. We also found a negative correlation between HbA1c levels and nerve conduction velocity. This shows that poor metabolic control causes early onset and rapid progression of neuropathy.

We conclude the study with the observation that nerve conduction study can be used as a screening tool to diagnose neuropathy in subclinical stages and overweight diabetics should be considered at risk category for aggressive glycemic control by diet, drugs and life style modification to prevent progression of neuropathy.

RECOMMENDATIONS: Further studies with a larger sample size are required to study the correlation between Nerve conduction parameters and glycosylated haemoglobin and document serial changes with duration of diabetes in relation to glycemic control more precisely.

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