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NERVE CONDUCTION VELOCITY AMONG CLINICALLY ASYMPTOMATIC PATIENTS OF TYPE 2 DIABETES MELLITUS

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ABSTRACT **Objective:** Diabetes Mellitus is worldwide disease and diabetic peripheral neuropathy (DPN) is one of the most prevalent micro-vascular complication seen during the progress of disease. Frequently there is subclinical involvement seen for peripheral nerve functions which is revealed on nerve conduction study. Present study aims to assess nerve conduction velocity changes among asymptomatic patients with type 2 diabetes mellitus. **Material And Methods:** Total 60 subjects were included for the study, 30 of them were of type 2 diabetes mellitus on regular treatment with disease duration of less than 5 years and asymptomatic for neuropathy and 30 were controls. All of the participants were subjected to nerve conduction studies. **Results:** Nerve Conduction Velocity of Sural sensory nerve (44.42±6.93) was found to be significantly decreased in diabetic patients as compared to controls (50.31±6.65). No significant reduction of NCV was seen for motor nerves. **Conclusion:** Sensory NCV was significantly decreased in the Sural nerve compared to upper limb sensory nerves which suggests that long nerves are more prone to neuropathic changes even in asymptomatic diabetic patients. However no significant decrease was observed in Motor NCV among these patients.

KEYWORDS : Diabetes Mellitus, Nerve Conduction Velocity, Nerve Conduction Studies, Peripheral Neuropathy

INTRODUCTION

Diabetes mellitus (DM) is a chronic disease that occurs either when the body does not produce enough insulin or it cannot effectively utilize the insulin it produces. Type 2 DM is a heterogeneous group of disorders characterised by variable degrees of insulin resistance, impaired insulin secretion, along with Hyperglycaemia. Chronic complications of diabetes include various microvascular, macrovascular and non-vascular complications. One of the most prevalent microvascular complications among these is the diabetic peripheral neuropathy (DPN).

Globally, there were 366 million people with diabetes in 2011, and this is expected to rise to 552 million by 2030[1]. It is predicted that by 2030 diabetes mellitus may rise up to 79.4 million individuals in India [2].

DPN is one of the more common and troublesome complications affecting individuals with diabetes. Previous studies have demonstrated that the DPN is often associated with both diabetes duration and level of hyperglycaemia. There may be reduction to nerve conduction velocity (NCV) due to demyelination or reduction of amplitude due to axon loss in diabetic patients [3]. Usually, the symptoms develop at any stage of neuropathic impairment or may not develop at all [4].

Nerve Conduction Studies (NCS) are frequently used for the diagnosis of peripheral nerve disorders. These neurophysiological measurements have been established to be sensitive, specific and reproducible measures of the presence and severity of peripheral neuropathy. They also correlate with the morphological findings of nerve biopsy and thus define quantitative nerve dysfunction. Previous studies has shown that Nerve conduction abnormalities can be seen before manifestations of clinical symptoms in diabetic patients [4].

The early and precise detection of DPN can help in better understanding the pattern of pathophysiological changes as well as in controlling crippling illness like DPN. Therefore present study was designed to assess the NCV changes in asymptomatic patients with type 2 diabetic mellitus and compare them with controls [5].

MATERIAL AND METHODS

The present study was conducted in department of physiology NIMS Medical College and Hospital, Jaipur, Rajasthan as a part of post graduate programme pre-requisite. Total 60 subjects were included in the study out of which 30 were type 2 diabetes mellitus patients and 30 controls. Patients with Type 2 diabetes mellitus attending OPD, Department of Medicine and taking regular anti-diabetic medication were included in the study. All the participants were subjected to Nerve conduction studies.Motor nerve conduction velocity (MNCV) and sensory nerve conduction velocity (SNCV) was recorded for upper and lower limb nerves using standard recording protocol. [6, 7] They were compared for motor and sensory nerve conduction velocity with healthy controls (n=30) which includes patients relative and hospital staff. Medicaid systems EMG/NCV equipment with neuro-perfect software was used to perform the nerve conduction study.

Inclusion Criteria:

Control:-

- Informed consent
- Age and BMI matched.
- Normal healthy male aged 30-60 years.

Case:-

- Informed consent
- Diagnosed Type 2 Diabetic males with history of disease up to 2-5 years and no symptoms of neuropathy aged 30-60.
- The diagnosis of diabetes was made on the basis of (Revised American Diabetic Association criteria): Fasting glucose >126mg/dl and 2hr postprandial plasma glucose >200mg/dl.

Exclusion Criteria:

- Previous history of any systemic condition related to peripheral neuropathy (Hypertension. Malnutrition, Alcoholic neuropathy, Renal failure)
- Neuromuscular disorders such as myopathy, familial polyneuropathy or chronic polyneuropathy.
- Neuropathies associated with exogenous toxic agents, metals or drugs.
- Skin lesions or swelling that would interfere with NCS.
- Trauma in the course of nerve to be examined.

Statistical Analysis:

It was done using Statistical Package for Social Sciences version 17.0 (SPSS) software. Unpaired t–test was used and applied for the obtained data and p value <0.05 was taken as significant.

RESULTS:

SNCV:

A bilaterally reduced Sensory NCV was seen for Median and Ulnar nerves of upper limb and Sural sensory nerves of lower limbs in diabetic cases as compared to the control subjects but this decrease in SNCV was statistically significant (P value < 0.05) only for lower limb Sural sensory nerves. (Table -1)

MNCV:

Reduced nerve conduction velocity was observed for Median,

Table-1:Bilateral Comparison Between Mncv And Sncv Of Various Nerves In Control And Diabetics

PARAMETER	NERVE	CONTROLS (n=30) M ± SD (m/s)	DIABETICS (n=30) M ± SD (m/s)	P VALUE
Motor NCV	Median MNCV(Right)	55.89±3.21	55.36 ±3.29	>0.05
	Median MNCV (Left)	55.82±3.19	55.31±3.31	>0.05
	Ulnar MNCV(Right)	60.23±4.10	59.29±4.05	>0.05
	Ulnar MNCV (Left)	60.21±4.08	59.20±4.06	>0.05
	Common Peroneal MNCV(Right)	52.52±4.46	51.23±4.53	>0.05
	Common Peroneal MNCV (Left)	52.49±4.23	51.18±4.50	>0.05
Sensory NCV	Median SNCV(Right)	57.44±6.68	56.04±6.51	>0.05
	Median SNCV(Left)	57.32±6.42	55.94±6.49	>0.05
	Ulnar SNCV(Right)	56.41±7.04	55.10±7.00	>0.05
	Ulnar SNCV(Left)	56.21±7.13	55.02±6.98	>0.05
	Sural SNCV(Right)	50.31±6.65	44.42±6.93	**<0.05**
	Sural SNCV(Left)	51.00±6.52	46.08 ±5.32	**<0.05**

Ulnar nerves and Common Peroneal nerve in diabetic cases as compared to the control subjects but this decrease in NCV for motor nerve was not found to be statistically significant (p value > 0.05). (Table -1)

Data was presented as Mean ± Standard Deviation. Analysis was done by unpaired 'T- test' 'P' < 0.05 significant.

DISCUSSION

Present study was carried out to assess the NCV changes among asymptomatic type 2 diabetic mellitus patients. In this study more deterioration of NCV was found in nerves of lower limb as compared to nerves of upper limb. Similarly a study observed that nerves of lower limbs are more susceptible to diabetes assault as compared to upper limb suggesting that long nerves are commonly affected [8].

Decrease in SNCV in this study is consistent with the observation of (Tupkovic E et al) that showed SNCV was higher in control group as compared to diabetics whereas there was no significant difference in conduction velocity of diabetics and control groups in MNCV [9]. In present study motor conduction velocity was not significantly reduced in diabetic patients as that was seen for sensory nerves. These findings are in agreement with the observations made by previous studies available in literature. (Wakode et al) (Tayede et.al.) [7, 10].

The pathological mechanisms implicated in diabetic neuropathy, include micro-vascular damage, metabolic disorders, and changes in the interactions between neuronal and immunological systems in parallel with glial cell activation [17].

Changes in the blood vessels supplying the peripheral nerves underlie the mechanisms involved in micro-vascular damage and hypoxia. These include increase in wall thickness with the hyalinization of the vessel walls and the basal lamina of arterioles and capillaries, leading to nerve ischemia [11]. Metabolic disorders are common in diabetes. In hyperglycaemic state there is up-regulation of the NADPH oxidase complex that results in oxidative stress through reduced glutathione production, decreased nitric oxide concentrations and increased reactive oxygen species concentrations [12]. A growing body of evidence indicates that the activation of non-neuronal cells (microglia, astrocytes and immune cells) plays an important role in the development of neuropathic pain , and these cells are activated under hyperglycaemic conditions in the spinal cord [13,14].

There are many reports implicating the release of pro-inflammatory cytokines from glia and immune cells as a pathological mechanism for diabetic neuropathy such as IL-1b, IL-6 and TNFa [15].

Despite efforts to normalize the metabolic disturbances in diabetes, a large number of patients develop symptoms due to generalized vascular disease [16]. Long-term diabetic patients often suffer from advanced nonspecific atherosclerotic manifestations (macroangiopathy) as well as widespread lesions in the small vessels and capillaries (microangiopathy) [18].

CONCLUSION

Present study concludes that sensory nerves are more susceptible to diabetic neuropathy and lower limbs are more affected suggesting long nerves are more susceptible to neuropathic changes in type 2 diabetic mellitus patients, and frequently this involvement is subclinical.

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