VOLUME - 9, ISSUE - 7, JULY - 2020 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Physiology

TO ASSESS THE SENSORY NERVE CONDUCTION STUDY IN TYPE II DIABETES MELLITUS PATIENTS IN RAJASTHAN REGION

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ABSTRACT Diabetic neuropathies are nerve disorders associated with diabetes, which affect approximately half of all diabetes patients. The most common complication of diabetes is caused by hyperglycemia which can damage nerve fibers throughout the body. Depending on the types of nerves involved, diabetic neuropathies can be categorized as peripheral, autonomic, proximal, focal neuropathies. In this study, we have analyzed effect of diabetes on various parameters of upper limb nerve conduction study in 340 Type II diabetes mellitus subjects. All Type II diabetes mellitus subjects who recruited from OPD of the Department of Medicine, National Institute of Medical Sciences and research, Hospital, NIMS University Rajasthan Jaipur, Rajasthan. All the subjects are on hypoglycemic medicine. Performing Nerve conduction study (NCS) in all the subjects. Medicaid System's EMG/NCV equipment with Neurostim software was used for nerve conduction velocity. Prolongation of distal latency (DL) and reduced the amplitude and conduction velocity highly significant in Type II diabetes mellitus subjects as compared to healthy subjects of sensory nerve of upper limbs. So this study demonstrated that various parameters of nerve conduction study can be affected by hyperglycemia.

KEYWORDS : Type II diabetes mellitus, Nerve conduction study, Distal latency.

INTRODUCTION

Diabetes Mellitus is the most common non-communicable disease worldwide and has a huge impact on quality of life. In India about 62 million of people affected by diabetes in 2011which is projected to rise to 101 million by 2030 [1,2].

TIIDM is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production, and abnormal fat metabolism. TIIDM slowly develops in middle aged people and is usually milder and more stable[3, 4]. Peripheral neuropathy in diabetes may manifest indifferent forms, including sensory, motor, focal/multifocal, and autonomic neuropathies. About 80% of amputations occur after foot ulceration or injury, which can result of diabetic neuropathy[5].

Diabetic neuropathy is the more common complication of diabetes. It can lead to sensory loss and damage to the limbs and also leading cause of lower extremity amputations not related to injury. 50% of diabetes subjects have some form of neuropathy, and develop nerve problems at any time, but longer duration a person has diabetes, the high risk[6]. World Health Organization, studies suggested that up to 50% of people with diabetes are affected with nerve dysfunction. To some degree nerve destruction can lead to sensory depletion and affect to the limbs[7].

Nerve conduction Study is the standard measurement of diabetic neuropathy. It is non –invasive, least subjective single criterion[8]. In many instances if the diabetic peripheral neuropathy is diagnosed earlier, it can help in better understanding the pattern of pathophysiological changes as well as in controlling crippling illness like peripheral neuropathy[9].

After many years of diabetes some patients develop symptoms of peripheral neuropathy. It was further observed that nerve function is considerably disturbed in most long-term diabetics and nerve structure is severely damaged including segmental demyelination and axonal loss[10].

Hyperglycemia causes nerve damage by inducing the activation of the polyol, protein kinase C, hexosamine pathways and the accumulation of advanced glycation end products[11,12]. The oxidative stress depletes nitric oxide within the peripheral nerves and endothelium of the microvasculature by reducing endothelial nitric oxide synthase, altering nerve perfusion. In addition, there is deficiency of or a poor response to neurotrophic factors. However, there is now increasing evidence to suggest that autoimmunity has a role to play in the development and progression of diabetic neuropathies[13].

MATERIAL AND METHOD

The study was done in the Department of Physiology in National Institute of Medical Sciences and research, NIMS University Rajasthan, Jaipur. After approval of the ethical committee, 340 subjects were selected for study. All subjects were diagnosed TIIDM subjects. A brief explanation of the procedure was given to the subjects and voluntary informed consent was taken. Medicaid System's EMG/NCV equipment with Neurostim software was used for nerve conduction study.

Exclusion criteria:

- Trauma in the course of nerve to be examined.
- No previous history of any systemic condition related to peripheral neuropathy (Hypertension, Alcoholic neuropathy, Renal failure)
- Any neuromuscular disorders such as myopathy, familial polyneuropathy or chronic polyneuropathy.
- Neuropathies associated with exogenous toxic agents, metals ordrugs.

Inclusion criteria:

- Willingness (informed consent)
- Only Diagnosed cases of male and female Type II Diabetic aged 30-50 suffering from more than 2yrs.
- The diagnosis of diabetes is made on the basis of (Revised American Diabetic Association criteria) [14].

- Sensory Nerves of Upper limb was taken into the consideration and test was performed.

Statistical Analysis

Result was done using Statistical Package for Social Sciences version 17.0 (SPSS) software. Unpaired t–test was used for Nerve conduction velocity, latency and amplitude and applied for the obtained data and p value <0.05 is taken as significant.

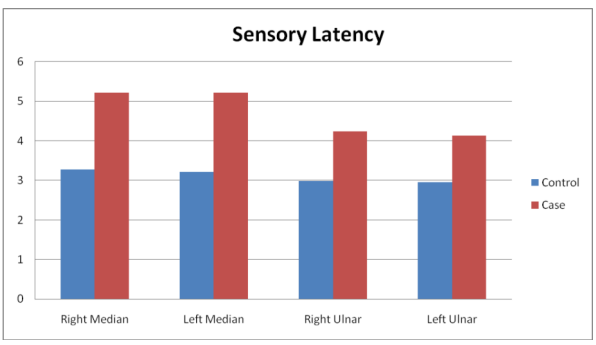
Observations and Results

Data was statistically analyzed using appropriate tests for sensory nerve study of upper limb in healthy and TIIDM subjects. The table no. 1 showed the mean (± SD) of various sensory nerve variables like latency, amplitude and nerve conduction velocity between control and cases. When compared these variables between controls and cases was statistically highly significant (p<0.001). Comparison of various sensory nerve variables like latency, amplitude and nerve conduction velocity between control and cases are graphically shown in graph -1, 2 and 3.

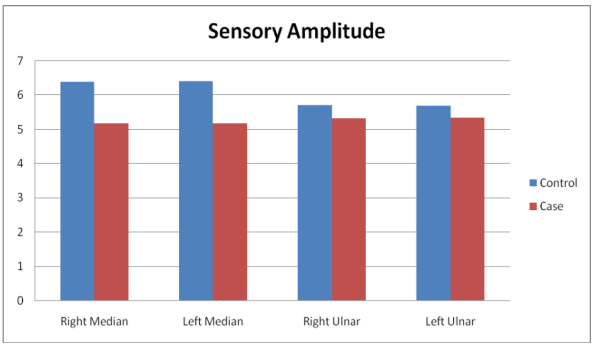
Table No. 1: Comparison of Various Sensory Nerve Studies of upper limb in Controls and Cases

Nerves	Parameters	Control (n-340)	Case (n-340)	P Value
		Mean± SD	Mean± SD	
Right Median Nerve	Latency (ms)	3.28 ± 0.71	5.22 ± 0.57	< 0.001
	Amplitude (mv)	6.40 ± 0.92	5.18 ± 0.56	< 0.001
	NCV (m/s)	54.42 ± 3.49	50.01 ± 2.30	< 0.001
Left Median Nerve	Latency (ms)	3.21 ± 0.69	5.22 ± 0.57	< 0.001
	Amplitude (mv)	6.41 ± 0.92	5.18 ± 0.56	< 0.001
	NCV (m/s)	54.48 ± 3.48	50.03 ± 2.29	< 0.001
Right Ulnar Nerve	Latency (ms)	2.98 ± 0.46	4.24 ± 0.43	< 0.001
	Amplitude (mv)	5.71 ± 0.88	5.33 ± 0.41	< 0.001
	NCV (m/s)	52.32 ± 2.99	49.95 ± 2.92	< 0.001
Left Ulnar Nerve	Latency (ms)	2.96 ± 0.36	4.14 ± 0.48	< 0.001
	Amplitude (mv)	5.69 ± 0.89	5.34 ± 0.42	< 0.001
	NCV (m/s)	52.27 ± 3.02	49.95 ± 2.93	< 0.001

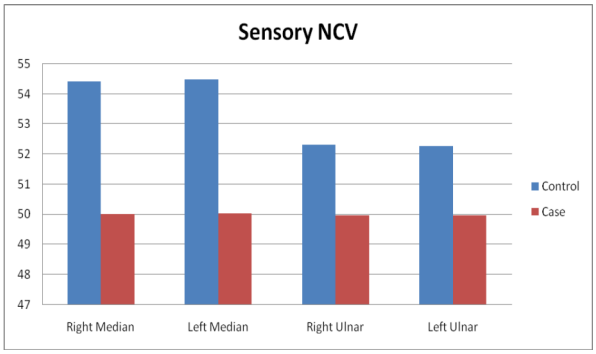
Graph: 1



Graph: 2



Graph: 3



DISCUSSION

Chronic peripheral nerve dysfunction is the important manifestation of the hyperglycemic neuropathies which is usually harmful in origin and can be the presenting feature in people with TIIDM. [15]

It was observed that latency of median, ulnar nerves of both sides was prolonged. These highly significant (p<0.001) when compared between control and cases. The amplitude of both nerves of both side were also diminished. These highly significant (p<0.001) when compared between control and cases. It also observed that Conduction velocities of all sensory nerves were also reduced. These observation highly significant (p<0.001) when compared between control and cases.

Similar results were also found by Rota E (2005)[16] found reduced sensory nerve action potential amplitude of median nerve in 70 % of the diabetic patients, ulnar nerve in 69% of diabetics. Leventoglu A (2009)[17] found decreased amplitude, conduction velocity and prolonged distal latency of median sensory nerve in diabetes.

Malhotra V (2002)[18] found decrease conduction velocity, distal amplitude and an increase in distal latency of median nerve in diabetics respectively as compared to control subjects. Kimura J (1979)[19] also found increased latency and decreased conduction velocity of ulnar, peroneal and tibial nerves in diabetics as compared to normal subjects. Our finding is in agreement with previous report done by Prasad NB (2011)[20] it was observed that sensory latency of median and ulnar nerves of both sides were significantly more in the diabetic group than the control group. Amplitude of all sensory nerves was found to be reduced in cases. CV of median and ulnar nerve was found to be significantly less in cases as related to controls.

Also, the present study was similar and comparable to the study done by Verma A (2005)[21] that showed that in comparison to normal healthy volunteers, amplitude and conduction velocity range is significantly decreased in diabetic patients. Similar findings were reached by Drory VE (1999)[22] reported that hypertriglyceridemia might be associated with mild axonal polyneuropathy.

We concluded that diabetic neuropathy is caused by dysfunction of the peripheral or central nervous system associated with abnormally high levels of blood glucose. It is often chronic and disabling. Due to the poorly understood mechanism, effective therapies that can cure diabetic neuropathy remain elusive. However, there exist various options to prevent or treat the disease. To date, the fundamental treatment for diabetic neuropathy is to keep blood glucose levels under control to prevent further nerve damage. Moreover, healthy lifestyle, quitting smoking will be beneficial to diabetic neuropathy and each diabetic neuropathy patient needs to be investigated thoroughly.

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