International Journal of Physiotherapy and Research,
Int J Physiother Res 2019, Vol 7(2):3011-15. ISSN 2321-1822
DOI: https://dx.doi.org/10.16965/ijpr.2019.102

Original Research Article

NEUROPHYSIOLOGICAL CHANGES IN PERSON WITH INSULIN DEPENDENT AND NON INSULIN DEPENDENT DIABETES MELLITUS

Maitrey Pandya *[1], Miral Damani [2].

*[1,2] MPT, Lecturer, Kamdar institute of physiotherapy, Rajkot, Gujrat, India.

ABSTRACT

Background: Diabetes mellitus is a disease caused by an inability of the body to metabolize glucose properly. The prevalence of diabetes is rapidly rising all over the globe at an alarming rate. As Insulin and non-insulin dependent diabetes shows their effects on various physiological systems includes central, peripheral and autonomic nervous systems, musculoskeletal, cardiovascular and other vital systems. A common complications due to the IDDM and NIDDM includes peripheral neuropathy, retinopathy, nephropathy and vascular complication. Insulin and non-insulin dependent diabetes mellitus, both affect the peripheral nervous system significantly. Therefore we would like to find out neurophysiological changes on peripheral nervous systems between insulin and non-insulin dependent diabetes mellitus.

Aim: To find out the Neuro-physiological changes between IDDM and NIDDM.

Materials and Method: 120 individuals screened with SF36 (general health good and above) were included with age limit between 25 to 60 years. Those individuals having a history of hospitalization in last 1 year, acute fever, present history of radiculopathy and open wound were excluded. They were divided into 2 groups IDDM and NIDDM. For nerve conduction study–distal latency, amplitude and NCV of sensory and motor nerves were performed. Nerve conduction studies of common peroneal, tibial and sural nerves were examined in both groups. Latency, NCV and CMAP/SNAP were taken as outcome measures.

Result and Discussion: Bio-statistical analysis has been done using Mann-Whitney test. Result suggest that there is a significant difference in Neurophysiological changes ($p<0.05$) between IDDM and NIDDM groups. **Conclusion:** In context to our study and neurophysiological findings, individuals with IDDM must be taken into consideration for promotion, prevention, and care as compared to NIDDM for secondary complications.

KEY WORDS: Insulin dependent diabetes mellitus (IDDM), Non-insulin dependent diabetes mellitus (NIDDM), Neurophysiological changes.

Address for correspondence: Dr. Maitrey Pandya, MPT, Lecturer, Kamdar institute of physiotherapy, Rajkot,Gujrat, India. **E-Mail:** maitreypandya07@gmail.com

Access this Article online	Journal Information
Quick Response code	**International Journal of Physiotherapy and Research**
	ICV for 2016 86.93
	ISSN (E) 2321-1822 | ISSN (P) 2321-8975
	https://www.ijmhr.org/ijpr.html
	DOI-Prefix: https://dx.doi.org/10.16965/ijpr
DOI: 10.16965/ijpr.2019.102	**Article Information**
	Received: 10 Jan 2019 Accepted: 15 Feb 2019
	Peer Review: 10 Jan 2019 Published (O): 11 Mar 2019
	Revised: None Published (P): 11 Apr 2019

INTRODUCTION

Diabetes mellitus is characterised by chronic hyperglycaemia with disturbance of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action or both. The two broad categories of diabetes are designated Insulin dependent and non-dependent. IDDM is the result of complete or near-total insulin deficiency. NIDDM is a heterogeneous group of disorders characterized by varying degrees of insulin resistance, impaired insulin secretion, and increased glucose production [1].

According to the Diabetes Atlas 2006 published by the International Diabetes Federation, the number of people with diabetes in India currently

around 40.9 million is expected to rise to 69.9 million by 2025 unless urgent preventive steps are taken [2].

Insulin dependent and non insulin dependent diabetes shows their effects on various physiological system like central nervous system, peripheral nervous system, musculoskeletal system, autonomic nervous system and cardiovascular system [3,4]. Common complications due to the IDDM and NIDDM includes peripheral neuropathy, retinopathy, nephropathy and vascular complication. Insulin dependent and non insulin dependent diabetes mellitus, both affect the peripheral nervous system significantly [5]. Diabetic peripheral neuropathy (DPN) is among the most distressing of all the chronic complications of diabetes and is a cause of significant disability and poor quality of life [6]. The presence of peripheral diabetic neuropathy is suggested by complaints of numbness, pain, or both, usually in a symmetrical distribution and noticed first in the toes [7].

Electrophysiological studies have revealed a number of abnormalities in diabetic neuropathy [8-11]. Patients with signs of neuropathy have slower nerve conduction velocities and smaller amplitudes than those without symptoms [12,13], showing a close correlation between clinical findings and the degree of conduction changes [14,15].

Insulin dependent and non insulin dependent diabetes mellitus shows their effect on various physiological systems. Common complications due to IDDM and NIDDM include peripheral neuropathy, retinopathy, nephropathy and vascular complications. IDDM and NIDDM both affect the peripheral nervous system significantly. Therefore, we would like to find out neurophysiological changes on the peripheral nervous system between individual with IDDM and NIDDM.

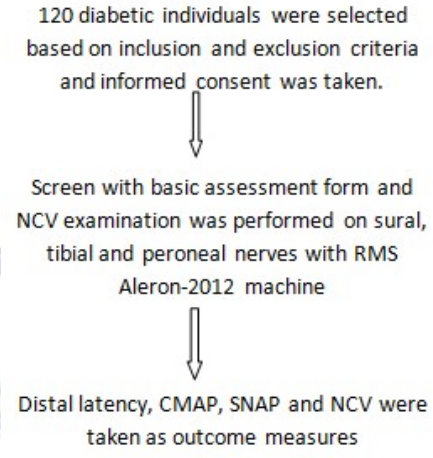
Aim of the study is to find the neurophysiological changes between IDDM and NIDDM individuals.

Review of literature:

Arindam [16] has observed that there is significant correlation between peripheral neuropathy and duration of diabetes, age of patients and postprandial blood glucose level. Kari et al [17] has found that after 40 years of type 1 diabetes small fiber sensory neuropathy is a major manifestation in type 1 diabetes. AL Kakrani [1] had observed that involvement of lower limbs (tibial and sural) is more common than upper limbs.

MATERIALS AND METHODS

Flow chart 1: Procedure and data collection.



This was an observational cross sectional study which included 120 subjects. (IDDM – 40 and NIDDM – 80) Subjects between 25 to 60 years of age and individuals with IDDM and NIDDM screen with SF-36, general health good and above were included in the study. Subjects having present history of lower limb Radiculopathy, history of hospitalization in last 1 year, open wound and acute fever were excluded from the study. The room temperature was maintained between 21c to 23c. The skin resistance was reduced by cleaning with spirit. Supra maximal stimulation was given for the nerve conduction examination.

120 diabetic individuals were selected based on inclusion and exclusion criteria and informed consent was taken.

Screen with basic assessment form and NCV examination was performed on sural, tibial and peroneal nerves with RMS Aleron-2012 machine

Distal latency, CMAP, SNAP and NCV were taken as outcome measures

Surface recordings for common peroneal nerve were obtained from extensor digitorum brevis and stimulation was given at the ankle and at the neck of the fibula. For Sural nerve examination, the surface electrode between lateral malleolus and tendoachilles records nerve

conduction of sural nerve. The nerve was stimulated antidromically 10-16 cm proximal to the recording electrode, distal to the lower border of gastrocnemius at the junction of the middle and lower third of the leg. The active surface recording electrode for Tibial nerve was placed on abductor hallusis or abductor digiti quiniti slightly below and anterior to navicular tuberosity. Surface stimulation was used behind and proximal to the medial malleolus and in the popliteal fossa and slightly lateral to the midline in the popliteal fossa.

RESULTS

Bio-Statistical analysis was done using SPSS (version 21). Mann-Whitney test was used for data analysis. Level of significance was 0.05.

Table 1: Demographic data which include age, blood glucose control and duration of IDDM and NIDDM groups.

GROUP	AGE IN YEARS	BLOOD GLUCOSE			DURATION
A-IDDM B-NIDDM		FBS	PPBS	HbA1c %	In years
A (n-40)	42.1 ± 8.78	144.53 ± 31.66	198.35 ± 66.4	7.91 ± 1.75	10.85 ± 5.28
B (n-80)	50.44 ± 6.72	134.51 ± 23.01	175.04 ± 32.8	6.79 ± 1.06	6.51 ± 2.9

Table 2: Neurophysiological changes in Sural nerve.

GROUP	SURAL NERVE		
A-IDDM	Latency	SNAP	NCV
B-NIDDM	ms	µv	m/s
A (n-40)	3.5 ± 0.75	10.24 ± 5.92	39.17 ± 3.09
B (n-80)	3.06 ± 0.65	14.16 ± 5.94	41.3 ± 2.47
Mann Whitney test (P < 0.05)	0.0033*	0.0002*	0.020*

Table 3: Neurophysiological changes in Tibial nerve

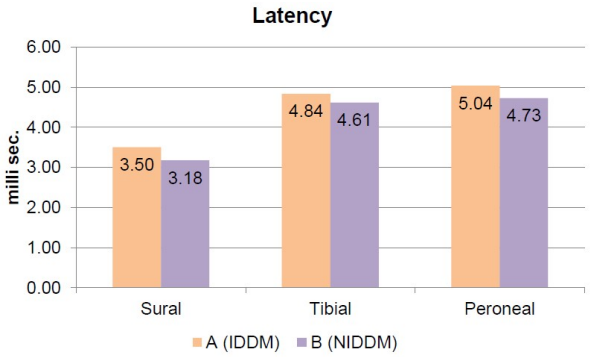
GROUP	TIBIAL NERVE		
A-IDDM	Latency	CMAP	NCV
B-NIDDM	ms	mv	m/s
A (n-40)	5.24 ± 0.69	5.21 ± 1.63	38.11 ± 2.53
B (n-80)	4.61 ± 0.67	6.04 ± 1.32	40.93 ± 2
Mann Whitney test (P < 0.05)	0.05*	0.0158*	0.029*

Table 4: Neurophysiological changes in Peroneal nerve.

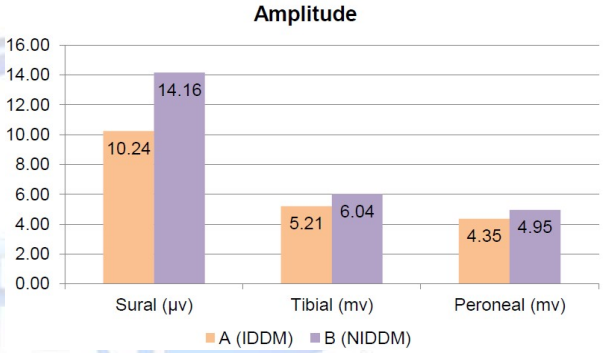
GROUP	PERONEAL NERVE		
A-IDDM	Latency	CMAP	NCV
B-NIDDM	ms	mv	m/s
A (n-40)	5.04 ± 0.72	4.35 ± 1.32	38.6 ± 2.91
B (n-80)	4.73 ± 0.65	4.95 ± 0.9	40.89 ± 2
Mann Whitney test (P < 0.05)	0.0149*	0.01*	0.0169*

*=suggest significant difference

Graph 1: Latency of Sural, Tibial and Peroneal nerve which suggest that latency is higher in IDDM group compare to NIDDM group.



Graph 2-Amplitude of Sural, Tibial and Peroneal nerve which suggest that amplitude is lower in IDDM group compare to NIDDM group



Graph 3: NCV of Sural, Tibial and Peroneal nerve which suggest that NCV is lower in IDDM group compare to NIDDM group.

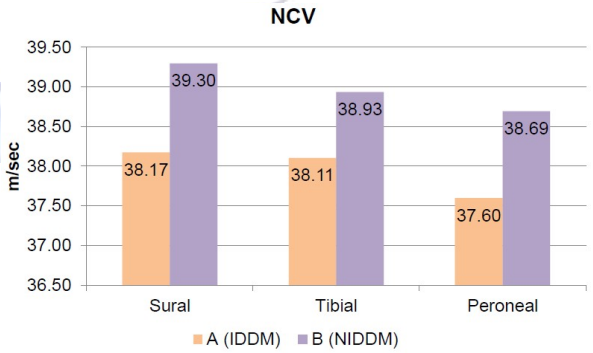


Table 2-4 and graph 1-3 suggest that there is significant difference in neurophysiological changes between IDDM and NIDDM group. IDDM group is more affected than NIDDM. Latency, amplitude and NCV show significant difference ($p<0.05$). Latency is higher in IDDM group compare to NIDDM group. Amplitude is lower in the IIDM group compare to the NIDDM group. NCV is lower in the IDDM group compare to the NIDDM group.

DISCUSSION

In the present study significant impairment was observed in IDDM group on neurophysiological

changes because IDDM group has longer duration of exposure. IDDM has poor glycemic control; high glucose variability which is characterised by improper synthetic insulin dosage compare to the amount of glucose intake .IDDM has longer duration of exposure of diabetes mellitus.

Poor glycemic control has more effect on peripheral nerve in IDDM group compared to NIDDM. Because of poor glycemic control there is an increase in the amount of Sorbitol. In hyperglycemias, glucose shunted through the Sorbitol pathway, causes the accumulation of sorbitol in Schwann cells, which undergo osmotic damage leading to segmental demyelination. This finding would support the sorbitol pathway hypothesis [18]. Other factors considered important in the pathogenesis include insulin deficiency and altered myoinositol metabolism. Vascular insufficiency quantitatively aggravates diabetic neuropathy [19]. Insufficient to cause infarction may result in measurable functional and morphological abnormalities in peripheral nerves [20]. Ischemic changes in the nerve presumably result from the proliferation of the endothelial in the blood vessels and abnormalities of the capillaries [21]. Because of poor glycemic control, IDDM group has major impact on neurophysiological changes.

Duration of exposure of IDDM group is more compared to NIDDM however the mean age is low. Duration of exposure has a significant effect on the peripheral nervous system. Our finding is consistent with the previous findings of Vinik,Gregersen , Valensi et al [22-24]. Knuiman et al [25] also reported that sensory neuropathy is more common in long standing diabetic subjects especially in those who develop the disease late in life. No significant sensory nerve dysfunction was found in the diabetic group with relatively short duration of diabetes.

Summary: Prevalence of diabetes is increasing worldwide. IDDM and NIDDM both, affects Physiological systems of our body. We did an NCV examination of IDDM and NIDDM individuals. Results suggest that IDDM people have major affection compared to NIDDM. So, individuals with IDDM must be taken into consideration for promotion, prevention, and care as compared to NIDDM for secondary complications.

CONCLUSION

Neurophysiological changes in IDDM show major impact on peripheral nerves compare to NIDDM. Therefore, individuals with IDDM must be taken into consideration for promotion, prevention, and care as compared to NIDDM for secondary complications.

Future study scope: The study can be repeated to find the therapeutic effect of exercise in diabetic individuals in context to neurophysiological changes. The study can be repeated to find proximal nerve involvement in individual with IDDM and NIDDM.

Conflicts of interest: **None**

REFERENCES

[1]. AL Kakrani, Gokhale, Karan Vohra et al. Clinical and Nerve Conduction Study Correlation in Patients of Diabetic Neuropathy. Journal of the association of physicians of India. .2014; 62: 24-7.

[2]. Sarah Wild, Gojka, Anders et al. Global Prevalence of Diabetes-Estimates for the year 2000 and projections for 2030. The American diabetes association. 2004; 27: 1047-53.

[3]. Lindsay, Bone, Callander. Neurology and neurosurgery illustrated. 3rd edition. 1997:525.

[4]. Aspi and Sharukh Golwala. Medicine for students. 22nd edition. 2009; 437-44.

[5]. Mamta , Abigail, Catherine et al. Peripheral Neuropathy in Adolescents and Young Adults With Type1 and Type2 Diabetes From the search for Diabetes in Youth Follow-up Cohort. American diabetes association. 2013; 36:3903-9.

[6]. Jie Ding, Carol, Kamran et al. Early Retinal Arteriolar Changes and Peripheral Neuropathy in Diabetes. The American Diabetes Association. 2012; 35:1098-1104.

[7]. Callaghan, Little, Feldman et al. Enhanced glucose control for preventing and treating diabetic neuropathy (Review). The cohort collaboration.2012; 6:1-55.

[8]. Jun Kimura M. D. Electro diagnosis in Diseases of Nerve and Muscle. Principles and Practice. 3rd Ed.; 1987: 653.

[9]. Claus, Mustafa, Vogel et al. Assessment of diabetic neuropathy. Definition of norm and discrimination of abnormal nerve function. Muscle nerve 1993; 16:757–768.

[10]. Dyck, Karnes, Daube et al. Clinical and Neuro pathological criteria for the diagnosis and staging of diabetic polyneuropathy. Brain. 1985; 108:861–880.

[11]. Mackel. Properties of cutaneous afferents in diabetic neuropathy. Brain. 1989; 112:1359–1376.

[12]. Niakan, Harati. Sympathetic skin response in diabetic peripheral neuropathy. Muscle nerve. 1988;11:261–264.

[13]. Le Quesne, Fowler, Parkhouse . Peripheral neuropathy profile in various groups of diabetics. Journal of neurology neurosurgery and psychiatry. 1990; 53:558–563.

[14]. Halar, Graf, Halter et al. Diabetic neuropathy, A clinical, laboratory and electro diagnostic study. Archives of physical medicine and rehabilitation. 1982; 63:298–303.

[15]. Conrad B, Aschoff JC, Fischler M.neurophysiological changes in patients with long duration diabetes.J Neurology. 1975;210:151–159.

[16]. Arindam, Santa, Premchandra. Prevalence of peripheral neuropathy in newly diagnosed type 2 diabetics. International Journal of diabetes devloping country. 2005;25:30-3.

[17]. Kari,Mortin, Bassam et al. Small and large fiber neuropathy after 40 years of type 1 diabetes. Diabetes Care.2013; 36:3712-17.

[18]. Fagius, Jameso. Effects of aldose reductase inhibitor treatment in diabetic polyneuropathy. Journal of neurology neurosurgery and psychiatry.1981; 44:991–1001.

[19]. Ram, Sadeh, Walden et al. Vascular insufficiency quantitatively aggravates diabetic neuropathy. Archives of neurology. 1991;48:1239–1242.

[20]. Sladky, Tschoepe, Greenberg et al. Peripheral neuropathy after chronic endoneurial ischemia. Annals of neurology. 1991; 29:272–278.

[21]. Malik, Veves, Masson et al. Endoneurial capillary abnormalities in mild human diabetic neuropathy. Journal of neurology neurosurgery and psychiatry. 1992;55:557–561.

[22]. Vinik, Holland, Beua et al. Diabetic neuropathies. Diabetes care 1992;15 (12): 1926-1951.

[23]. Gregersen.Diabetic neuropathy, influence of age, sex, metabolic control and duration of diabetes on motor conduction velocity. Neurology 1967; 17: 972-980.

[24]. Valensi, Giroux, Seeboth et al. Diabetic peripheral neuropathy: effects of age, duration of diabetes, glycemic control and vascular factors. Journal of Diabetes and its Complication. 1997; 11: 27-34.

[25].Kuniman, Wilborn, Mc Cann, et al. Prevalence of diabetic complications in relation to risk factors. Diabetes 1986; 35: 1332-1339.

How to cite this article:

Maitrey Pandya, Miral Damani. NEUROPHYSIOLOGICAL CHANGES IN PERSON WITH INSULIN DEPENDENT AND NON INSULIN DEPENDENT DIABETES MELLITUS. Int J Physiother Res 2019;7(2):3011-3015. **DOI:** 10.16965/ijpr.2019.102