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Original Article

Physiology Section

Nerve Conduction among Middle-aged Indian Diabetic Males without Clinical Neurodeficit: A Cross-sectional Study

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ABSTRACT

Introduction: Peripheral neuropathy is one of the dreaded complications of diabetes mellitus. Data pertaining to subclinical neuropathies among diabetics are deficient, especially in the Indian context.

Aim: The aim of this study is to evaluate the Nerve Conduction Study (NCS) parameters in middle-aged Indian males with diabetes who do not exhibit any clinical neurodeficits, and to compare the findings with age-matched, non diabetic healthy controls. The present study also explores the correlations between NCS parameters and glycaemic control (HbA1c) in the study population.

Materials and Methods: This cross-sectional study was conducted at IPGME&R Kolkata, West Bengal, India from February 2021 to July 2022. A total of 165 diabetic males without any neurological symptoms and 54 age-matched non diabetic controls were included in this study. Onset latencies, amplitudes, and conduction velocities of the median, ulnar, tibial, peroneal, and sural nerves were compared between the groups using an unpaired Student's t-test. The correlation between NCS parameters and HbA1c levels among diabetics was checked using the Pearson correlation coefficient. A p-value of <0.05 was considered significant.

Results: In the present study, 165 diabetic individuals and 54 non diabetic controls were included. When comparing the nerve conduction parameters, the diabetic individuals showed significant delays in sensory and motor nerve conduction across all peripheral nerves examined. For instance, the conduction velocities (m/s) of the motor and sensory components of the median nerve were found to be 56.73±4.96 and 65.85±9.84 in the non diabetic controls, whereas they were 53.71±7.23 and 59.71±11.87 in the diabetic patients ($p<0.05$ in each case). Except for the motor amplitudes of the tibial and ulnar nerves, all other peripheral nerves in both the upper and lower limbs showed significantly higher amplitudes of motor and sensory action potentials among the non diabetic controls. HbA1c was found to have significant positive correlations with NCS parameters, including the motor onset latencies of the median, ulnar, tibial, and peroneal nerves, as well as the sensory onset latencies of the median, ulnar, and sural nerves.

Conclusion: Peripheral neuronal conduction is affected in diabetic patients even before any neurological symptoms appear, and the degree of neurodeficit is dependent on glycaemic control.

Keywords: Diabetic neuropathy, Glycosylated haemoglobin, Peripheral nerves, Subclinical neuropathy

INTRODUCTION

Diabetes mellitus refers to a group of common metabolic disorders that cause various metabolic dysregulations in our body by reducing insulin secretion, decreasing peripheral glucose utilisation, and increasing glucose production [1]. It has become a global epidemic over the last few decades. In 2024, the International Diabetes Federation estimates the global prevalence of diabetes among adults aged 20-79 years at 589 million (11.1%) [2].

The metabolic dysregulation associated with diabetes causes secondary pathophysiological changes in multiple organ systems [1]. Morbidity and mortality in diabetes are primarily related to its complications. Complications associated with diabetes can be divided into vascular and non vascular categories, and vascular complications can be further subdivided into micro-vascular and macro-vascular complications [1]. Peripheral neuropathy is one of the most serious microvascular complications of diabetes. The prevalence of peripheral neuropathy is estimated to range between 6% to 51% among diabetic individuals, depending on age, duration of diabetes, glycaemic control, and type of diabetes [3]. Distal symmetrical polyneuropathy usually manifests as burning or stabbing pain or deep ache in the limbs, which may often worsen at night. However, these neurological symptoms can vary widely, from subclinical neuropathy to extremely severe neuropathy with life-threatening autonomic dysfunction [4].

Measurement of glycosylated haemoglobin (HbA1c) is considered as the standard method of assessing long-term glycaemic control, it is established that improvement in glycaemic control can lower the risk of diabetic complications, especially the microvascular complications. As erythrocytes have an average life span of 120 days, HbA1c reflects consistently elevated plasma glucose level over previous 2-3 months [1]. A NCS is considered a reliable, sensitive, and non invasive objective technique for diagnosing neuropathy [5,6]. The parameters of nerve conduction, such as onset latency, amplitude, and conduction velocity, are valuable for identifying neuronal deficiencies [6].

The Modified Toronto Clinical Neuropathy Score (mTCNS) is a valid tool for monitoring and diagnosing distal symmetric polyneuropathy, demonstrating considerably high sensitivity and specificity. It combines symptoms (including foot pain, numbness, tingling, weakness, ataxia, and upper limb symptoms) with physical examination findings (such as pain, temperature, light touch, vibration sense, and position sense). Each component of this scoring system is graded from 0-3 for discrete evaluation [7]. A score of 0-5 is considered indicative of 'no or minimal neuropathy' [8].

Although there is significant interest among researchers regarding diabetic neuropathy and its prevention, most studies globally have focused on clinically evident or overt diabetic neuropathy [9-12]. There is a substantial lack of information regarding the occurrence of delayed nerve conduction in neurologically asymptomatic

diabetic individuals and its correlation with glycaemic control in India. Since the majority of diabetics belong to the age group of 40-60 years [1,13], this demographic was identified as the most suitable for exploring the targeted objectives. Several studies have shown that postmenopausal women are at an increased risk of developing peripheral neuropathy due to lower levels of estrogen and progesterone [14,15]. Additionally, gender differences in nerve conduction parameters have also been documented [16]. Therefore, female participants were excluded from the current study to avoid significant variable factors due to time constraints.

Consequently, the present study was conducted to evaluate the NCS parameters in middle-aged Indian males with diabetes who do not exhibit any clinical neurodeficit, and to compare the findings with those of non diabetic healthy controls. This study also explored the correlations between NCS parameters and HbA1c in the study population.

MATERIALS AND METHODS

This analytical cross-sectional study was conducted in the Physiology Department in collaboration with the Endocrinology Department of the Institute of Post Graduate Medical Education and Research, Kolkata, West Bengal, India over a period of eighteen months from February 2021 to July 2022. The study was conducted after obtaining ethical clearance from the Institutional Ethics Committee (Memo No. IPGME&R/IEC/2021/171, date: 17/02/2021). Written informed consent was obtained from all participants.

Inclusion criteria: In this study, newly diagnosed middle-aged (40-60 years) diabetic male participants who did not exhibit symptoms of clinical neurodeficit were included as cases while attending the diabetic clinic at the institute. The study sample consisted of established cases of diabetes mellitus, as per criteria laid down by the American Diabetes Association [17]. The mTCNS was used to assess neuropathy clinically; participants with scores of 0-5 (indicating no clinical neuropathy) were included in this study. As the control group, healthy male volunteers in the same age range (40-60 years) without diabetes or any neurodeficits were included.

Exclusion criteria: The exclusion criteria for both groups were:

- Patients with chronic symptoms related to diabetic complications.
- Patients diagnosed with any form of neurological disease.
- Participants with any history of previous injury or gastric bariatric surgery, as gastric bariatric surgery affects the intestinal absorption of dietary Vitamin B12 [18].
- Participants with a history of addiction to alcohol, cannabis, or tobacco. Several studies have indicated that addiction to these substances may affect peripheral neuronal conduction [19-21].
- Participants with a history of certain drug intake (such as isoniazid, ethambutol, antiretroviral drugs, chemotherapy, etc.) or a history of toxicity (such as industrial exposure, heavy metals, zinc toxicity, etc.).
- Obesity {Body Mass Index (BMI) ≥30 kg/m^2}, as obesity is considered an important risk factor for neuropathy [22].
- Individuals who were strict vegans. Various studies have shown that dietary deficiency of Vitamin B12 can cause peripheral neuropathy [23]. Other literature suggests that vegans are more likely to experience a dietary deficiency of Vitamin B12 [24].

Sample size calculation: Sample size (N) was calculated from the formula, $N=Z^2pq/d^2$,

Where, Z is the statistic corresponding at the level of confidence. At 95% level of confidence, Z=1.96~2, Z^2~4. In this equation, p is present prevalence and present prevalence of diabetes in the world among 20-79 age-group estimated for 2024 is 11.1% [2], q is (100-p) and d is allowable error of p (5%).

Hence, N=(4) (11.1) (100-11.1)/ $(5)^2$=157.9. We took sample size of 219 male participants where 165 diabetic individuals and 54 non diabetic healthy male individuals of the same age group, using simple random sampling without replacement via computer-generated random number tables.

Cases were matched with the control group concerning age and BMI, except for glycaemic status.

Study Procedure

Detailed clinical history and thorough clinical examination were conducted for all participants after obtaining their written informed consent. All selected participants then underwent Nerve Conduction Studies (NCS) on the median, ulnar, tibial, peroneal, and sural nerves. NCS was performed using an RMS EMG machine (RMS Aleron 201 EMG machine) with surface electrodes, following standardised techniques [6] at the Neurophysiology Laboratory of our Physiology Department. The room temperature was maintained at a normal ambient temperature (21°C - 23°C) [6].

In motor NCS, surface stimulation of the nerve was administered using a square wave pulse of 0.1 ms duration with an intensity of 5-40 mA. The filter setting for motor NCS was 5 Hz to 10 kHz, and the sweep speed was set at 2-5 ms/division. A ground electrode was placed between the stimulator and the recording electrode, resulting in a biphasic action potential being recorded. In sensory NCS, the filter setting was 10 Hz to 2 kHz, sweep speed was 1-2 ms/division, and gain was set to 1-5 mV/division [6]. For each nerve, NCS was performed on one side only.

The present glycaemic control for each participant was assessed by measuring their current HbA1c levels.

STATISTICAL ANALYSIS

All data were computed using Microsoft® Excel® 2016 MSO (Version 2207 Build 16.0.15427.20182). The data for each parameter of NCS were expressed as mean±SD. Study variables were compared between groups using an unpaired Student's t-test. Correlations between the blood level of HbA1c and NCS parameters among the diabetic participants in our test group were assessed using the Pearson correlation coefficient (r-value). The p-values of Pearson correlation coefficients at the specific degrees of freedom for this study were calculated using Statistical Package for the Social Sciences (SPSS) version 25.0. A "p-value" of less than 0.05 was regarded as statistically significant; very small p-values were reported as <0.001.

RESULTS

In the present study, 165 diabetic participants without clinical neurodeficit and 54 age-matched non diabetic controls were included. Among both groups, age and anthropometric parameters (height, weight, BMI) were comparable [Table/Fig-1].

Participants' particulars	Diabetic patients without clinical neuropathy (Mean±SD)	Non diabetic control (Mean±SD)	Result of unpaired t-test (p-value)
Age (years)	47.84±5.49	47.56±4.94	0.739
Height (cm)	159.25±9.33	160.72±7.14	0.291
Weight (kg)	62.12±6.41	62.05±8.23	0.948
BMI (kg/m^2)	24.85±4.56	24.11±3.45	0.277
HbA1c (%)	7.67±1.78	4.78±0.38	<0.001

[Table/Fig-1]: Comparison of baseline data between diabetic and non diabetic group.

When compared with an age-matched control group, diabetic individuals showed significant differences in both motor and sensory onset latencies of the median and ulnar nerves, motor onset latency of the tibial and peroneal nerves, and sensory onset latency of the sural nerves [Table/Fig-2]. Significant differences were also observed

Parameters under study (motor or sensory onset latency)	Diabetic patients without clinical neuropathy (Mean±SD)	Non diabetic control (Mean±SD)	Result of unpaired t-test (p-value)
Median motor onset latency (ms)	4.05±1.36	3.37±0.26	<0.001*
Ulnar motor onset latency (ms)	2.68±0.9	2.3±0.63	0.004*
Tibial motor onset latency (ms)	4.15±0.53	3.84±0.54	<0.001*
Peroneal motor onset latency (ms)	3.94±0.99	3.44±0.5	<0.001*
Median sensory onset latency (ms)	3.01±0.72	2.62±0.26	<0.001*
Ulnar sensory onset latency (ms)	2.58±1.23	2.17±0.53	0.019*
Sural sensory onset latency (ms)	4.21±1.51	3.43±0.63	<0.001*

[Table/Fig-2]: Comparison of motor and sensory onset latencies between diabetic and non diabetic group.
*Significant p-value of unpaired t-test.

among diabetics in the motor amplitude of the median and peroneal nerves, as well as sensory amplitude of the median, ulnar, and sural nerves [Table/Fig-3]. Similarly, statistically significant changes were noted among diabetic participants in the motor conduction velocity of the median, ulnar, tibial, and peroneal nerves, and the sensory conduction velocity of the median, ulnar, and sural nerves [Table/Fig-4]. In our study, with 165 diabetic participants (n), the degrees of freedom in these correlations are 163 (n-2=163). Significant positive correlations were found between HbA1c and NCS parameters, such as motor onset latencies of the median, ulnar, tibial, and peroneal nerves, and the sensory distal latencies of the median, ulnar, and sural nerves. In contrast, significant negative correlations were found between HbA1c and NCS parameters, including the motor conduction velocities of the median, ulnar, and tibial nerves, sensory conduction velocities of the median, ulnar, and sural nerves, as well as the amplitudes of motor action potentials of the median, ulnar, and peroneal nerves and the sensory amplitude of the median nerve only [Table/Fig-5].

Parameters under study (motor or sensory amplitude)	Diabetic patients without clinical neuropathy (Mean±SD)	Non diabetic control (Mean±SD)	Result of unpaired t-test (p-value)
Median motor amplitude (mV)	8.95±4.03	11.05±2.42	<0.001*
Ulnar motor amplitude (mV)	8.49±2.94	9.23±2.69	0.106
Tibial motor amplitude (mV)	10.21±4.87	11.23±7.04	0.235
Peroneal motor amplitude (mV)	4.32±2.97	5.67±2.24	0.002*
Median sensory amplitude (µV)	31.08±15.86	38.67±11.62	0.001*
Ulnar sensory amplitude (µV)	31.09±25.48	42.59±16.28	0.002*
Sural sensory amplitude (µV)	11.09±8.04	16.24±11.62	<0.001*

[Table/Fig-3]: Comparison of motor and sensory amplitudes between diabetic and non diabetic group.
*Significant p-value of unpaired t-test.

Parameters under study (motor or sensory conduction velocity)	Diabetic patients without clinical neuropathy (Mean±SD)	Non diabetic control (Mean±SD)	Result of unpaired t-test (p-value)
Median motor conduction velocity (m/s)	53.71±7.23	56.73±4.96	0.005*
Ulnar motor conduction velocity (m/s)	57.07±6.41	60±5.08	0.003*
Tibial motor conduction velocity (m/s)	43.49±4.6	45.33±2.91	0.006*
Peroneal motor conduction velocity (m/s)	44.29±6.36	46.45±4.91	0.023*
Median sensory conduction velocity (m/s)	59.71±11.87	65.85±9.84	<0.001*
Ulnar sensory conduction velocity (m/s)	54.26±19.25	64.84±19.26	<0.001*
Sural sensory conduction velocity (m/s)	41.4±9.74	46.36±4.91	<0.001*

[Table/Fig-4]: Comparison of motor and sensory conduction velocities between diabetic and non diabetic group.
*Significant p-value of unpaired t-test.

Correlations among parameters (HbA1c and onset latency)	r value/ p-value	Correlations among parameters (HbA1c and amplitude)	r value/ p-value	Correlations among parameters (HbA1c and conduction velocity)	r value/ p-value
Median motor onset latency (ms)	+0.399 <0.001*	Median motor amplitude (mV)	-0.277 0.001*	Median motor conduction velocity (m/s)	-0.474 <0.001*
Ulnar motor onset latency (ms)	+0.496 <0.001*	Ulnar motor amplitude (mV)	-0.340 <0.001*	Ulnar motor conduction velocity (m/s)	-0.344 <0.001*
Tibial motor onset latency (ms)	+0.713 <0.001*	Tibial motor amplitude (mV)	-0.062 0.567	Tibial motor conduction velocity (m/s)	-0.607 <0.001*
Peroneal motor onset latency (ms)	+0.313 <0.001*	Peroneal motor amplitude (mV)	-0.176 0.027*	Peroneal motor conduction velocity (m/s)	-0.097 0.321
Median sensory onset latency (ms)	+0.436 <0.001*	Median sensory amplitude (µV)	-0.651 <0.001*	Median sensory conduction velocity (m/s)	-0.695 <0.001*
Ulnar sensory onset latency (ms)	+0.421 <0.001*	Ulnar sensory amplitude (µV)	-0.007 0.995	Ulnar sensory conduction velocity (m/s)	-0.236 0.002*
Sural sensory onset latency (ms)	+0.237 0.014*	Sural sensory amplitude (µV)	-0.061 0.502	Sural sensory conduction velocity (m/s)	-0.490 <0.001*

[Table/Fig-5]: Correlations between the Glycosylated Haemoglobin (HbA1c) and NCS parameters in diabetic group.
*Significant p-value of Pearson correlation.

DISCUSSION

In the present study, while comparing the nerve conduction parameters of neurologically asymptomatic diabetic individuals with an age-matched non diabetic control group, significant changes in NCS parameters in the diabetic group were found. These changes include motor onset latencies of the median, ulnar, tibial, and peroneal nerves, sensory onset latencies of the median, ulnar, and sural nerves, motor amplitudes of the median and peroneal nerves, sensory amplitudes of the median, ulnar, and sural nerves, motor conduction velocities of the median, ulnar, tibial, and peroneal nerves, and sensory conduction velocities of the median, ulnar, and sural nerves.

Studies by Agarwal S et al., Chidri SV and Vidya G, Al-Taweel YA et al., and Zhang YQ et al., show findings that corroborate with those of the present study [25-28]. They also found that neurologically asymptomatic diabetic patients had significant differences in several NCS parameters of peripheral nerves compared to their control group. The probable changes in peripheral nerves due to hyperglycemia include microvascular constriction, alterations in Schwann cell-axon transport, changes in protein expression in the dorsal root ganglia, demyelination, and degeneration of axons [29]. The obvious consequence of demyelination is slow, continuous impulse conduction through axons.

In our study, we did not observe significant changes in the motor amplitudes of the ulnar and tibial nerves. This finding is consistent

with the study conducted by Zhang YQ et al., which showed that sensory nerves are affected to a greater extent than motor nerves [28]. This may be attributed to the greater vulnerability of sensory neurons to the insults of diabetes compared to motor neurons. C fiber axons are unmyelinated and thin, and the dorsal root ganglia are not protected by a blood-nerve barrier, unlike motor neurons [29].

On the contrary, findings from a few studies do not corroborate our results. Karsidag S et al., and Bhowmik NB et al., showed in their studies that only the conduction velocity of distal peripheral nerves in the extremities is affected in diabetic patients [30,31]. They concluded that in the early stage of diabetes, the axoplasmic flow may be impaired; hence, the conduction velocity in long peripheral nerves of the extremities may also be affected [30,31].

In our study, we also found significant correlations between the HbA1c levels of diabetic patients and their peripheral nerve conduction parameters. Our findings align with those of other researchers (Al-Taweel YA et al., [27], Agarwal S et al., [25], Karsidag S et al., [30]), who also found significant correlations between HbA1c and peripheral nerve conduction dysfunction. The probable pathophysiology behind these correlations may be due to dysregulation of cellular metabolism resulting from the excessive entry of glucose. This leads to depletion of ATP production and increased production of reactive oxygen species. Consequently, the continued excess of substrates can cause loss of axonal mitochondrial function, resulting in the degradation of axonal structure and function, which in turn leads to diabetic neuropathy [29].

On the contrary to our findings, Akaza M et al., Pinto MV et al., and Su J et al., concluded that increased blood glucose variability is closely associated with peripheral neuropathy in type 2 diabetics and could be considered a potent indicator for diabetic peripheral neuropathy in these patients [32-34]. To support their conclusion, the study by Wentholt IM et al., can be mentioned, which demonstrated that increased glucose variability can activate the overproduction of reactive oxygen species, increase the production of inflammatory cytokines, induce cell apoptosis, and stimulate epigenetic changes that might heighten the risk of diabetic peripheral neuropathy [35].

Limitation(s)

Age-wise stratification of the diabetic population could not be performed. Follow-up for NCS parameters concerning glycaemic control could not be completed. We relied on patients' history for the duration of the disease through verbal information in most cases. Histopathological examination of peripheral nerve tissue could not be conducted to objectively prove diabetes-induced neuronal damage.

CONCLUSION(S)

The present study indicates that compared to non diabetic controls, nerve conduction is significantly impaired in diabetic patients even without clinical neurodeficit. Impairment of nerve conduction is significantly correlated with poor glycaemic control in those patients.

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