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Original Research Article

The Impact of Diabetes Mellitus on Nerve Conduction: A Cross Sectional Study

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Abstract:

Background and Objectives: A serious ailment that impairs quality of life and has societal ramifications is diabetic peripheral neuropathy. Early detection and intervention might alter the course of neuropathic processes and dramatically reduce associated morbidity and mortality. This study looks at how long-term blood sugar management affects diabetic peripheral neuropathy in people with type 2 diabetes.

Material and Methods: Medical College Hospital in India hosted a hospital-based study. All participants older than eighteen who have suffered from Type 2 diabetes for no more than ten years were recruited. The BMI, HbA1c level, and nerve conduction studies (NCS) were computed using established methodologies. The data were analyzed using SPSS, version 25.0, a statistical programme for social sciences. P-values less than 0.05 were deemed statistically significant.

Results: Of the 95 T2DM patients, 52 were male. Our findings indicate that as the duration of diabetes grew, the motor nerve reduced from 63.392 ± 2.378 to 53.868 ± 2.082 (P = 0.0029), and the sensory velocity fell from 64.068 ± 3.222 to 54.002 ± 5.338 (P = 0.0502, P = 0.0032, respectively). Additionally, greater duration of diabetes was associated with a substantial drop in motor nerve amplitude from 8.788 ± 3.108 to 6.942 ± 1.838 (P = 0.0502) and sensory nerve amplitude from 25.712 ± 5.702 to 19.512 ± 6.508 (P = 0.0032). In addition, both NCS parameters (amplitude and velocity) decreased when HbA1c was greater than 6: sensory velocity decreased from 63.962 ± 2.358 to 55.488 ± 2.432 (P = 0.028), and motor velocity decreased from 63.002 ± 2.588 to 51.438 ± 1.662 (P = 0.022). Additionally, the sensory amplitude decreased from 26.908 ± 1.262 to 20.848 ± 2.102 (P = 0.0508), whereas the motor amplitude decreased from 6.878 ± 3.552 to 6.608 ± 3.288 (P = 0.0508). Moreover, a significant (P = 0.0508) correlation has been seen between the motor and sensory amplitudes and BMI.

Conclusion: Elevated BMI and inadequately managed (elevated HbA1c) chronic diabetes had a detrimental impact across all of the nerve conductivity evaluation metrics.

Keywords: Amplitude; Diabetes mellitus; Motor nerve conduction; Peripheral neuropathy; Sensory nerve velocity.

Introduction

Individuals with diabetes mellitus (DM), a metabolic disorder, are more prone to experiencing microvascular and macrovascular complications [1]. With the increasing global diabetes epidemic, the prevalence of diabetes-related issues is bound to rise. Peripheral neuropathy, a result of diabetes, can significantly impair social, financial, and overall quality of life aspects. [2] Worldwide, 1.91% of diabetics have diabetic neuropathy (DN) [3]. DN ranged in Northern Africa from 21.9% to 60%. [4] According to Awadalla's (2017) calculations, diabetic polyneuropathy affected 68.2% of Sudanese population [5]. Uncontrolled glucose levels are a significant risk factor for the emergence of diabetic neuropathy (DN) in individuals with type 2 diabetes (T2DM). [6] Early detection and effective glucose control can significantly alter the progression of diabetic neuropathy and lower morbidity and mortality. Through its effects on inflammation and

metabolism, type 2 diabetes and degenerative DN may hasten the aging-related loss of strength and muscle mass. As a result, the negative cycle of diabetes and ageing will hasten the emergence of limitations related to daily living tasks and hasten the loss of autonomy. [7] In those with type 2 diabetes, DPN was found to be influenced by the duration of DM and glycemic control. The findings and implications of the study will emphasize how important it is to control diabetes mellitus as soon as possible in order to lessen or delay the pathophysiology that causes PDN.

Aim and Objectives: This study looks at how long-term blood sugar management affects the development of diabetic peripheral neuropathy in people with type 2 diabetes.

Material and Methods

This cross-sectional study was carried out in a medical facility. The Medical College Hospital in India served as the study's site. Each participant provided informed, written consent. The parameters that were evaluated yielded findings that were given to the participants. 95 adults over the age of 18 who had been diagnosed with T2DM 10 years prior were included in this research. Patients having a history of myopathies, drug-induced neuropathy, known-cause peripheral neuropathy, cerebrovascular stroke, and those with type 1 diabetes were among the exclusion criteria. Other known aetiologies of peripheral neuropathy included hereditary neuropathy and neuromuscular diseases. [8]

Metrics anthropometric: Weight was calculated using a calibrated weight and height measuring device in compliance with authorized procedures. The following formulae were used to calculate BMI: height squared in metres times' body weight (in kilograms). Following fulfilment of the selection criterion, BMI was classified as kg/m2 as follows: Normal weight was defined as 18.5–24.9; overweight was defined as 25–29.9; obesity I was defined as 30–34.9; obese II as 35–39.9; and obese III as ≥40.

Chemical analysis: A volume of five millilitres of venous blood was extracted from the antecubital vein of each participant, ensuring a sterile environment. Each participant's HbA1c level was determined using the NycoCard©Reader, an enzyme-linked immunosorbent assay (ELISA) reader that was specifically made for this purpose.

Electrophysiological investigation: A solitary technician conducted nerve conduction studies (NCS) using widely accepted methodologies [7]. The Neurowerk EMG device, developed by Neuroevolution, Sistemas Medicos Lda in Germany, is a surface stimulator specifically designed for nerve conduction studies (NCS). Recordings of potentials were obtained using circular disc electrodes of 10 mm in diameter. The examination required the use of both the upper and lower extremities. Measurements were taken for distal motor and sensory latencies, as well as sensory and motor amplitudes. Typically, we received a minimum of ten responses. The gadget autonomously computed the latencies and amplitudes. To calculate the conduction velocity (CV) of both motor and sensory functions, divide the measured distance by the delay of onset.

Measurements of nerve conduction in the upper limbs: Only the sensory functions of the radial nerve were evaluated, whereas both the sensory and motor functions of the median and ulnar nerves were examined. An electrode was used to assess the medial motor nerve. The electrode was placed precisely at the halfway between the distal wrist crease and the very first metacarpophalangeal joint.

The electrodes for the ulnar motor nerve were positioned around the 1st metacarpophalangeal joint, though the electrodes for the extensor carpi ulnaris were put on the thumb and in the middle of the forearm. The median sensory nerve innervated the second and third digits. Both the radial and ulnar edges of the finger under examination have a ring electrode implanted. The electrode was placed slightly closer to the finger's tip than its base. There were four centimetres separating the reference electrode and the active electrode.

Lower limb nerve conduction measurements: Sensory information of the sural nerve was assessed in both lower limbs, whereas motor information of the peroneal as well as tibial nerves was examined. Electrodes were placed on the lateral malleolus (ankle bone) and calf to evaluate the functioning of the sural nerve. The electrodes were positioned at the knee and ankle to specifically stimulate the peroneal nerves, which are located at the tip of the fibula, and the tibial nerves, which are located near the ankle. Measurements were conducted to ascertain the magnitude of the action potential and the speeds at which both motor and sensory activities are conducted.

Inclusion criteria: According to the WHO definition, individuals with type 2 diabetes who had been diagnosed for 10 years or longer and were above the age of eighteen were included. Participants in this study who were able and willing to provide permission were both male and female.

Exclusion criteria: People with type-1 diabetes or a history of peripheral neuropathy unrelated to diabetes who were younger than eighteen were not eligible. The group did not include those with a history of myopathies, neuromuscular diseases, drug-induced neuropathy, genetic neuropathy, or

peripheral neuropathy with a known aetiology, such as cerebrovascular stroke.

Statistical Analysis: The statistical analysis was carried out using the social science statistical programmes (SPPS). For the continuous variable, the mean was shown, and for the categorical variables, the number of occurrences with percentages was presented. Statistically significant was defined as having a P-value of less than 0.05. When comparing data between more than two groups, an ANOVA was employed.

Results

This study included 95 T2DM patients, with a 54.7% male prevalence. With a mean age of 49, the participants' ages varied from 30 to 79 years old. The individuals' average height was 91.668 ± 3.168 kg, and their average weight was 169.732 ± 7.228 cm. The individuals' average HbA1c was 8.04% and their average BMI was 30.562 ± 3.178. [Table 1]

Table 1: Demographic profile

	Minimum value	Maximum value	Mean ± SD
Age (years)	30	79	49.011±11.489
BMI	17.3	50.1	28.709±6.591
HbA1c (%)	5.7	13.0	8.009±1.819
Fasting blood glucose (gm/dl)	78	406	184.789±77.111

Our findings showed a significant reduction in the velocities of both the motor and sensory nerves as the duration of diabetes grew beyond five years (P-value = 0.0032; P-value = 0.0502, respectively).

Furthermore, the findings showed that both the sensory nerve amplitude (P-value = 0.0032) as well as motor nerve amplitude (P-value = 0.0502) significantly decreased as the duration of diabetes raised beyond five years, but the mean HbAIc did not significantly differ between the two groups after five years.

Our results showed a significant correlation (P-value < 0.05) between the amplitudes of the sensory and motor nerves and BMI for underweight, normal weight, overweight, and obese persons. [Table 2, 3]

Table 2: Nerve velocities and amplitude among participants

Variables	<5 years (Mean ± SD)	5–10 years (Mean ± SD)	P-value
Age (years)	49.289 ± 8.111	53.209 ± 10.789	0.0329
HbA1c	7.709 ± 1.589	8.189 ± 1.711	0.2909
Sensory nerve velocity (m/sec)	64.068 ± 3.222	54.002 ± 5.338	0.0502
Motor nerve velocity (m/sec)	63.392 ± 2.378	53.868 ± 2.082	0.0029
Sensory nerve amplitude (mV)	25.712 ± 5.702	19.512 ± 6.508	0.0032
Motor nerve amplitude (mV)	8.788 ± 3.108	6.942 ± 1.838	0.0502

Table 3: Nerve conduction and HbA1c

Variables	HbA1c < 6 (Mean ± SD)	HbA1c > 6 (Mean ± SD)	P-value
Sensory nerve velocity (m/sec)	63.962 ± 2.358	55.488 ± 2.432	0.028
Motor nerve velocity (m/sec)	63.002 ± 2.588	51.438 ± 1.662	0.022
Sensory nerve amplitude (mV)	26.908 ± 1.262	20.848 ± 2.102	0.0508
Motor nerve amplitude (mV)	6.878 ± 3.552	6.608 ± 3.288	0.0508

Discussion

Diabetic foot consequences most commonly and problematically result in diabetic peripheral neuropathy (DPN). Diabetic persons who show symptoms and/or proof of peripheral nerve injury have been diagnosed with diabetic neuropathic pain (DN) after excluding all other possible causes. This pain can either be confined or spread across the body. [9, 10] Regardless of its clinical spectrum, neurological symptoms have long been linked to diabetes. As a matter of fact, there is an abundance of clinical evidence about the neurological effects of diabetes, and it is evident that the central nervous system, peripheral nervous system, and ocular symptoms are frequently involved and may even have been recorded [11]. The clinical manifestations of DPN depend on the extent of nerve injury. NCS has been proposed as a means of validating DPN findings [12]. The NCS study found that individuals with diabetes for more than five years had a decrease in both the speed and size of sensory and motor nerve fibres.

The statistical analysis showed a significant reduction in these measures with a P-value of 0.05. The findings were consistent with a five-year assessment of patients with T2DM carried out in Jordan by Khawaja et al. They found that the duration of diabetes had the greatest impact on the

development of DPN. These findings demonstrated a link between neuropathy and the duration of diabetes. However, our data also showed a significant (P-value 0.05) association among BMI & the motor as well as sensory amplitudes for both motor & sensory fibres in individuals with T2DM, which aligns with the results reported by Khawaja et al. [13]. In addition, Zhang et al. discovered that the median, ulnar, posterior tibial, and common peroneal nerves in the diabetic groups showed increased distal motor latency, reduced sensory nerve conduction velocity, and substantially reduced sensory nerve action potential along with compound muscle action potential amplitude compared to the control group [15].

The results of our research align with a study conducted by Mayeda et al., which concluded that continuous monitoring of glucose is more effective than haemoglobin A1c (HbA1c) testing in recognising potential complications, including diabetic peripheral neuropathy (DPN). Additionally, our findings demonstrated that regular HbAIc monitoring and well-managed diabetes mellitus can help avoid neurological problems [16]. Another study, which demonstrated that those with HbA1c levels more than 10% had reduced cardiovascular (CV) and amplitude potential compared to those with HbA1c levels of 10%, likewise yielded findings consistent with our own research [17]. Partanen et al.'s study revealed that individuals with NIDDM had diminished sensory amplitude in the radial nerve, as well as decreased motor amplitude in the median nerve. Furthermore, these individuals also had shown reduced nerve conduction velocity in both sensory and motor nerves.

These findings are consistent with the significant unfavourable association between type II diabetes mellitus and our study's findings. [18] In addition, EmanAbd El Aziz Galbat et al. discovered that inadequate management of blood sugar levels was linked to a more severe form of diabetic peripheral neuropathy (DPN).

A study on nerve conduction revealed a significant positive association among motor and sensory conduction velocity (CV) & amplitude, as well as a robust negative connection between motor and sensory delay. [19] To prevent more serious consequences and enhance patients' quality of life, primary care doctors must educate their patients, recognise issues early, and administer the proper therapy.

Limitations of study: Since the cross-sectional design makes it difficult to investigate the chronology for the genesis of the observed deviations, we are left with only assumption. Additionally, the sample size for this study was rather little; it could be better to look at a larger sample in the future.

Conclusion

The aim of our study was to examine the relationship between BMI and the duration of DM in patients with diabetic neuropathy (DN) who were having nerve conduction exams. Both variables exhibited statistical significance at a significance level of $P < 0.05$. Together, these findings provide a vital understanding of the neurological symptoms those individuals with DN experience.

Our research indicates that continuous inflammation can lead to damage to the axons, resulting in muscular weakness and neuropathic pain. This can have a substantial impact on the quality of life for those with DN. Additionally; it may be beneficial to elucidate the neurological implications in individuals with DN.

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