

Research Article

Effect of Uncontrolled Type-II Diabetes Mellitus and Its Duration on Nerve Conduction Parameters in Adult Sudanese Patients in Khartoum State

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Abstract

Background: Peripheral neuropathy is a serious complication of diabetes, which has socioeconomic consequences as well as a reduced quality of life. Early neuropathic process recognition and management could alter its course and considerably reduce the associated morbidity and mortality. This study determines the effect of long-term glycemic control on diabetic peripheral neuropathy in people with type 2 diabetes (T2DM).

Methods: A hospital-based study was carried out at the National Centre of Neurosciences and Ibrahim Malik Hospital in Khartoum. All individuals who were older than 18 years and have had T2DM for less than 10 years were recruited. Using accepted techniques, the BMI, HbA1c level, and nerve conduction studies (NCS) were measured. Data were analyzed using the Statistical Package for Social Sciences (SPSS), version 25.0 software. P -value ≤ 0.05 was considered significant.

Results: Of the 95 patients with T2DM, 52 were male patients. Our findings showed that as the duration of diabetes increased, the sensory velocity reduced from 64.07 ± 3.22 to 54.00 ± 5.34 and the motor nerve from 63.39 ± 2.38 to 53.87 ± 2.08 ($P = 0.05$, $P = 0.003$, respectively). Additionally, with increased duration of diabetes, a significant decrease was seen in both motor nerve amplitude from 8.79 ± 3.11 to 6.94 ± 1.84 ($P = 0.05$) and sensory nerve amplitude from 25.71 ± 5.70 to 19.51 ± 6.51 ($P = 0.003$). Also, all parameters of NCS (velocity and amplitude) decreased when Hb A1c was >6 – sensory velocity from 63.96 ± 2.36 to 55.49 ± 2.43 ($P = 0.03$) and motor velocity from 63.00 ± 2.59 to 51.44 ± 1.66 ($P = 0.02$). And sensory amplitude decreased from 26.91 ± 1.26 to 20.85 ± 2.1 ($P = 0.05$), while motor amplitude decreased from 6.88 ± 3.55 to 6.61 ± 3.29 ($P = 0.05$). Additionally, there is a substantial ($P = 0.05$) correlation between sensory and motor amplitudes and the BMI.

Conclusion: High BMI and poorly controlled (high HbA1c) long-term diabetes had a negative impact on all nerve conduction study parameters.

Keywords: diabetes mellitus, peripheral nerve neuropathy, body mass index

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1. Introduction

Microvascular and macrovascular problems are more common in people with diabetes mellitus (DM), a metabolic condition [1]. As the worldwide diabetes epidemic grows, diabetes complications will definitely surge. Peripheral neuropathy, one of the diabetes complications, can have serious social, economic, and quality-of-life consequences [2]. Globally, 1.91% of people with diabetes suffer from diabetic neuropathy (DN) [3]. In Northern Africa, including Morocco, Algeria, Tunisia, Libya, Egypt, Sudan, South Sudan, and Western Sahara, DN ranged from 21.9% to 60% [4]. In 2017, Awadalla estimated the prevalence of diabetic polyneuropathy in Sudan to be around 68.2% [5]. Poor glucose control is one of the risk factors strongly associated with the advancement of DN in type 2 diabetes (T2DM) patients [6]. Early detection of DN and good glycemic control can significantly alter its course, resulting in significantly lower morbidity and mortality. The impact of both type 2 DM and DN on metabolism and inflammation could hasten the aging-related loss of muscle mass and strength. As a result, this vicious cycle of diabetes and ageing will speed up the loss of independence and lead to an increase in the development of disabilities in daily living tasks [7]. The study determined the effect of DM duration and glycemic control on DPN in persons with type 2 DM. The findings/outcome of this research will emphasize the early detection of DM and management to abate or retard pathophysiology leading to PDN.

2. Methods

This study was a hospital-based cross-sectional study. The research work was carried out between 2020 January and 2021 January at Ibrahim

Malik Hospital in Khartoum city, which provides endocrinology and diabetes services, and the National Center of Neurosciences, in Khartoum city which provides neurology services to patients from Khartoum State and referrals from other states.

Each subject provided a written informed consent. Results of tested parameters were delivered to participants.

A total of 95 participants, aged >18 years, diagnosed with T2DM (<10 years) were included in this study. Those with type 1 DM, peripheral neuropathy of known etiologies, that is, neuromuscular diseases, inherited neuropathy, alcoholic patients with history of myopathy, drugs-induced neuropathy, peripheral neuropathy with known cause, and cerebrovascular stroke were excluded [8].

2.1. Anthropometric measures

Weight was measured using a calibrated weight and height measuring scale following standard procedures. BMI was calculated according to formulae; body weight (in kgs) over the height squared in meters. Once the selection criteria were met, BMI were classified as follows: 18.5–24.9 as normal weight; 25–29.9 as overweight; 30–34.9 as obese I; 35–39.9 as obese II; and ≥ 40 as obese III in kgs/m^2 .

2.2. Chemical analysis

Five milliliters of venous blood were drawn from each participant's antecubital vein under sterile conditions, and HbA1c levels were tested using a customized enzyme-linked immunosorbent assay (ELISA) reader known as the NycoCard[®] Reader.

2.3. Electrophysiological investigation

One technician used normal methods to perform nerve conduction studies (NCS) [7]. Neurowerk EMG system electromyography, a surface stimulator (Neuroevolution, Sistemas Medicos Lda, Germany), was utilized for NCS. Potentials were recorded using round disk electrodes with a diameter of 10 mm. The test was carried out on both the upper and lower limbs. Distal motor and sensory latencies, as well as sensory and motor amplitudes were measured. An average of 10 or more replies were obtained. The latencies and amplitudes were computed automatically by the device. By dividing the measured distance by the onset delay, the motor and sensory conduction velocity (CV) was estimated.

2.4. Upper limbs nerve conduction measurement

The motor and sensory nerves of both upper limbs were evaluated, with the median and ulnar nerves testing for both motor and sensory, and the radial nerve testing for sensory only. An electrode was used to assess the medial motor nerve. The electrode was placed halfway between the distal wrist crease and the first metacarpophalangeal joint. Electrodes for the ulnar motor nerve were placed slightly distal to the first metacarpophalangeal joint, while electrodes for the extensor carpi ulnaris were placed at the mid-forearm and on the thumb. The median sensory nerve connected the second and third digits. A ring electrode was implanted on the radial and ulnar sides of the digit being evaluated, slightly distal to the digit's base. While the reference electrode was 4 cm away from the active electrode.

2.5. Lower limb nerve conduction measurement

The peroneal and tibial nerves for motor and the sural nerve for sensory were evaluated in both lower limbs. Electrodes were placed on the calf and lateral malleolus to examine the sural nerve. Electrodes were put at the knee and ankle for peroneal nerves and near the tip of the fibula and ankle for tibial nerves. The amplitude of the action potential, as well as the motor and sensory conduction velocities were all measured.

2.6. Inclusion criteria

Patients aged >18 years, with a known case of T2DM (diagnosed as per the WHO criteria) for ≤10 years were included. Both males and females, willing and capable of providing consent, were included in this study.

2.7. Exclusion criteria

Patients aged <18 years, with type 1 DM, a known case of peripheral neuropathy other than DM were excluded. Patients with neuromuscular diseases, inherited neuropathy, alcoholic patients with history of myopathy, drugs-induced neuropathy, peripheral neuropathy with known cause, that is, cerebrovascular stroke were excluded.

2.8. Statistical analysis

Statistical analysis was performed using the statistical packages of the social science (SPSS). The categorical variables were shown as the number of cases with percentages and the continuous variable was shown as mean. *P*-value < 0.05 was considered statistically significant. To compare between more than two groups, ANOVA was used.

TABLE 1: Descriptive distribution of participants.

	Minimum	Maximum	Mean	Std. Deviation
Age (yr)	30	79	49.0	11.5
BMI	17.3	50.1	28.7	6.6
HbA1c (%)	5.7	13.0	8.04	1.81
Fasting blood glucose (gr/dl)	78	406	184.8	77.1

TABLE 2: Sensory and motor nerve velocity and amplitude among diabetic participants.

Variables	<5 years	5–10 years	P-value
	Mean \pm SD		
Age (yr)	49.3 \pm 8.1	53.2 \pm 10.8	0.032
HbA1c	7.7 \pm 1.6	8.2 \pm 1.7	0.29
Sensory velocity (m/s)	64.07 \pm 3.22	54.00 \pm 5.34	0.05
Motor velocity (m/s)	63.39 \pm 2.38	53.87 \pm 2.08	0.003
Sensory amplitude (mv)	25.71 \pm 5.70	19.51 \pm 6.51	0.003
Motor amplitude (mv)	8.79 \pm 3.11	6.94 \pm 1.84	0.05

TABLE 3: Association between various nerve conduction and HbA1c.

Variables	HbA1c < 6	HbA1c > 6	P-value
	Mean \pm SD		
Sensory velocity (m/s)	63.96 \pm 2.36	55.49 \pm 2.43	0.03
Motor velocity (m/s)	63.00 \pm 2.59	51.44 \pm 1.66	0.02
Sensory amplitude (mv)	26.91 \pm 1.26	20.85 \pm 2.17	0.05
Motor amplitude (mv)	6.88 \pm 3.55	6.61 \pm 3.29	0.05

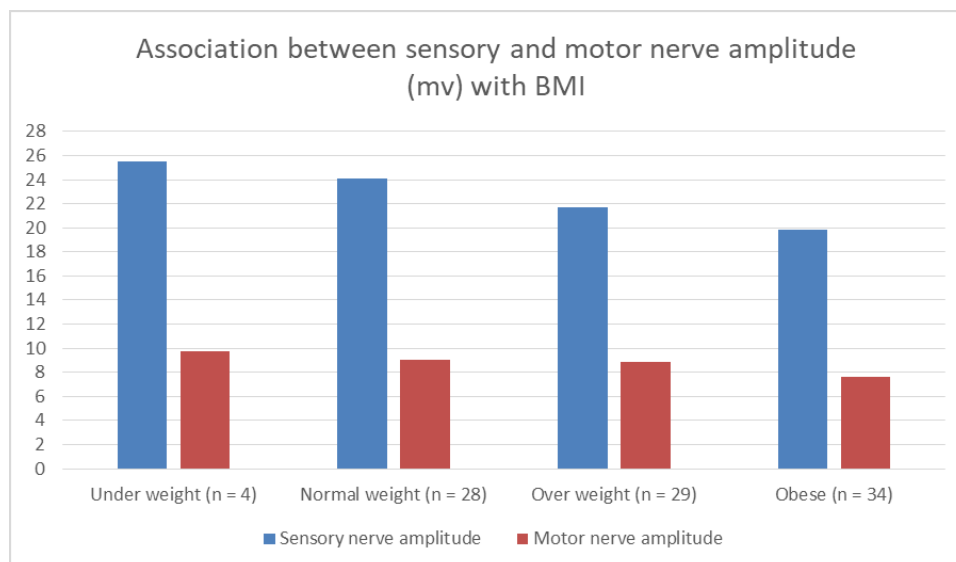


Figure 1: Association between sensory and motor nerve amplitudes and BMI.

3. Results

3.1. Sociodemographic factors

This study involved 95 individuals with T2DM with a male preponderance (54.7%). The mean age of the participants was 49 years – the minimum age being 30 and the maximum being 79 years. The participants' average height and weight were 169.73 ± 7.23 cm and 91.67 ± 3.17 kg, respectively. The mean BMI of the participants was 30.56 ± 3.18 , and the mean HbA1c was 8.04% (Table 1).

Our result showed a significant reduction in both sensory and motor nerve velocities with increased duration of diabetes of >5 years (P -value = 0.05; P -value = 0.003, respectively). Also, the results showed a significant reduction in both sensory nerve amplitude (P -value = 0.003) and motor nerve amplitude (P -value = 0.05) with increase in the duration of diabetes of >5 years, while there was no significant difference in the mean HbA1c between the two groups of duration <5 and >5 years (Table 2).

Our results showed a significant association between sensory and motor nerve amplitudes for underweight, normal weight, overweight, and obese BMI (P -value \leq 0.05; Figure 1).

4. Discussion

Diabetic peripheral neuropathy (DPN) is the most prevalent and troublesome consequence of diabetes. After ruling out every alternative etiology, DN, which can be localized or diffuse, is diagnosed when diabetic patients complain of symptoms and/or demonstrate evidence of peripheral nerve damage [9, 10]. Regardless of the clinical spectrum of diabetes, it has traditionally been linked with neurological manifestations. Indeed, there is a wealth of clinical information on the effects of diabetes on the nervous system, and it is evident

that peripheral nervous system, ocular manifestations, and central nervous system involvement are common and may have been published [11]. DPN clinical symptoms vary depending on the level of nerve injury. The use of NCS to corroborate DPN findings has been proposed [12]. The current study discovered a decrease (P -value = 0.05) in velocity and amplitude of both sensory and motor fibers in diabetic patients with >5 years of diabetes, as evaluated by NCS. These results were congruent with those of a Jordanian study by Khawaja *et al.*, which discovered that diabetes duration was the strongest predictor of DPN; the study compared individuals with T2DM for <5 years and those with diabetes duration of 5–11 years and reported a higher risk of DPN in the latter [13]. Furthermore, similar findings were seen in the study by Amelia *et al.* [14] in Indonesia, which indicated that the majority of diabetic patients had a duration of illness of >5 years. These findings demonstrated a link between diabetes duration and neuropathy. On the other hand, our results also demonstrated a significant (P -value \leq 0.05) association between BMI and sensory, motor amplitudes of both sensory and motor fibers in T2DM patients, which is in accordance with the study of Khawaja *et al.* [13]. Furthermore, Zhang *et al.* also found that distal motor latency was longer, sensory nerve CV was slower, and sensory nerve action potential and amplitude of compound muscle action potential were significantly lower in the diabetic groups compared to controls in the median, ulnar, posterior tibial, and common peroneal nerves [15]. Our results showed that a well-controlled DM and regular hemoglobin A1c (HbA1c) testing can promote the prevention of neurological complications; these findings were in accordance to the study of Mayeda *et al.*, who found that compared with HbA1c, continuous glucose monitoring may better capture risk of complications, including DPN [16]. Other study, with

similar findings as ours, revealed that CV and amplitude potential were found to be lower in HbA1C > 10% than in HbA1C 10% [17]. The findings of our study revealed strong inverse association with type II diabetes mellitus in line with findings of Partanen *et al.* who showed that all values for nerve CV in sensory and motor nerves were slower, and the sensory amplitude of the radial nerve and the motor amplitude of the median nerve were lower in the group with NIDDM [18]. In addition, Eman Abd El Aziz Galbat *et al.* found in their study that poor glycemic control was associated with a greater severity of DPN. Studies on nerve conduction demonstrated a highly significant positive link between motor and sensory CV and amplitude and a highly significant negative correlation between motor and sensory delay [19]. We emphasize that primary care physician have a duty to educate patients, identify problems early, and treat them appropriately to avoid more serious complications and improve patients' quality of life.

5. Limitations

Due to its cross-sectional design, it is impossible to study the timeline for the emergence of the observed differences, leaving us with only conjecture. Also, the sample size of the current study was relatively small, and it might be better if a larger sample is studied in the future.

6. Conclusion

Our findings explored the association of BMI and the duration of DM in DN patients with nerve conduction studies, which were both statistically significant ($P \leq 0.05$). These findings, taken together, provide an essential understanding of neurological manifestations in DN patients. Our findings may provide a better understanding

of the etiology of neurological involvement in patients with DN and may lead to hypothesis that chronic inflammatory response may cause axonal injury which can cause muscle weakness and neuropathic pain that affect DN patient's life considerably.

Acknowledgements

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Ethical Considerations

The study was approved by the Faculty of Medicine, International University of Africa, and the Federal Ministry of Health's ethical commission. Each subject provided written informed permission. Results of tested parameters were delivered to the participants.

Competing Interests

The authors declare that they have no conflicts of interest in relation to this study.

Availability of Data and Material

The data and materials used in this study are available upon request from the corresponding author.

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