Predictors of distal motor nerve conduction velocity in patients with diabetic neuropathy: a cross-sectional study

Predictores de la velocidad de conducción del nervio motor distal en pacientes con neuropatía diabética: un estudio transversal

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Received: 06-05-26
Accepted: 05-06-26

How to cite in APA

Ibrahim, A. M., Abdelrazik, R. K., Zedan, A. M. E., Alshimy, A. M., Abdelrahim, R. M., Moubarak, E., Nassar, R. N., Ammaar, A. H. F., & Shalash, K. A. (2026). Predictors of distal motor nerve conduction velocity in patients with diabetic neuropathy: a cross-sectional study. *Retos, 81*, 797-808. https://doi.org/10.47197/retos.v81.119409

Abstract

Background: Type 2 diabetes mellitus reduces nerve conduction velocity (NCV) leading to diabetic neuropathy (DN) with sensory, motor, and autonomic manifestations. Predictors of NCV need to be studied for better assessment, treatment, and prediction of the affected patients.
Objective: To determine predictors of distal motor NCV (tibial and peroneal nerves) in patients with DN.
Methods: Forty-four patients with DN from an outpatient clinic were enrolled. Besides demographic data, they were assessed for motor NCV of tibial and peroneal nerves and toe flexor muscle strength using dynamometer. Correlations using Pearson's correlation coefficient and linear regression model were used.
Results: Univariate analysis showed that strength and BMI are related to Peroneal motor NCV and age is related to tibial motor NCV at $p \leq 0.2$. Regression model showed that no significant predictor for peroneal or tibial motor NCV at $p < 0.05$.
Conclusion: Toes flexor isometric strength, age, and BMI are not independent predictors of distal motor NCV in patients with DN. Larger studies are needed to explore potential clinical screening proxies.

Keywords

T2DM, diabetic peripheral neuropathy, nerve conduction velocity, intrinsic foot muscle strength, cross-sectional study.

Resumen

Fundamento: La diabetes mellitus tipo 2 reduce la velocidad de conducción nerviosa (VNC) dando lugar a neuropatía diabética (ND) con manifestaciones sensoriales, motoras y autonómicas. Es necesario estudiar los predictores del VNC para una mejor evaluación, tratamiento y predicción de los pacientes afectados.
Objetivo: Determinar predictores del VNC motor distal (nervios tibial y peroneo) en pacientes con ND.
Métodos: Se inscribieron cuarenta y cuatro pacientes con ND de consulta externa. Además de los datos demográficos, se evaluaron para determinar el NCV motor de los nervios tibial y peroneo y la fuerza del músculo flexor del dedo del pie mediante dinamómetro. Se utilizaron correlaciones utilizando el coeficiente de correlación de Pearson y el modelo de regresión lineal.
Resultados: El análisis univariado mostró que la fuerza y el IMC están relacionados con el VNC motor peroneo y la edad está relacionada con el VNC motor tibial a $p \leq 0,2$. El modelo de regresión mostró que no hubo predictor significativo de VNC motora peronea o tibial a $p < 0,05$. Conclusión: La fuerza isométrica flexora de los dedos de los pies, la edad y el IMC no son predictores independientes del VNC motor distal en pacientes con ND. Se necesitan estudios más amplios para explorar posibles alternativas de detección clínica.

Palabras clave

DM2, neuropatía periférica diabética, velocidad de conducción nerviosa, fuerza muscular intrínseca del pie, estudio transversal.

Introduction

Diabetes mellitus (DM) is the most prevalent metabolic disorder that affects >9% of the population all over the world. In Egypt, diabetes mellitus (DM) is a significant public health concern (Abouzid et al., 2022), affecting >20% in one Governorate (AlSawahli et al., 2021). It has several types and etiologies. Among them, Type 2 DM (T2DM) is due to insulin resistance that impairs metabolism of carbohydrates, leading to chronic hyperglycemia. It is commonly associated with neuropathy (up to 45%) that impairs sensory, motor, and autonomic functions causing tingling/numbness/pain, weakness, sudomotor symptoms, impaired proprioception, balance and life quality, ulceration, amputation, morbidity, and mortality (Reeves et al., 2013; Richardson et a., 2014; Almurdhi et al., 2016; Yokoyama et al., 2020; Seiglie et al., 2020; Ponirakis et al., 2021; Bodman & Varacallo, 2023; Olimzhonovna, 2024). This leads to high healthcare expenditures (several hundred billion dollars worldwide). (IDF 2019).

Factors that affect (increase) the prevalence and severity of DPN include age (old) and duration of DM (long) (Tesfaye et al., 2005; Haji Naghi Tehrani, 2020; Sánchez-Pozos et al., 2021; Mohamed et al., 2020) and gender (male) (Haji Naghi Tehrani, 2020), weight (obese) (Andersen et al., 2018; Abouzid et al., 2022), and height (tall) (Feldman et al., 2019). In contrast, Kim et al. (2019) showed that DM duration did not affect amplitude of NCV.

Although muscle strength deficits (including ankle plantar/dorsiflexors and quadriceps) are common among patients with PN (Darwesh et al., 2017; Younesian et al., 2020), there is controversy among literatures about its relation to severity of PN (Van Schie et al., 2004; Andersen et al., 2004; Andreassen et al., 2009; Liu et al., 2015; Almurdhi et al., 2016).

Nerve conduction velocity (NCV) study is a gold standard in diagnosing and predicting DN (Dyck et al., 2010; Tesfaye et al., 2010); patients should have one or more abnormal sign in NCV combined with sign/symptom of neuropathy to have DN (Tesfaye et al., 2010). However, it is time-consuming, operator-dependent, uncomfortable for the patient, and costly (Weisman et al., 2013; Callaghan et al., 2013).

However, some studies addressed factors affecting NCV in patients with DN, they were limited by assessing only sensory NCV (Yokomaya et al., 2020) or assessed effect of muscle strength only (Andersen et al., 1998).

Although DPN is known to cause proximal lower-limb weakness, intrinsic foot muscles, especially the toe flexors, are frequently weakened early in the course of the disease, which increases the risk of falls, impairs balance, and alters gait mechanics. Distal motor function can be quickly, affordably, and practically measured with the toe flexor dynamometer. Despite its potential, little is known about the connection between intrinsic foot muscle strength and motor NCV; previous research has mostly concentrated on proximal muscle groups or sensory NCV. By elucidating this relationship, accessible screening methods for early motor involvement in DPN may be developed, thereby lowering the need for resource-intensive electrophysiological testing.

Studying factors that may be strongly related to NCV in patients with DN may reduce dependency on NCV and hence reduce its drawbacks/limitations. In addition, it may help early diagnosis of this condition to prevent the development and progression of diabetic neuropathy (Li et al., 2021). Furthermore, it helps implementing personalized treatment strategies (including endurance and sensorimotor training) to enhance health and reduce costs (Battesha et al., 2020; Streckmann et al., 2023). Therefore, this study aimed to determine the predictors of distal motor NCV in patients with T2DM.

Method

Design and setting

This cross-sectional study was carried out at the out-patient clinic of faculty of physical therapy-Benha University from October 2024 to February 2025. Patients were referred by neurologists from surrounding hospitals in Cairo and Qaluybia. Principles of Declaration of Helsinki were followed in this study which got its ethical approval from the ethics committee of faculty of physical therapy-AlSalam University [NO.SREC.PT.SUE(11)625]. The informed consent was signed by each patient prior to participation. In order to detect moderate-large effect size (0.4 on average) based on study of Mao et al., 2018 - for the effect of age on diabetic polyneuropathy with 80% power and 5% significance level - G*power software (version 3.1.9.7, Franz Faul, Universitat Kiel, Germany) suggests that 44 participants needed using correlation: point biserial model.

Sample size was calculated using G*Power v3.1.9.7 (Franz Faul, Universität Kiel, Germany). Based on Mao et al. (2018), who reported a moderate-to-large correlation (r ≈ 0.40) between age and diabetic polyneuropathy, we set the expected effect size at r = 0.40. With α = 0.05 (two-tailed) and 80% power, the required sample size for a bivariate normal model is 44 participants. This calculation ensures adequate power to detect clinically meaningful associations while minimizing Type II error.

Patients

Inclusion required a confirmed diagnosis of DPN based on the Toronto Consensus criteria, defined as the presence of neuropathic symptoms (e.g., tingling, numbness, pain) or clinical signs combined with at least one abnormal motor or sensory nerve conduction parameter (Tesfaye et al., 2010). Patients with chronic (>10 years), controlled T2DM and aged > 18 years old with abnormal NCV tests were included in this study. While, patients with other types of DM, severe physical disease; kidney/liver failure; foot ulceration/amputation; and other diseases affecting nerves (e.g. radiculopathy and stroke) were excluded from the study (Alshimy et al., 2023).

Procedures

Baseline data such as gender, age, weight, height, body mass index, and duration of disease were taken from and/or measured for each patient.

Next, isometric toes flexor muscle strength was measured using toe strength dynamometer (American Weigh Scales, Georgia, USA). It showed moderate validity and reliability in measuring toes flexor muscle strength. Firstly, assessor instructed the patients how to do the movement. The assessor asked the patients to straighten their toes and slid the plastic card under the toe(s) to a level before the metatarsal head(s), Then asked them again to rest their toes on the card, while the assessor stabilized the foot top. Patients were asked to push on the card firmly keeping it in place "as hard as they could" for 3 seconds (three counts), while the assessor pulled the handle of the device, increasing the force of the pull slowly so that the card slid out on "3", keeping the device on the ground with the line of pull in the sagittal plane. All assessments were conducted on the same surface. Patients were in a hook-lying position, feet flat on the ground and arms across the chest (Xu et al., 2023).

Then, NCV was recorded under standard surface stimulation using a device (Nihon Kohen, Japan, MEB-9200/9300). During the measurement, the electrode was coated with conductive glue and fixed with tape. NCV was measured on both sides of the limbs. The tests were conducted at room temperature (20–25◦ C), with a stimulation frequency of 1 Hz, a stimulation pulse width of 0.1 ms, and a sensitivity of 5 mV/lattice. Tibial and common peroneal nerves were tested. Tibial motor nerve conduction velocity studies were performed by placing an active electrode on the abductor hallucis brevis muscle. Stimulating electrodes were placed in two locations (ankle and popliteal fossa), with an earth electrode placed between the stimulator cathode and the active pickup electrode, and a reference electrode was placed over the big toe's metatarsalphalangeal joint. (Li et al., 2021; Alshimy et al., 2023). NCV studies are valid (Weisman et al., 2013) and reliable (Litchy et al., 2014) in identification and prediction of DN.

Statistical analysis:

Descriptive statistics were shown as mean±standard deviation and counts for scale and categorical data, respectively. Shapiro-Wilk test was used to verify the normal distribution of the data and showed that almost all data were normally distributed ($p>0.05$). Pearson's correlation coefficient was used to assess univariate correlations between studied variables. Significantly correlated variables (at $p\leq0.2$) were entered a linear regression model. This higher threshold was used so as not to lose significant predictors prematurely.

Results

Baseline characteristics

Descriptive statistics for demographic data of all patients were shown in Table (1).

Table 1. Descriptive statistics for demographic data for all patients.

Variable	Mean±SD
Age (year)	51.7±4.7
Weight (kg)	67.8±2.7
Height (cm)	171±2.7
BMI (kg/m²)	23.1±1.03
Gender (M/F)	22/22
Toes flexor isometric strength (lbs)	67.91±4
Tibial motor NCV (m/s)	38.68±3.1
Peroneal motor NCV (m/s)	36.93±4.28

SD: Standard deviation, BMI: Body mass index, M: Male, F: Female, NCV: Nerve conduction velocity, lbs: pounds, m/s: meter per second.

Univariate analysis

Pearson correlation coefficient showed that only strength and BMI were correlated with Peroneal motor NCV and age was correlated with Tibial motor NCV at $p \leq 0.2$.

Table 2. Univariate analysis (Pearson correlation coefficients) for the relation between strength, age, BMI and distal motor NCV

	Correlation	Peroneal motor NCV (m/s)	Tibial motor NCV (m/s)
Toes flexor isometric strength (lbs)	r	.18	.058
	p	.12	.4
BMI (kg/m²)	r	-.205	-.064
	p	.183	.34
Age (years)	r	-.046	.129
	p	.768	.2

BMI: Body mass index, r: Pearson correlation coefficient value, p: Probability value.

Uni- and multi-variate regression analysis

Regression analysis did not show any significant predictor for peroneal or tibial motor NCV at $p<0.05$. However, the models were insignificant (ANOVA F values were 2 and 0.7, respectively, $p<0.05$), the models showed that toe flexor strength and BMI explained 9% of change in peroneal motor NCV and age explained only 1.7% of change in tibial motor NCV. (Tables 3-5) (Figures 1-3).

Table 3. Model summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
Peroneal motor NCV	.299[a]	.090	.045	4.15016
Tibial motor NCV	.129[a]	.017	-.007	3.14403

Table 4. ANOVA results

Model	Sum of Squares	df	Mean Square	F	p
Peroneal motor NCV	69.554	2	34.777	2.019	.15
Tibial motor NCV	7.078	1	7.078	.716	.40

Table 5. Coefficients

Model		Unstandardized Coefficients		Standardized Coefficients	t	p	95.0% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
Peroneal motor NCV	Constant	45.24	17.09		2.65	.01	10.73	79.76
	Toe flexor strength	.21	.16	.20	1.35	.19	-.11	.53
	BMI	-.99	.62	-.24	-1.60	.12	-2.23	.26
Tibial motor NCV	Constant	34.22	5.3		6.46	<.01	23.53	44.91
	Age	.09	.10	.13	.85	.40	-.12	.29

NCV: Nerve conduction velocity, BMI: Body mass index, p: Probability value.

Figure 1. Scatter plot for relation between toe flexor strength (pounds) and peroneal motor NCV (m/s).

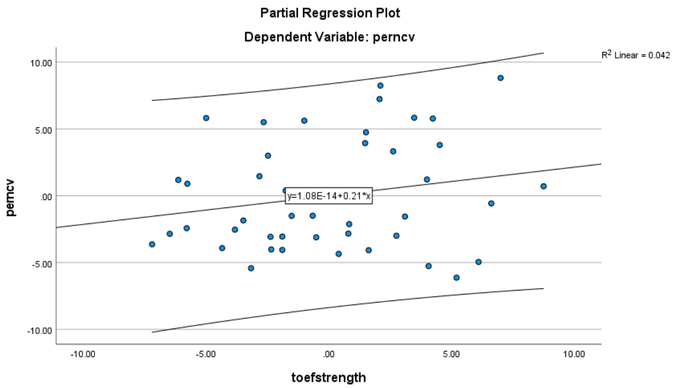


Figure 2. Scatter plot for relation between BMI and peroneal motor NCV (m/s).

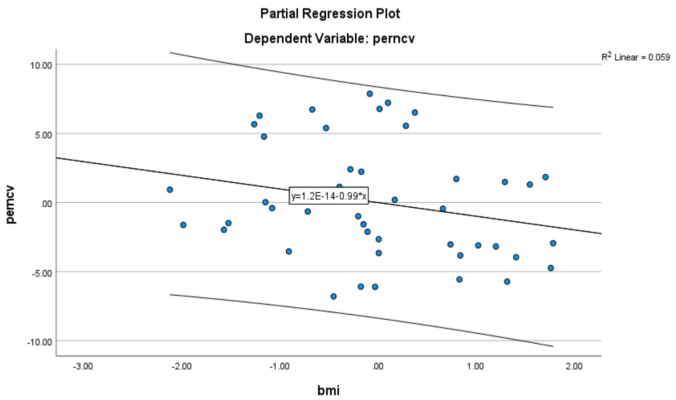
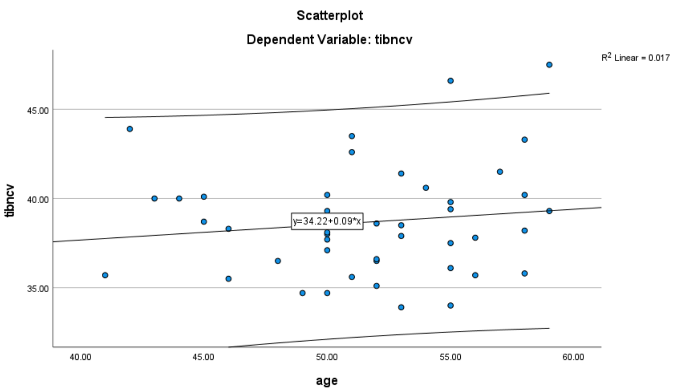


Figure 3. Scatter plot for relation between age and Tibial motor NCV (m/s).



Discussion

In patients with diabetic peripheral neuropathy (DPN), this cross-sectional study investigated possible relationships between motor nerve conduction velocity (NCV) of the tibial and peroneal nerves and toe flexor isometric strength, age, and body mass index (BMI). In contrast to our original prediction, there were no statistically significant correlations found in the current cohort between these factors and motor NCV (r range: 0.05–0.30; p > 0.05). These results show that in patients with established DPN, individual measures of distal intrinsic foot muscle strength, age, or BMI do not show a linear connection with motor NCV within the methodological limitations of this investigation.

Toe Flexor Strength and Motor NCV: The lack of a substantial correlation between motor NCV and toe flexor strength calls for pathophysiological and biomechanical analysis. Conduction slowing is usually a sign of demyelination, axonal loss, or compromised nodal ion channel function. Motor NCV mainly represents the structural and functional integrity of massive, myelinated alpha motor axons. (Yang et al., 2022). In contrast, Peripheral nerve conduction, central motor drive, neuromuscular junction transmission, muscle fiber composition, cross-sectional area, and compensatory motor unit recruitment are all factors that influence voluntary muscle strength. (Laginestra et al., 2022 ; Abd elrazik et al., 2021). In early to moderate stages of DPN, neural plasticity and collateral sprouting may preserve functional strength despite measurable electrophysiological slowing, effectively decoupling NCV from clinical force generation (Gracia et al., 2025).

Previous literature presents mixed evidence regarding strength-NCV relationships. Studies supporting a positive association have predominantly examined proximal muscle groups rather than intrinsic foot muscles. For instance, Andersen et al. (1998) demonstrated moderate-to-strong inverse correlations between ankle/knee extensor strength and electromyographic measures of reinnervation (macro-MUP amplitude: r = −0.63, p < 0.001) in patients with diabetic neuropathy. Similarly, Andersen et al. (2004) reported significant inverse correlations between neuropathy severity scores and ankle dorsiflexor (r = −0.66, p < 0.001) and plantar flexor strength (r = −0.51, p < 0.001). However, these studies assessed proximal muscles and used electromyographic rather than motor NCV outcomes.

More recently, Yoshida et al. (2023) demonstrated that diabetic polyneuropathy (confirmed by nerve conduction studies) was independently associated with reduced toe grip strength after adjusting for age, sex, and BMI. Conversely, several investigations align with our null findings. Van Schie et al. (2004) found no significant correlation between foot muscle strength and neuropathy severity scores in Caucasian men with diabetes. Almurdhi et al. (2016) reported that while lower-limb muscle volume was reduced in patients with DPN, the relationship with nerve conduction parameters was inconsistent and depended on vitamin D status and intramuscular fat infiltration—highlighting the potential mediating role of unmeasured confounders.

There are a number of reasons why our results differ from earlier favorable reports: (1) Muscle group specificity: Proximal muscles, which may show distinct susceptibility patterns from distal intrinsic foot muscles, were the focus of earlier research showing strength-NCV relationships; (2) Variations in outcome variables: (3) Measurement methodology: Toe flexor strength was evaluated using a novel dynamometer with moderate validity, whereas previous studies used isokinetic dynamometry or manual muscle testing; (4) Disease stage: Our cohort included patients with established DPN; in advanced neuropathy, the relationship between strength and NCV may plateau due to compensatory mechanisms. Many studies used sensory NCV, amplitude measures, or composite neuropathy scores rather than motor NCV specifically.

Age and Motor NCV: One of the most investigated and documented predictor factors for the development of DN is age (Bansal et al., 2014). The current study did not support this finding as there was no significant correlation between age and NCV. This may be due to small sample and including specific age category.

In same line with our study Mao et al. (2019) identified age as an independent risk factor for diabetic peripheral neuropathy in a large Chinese cohort with older patients exhibiting significantly slower sensory and motor NCV. Haji Naghi Tehrani (2020) reported significant negative correlations between age and tibial/peroneal motor NCV in a cross-sectional study of 120 diabetic patients. Kim et al. (2019) found that diabetes duration, but not age, significantly predicted nerve conduction abnormalities in lower limbs of diabetic patients, suggesting that metabolic exposure may outweigh chronological age in determining neuropathic severity.

Our null finding regarding age may reflect: (1) Restricted age range: Our sample had a narrow age distribution (mean = 51.7 ± 4.7 years), limiting variability necessary to detect age-related gradients; (2) Cross-sectional design: Age-related NCV decline is gradual and cumulative; cross-sectional snapshots may fail to capture longitudinal trajectories; (3) Confounding by diabetes duration: Age and diabetes duration are often collinear; without adjusting for exact disease duration, the independent effect of age may be obscured; (4) Population-specific factors: Genetic, environmental, or healthcare access differences may modify age-NCV relationships.

BMI and Motor NCV: Obesity is increasingly recognized as an independent metabolic risk factor for neuropathy. Andersen et al. (2018), in a 13-year prospective cohort, identified elevated BMI as a predictor of incident DPN independent of glycemic control. Awang et al. (2006), reported that higher BMI was associated with reduced sural nerve conduction velocity Conversely, Romero et al. (1999), align with our null findings. and failed to establish a link between obesity and autonomic neuropathy The absence of a BMI-NCV correlation in our study may be explained by: (1) Limited BMI variability: Our cohort had a narrow BMI range (mean = 23.1 ± 1.03 kg/m^2), predominantly within the normal-to-overweight category; (2) BMI as an imperfect proxy: BMI does not distinguish between visceral adiposity, subcutaneous fat, or lean mass—factors with divergent metabolic implications; (3) Mediation by glycemic control: The relationship between adiposity and neuropathy may be mediated by insulin resistance and chronic hyperglycemia; without HbA1c data, we could not test this mediation pathway; (4) Sample size and power: As noted below, the study was powered to detect moderate-to-large effects ($r \geq 0.40$); small but clinically meaningful associations ($r \approx 0.20–0.30$) may have been missed.

It is critical to interpret these negative findings within their statistical context. The a priori sample size calculation targeted a moderate-to-large effect size ($r \geq 0.40$) with 80% power. Consequently, the study was underpowered to detect small or weak associations ($r < 0.30$), increasing the likelihood of a Type II error. The absence of statistical significance should therefore not be equated with clinical irrelevance; rather, it highlights the need for larger, adequately powered cohorts to explore potential nonlinear or threshold-dependent relationships.

Additionally, the cross-sectional design precludes causal inference and limits our ability to determine whether alterations in strength, age-related changes, or adiposity precede, coincide with, or follow motor NCV decline. Longitudinal studies are needed to clarify temporal relationships and identify potential causal pathways.

Clinical Implications and Future Directions

From a clinical standpoint, these results suggest that toe flexor isometric strength, age, and BMI, when evaluated in isolation, cannot substitute for electrophysiological testing in the assessment of motor nerve involvement in DPN. However, this does not diminish the potential utility of strength assessment as a component of a multimodal, low-cost screening protocol. In resource-limited settings where NCV equipment is unavailable or prohibitively expensive, combining distal strength measures with validated clinical tools (e.g., Michigan Neuropathy Screening Instrument, 10-g monofilament testing, vibration perception threshold) may improve early identification of patients warranting formal neurophysiological evaluation (Weisman et al., 2013; Li et al., 2021).

Future research should prioritize: (1) Larger, prospective cohorts with comprehensive metabolic profiling (HbA1c, lipid panels); (2) Incorporation of detailed neuropathy staging and validated severity scales; (3) Advanced statistical modeling (e.g., multivariate regression, structural equation modeling, machine learning) to disentangle direct, indirect, and interactive effects; (4) Examination of both motor and sensory nerve parameters to capture the full spectrum of peripheral nerve involvement in DPN.

Study Limitations

The modest sample size ($n = 44$) restricts generalizability and increases vulnerability to Type II error. Key clinical confounders, including HbA1c, exact diabetes duration, pharmacological management, physical activity levels, and standardized neuropathy severity scores, were not systematically recorded, limiting our ability to adjust for metabolic and lifestyle influences. The focus on two motor nerves (tibial and peroneal) may not capture the full spectrum of peripheral nerve involvement in DPN, which often affects sensory fibers earlier and more severely. Finally, the cross-sectional design cannot establish temporal or causal relationships between the variables examined.

Conclusions

Muscle strength, age, and BMI are not independent predictors for NCV in patients with DN. More predictors need to be studied in future large studies.

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