ORIGINAL ARTICLE

Physical Activity and BMI in Relation to Diabetic Neuropathy: Which Factor Plays a Greater Role?

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ABSTRACT

Introduction: The rising prevalence of obesity indices, such as body mass index (BMI), alongside persistently low levels of physical activity, contributes significantly to the development of diabetic microvascular complications, including somatic and autonomic neuropathy. This study aims to analyse the relationship between body mass index (BMI) and physical activity with the incidence of diabetic neuropathy in patients with type 2 diabetes mellitus. **Method**: This study employed a cross-sectional design in 55 patients diagnosed with type 2 diabetes mellitus in 2024 who met the inclusion and exclusion criteria and were selected using consecutive random sampling. The research instruments used were weight and height scales, the Global Physical Activity Questionnaire (GPAQ), and the Michigan Neuropathy Screening Instrument (MNSI). **Results**: The neuropathy-positive group had higher BMI (27.78 ± 4.708 vs. 23.70 ± 3.444, $p < 0.05$) and lower physical activity (905.05 ± 911.871 vs. 2,537.11 ± 2,126.107, $p < 0.05$). BMI status ($r=0.461$, $p<0.05$) and physical activity level ($r=0.450$, $p<0.05$) had a positive and moderate association with the incidence of diabetic neuropathy. Logistic regression results showed that physical activity (Wald = 8.167) and BMI (Wald = 5.530) had a more significant influence than BMI on the incidence of diabetic neuropathy. **Conclusion**: A significant relationship was identified between Body Mass Index (BMI) and physical activity with the incidence of diabetic neuropathy in type 2 diabetes mellitus patients.
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Keywords: Body Mass Index (BMI), Diabetic Neuropathy, Physical Activity, Type 2 Diabetes Mellitus, Risk Factors

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INTRODUCTION

As a metabolic disease, diabetes is characterised by elevated blood glucose levels and remains a growing global health challenge (1). Behavioural, socio-demographic and lifestyle factors, as well as clinical or psychological conditions, can influence the incidence of diabetes (2). Diabetic neuropathy is among the most common complications arising from elevated blood glucose and lipid levels that damage peripheral nerves. It is widely acknowledged that the most prevalent type of diabetic neuropathy is diabetic peripheral neuropathy (3).

A study of global epidemiological data, as reported in the Global Burden of Disease (GBD) study, showed a global prevalence of type 2 diabetes of 462 million cases in 2017. This finding was approximately 6.28% of the worldwide population, with a further breakdown of 4.4% of people aged 15-49, 15% of those aged 50-69, and 22% of those aged >70. Projections for the global prevalence of type 2 diabetes indicate an increase to 7079 cases per 100,000 by 2030 (4). The national prevalence of diabetes mellitus increased from 10.9% in 2018 to 11.7% in 2023, with a higher incidence reported among individuals aged over 35, particularly within the 45-64 age group (5). Globally, 22% to 46.5% of diabetic patients have diabetic neuropathy (6).

A prospective study reported that rising levels of obesity indices, such as body mass index (BMI), are significantly associated with an increased risk of both macrovascular and microvascular complications among individuals with diabetes (7). In addition, systematic analyses of dietary and physical activity interventions furnished further evidence that corroborated the effectiveness of these interventions in reducing the severity of somatic and autonomic neuropathies. However, the findings from these studies should be analysed with caution, as most were based on small sample sizes and non-randomised designs, and not all outcome measures were reliable and clinically relevant (8). The importance of the comprehensive management of modifiable risk factors, along with the implementation of lifestyle modifications and system-level measures, including

systematic screening, educational programs, and increased awareness, cannot be overstated when it comes to enhancing quality of life (9).

Among the various modifiable risk factors, body mass index (BMI) and physical activity have received particular attention due to their significant influence on metabolic control, vascular health, and nerve function. Elevated BMI has been linked to increased oxidative stress and microvascular impairment, which contribute to nerve damage (10,11). Conversely, physical activity has demonstrated benefits in improving glycaemic control, reducing inflammation, and enhancing peripheral nerve perfusion (12–14). These characteristics render both variables highly relevant for investigation in relation to diabetic neuropathy, particularly in low-resource settings where preventive strategies must prioritise modifiable and cost-effective targets.

Although diabetic neuropathy is a prevalent and serious complication of type 2 diabetes mellitus, existing evidence regarding the role of modifiable risk factors such as body mass index and physical activity in its development remains inconsistent and inconclusive (15,16). This gap in understanding is particularly relevant in low- and middle-income countries, where lifestyle patterns and access to preventive healthcare may differ markedly from those in high-income countries (17). This study aims to analyse the relationship between body mass index (BMI) and physical activity with diabetic neuropathy in individuals with type 2 diabetes. Identifying modifiable risk factors aims to support the development of targeted prevention and intervention strategies to reduce the burden of diabetic neuropathy.

MATERIALS AND METHODS

The present study employed a cross-sectional design involving patients diagnosed with type 2 diabetes mellitus. Ethical approval was obtained from Siti Khodijah Hospital's Ethics Committee (Number: 21/KET-KEPK/8-2024). Participants were selected through simple random sampling. As the study involved human subjects, informed consent was obtained before data collection. The inclusion criteria comprised patients aged 19 to 70 who received either single or combination anti-diabetic therapy. Exclusion criteria included absence during data collection, gestational diabetes, sensory impairments, a history of stroke or Morbus Hansen, and the use of neurotoxic medications (Topiramate, Rufinamide, Oxcarbazepine, Gabapentin, Vigabatrin).

The sample size was calculated using the ordinal-nominal correlation formula, resulting in a final sample of 55 participants. The primary objective was to assess the characteristics of respondents, including age, gender, duration of diabetes mellitus, genetic history, history of hypertension, smoking status, and measurement of physical activity, as well as the weight and height of respondents. The secondary objective was to analyse the relationship between Body Mass Index (BMI) and physical activity in patients with type 2 diabetes mellitus and to determine various patient characteristics.

The use of the Global Physical Activity Questionnaire (GPAQ) instrument in this study was based on its established validity and reliability in assessing physical activity among adult populations across various countries, including individuals with chronic conditions such as diabetes mellitus. The GPAQ has demonstrated adequate internal consistency and significant correlations with objective measures of physical activity in individuals with metabolic disorders (18). Meanwhile, the Michigan Neuropathy Screening Instrument (MNSI) was chosen for assessing diabetic neuropathy due to its high sensitivity, specificity, and ease of application in clinical and research settings (19).

The overall research procedure began with ethical approval, followed by participant recruitment and screening based on inclusion and exclusion criteria. Eligible participants were selected through simple random sampling. Diabetic neuropathy was assessed using the MNSI, while data on body mass index (BMI) and physical activity were collected using BMI measurement and the Global Physical Activity Questionnaire (GPAQ). The collected data were subsequently organised and analysed to examine the relationship between BMI, physical activity, and diabetic neuropathy. The research flow is illustrated in Figure 1[IB1.1][IB1.2]. (See Figure 1)

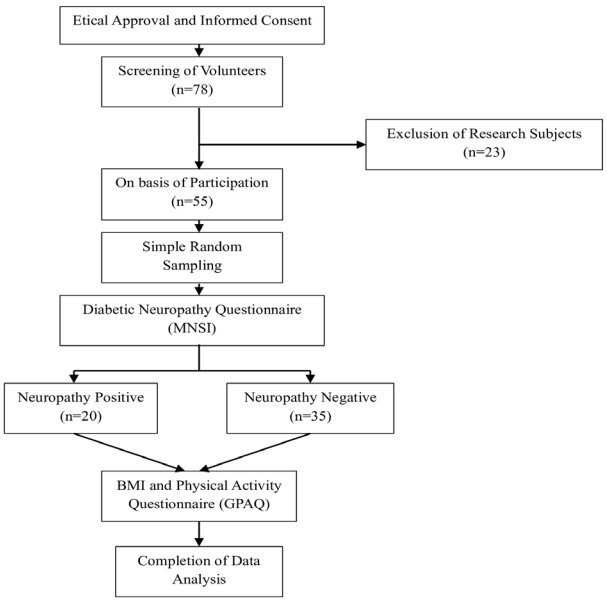

Figure 1: Flow chart of the research design.

Physical Activity Questionnaire

The Global Physical Activity Questionnaire (GPAQ) is a 16-item survey designed to assess various domains of physical activity, including work-related, transportation-related, recreational, and sedentary activities, over the preceding week. The calculated data were converted into MET minutes per week, as outlined in the WHO GPAQ version 2 analysis guide. Specifically, heavy activity data were multiplied by 8 METs, and moderate activity data were multiplied by 4 METs. The results of these calculations were then categorised as follows: heavy (MET ≥ 3000), moderate (3000 > MET ≥ 600), and light (600 < MET) (20,21). Previous studies have confirmed that the Indonesian version of the GPAQ questionnaire is both valid and reliable (22).

Diabetic Neuropathy Questionnaire

The Michigan Neuropathy Screening Instrument (MNSI) was divided into two parts: the first was a patient questionnaire consisting of 15 questions, and the second was an examination by medical personnel that included inspection (checking for deformity, dry skin, callus, infection, fissures, and ulcers), Achilles tendon reflex, and vibration sensation (23). Patients were diagnosed with neuropathy if their MNSI questionnaire score was ≥ 7 and their examination score was ≥ 2.5. Conversely, patients were considered to have a negative diagnosis for diabetic neuropathy if their MNSI questionnaire score was < 7 and their examination score was < 2.5 (24). As evidenced by existing literature, the Indonesian version of the MNSI questionnaire has a documented sensitivity of 95.0% and specificity of 90.5% (25).

BMI Measurement

The researcher conducted BMI measurements on the respondents, adhering to standard procedures for measuring weight and height (Sojikyo HS 200; Japan). The researcher took measurements at least three times to obtain an average value and then calculated the respondents' Body Mass Index (BMI) and classified them based on their weight status. The Body Mass Index category for the Asia-Pacific region was proposed and developed by the World Health Organisation, specifically by the Regional Office for the Western Pacific (WPRO), in 2000 and is used as a standard reference to categorise BMI for populations in the Asia-Pacific region (26). (See Table I)

Table I: Asia-Pacific BMI Classification

Classification	Asia-Pacific
Underweight	< 18.5
Normal Weight	18.5 – 22.9
Overweight	23 – 24.9
Obesity (Class I)	25 – 29.9
Obesity (Class II)	≥ 30

Statistical Analysis

The data were analysed using SPSS version 25.0. Frequencies and percentages were used to present categorical data. The Mann-Whitney U test was used to compare BMI and physical activity levels between individuals with and without diabetic neuropathy, as the data were not normally distributed. The chi-square test was used to analyse the association between BMI, physical activity, and diabetic neuropathy. The contingency coefficient was used to measure the variables' association strength. A regression analysis was then conducted to ascertain BMI and the influence of activity on diabetic neuropathy risk. A significance level of $p<0.05$ was predetermined for all analyses.

RESULTS

Socio-Demographic and Clinical Characteristics of Type 2 Diabetes Mellitus Patients

The majority of respondents were female (85.5%), in the age range of 61-70 (43.7%), had a normal body mass index (43.6%), had light physical activity level (43.6%), had been diagnosed with diabetes mellitus for > 5 years (60%), had a family history of diabetes (63.8%), had no history of hypertension (50.9%), had no smoking habit (98.2%), and had no diabetic neuropathy (63.6%) (See Table II).

Table II: Socio-Demographic and Clinical Characteristics of Type 2 Diabetes Mellitus Patients

Characteristic		Total (%) (N=55)
Age	40-50 years	9 (16.4%)
	51-60 years	22 (40%)
	61-70 years	24 (43.7%)
Gender	Male	8 (14.5%)
	Women	47 (85.5%)
Duration of Diabetes	> 5 years	33 (60%)
	≤ 5 years	22 (40%)
Family History of Diabetes	Yes	35 (63.8%)
	No	20 (36.4%)
History of Hypertension	Yes	27 (49.1%)
	No	28 (50.9%)
Smoking	Yes	1 (1.8%)
	No	54 (98.2%)
BMI	Normal	24 (43.6%)
	Overweight	7 (12.7%)
	Obesity I	16 (29.1%)
	Obesity II	8 (14.5%)
Physical Activity	Light	24 (43.6%)
	Moderate	15 (27.3%)
	Vigorous	16 (29.1%)
Diabetic Neuropathy	(+)	20 (36.4%)
	(-)	35 (63.6%)

Socio-Demographic and Clinical Characteristics of Patients Based on the Incidence of Diabetic Neuropathy

The results showed that 20% of patients aged 61–70 years had diabetic neuropathy, with a predominance of female gender (32.7%). It was found that a duration of diabetes exceeding five years was associated with a 23.6% incidence. Furthermore, a family history of diabetes was found to be a contributing factor in 21.9% of cases. In contrast, no significant difference was found between patients with and without a previous history of hypertension, with an incidence of 18.2%. It was noteworthy that all patients who experienced diabetic neuropathy in this study were non-smokers, accounting for 36.4% of the total participants. Furthermore, an 18.2% incidence of diabetic neuropathy was identified in patients with class I obesity. In contrast, a 27.25% incidence of diabetic neuropathy was identified in patients with mild physical activity levels in this study (See Table III).

Table III: Socio-Demographic and Clinical Characteristics of Patients Based on the Incidence of Diabetic Neuropathy

Characteristic		Diabetic Neuropathy	
		+	-
Age	40-50 years	4 (7.3%)	5 (9.1%)
	51-60 years	5 (9.1%)	17 (30.9%)
	61-70 years	11 (20%)	13 (23.7%)
Gender	Male	2 (3.6%)	6 (10.9%)
	Female	18 (32.7%)	29 (52.8%)
Duration of Diabetes	> 5 years	13 (23.6%)	20 (36.4%)
	≤ 5 years	7 (12.7%)	15 (27.3%)
Family History of Diabetes	Yes	12 (21.9%)	23 (42%)
	No	8 (14.6%)	12 (21.8%)
History of Hypertension	Yes	10 (18.2%)	17 (32.9%)
	No	10 (18.2%)	18 (32.7%)
Smoking	Yes	0	1 (1.8%)
	No	20 (36.4%)	34 (61.9%)
BMI	Normal	2 (3.6%)	22 (40%)
	Overweight	4 (7.3%)	3 (5.4%)
	Obesity I	10 (18.2%)	6 (10.9%)
	Obesity II	4 (7.25%)	4 (7.25%)
Physical Acivity	Light	15 (27.25%)	9 (16.35%)
	Moderate	4 (7.3%)	11 (20%)
	Vigorous	1 (1.8%)	15 (27.3%)

The Comparison of Body Mass Index [IB2.1][IB2.2] and Physical Activity Levels Between Patients With and Without Diabetic Neuropathy in Type 2 Diabetes Mellitus Patients

Figure 2a presents a comparison of Body Mass Index (BMI) between patients with and without diabetic neuropathy. The neuropathy-positive group demonstrated a higher mean BMI of 27.78 (±4.708), in contrast to a lower mean of 23.70 (±3.444) in the neuropathy-negative group. This difference was statistically significant ($p = 0.000$), as indicated by the Mann–Whitney U test, suggesting that increased BMI is associated with the presence of diabetic neuropathy. (See Figure 2a)

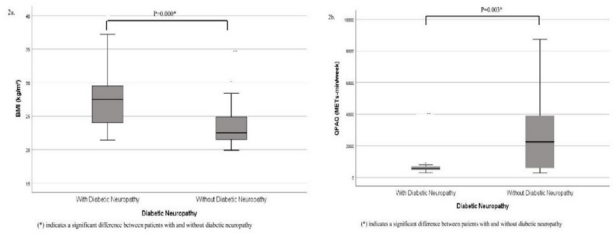

Figure 2: (a) The comparison of Body Mass Index (BMI) values between patients with and without diabetic neuropathy. (b) The comparision of total physical activity levels (measured in MET- minutes per week) between patients with and without diabetic neuropathy.

Figure 2b displays the comparison of total physical activity levels between patients with and without diabetic neuropathy. The neuropathy-positive group exhibited a markedly lower mean physical activity level of 905.05 (±911.871), whereas the neuropathy-negative group recorded a higher mean of 2,537.11 (±2,126.107). This difference was also statistically significant (p = 0.003), indicating that lower levels of physical activity are associated with the presence of diabetic neuropathy. (See Figure 2b)

The Relationship Between BMI and Physical Activity with the Incidence of Diabetic Neuropathy in Type 2 Diabetes Mellitus Patients

The results indicated that the body mass index (BMI) of patients with diabetic neuropathy predominantly fell within the Obesity I category. The significance value of 0.002 ($p < 0.05$) and the correlation coefficient of 0.461 obtained in this study signified a moderate correlation between BMI and the prevalence of diabetic neuropathy, suggesting that an elevated BMI was associated with an increased risk of diabetic neuropathy. The physical activity of patients with diabetic neuropathy was predominantly in the category of light physical activity, as indicated by a significance value of 0.001 ($p < 0.05$) and a correlation coefficient of 0.450. This result suggested a moderate correlation between physical activity and the incidence of diabetic neuropathy, indicating that lower physical activity would lead to an increasing risk of diabetic neuropathy. (See Table IV)

Table IV: The Relationship Between BMI and Physical Activity with the Incidence of Diabetic Neuropathy in Type 2 Diabetes Mellitus Patients

	Classification	Diabetic Neuropathy		Total	Contingency Coefficient Test
		+	-		
BMI	Normal	2 (3.6%)	22(40%)	24 (43.6%)	p = 0.002 r = 0.461
	Overweight	4 (7.3%)	3(5.4%)	7 (12.7%)	
	Obesity I	10 (18.2%)	6(10.9%)	16 (29.1%)	
	Obesity II	4 (7.25%)	4(7.25%)	8 (14.5%)	
Physical Activity	Light	15 (27.2%)	9(16.4%)	24 (43.6%)	p = 0.001 r = 0.450
	Moderate	4 (7.3%)	11(20%)	15 (27.3%)	
	Vigorous	1 (1.8%)	15(27.3%)	16 (29.1%)	

Logistic Regression Analysis to Determine the Dominant Factors Between BMI and Physical Activity on the Incidence of Diabetic Neuropathy in Type 2 Diabetes Mellitus Patients

The Wald test values for physical activity and BMI were 8.167 ($p < 0.05$) and 5.530 ($p < 0.05$), respectively. These values indicated that physical activity exerted a more significant influence than BMI on the development of diabetic neuropathy. (See Table V)

Table V: Logistic Regression Analysis to Determine the Dominant Factors Between BMI and Physical Activity on the Incidence of Diabetic Neuropathy in Type 2 Diabetes Mellitus Patients

Variable	Wald	p-*value*
Pysical Activity	8.167	0.001
Body Mass Indeks (BMI)	5.530	0.002

DISCUSSION

A substantial proportion 43.7% of respondents were aged between 61 and 70 years, with a recorded incidence of diabetic neuropathy of 20%. Advancing age is associated with neural degeneration affecting both large and small fibres, thereby increasing the risk of neuropathy (27). Particularly in the 45–65 age group (28). Elevated lipid peroxide levels and altered enzyme activity exemplify the degenerative changes in diabetes mellitus. This condition progresses with age and heightens the risk of complications such as diabetic neuropathy (29).

The current study revealed that 85.5% of the participants were female, of whom 32.7% exhibited diabetic neuropathy. The higher prevalence of diabetes mellitus in women is partly explained by the increased mortality among men, primarily due to cardiovascular and renal complications. Estrogen confers cardiovascular protection by inhibiting TNF-α production and upregulating prostacyclin and nitric oxide (NO), thereby preserving endothelial integrity (30). The present findings were consistent with the results of other studies, which demonstrated that gender exerted an influence on the incidence of complications arising from neuropathy (31).

It was a well-documented fact that 60% of patients in this study had had diabetes for more than five years, and that duration of diabetes exceeding this timeframe contributed to 23.6% of cases of diabetic neuropathy. Prolonged hyperglycaemia induces AGE accumulation, which interacts with RAGE and activates PKC pathways, leading to vascular dysfunction and sustained inflammation (32). Evidence indicates that the risk of diabetic neuropathy rises markedly after five years of disease duration, by which time vascular and metabolic damage is often already substantial (6).

A majority of patients 63.8% had a family history of diabetes, and 21.9% of them developed diabetic neuropathy. Genetic factors have been shown to influence the development of type 2 diabetes mellitus, with specific genetic variants potentially accelerating disease onset, particularly in the presence of risk factors such as obesity and physical inactivity (33). Moreover, specific genetic polymorphisms have been implicated in the development of diabetic neuropathy, although the underlying mechanisms remain incompletely understood (34). These include variations in genes such as methylenetetrahydrofolate reductase (MTHFR) and nitric oxide synthase 1 (NOS1), which have been associated with increased susceptibility to neuropathic complications in individuals with diabetes (35,36).

Among patients with hypertension[IB3.1][IB3.2] 49.1%, 18.2% experienced diabetic neuropathy. Hypertension contributes to neuropathic damage by impairing microvascular integrity, including vessels that supply peripheral nerves. It accelerates atherosclerosis and induces endothelial dysfunction, which together restrict oxygen and nutrient delivery to nerve tissues (37). Furthermore, elevated blood pressure promotes the generation of reactive oxygen species (ROS), exacerbating oxidative stress and causing structural damage to neural components such as lipids, proteins, and DNA (38). These combined vascular and oxidative insults are well-documented risk factors for diabetic neuropathy, and patients with hypertension are consistently shown to be at higher risk of developing this complication (39).

None of the smoking patients in this study were found to have diabetic neuropathy. However, the nicotine content in cigarettes is known to cause vasoconstriction, reducing peripheral nerve perfusion and thereby increasing the risk of nerve damage in individuals with diabetes (40). Previous studies identified smoking as an independent risk factor for diabetic neuropathy due to its ability to generate free radicals, which cause oxidative stress that contributes to nerve damage (41).

Among the patients, 56.3% presented with an abnormal body mass index, contributing to 32.75% of diabetic neuropathy cases, whereas 43.6% demonstrated low levels of physical activity, of whom 27.25% were affected by diabetic neuropathy. Excessive BMI, particularly central or abdominal obesity, plays a significant role in the development of diabetic neuropathy through complex metabolic and inflammatory pathways. Visceral adipose tissue acts as a metabolically active endocrine organ, releasing free fatty acids and pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which impair insulin signalling and perpetuate systemic insulin resistance, thus sustaining chronic hyperglycaemia (10). Persistent hyperglycaemia triggers several neurotoxic cascades, including the formation of advanced glycation end-products (AGEs), which bind to RAGE receptors, initiating oxidative stress and chronic inflammation. Concurrently, activation of protein kinase C (PKC), particularly the β isoform, disrupts microvascular homeostasis by reducing nitric oxide bioavailability and increasing endothelial permeability, thereby impairing perfusion to peripheral nerves (42). Excess intracellular glucose is also diverted into the polyol pathway, resulting in sorbitol and fructose accumulation, osmotic imbalance, and neuronal injury (10). These mechanisms undermine microvascular integrity, restrict oxygen and nutrient delivery, and contribute to axonal degeneration and demyelination, culminating in peripheral nerve dysfunction (43).

Insufficient physical activity likewise contributes to the progression of diabetic neuropathy by exacerbating insulin resistance and promoting a pro-inflammatory state. Sedentary behaviour limits skeletal muscle glucose uptake, reduces insulin sensitivity, and increases visceral adiposity, amplifying inflammatory mediator release and metabolic disruption (11). Moreover, physical inactivity impairs endothelial function by eliminating exercise-induced shear stress, which usually promotes nitric oxide synthesis and vasodilation, thereby compromising peripheral circulation (44). This vascular dysfunction diminishes neural oxygenation and nutrient supply, facilitating neuropathic changes. Furthermore, physical inactivity lowers the expression of neurotrophic factors such as brain-derived neurotrophic factor (BDNF) and insulin-like growth factor-1 (IGF-1), which are essential for neuronal maintenance, synaptic plasticity, and repair (14). The resultant decline in neuroregenerative

capacity, compounded by ongoing metabolic insult, accelerates nerve fibre degeneration and sensory deficits (12).

Conversely, maintaining a healthy BMI reduces adipose-derived inflammation and preserves insulin sensitivity, thereby protecting against hyperglycaemia-induced neural injury (45). Regular physical activity confers neuroprotective benefits by enhancing glycaemic control, improving endothelial function, and supporting peripheral nerve perfusion (13). Moreover, exercise stimulates neurotrophic factor expression and promotes neuroplasticity, facilitating axonal repair and remyelination (14). Thus, optimal body composition and sustained physical activity serve as modifiable protective factors that mitigate the metabolic, vascular, and degenerative processes underlying diabetic neuropathy (46).

The finding that physical activity exerts a more significant influence on the incidence of diabetic neuropathy than body mass index (BMI) provides essential clinical implications for the development of strategies to prevent and manage diabetic complications. Specifically, structured physical activity interventions, such as diabetes-specific exercise programs, walking groups, and community-based fitness initiatives, can be more effective than focusing solely on weight reduction. These approaches will improve glycaemic control, enhance nerve function, and reduce the risk of neuropathy (47).

CONCLUSION

The results of this study showed that BMI ($r = 0.461$, $p<0.05$) and physical activity ($r = 0.450$, $p<0.05$) were significantly associated with diabetic neuropathy in patients with type 2 diabetes mellitus. However, other factors such as age, gender, duration of diabetes, family history of diabetes, hypertension and smoking must also be considered in their role to the development of diabetic neuropathy. A comprehensive, multifactorial approach was needed to prevent and treat diabetic neuropathy.

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