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Original Article

Differences in The Severity of Diabetic Neuropathy Based on Electromyography in Type 2 Diabetes Mellitus Patients with and without Comorbidities

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Abstract

Background : Diabetic neuropathy is one of the most common complications of type 2 Diabetes Mellitus. Hyperglycemia causes axonal abnormalities and impaired schwan cell metabolism. Hypertension and hyperlipidemia associated with atherosclerosis, lipid metabolism and arterial compliance. This study aims to determine the difference in the severity of diabetic neuropathy based on electromyography between type 2 DM patients with and without comorbidities.
Methods : This study used a cross sectional design. A total of 78 diabetic neuropathy subjects from Outpatient Installation of Dr. Kariadi Hospital Semarang were divided into 3 groups, 26 subjects without comorbidities, 25 subjects with comorbid hypertension and 27 subjects with comorbid hypertension and hyperlipidemia. Inclusion criteria were aged 40–80 years, distal symmetrical polyneuropathy classification, without comorbid and with comorbid hypertension and hyperlipidemia. Exclusion criteria were patients with chemotherapy, HNP, CKD stage 5, incomplete medical records. Data were analyzed using Kruskal-Wallis comparative test.
Results : There is a difference in the severity of diabetic neuropathy based on EMG between type 2 DM patients without comorbidities and with comorbidities (p<0.01). No difference in the severity between patients with type 2 DM without comorbidities and with comorbid hypertension (p 0.058). There is a difference in the severity between patients with type 2 DM without comorbidities and with comorbid hypertension and hyperlipidemia (p<0.01).
Conclusion : There is a difference in the severity of diabetic neuropathy based on EMG between patients with type 2 DM without comorbidities, with comorbid hypertension and with comorbid hypertension and hyperlipidemia. The more comorbidities, the greater the severity of diabetic neuropathy.

Keywords : Type 2 Diabetes Mellitus, Diabetic Neuropathy, Electromyography, Hypertension, Hyperlipidemi

INTRODUCTION

Diabetes mellitus is a major metabolic disorder, where almost more than 1 billion people suffer from diabetes worldwide.[1] Diabetes mellitus is characterized by hyperglycemia and glucose intolerance.[2] Type 2 diabetes mellitus can cause microvascular and macrovascular complications. One of them is diabetic neuropathy. Diabetic neuropathy is the most common cause of neuropathy worldwide and is estimated to affect approximately half of people with diabetes, increasing morbidity, impairing quality of life and increasing mortality. The underlying pathophysiology of diabetic neuropathy is the result of hyperglycemia and microangiopathy. There is many type of diabetic neuropathy, however, distal symmetric sensorimotor polyneuropathy is the most common form. Thist suggests than the disease primarily involves the most distal regions of the body, particularly the feet, while the term "symmetric" implies that the signs and symptoms are present on both sides of the body.[3,4] Hyperglycemia increases vascular endothelial resistance, decreased neurovascular flow, decreased myoinositol in the nervous system and oxidative stress which cause neuronal, axonal and metabolic abnormalities of schwan cells that interfere with axonal transport processes.[5,6]

Hyperlipidemia is a metabolic problem associated with abnormal lipid metabolism which is directly related to cell membranes and affects the myelin sheath. Hyperlipidemia resulting in vascular lumen stenosis and impairment of microcirculation, which consequently leads to ischemia of peripheral nerves. In cases of hyperlipidemia, hyperstimulation of glutamatergic receptors may occur, disrupting intracellular calcium homeostasis and leading to toxic calcium accumulation that subsequently causes neuronal damage. Hyperlipidemia is also associated with increased leves of plasma oxLDL, which binds to oxLDL receptor on the neuronal membrane and activates NOXes in cells. Activation of NOXes leads to the generation of ROS within neurons, resulting in cellular oxidative stress.[7]

Hypertension is also associated with the severity of diabetic neuropathy. There are two main pathologies of hypertension, namely atherosclerosis and decreased arterial compliance. Vascular tone may increase due to enhanced stimulation of α-adrenoreceptors or elevated levels of peptides such as angiotensin or endothelin. The final pathway involves an increase in cytosolic calcium within vascular smooth muscle cells, leading to vasoconstriction. Several growth factors, including angiotensin and endothelin, contribute to the proliferation of vascular smooth muscle cells, a process known as vascular remodeling. This is associated with impaired microvascular flow, characterized by reduced endothelium-dependent vasodilatation and decreased nitric oxide (NO) levels in hypertensive condition. There is an association between diabetic neuropathy and comorbid factors of hypertension and hyperlipidemia. A study conducted by Lingning Huang *et al*., demonstrated that systolic blood pressure and HbA1C levels in patients with diabetic peripheral neuropathy were found to be higher than in the non-DPN group.[8–11]

Age is a risk factor for diabetic polyneuropathy. Several study groups have demonstrated that age serves as an independent factor in -patients with diabetic polyneuropathy, indicating a progressive increase in its prevalence with each decade of life. Several study found that female patients with diabetes have a higher risk of developing diabetic polyneuropathy compared to male patients. Height is also associated with diabetic polyneuropathy due to length-dependent pattern of the disease, which is related to the length of the nerve fibers. In diabetic neuropathy, these disorders are more often found in sensory nerves than in motor nerves, which is related to the morphology of these nerves.[12–14] From the examination of nerve conductivity in diabetic neuropathy patients, we often found demyelination disorders with a decrease in amplitude associated with axonopathy.

Studies comparing the severity of diabetic neuropathy based on multiple comorbidities remain limited because prior research only focused with single comorbidity such as hypertension or dyslipidemia. The purpose of this study was to determine the differences in the severity of diabetic neuropathy based on electromyographic examination between patients with type 2 diabetes mellitus without comorbidities, type 2 diabetes mellitus with comorbid hypertension and type 2 diabetes mellitus with comorbid hypertension and hyperlipidemia, then determine the relationship between distal latency and amplitude in diabetic neuropathy patients with and without comorbidities and analyze differences in the severity of diabetic neuropathy based on age, height and HbA1C in type 2 diabetes mellitus patients with and without comorbidities. Therefore, this study provides novelty by evaluating the severity of diabetic neuropathy using electrophysiological parameters between patient without comorbidities, with hypertension and with both hypertension and hyperlipidemia.

METHODS

This study is an analytical descriptive study with a cross sectional approach and was conducted at the Outpatient Installation and Medical Records of Dr. Kariadi Hospital Semarang. This study was approved by the Health Research Ethics Committee (HREC) of the Faculty of Medicine, Diponegoro University, under Ethical Clearance number 600/EC/KEPK/FK-UNDIP/XII/2023. All costs related to the research were covered by the researcher. The identities of the subjects were kept confidential throughout the study.

The population of this study were patients with type 2 diabetes mellitus with diabetic neuropathy who received treatment at Dr. Kariadi Hospital Semarang for the period January 1, 2021 – December 31, 2023. The sample size for this study was determined using the formula for unpaired categorical analytical studies and met the criteria for statistical testing. The calculation yielded a required sample size of 25 subjects per group.

The inclusion criteria were diabetic neuropathy patients aged 40–80 years, diabetic neuropathy patients with distal symmetrical polyneuropathy classification, diabetic neuropathy patients without comorbidities and with comorbid hypertension and hyperlipidemia, while the exclusion criteria in this study were diabetic neuropathy patients with a history of chemotherapy, diabetic neuropathy patients with Hernia Nucleus Pulposus (HNP), diabetic neuropathy patients with CKD stage 5 (eGFR <15 mL/min/1. 73 m) or doing hemodialysis, and incomplete medical records.

The severity of diabetic neuropathy in this study is based on electromyographic examination which is measured based on latency, amplitude and nerve conductivity on the unilateral side and based on the number of peripheral nerves involved. Where grade 0 is no nerve involved, grade 1 is mild if the sensory nervus suralis is involved, grade 2 is moderate if the sensory nervus suralis and motor of the peroneal nerve and or tibial nerve are involved, grade 3 is moderate to severe, if the suralis nerve, peroneal nerve, tibial nerve and sensory median and or ulnar nerve are involved, while grade 4 is severe if the suralis nerve, peroneal nerve, tibial nerve, sensory and motor median and or ulnar nerve are involved.

The collected data were analyzed using SPSS Statistics for Windows version 26. Data analysis was conducted in two stages: descriptive statistics and analytical statistics. To evaluate differences in the severity of diabetic neuropathy, an unpaired categorical comparative test was employed using the Chi-square test when assumptions were met. If the assumptions were not fulfilled, the KruskalWallis test was applied as an alternative. The data collected were then divided into 3 groups of neuropathy patients, namely diabetic neuropathy patients without comorbidities, with comorbid hypertension and with comorbid hypertension and hyperlipidemia. In this study, data regarding HbA1C, age of the subject which was divided into 3 groups, namely 40–50 years, 51–60 years and ≥ 61 years, and height of the subject were included.

RESULTS

From data collection through medical records on patients with a diagnosis of diabetic neuropathy who received treatment at the neurology clinic and underwent electromyographic examination at Dr. Kariadi Hospital from 2021 to 2023, 78 subjects were obtained who met the inclusion criteria. After that, they were grouped into groups of diabetic neuropathy patients without comorbidities as many as 26 people, diabetic neuropathy patients with comorbid hypertension as many as 25 people, and diabetic neuropathy patients with comorbid hypertension and hyperlipidemia as many as 27 people.

This study included 78 patients with diabetic neuropathy who fulfilled the inclusion criteria. The mean age of the participants was 56 years, and the majority were female. Based on the duration of diabetes mellitus (DM), the majority of cases were found in patients with a disease onset of ≥5 years. The mean body mass index (BMI) was 25.14 and the mean HbA1C level was 8.17. Differences in electromyography results in diabetic neuropathy patients in this study were associated with severity. Severity is measured based on latency, amplitude and nerve velocity. The severity of diabetic neuropathy based on electromyographic examination is divided based on the number of peripheral nerves involved.

Table 2 shows the result of p=0.001. There is significant difference between the severity of diabetic neuropathy in DM subjects without comorbidities, DM subjects with comorbid hypertension, and DM subjects with comorbid hypertension and hyperlipidemia.

Table 3 shows the result of p=0.058. There is no statistically significant difference between the severity of diabetic neuropathy in DM subjects without comorbidities and DM subjects with comorbid hypertension.

Table 4 shows the results of p<0.01. There is significant difference between the severity of diabetic neuropathy in DM subjects without comorbidities and DM subjects with comorbid hypertension and hyperlipidemia.

Table 5 shows that there is a significant correlation between latency and amplitude in patients with DM without comorbidities with a very strong correlation in Median Sensory Nerve (rho -0.903) and Suralis Sensory Nerve (rho -0.894). There was a strong correlation in Ulnar Sensory Nerve (rho -0.692) and moderate correlation in Median Motoric Nerve (rho - 0.411). Beside that, there is a significant correlation between distal latency and amplitude in patients DM with comorbidities with a very strong correlation in Suralis Sensory Nerve (rho - 0.956), strong correlation in Median Sensory Nerve (rho -0.774) and Ulnar Sensory Nerve (rho -0.676). There was a moderate correlation in Median Motoric Nerve (rho -0.423), Peroneal Motoric Nerve (rho -0.594) and Tibial Motoric Nerve (rho -0.507).

Based on theory, age, height and HbA1C are factors that can affect the severity of diabetic neuropathy in both DM patients without comorbidities and with comorbidities (Table 6).

TABLE 1
Demographic and Clinical Characteristics of Subjects

Variable	Frequency	%	Mean ± SD	Median (Min–Max)
Demographics Data				
Age (year)			56.09 ± 8.53	56.5 (40–74)
40-50	20	25.6		
51-60	32	41		
≥ 61	26	33.3		
Gender				
Male	33	42.3		
Female	45	57.7		
Clinical Data				
Group of diseases				
DM	26	33.3		
DM and hypertension	25	32.1		
DM, hypertension & hyperlipidemia	27	34.6		
DM onset				
< 5 years	29	37.2		
≥ 5 years	49	62.8		
Weight			65.92 ± 11.79	65 (33–100)
Height			161.77 ± 7.3	160 (145–180)
IMT			25.14 ± 4	24.7 (15.7–39.06)
HbA1C			8.17 ± 2.23	7.55 (5.2–17.6)

TABLE 2
Differences in Severity of Diabetic Neuropathy in DM Subjects without comorbid, comorbid hypertension and comorbid hypertension and hyperlipidemia

		Group of Diseases			*P* Value
		DM (n=26)	DM & hypertension (n=25)	DM, hypertension & hyperlipidemia (n=27)	
Severity of Diabetic Neuropathy	Grade 1	4 (100%)	0 (0%)	0 (0%)	0.001*
	Grade 2	5 (41.7%)	5 (41.7%)	2 (16.7%)	
	Grade 3	13 (40.6%)	11 (34.4%)	8 (25%)	
	Grade 4	4 (13.3%)	9 (30%)	17 (56.7%)	
	Total	26 (33.3%)	25 (32.1%)	27 (34.6%)	

Notes : *significant ($p < 0.05$) using Kruskal-Wallis test

TABLE 3
Differences in Severity of Diabetic Neuropathy in DM Subjects without Comorbidities and with Comorbid Hypertension

		Group of Diseases		*P* Value
		DM (n=26)	DM & hypertension (n=25)	
Severity of Diabetic Neuropathy	Grade 1	4 (100%)	0 (0%)	0.058
	Grade 2	5 (50%)	5 (50%)	
	Grade 3	13 (54.2%)	11 (45.8%)	
	Grade 4	4 (30.8%)	9 (69.2%)	
	Total	26 (51%)	25 (49%)	

Notes : *significant ($p < 0.05$) using Kruskal-Wallis test

TABLE 4
Differences in Severity of Diabetic Neuropathy in DM Subjects without Comorbidities and with Comorbid Hypertension and Hyperlipidemia

		Group of Diseases		*P* Value
		DM (n=26)	DM, hypertension & hyperlipidemia (n=27)	
Severity of Diabetic Neuropathy	Grade 1	4 (100%)	0 (0%)	< 0.01*
	Grade 2	5 (71.4%)	2 (28.6%)	
	Grade 3	13 (61.9%)	8 (38.1%)	
	Grade 4	4 (19%)	17 (81%)	
	Total	26 (49.1%)	27 (50.9%)	

Notes : *significant ($p < 0.05$) using Kruskal-Wallis test

TABLE 5
Correlation between Distal Latency and Amplitude in DM Subjects without Comorbidities and with Comorbidities

		DM without Comorbidities		DM with Comorbidities	
	Amplitude Score	rho	*p*	rho	*p*
Distal Latency	Median Sensory Nerve	-0.903	< 0.01	-0.774	< 0.01
	Ulnar Sensory Nerve	-0.692	< 0.01	-0.676	< 0.01
	Median Motoric Nerve	-0.411	0.037	-0.423	0.002
	Ulnar Motoric Nerve	0.304	0.130	-0.173	0.219
	Suralis Sensory Nerve	-0.894	< 0.01	-0.956	< 0.01
	Peroneal Motoric Nerve	-0.249	0.220	-0.594	< 0.01
	Tibial Motoric Nerve	-0.301	0.136	-0.594	< 0.01

Notes : *significant ($p < 0.05$) using Spearman Correlation test

TABLE 6
Differences in Severity of Diabetic Neuropathy by Age Group in DM Subjects with and Without Comorbidities

Variable	Severity of Diabetic Neuropathy					Total	*P* Value
	Grade 0	Grade 1	Grade 2	Grade 3	Grade 4		
DM without comorbidities							
Age 40–50	0 (0%)	2 (33.3%)	1 (16.7%)	3 (50%)	0 (0%)	6	
Age 51–60	0 (0%)	1 (8.3%)	4 (33.3%)	5 (41.7%)	2 (16.7%)	12	0.405
Age ≥ 61	0 (0%)	1 (12.5%)	0 (0%)	5 (62.5%)	2 (25%)	8	
Height	0	164.25 ± 5.38	163 ± 5.43	158.15 ± 7.24	162.25 ± 5.32		0.394
HbA1C	0	8.05 ± 2.9	8.26 ± 2.6	7.85 ± 2.26	10.23 ± 5.23		0.903
DM with comorbidities							
Age 40–50	0 (0%)	0 (0%)	2 (14.3%)	6 (42.9%)	6 (42.9%)	14	
Age 51–60	0 (0%)	0 (0%)	4 (20%)	7 (35%)	9 (45%)	20	0.511
Age ≥ 61	0 (0%)	0 (0%)	1 (5.6%)	6 (33.3%)	11 (61.1%)	18	
Height	0	0	162.43 ± 9.48	160.95 ± 7.4	163.3 ± 7.43		0.74
HbA1C	0	0	8.16 ± 2.15	7.88 ± 1.61	8.24 ± 1.94		0.389

Notes : *significant ($p < 0.05$) using Kruskal-Wallis test

Table 6 shows there is no significant difference between age, height and HbA1C with the severity of diabetic neuropathy. However, the older age group (age ≥ 61 years) was found to have more grade 3 (62.5%) and grade 4 (25%) diabetic neuropathy. For height, grade 3 diabetic neuropathy was 158.15 ± 7.24 and grade 4 diabetic neuropathy was 162.25 ± 5.32. While based on HbA1C levels obtained for grade 4 of 10.23 ± 5.23.

In the group of DM subjects with comorbidities also showed the results there is no significant difference between groups of age, height and HbA1C with the severity of diabetic neuropathy. In the older age group (age ≥ 61 years), there were more grade 3 (33.3%) and grade 4 (61.1%) diabetic neuropathy. For height, grade 3 diabetic neuropathy was found at 160.95 ± 7.4 and grade 4 diabetic neuropathy at 163.3 ± 7.43. While based on HbA1C levels 8.24 ± 1.94.

DISCUSSION

This study involved 78 diabetic neuropathy patients who met the inclusion criteria. Based on demographic data, the majority of patients with diabetic neuropathy in this study were female (57.7%). A study conducted by Dipika Bansal *et al*. which also reported that most patients were female (50.7%). This finding is consistent with studies by Yanhui Lu *et al*. and Zohaib Igbal *et al*., which demonstrated that female patients with diabetes mellitus are more likely to develop diabetic neuropathy compared to males. Furthermore, their study found that women have a threefold higher risk of developing diabetic neuropathy than men. This difference may be related to higher physical activity levels among men. But, there is another study who reported that the onset of diabetic neuropathy occurs earlier in men than in women. This may be attributed to greater exposure to stressors and a decline in androgen hormens in male patients with diabetes mellitus, as these hormones exert neuroprotective effects on both the central and peripheral nervous systems.[15]

In this study, the average age of diabetic neuropathy patients in this study was 56.09 years. This agrees with the study of Fipika Bansal *et al* in 2014 obtained similar results, namely the average age of patients with diabetic neuropathy was 57.1 years.[15] This is also in accordance with studies which state that diabetic neuropathy tends to occur at the age of more than 50 years.[1] Hyperglycemia conditions take time to cause damage to the nerves so that increasing age is indirectly related to an increased risk of diabetic neuropathy in patients with type 2 diabetes mellitus.[1,16]

In this study there was a significant difference between the severity of diabetic neuropathy in Diabetes Mellitus patients without comorbidities and Diabetes Mellitus patients with comorbidities. Some comorbidities are associated with the severity of diabetic neuropathy such as hyperlipidemia, smoking, hypertension and obesity. This is in agreement with the study of Shafina

Sachedina *et al*, who found that some of the comorbidities were associated with the severity of diabetic neuropathy in patients with type 2 diabetes mellitus.[17]

There was no statistically significant difference between the severity of diabetic neuropathy in patients with DM without comorbidities and DM with comorbid hypertension. However, the data showed that the number of patients with grade 4 severity was higher among patients who also had comorbid hypertension. In addition, there were no cases of grade 1 diabetic neuropathy in patients with diabetes mellitus with comorbid hypertension. This is in agreement with the study of Shafina Sachedina *et al*, who found that hypertension did not provide a significant difference with the severity of diabetic neuropathy in patients with type 2 DM.[17] However, this result is different from the study of Lingning Huang *et al*, where a significant difference was found between diabetic neuropathy patients in DM patients with and without hypertension.[8] Meanwhile, the results of a study conducted by Sethi Y, et. al where found that hypertension is a modifiable risk factor for the development of diabetic neuropathy in patients with type 2 DM.[18] Hypertension is associated with decreased nerve perfusion, endoneural hypoxia and structural changes in nerve microvasculature. Growth factors such as angiotensin and endothelin also cause vascular remodelling where there is an increase in vascular smooth muscle and associated with changes in vasoconstriction function and decreased vasodilation or vascular elasticity and associated with the occurrence of subclinical atherosclerosis due to impaired arterial compliance.[8]

There was a statistically significant difference between the severity of diabetic neuropathy in patients DM and DM with comorbid hypertension and hyperlipidemia. Hypertension and hyperlipidemia are 2 important cardiovascular risk factors as predictors of severe diabetic complications.[19] Based on a study by Nidhi Yadav *et al*., diabetic neuropathy patients with hyperlipidemia showed differences in the severity of diabetic neuropathy. The progression and severity of diabetic neuropathy is related to factors including elevated triglycerides, smoking, hypertension and obesity. The presence of hypertension is associated with vascular remodeling and leads to impaired perfusion in the nerve vasculature.[18] Meanwhile, hyperlipidemia is associated with oxidative stress in the dorsal root ganglia which plays a major role in the occurrence of neurodegeneration in diabetes.[17]

This study found a significant correlation between distal latency and amplitude in patients with DM without comorbidities with a very strong correlation in Median Sensory Nerve and Suralis Sensory Nerve. In addition, there was a strong correlation in Ulnar Sensory Nerve and a moderate correlation in Median Motoric Nerve. In DM patients with comorbidities, there was a significant correlation between distal latency and amplitude with a very strong correlation in Suralis Sensory Nerve, strong correlation in Median Sensory Nerve and Ulnar Sensory Nerve. In addition, there was also a moderate correlation in Median Motoric Nerve, Peroneal Motoric Nerve, and Tibial Motoric Nerve. In DM patients, sensory nerve abnormalities are more prominent than motor nerve abnormalities. The vulnerability in sensory nerves is associated with thinner and longer nerve types compared to motor nerves.[20,21]

Latency is related to the presence or absence of myelin destruction while amplitude is related to axonal. Amplitude indicates how many nerve fibers are stimulated.[12,22] Study from Ruchi *et al*, found that axonal-type damage of the ulnar nerve was found in patients with diabetic neuropathy and type 2 diabetes mellitus. In a study conducted by Anwar H. Siddique *et al*., the amplutides of the median and sural nerves were significantly lower in patients with symptomatic diabetic neuropathy compared to those with asymptomatic diabetic neuropathy.[20] Another study demonstrated a decrease in sensoryh and motor nerve amplitudes due to axonopathy, which is more commonly observed in diabetic patients. Axonal neuropathy results in reduced amplitude, whereas demyelinating neuropathy is characterized by slowed conduction. Previous studies have shown that diabetic neuropathy involves both of these components. Study by Raju Panta *et al*, found that a decrease in the amplitude of the sensory nerve was often found in demyelinating lesions, a decrease in the amplitude of the Suralis Nerve examination indicates axonal loss which is often also found in demyelinating disorders. The presence of amplitude disorders (axonal neuropathy) is the most powerful measure related to the severity of neuropathic disorders.[23]

When associated with age, there was no significant difference between DM patient groups without comorbidities and with comorbidities. However, descriptively, it was found that the older age group had more grade 3 and 4 diabetic neuropathy severity in both the DM without comorbidities and with comorbidities groups. This may be related to medication compliance in elderly patients and the association with other risk factors such as HbA1C variability and the number of comorbidities in each age group in the DM with comorbidities group. Several study have indicated that age serves as an independent factor in patients with diabetic polyneuropathy, suggesting a progressive increase in its prevalence with each decade of life. Animal studies have shown that the severity of diabetic neuropathy is associated with nerve conduction velocity. In young rats, no changes in conduction velocity are observed initially, but a subsequent decline occurs over time. With increasing age, there is an accumulation of nerve damage, in which patients with diabetic neuropathy may experience injury to both large and

small nerve fibers, caused by axonal damage and demyelination.[16]

Similarly, with height, we found no statistically significant difference between height and severity of diabetic neuropathy in both groups of DM patients without and with comorbidities. This may also be related to other risk factors such as age and HbA1C variability.[24] The San Luis Valley study also found that height was not associated with the occurrence of diabetic neuropathy.[25] Study by Toeko Matsumoto, *et al* also found that height was not associated with neuropathy. These results differ from a population study in Mauritius where height gave significant results in increasing the risk of diabetic neuropathy. Height is associated with diabetic polyneuropathy due to the length-dependent pattern of the disease, which is related to the length of the nerve fibers. The taller the individual, the longer the segment of axonal tapering and the slower the conduction velocity. In addition, the internodal distance becomes shorter, axonal transport slows down and alterations in cell membrane properties occur.

In this study, there was no statistically significant difference between HbA1C and the severity of diabetic neuropathy in both groups of DM patients without comorbidities and with comorbidities. However, this study found that patients with diabetes mellitus (DM) without comorbidities have higher HbA1C levels compared to those with comorbid conditions. This may be related to age as a risk factor and to the higher level of treatment adherence observed among patients with comorbidities compared to those without. Based on study by Dipika Bansal, *et al*, it was found that high levels of HbA1C were not significantly associated with diabetic polyneuropathy. Increased variability of HbA1C in patients with diabetes mellitus is associated with increased severity of diabetic neuropathy. Study by Caprnda *et al*, was found that there was no association between short-term variability of glycemic levels and micro- and macrovascular complications in type 2 DM.[26] Long-term variability of glycemic levels assessed by HbA1C variability can increase oxidative stress leading to cell and tissue damage.[27]

CONCLUSION

This study found differences in the severity of diabetic neuropathy based on EMG examination between patients with type 2 diabetes mellitus without comorbidities, type 2 diabetes mellitus with comorbid hypertension and type 2 DM with comorbid hypertension and hyperlipidemia. There was also a difference in the severity of diabetic neuropathy between patients with type 2 diabetes mellitus without comorbidities and with comorbid hypertension and hyperlipidemia. However, no significant differences between diabetic neuropathy without comorbidities and comorbid hypertension alone. The more comorbidities in patients with diabetic neuropathy, the greater the severity of diabetic neuropathy. This study found a correlation between distal latency and amplitude in type 2 DM patients without comorbidities and with comorbidities, which shows that diabetic neuropathy can have both components according to severity. When associated with other risk factors such as age, height and HbA1C, there was no significant difference in the severity of diabetic neuropathy in both patients with type 2 DM without comorbid and with comorbid.

Based on this study, the following limitations were found: data collection from medical records where electromyography examination may not be carried out ideally, the number of samples is limited to patients with Diabetes mellitus with 1 type of comorbid, this study does not consider diabetes mellitus therapy obtained by patients and physical activity of patients. Other risk factors such as the onset of type 2 diabetes mellitus cannot be precisely ascertained considering that the data were taken based on medical records. Long-term variability of HbA1C is closely related to the severity of diabetic neuropathy. However, in this study, HbA1C data collection was only done in the short term.

CONFLICT OF INTEREST

The authors declared no conflict of interest.

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REFERENCES

1. Yavuz DG. Chapter 1 - Classification, risk factors, and clinical presentation diabetic neuropathy. In: Tavakoli M, editor. Diabetic Neuropathy [Internet]. Elsevier; 2022. p. 1–9. Available from: https://www.sciencedirect.com/science/article/pii/B9780128206690000141
2. Ohiagu F, Chikezie P, Chikezie C. Pathophysiology of diabetes mellitus complications: Metabolic events and control. Biomedical Research and Therapy. 2021;8(3):4243–57. Available from: https://doi.org/10.15419/bmrat.v8i3.663
3. Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, *et al*. Pathophysiology of type 2 diabetes mellitus. Int J Mol Sci. 2020;21(17):6275. Available from: https://doi.org/10.3390/ijms21176275
4. Hansen C, Määttä L, Andersen S, Charles M. The epidemiology of diabetic neuropathy. In: Tesfaye S, Selvarajah D, editors. Diabetic Neuropathy. Humana Cham; 2023. p. 5–36. Available from: https://doi.org/10.1007/978-3-031-15613-7_2
5. Smith S, Normahani P, Lane T, Hohenschurz-Schmidt D, Oliver N, Davies AH. Pathogenesis of distal symmetrical polyneuropathy in diabetes. Life (Basel). 2022;12(7):1074. Available from: https://doi.org/10.3390/life12071074
6. Pang L, Lian X, Liu H, Zhang Y, Li Q, Cai Y, *et al*. Understanding diabetic neuropathy: focus on oxidative stress. Oxid Med Cell Longev. 2020;2020:1–13. Available from: https://doi.org/10.1155/2020/9524635

7. Iqbal Z, Bashir B, Ferdousi M, Kalteniece A, Alam U, Malik RA, *et al*. Lipids and peripheral neuropathy. Lipidology. 2021;32(4):249–57. Available from: https://doi.org/10.1097/MOL.0000000000000770
8. Huang L, Zhang Y, Wang Y, Shen X, Yan S. Diabetic peripheral neuropathy is associated with higher systolic blood pressure in adults with type 2 diabetes with and without hypertension in the chinese han population. Can J Diabetes. 2020;44(7):615–23. Available from: https://doi.org/10.1016/j.jcjd.2019.11.005
9. Harrison DG, Coffman TM, Wilcox CS. Pathophysiology of hypertension: the mosaic theory and beyond. Ciculation Research. 2021;128(7):847–63. Available from: https://doi.org/10.1161/circresaha.121.318082
10. Webb AJS, Werring DJ. New insights into cerebrovascular pathophysiology and hypertension. Stroke AHA. 2022;53(4):1054–64. Available from: https://doi.org/10.1161/STROKEAHA.121.035850
11. Lan YR, Chiu WC, Huang CC. HbA1C variability is strongly associated with the severity of peripheral neuropathy in patients with type 2 diabetes. Front Neurosci. 2019;13:90. Available from: https://doi.org/10.3389/fnins.2019.00090
12. Diwan J, Patel K, Goswami D, Patel V. Sensory nerve conduction velocity study in diabetic patients. Natl J Physiol Pharm Pharmacol. 2021;11(3):315–9. Available from: http://dx.doi.org/10.5455/njppp.2021.11.01028202122012021
13. Lu Y, Xing P, Cai X, Luo D, Li R, Lloyd C, *et al*. Prevalence and risk factors for diabetic peripheral neuropathy in type 2 diabetic patients from 14 countries: estimates of the INTERPRET-DD Study. Front Public Health. 2020;8:534372. Available from: https://doi.org/10.3389/fpubh.2020.534372
14. Rahman F, Siddiqui A, Singhal S, Ashraf H, Faraz A. Association of nerve conduction velocity with Total Body Fat Mass and Body Mass Index in type 2 diabetes mellitus physiology section. 2020;14(9):4–7. Available from: https://doi.org/10.7860/JCDR/2020/44524.1406
15. Bansal D, Gudala K, Muthyala H, Esam H prasad, Nayakallu R, Bhansali A. Prevalence and risk factors of development of peripheral diabetic neuropathy in type 2 diabetes mellitus in a tertiary care setting. J Diabetes Investig. 2014;5(16):714–21. Available from: https://doi.org/10.1111/jdi.12223
16. Mao F, Zhu X, Liu S, Qiao X, Zheng H, Lu B, *et al*. Age as an independent risk factor for diabetic peripheral neuropathy in Chinese patients with type 2 diabetes. Aging Dis. 2019;10(3):592–600. Available from: https://doi.org/10.14336/AD.2018.0618
17. Sachedina S, Toth C. Association of comorbidities with increasing severity of peripheral neuropathy in diabetes mellitus. World J Diabetes. 2013;4(4):135–44. Available from: https://doi.org/10.4239/wjd.v4.i4.135
18. Sethi Y, Uniyal N, Vora V, Agarwal P, Murli H, Joshi A, *et al*. Hypertension the "Missed Modifiable Risk Factor" for diabetic neuropathy: a systematic review. Curr Probl Cardiol. 2023;48(4):101581. Available from: https://doi.org/10.1016/j.cpcardiol.2022.101581
19. Lord J, Reid K, Duclos C, Mai A, Odoi A. Investigation of predictors of severity of diabetes complications among hospitalized patients with diabetes in Florida, 2016–2019. BMC Public Health [Internet]. 2023;23(1):2424. Available from: https://doi.org/10.1186/s12889-023-17288-x
20. Siddiqui AH, Tauheed N, Ashraf H, Ahmad J. Association of sensory nerve action potential amplitude and velocity with type 2 diabetic peripheral neuropathy. Cureus. 2023;15(10):1–10. Available from: https://doi.org/10.7759/cureus.46501
21. Park JH, Park JH, Won JC. Associations of nerve conduction study variables with clinical symptom scores in patients with type 2 diabetes. Ann Clin Neurophysiol. 2019;21(1):36–43. Available from: https://doi.org/10.14253/acn.2019.21.1.36
22. Nurlaela S, Kurniawan SN, Husna M. Electroneuromyography examination of diabetic polyneuropathy patients.Malang Neurology Journal. 2019;5(1):1–4. Available from: https://mnj.ub.ac.id/index.php/mnj/article/view/305
23. Shrivastava R, Saluja A, Sharma B, Dave R, Shrivastava A. To evaluate peripheral neuropathy of ulnar sensory nerve through nerve conduction studies in Type 2 diabetic patients. Natl J Physiol Pharm Pharmacol. 2023;13(5):935–8. Available from: https://doi.org/10.5455/njppp.2023.13.09425202220092022
24. Nozawa K, Ikeda M, Kikuchi S. Association between HbA1c levels and diabetic peripheral neuropathy: a case-control study of patients with type 2 diabetes using claims data. Drugs Real World Outcomes [Internet]. 2022;9(3):403–14. Available from: https://doi.org/10.1007/s40801-022-00309-3
25. Elafros MA, Callaghan BC. Diabetic neuropathies. Continuum (Minneap Minn). 2023;29(5):1401–17. Available from: https://doi.org/10.1212/con.0000000000001291
26. Caprnda M, Mesarosova D, Ortega P, Krahulec B, Egom E, Rodrigo L, *et al*. Glycemic variability and vascular complications in patients with type 2 Diabetes Mellitus. Folia Med (Plovdiv). 2017;59(3):270–7. Available from: https://doi.org/10.1515/folmed-2017-0048
27. Su JB, Zhao LH, Zhang XL. HbA1c variability and diabetic peripheral neuropathy in type 2 diabetic patients. Cardiovasc Diabetol. 2018;17(1):47. Available from: https://doi.org/10.1186/s12933-018-0693-0