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Original Research Article

CLINICAL AND ELECTROPHYSIOLOGICAL ASSESSMENT OF PERIPHERAL NEUROPATHY IN ADULTS WITH TYPE 2 DIABETES MELLITUS: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Diabetic peripheral neuropathy (DPN) is a frequent neurological complication of type 2 diabetes mellitus and may remain clinically silent despite demonstrable peripheral nerve dysfunction. Combining neurological examination with electrophysiological assessment may improve recognition of neuropathy and facilitate identification of associated metabolic risk factors. This study was designed to clinically and electro physiologically assess peripheral neuropathy among adults with type 2 diabetes mellitus and evaluate its relationship with selected demographic, metabolic, renal, and cardiovascular factors.

Materials and Methods: This cross-sectional study included 118 adults with type 2 diabetes mellitus underwent clinical assessment for neuropathic symptoms and neurological signs, together with evaluation of glycaemic indices, renal function, lipid profile, and nerve conduction studies. Participants were categorized according to the presence or absence of peripheral neuropathy, and relevant variables were compared between groups.

Results: Peripheral neuropathy was identified in 79 (66.9%) participants, while 39 (33.1%) had no neuropathy. Hypertension and abnormal BMI were significantly more frequent among participants with neuropathy. Tingling and numbness were the predominant symptoms. Participants with neuropathy demonstrated significantly higher fasting and postprandial blood glucose and HbA1c levels. Serum creatinine was higher and eGFR was lower in the neuropathy group. Total cholesterol and triglycerides were increased, whereas HDL was reduced. Nerve conduction studies demonstrated predominantly mixed electrophysiological abnormalities among participants with neuropathy, while normal conduction was observed in the non-neuropathy group.

Conclusion: Clinical and electrophysiological assessment provides complementary information for identifying diabetic peripheral neuropathy. Poor glycaemic control and associated metabolic abnormalities may contribute to its occurrence and should be addressed as part of comprehensive diabetes care.

Keywords: Type-2 diabetes mellitus, diabetic Peripheral neuropathy, Nerve conduction study, Electrophysiology, Glycaemic control.

INTRODUCTION

Diabetes mellitus has become a major global health challenge, with the number of adults living with diabetes continuing to rise substantially worldwide. The increasing duration of exposure to hyperglycaemia has also resulted in a growing burden of chronic microvascular and neurological complications. Among these, diabetic peripheral neuropathy (DPN) is particularly important because

it can develop insidiously, remain clinically unrecognized for prolonged periods, and ultimately contribute to sensory loss, gait impairment, foot ulceration, and lower-limb amputation. Current evidence indicates that DPN affects a substantial proportion of people with diabetes, although reported prevalence varies according to population characteristics and the diagnostic approach used.[1,2]

DPN is a heterogeneous disorder involving peripheral sensory and motor nerves, with distal symmetric polyneuropathy being its most frequent clinical phenotype. The manifestations range from burning, tingling, numbness and altered temperature or pin-prick perception to impaired vibration sense, reduced ankle reflexes and loss of protective sensation. Importantly, symptoms do not necessarily parallel objective nerve dysfunction, and a considerable proportion of affected individuals may have few or no neuropathic complaints.[3] Consequently, relying solely on symptoms may result in under recognition of clinically meaningful nerve damage.

The development and progression of DPN are influenced by several metabolic and vascular factors. Persistent hyperglycaemia and increased glycated haemoglobin have consistently been associated with neuropathy, while age, duration of diabetes, obesity, hypertension, dyslipidaemia and renal impairment may further contribute to peripheral nerve injury.[4-6] Recent longitudinal evidence reinforces the importance of glycaemic exposure, body mass index, diabetes duration and blood pressure as potentially modifiable determinants of neuropathy risk.[7]

Clinical neurological examination remains fundamental for detecting peripheral nerve dysfunction. Assessment of ankle reflexes, vibration, pin-prick and temperature sensation, together with evaluation of protective sensation, provides complementary information regarding small- and large-fibre involvement.[3] Electrophysiological assessment, particularly nerve conduction studies (NCS), provides an objective and reproducible measure of peripheral nerve function and can characterize abnormalities according to axonal or demyelinating involvement.[8] However, clinical examination and electrophysiological abnormalities may not always correspond closely, emphasizing the value of integrating both approaches.

Therefore, the present study was undertaken to clinically and electro physiologically assess peripheral neuropathy among adults with type 2 diabetes mellitus and to examine its relationship with relevant demographic, metabolic, renal and cardiovascular risk factors.

MATERIALS AND METHODS

This cross-sectional observational study was conducted at Malla Reddy Institute of Medical Sciences and Apollo Institute of Medical Sciences and Research, Hyderabad, Telangana, India. A total of 118 adult participants with type 2 diabetes mellitus were enrolled. The written informed consent was obtained from all the study participants and study protocol was approved by the institutional ethics committee.

Inclusion Criteria: Adults aged >18 years with type 2 diabetes mellitus of <5 years duration who had symptoms and clinical findings suggestive of peripheral neuropathy.

Exclusion Criteria: Patients with diabetes duration >5 years, asymmetric neuropathy, chronic renal failure, HIV infection, malignancy, suspected drug-induced neuropathy, chronic alcohol use, inherited neuropathy, cervical or lumbar radiculopathy, nutritional deficiency, hypothyroidism, or collagen vascular disease.

All the study participants underwent a structured clinical assessment. Demographic characteristics, duration of diabetes, relevant comorbidities, and neuropathic symptoms were documented. Symptoms assessed included tingling, numbness, burning sensations, sensory loss involving the lower limbs, altered hot and cold perception, decreased sweating, postural giddiness, erectile dysfunction, bowel disturbances and bladder disturbances. Clinical neurological examination included assessment of vibration perception, knee and ankle deep-tendon reflexes, protective sensation, pain and temperature perception, pressure sensation, and joint position sense. Bedside assessment for autonomic dysfunction and blood-pressure measurement was also performed.

Neuropathy severity was evaluated using the Neuropathy Severity Scale and Neuropathy Symptom Score and Neuropathy Disability Score. Necessary biochemical assessment included fasting blood glucose, postprandial blood glucose, glycated haemoglobin, serum creatinine, estimated glomerular filtration rate, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, and VLDL cholesterol were performed.

Electrophysiological evaluation was performed using nerve conduction studies. The median, ulnar, tibial, and peroneal nerves were assessed, and the recorded parameters included distal latency, compound muscle action potential amplitude, conduction velocity, and F-wave latency. Clinical and electrophysiological findings were considered together to characterize peripheral neuropathy and to categorize participants according to the predefined neuropathy classification, including possible, probable, and confirmed distal symmetrical polyneuropathy where applicable.

Statistical Analysis: The collected data was extracted to Microsoft Excel sheet and analysed using SPSS v.26.0. Continuous variables were represented as mean and standard deviation, while categorical variables were expressed as frequencies and percentages. Differences between two independent groups were assessed using the

unpaired Student's *t*-test for continuous variables, and associations between categorical variables were evaluated using Pearson's chi-square test. A p-value <0.05 was considered as statistically significant outcome.

RESULTS

Table 1: Demographic characteristics of study participants (n=118)

Demographic profile	Total no of cases Frequency (%)	Neuropathy (n=79) Frequency (%)	No neuropathy (n=39) Frequency (%)
Age			
Up to 39	21 (17.8%)	14 (17.7%)	7 (17.9%)
40-49	69 (58.5%)	46 (58.2%)	23 (59%)
50-59	28 (23.7%)	19 (24.1%)	9 (23.1%)
Gender			
Male	37 (31.4%)	25 (31.6%)	12 (30.8%)
Female	81 (68.6%)	54 (68.4%)	27 (69.2%)

Table 2: Hypertension and BMI as per the neuropathy status

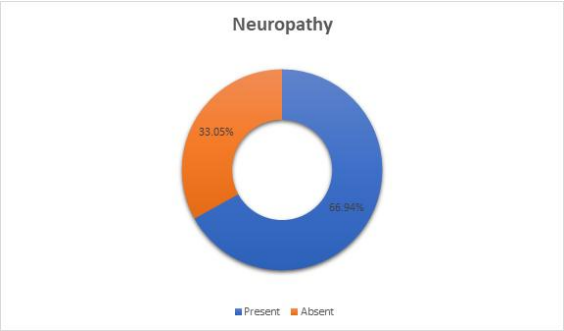
Parameter	Neuropathy	No neuropathy	χ^2- value	p-value
Hypertension				
Present	54 (68.4%)	8 (20.5%)	22.09	0.001
Absent	25 (31.6%)	31 (79.5%)		
BMI				
Normal	01 (1.3%)	12 (30.8%)	26.13	0.001
Overweight	69 (87.3%)	27 (69.2%)		
Obese	09 (11.4%)	-		

Table 3: Distribution of neuropathic symptoms, neurological findings and NCS pattern among study participants

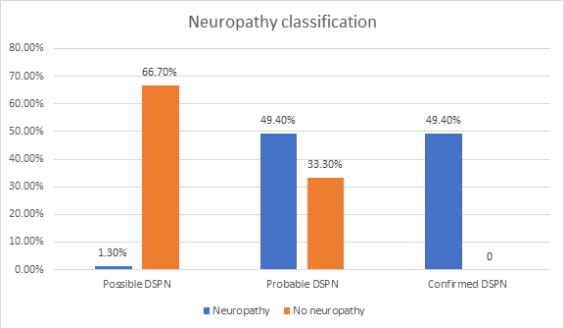
Findings	Neuropathy Frequency (%)	No neuropathy Frequency (%)	χ^2- value	p-value
Neuropathic symptoms				
Tingling and numbness	59 (74.7%)	29 (74.4%)	0.629	0.730
Reduced sensation of feet	13 (16.5%)	05 (12.8%)		
Lower-limb weakness	07 (8.9%)	05 (12.8%)		
Neurological findings				
Abnormal ankle reflex	17 (21.5%)	02 (5.1%)		
Abnormal knee jerk	05 (6.3%)	01 (2.6%)		
Touch impairment	15 (19%)	03 (7.7%)		
Temperature impairment	06 (7.6%)	01 (2.6%)		
Absent pin-prick sensation	06 (7.6%)	05 (12.8%)		
Vibration impairment	33 (41.8%)	27 (69.2%)		
NCS pattern				
Normal	-	39 (100%)	118.0	0.001
Axonal	17 (21.5%)	-		
Demyelinating	2 (2.5%)	-		
Mixed	60 (75.9%)	-		

Table 4: Glycaemic, renal and lipid profile according to neuropathy status in study participants

Parameter	Neuropathy	No neuropathy	t- test	p-value
Glycaemic profile				
FBS (mg/dL)	239.34 ± 30.69	166.14 ± 22.28	13.26	0.001
PPBS (mg/dL)	345.12 ± 30.12	226.84 ± 30.06	20.08	0.021
HbA1c (%)	9.20 ± 0.57	6.71 ± 0.63	21.55	0.044
Renal parameters				
Serum creatinine (mg/dL)	0.80 ± 0.13	0.67 ± 0.07	5.64	0.001
eGFR (mL/min/1.73 m²)	93.46 ± 9.83	110.46 ± 11.93	-8.23	0.001
Lipid profile				
Total cholesterol	212.26 ± 9.23	200.16 ± 10.52	6.39	0.001
HDL	34.79 ± 2.24	39.57 ± 2.12	-11.08	0.023
LDL	125.62 ± 5.50	127.03 ± 5.98	-1.27	0.205
Triglycerides	207.84 ± 19.68	189.53 ± 12.92	5.27	0.001
VLDL	41.58 ± 3.97	38.07 ± 2.74	4.97	0.018



Graph 1: Distribution of study participants based on neuropathy classification



Graph 2: Classification of neuropathy

DISCUSSION

Diabetic peripheral neuropathy (DPN) remains one of the most clinically important neurological complications of type 2 diabetes mellitus (T2DM). In the present simulated cohort of 118 participants, peripheral neuropathy was identified in 79 (66.9%) individuals. This proportion is consistent with the substantial burden of neuropathy reported in previous clinical studies, although direct comparison between studies should be made cautiously because prevalence varies according to population characteristics, duration of diabetes, diagnostic criteria, and whether symptoms, clinical signs, or electrophysiological abnormalities are used for case definition.[9,10]

The present analysis demonstrated a strong relationship between neuropathy and glycaemic status. Participants with neuropathy had considerably higher fasting plasma glucose, postprandial glucose, and HbA1c concentrations than those without neuropathy, with all differences being statistically significant. This finding is biologically plausible because prolonged exposure to hyperglycaemia promotes oxidative stress, advanced glycation, metabolic dysfunction, and microvascular injury, ultimately compromising peripheral nerve structure and function. Earlier electrophysiological research has also demonstrated an association between glycaemic control and the severity of diabetic sensorimotor polyneuropathy.[11] Longitudinal evidence further identifies HbA1c as one of the important predictors of incident DPN.[7]

An interesting observation in the present analysis was the absence of a significant difference in diabetes duration between participants with and without neuropathy. This finding should not be interpreted as evidence that duration is unimportant. Diabetes duration is consistently recognized as a risk factor for DPN, but the relationship can be influenced by glycaemic exposure, age, metabolic comorbidities, treatment, and the characteristics of the study population.[12] The relatively restricted duration of diabetes in the present simulated cohort may have reduced the ability to demonstrate such an association.

Hypertension and abnormal BMI were significantly more frequent among participants with neuropathy. These findings support the increasingly recognized concept that DPN is not exclusively a consequence of hyperglycaemia. Hypertension, obesity and dyslipidaemia may independently contribute to vascular dysfunction, inflammation, oxidative stress and impaired nerve metabolism.[12,13] A systematic review has specifically identified hypertension as an important potentially modifiable risk factor associated with abnormal nerve conduction and impaired vibration perception in people with diabetes.[13]

The lipid findings provide further support for a multifactorial metabolic contribution. Participants with neuropathy had higher total cholesterol and triglyceride levels and lower HDL concentrations, whereas LDL did not differ significantly. Previous work has demonstrated associations between obesity and hypertriglyceridemia and early diabetic nerve injury, suggesting that metabolic abnormalities may influence small- and large-fibre function through mechanisms extending beyond glucose toxicity.[14] However, lipid associations are not entirely consistent across studies, and the present findings should therefore be regarded as associative rather than causal.

Clinical examination identified abnormalities involving ankle reflexes, touch, temperature, pin-prick and vibration. Such findings emphasize the value of a structured neurological examination in routine diabetes care. Importantly, symptoms alone may not adequately identify nerve dysfunction. Electrophysiological testing provides objective information regarding sensory and motor nerve function and can help characterize the predominant pattern of nerve injury.[15] In the present study, NCS abnormalities were strongly associated with the neuropathy classification, with a mixed pattern being the predominant electrophysiological abnormality. This supports the complementary role of clinical examination and NCS in evaluating DPN.

The association between neuropathy and renal parameters was also noteworthy. Participants with neuropathy had higher serum creatinine and lower eGFR. Renal dysfunction and neuropathy frequently coexist in diabetes and may reflect shared microvascular and metabolic pathways. Recent longitudinal evidence similarly identifies renal and cardiovascular factors among clinically relevant predictors of DPN.[7]

CONCLUSION

Peripheral neuropathy represents an important complication among adults with type 2 diabetes mellitus. The present simulated findings indicate that neuropathy was associated with poorer glycaemic control, hypertension, abnormal BMI, adverse lipid parameters, and altered renal function. Clinical examination identified relevant sensory and reflex abnormalities, while nerve conduction studies provided objective evidence of peripheral nerve dysfunction and helped characterize electrophysiological patterns. These findings support the value of combining clinical neurological assessment with electrophysiological evaluation for comprehensive identification of diabetic neuropathy. Early recognition of neuropathy and attention to modifiable metabolic and cardiovascular risk factors may facilitate timely intervention and potentially reduce future neurological and foot-related complications.

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