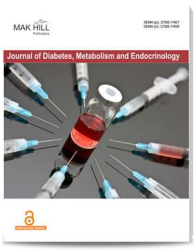

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Research Article

Electrophysiological Evaluation of Diabetic Polyneuropathy in Individuals Aged 40 Years and Above

Sukhajit Bagchi[1*], Serina Khatun[2] and Tamal Chakraborty[3]

1Senior Resident, Department of Physiology, Maharaja Jitendra Narayan Medical College and Hospital, Cooch Behar, India

[2]Assistant Professor, Department of Physiology, Calcutta National Medical College, India

[3]Associate Professor, Department of Physiology, North Bengal Medical College, India

***Corresponding Author**
Dr. Sukhajit Bagchi
(emailsukhajit@gmail.com)

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Abstract: Introduction: Diabetic polyneuropathy (DPN) is one of the important neurological complications of diabetes mellitus and may remain clinically asymptomatic despite demonstrable peripheral nerve dysfunction. Nerve conduction studies provide an objective method for evaluating peripheral nerve involvement and may help identify electrophysiological abnormalities before the development of overt clinical manifestations. **Objectives:** To evaluate nerve conduction study parameters in diabetic individuals aged 40 years and above, determine the prevalence and electrophysiological pattern of diabetic polyneuropathy, and assess the relationship of nerve conduction abnormalities with clinical symptoms, age, duration of diabetes, and fasting blood sugar. **Methodology:** This cross-sectional analytical study was conducted in the Outpatient Departments of Neurology, General Medicine and Endocrinology and the Department of Physiology, Medical College and Hospital, Kolkata, over 18 months from April 2017 to November 2018. A total of 31 confirmed diabetic individuals aged 40 years and above were evaluated. Clinical assessment was performed using the Toronto Clinical Scoring System, along with assessment of sensory, motor and autonomic symptoms. Nerve conduction studies were performed bilaterally on the median, peroneal and sural nerves, assessing latency, amplitude and conduction velocity. Data were analyzed using Microsoft Excel Statistical Package, with $p<0.05$ considered statistically significant. **Results:** The study population comprised 26 males and 5 females, with a mean age of 55.39±9.74 years. DPN was detected in 16 of 31 participants, giving an overall prevalence of 51.61%. Notably, 23 participants (74.19%) were asymptomatic, yet nerve conduction studies detected electrophysiological abnormalities consistent with DPN in 8 of these 23 asymptomatic individuals (34.78%). All 8 symptomatic participants demonstrated DPN. The predominant electrophysiological pattern was mixed sensory-motor axonal polyneuropathy (75%), followed by predominantly motor involvement (18.75%) and pure sensory involvement (6.25%). Significant abnormalities were observed in motor nerve amplitude and conduction velocity, while bilateral sural sensory nerve parameters showed significant differences between DPN and non-DPN groups. Mean fasting blood sugar was significantly higher in the DPN group (184.0±96.69 vs. 133.6±17.07 mg/dL; $p = 0.028$). Duration of diabetes and fasting blood sugar showed negative correlations with sural nerve amplitude, although these were not statistically significant. **Conclusion:** Diabetic polyneuropathy was common among diabetic individuals aged 40 years and above, with a substantial proportion of electrophysiological abnormalities occurring in clinically asymptomatic subjects. The predominant pattern was mixed sensory-motor axonal polyneuropathy, with prominent abnormalities in sural sensory nerve conduction parameters. Nerve conduction studies may therefore be valuable for objective detection of diabetic polyneuropathy, particularly in asymptomatic or minimally symptomatic patients. Larger studies are required to further substantiate these findings.

Keywords: Diabetic Polyneuropathy, Diabetes Mellitus, Nerve Conduction Studies, Nerve Conduction Velocity, Sural Nerve, Peripheral Neuropathy, Asymptomatic Neuropathy

INTRODUCTION

Diabetes mellitus is a progressive metabolic disorder associated with multiple systemic complications and represents an increasing global health burden. The available data cited in the study indicate that diabetes affected approximately 415 million people worldwide, corresponding to about one in 11 adults, with a substantial proportion remaining undiagnosed. The number of people with diabetes was projected to increase further, reaching approximately 642 million by 2040. In India, diabetes has similarly assumed epidemic proportions, with more than 62 million diagnosed individuals and a projected increase to 79.4 million by 2030 [1,2].

The complications of diabetes involve multiple organ systems and are broadly categorized into macrovascular and microvascular complications. Neuropathy represents one of the important microvascular complications, and diabetic neuropathies may be serious and disabling. Diabetic polyneuropathy (DPN) occurs in approximately 50% of individuals with type 1 and type 2 diabetes, with distal symmetric polyneuropathy being the commonest form. DPN is a chronic

distal symmetric sensorimotor polyneuropathy and contributes substantially to morbidity through foot ulceration, lower-limb amputation, pain, impaired quality of life, reduced ability to perform activities of daily living, and increased susceptibility to falls and fractures [3, 4].

Diabetic polyneuropathy may involve motor, sensory and autonomic nerve fibres. Motor involvement may manifest as proximal muscle atrophy and weakness, while sensory involvement may produce diminished perception of touch, pain, temperature, position and vibration. Autonomic involvement may result in abnormalities such as loss of sweating, gastroparesis and cardiac dysrhythmia. The resulting impairment may progress to the characteristic "glove and stocking" distribution, Charcot neuropathy, foot ulceration, gangrene and amputation. Neural damage has been associated with impaired fasting glucose, impaired glucose tolerance and subsequent hyperglycaemia. The neuropathy is also described as length dependent, with the longest fibres of the lower limbs being affected first [3].

The development of diabetic polyneuropathy is related to several clinical factors, including age, duration of diabetes and glycaemic control, with increased body mass index and cardiovascular risk factors also described as contributing factors. In the Indian setting, delayed detection and inadequate glycaemic control may further increase the risk of neurological complications. The study population was specifically selected from diabetic individuals aged 40 years and above because middle age represents an important period in which many Indian patients present for diagnosis and treatment, after potentially prolonged periods of pre-diabetes or overt diabetes [3, 5].

An important characteristic of DPN is that neurological dysfunction may develop before clinically appreciable symptoms or signs become evident. Consequently, assessment based only on symptoms may have limited value for detecting early neuropathy. The study background emphasizes that DPN may remain asymptomatic and that electrophysiological assessment provides quantitative information regarding peripheral nerve dysfunction. Nerve conduction studies (NCS) are sensitive, specific and reproducible methods for assessing the presence and severity of peripheral neuropathy and can provide quantitative evidence of nerve dysfunction [6,7].

Nerve conduction velocity remains relatively stable during adulthood but begins to decline gradually with advancing age. This age-related change is particularly relevant when evaluating older diabetic individuals, since age itself may influence nerve conduction parameters. The study background notes that conduction velocity remains stable through early adulthood and subsequently shows a gradual decline, while the reduction is relatively limited even in advanced age [8, 9].

NCS evaluates parameters including latency, amplitude and conduction velocity and can identify abnormalities affecting axonal or myelin function. Reduction in motor and sensory amplitudes is particularly relevant to axonal neuropathy, while slowing of conduction velocity may accompany the electrophysiological abnormalities. The study specifically emphasized that reduced amplitude may occur along with reduced conduction velocity before clinically appreciable manifestations of polyneuropathy.

The selection of appropriate nerves is important in electrophysiological assessment of diabetic neuropathy. Upper-limb nerves may be relatively less affected and may also be influenced by entrapment at the wrist or neck. Lower-limb nerves are therefore important in the assessment of DPN. In the present work, the median nerves of both upper limbs and the peroneal and sural nerves of both lower limbs were evaluated. The sural nerve is particularly relevant because it is exclusively sensory and is considered to be affected early in various pathological conditions associated with DPN.
Previous observations cited in the study have also demonstrated the importance of age and sex in interpreting nerve conduction parameters. Falco et al. reported a significant effect of age on nerve conduction values in healthy elderly individuals, while Karnain et al. demonstrated gender-related differences in upper-limb nerve conduction parameters among healthy individuals from North India [9,10].

Thus, the assessment of nerve conduction parameters in diabetic individuals aged 40 years and above is important for identifying electrophysiological abnormalities that may not be apparent clinically. The present study was undertaken to evaluate motor and sensory nerve conduction parameters of the bilateral median, peroneal and sural nerves in diabetic individuals aged 40 years and above, to estimate the occurrence of diabetic polyneuropathy and to examine its relationship with age, duration of diabetes and the presence or absence of neuropathic symptoms. The study also sought to determine whether NCS could identify electrophysiological abnormalities among apparently asymptomatic diabetic individuals.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted in the Outpatient Departments of Neurology, General Medicine and Endocrinology and in the Department of Physiology, Medical College and Hospital, Kolkata, over 18 months. Data collection was undertaken from April 2017 to March 2018, followed by tabulation from April to October 2018 and data analysis in November 2018. The study protocol was reviewed and approved by the Institutional Ethics Committee, Medical College, Kolkata (Ref. No. MC/Kol/IEC/Non-spon/553/03-2017; dated 29 April 2017), and the study was conducted after acceptance of the protocol. Written informed consent was obtained from all participants before enrolment.

The study population comprised confirmed diabetic patients aged 40 years and above, recruited randomly using a computer-generated random number table from the General OPD, Diabetic OPD and Neurology OPD. Patients with type 1 or type 2 diabetes were eligible when diabetes was confirmed by OPD diagnosis, treatment history and documentary evidence of fasting and postprandial hyperglycaemia. Patients were excluded when clinical assessment and relevant investigations indicated other causes of neuropathy, including infectious or inflammatory disorders such as leprosy, HIV, hepatitis C, Guillain-Barré syndrome, chronic inflammatory demyelinating polyneuropathy and primary biliary cirrhosis; vascular disorders including peripheral vascular disease and necrotizing vasculitis; endocrine disorders including hypothyroidism and acromegaly; metabolic disorders including hepatic cirrhosis, chronic renal failure and amyloidosis; deficiency states involving vitamin B or vitamin E; exposure to antineoplastic or antiretroviral therapy or ethanol; and miscellaneous conditions including carpal tunnel syndrome, lumbar spondylosis and limb amputation. Patients at the extremes of age were also excluded. No control group was included.

A sample size of 31 subjects was obtained using the study prevalence estimate of diabetic polyneuropathy of 27.4%, with 95% confidence, 80% statistical power and the specified difference between affected and non-affected groups. The variables assessed included age, sex, duration of diabetes, fasting blood sugar, postprandial blood sugar and nerve conduction parameters. Nerve conduction studies evaluated amplitude, latency and conduction velocity of the motor and sensory modalities of the bilateral median nerves, bilateral peroneal nerves and bilateral sural nerves. Blood sugar values were obtained from the respective outpatient departments.

Clinical assessment of neuropathy included a structured history of sensory, motor and autonomic symptoms, including paresthesia, pricking, tingling, pins and needles, burning, pain, numbness, sleep disturbance, allodynia, joint movement difficulty, falls, palpitations, loss of sweating, altered bowel movements and erectile dysfunction. The Toronto Clinical Scoring System (TCSS) was used to assess neuropathic symptoms and signs. Sensory function was evaluated using a 10-g Semmes-Weinstein monofilament and vibration sense with a 128-Hz tuning fork; tendon reflexes and other clinical findings were also assessed. The TCSS score ranged from 0 to 19 and was categorized as no neuropathy (0–5), mild neuropathy (6–8), moderate neuropathy (9–11) and severe neuropathy (≥12). For the present analysis, a score above 5 was considered symptomatic neuropathy.

Nerve conduction studies were performed using the RMS ALERON 201-2CH system. Participants were examined in the supine position in a physically and mentally relaxed state. After locating the nerve, the skin was cleaned with spirit and recording electrodes were attached using conducting paste. Stimulating electrodes were positioned at predetermined anatomical sites. A brief electrical stimulus of 60 Hz with a duration of 0.1 ms was delivered, and the evoked response was displayed and printed for analysis. Motor nerves were stimulated at two points along their course using supramaximal stimulation, with recording over the appropriate motor point. Compound muscle action potentials were recorded. For sensory studies, antidromic conduction was used. Median motor conduction was assessed by stimulation at the wrist and elbow with recording over the abductor pollicis brevis, while sensory conduction was assessed by wrist stimulation with recording over the second or third digits. Peroneal motor conduction was recorded from the extensor digitorum brevis with stimulation at the ankle and fibular neck. Sural sensory conduction was assessed by stimulation at the lower border of the gastrocnemius and recording between the lateral malleolus and tendo Achillis.

Motor conduction velocity was calculated as the distance between the two stimulation sites divided by the difference between proximal and distal latencies. Sensory conduction velocity was calculated by dividing the distance between the stimulating and recording electrodes by the latency period.

Data were compiled and statistically analysed using Microsoft Excel Statistical Package. Continuous variables were summarized using mean, median, mode, standard deviation and standard error, while categorical variables were expressed as frequencies and proportions. Comparisons between DPN and non-DPN, symptomatic and asymptomatic, and male and female groups were performed using appropriate statistical tests, including unpaired Student's t-test and chi-square test. Pearson correlation, linear regression and multiple regression analyses were used to examine relationships between nerve conduction parameters and demographic or clinical variables. A p-value <0.05 was considered statistically significant, while $p<0.01$ was considered highly statistically significant.

RESULTS

A total of 31 diabetic subjects aged 40 years and above were evaluated by clinical assessment and nerve conduction studies. The study population had a mean age of 55.39 ± 9.74 years, mean body weight of 57.79 ± 8.09 kg, mean duration of diabetes of 6.45 ± 4.92 years, and mean fasting blood sugar (FBS) of 159.61 ± 73.93 mg/dL. The corresponding median values were 55 years, 57 kg, 6 years and 132 mg/dL, respectively. The study population therefore showed a broad distribution of age, duration of diabetes and fasting blood glucose values. The thesis records these variables as normally distributed (Table 1).

Of the 31 subjects, 16 (51.61%) were classified as having diabetic polyneuropathy (DPN) and 15 (48.39%) were classified as non-DPN. Eight of the 31 subjects were symptomatic according to the Toronto Clinical Scoring System

(TCSS), while 23 (74.19%) were asymptomatic. Among the symptomatic subjects, all 8 had DPN. Importantly, among the 23 asymptomatic subjects, 8 demonstrated electrophysiological abnormalities consistent with DPN, corresponding to 34.78% of the asymptomatic subgroup. Thus, a substantial proportion of electrophysiologically detected neuropathy occurred in subjects without clinically evident neuropathic symptoms (Table 2).

The study population consisted of 26 males and 5 females. DPN was identified in 16 males and none of the female participants. The analysis reported a statistically significant difference in the distribution of neuropathy between the sexes ($p = 0.00199$). In contrast, the distribution of symptomatic and asymptomatic status did not differ significantly by sex on chi-square analysis ($p = 0.14987$). Assessment by TCSS demonstrated that 23 subjects (74.19%) were in the asymptomatic category with scores of 0–5. Three subjects had mild neuropathy scores (6–8), two had moderate scores (9–11), and three had severe scores (>12). Thus, the majority of the study population had no clinically appreciable neuropathic symptoms despite being diabetic (Table 3).

Electrophysiological comparison between DPN and non-DPN groups

Motor nerve conduction studies demonstrated clear differences between DPN and non-DPN subjects. In the right median nerve, the mean proximal latency was higher in the DPN group than in the non-DPN group (9.04 ± 1.95 vs. 7.91 ± 0.99), whereas the mean amplitude was substantially lower (5.94 ± 2.46 vs. 9.60 ± 2.26) and conduction velocity was lower (49.16 ± 6.46 vs. 56.03 ± 2.26). The differences in proximal latency, amplitude and conduction velocity were statistically significant, with p-values of 0.0273, <0.001 and <0.001, respectively; distal latency did not differ significantly.

A similar pattern was observed in the right peroneal motor nerve. The DPN group showed markedly lower amplitude (1.49 ± 1.78 vs. 4.17 ± 1.77) and conduction velocity (27.18 ± 19.72 vs. 47.62 ± 2.62) compared with the non-DPN group. Both differences were highly statistically significant ($p = 0.0001185$ and $p = 0.0002045$, respectively), whereas proximal and distal latencies did not show statistically significant differences (Table 4).

The sensory nerve studies demonstrated particularly marked abnormalities in the sural nerve. In the right sural nerve, DPN subjects had markedly reduced distal latency, amplitude and conduction velocity compared with non-DPN subjects. The mean amplitude was 4.03 ± 7.71 in DPN compared with 23.81 ± 11.58 in non-DPN subjects, while conduction velocity was 16.31 ± 30.12 compared with 70.79 ± 12.01, respectively. All three parameters showed highly significant differences.

The same pattern was observed in the left sural nerve, where DPN subjects had lower distal latency, amplitude and conduction velocity compared with non-DPN subjects. The differences were statistically significant for all three parameters, with p-values of <0.001 (Table 5).

Relationship of DPN with age, weight, duration of diabetes and fasting blood sugar

When clinical and metabolic parameters were compared between the DPN and non-DPN groups, the mean age was 55.75 ± 9.59 years in the DPN group and 55.00 ± 10.22 years in the non-DPN group ($p = 0.417244$). Mean body weight was 56.75 ± 6.07 kg and 58.90 ± 9.90 kg, respectively ($p = 0.234407$). Mean duration of diabetes was longer among DPN subjects (7.31 ± 4.87 years) than non-DPN subjects (5.53 ± 4.97 years), although the difference was not statistically significant ($p = 0.161187$). In contrast, fasting blood sugar was significantly higher in the DPN group (184.0 ± 96.69 mg/dL vs. 133.6 ± 17.07 mg/dL; $p = 0.028171$) (Table 6).

Correlation analysis of right sural amplitude showed weak associations with age, weight, duration of diabetes and FBS. For right sural amplitude, the correlation coefficients were 0.005 for age, 0.284 for weight, −0.332 for duration of diabetes and −0.380 for FBS; none reached statistical significance. A similar pattern was observed for left sural amplitude, with r values of 0.0117, 0.193, −0.329 and −0.361 for age, weight, duration of diabetes and FBS, respectively. The negative correlations of amplitude with duration of diabetes and FBS were therefore not statistically significant in the present sample (Figure 1).

The analysis also reported an ANOVA comparing age, weight, duration of diabetes and FBS among the DPN cases. The overall ANOVA was highly significant ($F = 38.53151$, $p = 5.23 \times 10^{-14}$), indicating significant variation among the four parameter groups; however, this analysis should be interpreted cautiously because the variables are measured on different scales and are not independent clinical groups (Table 7).

Table 1: Baseline Characteristics of the Study Population

Parameter	Mean	Median	Mode	Standard deviation	Standard error
Age (years)	55.39	55	50	9.74	1.75
Weight (kg)	57.79	57	57	8.09	1.45
Duration of diabetes (years)	6.45	6	2	4.92	0.88
Fasting blood sugar (mg/dL)	159.61	132	130	73.93	13.28

Table 2: Distribution of Diabetic Polyneuropathy According to Symptoms and Sex

Group	Symptomatic	Asymptomatic	Total
DPN	8 (50.00%)	8 (50.00%)	16 (100.00%)
Non-DPN	-	15 (100.00%)	15 (100.00%)
Total	8 (25.80%)	23 (74.2%)	31 (100.00%)
Sex	DPN	Non-DPN	Total
Male	16 (61.54%)	10 (38.46%)	26 (100.00%)
Female	-	5 (100.00%)	5 (100.00%)
Total	16 (51.61%)	15 (48.39%)	31 (100.00%)

Table 3: Distribution of Subjects According to Toronto Clinical Scoring System (TCSS)

TCSS category	TCSS score	Number of subjects	Percentage
Asymptomatic	0–5	23	74.19%
Mild	6–8	3	9.68%
Moderate	9–11	2	6.45%
Severe	>12	3	9.68%
Total	—	31	100%

Table 4: Comparison of Selected Motor Nerve Conduction Parameters Between DPN and non-DPN groups

Nerve and parameter	DPN	Non-DPN	p-value
Right median – Proximal latency	9.04 ± 1.95	7.91 ± 0.99	0.027284
Right median – Distal latency	4.19 ± 1.30	3.79 ± 1.02	0.172599
Right median – Amplitude	5.94 ± 2.46	9.60 ± 2.26	0.000087
Right median – Velocity	49.16 ± 6.46	56.03 ± 2.26	0.000260
Right peroneal – Proximal latency	8.98 ± 6.64	10.51 ± 0.75	0.190638
Right peroneal – Distal latency	3.06 ± 2.35	3.51 ± 0.59	0.238084
Right peroneal – Amplitude	1.49 ± 1.78	4.17 ± 1.77	0.000119
Right peroneal – Velocity	27.18 ± 19.72	47.62 ± 2.62	0.000204

Table 5: Comparison of sensory nerve conduction parameters between DPN and non-DPN groups

Nerve and parameter	DPN	Non-DPN	p-value
Right median – Distal latency	2.26 ± 1.29	2.90 ± 0.69	0.050196
Right median – Amplitude	27.29 ± 18.67	43.50 ± 19.28	0.003368
Right median – Velocity	45.48 ± 25.35	41.50 ± 9.96	0.178755
Right sural – Distal latency	0.52 ± 0.95	1.74 ± 0.34	0.000030
Right sural – Amplitude	4.03 ± 7.71	23.81 ± 11.58	0.000002
Right sural – Velocity	16.31 ± 30.12	70.79 ± 12.01	<0.000001
Left sural – Distal latency	0.52 ± 0.95	2.05 ± 0.29	<0.000001
Left sural – Amplitude	4.28 ± 8.96	24.35 ± 11.63	<0.000005
Left sural – Velocity	16.05 ± 29.28	59.23 ± 7.35	<0.000003

Table 6: Comparison of demographic and metabolic parameters between DPN and non-DPN groups

Parameter	DPN	Non-DPN	p-value
Age (years)	55.75 ± 9.59	55.00 ± 10.22	0.417244
Weight (kg)	56.75 ± 6.07	58.90 ± 9.90	0.234407
Duration of diabetes (years)	7.31 ± 4.87	5.53 ± 4.97	0.161187
Fasting blood sugar (mg/dL)	184.00 ± 96.69	133.60 ± 17.07	0.028171

Table 7: Association of demographic and metabolic parameters with diabetic polyneuropathy and sural nerve amplitude

Analysis / Parameter	N	Mean	Variance	r-value	r^2-value	p-value
ANOVA among DPN cases						
Age (years)	16	55.75	91.933	—	—	—
Weight (kg)	16	56.75	36.867	—	—	—
Duration of diabetes (years)	16	7.31	23.696	—	—	—
Fasting blood sugar (mg/dL)	16	184.00	9349.733	—	—	—
Overall ANOVA	64 observations	—	—	—	—	5.23×10^{-14}
Correlation with right sural nerve amplitude						
Age (years)	16	—	—	0.005	0.000025	0.990
Weight (kg)	16	—	—	0.284	0.081	0.290
Duration of diabetes (years)	16	—	—	−0.332	0.110	0.210
Fasting blood sugar (mg/dL)	16	—	—	−0.380	0.144	0.147
Correlation with left sural nerve amplitude						
Age (years)	16	—	—	0.0117	0.0001	0.966
Weight (kg)	16	—	—	0.193	0.037	0.475
Duration of diabetes (years)	16	—	—	−0.329	0.108	0.214
Fasting blood sugar (mg/dL)	16	—	—	−0.361	0.130	0.170

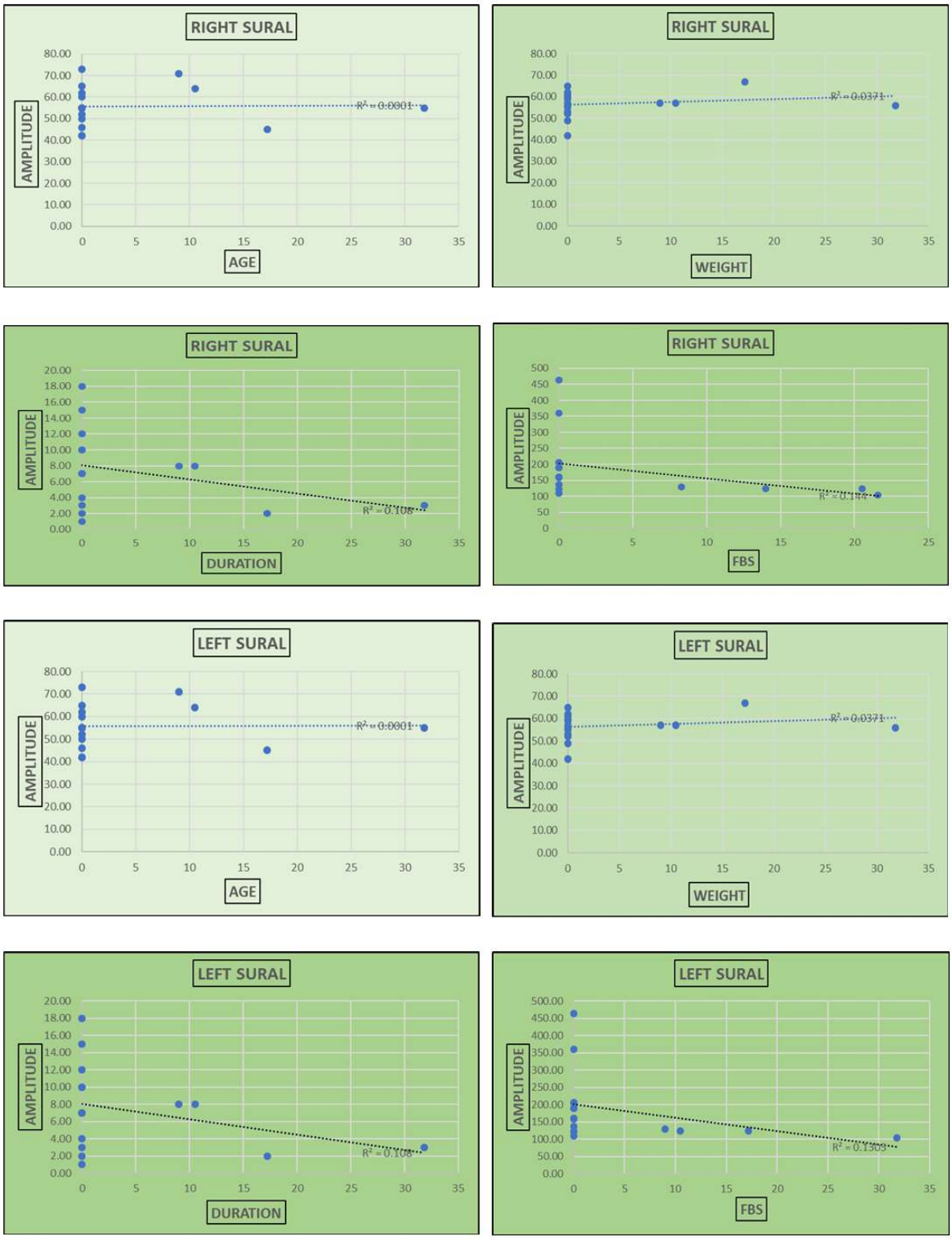

Figure 1: Graphically shows the Value of Tables

DISCUSSION

The present study evaluated electrophysiological abnormalities among 31 confirmed diabetic individuals aged 40 years and above. The study focused on the prevalence of diabetic polyneuropathy (DPN), the relationship between electrophysiological abnormalities and clinical symptoms, and the association of nerve conduction parameters with age,

weight, duration of diabetes and fasting blood sugar. Among the 31 participants, 26 were males and 5 were females. Sixteen subjects were classified as having DPN, giving an overall prevalence of 51.61%. The age of the participants ranged from 42 to 73 years, with a greater concentration in the younger part of the studied age range [11].

The prevalence of DPN observed in the present study indicates a considerable burden of peripheral nerve involvement among diabetic individuals aged 40 years and above. The findings are clinically important because diabetic neuropathy may develop progressively and may remain clinically silent for a considerable period. In the present study, 23 of 31 participants (74.19%) were asymptomatic according to the Toronto Clinical Scoring System (TCSS), while 8 were symptomatic. Electrophysiological abnormalities were detected in all 8 symptomatic subjects and in an additional 8 of the 23 asymptomatic subjects. Thus, 34.78% of the clinically asymptomatic participants had electrophysiologically detectable DPN. This finding demonstrates that the absence of neuropathic symptoms does not necessarily exclude peripheral nerve involvement and supports the importance of objective electrophysiological evaluation in diabetic patients [12].

The electrophysiological pattern in the present study was predominantly mixed sensory-motor axonal polyneuropathy, which was observed in 12 of the 16 DPN cases (75%). Three cases showed predominantly motor axonal involvement and one case showed pure sensory involvement. The predominance of axonal involvement is consistent with the description of DPN as a neuropathy in which loss of nerve fibres is a major pathological feature. The study findings further showed that reduction in nerve amplitude was an important electrophysiological abnormality. In particular, amplitude reduction was observed across the nerves examined and was associated with the presence of DPN. This pattern is compatible with the principles of electrodiagnostic interpretation described by Falck and Stålberg, in which the amplitude of the evoked response provides important information regarding the number of functioning nerve fibres [13].

The present study demonstrated significant abnormalities in both motor and sensory nerve conduction parameters. In the right median nerve, the DPN group showed a lower mean amplitude and conduction velocity compared with the non-DPN group, while proximal latency was also significantly different. In the right peroneal nerve, amplitude and conduction velocity were significantly reduced in DPN. These findings indicate that diabetic peripheral nerve involvement was not restricted to a single nerve or a single limb. The involvement of both upper- and lower-limb nerves also supports the diffuse nature of diabetic polyneuropathy. Normative studies by Hennessey *et al*. [11] and Pawar *et al.* [12] have demonstrated the importance of establishing appropriate reference values for interpretation of upper-limb nerve conduction parameters, while Chouhan reported normative motor conduction characteristics of the median and common peroneal nerves [14-17].

The sensory nerve findings were particularly prominent in the sural nerves. In the right sural nerve, distal latency, amplitude and conduction velocity were significantly different between the DPN and non-DPN groups. The same pattern was observed in the left sural nerve, with all three parameters showing highly significant differences. The mean left sural amplitude was 4.28 ± 8.96 in the DPN group compared with 24.35 ± 11.63 in the non-DPN group, while conduction velocity was 16.05 ± 29.28 compared with 59.23 ± 7.35, respectively. These findings indicate substantial sensory nerve involvement in DPN. The sural nerve findings are particularly relevant because sensory nerve action potential abnormalities can provide objective evidence of peripheral nerve dysfunction even when clinical manifestations are limited.

A similar difference was demonstrated when symptomatic and asymptomatic participants were compared. In the left sural nerve, distal latency, amplitude and conduction velocity were all significantly different between symptomatic and asymptomatic subjects, with p-values of 0.0135, 0.0059 and 0.0069, respectively. Although the asymptomatic group had fewer clinical manifestations, their nerve conduction parameters were nevertheless abnormal in a proportion of subjects. This finding reinforces the observation that electrophysiological abnormalities may precede clinically evident neuropathy. The usefulness of nerve conduction studies for objectively demonstrating peripheral nerve dysfunction has been emphasized in electrodiagnostic literature, including the work of Kimura [8] and Falck and Stålberg [18].

The marked reduction in amplitude compared with the relatively less consistent changes in latency and conduction velocity is also noteworthy. The present study reported approximately 50% reduction in amplitude in the examined nerves according to the applied criteria. Multiple-nerve involvement was found in several subjects, with some demonstrating abnormalities in four nerves, while others had involvement of three, two or a single nerve. The overall pattern was therefore heterogeneous in distribution but predominantly axonal in character. This finding is consistent with the electrophysiological interpretation used in the study, wherein a markedly reduced amplitude with relatively preserved latency and only borderline slowing of conduction velocity favours axonal loss, whereas conduction slowing with preserved amplitude and latency is more suggestive of demyelination.

Age is an important factor when interpreting nerve conduction studies. In the present study, the mean age of the DPN group was 55.75 ± 9.59 years compared with 55.00 ± 10.22 years in the non-DPN group, and the difference was not statistically significant ($p = 0.417244$). Thus, within this relatively small cohort, age alone did not distinguish the DPN from the non-DPN group. Nevertheless, age-related variation in nerve conduction parameters must be considered

when interpreting electrophysiological findings. Falco *et al.* [9] reported age-related effects on nerve conduction values in healthy elderly individuals, while Karnain *et al.* [10] demonstrated that demographic factors such as sex can also influence upper-limb nerve conduction parameters. These observations emphasize the importance of considering physiological variation while assessing pathological changes in diabetic patients.

Duration of diabetes was higher in the DPN group than in the non-DPN group, with mean durations of 7.31 ± 4.87 years and 5.53 ± 4.97 years, respectively. However, this difference did not reach statistical significance ($p = 0.161187$). Although the present sample did not demonstrate a statistically significant group difference, the direction of the finding is compatible with the clinical concept that longer exposure to diabetes may be associated with greater peripheral nerve involvement. The correlation analysis also showed a negative relationship between duration of diabetes and sural nerve amplitude, with r values of −0.332 for the right sural nerve and −0.329 for the left sural nerve. These correlations were not statistically significant, but the direction of the association suggests that increasing duration of diabetes may be accompanied by reduction in sensory nerve amplitude.

Fasting blood sugar showed a more definite association with DPN. The mean fasting blood sugar was 184.0 ± 96.69 mg/dL in the DPN group compared with 133.6 ± 17.07 mg/dL in the non-DPN group, and this difference was statistically significant ($p = 0.028171$). Furthermore, fasting blood sugar demonstrated a negative correlation with sural nerve amplitude, with $r = −0.380$ for the right sural nerve and $r = −0.361$ for the left sural nerve. Although these individual correlations did not reach statistical significance, their consistent negative direction suggests a relationship between higher fasting glycaemia and reduced sensory nerve amplitude. The findings therefore support the importance of glycaemic status in relation to peripheral nerve dysfunction.

The overall statistical analysis further demonstrated significant variation among age, weight, duration of diabetes and fasting blood sugar within the DPN group, with the ANOVA yielding $F = 38.53151$ and $p = 5.23 \times 10^{-14}$. However, because these variables represent different clinical characteristics rather than independent clinical groups, this overall ANOVA should primarily be regarded as demonstrating variation among the measured parameters rather than as establishing a direct causal relationship between them. The individual group comparison provides the more clinically interpretable finding that fasting blood sugar differed significantly between DPN and non-DPN subjects.

A significant association was also observed between sex and DPN, with DPN identified in all 16 affected subjects being male and none of the 5 female participants. The statistical test demonstrated a highly significant difference. However, this finding should be interpreted cautiously because of the marked imbalance in the sex distribution and the very small number of female participants. Sex-related differences in nerve conduction parameters have previously been described by Karnain et al.[10], while normative studies by Hennessey et al. [11] and Salerno *et al.* [18] have also emphasized the importance of demographic characteristics when interpreting nerve conduction measurements.

An important clinical observation of the present study was that the presence of symptoms did not provide a complete representation of electrophysiological nerve involvement. Although the majority of the study population, 23 out of 31 participants (74.19%), remained asymptomatic, nerve conduction studies detected electrophysiological abnormalities consistent with DPN in 8 of these 23 asymptomatic individuals (34.78%). Although all symptomatic subjects had DPN, the detection of DPN in more than one-third of the asymptomatic population highlights the ability of nerve conduction studies to identify clinically silent peripheral nerve dysfunction. The association between symptomatic status and neuropathy was statistically significant ($p = 0.001475714$); however, these findings clearly demonstrate that symptom-based assessment alone may fail to identify a proportion of affected individuals. This is particularly relevant in diabetic patients because electrophysiological abnormalities may be present before patients recognize or report neuropathic symptoms [19, 20].

CONCLUSION

The present study evaluated nerve conduction parameters among diabetic individuals aged 40 years and above and demonstrated a substantial burden of electrophysiologically detectable diabetic polyneuropathy. Among the 31 participants evaluated, 16 were identified as having DPN, giving an overall prevalence of 51.61%. The predominant electrophysiological pattern was mixed sensory-motor axonal polyneuropathy, observed in 12 of the 16 affected subjects (75%), while 3 had predominantly motor axonal involvement (18.75%) and 1 had pure sensory involvement (6.25%). Reduction in nerve conduction amplitudes was a prominent finding, supporting the predominance of axonal involvement in the study population.

The sensory nerve abnormalities were particularly prominent in the bilateral sural nerves, with significant differences in distal latency, amplitude and conduction velocity between DPN and non-DPN groups. Motor abnormalities were also demonstrated, particularly involving the median and peroneal nerves. Fasting blood sugar was significantly higher among subjects with DPN than among those without DPN (184.0±96.69 vs. 133.6±17.07 mg/dL; $p = 0.028171$). Duration of diabetes and fasting blood sugar showed negative correlations with right and left sural nerve amplitude; however, these individual correlations did not reach statistical significance.

Limitations

The study has several limitations that should be considered while interpreting its findings. First, the sample size was relatively small, comprising only 31 diabetic participants. This limits the statistical power of subgroup analyses and restricts the generalizability of the findings to the broader diabetic population.

Second, there was a marked imbalance in sex distribution, with 26 male and only 5 female participants. Consequently, although a statistically significant association between sex and DPN was observed, the small number of female participants makes it difficult to draw reliable conclusions regarding sex-related differences in the prevalence or electrophysiological characteristics of DPN.

Third, a technical breakdown of the RMS ALERON nerve conduction machine toward the end of the study resulted in loss of some data. In addition, complete recordings from all intended nerves could not always be obtained because of fear or non-cooperation among some participants. These factors may have reduced the completeness of the electrophysiological dataset.

Finally, nerve conduction studies primarily assess large myelinated peripheral nerve fibres and may not detect abnormalities involving unmyelinated fibres or very mild neuropathy. Therefore, normal or borderline nerve conduction findings cannot completely exclude early diabetic neuropathy. Larger studies with more balanced participant characteristics and more comprehensive electrophysiological assessment would be required to validate and extend the present findings.

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