Section: General Medicine

Original Research Article

TO STUDY THE CORRELATION BETWEEN ELECTROPHYSIOLOGICAL CHANGES OF DIABETIC PERIPHERAL NEUROPATHY AND DURATION OF DIABETES IN ASYMPTOMATIC DIABETIC PATIENTS

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ABSTRACT

Background: Diabetic peripheral neuropathy (DPN) is the commonest chronic complications of diabetes mellitus (DM). Early identification of DPN is vital, as prompt glycaemic and risk factor management can slow progression and prevent irreversible nerve damage. As asymptomatic patients, are underrepresented in neuropathy screening programs, understanding the electrophysiological changes in these patients can provide an opportunity for early diagnosis, intervention, and prevention of long-term disability. **Aim:** To study the electrophysiological changes in asymptomatic diabetic patients and to correlate them with the duration of diabetes. **Objectives** 1. To look for the effect of the duration of diabetes on nerve conduction studies in asymptomatic diabetic patients. 2. To look for early changes in nerve conduction in common peroneal, posterior tibial, and sural nerves in patients not exhibiting symptoms of diabetic neuropathy.

Materials and Methods: Cross-sectional study was done in 100 asymptomatic DM patients attending general medicine department. After obtaining institutional ethical committee clearance (IHEC/ ECR/300/inst/AP/2013/RR-24) and patient's informed consent data on detailed medical history, comprehensive clinical examination and nerve conduction studies (NCS) (common peroneal, posterior tibial and sural nerve) were recorded. Statistical analysis was done using independent sample t test, chi-square, multinomial logistic regression. $P < 0.05$ was considered as statistically significant.

Results: A statistically significant association was observed between duration of diabetes and presence of neuropathy ($\chi^2 = 22.4$, $p = 0.0016$), with prevalence increasing from 19% in those with ≤5 years of diabetes to 71% in those with >10 years. Progressive worsening was evident in all three nerves examined. multivariate logistic regression shows, duration of diabetes (OR = 1.42, $p < 0.001$) and HbA1c levels (OR = 1.98, $p = 0.005$) as significant independent predictors.

Conclusion: NCS coupled with tight glycemic control, may help in delaying or preventing the onset of subclinical diabetic peripheral neuropathy.

Keywords: Diabetic peripheral neuropathy, correlation, electrophysiological changes, asymptomatic, duration, Diabetes Mellitus type II.

INTRODUCTION

Diabetic peripheral neuropathy (DPN) is a chronic complication in patients with Diabetes mellitus characterised by progressive loss of nerve function.[1,2] DPN causes significant morbidity by causing impaired gait, decreased sensory perception and raising the risk of foot ulcerarion.[3,4] Unlike symptomatic neuropathy which is readily recognised on clinical examination, asymptomatic DPN poses a hidden challenge, with underlying nerve injury progressing unnoticed until significant complications develop.[5] Hence, early identification of DPN is essential, which may lead to glycaemic control and management of associated risk factors. Thus, delaying disease progression and minimizing permanent nerve damage.[6]

Recent estimates in India shows about 77 million people living with diabetes, which may increase further in the future.[7] Consequently, increasing the prevalence of DPN which can impose a considerable burden on our health care systems, with many people who may have subclinical DPN goes unnoticed till they reach clinical stage, leading to morbidity status.[8] As asymptomatic patients are not represented in neuropathy screening programs, thus assessing electrophysiological changes in them, will be an opportunity to diagnose, intervene and prevent long term disability.[9-11]

In India, especially in Telangana, there is a lack of published evidence showing the association of duration of diabetes mellitus and electrophysiological changes in asymptomatic patients. Therefore, this study seeks to address this knowledge gap by generating region-specific evidence that may contribute to the development of appropriate local screening strategies.

Thus, the present study is designed to investigate the relationship between the duration of diabetes and electrophysiological indicators of peripheral neuropathy in asymptomatic individuals.

Aims and Objectives

Aim

1. To study the electrophysiological changes in asymptomatic diabetic patients and to correlate them with the duration of diabetes.

Objectives

1. To determine for the effect of the duration of diabetes on nerve conduction studies in
2. asymptomatic diabetic patients.
3. To Identify for early changes in nerve conduction in common peroneal, posterior tibial, and sural nerves in asymptomatic patients of diabetic neuropathy.

MATERIALS AND METHODS

A cross-sectional observational study conducted over a period of 18 months in the inpatient and outpatient departments of General Medicine at a tertiary care teaching hospital. Asymptomatic patients with Type 2 Diabetes Mellitus (T2DM), attending tertiary care hospital were the study population selected based on the following selection criteria.

Inclusion Criteria

- Patients aged 18 to 60 years of either gender
- Diagnosed with Type 2 Diabetes Mellitus
- HbA1c levels between 6.5% and 7.5%
- Absence of clinical symptoms of diabetic peripheral neuropathy

Exclusion Criteria

- Type 1 Diabetes Mellitus
- Symptomatic T2DM patients
- Known cases of HIV infection
- Malignancy
- Chronic alcohol use
- Diagnosed cases of inherited neuropathies
- Lumbar or cervical radiculopathy
- Nutritional deficiencies (e.g., Vitamin B12 deficiency)
- Hypothyroidism

Sampling size calculation: Sample size was calculated using formula for finite population. Where, Z α is the standard normal deviate, 1.96 at 95% confidence interval.

As per study by Gao L et al prevalence of subclinical DPN in asymptomatic diabetes mellitus patients was 47.5% [5].

Hence P = Prevalence is 47.5%. i.e P = 0.475, 1-P = (1-0.475)

e = allowable error was 10%

N = study population (Patients with type 2 DM who were asymptomatic attending OPD in the institution in the past 6 months) = 2543,

Sample size(n)= ((z^2 X p(1-p))/e^2)/(1+(z^2 X p(1-p))/(e^2 N))

Sample size(n)= (((1.96)〗 ^2 X 0.475(1-0.475))/〖(0.10)〗 ^2)/(1+(〖(1.96)〗 ^2 X 0.475(1-0.475))/((〖0.1)〗 ^2 2543))

Sample size(n)required was=93, Which was rounded up to 100.

Thus 100 patients meeting the inclusion and exclusion criteria, were enrolled in to the study by purposive sampling method.

Data collection and procedure: Data collection was done after obtaining institutional ethical committee clearance (IHEC/ ECR/300/inst/AP/2013/RR-24) and patient's informed consent. Patients who met the selection criteria were included in the study and data collected using a semi structured questionnaire which has the following:

1. Demographic and medical history in detail which includes neuropathic symptoms to rule out DPN.
2. Detailed findings on clinical examination, vital signs, systemic examination and findings and also assessment on glycaemic status.
3. findings of Nerve conduction studies (NCS) of the lower limbs. Peripheral nerves evaluated were:

- Common peroneal nerve (motor)

- Posterior tibial nerve (motor)
- Sural nerve (sensory)

NCS was conducted using standard surface electrodes and percutaneous stimulation, following established protocols for latency, amplitude, and conduction velocity. All procedures were carried out by trained personnel using calibrated neurophysiological equipment.

Statistical analysis: Data was entered in to MS excel 2019, and analysed using SPSS version 28. Continuous data was represented as mean and SD when normally distributed and categorical data as frequencies and percentages. Statistical analysis was done using Kruskal–Wallis test, chi-square, Spearmans correlation and multinomial logistic regression. $P < 0.05$ was considered as statistically significant.

RESULTS

A total of 100 participants were included in the study. The majority belonged to the 51–60 years age group (47, 47%), followed by 41–50 years (25, 25%), 61–70 years (15, 15%), 31–40 years (8, 8%), 71–80 years (4, 4%), and 21–30 years (1, 1%). The gender distribution was nearly equal, with 51 (51%) males and 49 (49%) females. (shown in table 1).

Table 1: Distribution of study participants by age and gender

Variables	Sub variable	Frequency	Percentage
Age group	21-30	1	1
	31-40	8	8
	41-50	25	25
	51-60	47	47
	61-70	15	15
	71-80	4	4
Gender	MALE	51	51
	FEMALE	49	49

Over half of the participants (54%) exhibited no detectable neuropathy, while 40% showed a sensorimotor pattern, indicating subclinical involvement. Less common were pure sensory (4%) and autonomic (2%) patterns. (figure 1)

Figure 1: Prevalence of Neuropathy Patterns in Asymptomatic Diabetic Patients

Among those with duration of diabetes mellitus ≤5 years, 81% had no neuropathy, while in the >10 years group, 71% exhibited neuropathy. The chi-square value of 22.4 and a p-value of 0.0016 indicate a statistically significant relationship. (table 2)

Table 2: Association Between Duration of Diabetes and presence of Neuropathy.

DM Duration Group		
0 (No		
Neuropathy)		
1 (Neuropathy Present)		
Chi-square		
P-value		
<=5	34 (81%)	8 (19%)
22.4		
0.0016		
10-Jun	13 (38.2%)	21(61.8%)
>10	7 (29.2%)	17 (70.8%)

Nerve conduction parameters showed significant deterioration with increasing duration of diabetes. In the common peroneal nerve, latency increased from 6.41 ms to 6.94 ms and demonstrated a moderate positive correlation ($r = 0.56$, $p = 0.02$), whereas amplitude decreased from 3.64 mV to 2.89 mV with a moderate negative correlation ($r = -0.42$, $p = 0.04$). NCV declined from 41.62 m/s to 37.35 m/s, exhibiting a strong negative correlation ($r = -0.71$, $p = 0.001$).

For the posterior tibial nerve, latency increased from 6.03 ms to 6.43 ms, showing a weak positive correlation ($r = 0.34$, $p = 0.012$). Amplitude decreased from 3.75 mV to 2.83 mV, with a moderate negative correlation ($r = -0.57$, $p = 0.04$), while NCV decreased from 42.14 m/s to 36.57 m/s, demonstrating a moderate negative correlation ($r = -0.61$, $p = 0.001$).

In the sural nerve, latency increased from 3.91 ms to 4.35 ms, showing a moderate positive correlation ($r = 0.47$, $p = 0.008$). Amplitude declined from 5.72 mV to 3.84 mV ($r = -0.72$, $p = 0.037$) and NCV from 42.01 m/s to 35.92 m/s ($r = -0.71$, $p = 0.001$), both demonstrating strong negative correlations. (table 3)

Table 3: Correlation of Duration of Diabetes and Nerve Conduction Parameters assessed in the current study

Nerve assessed				
DM Duration Group				
Latency (ms)				
Amplitude (mV)				
NCV (m/s)				
Common Peroneal Nerve (Motor)	<=5	6.41	3.64	41.62
	6–10	6.72	3.27	39.1
	>10	6.94	2.89	37.35

Spearmans correlation, p vlaue r=0.56. P =0.02, moderate positive correlation r= -0.42. P =0.04, Weak negative correlation r= -0.71. P =0.001, Strong negative correlation

Posterior Tibial Nerve (Motor)

<=5 6.03 3.75 42.14

6–10 6.23 3.27 38.41

>10 6.43 2.83 36.57

Spearmans correlation, p vlaue r=0.34. P =0.012, Weak positive correlation r= -0.57. P =0.04, Moderate negative correlation r= -0.61. P =0.001, Moderate negative correlation

Sural Nerve (Sensory) <=5 3.91 5.72 42.01

6–10 4.29 4.55 37.91

>10 4.35 3.84 35.92

Spearmans correlation, p vlaue r=0.47. P =0.008, Weak positive correlation r= -0.72. P =0.037, Strong negative correlation r= -0.71. P =0.001, Strong negative correlation

The mean HbA1c level increased with the severity of neuropathy. Participants without neuropathy (n = 54) had a mean HbA1c of 6.83 ± 0.48 (range: 6.2–7.8). Higher mean HbA1c values were observed in those with sensorimotor neuropathy (n = 40; 7.47 ± 0.54, range: 6.7–8.9), pure sensory neuropathy (n = 4; 7.35 ± 0.57, range: 6.8–8.0), and autonomic neuropathy (n = 2; 7.65 ± 0.35, range: 7.4–8.1). The overall mean HbA1c for the study population was 7.01 ± 0.31 (range: 6.5–7.5). The difference in HbA1c levels across neuropathy patterns was statistically significant (t = 3.46, p = 0.0016), indicating an association between poorer glycemic control and the presence of neuropathy. (table 4)

Table 4: Association Between HbA1c Levels and Neuropathy Pattern

Neuropathy Pattern	count	Mean HbA1C	std	min	max	Unpaired sample t test	P-value
None	54	6.83	0.48	6.2	7.8	3.46	0.0016
Sensorimotor	40	7.47	0.54	6.7	8.9		
Pure Sensory	4	7.35	0.57	6.8	8		
Autonomic	2	7.65	0.35	7.4	8.1		
Overall	100	7.01	0.31	6.5	7.5		

Multivariable logistic regression analysis identified the duration of diabetes and HbA1c as independent predictors of subclinical neuropathy. Each additional year of diabetes was associated with a 42% increase in the odds of subclinical neuropathy (OR = 1.42; 95% CI: 1.18–1.70; p < 0.001). Likewise, every 1% increase in HbA1c nearly doubled the likelihood of subclinical neuropathy (OR = 1.98; 95% CI: 1.23–3.17; p = 0.005). In contrast, age was not significantly associated with subclinical neuropathy (OR = 1.03; 95% CI: 0.97–1.09; p = 0.29), and gender also showed no significant effect (OR = 1.22; 95% CI: 0.58–2.58; p = 0.60). (table 5)

Table 5: Multivariate Logistic Regression – Predictors of Subclinical Neuropathy

Independent Variable	Odds Ratio (OR)	95% Confidence Interval (CI)	p-value
Duration of Diabetes (years)	1.42	1.18 – 1.70	<0.001
HbA1c (%)	1.98	1.23 – 3.17	0.005
Age (years)	1.03	0.97 – 1.09	0.29
Gender (Male vs. Female)	1.22	0.58 – 2.58	0.6

DISCUSSION

The findings from this study assessed prevalence of subclinical DPN and its pattern in asymptomatic patients with DM, emphasizing the relationship of duration of DM with electrophysiological changes to identify the changes at the earliest.

In our study 100 asymptomatic patients with DM were included, of which 51% were males and 49% were females, showing almost equal distribution. Whereas study by, Salata Mohapatra et al. (males = 66.7%),[12] and Shalini Kaur et al,[13] (males were 63.64%) reported males' predominance. On the other side, Waseem S. Hamid et al,[14] reported a female predominance, with 55.6% of the study population being women. These variations could be due to, differences in study populations, geographic

settings, referral patterns, or healthcare-seeking behavior.

Our study participants have a mean age of 51.38 ± 10.54 years, showing a predominantly middle-aged diabetic population frequently encountered in clinical practice. Similar findings were reported by Shivani Agarwal et al,[15] (mean age of 52.16 ± 10.12 years), and Suslata Mohapatra et al,[12] (mean age of 52.42 ± 7.39 years) showing the comparability across the studies. In contrast, Waseem S. Hamid et al,[14] documented a higher mean age of 57.71 ± 12.2 years, indicating an older study population. Such differences in age may partly explain the variation in the prevalence and severity of neuropathy observed between studies. The age profile of the present cohort is clinically relevant because it represents the stage at which diabetic neuropathy may already be developing despite the absence of overt symptoms.

The study population had a mean HbA1c of 7.01 ± 0.31%, indicating moderately controlled glycemia according to the American Diabetes Association (ADA) criteria in our study. This value was slightly lower than those reported by Shivani Agarwal et al,[15] (7.63 ± 2.4%) and Waseem S. Hamid et al,[14] (7.61 ± 1.76%), with the present study also demonstrating less variability in HbA1c values. In comparison, Suslata Mohapatra et al,[12] observed a substantially higher mean HbA1c of 8.96 ± 1.97%, reflecting poorer glycemic control and a potentially increased susceptibility to diabetic neuropathy.

When comparing neuropathy patterns and prevalence these differences in glycaemic status of the study population can have their impact. Higher HbA1c levels are generally associated with a greater likelihood of developing more severe or clinically apparent neuropathy, whereas the participants in the present study were asymptomatic with relatively better glycemic control.

In the present study, 40% of participants exhibited a sensorimotor neuropathy pattern, suggesting subclinical nerve involvement. Pure sensory neuropathy and autonomic neuropathy were identified in 4% and 2% of patients, respectively, resulting in an overall subclinical diabetic peripheral neuropathy (DPN) prevalence of 46%. These findings are comparable to those of Li Gao et al,[5] who reported that 142 of 311(47.5%) asymptomatic patients with Type 2 Diabetes Mellitus (45.7%) had abnormal nerve conduction studies consistent with subclinical diabetic peripheral neuropathy (sDPN). The similarity in prevalence across both studies supports the observation that a substantial proportion of asymptomatic individuals with diabetes already have electrophysiological evidence of peripheral nerve dysfunction despite the absence of clinical manifestations.

In the present study, the mean nerve conduction velocities were 39.36 m/s for the common peroneal nerve, 39.04 m/s for the posterior tibial nerve, and 38.61 m/s for the sural nerve. These values were lower than those reported in several comparable studies. Suslata Mohapatra et al,[12] documented substantially higher nerve conduction velocities, with 48.54 m/s for the peroneal nerve and 50.9 m/s for the sural nerve. Likewise, Waseem S. Hamid et al,[14] and Shivani Agarwal et al,[15] reported higher conduction velocities, with sural nerve values exceeding 43 m/s in both studies. Among the nerves evaluated in the present study, the posterior tibial nerve demonstrated the lowest conduction velocity (39.04 m/s), suggesting that motor nerve fibers may exhibit electrophysiological impairment even before the appearance of clinical symptoms. The observed differences between studies may be related to variations in study populations, duration of diabetes, glycemic control, demographic characteristics, and participant selection criteria.

The mean compound muscle action potential (CMAP) amplitude in the present study was 2.18 mV, which was lower than the values reported in most of the published literature. Shivani Agarwal et al,[15] reported a mean CMAP amplitude of 3.55 mV, whereas Waseem S. Hamid et al,[14] documented the highest value of 7.8 ± 4.04 mV, indicating better preservation of motor axonal function in their study population. In comparison, Shalini Kaur et al,[13] reported a mean amplitude of 2.78 ± 2.36 mV, which was more comparable to the findings of the present study. The relatively lower CMAP amplitudes observed in our asymptomatic cohort suggest that axonal dysfunction may develop before the onset of clinical manifestations, supporting the concept that subclinical nerve fiber damage can be detected through electrophysiological evaluation.

The present study demonstrated that the mean HbA1c was significantly higher among participants with neuropathy (7.47%) than among those without neuropathy (6.83%), a finding consistent with the observations of Masood A et al.[16] Multivariable logistic regression further identified the duration of diabetes and HbA1c as independent predictors of subclinical neuropathy, with odds ratios (ORs) of 1.42 and 1.98, respectively. In contrast, age and gender were not significantly associated with neuropathy in the present cohort.

These findings differ partly from those reported by Allam et al,[16] who found that increasing age (OR: 1.06; 95% CI: 1.03–1.08; $p < 0.001$), higher HbA1c levels (OR: 2.2; 95% CI: 1.7–2.9; $p < 0.001$), and the presence of subclinical hypothyroidism (SCH) (OR: 7.7; 95% CI: 3.6–15.5; $p < 0.001$) were independently associated with greater severity of diabetic peripheral neuropathy. The absence of a significant association between age and neuropathy in the present study may be related to differences in study population, sample characteristics, or disease stage.

Similarly, Allam M A et al,[17] reported significant differences between patients with and without subclinical diabetic peripheral neuropathy (sDPN) in terms of age, duration of diabetes, and many others (all $p < 0.05$). Their multivariable analysis identified

longer diabetes duration (OR: 1.062; 95% CI: 1.016–1.110), as independent predictors of sDPN ($p < 0.05$). Furthermore, their combined prediction model demonstrated good discriminatory ability, with an AUC of 0.6808 (95% CI: 0.6212–0.7404; $p < 0.0001$). Despite differences in the predictors evaluated, both the present study and the findings of Allam M A et al. underscore the importance of longer diabetes duration as a consistent independent risk factor for subclinical neuropathy, while emphasizing the contribution of poor glycemic control to early nerve dysfunction.

CONCLUSION

The prevalence of electrophysiological changes was seen in 46%, with sensorimotor neuropathy being the predominant pattern (40%), followed by pure sensory (4%) and autonomic (2%) neuropathy. The prevalence of neuropathy increased significantly with the duration of diabetes, rising from 19% in patients with ≤5 years of disease to 71% in those with >10 years ($\chi^2 = 22.4$, $p = 0.0016$). Progressive electrophysiological deterioration was observed in the common peroneal, posterior tibial, and sural nerves, characterized by increasing distal latencies and declining nerve conduction velocities with longer disease duration. The sural nerve exhibited the greatest reduction in conduction velocity (42.01 to 35.92 m/s) and amplitude, indicating early involvement of distal sensory fibers. Correlation analysis further confirmed significant positive associations between diabetes duration and latency, and negative associations with amplitude and NCV, with the strongest relationships observed for conduction velocity. In addition, the mean HbA1c was higher among patients with neuropathy (7.47%) than among those without neuropathy (6.83%). Collectively, these findings suggest that diabetic neuropathy is a length-dependent, predominantly axonal process that begins before the onset of clinical symptoms. The marked involvement of the sural nerve supports its value as a sensitive marker for early detection, highlighting the role of nerve conduction studies and optimal glycemic control in identifying subclinical neuropathy and potentially delaying the development of overt diabetic peripheral neuropathy.

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