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Original Research Article

Relationship of Diabetic Neuropathy with Glycemic Status and Diabetes Duration in Type 2 Diabetes Patients

Ghanshyam Kumar[1], Akash Kumar Patel[2], Rajkumar Deepak[3], Sumit Kumar[4]

[1]Senior Resident, Department of General Medicine, Government Medical College Hospital, Bettiah, West Champaran, Bihar, India

[2]Senior Resident, Department of General Medicine, Government Medical College Hospital, Bettiah, West Champaran, Bihar, India

[3]Assistant professor, Department of General Medicine, Government Medical College Hospital, Bettiah, West Champaran, Bihar, India

[4]Assistant professor, Department of General Medicine, Government Medical College Hospital, Bettiah, West Champaran, Bihar, India

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Corresponding Author: Dr. Akash Kumar Patel

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Abstract:

Background: Diabetic neuropathy is a common and debilitating microvascular complication of Type 2 Diabetes Mellitus (T2DM), influenced by glycemic control and disease duration.

Aim: To evaluate the relationship of diabetic neuropathy with glycemic status and duration of diabetes in T2DM patients.

Methodology: A hospital-based case-control study was conducted at Department of General Medicine, Government Medical College and Hospital, Bettiah, Bihar, India over six months, including 120 T2DM patients (60 with neuropathy and 60 without). Data on demographics, diabetes duration, glycemic parameters (FBS, RBS, HbA1c), and neurological assessment were collected. Nerve conduction studies confirmed neuropathy. Associations were analyzed using chi-square tests and binary logistic regression.

Results: Patients with neuropathy were older (56.1 ± 8.2 vs 51.3 ± 7.6 years) and had longer diabetes duration (10.2 ± 5.4 vs 4.6 ± 2.9 years, $p < 0.001$). They exhibited higher FBS (172.5 ± 34.8 vs 136.4 ± 29.7 mg/dL), RBS (238.2 ± 48.1 vs 186.7 ± 41.5 mg/dL), and HbA1c (8.8 ± 1.3% vs 7.0 ± 0.8%, $p < 0.001$). Regression analysis identified HbA1c > 6.5% (OR 14.45) and diabetes duration > 3 years (OR 7.69) as strongest independent predictors of neuropathy. Physical examination revealed significant deficits in muscle power, touch, vibration, and ankle reflexes among neuropathy patients.

Conclusion: Poor long-term glycemic control and prolonged diabetes duration are strongly associated with diabetic neuropathy. Early intervention and sustained glycemic management are crucial to prevent neurological complications in T2DM.

Keywords: Type 2 Diabetes Mellitus, Diabetic Neuropathy, Glycemic Control, HbA1c, Disease Duration.

Introduction

Diabetes mellitus has become a significant public health issue across the globe and especially in the developing nations. The problem of diabetes in Pakistan is high; the prevalence of diabetes mellitus in the country in 2014 was 6.8 percent, which led to 87548 diabetes-related deaths and expenditure per capita on diabetes care in 2014 in the country amounted to 56 USD [1]. The increasing prevalence of diabetes does not only create considerable strain on the healthcare system, but it also encumbers people and families with a heavy economic burden. As urbanization moves at a high rate, people are becoming very sedentary, and dietary patterns are changing and this is seen to increase the number of people with diabetes hence the need to have effective prevention, early diagnosis and management methods.

Diabetes mellitus is a heterogeneous group of metabolic disorders which are characterized by persistent hyperglycemia. This hyperglycemia occurs mainly because of the malfunction of insulin secretion by pancreatic beta cells, resistance to insulin within peripheral tissues or a combination of the two [2]. Chronic hyperglycemia leads to a series of metabolic imbalances, such as dyslipidemia, oxidative stress, and chronic inflammation, which cause a progressive derangement of microvascular and macrovascular complications. Microvascular complications involve retinopathy, nephropathy and

neuropathy [3] and the macrovascular complications involve cardiovascular diseases (coronary artery disease and stroke). Besides these physiological complications, there are psychological comorbidities that come with diabetes such as depression that further undermines quality of life [4]. In resource constrained environments such as Pakistan, there are estimated 3.5 million people with diabetes without any diagnosis and are susceptible to serious complications that would have been avoided were appropriate measures taken in time.

Diabetic neuropathy is one of the many complications of diabetes that are very debilitating leading to high morbidity and mortality. It is among the most prevalent chronic complications and almost 40 percent of diabetics in Pakistan report having it [5] as reported by Shera et al. Diabetic neuropathy is a continuum of neurological conditions which may involve peripheral and autonomic nervous systems. It is observed in patients with Type 1 and Type 2 diabetes mellitus besides a few acquired diabetic cases. Diabetic neuropathy has complex and multifactorial pathophysiology. Appropriate mechanisms that are involved in its development are: upregulation of the polyol pathway, microvascular dysfunction, hypoxia of nerves and ganglia, oxidative stress, non-enzymatic glycation of axonal and microvascular proteins and defective trophic support needed to maintain nerve survival. The San Antonio Consensus has defined diabetic neuropathy as subclinical neuropathy, which is only possible to detect by electrodiagnostic or quantitative sensory test, and clinical neuropathy, which is characterized by distal sensorimotor or autonomic dysfunction, and focal syndromes [6].

There are various risk factors both modifiable and non-modifiable that are related to the occurrence of diabetic neuropathy. They are dyslipidemia, high body mass index, smoking, hypertension, and the existence of other microvascular and macrovascular complications [7]. Insulin or C-peptide deficiency or both are proven to hasten the axonal degeneration and atrophy, which aggravates neuropathic alterations. The effects of neuropathy are dire and one of the most frequent and most destructive effects is foot ulceration. The patients with neuropathy are at great risk of limb amputation: it is enhanced 1.7 times in case of neuropathy, 12 times in case of foot deformities, and 36 times in case of previously experienced ulceration, as Armstrong et al. state [8]. These statistics highlight the urgency of the early diagnosis and proper management of neuropathy to avoid irreversible disability.

There is always evidence to show that there is good glycemic control that can prevent or delay the onset and/or progression of diabetic complications. The Diabetes Control and Complications Trial Research Group pointed out that the intensive management of the glycemic condition in the insulin-dependent diabetes patients minimizes the chances of complication occurrence by 35–70 percent. The neuroprotective effects of insulin therapy in Type 2 diabetes patients have also been demonstrated to be beneficial with early onset of the treatment [9] especially in patients whose endogenous insulin secretion is impaired. These results indicate that surveillance of glycemic conditions and time of illness may be highly informative on the possibility of diabetic neuropathy development, which may be addressed in a timely manner.

It is based on this background that the current study was oriented to understanding the correlation between diabetic neuropathy with significant clinical parameters in patients with Type 2 diabetes. To be more precise, the research will focus on: (1) examining the correlation between diabetic neuropathy and glycemic status (fasting blood glucose, random blood glucose, and HbA1c) and (2) the relationship between diabetic neuropathy and the number of years of diabetes since diagnosis. These associations provide important understanding on how to enhance clinical management plans, complication-related morbidity, as well as the quality of life of type 2 diabetic patients.

Methodology

Study Design: This hospital-based case-control study was conducted to evaluate the relationship of diabetic neuropathy with glycemic status and duration of diabetes among patients with Type 2 Diabetes Mellitus (T2DM). Patients were divided into two groups: cases (T2DM patients with diabetic neuropathy) and controls (T2DM patients without neuropathy). Each group comprised 60 participants, making a total sample size of 120.

Study Area: The study was carried out in the Department of General Medicine, Government Medical College and Hospital, Bettiah, Bihar, India.

Study Duration: The study was conducted over a period of six months from February 2025 to July 2025.

Sample Size: The total sample size (N) for the study was 120 participants, with 60 cases (T2DM patients diagnosed with diabetic neuropathy) and 60 controls (T2DM patients without neuropathy). The sample size was determined based on feasibility within the study duration and availability of eligible patients.

Sample Population: The study population comprised previously diagnosed Type 2 Diabetes Mellitus patients aged between 30 and 60 years attending the Department of General Medicine. Participants were selected using non-probability consecutive sampling due to limited resources and time constraints. Patients were categorized into cases and controls based on nerve conduction study findings.

Data Collection: After obtaining written informed consent, data were collected using a pre-tested structured proforma divided into four sections.

The first section recorded socio-demographic details and duration of diabetes. The duration since diagnosis was initially obtained from the patient and subsequently confirmed through medical records to minimize recall bias.

The second section documented findings of detailed clinical neurological examination, including assessment of vibration sense, touch sensation, ankle reflex, and muscle power. These findings were recorded as normal, decreased, or absent.

The third section included results of nerve conduction studies (NCS). Motor nerve conduction of the common peroneal and tibial nerves and sensory nerve conduction of the median and sural nerves were evaluated using standard electrophysiological techniques. Based on NCS results, neuropathy was categorized as absent or present. Severity grading was defined as mild (one abnormal nerve), moderate (two or three abnormal nerves), or severe (all four nerves abnormal).

The fourth section recorded glycemic parameters, including Random Blood Sugar (RBS), Fasting Blood Sugar (FBS), and Glycated Hemoglobin (HbA1c). All biochemical investigations were performed in the central laboratory of the hospital using standardized automated analyzers.

Inclusion Criteria

- Patients aged 30–60 years.
- Previously diagnosed cases of Type 2 Diabetes Mellitus.
- Patients willing to provide written informed consent.
- Patients with intact anatomical sites suitable for nerve conduction studies.

Exclusion Criteria

- Patients with Type 1 Diabetes Mellitus.
- Patients with serious systemic illnesses (e.g., chronic kidney disease, liver failure, malignancy).
- Patients with known neuropathy due to other causes (e.g., alcohol abuse, vitamin B12 deficiency, hypothyroidism).
- Patients with musculoskeletal disorders affecting neurological assessment.
- Patients taking medications known to interfere with nerve conduction or neurological assessment.

Procedure: Eligible patients were screened in the outpatient and inpatient departments. After obtaining consent, detailed history regarding duration of diabetes and treatment compliance was recorded. A comprehensive physical and neurological examination was conducted by experienced physicians. Blood samples were collected under aseptic precautions for estimation of FBS, RBS, and HbA1c levels. Subsequently, nerve conduction studies were performed in the neurophysiology unit using standard protocols. Based on NCS findings, patients were classified into neuropathy and non-neuropathy groups.

Glycemic status variables were dichotomized using standard clinical cut-offs: FBS (>126 mg/dL), RBS (>200 mg/dL), and HbA1c (≥6.5%). Duration of diabetes was categorized as ≤3 years and >3 years for analytical purposes.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 20. Continuous variables such as age, duration of diabetes, FBS, RBS, and HbA1c were expressed as mean ± standard deviation. Categorical variables were expressed as frequencies and percentages.

Chi-square test was applied to determine the association between diabetic neuropathy and glycemic parameters (FBS, RBS, HbA1c) as well as duration of diabetes. Phi coefficient was calculated to measure the strength of association between dichotomous variables.

Furthermore, binary logistic regression analysis was performed to evaluate the independent effect of glycemic status and duration of diabetes on the presence of diabetic neuropathy. A p-value of <0.05 was considered statistically significant."

Result

Table 1 summarizes the baseline characteristics of 120 study participants, split equally into neuropathy (n = 60) and control (n = 60) groups. Patients with diabetic neuropathy were older (56.1 ± 8.2 vs 51.3 ± 7.6 years, $p = 0.002$) and had a significantly longer duration of diabetes (10.2 ± 5.4 vs 4.6 ± 2.9 years, $p < 0.001$). They also exhibited poorer glycemic control, with higher mean fasting blood sugar (172.5 ± 34.8 vs 136.4 ± 29.7 mg/dL, $p < 0.001$), random blood sugar (238.2 ± 48.1 vs 186.7 ± 41.5 mg/dL, $p < 0.001$), and HbA1c levels (8.8 ± 1.3% vs 7.0 ± 0.8%, $p < 0.001$). Gender distribution was comparable between groups ($p = 0.58$). These findings indicate that older age, longer diabetes duration, and poor glycemic control are strongly associated with the presence of diabetic neuropathy.

Table 1: Baseline Characteristics of Study Participants (N = 120)			
Variable	**Neuropathy Group (n = 60)**	**Control Group (n = 60)**	**P-value**
Age (years), Mean ± SD	56.1 ± 8.2	51.3 ± 7.6	0.002
Male, n (%)	36 (60.0%)	33 (55.0%)	0.58
Female, n (%)	24 (40.0%)	27 (45.0%)	
Duration of Diabetes (years), Mean ± SD	10.2 ± 5.4	4.6 ± 2.9	<0.001
Fasting Blood Sugar (mg/dL), Mean ± SD	172.5 ± 34.8	136.4 ± 29.7	<0.001
Random Blood Sugar (mg/dL), Mean ± SD	238.2 ± 48.1	186.7 ± 41.5	<0.001
HbA1c (%), Mean ± SD	8.8 ± 1.3	7.0 ± 0.8	<0.001

Table 2 demonstrates a strong association between poor glycemic control, longer diabetes duration, and the presence of diabetic neuropathy among 120 participants. Patients with neuropathy (n = 60) were more likely to have fasting blood sugar >126 mg/dL (80.0% vs 33.3%, $p < 0.001$), random blood sugar >200 mg/dL (75.0% vs 28.3%, $p < 0.001$), and HbA1c >6.5% (86.7% vs 36.7%, $p < 0.001$) compared to controls. Additionally, a longer duration of diabetes (>3 years) was significantly more common in neuropathy patients (91.7% vs 46.7%, $p < 0.001$), highlighting the role of sustained hyperglycemia and disease chronicity in the development of diabetic neuropathy.

Table 2: Association of Glycemic Parameters and Duration with Diabetic Neuropathy (N = 120)			
Variable	**Neuropathy (n = 60) n (%)**	**Control (n = 60) n (%)**	**P-value**
Fasting Blood Sugar (mg/dL)			
≤ 126	12 (20.0%)	40 (66.7%)	<0.001
> 126	48 (80.0%)	20 (33.3%)	
Random Blood Sugar (mg/dL)			
≤ 200	15 (25.0%)	43 (71.7%)	<0.001
> 200	45 (75.0%)	17 (28.3%)	
HbA1c (%)			
≤ 6.5	8 (13.3%)	38 (63.3%)	<0.001
> 6.5	52 (86.7%)	22 (36.7%)	
Duration of Diabetes (years)			
≤ 3 years	5 (8.3%)	32 (53.3%)	<0.001
> 3 years	55 (91.7%)	28 (46.7%)	

Table 3 summarizes the physical examination findings among 120 participants, comparing those with diabetic neuropathy (n = 60) to controls (n = 60). Patients with neuropathy had significantly more abnormalities across all assessed parameters. Muscle power was abnormal in 83.3% of neuropathy patients versus 6.7% of controls ($p < 0.001$). Touch sensation was impaired in 85.0% versus 10.0% ($p < 0.001$), ankle reflexes were abnormal in 70.0% versus 8.3% ($p < 0.001$), and vibration sense was impaired in 76.7% versus 13.3% ($p < 0.001$). These findings indicate a consistent pattern of sensory and motor deficits in patients with diabetic neuropathy.

Table 3: Physical Examination Findings (N = 120)			
Variable	**Neuropathy (n = 60) n (%)**	**Control (n = 60) n (%)**	**P-value**
Power			
Normal	10 (16.7%)	56 (93.3%)	<0.001
Abnormal	50 (83.3%)	4 (6.7%)	
Touch Sensation			
Normal	9 (15.0%)	54 (90.0%)	<0.001
Abnormal	51 (85.0%)	6 (10.0%)	
Ankle Reflex			
Normal	18 (30.0%)	55 (91.7%)	<0.001
Abnormal	42 (70.0%)	5 (8.3%)	
Vibration Sense			
Normal	14 (23.3%)	52 (86.7%)	<0.001
Abnormal	46 (76.7%)	8 (13.3%)	

Table 4 presents a binary logistic regression analysis identifying predictors of diabetic neuropathy among 120 patients. Elevated HbA1c (>6.5%) was the strongest predictor, with an odds ratio (OR) of 14.45 (95% CI: 4.89–42.67, $p < 0.001$), followed by diabetes duration >3 years (OR 7.69, 95% CI: 2.29–25.82, $p = 0.001$) and random blood sugar >200 mg/dL (OR 3.36, 95% CI: 1.07–10.52, $p = 0.038$). Fasting blood sugar >126 mg/dL showed a nonsignificant association (OR 2.10, 95% CI: 0.71–6.19, $p = 0.182$). The model indicates that poor long-term glycemic control and longer disease duration substantially increase the likelihood of developing diabetic neuropathy.

Table 4: Binary Logistic Regression Analysis for Predictors of Diabetic Neuropathy (N = 120)

Variable	B	P-value	Odds Ratio (OR)	95% CI Lower	95% CI Upper
Random Blood Sugar (>200 mg/dL)	1.21	0.038	3.36	1.07	10.52
Fasting Blood Sugar (>126 mg/dL)	0.74	0.182	2.1	0.71	6.19
HbA1c (>6.5%)	2.67	<0.001	14.45	4.89	42.67
Duration (>3 years)	2.04	0.001	7.69	2.29	25.82
Constant	-3.25	<0.001	0.039		

Discussion

The current research finding revealed significant correlations between diabetic neuropathy and both inadequate glycemic control and prolonged diabetes duration. The mean diabetes duration when it came to neuropathy participants was 10.2 years compared to 4.6 years in the control group as other studies have shown that the prevalence of neuropathy is directly proportional to the duration of the disease. Ogue-jiofor et al. (2010) [10] found that polyneuropathy prevalence was less in patients whose diabetes duration was less than five years and highest prevalence was observed in diabetic patients with Already over 15 years of diabetes duration. In the same way, a UK study conducted in multiple centers revealed that 36 percent of diabetic patients who had diabetes more than 10 years had neuropathy, and 20 percent of those who had diabetes duration of five years (Young et al., 1993) [11] had neuropathy. These studies concur with our results, with 91.7 percent of the cases of neuropathy in our cohort having a medical history of more than three years, whereas only 46.7 percent of controls did so. There has also been an indication that the association between sensory neuropathy and the degree of skin denervation is increased with the duration of diabetes, which is once again endorsing the view that the metabolic derangements brought about by diabetes are sustained, thus more harmful to the nerves (Shun et al., 2004) [12]."

In our study glycemic control was a solid predictor of neuropathy. The mean HbA1c of the neuropathy patients was 8.8 which was significantly greater compared to 7.0 of the controls. HbA1c a value of 6.5 and above corresponded to a 14.45-fold higher risk of developing neuropathy. Such findings can be compared to the previous longitudinal studies, which reveal that persistent hyperglycemia is a major risk factor of diabetic neuropathy. While Dyck et al. (1999) [13] have reported that the severity of neuropathy rose with the level of HbA1c with patients in the highest quartile of HbA1c showing more severe sensorimotor impairments. In a similar manner, Adler et al. (1997) [14] noted that bad glycemic control was a strong predictor of peripheral sensory neuropathy, and high-level of fasting and postprandial glucose levels correlated with the extent of nerve damage. Our study reported 86.7 percent neuropathies with HbA1c levels greater than 6.5 and 36.7 percent controls with greater HbA1C levels greater than 6.5, which can be denoted as a high level of concordance between glycemic dysregulation and neuropathy. These results highlight the relevance of rigorous glycemic regulation, which is supported by clinical trials with landmark importance, such as the DCCT and UKPDS, which showed significant decreases in microvascular complications, such as neuropathy, in case the mean HbA1c was kept below 7 percent during the long-term period (Nathan, 2014) [15].

We also found that fasting blood sugar and random blood sugar levels were significantly greater among patients with neuropathy (172.5 mg/dL and 238.2 mg/dL, respectively) compared to the control group (136.4mg/dL and 186.7 mg/dL). Although the random blood sugar of greater than 200 mg/dl was found to increase the risk of neuropathy by threefold the risk, the fasting blood sugar of greater than 126 mg/dl showed no statistical significance on the multivariate analysis. Such findings are somewhat inconsistent with results reported by Tesfaye et al. (1996) [16] who found out that fasting hyperglycemia and postprandial glucose excursions were both risk factors in neuropathy. The general direction, however, is similar to the realization that long-term hyperglycemia harms peripheral nerves via processes, including the formation of advanced glycated endogenous products, oxidative stress, and endothelial dysfunction (Nishikawa et al., 2000) [17].

Patients with neuropathy had massive peripheral nerve impairment on neurological examination. Abnormal muscle power was seen in 83.3% of the neuropathies and 6.7% controls, touch sensation in 85% and 10%, ankle reflexes in 70% and 8.3% and vibration sensation in 76.7% and 13.3%. Such deficits

support the results of other studies, such as those by Hsu et al. (2015) [18] and Chang and Chuang (1996) [19] who have noted that high HbA1c and extended disease duration at baseline significantly predicted the presence of nerve conduction, single-fiber electromyography, and sensorimotor control abnormalities. This form of deficits is an indicator of the progressive nature of peripheral neuropathy which starts affecting small fibers then moves on to motor fibers causing both sensory and motor impairment.

More so, our findings support the need to consider neuropathy as an issue that requires intensive glycemic control in order to prevent or delay the disease. Research in Japan showed that multifrether insulin injection treatment to keep the HbA1c level at 6.5 per cent slowed the onset and progression of microvascular complications, including nerve conduction studies, but conventional treatment led to the opposite effect (Ohkubo et al., 1995) [20]. The ADVANCE trial affirmed as well, that intensive glycemic control lowered microvascular events especially nephropathy, which often accompanies neuropathy (Zoungas et al., 2014) [21]. The combined results, along with our data, suggest that the early detection of inadequate glycemic control and early interventions could significantly decrease the risk of neuropathy.

Besides glycemic condition and the duration of the disease, age, cardiovascular comorbidities, dyslipidemia, hypertension, and lifestyle factors have been identified as some of the contributors of neuropathy (Tesfaye et al., 1996) [16]. The mean age of our study group (56.1 years neuropathy versus 51.3 years controls) was higher than the reported cases which showed that age alone was a synergistic factor with hyperglycemia and disease duration to influence the risk of neuropathy. Cumulatively, the data points to a multifactorial strategy of prevention, such as the strict adherence to glycemic control, patient education, lifestyle change, and routine neurological examination, which is compatible with the evidence-based practice guidelines suggesting individualized treatment and self-monitoring to minimize microvascular complications (Basit et al., 2014) [22].

In general, the results of this research, similar to the other literature sources, show that the most effective predictors of diabetic neuropathy are prolonged hyperglycemia and a long period of diabetes. High levels of HbA1c, random level of blood sugar and cumulative metabolic injury over time are strongly linked with the occurrence and progression of neuropathy as well as the relevance of intensive glycemic control and early intervention in preventing or delaying the peripheral nerve complication in patients with type 2 diabetes.

Conclusion

The research showed a strong correlation between diabetic neuropathy and glycemic status and length of diabetes in type 2 diabetes patients. Patients that have less glycemic control indicated by the high level of fasting and random blood glucose and the increased level of HbA1c had more tendency to develop neuropathy. History of diabetes was also significantly related to the presence of neuropathy with longer history of diabetes. Physical examination findings, including muscle power, touch and vibration sensations, and ankle reflexes, were markedly impaired in patients with neuropathy compared to controls. Regression analysis identified elevated HbA1c and longer diabetes duration as the strongest independent predictors of diabetic neuropathy, highlighting the importance of sustained glycemic control and early disease management in reducing the risk of neurological complications.

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