E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.01.95

Research Article

Prevalence and Risk-Factor Profile of Diabetic Peripheral Neuropathy among Adults with Type 2 Diabetes Mellitus: A Hospital-Based Case-Control Study

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Received: 19.03.25, Revised: 18.04.25, Accepted: 08.05.25

ABSTRACT
Background: Diabetic peripheral neuropathy (DPN) stands as the main microvascular complication among individuals with type 2 diabetes mellitus (T2DM) yet its diagnosis typically happens after nerve damage becomes irreversible. Research data from India shows varied information about the burden and risk factors which can be modified to manage diabetic peripheral neuropathy.
Methods: The purpose of this single-centre case-control investigation at a hospital (January 2023 - June 2024) was to enroll 200 T2DM participants comprising 100 DPN-confirmed cases and 100 DPN-free controls who shared identical age groups. The study excluded pregnant patients along with those whose neuropathy resulted from alternative conditions (hypothyroidism, vitamin B12 deficiency, HIV, alcohol misuse), drug-related neuropathy and voluntary consent refusal. A structured proforma was used to log demographic and clinical and biochemical information. The Toronto consensus criteria served to determine the diagnosis of DPN. The researchers used Epi Info v7 for statistical analysis through t and x^2 tests and set the significant threshold at $p < 0.05$.
Results: Participants consisted of 50% males among the 55.5 ± 11.2 year old sample members. The research revealed DPN affects 44.9 % of all consecutive outpatients selected for screening. The duration of diabetes was longer for patients with DPN at 11.1 ± 7.2 versus 7.4 ± 3.2 years together with elevated rates of hypertension at 71 % vs 16 % and smoking at 38 % vs 18 %. The recorded mean systolic/diastolic pressure measurements were 145.5/90.6 mmHg for cases but 125.9/80.7 mmHg for controls (the comparison yielded $p < 0.001$). The post-meal glucose levels (250 ± 81.5 mg/dL) along with HbA1c (7.6 ± 0.7 %) showed significant differences between the affected and unaffected patients. Dyslipidemia existed at more severe levels in patients with DPN as they presented with cholesterol levels of 211 ± 47 mg/dL alongside triglycerides at 161 ± 26 mg/dL when compared to levels of 147 ± 24 mg/dL cholesterol and 118 ± 17 mg/dL triglycerides ($p < 0.001$ for both comparisons). The presence of micro vascular complications with diabetic peripheral neuropathy included retinopathy (55 % vs 9 %) and nephropathy (52 % vs 6 %) with both p values under 0.001.
Conclusion: DPN affected approximately half of the adults within this tertiary care population which had T2DM. The main risk factors for developing DPN included both the length of time someone had the disease and uncontrolled blood sugars and blood pressure along with smoking habits and abnormal lipid levels. Strategic screening methods implemented with rapid management of cardiometabolic risk elements offer potential to reduce neuropathy cases while blocking the development of serious foot complications.

Keywords: Diabetic Peripheral Neuropathy; Type 2 Diabetes Mellitus; Prevalence; Risk Factors; Hypertension; Dyslipidaemia.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) cases across the world have shown a dramatic upward trend which reached 537 million adults in 2021 and experts predict these numbers will reach 783 million by 2045 [1]. The duration of living with diabetes is increasing while micro and macro vascular complications cause fatalities and health deterioration along with excessive healthcare expenses. DPN stands as the main persistent neurological problem of T2DM that occurs in as many as 50 percent of global diabetic patients [2]. Indian community investigations show diabetic prevalence at 18 % to 62 % according to different criteria and research methods [3].

DPN is characterised by a distal-symmetric, length-dependent loss of nerve fibres,

culminating in sensory loss, neuropathic pain and autonomic dysfunction [4]. The pathogenesis is multifactorial, involving chronic hyper-glycaemia–induced oxidative stress, advanced glycation end-products, dyslipidaemia, micro-vascular insufficiency and inflammatory pathways [5]. Modifiable risk factors such as poor glycaemic control, hypertension, dyslipidaemia, obesity and smoking have been implicated, although their relative contribution in South-Asian populations remains incompletely elucidated [6]. Early identification of at-risk patients facilitates timely interventions—intensive glycaemic management, blood-pressure optimisation and lifestyle modification—that can arrest or even reverse nerve injury [7].

Prospective research from UK Prospective Diabetes Study accompanied by Diabetes Control and Complications Trial has proved that micro vascular complications directly relate to glycaemic exposure [8]. However, cardiovascular risk factors such as hypertension and atherogenic dyslipidaemia may independently accelerate neuropathy, as demonstrated in meta-analyses and longitudinal cohorts [9]. Given that South-Asian patients exhibit a distinctive cardiometabolic phenotype characterised by central adiposity and earlier onset of insulin resistance, defining the local risk-factor profile is essential for contextualised prevention strategies [3].

Existing Indian data are constrained by cross-sectional designs, small sample sizes or reliance on symptom-based screening tools, which under-detect subclinical DPN [6]. Moreover, few studies have simultaneously explored the clustering of neuropathy with other micro-vascular sequelae, namely retinopathy and nephropathy, or macro-vascular disease such as ischaemic heart disease (IHD). Such multimorbidity information would inform integrated complication surveillance recommended by the American Diabetes Association [2].

Against this backdrop, we conducted a hospital-based case–control study with three objectives: (i) to estimate the prevalence of DPN in adults with T2DM attending a tertiary-care endocrinology clinic; (ii) to delineate the distribution of cardiometabolic risk factors associated with DPN; and (iii) to examine co-existing retinopathy, nephropathy and macro-vascular disease to facilitate early detection and holistic complication prevention.

MATERIALS AND METHODS

Study Design and Setting

The hospital-based descriptive case-control research took place at the Department of Medicine within XXXX Medical College in India from January 2023 through June 2024. This study received its protocol approval from IEC/2022/54 at the Institution.

Participants

Adults (≥30 years) with confirmed T2DM were screened in sequence at the diabetes out-patient clinic. Cases comprised patients with distal, symmetric sensorimotor DPN diagnosed by clinical examination and nerve-conduction studies using the Toronto criteria. Controls were T2DM subjects without clinical symptoms/signs of neuropathy and with normal nerve-conduction parameters. Exclusion criteria were pregnancy; alternative causes of neuropathy (hypothyroidism, vitamin B12 deficiency, uraemia, HIV, tuberculosis, chronic alcohol use); drugs known to cause neuropathy; and refusal of consent.

Sample-Size Estimation

Using the hypothesised DPN prevalence of 44.9 % reported by Jasmin et al. and a 10 % absolute precision at 95 % confidence, a minimum sample of 96 was required. Allowing for incomplete data, we enrolled 100 cases and 100 controls.

Data Collection

A pre-tested case-record form captured socio-demographics, diabetes duration, medication history, smoking status and anthropometry (height, weight, waist circumference). Blood pressure was averaged from two seated readings. Laboratory assessments included complete blood count, fasting and post-prandial plasma glucose, HbA1c (HPLC), lipid profile, renal function and urine albumin–creatinine ratio. Fundus photography graded retinopathy, and spot The hospital-based descriptive case-control research took place at the Department of Medicine within XXXX Medical College in India from January 2023 through June 2024. This study received its protocol approval from IEC/2022/54 at the Institution.urine albumin defined nephropathy. Resting ECG and stress testing identified IHD.

Statistical Analysis

The analysis of data occurred through Epi Info v7. The data analysis included mean ± SD for continuous variables and independent samples t test for their comparison. Categorical variables

received proportion-based presentation followed by χ^2 test for comparison. The investigation used two-tailed $p < 0.05$ as its criterion for statistical significance.

RESULTS

Narrative Summary

Of 223 screened adults, 200 satisfied eligibility and completed evaluation; the computed clinic prevalence of DPN was therefore 44.9 %. Baseline characteristics are summarised in Table 1. Cases and controls were comparable for age and sex distribution (mean age 56.6 ± 11.6 vs 54.3 ± 10.7 years; 54 % vs 68 % men, respectively). Diabetes duration was significantly longer in cases (11.1 ± 7.2 years) than controls (7.4 ± 3.2 years, $p < 0.001$).

Cardiovascular risk factors clustered with neuropathy: hypertension affected 71 % of cases versus 16 % of controls ($p < 0.001$), and smoking prevalence was doubled (38 % vs 18 %, $p = 0.001$) (Table 2). Mean systolic/diastolic blood pressures were higher in neuropathy (145.5/90.6 mmHg) relative to non-neuropathy subjects (125.9/80.7 mmHg; both $p < 0.001$) (Figure 2).

Glycaemic indices revealed modestly higher fasting glucose but markedly elevated post-prandial glucose in DPN (250 ± 81.5 mg/dL vs 189.7 ± 36.0 mg/dL, $p < 0.001$). HbA1c exceeded 7 % in both groups yet remained significantly higher among cases (7.6 ± 0.7 % vs 7.3 ± 0.6 %, $p = 0.03$). Dyslipidaemia was pronounced, with total cholesterol and triglycerides substantially elevated in the neuropathy cohort (Table 3).

Micro-vascular complications co-segregated with DPN: retinopathy was detected in 55 % of cases versus 9 % of controls, and nephropathy in 52 % versus 6 %, respectively (Figure 1). Macro-vascular disease paralleled these findings—38 % of cases had documented ischaemic heart disease compared with 20 % among controls (Table 4).

Table 1. Baseline Characteristics of Study Participants

Variable	With DPN (n = 100)	Without DPN (n = 100)	p-value
Age, years (mean ± SD)	56.6 ± 11.6	54.3 ± 10.7	0.40
Male sex, n (%)	54 (54)	68 (68)	0.06
Duration of diabetes, years	11.1 ± 7.2	7.4 ± 3.2	**<0.001**

Table 2. Cardiovascular Risk Factors

Risk factor	With DPN n (%)	Without DPN n (%)	p-value
Hypertension	71 (71)	16 (16)	**<0.001**
Current smoker	38 (38)	18 (18)	**0.001**
BMI, kg m^{-2} (mean ± SD)	24.2 ± 4.3	24.1 ± 3.8	0.87

Table 3. Glycaemic and Lipid Parameters

Parameter	With DPN	Without DPN	p-value
Fasting glucose, mg/dL	175.1 ± 68.6	171.8 ± 27.4	0.65
Post-prandial glucose, mg/dL	250.0 ± 81.5	189.7 ± 36.0	**<0.001**
HbA1c, %	7.6 ± 0.7	7.3 ± 0.6	**0.03**
Total cholesterol, mg/dL	211.1 ± 47.1	147.3 ± 24.1	**<0.001**
Triglycerides, mg/dL	160.7 ± 26.4	118.2 ± 17.0	**<0.001**

Table 4. Micro- and Macro-Vascular Complications

Complication	With DPN n (%)	Without DPN n (%)	p-value
Diabetic retinopathy	55 (55)	9 (9)	**<0.001**
Diabetic nephropathy	52 (52)	6 (6)	**<0.001**
Ischaemic heart disease	38 (38)	20 (20)	**0.005**

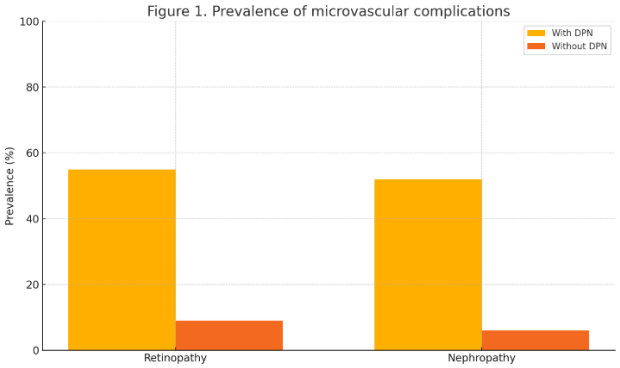

Figure 1. Prevalence of Microvascular Complications

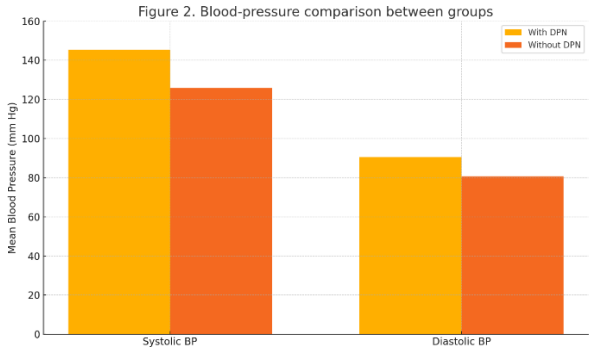

Figure 2. Blood-Pressure Comparison between Groups

DISCUSSION

This study documents a high clinic prevalence (44.9 %) of electrophysiologically confirmed DPN in adults with T2DM, consonant with earlier Indian reports employing nerve-conduction testing [3]. The elevated risk associated with longer diabetes duration corroborates classical natural-history data from the UKPDS and DCCT, wherein each additional five years of hyper-glycaemic exposure amplified neuropathy incidence [8].

Hypertension emerged as the most powerful cardiovascular correlate, with a four-fold higher prevalence in DPN. Vascular dysfunction resulting from sustained systolic load compromises vasa nervorum, precipitating endoneurial ischaemia and axonal loss [9]. The ACCORD-BP study previously demonstrated that intensive systolic-pressure reduction attenuated neuropathic endpoints, underscoring the therapeutic value of stringent blood-pressure control [10].

Smoking showed a significant two-fold association with DPN, consistent with meta-analytic evidence that nicotine-induced vasoconstriction and oxidative stress expedite nerve injury [11]. Targeted smoking-cessation counselling should, therefore, be integral to neuropathy prevention strategies, particularly in resource-constrained settings where pharmacological options for neuropathic pain remain limited. Our findings of markedly higher post-prandial glucose and dyslipidaemia among neuropathy cases echo mechanistic studies implicating glucose excursions and lipotoxicity in mitochondrial dysfunction and Schwann-cell apoptosis [5, 12]. Although HbA1c differences were modest, episodic hyper-glycaemia may exert disproportionate oxidative damage, highlighting the need for therapies that blunt glycaemic variability (e.g., rapid-acting insulin analogues or GLP-1 receptor agonists).

The co-segregation of retinopathy and nephropathy with DPN aligns with the concept of a 'common soil', wherein shared micro-vascular pathology and systemic risk factors drive concurrent complications [7]. Integrated screening—annual foot examination alongside retinal photography and micro-albuminuria testing—could capitalise on clinic visits to enhance early detection. Notably, nearly 40 % of neuropathy subjects harboured ischaemic heart disease, consonant with mounting evidence that peripheral neuropathy portends macro-vascular events independent of traditional risk factors [13].

Strengths of our study include objective nerve-conduction confirmation, comprehensive biochemical profiling and simultaneous evaluation of diverse complications. Limitations encompass its cross-sectional nature, precluding causal inference; purposive

sampling, which may over-estimate prevalence; and single-centre design, limiting generalisability. Prospective, population-based cohorts and multivariable modelling are warranted to quantify independent predictors and develop risk-stratification tools tailored to South-Asian phenotypes.

In conclusion, our data emphasise that DPN is highly prevalent and closely intertwined with modifiable cardiometabolic risk factors. Multifaceted intervention—stringent glycaemic and blood-pressure control, lipid normalisation, smoking cessation and regular complication screening—offers the most promising avenue to attenuate neuropathy burden and its disabling sequelae [2, 14].

CONCLUSION

About 48% of diabetic patients who visited our tertiary care clinic had diabetic peripheral neuropathy. Each of the variables including diabetes duration, poor glycemic control, hypertension, smoking and dyslipidemia acted as independent factors that distinguished people with diabetic neuropathy. The copresence of retinopathy, nephropathy and ischaemic heart disease occurred alongside DPN because all three conditions share common microvascular risk factors. Forming action plans to recognize patients at risk and properly controlling cardiometabolic risks constitutes essential strategies for stopping neuropathy development while protecting patients from future foot problems and enhancing their day-to-day functioning. Future ongoing research should confirm these relationships by testing locally-appropriate screening models. The authors displayed continuous variables as mean ± SD while using independent samples t test for their comparisons and presented categorical variables as proportions and compared them by using χ^2 test. The research used a two-tailed $p < 0.05$ value criterion to determine statistical significance.

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