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Original Research Article

TO ESTIMATE THE PREVALENCE OF DIABETIC NEUROPATHY IN TYPE 2 DIABETES MELLITUS PATIENTS AND TO STUDY ITS CORRELATION WITH RISK FACTORS

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ABSTRACT

Background: Aim: This cross-sectional study aimed to estimate the prevalence of diabetic peripheral neuropathy (DPN) in type 2 diabetes mellitus (T2DM) patients presenting to a tertiary care centre and to study its correlation with cardiometabolic risk factors, including duration of diabetes, glycated haemoglobin (HbA1c), hypertension, body mass index (BMI), smoking, tobacco use, and lipid profile. Additionally, the study sought to characterise the pattern of neuropathy—including limb distribution, type of nerve fibre involvement, and electrophysiological pattern—using nerve conduction studies (NCS).

Materials and Methods: A cross-sectional observational study was conducted at the Department of General Medicine, NSCB Medical College and Hospital, Jabalpur, Madhya Pradesh, from March 2021 to August 2022. A total of 150 patients with established T2DM aged 30 years and above were enrolled using consecutive sampling after fulfilling predefined inclusion and exclusion criteria. All participants were evaluated clinically using the Neuropathy Symptom Score (NSS) and Neuropathy Disability Score (NDS). Nerve conduction velocity (NCV) was recorded in the median, ulnar, radial, posterior tibial, sural, and peroneal nerves using an RMS Aleron 201 electromyography machine. Risk factor assessment included fasting blood sugar (FBS), post-prandial blood sugar (PPBS), HbA1c, lipid profile, anthropometric measurements, and blood pressure. Statistical analysis was performed using Chi-square test, Fisher's exact test, independent t-test, and odds ratio with 95% confidence interval.

Results: Out of 150 T2DM patients, 102 (68%) were diagnosed with diabetic peripheral neuropathy. The prevalence of DPN increased significantly with age ($p=0.011$), duration of diabetes ($p<0.001$), and HbA1c levels ($p<0.001$). Among patients with >10 years of diabetes, 92.3% had DPN. Hypertension showed a statistically significant association with DPN ($p=0.027$). Gender, BMI, smoking, tobacco, diet, and lipid parameters did not show significant independent association. Lower limb involvement was found in 60.7% while combined upper and lower limb involvement was noted in 34.3% patients. Axonal neuropathy was the predominant electrophysiological pattern (69.6%), and polyneuropathy was the commonest clinical presentation (76.4%).

Conclusion: The prevalence of diabetic peripheral neuropathy is high (68%) in T2DM patients at a tertiary care centre in central India. Duration of diabetes, advancing age, poor glycaemic control (high HbA1c), and hypertension are significant independent risk factors for DPN. Routine screening using NSS and

NDS followed by confirmation with NCS is recommended for all T2DM patients at the time of presentation, enabling early detection and preventive intervention to reduce DPN-related morbidity.
Keywords: Diabetic peripheral neuropathy, Type 2 diabetes mellitus, Nerve conduction study, Neuropathy Symptom Score, Glycated haemoglobin.

INTRODUCTION

Diabetes mellitus (DM) is one of the most challenging global health crises of the twenty-first century. India occupies a particularly alarming position in global diabetes epidemiology, ranking second in the world in terms of the total number of adults living with diabetes. Estimates from the ICMR-INDIAB study indicate that approximately 62.4 million Indians are affected by DM, with around 4.39 crore individuals unaware of their diabetic status, highlighting a massive unmet diagnostic gap.

Peripheral neuropathy represents one of the earliest and most prevalent complications of diabetes mellitus. The estimated global prevalence of diabetic peripheral neuropathy (DPN) ranges widely from 6% to 51% among adults with diabetes, depending on the patient population studied, diagnostic criteria applied, and disease characteristics including age, duration of diabetes, and type of DM. The spectrum of diabetic neuropathy encompasses several distinct syndromes, ranging from focal and multifocal neuropathies to diffuse, symmetrical sensorimotor and autonomic neuropathies. The hallmark and commonest form is distal symmetrical peripheral neuropathy (DSPN), which accounts for approximately 75% of all diabetic neuropathies.

Given the high prevalence of diabetes in India and the disproportionate burden of its complications, there is an urgent need for robust local epidemiological data on DPN, particularly from central India, where data remain sparse. Understanding the prevalence and risk factor associations of DPN in a tertiary care setting enables targeted screening, early intervention, and optimisation of glycaemic and cardiovascular risk factor management to reduce the morbidity—including foot ulcerations, amputations, gait disturbances, and impaired quality of life—that accompanies DPN. The present study was therefore undertaken to estimate the prevalence of diabetic peripheral neuropathy in T2DM patients presenting to a tertiary care centre in Jabalpur, Madhya Pradesh, and to assess the correlation of DPN with established cardiometabolic and demographic risk factors.

MATERIALS AND METHODS

Study Design and Setting This was a cross-sectional, observational study conducted in the Department of General Medicine, Netaji Subhash Chandra Bose (NSCB) Medical College and Hospital, Jabalpur, Madhya Pradesh, India—a tertiary care teaching institution. The study period extended from 1st March 2021 to 31st August 2022, spanning one and a half years.

Study Population and Sample Size The target population comprised patients with established type 2 diabetes mellitus presenting to the outpatient and inpatient departments of General Medicine. The estimated minimum required sample size was 150 patients, and this was the final enrolled sample.

Inclusion and Exclusion Criteria Inclusion criteria were: (i) patients with confirmed T2DM presenting to NSCB Medical College, Jabalpur; and (ii) age 30 years and above. Exclusion criteria were: (i) patients with peripheral vascular disease; (ii) chronic alcoholism; (iii) leprosy; (iv) pregnant women; and (v) patients receiving medications known to impair nerve function (e.g., chemotherapeutic agents, isoniazid without pyridoxine supplementation).

- **Clinical Assessment:** All enrolled patients underwent a detailed history and general physical examination. Neuropathy was screened clinically using two validated scoring instruments: Neuropathy Symptom Score (NSS) and Neuropathy Disability Score (NDS).
- Neurophysiological Assessment
- Cardiometabolic Risk Factor Assessment

Ethical Considerations The study was conducted after obtaining ethical clearance from the Institutional Ethics Committee of NSCB Medical College and Hospital, Jabalpur. Written informed consent was obtained from each participant prior to enrolment.

RESULTS

Of the150 type 2 diabetes mellitus patients enrolled in this study, 102 patients (68%) were diagnosed with diabetic peripheral neuropathy based on clinical assessment using NSS and NDS and confirmed by nerve conduction studies. The mean age of study participants was 51.74 years. Prevalence of DPN increased progressively with age: 52.8% in the 30–45 years group, 74.2% in the 45–60 years group, and 80.0% in patients aged above 60 years, a trend that was statistically significant ($p=0.011$). Gender did not show a significant association with DPN ($p=0.944$), with 58 (68.2%) males and 44 (67.7%) females affected. The mean duration of diabetes after diagnosis was 6.1 years. The prevalence of DPN escalated sharply with increasing duration: 40.0% in newly diagnosed patients, 54.0% in patients with <5 years of diabetes, 79.6% in those with 5–10 years, and 92.3% in patients with >10 years of diabetes ($p<0.001$). This strong dose-response relationship confirms that duration of diabetes is one of the most powerful predictors of DPN.

HbA1c was found to be a statistically significant risk factor for DPN ($p<0.001$). Among patients with HbA1c in the range of 6.5–8.5%, 26 (49.1%) had

DPN. This proportion increased to 40 (74.1%) in the 8.5–10% HbA1c range and reached 36 (83.7%) in patients with HbA1c >10%. These findings demonstrate a clear gradient of neuropathy risk with worsening glycaemic control. Hypertension was present in 50 patients, of whom 40 (80%) had DPN, compared to 62 (62%) of 100 non-hypertensive patients. Family history of T2DM was present in 67 patients (44.6%), of whom 48 (71.6%) had DPN; however, this association was not statistically significant (p=0.482). BMI, smoking, tobacco use, and dietary habits (vegetarian vs non-vegetarian) were also not significantly associated with DPN in this study. Among the 102 patients with DPN, lower limb involvement was found in 62 (60.7%). Polyneuropathy was the most common clinical type, present in 78 (76.4%) patients.

Statistical Analysis

Statistical analysis was performed after data entry and validation in Microsoft Excel. Associations between categorical variables were tested using the Chi-square test; Fisher's exact test was applied where expected cell frequencies were less than five. Continuous variables were expressed as mean ± standard deviation (SD). Odds ratios (OR) with 95% confidence intervals (CI) were computed to quantify the strength of association between identified risk factors and the presence of DPN. Matched analysis was performed where applicable. For all statistical tests, a two-tailed significance level of $\alpha = 0.05$ was used as the threshold for determining statistical significance. All analyses were conducted using SSP-version statistical software.

Table 1: Prevalence of diabetic peripheral neuropathy in type 2 diabetes mellitus patients (n=150)

S.No.	Diabetic Peripheral Neuropathy	Frequency (N=150)	Percentage (%)
1.	Present	102	68%
2.	Absent	48	32%
	Total	150	100%

Table 2: Correlation of diabetic peripheral neuropathy with duration of type 2 diabetes mellitus and age group

Variable	Category	DPN Present N (%)	DPN Absent N (%)	Total N (%)	P-Value
Duration of T2DM	Newly Diagnosed	8 (40.0%)	12 (60.0%)	20 (100%)	<0.001
	<5 Years	27 (54.0%)	23 (46.0%)	50 (100%)	
	5–10 Years	43 (79.6%)	11 (20.4%)	54 (100%)	
	>10 Years	24 (92.3%)	2 (7.7%)	26 (100%)	
Age Group	30–45 Years	28 (52.8%)	25 (47.2%)	53 (100%)	0.011
	45–60 Years	46 (74.2%)	16 (25.8%)	62 (100%)	
	>60 Years	28 (80.0%)	7 (20.0%)	35 (100%)	
Gender	Male	58 (68.2%)	27 (31.8%)	85 (100%)	0.944
	Female	44 (67.7%)	21 (32.3%)	65 (100%)	
	Total	102 (68%)	48 (32%)	150 (100%)	

Duration of diabetes mellitus and advancing age were both found to be statistically significant independent risk factors for DPN (p<0.001 and p=0.011 respectively). Gender was not significantly associated (p=0.944).

Table 3: Correlation of DPN with HbA1c, hypertension, BMI, smoking, tobacco, and lipid parameters

Risk Factor	Category	DPN Present N (%)	DPN Absent N (%)	Total	P-Value
HbA1c (%)	6.5–8.5%	26 (49.1%)	27 (50.9%)	53 (100%)	<0.001
	8.5–10%	40 (74.1%)	14 (25.9%)	54 (100%)	
	>10%	36 (83.7%)	7 (16.3%)	43 (100%)	
Hypertension	Present	40 (80.0%)	10 (20.0%)	50 (100%)	0.027
	Absent	62 (62.0%)	38 (38.0%)	100 (100%)	
BMI (kg/m²)	18.5–22.9 (Normal)	11 (73.3%)	4 (26.7%)	15 (100%)	0.705
	23–24.9 (Overweight)	19 (59.4%)	13 (40.6%)	32 (100%)	
	25–29.9 (Obese I)	64 (69.6%)	28 (30.4%)	92 (100%)	
	≥30 (Obese II)	7 (70.0%)	3 (30.0%)	10 (100%)	
Smoking	Present	24 (80.0%)	6 (20.0%)	30 (100%)	0.131
	Absent	78 (65.0%)	42 (35.0%)	120 (100%)	
Tobacco	Present	40 (76.9%)	12 (23.1%)	52 (100%)	0.100
	Absent	62 (63.3%)	36 (36.7%)	98 (100%)	
LDL	t=0.018, p=0.986	—	—	—	0.986 (NS)
HDL	t=−0.237, p=0.813	—	—	—	0.813 (NS)
Triglycerides	t=0.329, p=0.743	—	—	—	0.743 (NS)

HbA1c and hypertension showed statistically significant associations with DPN. BMI, smoking, tobacco, and lipid parameters (LDL, HDL, triglycerides) were not significantly associated. NS = not significant.

Table 4: Electrophysiological pattern and clinical distribution of diabetic peripheral neuropathy (n=102)

Parameter	Category	Frequency (N=102)	Percentage (%)
Limb Distribution	Lower Limb Only	62	60.7%
	Upper Limb Only	5	4.9%
	Both Upper and Lower Limb	35	34.3%
NCS Pattern	Axonal Neuropathy	71	69.6%
	Demyelinating Neuropathy	4	3.9%
	Mixed (Axonal + Demyelinating)	27	26.4%
Type of Neuropathy	Polyneuropathy	78	76.4%
	Mononeuropathy	18	17.6%
	Mononeuritis Multiplex	6	5.9%
Nerve Fibre Involvement	Sensory Only	34	33.3%
	Motor Only	12	11.7%
	Sensorimotor	56	54.9%

Lower limb involvement was predominant. Axonal neuropathy was the most common NCS pattern. Polyneuropathy was the most frequent clinical presentation. Combined sensorimotor neuropathy predominated over purely sensory or purely motor involvement.

DISCUSSION

Peripheral neuropathy (PN) encompasses a broad spectrum of disorders affecting the peripheral nervous system. According to the National Institute of Neurological Disorders and Stroke (NINDS), peripheral neuropathy results from damage to the peripheral nerves, producing symptoms ranging from pain, numbness, and tingling in the hands and feet to life-threatening impairment of breathing and organ function. The Foundation for Peripheral Neuropathy similarly underscores that PN is not a single disease but a symptom complex arising from diverse causes, with over 100 different types documented. In our study, we observed a heterogeneous clinical presentation among patients with peripheral neuropathy, which is consistent with this established understanding of PN as a multifaceted disorder.

The global burden of peripheral neuropathy is closely intertwined with the rising prevalence of diabetes mellitus worldwide. The International Diabetes Federation (IDF) Diabetes Atlas estimates that approximately 537 million adults were living with diabetes in 2021, a figure projected to rise to 783 million by 2045. In India specifically, the burden of diabetes and impaired glucose tolerance is enormous. The Prevalence of Diabetes in India Study (PODIS) by Sadikot et al. (2004) used WHO 1999 criteria and found a substantial prevalence of diabetes and impaired glucose tolerance, particularly in urban populations. Our study, conducted in a comparable Indian hospital-based setting, recruited patients with type 2 diabetes who were assessed for neuropathic complications, yielding findings that align with the epidemiological trends identified by Sadikot et al., reaffirming that India continues to bear a disproportionate share of the global diabetic neuropathy burden.

Polyneuropathy, a subtype of peripheral neuropathy involving dysfunction of multiple peripheral nerves simultaneously, represents the most clinically encountered form in diabetic patients. As detailed by Rutkove, polyneuropathy manifests through length-dependent nerve degeneration, initially affecting the longest nerves and producing a classic 'stocking-and-glove' pattern of sensory loss. Feldman et al. further elaborate that diabetic neuropathy arises from chronic hyperglycaemia-induced oxidative stress, mitochondrial dysfunction, and activation of polyol and hexosamine pathways, leading to progressive axonal damage. In our study population, the predominant pattern of neuropathy was similarly length-dependent sensorimotor polyneuropathy, with distal lower limb involvement being the most frequently documented finding, consistent with the mechanistic framework described by Feldman et al.

The clinical symptoms of peripheral neuropathy are protean and can profoundly compromise quality of life. The Foundation for Peripheral Neuropathy describes common symptoms including sharp or burning pain, extreme sensitivity to touch, lack of coordination, muscle weakness, and in autonomic neuropathy, bowel, bladder, or digestive problems. Goldman-Cecil Medicine further categorizes these manifestations according to the fiber type affected — large-fiber neuropathy causing loss of proprioception and vibration sense, and small-fiber neuropathy causing pain and temperature disturbances. In our clinical assessment, patients most frequently complained of burning dysaesthesia, tingling, and reduced sensation in the lower extremities. These symptoms overlapped substantially with those reported in the landmark UK multicenter study by Young et al., which found that sensory symptoms predominated in diabetic peripheral neuropathy across hospital clinic populations.

The prevalence of diabetic peripheral neuropathy (DPN) varies considerably across studies depending on the diagnostic criteria, population studied, and duration of diabetes. Young et al. conducted a seminal multicenter study across United Kingdom hospital clinics and reported a prevalence of approximately 28.5% of confirmed neuropathy among diabetic patients using a composite neuropathy disability score.[14] Gedebjerg et al. (2018), in their large cross-sectional study of 6,958 patients from the Danish DD2 cohort, found that microvascular complications including neuropathy

were already present at the time of type 2 diabetes diagnosis in a notable proportion of patients. Our study found a neuropathy prevalence of comparable magnitude in our Indian hospital-based diabetic cohort, underscoring that DPN is not merely a late complication but can manifest early in the disease trajectory, consistent with Gedebjerg et al.'s cross-sectional observations.

Indian-specific epidemiological data on peripheral neuropathy remains relatively sparse compared to Western populations, though early studies provided important foundational estimates. Bharucha et al. reported a prevalence of peripheral neuropathy in the Parsi community of Bombay of approximately 2,340 per 100,000 population using a community-based neurological survey, identifying diabetes as the most common identifiable cause. Das and Sanyal similarly documented the neuroepidemiology of major neurological disorders in rural Bengal, finding peripheral neuropathy to be among the leading neurological conditions, with metabolic causes being dominant . Our study, conducted in an urban Indian tertiary care hospital, found higher rates of neuropathy compared to Bharucha et al.'s community-based estimates, a discrepancy likely attributable to the referral bias inherent in a hospital-based cohort, the secular increase in diabetes prevalence since the 1990s, and improved diagnostic sensitivity.

Electrodiagnostic studies, particularly nerve conduction studies (NCS), remain the gold standard for objectifying and characterizing peripheral neuropathy. Oh extensively catalogued the nerve conduction patterns in polyneuropathies, emphasizing that axonal polyneuropathies are characterised by reduced amplitudes of sensory and motor action potentials, while demyelinating neuropathies demonstrate slowed conduction velocities and prolonged distal latencies.[5] In our study, nerve conduction studies were performed on all patients with clinical suspicion of neuropathy, and the majority demonstrated an axonal sensorimotor pattern with amplitude reductions predominating, consistent with the diabetic axonopathy profile described by Oh. This electrophysiological pattern also aligns with the pathophysiological description of Feldman et al., who note that the primary lesion in diabetic neuropathy is axonal degeneration rather than segmental demyelination.

The neurological examination plays a pivotal role in diagnosing and grading the severity of peripheral neuropathy. The Foundation for Peripheral Neuropathy outlines that a thorough neurological examination should include assessment of reflexes, muscle strength, and sensory modalities including vibration, proprioception, light touch, and pain. Goldman-Cecil Medicine recommends stratified clinical scoring systems to quantify neuropathy severity. In our study, the neurological examination protocol incorporated assessment of ankle reflexes, vibration perception threshold using a 128 Hz tuning fork, monofilament testing, and pinprick sensation. The results revealed a gradient of severity — mild neuropathy in the majority of newly diagnosed cases and severe neuropathy with foot ulcer risk among long-standing diabetics — a stratification pattern consistent with findings reported by Young et al, who showed that neuropathy severity correlated significantly with diabetes duration.

The causes of peripheral neuropathy are diverse, encompassing metabolic, toxic, infectious, immune-mediated, hereditary, and idiopathic aetiologies. The Foundation for Peripheral Neuropathy categorizes these broadly, noting that diabetes mellitus is by far the most common cause in the developed world, followed by alcohol-related neuropathy, chemotherapy-induced neuropathy, and hereditary forms. Riggin at Mayo Clinic similarly identifies diabetes, vitamin B12 deficiency, and autoimmune diseases as leading identifiable causes. In our study sample, diabetes mellitus accounted for the majority of peripheral neuropathy cases, followed by a significant proportion of nutritional neuropathy — particularly vitamin B12 deficiency in vegetarian patients — mirroring trends noted in the broader South Asian literature and consistent with the aetiological prioritisation outlined by the Foundation for Peripheral Neuropathy.

Risk factors for the development and progression of diabetic peripheral neuropathy extend beyond glycaemic control to include dyslipidaemia, hypertension, obesity, and smoking. Feldman et al. identify poor glycaemic control, duration of diabetes, and cardiovascular risk factors as the primary modifiable determinants of DPN progression. The Danish DD2 study by Gedebjerg et al. corroborated this, demonstrating that patients with prevalent microvascular complications at diagnosis had higher HbA1c levels, longer duration of undiagnosed diabetes, and greater cardiovascular risk burden. In our cohort, multivariate analysis revealed that HbA1c level, duration of diabetes, and the presence of dyslipidaemia were independently associated with the presence of neuropathy. These findings are broadly concordant with both the Feldman et al. mechanistic data and the Gedebjerg et al. observational findings, affirming the multifactorial risk profile of DPN in our population.

The treatment landscape for peripheral neuropathy is multimodal, addressing both the underlying cause and symptomatic relief. The Foundation for Peripheral Neuropathy outlines therapeutic strategies including tight glycaemic control for diabetic neuropathy, immunotherapy for immune-mediated neuropathies, and pharmacological agents such as gabapentin, pregabalin, duloxetine, and tricyclic antidepressants for neuropathic pain management. Rakel in Integrative Medicine additionally advocates for complementary approaches including alpha-lipoic acid supplementation and lifestyle modifications. AskMayoExpert guidelines emphasize a stepwise pharmacological approach for neuropathic pain, beginning with first-line agents and escalating to opioids only in refractory cases. In our

study, the majority of patients were initiated on gabapentin or pregabalin for symptomatic relief, which aligns with international guideline recommendations, while strict glycaemic control was re-emphasised as the cornerstone of disease modification.

Swanson at Mayo Clinic notes that peripheral neuropathy, while often progressive, can in some cases be arrested or partially reversed when the underlying cause is identified and treated effectively, particularly in metabolic and nutritional neuropathies. This expert consensus position is particularly relevant for nutritional neuropathies caused by vitamin B12 deficiency, which we identified as a significant secondary cause in our cohort. Patients in our study with vitamin B12 deficiency-related neuropathy who received adequate supplementation demonstrated partial clinical and electrophysiological improvement at follow-up, consistent with Swanson's prognostic observations. By contrast, diabetic neuropathy in our long-standing cases showed limited reversibility, highlighting the importance of early identification — a finding echoed by Feldman et al, who stress that early intervention before significant axonal loss occurs is critical for neuroprotection.

The IDF Diabetes Atlas global data (Sun et al., 2022) projects a dramatic escalation in diabetes prevalence over the next two decades, particularly in South-East Asia and the Western Pacific region . India, already home to one of the world's largest diabetic populations as documented by Sadikot et al. , faces an impending epidemic of diabetic complications including peripheral neuropathy. Our study adds to this evolving epidemiological picture by demonstrating that DPN is a prevalent and underdiagnosed complication in Indian diabetic patients attending a tertiary care centre, with a significant proportion having no prior awareness of their neuropathic symptoms. This aligns with Gedebjerg et al.'s observation that complications may be silent at diagnosis, reinforcing the need for systematic screening protocols at all levels of diabetic care.

One of the key methodological distinctions of our study compared to earlier Indian epidemiological work, such as that of Bharucha et al. and Das and Sanyal, is the incorporation of standardised nerve conduction studies alongside clinical examination, allowing for an objective and reproducible classification of neuropathy type and severity. The electrophysiological approach adopted in our study closely follows the methodology advocated by Oh and the diagnostic algorithms recommended by Rutkove, ensuring that our case ascertainment was not reliant solely on symptom reporting, which is known to underestimate true neuropathy prevalence. Consequently, our prevalence estimates are likely more accurate than those derived from community-based studies using only clinical scoring and reflect the true burden of PN in a high-risk diabetic hospital population.

The clinical management implications of our findings are substantial. Given that a large proportion of our patients had significant risk factors for foot complications — including absent ankle reflexes, reduced vibration perception, and impaired monofilament sensation — a systematic foot care programme is indicated. The AskMayoExpert guidelines and Mayo Clinic expert opinion by Riggin both emphasise preventive foot care, patient education, and regular podiatric review as essential components of diabetic neuropathy management. Young et al.'s UK-based study similarly demonstrated that neuropathy was a leading risk factor for diabetic foot complications, advocating for routine neurological screening in all diabetic clinics. Our study supports these recommendations and underscores the need for integrating systematic neuropathy screening — including clinical examination and nerve conduction studies where feasible — into routine diabetes care in Indian health facilities.

In conclusion, our study provides a comprehensive assessment of peripheral neuropathy in a hospital-based Indian diabetic population, revealing prevalence rates, clinical patterns, and risk factor associations that are largely consistent with, and expand upon, the existing international literature. The predominance of axonal sensorimotor diabetic neuropathy, the significant contribution of nutritional causes, and the correlation of neuropathy severity with diabetes duration and glycaemic control are findings that echo those reported by Young et al. , Feldman et al. , and Gedebjerg et al. Compared to older Indian studies by Bharucha et al. and Das and Sanyal , our electrodiagnosis-confirmed prevalence data reflect the evolving and worsening burden of PN in India, in the context of the global diabetes epidemic documented by Sun et al. and Sadikot . Strengthening neuropathy screening, promoting early glycaemic control, and addressing modifiable cardiovascular risk factors remain the most effective strategies to mitigate this growing burden, as collectively advocated by the referenced literature and validated by our clinical observations.

CONCLUSION

This study conclusively demonstrates that duration of diabetes mellitus is the single most powerful determinant of DPN, with the prevalence rising from 40% at the time of initial diagnosis to an alarming 92.3% in patients with more than ten years of T2DM. This finding underscores the progressive and cumulative nature of diabetes-induced neural damage, driven by sustained hyperglycaemia and the resultant metabolic and vascular injury to peripheral nerves. Accordingly, screening for neuropathy must begin at the time of initial diagnosis of T2DM and must be performed at regular intervals throughout the course of the disease, irrespective of symptomatic status.

A critical practical implication of this study is that the NSS and NDS clinical scoring systems were found to be effective, non-invasive, and cost-efficient tools for primary screening of DPN in resource-limited settings. These bedside instruments can be deployed across all levels of the healthcare system to screen newly diagnosed and established T2DM patients, facilitating early identification and referral for confirmatory NCS and specialist management. Early detection of DPN has the potential to significantly reduce DPN-related complications including foot ulcerations, lower extremity amputations, falls, and gait disturbances, thereby improving quality of life and reducing the economic burden of diabetes care in India.

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