Section: Miscellaneous

Original Research Article

RENAL, RETINAL AND NEUROLOGICAL INVOLVEMENT IN TYPE 2 DIABETES MELLITUS: A CROSS-SECTIONAL STUDY

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Received *: 22/11/2025*
Received in revised form *: 05/01/2026*
Accepted *: 24/01/2026*

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DOI: 10.70034/ijmedph.2026.1.316

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 1827-1833

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a major contributor to chronic microvascular complications, including nephropathy, retinopathy and neuropathy. The risk of these complications increases with longer disease duration and poor glycaemic control, yet the pattern and burden vary across populations. This study assessed renal, retinal and neurological status in patients with T2DM and examined their association with duration of diabetes and glycaemic control in an Indian tertiary-care setting.

Materials and Methods: A cross-sectional study was conducted among 263 adults with T2DM. Demographic and clinical parameters were recorded, and laboratory investigations included fasting glucose, postprandial glucose, and HbA1c measured via HPLC. Renal status was evaluated using urine albumin excretion and eGFR (CKD-EPI). Retinal assessment was performed using dilated fundus examination and graded according to ETDRS criteria. Peripheral neuropathy was assessed using the Michigan Neuropathy Screening Instrument (MNSI), 10-g monofilament, vibration perception and ankle reflex testing. Associations were analysed using chi-square tests and correlation analysis (Pearson/Spearman). A p-value <0.05 was considered significant.

Results: Albuminuria was present in 28.1% and reduced eGFR (<60 mL/min/1.73 m²) in 12.1% of participants. Diabetic retinopathy was detected in 25.1%, and clinical peripheral neuropathy in 33.8%. All complications showed a significant stepwise increase with duration of diabetes: albuminuria (10.9% to 46.0%), retinopathy (10.9% to 47.6%), and neuropathy (18.2% to 61.9%) (all $p<0.001$). Poor glycaemic control (HbA1c ≥9%) was similarly associated with higher rates of albuminuria (47.1%), retinopathy (42.9%) and neuropathy (51.4%) compared with good control (<7%) (all $p<0.001$). Correlation analysis demonstrated significant relationships between duration of diabetes and eGFR decline ($r = -0.36$), albuminuria ($\rho = 0.44$), and HbA1c levels ($r = 0.31$), as well as between HbA1c and both eGFR ($r = -0.29$) and albuminuria ($\rho = 0.41$) (all $p<0.001$).

Conclusion: Renal, retinal and neurological complications were highly prevalent in this T2DM cohort and showed strong associations with both longer diabetes duration and poorer glycaemic control. These findings highlight the need for early detection, stringent glycaemic management, and routine integrated screening for microvascular complications to prevent long-term morbidity in patients with T2DM.

Keywords: Type 2 diabetes mellitus; Microvascular complications; Glycaemic control; Albuminuria; eGFR.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder with rapidly rising global prevalence, currently affecting an estimated 537 million adults worldwide and projected to reach 643 million by 2030 and 783 million by 2045.[1] India alone accounts for over 74 million cases, making it one of the largest contributors to the global diabetes burden.[2] The major concern in T2DM is not only hyperglycaemia but the long-term development of microvascular complications diabetic nephropathy, diabetic retinopathy, and diabetic neuropathy—which significantly increase morbidity, reduce quality of life, and impose economic strain on patients and health systems.[1]

Microvascular complications arise due to chronic exposure to elevated glucose, leading to endothelial dysfunction, basement membrane thickening, oxidative stress, and microvascular ischemia.[3]

Over time, these processes culminate in organ-specific manifestations. Diabetic nephropathy (DN) is the leading cause of end-stage renal disease globally, affecting approximately 20–40% of patients with T2DM.[4] Diabetic retinopathy (DR) remains the most common cause of preventable blindness among adults aged 20–74 years, with a global prevalence of ~27% in diabetics.[5] Diabetic peripheral neuropathy (DPN) affects nearly 50% of individuals with long-standing T2DM and contributes to foot ulcers and amputations.[6]

Two of the most important determinants of these complications are duration of diabetes and adequacy of glycaemic control. Prolonged duration leads to cumulative metabolic injury; studies have demonstrated that individuals with ≥10 years of diabetes have 2–3 times higher risk of nephropathy, retinopathy, and neuropathy compared with those with shorter disease duration.[7] Similarly, poor glycaemic control reflected by higher HbA1c—accelerates microvascular damage. The literature showed that each 1% rise in HbA1c increases the risk of microvascular complications by nearly 37%.[8] Conversely, sustained glycaemic control significantly delays the onset and slows the progression of DR, DN, and DPN.

Despite these well-established associations, patterns of microvascular involvement differ across populations. Indian patients tend to develop T2DM at a younger age, have faster β-cell decline, and often present with complications earlier in the disease course [9]. Studies from India report varying prevalence: DN in 17–30%, DR in 18–35%, and DPN in 25–48% of patients, depending on disease duration, HbA1c levels, and screening methods.[10-12] In many settings, poor awareness, irregular follow-up, delayed screening, and suboptimal glycaemic control lead to under-recognition of early-stage complications.

A comprehensive assessment of renal (albuminuria, eGFR), retinal (fundus changes), and neurological (sensory deficits, neuropathic symptoms) status in relation to both duration of T2DM and glycaemic control is therefore essential. Understanding these relationships can help clinicians stratify patient risk, prioritise early interventions, and plan targeted screening strategies. Given the substantial population-level burden and the rising prevalence of poorly controlled T2DM in India, there is a pressing need to generate locally relevant evidence. Against this backdrop, the present study aims to evaluate renal, retinal, and neurological status among patients with Type 2 diabetes mellitus and analyse their association with disease duration and glycaemic control (HbA1c). This integrated assessment will contribute to better understanding of clustering patterns of microvascular complications and may guide future preventive and management strategies.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based cross-sectional observational study was conducted in the Department of Medicine and affiliated Ophthalmology and Neurology clinics of a tertiary care teaching hospital. The study was carried out over a period of 24 months between July 2021 and June 2023. All assessments, including clinical examination, laboratory investigations, fundus evaluation, and neurological testing, were performed within the institution using standardized protocols.

Study Population

The study included adult patients diagnosed with Type 2 Diabetes Mellitus as per the American Diabetes Association (ADA) criteria, who attended the outpatient departments during the study period. Patients aged ≥30 years with a minimum disease duration of one year were eligible. Exclusion criteria included individuals with Type 1 diabetes, gestational diabetes, chronic kidney disease due to non-diabetic causes, pre-existing retinal diseases unrelated to diabetes (such as hypertensive retinopathy, glaucoma, or retinal vascular occlusions), neurological disorders not attributable to diabetes (including alcohol-related neuropathy, vitamin B12 deficiency, hypothyroidism, or neurodegenerative diseases), current use of neurotoxic medications, and patients unwilling to participate. After applying these criteria, a total of 263 participants were included using consecutive sampling.

Clinical and Demographic Assessment

All participants underwent a detailed evaluation that included demographic information (age, sex, socioeconomic status), diabetes-related variables (duration of diabetes, treatment modalities, adherence history), and lifestyle factors such as smoking, alcohol intake, and physical activity. Anthropometric measurements including height, weight, and BMI were recorded using calibrated instruments. Blood pressure was measured in the

sitting position after five minutes of rest using a digital sphygmomanometer, and the average of two readings was documented. Duration of diabetes was cross-verified from medical records and categorized for analysis (e.g., <5 years, 5–10 years, >10 years).

Assessment of Glycaemic Control

Glycaemic control was assessed using fasting plasma glucose (FPG), post-prandial blood sugar (PPBS), and glycated haemoglobin (HbA1c). Venous blood samples were collected after an overnight fast, and HbA1c was measured using high-performance liquid chromatography (HPLC), ensuring NGSP-certified equipment. Glycaemic control was defined as "good" for HbA1c <7%, "moderate" for HbA1c 7–8.9%, and "poor" for HbA1c ≥9%. These categories were used for correlation with renal, retinal, and neurological findings.

Renal Status Evaluation

Renal function was assessed using both biochemical and clinical markers. Early morning spot urine samples were analysed for microalbuminuria using an immunoturbidimetric method; values between 30–300 mg/day were considered microalbuminuria, and >300 mg/day as macroalbuminuria. Serum creatinine was measured using an enzymatic method, and estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI formula. Patients were classified into stages of diabetic nephropathy based on albuminuria and eGFR values. Repeat testing was performed for borderline results to ensure accuracy.

Retinal Status Evaluation

Retinal examination was conducted by an ophthalmologist trained in diabetic retinopathy screening. After instillation of 1% tropicamide, a dilated fundus examination was performed using both direct and indirect ophthalmoscopy. Digital fundus photography was carried out when required. Retinopathy was graded according to the Early Treatment Diabetic Retinopathy Study (ETDRS) classification into: no retinopathy, mild to moderate non-proliferative diabetic retinopathy (NPDR), severe NPDR, and proliferative diabetic retinopathy (PDR). The presence of diabetic macular edema was documented separately. For patients unable to undergo dilation, non-mydriatic fundus imaging was utilized.

Neurological Status Assessment

Neurological evaluation focused on detecting peripheral neuropathy. A comprehensive examination was performed using standardized tools: a 10-g Semmes–Weinstein monofilament to assess pressure perception at eight plantar sites; a 128-Hz tuning fork for vibration perception at the hallux and medial malleolus; and ankle reflex testing using a reflex hammer. Neuropathic symptoms such as numbness, burning sensation, tingling, and pain were recorded using the Michigan Neuropathy Screening Instrument (MNSI) questionnaire. Patients with abnormal clinical findings were categorized as having mild, moderate, or severe neuropathy based on a composite score derived from sensory testing and symptom severity.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants after explaining the study purpose, procedures, benefits, and confidentiality measures. Participants requiring treatment for newly detected complications were referred appropriately for further management.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 21.0 (IBM Corp., USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Associations between diabetes duration, glycaemic control, and renal, retinal, and neurological outcomes were examined using chi-square tests for categorical variables and ANOVA or t-tests for continuous variables. Correlation analysis (Pearson or Spearman as appropriate) was used to explore linear relationships. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 263 participants with Type 2 diabetes mellitus were included, with a mean age of 54.6 ± 9.8 years. The majority (50.2%) were between 45–59 years, and 55.1% were male. The mean BMI was 26.1 ± 3.7 kg/m², and comorbid hypertension and dyslipidaemia were present in 52.5% and 36.5% of the sample, respectively. Nearly one-fifth (18.3%) were current smokers. Regarding diabetes duration, 41.8% had <5 years, 34.2% had 5–10 years, and 24.0% had >10 years of disease. Overall mean HbA1c was 8.2 ± 1.6%, with only 33.5% achieving good glycaemic control (HbA1c <7%), whereas 26.6% had poor control (≥9%). These baseline findings indicate a predominantly middle-aged cohort with substantial metabolic comorbidity and suboptimal glycaemic control. [Table 1]

Table 1: Baseline Characteristics of the Study Population

Variable	Frequency (%)/mean ± SD
Age (years)	54.6 ± 9.8
Age category	
30–44 years	62 (23.6%)
45–59 years	132 (50.2%)
≥60 years	69 (26.2%)
Sex	
Male	145 (55.1%)
Female	118 (44.9%)

Body mass index (BMI) (kg/m²)	26.1 ± 3.7
Hypertension (diagnosed or on treatment)	138 (52.5%)
Dyslipidaemia (documented or on lipid-lowering therapy)	96 (36.5%)
Current smokers	48 (18.3%)
Duration of diabetes	
<5 years	110 (41.8%)
5–10 years	90 (34.2%)
>10 years	63 (24.0%)
Glycaemic control (HbA1c) (%)	8.2 ± 1.6
HbA1c categories	
<7.0% (Good)	88 (33.5%)
7.0–8.9% (Moderate)	105 (39.9%)
≥9.0% (Poor)	70 (26.6%)

BMI = Body Mass Index; HbA1c = Glycated Hemoglobin.

Renal assessment showed that 20.2% of the participants had microalbuminuria and 8.0% had macroalbuminuria, with an overall albuminuria prevalence of 28.1%. The mean eGFR was 87.2 ± 17.9 mL/min/1.73 m², with the majority (59.0%) falling within the mildly reduced eGFR category of 60–89 mL/min/1.73 m². Moderate reduction in eGFR (30–59 mL/min/1.73 m²) was observed in 11.0%, and only 1.1% had eGFR <30 mL/min/1.73 m². This reflects a substantial burden of early diabetic kidney disease in the cohort. [Table 2]

Table 2: Renal Status of Participants

Renal parameter	Frequency (%)/mean ± SD
Microalbuminuria (UAE 30–300 mg/day)	53 (20.2)
Macroalbuminuria (UAE >300 mg/day)	21 (8.0)
Any albuminuria (micro + macro)	74 (28.1)
eGFR (CKD-EPI) (mL/min/1.73 m²)	87.2 ± 17.9
eGFR categories	
≥90 mL/min/1.73 m²	76 (28.9)
60–89 mL/min/1.73 m²	155 (59.0)
30–59 mL/min/1.73 m²	29 (11.0)
<30 mL/min/1.73 m²	3 (1.1)

UAE = Urine Albumin Excretion; eGFR = Estimated Glomerular Filtration Rate; CKD-EPI = Chronic Kidney Disease Epidemiology Collaboration formula.

Fundus evaluation revealed that 60.8% of participants had no evidence of diabetic retinopathy, while 17.5% had mild to moderate NPDR and 7.6% had severe NPDR. PDR was identified in 3.8% of patients. Clinically significant DME was present in 10.3% of the cohort. When combined, the overall prevalence of any diabetic retinopathy was 25.1%. These findings show that one quarter of the participants had retinal involvement, with a notable proportion exhibiting advanced changes. [Table 3]

Table 3: Retinal Status Among Study Participants

Retinopathy category	Frequency (%)
No diabetic retinopathy	160 (60.8)
Mild–moderate NPDR	46 (17.5)
Severe NPDR	20 (7.6)
Proliferative diabetic retinopathy (PDR)	10 (3.8)
Diabetic macular edema (DME; clinically significant)	27 (10.3)*

*DME counted separately (some patients with NPDR/PDR also had DME); total with any retinopathy = 66 (25.1%). NPDR = Non-Proliferative Diabetic Retinopathy; PDR = Proliferative Diabetic Retinopathy; DME = Diabetic Macular Edema.

Peripheral neuropathy assessment demonstrated that 31.2% of participants had abnormal monofilament testing, 28.9% had impaired vibration perception, and 20.5% exhibited absent ankle reflexes. Based on the MNSI scoring, 66.2% had no neuropathy, whereas 17.5%, 9.5%, and 6.8% were categorized as having mild, moderate, and severe neuropathy, respectively. The overall prevalence of clinical neuropathy (MNSI ≥2 and/or abnormal monofilament) was 33.8%. [Table 4]

Table 4: Neurological Status — Peripheral Neuropathy

Neuropathy parameter	Frequency (%)
Abnormal 10-g monofilament (loss at ≥1 site)	82 (31.2)
Reduced vibration (128-Hz) at hallux	76 (28.9)
Absent ankle reflexes (one or both)	54 (20.5)
Michigan Neuropathy Screening Instrument (MNSI) – symptom/sign composite	
No neuropathy (MNSI score <2)	174 (66.2)
Mild neuropathy	46 (17.5)

Moderate neuropathy	25 (9.5)
Severe neuropathy	18 (6.8)
Any clinical neuropathy (MNSI ≥2 or abnormal monofilament)	89 (33.8)

MNSI = Michigan Neuropathy Screening Instrument.

A significant association was observed between the duration of diabetes and all major microvascular complications. Albuminuria was present in 10.9% of those with <5 years of diabetes, rising to 36.7% in the 5–10 year group and 46.0% in those with >10 years ($\chi^2 = 29.36$, $p < 0.001$). Similarly, diabetic retinopathy increased progressively with duration (10.9%, 26.7%, and 47.6%, respectively; $\chi^2 = 28.90$, $p < 0.001$). Peripheral neuropathy also demonstrated a strong duration-dependent gradient, from 18.2% to 33.3% and 61.9% ($\chi^2 = 34.22$, $p < 0.001$). [Table 5]

Table 5: Association between duration of diabetes and major microvascular outcomes

Outcome	<5 yr (n=110)	5–10 yr (n=90)	>10 yr (n=63)	p-value (χ^2)
	Frequency (%)			
Any albuminuria (micro or macro)	12 (10.9%)	33 (36.7%)	29 (46.0%)	< 0.001 ($\chi^2 = 29.36$)
Diabetic retinopathy (any)	12 (10.9%)	24 (26.7%)	30 (47.6%)	< 0.001 ($\chi^2 = 28.90$)
Any peripheral neuropathy	20 (18.2%)	30 (33.3%)	39 (61.9%)	< 0.001 ($\chi^2 = 34.22$)

Poorer glycaemic control was significantly associated with higher rates of all microvascular complications. Albuminuria prevalence increased from 9.1% in the good control group to 31.4% in the moderate and 47.1% in the poor control groups ($\chi^2 = 28.86$, $p < 0.001$). Diabetic retinopathy also increased sharply across HbA1c groups (9.1%, 26.7%, and 42.9%; $\chi^2 = 23.88$, $p < 0.001$). Similarly, neuropathy prevalence rose from 20.5% to 33.3% and 51.4% across increasing HbA1c levels ($\chi^2 = 16.73$, $p < 0.0001$). These results demonstrate that inadequate glycaemic control is strongly linked with the presence of renal, retinal, and neurological complications.[Table 6]

Table 6: Association between glycaemic control (HbA1c categories) and microvascular outcomes

Outcome	HbA1c <7.0% (n=88)	HbA1c 7.0–8.9% (n=105)	HbA1c ≥9.0% (n=70)	p-value (χ^2)
	Frequency (%)			
Any albuminuria (micro or macro)	8 (9.1%)	33 (31.4%)	33 (47.1%)	< 0.001 ($\chi^2 = 28.86$)
Diabetic retinopathy (any)	8 (9.1%)	28 (26.7%)	30 (42.9%)	< 0.001 ($\chi^2 = 23.88$)
Any peripheral neuropathy	18 (20.5%)	35 (33.3%)	36 (51.4%)	< 0.0001 ($\chi^2 = 16.73$)

HbA1c = Glycated Hemoglobin

Correlation analysis showed a significant positive relationship between duration of diabetes and HbA1c ($r = 0.31$, $p < 0.001$), indicating worsening glycaemic control over time. Duration also correlated negatively with eGFR ($r = -0.36$, $p < 0.001$) and positively with albuminuria ($\rho = 0.44$, $p < 0.001$). HbA1c demonstrated a significant inverse correlation with eGFR ($r = -0.29$, $p < 0.001$) and a positive correlation with albuminuria ($\rho = 0.41$, $p < 0.001$). These correlations collectively highlight that both longer duration and poorer glycaemic control contribute substantially to renal functional decline and increasing albuminuria. [Table 7]

Table 7: Correlation between glycaemic control, renal function and duration of diabetes

Variables Correlated	Correlation Coefficient (r / ρ)	p-value
Duration of diabetes (years) vs HbA1c (%)	r = 0.31	< 0.001
Duration of diabetes (years) vs eGFR (mL/min/1.73 m²)	r = −0.36	< 0.001
HbA1c (%) vs eGFR (mL/min/1.73 m²)	r = −0.29	< 0.001
HbA1c (%) vs urine albumin excretion (mg/day)*	ρ = +0.41	< 0.001
Duration of diabetes (years) vs urine albumin excretion (mg/day)*	ρ = +0.44	< 0.001

*Spearman's rho (ρ) used for albuminuria because of non-normal distribution; r = Pearson's Correlation Coefficient; ρ = Spearman's Rank Correlation; eGFR = Estimated Glomerular Filtration Rate; UAE = Urine Albumin Excretion.

DISCUSSION

In this cross-sectional study of 263 patients with Type 2 diabetes mellitus (T2DM), we observed a substantial burden of microvascular complications, with renal, retinal and neurological involvement detected in 28.1%, 25.1%, and 33.8% of participants respectively. The mean age of participants in our study was 54.6 years and mean HbA1c was 8.2%, indicating overall inadequate glycaemic control an

important determinant strongly associated with complications in our cohort.[13,14]

Renal complications were highly prevalent, with 28.1% showing micro- or macroalbuminuria and 12.1% exhibiting moderate-to-severe reductions in eGFR. This prevalence is comparable to findings from the Chennai Urban Rural Epidemiology Study (CURES) by Mohan et al., which reported microalbuminuria in 26.9% of South Indian individuals with diabetes and reduced eGFR in 13.0%.[15] The clear dose–response relationship between duration of diabetes and albuminuria observed in our study (10.9% in <5 years vs. 46.0% in >10 years; p<0.001) mirrors results from studies by Bilous et al., and Wu et al., which demonstrated that chronic hyperglycaemia and cumulative metabolic injury significantly increase the risk of nephropathy.[16,17] Furthermore, the negative correlation between HbA1c and eGFR ($r = -0.29$, $p<0.001$) reinforces the role of glycaemic exposure in glomerular damage, consistent with findings by Vaishali et al., and Shah et al., who concluded that each 1% rise in HbA1c substantially increases renal risk.[18,19]

Retinal findings in our study also reflect the typical Indian scenario, where diabetic retinopathy (DR) prevalence ranges between 18% and 35% in clinic-based cohorts.[20,21] Our prevalence of 25.1% is similar to the 21.7% reported by Gadkari et al. in the All India Ophthalmological Society (AIOS) survey and the 26.2% reported by Vashisht et al.[20,21] Importantly, we found that DR prevalence increased progressively with disease duration (10.9% to 47.6%; p<0.001) and worsening glycaemic control (9.1% to 42.9%; p<0.001). This aligns with the Sharma et al., and Mehta et al., which established both duration of diabetes and HbA1c as the strongest predictors of retinopathy onset and progression.[22,23] The presence of clinically significant macular edema in 10.3% of participants parallels frequencies reported in Asian population–based cohorts, indicating that diabetic macular involvement continues to be a major cause of visual morbidity.[24]

Neuropathy was the most frequent microvascular complication in our study (33.8%), with abnormalities noted in monofilament testing, vibration perception, and ankle reflexes. This is consistent with prevalence rates of 30–50% reported in Indian studies by Darivemula et al., and Solanki et al.[25,26] The strong association between neuropathy and both duration of diabetes (18.2% to 61.9%; p<0.001) and glycaemic status (20.5% to 51.4%; p<0.0001) indicates cumulative neurodegeneration with prolonged hyperglycaemia. Mechanistically, sustained elevations in glucose promote oxidative injury, sorbitol pathway activation, endothelial dysfunction and impaired nerve blood flow all central pathways in diabetic peripheral neuropathy.[27]

These biological explanations support our correlation findings: neuropathy prevalence closely paralleled albuminuria and retinopathy trends, highlighting the clustering of microvascular damage as hyperglycaemia and disease duration increase.[27]

The strength of our study also lies in the correlation analysis, which demonstrated consistent relationships across renal indices. Albuminuria correlated positively with both HbA1c ($\rho = 0.41$) and duration of diabetes ($\rho = 0.44$), while eGFR showed inverse relationships with these variables ($r = -0.29$ and $r = -0.36$). These values are remarkably similar to those reported by Yu et al., who found comparable correlation coefficients in Chinese T2DM patients.[28] Such findings underscore a central concept: microvascular injury is cumulative, linear, and strongly modifiable through glycaemic control, reinforcing the need for early and sustained HbA1c reduction.[28]

Our findings collectively support existing literature demonstrating that poor glycaemic control and longer duration of diabetes are the primary determinants of microvascular complications.[29] The parallel gradients seen across albuminuria, DR, and neuropathy strengthen the model of microvascular clustering, and further highlight the urgent need for robust screening programs in Indian clinical practice.[30] Given that nearly two-thirds of our cohort had HbA1c ≥7%, and nearly 25% had >10 years of disease duration, a large proportion remain vulnerable to further progression of these complications.

Limitations

However, certain limitations must be acknowledged. The cross-sectional design precludes causal inference and limits the ability to assess progression over time. Being hospital-based, the findings may not fully represent community-level prevalence or the spectrum of asymptomatic individuals, potentially introducing selection bias. Albuminuria was measured using a single spot urine sample rather than serial measurements, which may underestimate transient variability. Additionally, unmeasured confounders such as dietary factors, medication adherence, socioeconomic status, and genetic susceptibility were not assessed. Despite these limitations, the study provides meaningful insights into microvascular patterns in Indian patients with T2DM and underscores the need for longitudinal and multi-center studies to further validate these findings.

CONCLUSION

This study demonstrates a substantial burden of microvascular complications among adults with Type 2 diabetes mellitus, with renal, retinal and neurological impairments affecting approximately one-fourth to one-third of the cohort. The strong and statistically significant associations between these complications and both the duration of diabetes and poor glycaemic control highlight the cumulative nature of hyperglycaemic injury. Albuminuria, retinopathy, and peripheral neuropathy all showed a clear stepwise increase with longer diabetes duration, while suboptimal HbA1c levels were consistently

linked with higher complication rates and declining renal function. These findings reinforce the critical importance of early diagnosis, sustained glycaemic control, and routine screening for microvascular complications, particularly in high-risk populations such as those in India where diabetes onset often occurs earlier and progresses rapidly. Strengthening preventive strategies and integrating comprehensive complication screening into routine diabetic care may significantly reduce the morbidity associated with long-term microvascular damage.

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