



Serum Magnesium and Its Association with Glycemic Control and Microvascular / Macrovascular Complications in Type 2 Diabetes Mellitus: A Cross-Sectional Study from the Bundelkhand Region

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ABSTRACT:

Background: Hypomagnesemia is frequent in type 2 diabetes mellitus (T2DM) and may aggravate insulin resistance and vascular injury; data from the Bundelkhand region are lacking.

Aims/Objectives: To determine serum magnesium levels in T2DM and their association with glycemic control and with microvascular and macrovascular complications.

Methodology: Hospital-based cross-sectional study of 250 consecutive patients with T2DM aged 30 years and above at a tertiary care teaching hospital in Jhansi (May 2025 to April 2026). Serum magnesium, HbA1c and blood glucose were estimated; complications were assessed by fundoscopy, urinary albumin, renal function tests and clinical examination. Chi-square tests, t-tests, Pearson correlation and multivariable logistic regression were applied.

Results: Hypomagnesemia (<1.6 mg/dL) was present in 61 patients (24.4%) and was associated with age, diabetes duration and body mass index (all $p < 0.001$) but not sex. Serum magnesium correlated inversely with HbA1c ($r = -0.378$). Crude odds ratios for hypomagnesemia were 2.71 for retinopathy, 2.57 for nephropathy and 2.19 for neuropathy; after adjustment only nephropathy remained significant (aOR 1.99, 95% CI 1.01–3.94). Macrovascular associations were not significant.

Conclusions: Hypomagnesemia affected one-quarter of patients and was associated with poorer glycemic control and, independently, with diabetic nephropathy.

Keywords: Hypomagnesemia; Glycemic Control; Diabetic Retinopathy; Diabetic Nephropathy; Diabetic Neuropathy; Cross-Sectional Studies



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INTRODUCTION:

India carries a disproportionate share of the global burden of type 2 diabetes mellitus (T2DM): the ICMR-INDIAB survey estimated that 101 million adults live with diabetes and a further 136 million with prediabetes [1], and the International Diabetes Federation Atlas places India among the countries with the largest and fastest-growing diabetic populations [2]. The Bundelkhand region, spanning southern Uttar Pradesh and northern Madhya Pradesh, is socio-economically disadvantaged and served by limited healthcare infrastructure, and delayed diagnosis means that chronic complications are frequently already present when patients first reach tertiary care.

The microvascular (retinopathy, nephropathy, neuropathy) and macrovascular (coronary, cerebrovascular and peripheral arterial) complications of T2DM arise from an interplay of chronic hyperglycemia, oxidative stress, low-grade inflammation and endothelial dysfunction. Because glycemic control alone does not fully account for the variation in complication burden between patients, attention has turned to modifiable micronutrient factors, of which magnesium is among the most biologically plausible. Magnesium is a cofactor for more than 300 enzymatic reactions, is required for insulin-receptor tyrosine kinase activity, regulates beta-cell calcium handling, maintains endothelial integrity and exerts antioxidant and anti-inflammatory effects [3].

Hypomagnesemia is correspondingly common in T2DM [4], with reported prevalence ranging from 11% in Palestinian primary care [5] to 19.1% in South India [6] and 44% in North India [7], depending on the diagnostic threshold, care setting and renal function. The relationship with glycemia appears bidirectional: low magnesium impairs insulin action [8], while chronic glycosuria drives renal magnesium wasting [3]. Supplementation trials report modest reductions in HbA1c [9,10], although a recent randomised trial found no effect on insulin sensitivity [11], so the causal direction remains unsettled. Hypomagnesemia has been associated with diabetic retinopathy in a meta-analysis of 17 studies [12] and in large clinical series [13,14], and with albuminuria and declining renal function [15]. For macrovascular disease the evidence is less consistent: in the largest prospective cohort, serum magnesium was inversely associated with heart failure, atrial fibrillation and microvascular events but not with myocardial infarction or stroke after adjustment [16].

Serum magnesium is not part of routine diabetes care in most Indian centres, and no data exist from Bundelkhand. Whether the associations reported elsewhere hold in a resource-limited, predominantly rural referral population with long-standing, poorly controlled disease is untested; the present study was undertaken to address this gap.

Objectives: The primary objective was to determine serum magnesium levels and the prevalence of hypomagnesemia among 250 adults with type 2 diabetes mellitus attending the Department of General Medicine of a tertiary care teaching hospital in Jhansi over the twelve months from May 2025 to April 2026. The secondary objectives were, in the same cohort and period, to determine the correlation of serum magnesium with glycated haemoglobin, fasting and postprandial blood sugar, and the association of serum magnesium status with diabetic retinopathy, nephropathy and neuropathy and with coronary artery disease, stroke and peripheral arterial disease.

MATERIALS AND METHODS:

Study design and setting:

This hospital-based, cross-sectional, observational study was carried out in the Department of General Medicine, Maharani Laxmi Bai Medical College and Associated Hospital, Jhansi, Uttar Pradesh, a tertiary care teaching hospital serving the Bundelkhand region, from May 2025 to April 2026. Patients were enrolled consecutively from the general medicine outpatient department and the hospital diabetic clinic.

Study population:

Adults aged 30 years and above with an established diagnosis of type 2 diabetes mellitus according to the American Diabetes Association criteria [17] (fasting plasma glucose ≥ 126 mg/dL, two-hour plasma glucose ≥ 200 mg/dL during an oral glucose tolerance test, HbA1c $\geq 6.5\%$, or random blood sugar ≥ 200 mg/dL with classical hyperglycaemic symptoms) who gave written informed consent were eligible. Patients were excluded if they had type 1 diabetes mellitus; chronic kidney disease of stage 4 or 5 or were receiving dialysis; any condition known to alter magnesium homeostasis (chronic diarrhoea, malabsorption syndromes or alcohol dependence); current use of magnesium supplements; pregnancy or lactation; or critical illness at the time of assessment. Consecutive patients with type 2 diabetes attending the outpatient department and diabetic clinic were screened against these criteria; those not meeting the eligibility criteria and those who declined consent were not enrolled, and recruitment continued until 250 eligible, consenting patients had been enrolled. All 250 completed the full assessment and were included in the analysis; there were no post-enrolment exclusions or missing data for the primary variables. The number of patients screened and the number declining consent were not systematically logged in the recruitment register, so the participation proportion cannot be stated precisely; this is acknowledged as a limitation.

Sample size:

The sample size was calculated using the formula for a single proportion, $n = Z^2 \times p(1 - p) / d^2$, with $Z = 1.96$ for 95% confidence, an anticipated hypomagnesemia prevalence of

35%, taken as an intermediate value from published hospital-based series reporting 19.1% to 44% [6,7], and an absolute precision of 6%. This yielded a minimum of 243 participants, and 250 patients were enrolled.

Data collection and measurements:

Each participant was assessed using a pre-designed, pre-tested structured proforma recording duration of diabetes, current antidiabetic treatment, comorbidities and associated risk factors, followed by a complete general and systemic examination. Height and weight were measured in light clothing without footwear, and body mass index (BMI) was categorised using the World Health Organization Asian criteria [18] as underweight ($<18.5 \text{ kg/m}^2$), normal ($18.5\text{--}22.9 \text{ kg/m}^2$), overweight ($23.0\text{--}24.9 \text{ kg/m}^2$) or obese ($\geq 25.0 \text{ kg/m}^2$).

Venous blood samples were drawn under aseptic precautions after an overnight fast of at least eight hours. Serum magnesium was estimated by the xylidyl blue colorimetric end-point method, and fasting blood sugar (FBS) and postprandial blood sugar (PPBS) by the glucose oxidase–peroxidase (GOD-POD) method, on a fully automated clinical chemistry analyser in the institutional central laboratory, calibrated daily and run with two-level internal quality control. HbA1c was estimated by cation-exchange high-performance liquid chromatography (HPLC) using an NGSP-certified, DCCT-aligned method. Serum creatinine (modified Jaffe kinetic method) and blood urea were measured on the same analyser, and urinary albumin-to-creatinine ratio was determined on a spot morning sample by immunoturbidimetry. All assays were performed in the same laboratory on the same instruments throughout the study period to minimise inter-assay variability. Hypomagnesemia was defined a priori as serum magnesium below 1.6 mg/dL , the threshold used in comparable published series [3,5], and glycemic control was classified as adequate at HbA1c below 7% and inadequate at 7% or above [17].

Diabetic retinopathy was diagnosed by dilated fundoscopic examination performed by an ophthalmologist and recorded as absent, non-proliferative or proliferative. Diabetic nephropathy was defined as a spot urinary albumin-to-creatinine ratio of 300 mg/g or above, interpreted together with serum creatinine and blood urea. Estimated glomerular filtration rate (eGFR; CKD-EPI 2021 creatinine equation) was calculated from serum creatinine solely to apply the exclusion criterion for stage 4–5 chronic kidney disease (eGFR $<30 \text{ mL/min/1.73 m}^2$). eGFR-based KDIGO categorisation was not used as an outcome because the protocol, fixed before data collection, defined nephropathy a priori by albuminuria alone and the analysis plan was not amended post hoc; the consequences of this choice are considered in the limitations. Diabetic neuropathy was diagnosed clinically from history

together with vibration perception (128-Hz tuning fork), protective sensation (10-g Semmes-Weinstein monofilament), pain and temperature sensation and ankle reflexes. Formal grading instruments (ETDRS retinal photography and validated composite neuropathy scores) were not used because fundus photography and nerve conduction facilities were not available for routine research use at this centre; bedside ascertainment by a uniform protocol was therefore adopted. Macrovascular complications were ascertained from clinical history, examination and relevant investigations: coronary artery disease from documented ischaemic heart disease, stroke from a documented cerebrovascular event, and peripheral arterial disease from claudication with absent or diminished peripheral pulses and supporting vascular assessment. All assessments were carried out by the investigating team using a single uniform protocol throughout the study period. Clinical assessment of complications, including fundoscopy, was completed at enrolment, before the laboratory report was available to the assessing investigators, who were therefore unaware of the patient's serum magnesium result at the time of assessment.

Statistical analysis:

Data were entered in Microsoft Excel and analysed using SPSS. Continuous variables were summarised as mean $\pm$ standard deviation and categorical variables as n (%). Associations between magnesium status and categorical variables were tested by the Pearson chi-square test, and means were compared by the independent-samples t -test, with the test statistic and degrees of freedom reported. Correlation between serum magnesium and glycemic parameters was assessed by the Pearson coefficient, with 95% confidence intervals from the Fisher z transformation. The association between hypomagnesemia and each complication was expressed as an odds ratio with 95% confidence interval, taking normal magnesium as reference, and multivariable binary logistic regression was fitted separately for retinopathy, nephropathy and neuropathy with age, duration of diabetes, sex and BMI as covariates. Subgroup analyses by duration of diabetes and age band, and a sensitivity analysis excluding underweight patients, were performed. All tests were two-tailed and $p < 0.05$ was considered significant.

RESULTS:

Of the 250 patients studied (Table 1), the mean age was 57.3 ± 12.6 years, 136 (54.4%) were men and 125 (50.0%) had diabetes of ten years' duration or longer. Glycemic control was poor, with a mean HbA1c of $9.10 \pm 1.52\%$ and only 4 patients (1.6%) below 7%. Mean serum magnesium was $1.72 \pm 0.20 \text{ mg/dL}$, and hypomagnesemia was present in 61 patients (24.4%).

Tables & Graphs: All six tables are placed within the Results section immediately after the text that first cites them.

Table 1: Demographic, anthropometric and biochemical profile of the study participants (n = 250)

Variable	Category	n (%)
Age group (years)	30–39	23 (9.2)
	40–49	42 (16.8)
	50–59	69 (27.6)
	60–69	71 (28.4)
	≥70	45 (18.0)
Sex	Male	136 (54.4)
	Female	114 (45.6)
Duration of diabetes (years)	<5	21 (8.4)
	5–9	104 (41.6)
	10–14	81 (32.4)
	15–19	43 (17.2)
	≥20	1 (0.4)
BMI category (Asian criteria)	Underweight (<18.5 kg/m ²)	13 (5.2)
	Normal (18.5–22.9 kg/m ²)	50 (20.0)
	Overweight (23.0–24.9 kg/m ²)	48 (19.2)
	Obese (≥25.0 kg/m ²)	139 (55.6)
Glycemic control	HbA1c <7%	4 (1.6)
	HbA1c ≥7%	246 (98.4)
Serum magnesium status	Hypomagnesemia (<1.6 mg/dL)	61 (24.4)
	Normal (≥1.6 mg/dL)	189 (75.6)

*BMI, body mass index; HbA1c, glycated haemoglobin.

Hypomagnesemia became steadily more frequent with advancing age, from 4 of 23 patients (17.4%) aged 30–39 years to 27 of 45 (60.0%) aged 70 years or above, and with longer disease duration, 50 of the 61 hypomagnesemic patients (82.0%) having had diabetes for ten years or more (Table 2). It was commonest among underweight patients (9 of 13, 69.2%) and declined across the normal (36.0%), overweight (18.8%) and obese (18.0%) strata. Sex was not associated with magnesium status.

Table 2: Association of participant characteristics with serum magnesium status (n = 250)

Variable	Hypomagnesemia (n=61) n (%)	Normal magnesium (n=189) n (%)	χ^2	df	p
Age group (years)					
30–39	4 (6.6)	19 (10.1)	47.94	4	<0.001
40–49	1 (1.6)	41 (21.7)			
50–59	9 (14.8)	60 (31.7)			
60–69	20 (32.8)	51 (27.0)			
≥70	27 (44.3)	18 (9.5)			
Sex					
Male	39 (63.9)	97 (51.3)	2.47	1	0.11
Female	22 (36.1)	92 (48.7)			
Duration of diabetes (years)					
<5	1 (1.6)	20 (10.6)	40.01	4	<0.001
5–9	10 (16.4)	94 (49.7)			
10–14	27 (44.3)	54 (28.6)			
15–19	22 (36.1)	21 (11.1)			
≥20	1 (1.6)	0 (0.0)			
BMI category					
Underweight	9 (14.8)	4 (2.1)	21.74	3	<0.001
Normal	18 (29.5)	32 (16.9)			
Overweight	9 (14.8)	39 (20.6)			
Obese	25 (41.0)	114 (60.3)			

*Pearson chi-square test. Percentages are column percentages within each magnesium group. BMI categories follow Asian criteria.

Serum magnesium correlated inversely with all three glycemic parameters, most strongly with HbA1c (Table 3). A categorical comparison of glycemic control between the magnesium groups was uninformative, since only 4 of 250 patients achieved an HbA1c below 7%.

Table 3: Correlation of serum magnesium with glycemic parameters (n = 250)

Glycemic parameter	Pearson r	95% CI	p
HbA1c (%)	−0.378	−0.480 to −0.267	0.001
Fasting blood sugar (mg/dL)	−0.216	−0.331 to −0.094	0.001
Postprandial blood sugar (mg/dL)	−0.311	−0.419 to −0.195	0.001

*Pearson correlation coefficient; 95% confidence intervals computed by the Fisher z transformation.

All three microvascular complications were significantly commoner in hypomagnesemic patients, with crude odds ratios of 2.71 for retinopathy, 2.57 for nephropathy and 2.19 for neuropathy (Table 4).

Table 4: Association of serum magnesium status with microvascular complications (n = 250)

Complication	Hypomagnesemia (n=61) n (%)	Normal magnesium (n=189) n (%)	Crude OR (95% CI)	χ^2 (df=1)	p
Retinopathy	34 (55.7)	60 (31.7)	2.71 (1.50–4.89)	11.31	0.001
Nephropathy	35 (57.4)	65 (34.4)	2.57 (1.42–4.63)	10.15	0.001
Neuropathy	37 (60.7)	78 (41.3)	2.19 (1.22–3.96)	6.98	0.008

*Pearson chi-square test. Percentages in the first two data columns are within magnesium group; OR, odds ratio, with normal serum magnesium as the reference category.

With serum magnesium as a continuous variable, mean magnesium was significantly lower in patients with retinopathy, nephropathy and neuropathy than in those without (Table 5). Mean HbA1c, by contrast, did not differ between patients with and without any complication ($p = 0.318$, 0.199 and 0.122 respectively), reflecting the narrow spread of HbA1c in this uniformly poorly controlled cohort.

Macrovascular complications were uncommon: coronary artery disease in 6 patients (2.4%), stroke in 5 (2.0%) and peripheral arterial disease in 3 (1.2%). Mean serum magnesium was numerically lower in each affected group but no difference approached significance (Table 5), and the corresponding odds ratios were too imprecise to be informative (1.57, 95% CI 0.28–8.78; 2.10, 95% CI 0.34–12.88; and 6.37, 95% CI 0.57–71.54).

Table 5: Mean serum magnesium by presence or absence of microvascular and macrovascular complications (n = 250)

Complication	Present n (%)	Serum Mg, present (mean±SD)	Absent n (%)	Serum Mg, absent (mean±SD)	t (df=248)	p	Mean difference (95% CI)
Retinopathy	94 (37.6)	1.647±0.216	156 (62.4)	1.756±0.181	−4.28	<0.001	−0.110 (−0.160 to −0.059)
Nephropathy	100 (40.0)	1.647±0.211	150 (60.0)	1.761±0.182	−4.55	<0.001	−0.114 (−0.163 to −0.064)
Neuropathy	115 (46.0)	1.674±0.202	135 (54.0)	1.750±0.195	−3.04	0.003	−0.076 (−0.126 to −0.027)
Coronary artery disease	6 (2.4)	1.683±0.194	244 (97.6)	1.716±0.202	−0.40	0.693	−0.033 (−0.197 to 0.132)
Stroke	5 (2.0)	1.700±0.308	245 (98.0)	1.716±0.200	−0.18	0.861	−0.016 (−0.195 to 0.164)
Peripheral arterial disease	3 (1.2)	1.633±0.231	247 (98.8)	1.716±0.202	−0.71	0.481	−0.083 (−0.314 to 0.148)

*Independent-samples t-test. Mg, magnesium; SD, standard deviation; CI, confidence interval. The mean difference is that in serum magnesium between patients with and without the complication. Percentages are of the whole cohort.

On multivariable logistic regression (Table 6), the association between hypomagnesemia and nephropathy persisted, whereas those with retinopathy and neuropathy were attenuated and no longer significant; age was independently associated with neuropathy and BMI inversely with retinopathy. On subgroup analysis the crude associations were confined to patients with diabetes of ten years' duration or longer (retinopathy odds ratio 3.00, 95% CI 1.43–6.30; nephropathy 5.34, 95% CI 2.45–11.61) and were absent in those of shorter duration. The association between BMI and magnesium status persisted after exclusion of the 13 underweight patients ($\chi^2 = 7.33$, $df = 2$, $p = 0.026$).

Table 6: Adjusted association between hypomagnesemia and microvascular complications (n = 250); unadjusted odds ratios are given in Table 4

Complication	Adjusted OR (95% CI)*	p	Other independent predictors
Retinopathy	1.61 (0.80–3.25)	0.185	BMI 0.85 per kg/m ² (0.77–0.95), p = 0.003
Nephropathy	1.99 (1.01–3.94)	0.048	None
Neuropathy	1.34 (0.67–2.67)	0.410	Age 1.06 per year (1.01–1.11), p = 0.013

*Binary logistic regression; reference category, serum magnesium ≥ 1.6 mg/dL. Adjusted for age (years), duration of diabetes (years), sex and body mass index (kg/m²). CI, confidence interval.

DISCUSSION:

In this cross-sectional study of 250 patients with type 2 diabetes mellitus from the Bundelkhand region, hypomagnesemia was present in 24.4%, rose steeply with age and disease duration, correlated inversely with every glycemic index measured, and was associated with all three microvascular complications before adjustment, but only with nephropathy after adjustment.

The prevalence of 24.4% lies within the mid-range of reported values. A meta-analysis confirmed that hypomagnesemia is a common accompaniment of T2DM worldwide while emphasising substantial heterogeneity between reports [4]. A North Indian outpatient study using a 1.7 mg/dL threshold found 44% [7], a Palestinian primary care study using our 1.6 mg/dL cut-off found 11% [5], a South Indian tertiary series reported 19.1% [6], a Pakistani hospital-based study found figures close to ours [19] and a western Indian series reported 37.7% [20]. This variation reflects the diagnostic threshold, the clinical setting and the renal and medication profile of the population sampled; our figure represents the burden in patients already in tertiary care, advanced kidney disease having been excluded.

The strong associations with age and disease duration are biologically expected. Ageing is accompanied by reduced dietary magnesium intake, diminished intestinal absorption and impaired tubular reabsorption, and older patients are more likely to receive proton pump inhibitors and diuretics [3], while sustained hyperglycaemia and glycosuria drive continuous urinary magnesium wasting that progressively depletes body stores. Sex was not associated with magnesium status, consistent with most previous reports; the Palestinian cohort, which identified female sex as an independent predictor, is the main exception [5].

The relationship with BMI deserves careful interpretation. Hypomagnesemia was commonest in underweight patients and fell progressively across the normal, overweight and

obese strata, pointing towards inadequate dietary magnesium intake in undernourished patients in a region where nutritional deficiency and diabetes frequently coexist. Studies in predominantly overweight or obese populations have generally not found BMI to be an independent predictor of hypomagnesemia [5], and the small number of underweight patients means this finding should be regarded as hypothesis-generating, although the gradient persisted when the underweight stratum was excluded.

The inverse correlation with glycemic parameters, strongest for HbA1c ($r = -0.378$), accords closely with the literature. Low magnesium has been proposed as an indicator of poor glycemic control in patients who have already developed complications [8], and a prospective cohort of 4,348 participants showed that HbA1c partially mediates the association between serum magnesium and diabetic complications [16]. Mechanistically, magnesium deficiency impairs insulin-receptor autophosphorylation and tyrosine kinase activity, while the resulting hyperglycaemia promotes further renal magnesium loss [3]. Whether this cycle can be interrupted pharmacologically is uncertain: meta-analyses of supplementation trials estimate HbA1c reductions of about 0.7% [9,10], but a randomised crossover trial in insulin-treated patients found no improvement in insulin sensitivity [11]. Our cross-sectional data cannot arbitrate between these positions.

Retinopathy showed the strongest crude association (odds ratio 2.71). A meta-analysis of 17 studies reported a more than fourfold increase in the odds of retinopathy with hypomagnesemia, with lower magnesium in proliferative than non-proliferative disease [12], a retrospective analysis of 2,222 patients found reduced magnesium in those with retinopathy [13], and a study restricted to patients with normal renal parameters, which removes renal wasting as a confounder, found the same relationship [14]. An Indian series reported non-proliferative retinopathy in 59.6% of hypomagnesemic against 23.3% of normomagnesemic patients [20]. Nephropathy showed the largest effect and was

the only association to persist after adjustment. Hypomagnesemia has been associated with albuminuria in a retrospective study of 1,178 patients [15] and with incident chronic kidney disease in the prospective cohort described above [16]. Causality here is bidirectional: reduced tubular reabsorption in diabetic kidney disease lowers serum magnesium, while magnesium deficiency may itself accelerate glomerular injury; exclusion of stage 4–5 disease reduces but does not eliminate the first mechanism. Neuropathy was the commonest complication (46.0%) and was more frequent in hypomagnesemia, as in a Pakistani inpatient series [19], although its continuous-variable association was the weakest of the three, which may reflect the clinical rather than electrophysiological basis of diagnosis.

The adjusted analysis is the most important qualification to these findings. Age and duration of diabetes are the dominant determinants both of hypomagnesemia and of retinopathy and neuropathy in this cohort, so a large part of the crude association is shared variance; the subgroup analysis makes the same point, the crude associations being confined to disease of ten years' duration or longer. That nephropathy alone survives adjustment is consistent with the close mechanistic link between renal tubular handling and magnesium balance. The prudent interpretation is that low serum magnesium marks a population with long-standing, poorly controlled disease and a high microvascular burden, and is an independent correlate only of nephropathy.

It is notable that mean HbA1c did not differ between patients with and without complications, whereas mean serum magnesium did. In a cohort in which 98.4% had an HbA1c of 7% or above, HbA1c has little residual capacity to discriminate, and such uniformly poor control is common in resource-limited settings [21]. Serum magnesium retained discriminatory value in exactly the setting in which an additional inexpensive marker would be most useful.

Macrovascular associations were in the expected direction but none attained significance. With only 6, 5 and 3 events, the study had essentially no power to detect associations of plausible magnitude, and even the largest prospective cohort found no association with myocardial infarction or stroke after adjustment [16]. These findings should be read as uninformative rather than as evidence of no association.

CONCLUSION:

Hypomagnesemia was common among patients with type 2 diabetes mellitus in this region and was associated with poorer glycemic control and with a higher unadjusted burden of diabetic retinopathy, nephropathy and neuropathy. After adjustment for age, duration of diabetes, sex and body mass index, an independent association persisted only with

nephropathy, and associations with macrovascular complications were not significant.

DECLARATIONS:

Ethical Approval: The study protocol was approved by the Institutional Ethical Committee of M.L.B. Medical College, Jhansi (approval number 916/IEC/PG-23-13/2026/SC-1, dated 08 May 2025), and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from every participant before enrolment in a language the participant understood. No patient identifiers were recorded in the analysis dataset.

Informed Consent: Informed consent was obtained from all participants prior to their inclusion in the study. Participants were informed that their data would be kept confidential and used solely for research purposes.

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Conflict of Interest: The authors declare that they have no conflicts of interest related to this study.

Data availability statement: The dataset presented in this study is available on request from the corresponding author.

AI Use Disclosure: No generative AI tools were used in the writing or data analysis. AI tools were limited to language assistance. The authors remain fully responsible for the content.

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