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Research Article

Glycaemic Variability and Urinary Albumin Excretion in Type 2 Diabetes Mellitus: A Retrospective Observational Study

Dr. Manikandan[1*], Dr. Parthiban[2], Dr.Anantharaj[3]
[1*]Assistant Professor, Department of General Medicine Kallakurichi Government Medical College Hospital, Government Medical College & Hospital, Kallakurichi, Tamil Nadu, India.
[2,3]Senior Resident, Department of General Medicine Kallakurichi Government Medical College Hospital, Government Medical College & Hospital, Kallakurichi, Tamil Nadu, India.
Corresponding Author: Dr. Manikandan
Assistant Professor, Department of General Medicine Kallakurichi Government Medical College Hospital, Government Medical College & Hospital, Kallakurichi, Tamil Nadu, India.
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ABSTRACT
Background: Albuminuria is an early and clinically meaningful expression of diabetic kidney involvement. Mean HbA1c reflects average glycaemic exposure, but it does not fully capture long-term oscillation in glycaemic control. Whether visit-to-visit HbA1c variability parallels urinary albumin excretion in routine general-medicine practice remains a practical question.
Objective: To evaluate the association between retrospective HbA1c variability and urinary albumin excretion rate (UAER) among patients with type 2 diabetes mellitus.
Materials and Methods: This retrospective observational study included 100 patients with type 2 diabetes mellitus evaluated in the Department of General Medicine between June 2025 and January 2026. Three consecutive annual HbA1c values were used to calculate mean HbA1c, standard deviation, and coefficient of variation (CV). UAER was classified as normoalbuminuria (<30 mg/24 h), microalbuminuria (30-300 mg/24 h), and macroalbuminuria (>300 mg/24 h). Group comparisons were performed using analysis of variance and chi-square tests. Spearman correlation assessed the relation of UAER with glycaemic, renal, and blood-pressure variables.
Results: The cohort had a mean age of 59.52 ± 8.06 years, with equal sex distribution. Normoalbuminuria, microalbuminuria, and macroalbuminuria were observed in 29%, 39%, and 32% of patients, respectively. Mean HbA1c increased from 7.24% in normoalbuminuria to 10.32% in macroalbuminuria ($p<0.001$). HbA1c CV also rose across the same groups (2.41% to 4.31%, $p<0.001$). Across HbA1c CV tertiles, mean UAER increased from 56.67 ± 41.79 to 559.41 ± 270.71 mg/24 h, while mean eGFR declined from 79.94 ± 15.52 to 43.50 ± 13.55 mL/min/1.73 m^2 (both $p<0.001$). Macroalbuminuria was concentrated in the high HbA1c CV tertile (82.4%).
Conclusion: Higher mean HbA1c and greater visit-to-visit HbA1c variability were strongly associated with higher UAER and lower eGFR in type 2 diabetes mellitus. Glycaemic consistency, not only point-in-time HbA1c control, may deserve greater emphasis during diabetic kidney disease risk assessment.

Keywords: Type 2 Diabetes Mellitus, Urinary Albumin Excretion Rate, Albuminuria, Hba1c Variability, Diabetic Kidney Disease, Egfr.

INTRODUCTION

Type 2 diabetes mellitus has become one of the most pressing non-communicable disease burdens in India, not only because of the number of affected adults but because renal and cardiovascular complications often appear during the most productive years of life. The ICMR-INDIAB national cross-sectional study estimated a substantially higher burden of metabolic non-communicable disease than earlier anticipated, with diabetes and prediabetes now forming a large reservoir for future microvascular disease [1]. In a busy Indian general-medicine outpatient clinic, this burden is seen in a familiar way: patients arrive with several years of diabetes, variable adherence, changing prescriptions, intermittent self-monitoring, and only occasional laboratory continuity. The renal signal is frequently the first measurable warning.

Albuminuria remains central to diabetic kidney disease assessment. KDIGO classifies chronic kidney disease using cause, glomerular filtration rate category, and albuminuria category, recognizing albuminuria as a marker of kidney damage and risk stratification even when eGFR is still relatively preserved [2]. The American Diabetes Association similarly places

urine albumin measurement and eGFR estimation at the centre of chronic kidney disease screening and monitoring among people with diabetes [3]. In clinical terms, a patient moving from normoalbuminuria to microalbuminuria is not merely crossing a laboratory threshold. The shift often changes counselling, blood-pressure targets, renin-angiotensin system blockade decisions, frequency of monitoring, and referral thresholds.

Guideline-driven diabetes care has traditionally focused on lowering mean glycaemic exposure. KDIGO guidance for diabetes management in chronic kidney disease emphasizes comprehensive care that includes glycaemic control, blood-pressure optimization, renin-angiotensin system inhibition where appropriate, and newer kidney-protective therapies [4]. The legacy of the UKPDS is still important here: increasing HbA1c exposure was strongly associated with microvascular and macrovascular complications in type 2 diabetes [5]. The ADVANCE trial later showed that intensive glucose control reduced new or worsening nephropathy, although the clinical challenge is to achieve benefit without creating avoidable hypoglycaemia or therapeutic fatigue [6].

Mean HbA1c, however, has one obvious limitation. It averages out fluctuation. Two patients may share the same annual mean HbA1c while having very different trajectories, one relatively stable and the other cycling through repeated periods of poor and improved control. The ADA glycaemic standards continue to treat HbA1c as a major monitoring tool, but clinical interpretation requires attention to treatment burden, hypoglycaemia risk, comorbidities, and the broader pattern of glucose exposure [7]. For diabetic kidney disease, this broader pattern is particularly relevant because endothelial injury, oxidative stress, glomerular hyperfiltration, and inflammatory signalling may be influenced by instability in glycaemic exposure, not only by the average value.

Longitudinal studies have therefore examined visit-to-visit HbA1c variability as an additional risk signal. Hsu et al. reported that HbA1c variability was associated with microalbuminuria development in type 2 diabetes over seven years [8]. Dorajoo et al. similarly observed that HbA1c coefficient of variation predicted new-onset albuminuria within three years [9]. Other work has linked intrapersonal HbA1c variation with progression of nephropathy, suggesting that long-term instability in glycaemic control may carry renal implications beyond the mean HbA1c itself [10]. The Indian literature also supports the clinical relevance of albuminuria screening in type 2 diabetes. Varghese et al. documented microalbuminuria among South Indian patients with type 2 diabetes attending a diabetes centre [11]. The Chennai Urban Rural Epidemiology Study later showed that microalbuminuria and overt nephropathy were not rare in urban Asian Indians and were related to diabetes duration, HbA1c, and blood pressure [12]. These findings matter because Indian patients often combine several risk amplifiers: long diabetes duration before specialist evaluation, high carbohydrate dietary patterns, variable affordability of drugs, fragmented follow-up, and late presentation to tertiary care. The present study was undertaken to evaluate whether retrospective HbA1c variability is associated with urinary albumin excretion among patients with type 2 diabetes mellitus in a general-medicine setting.

MATERIALS AND METHODS

Study Design and Setting

This was a retrospective observational analytical study conducted in the Department of General Medicine. The study period extended from June 2025 to January 2026.

Study Population

The study included 100 patients with type 2 diabetes mellitus for whom three consecutive annual HbA1c values, urinary albumin excretion rate, eGFR, blood-pressure measurements, treatment details, and smoking status were available for analysis.

Inclusion Criteria

Patients with type 2 diabetes mellitus were included when complete glycaemic history using three annual HbA1c values and renal assessment using UAER and eGFR were available during the study period.

Exclusion Criteria

Records lacking the minimum variables required for albuminuria classification or glycaemic variability calculation were not used for analysis.

Study Variables

The baseline variables were age, sex, duration of diabetes, body mass index, systolic and

diastolic blood pressure, eGFR, antihyperglycaemic treatment, ACE inhibitor or ARB use, and smoking status. Glycaemic exposure was represented by three annual HbA1c readings, mean HbA1c, HbA1c standard deviation, and HbA1c coefficient of variation. HbA1c CV was calculated as HbA1c SD divided by mean HbA1c, multiplied by 100.

Outcome Definition

The primary renal outcome was urinary albumin excretion rate measured as mg/24 h. Albuminuria was classified as normoalbuminuria (<30 mg/24 h), microalbuminuria (30-300 mg/24 h), and macroalbuminuria (>300 mg/24 h).

Statistical Analysis

Continuous variables were summarized as mean ± standard deviation and, where clinically useful, median with interquartile range. Categorical variables were summarized as frequency and percentage. Differences across albuminuria categories were assessed using one-way analysis of variance for continuous variables and chi-square test for categorical variables. HbA1c coefficient of variation was additionally divided into tertiles to examine whether increasing glycaemic variability was accompanied by a graded renal profile. Because several clinical variables were highly ordered across albuminuria categories, near-perfect rank correlations were not retained as primary inferential results. A p-value <0.05 was considered statistically significant.

Ethical Considerations

Institutional ethics approval details, including approval number and date, should be inserted before journal submission. Patient identifiers were handled as study codes during analysis.

RESULTS

A total of 100 patients with type 2 diabetes mellitus were included. The mean age was 59.52 ± 8.06 years, and the cohort had equal representation of men and women. The mean duration of diabetes was 14.77 ± 6.76 years. Baseline clinical and laboratory characteristics are summarized in Table 1.

Table 1. Baseline Clinical Profile of the Study Population (N=100)

Variable	Value
Age, years	59.52 ± 8.06
Male sex	50 (50.0)
Female sex	50 (50.0)
Duration of diabetes, years	14.77 ± 6.76
BMI, kg/m²	29.46 ± 2.50
Mean HbA1c, %	8.80 ± 1.31
HbA1c SD	0.28 ± 0.13
HbA1c CV, %	3.12 ± 1.12
UAER, mg/24 h, median (IQR)	95.00 (25.75-415.00)
eGFR, mL/min/1.73 m²	66.16 ± 23.77
SBP, mmHg	144.42 ± 14.16
DBP, mmHg	83.11 ± 6.43
ACEi/ARB use	72 (72.0)
Never smokers	50 (50.0)
Former smokers	27 (27.0)
Current smokers	23 (23.0)

Values are presented as mean ± SD, n (%), or median (IQR), as appropriate.

Albuminuria was distributed as normoalbuminuria in 29 patients, microalbuminuria in 39 patients, and macroalbuminuria in 32 patients. Figure 1 presents the albuminuria category distribution.

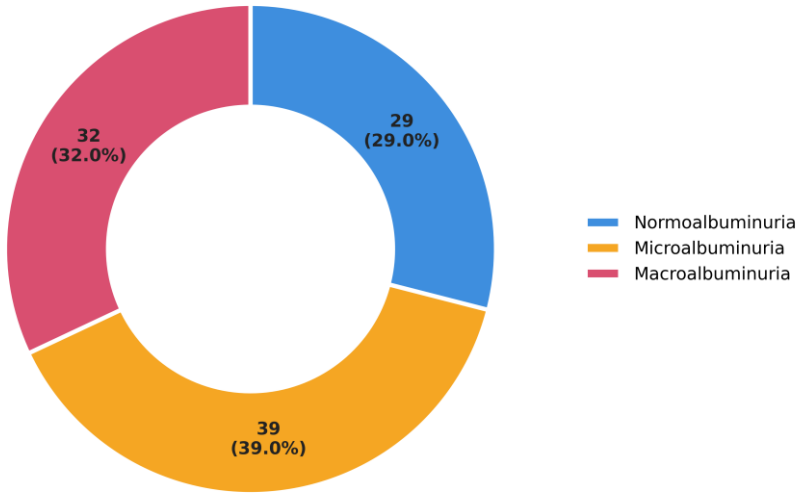

Figure 1. Distribution of Albuminuria Categories among Patients with Type 2 Diabetes Mellitus

Donut chart shows patient counts and percentages for normoalbuminuria, microalbuminuria, and macroalbuminuria.

Continuous variables showed a graded pattern across albuminuria categories. Patients with macroalbuminuria were older, had longer diabetes duration, higher mean HbA1c, greater HbA1c variability, higher blood pressure, and lower eGFR. These differences were statistically significant across all continuous variables shown in Table 2.

Table 2. Comparison of Continuous Clinical and Biochemical Variables by Albuminuria Category

Variable	Normoalbuminuria (N=29)	Microalbuminuria (N=39)	Macroalbuminuria (N=32)	P-Value
Age, years	50.17 ± 3.11	59.13 ± 4.22	68.47 ± 3.50	<0.001
Duration of diabetes, years	6.97 ± 1.92	14.21 ± 3.35	22.53 ± 3.08	<0.001
BMI, kg/m²	26.65 ± 1.58	30.59 ± 1.92	30.62 ± 1.68	<0.001
Mean HbA1c, %	7.24 ± 0.34	8.72 ± 0.60	10.32 ± 0.55	<0.001
HbA1c SD	0.17 ± 0.07	0.23 ± 0.08	0.44 ± 0.04	<0.001
HbA1c CV, %	2.41 ± 0.97	2.66 ± 0.81	4.31 ± 0.37	<0.001
UAER, mg/24 h	17.97 ± 6.27	117.28 ± 75.21	641.25 ± 225.10	<0.001
eGFR, mL/min/1.73 m²	95.76 ± 7.20	66.38 ± 10.57	39.06 ± 6.55	<0.001
SBP, mmHg	126.76 ± 4.46	144.54 ± 5.15	160.28 ± 6.16	<0.001
DBP, mmHg	75.38 ± 2.11	82.74 ± 2.19	90.56 ± 2.93	<0.001

Values are mean ± SD. p-values calculated using one-way analysis of variance.

Mean HbA1c and HbA1c CV increased stepwise across albuminuria strata. The gradient was clinically clear: mean HbA1c rose from 7.24% in normoalbuminuria to 10.32% in macroalbuminuria, while HbA1c CV rose from 2.41% to 4.31%. This pattern is illustrated in Figure 2.

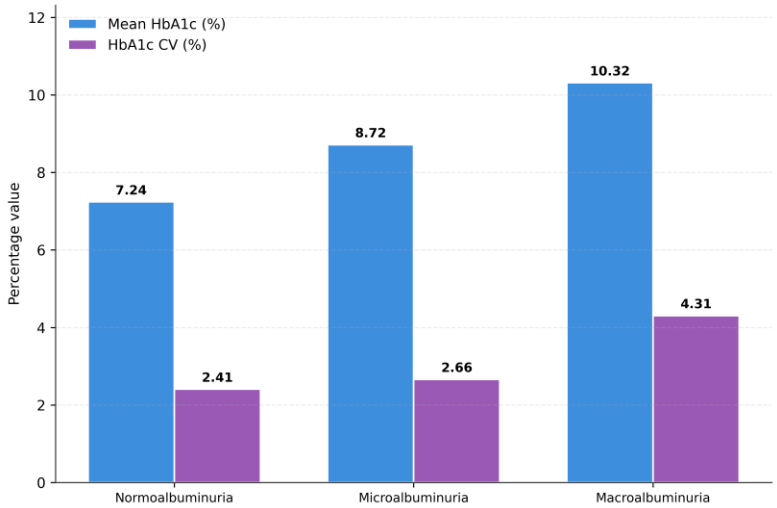

Figure 2. Mean Hba1c and Hba1c Coefficient of Variation across Albuminuria Categories

Grouped bars show mean percentage values; bold labels above bars indicate point estimates.

Renal function declined across albuminuria categories. Mean eGFR was 95.76 ± 7.20 mL/min/1.73 m² in normoalbuminuria, 66.38 ± 10.57 mL/min/1.73 m² in microalbuminuria, and 39.06 ± 6.55 mL/min/1.73 m² in macroalbuminuria (p<0.001). Figure 3 displays the eGFR gradient.

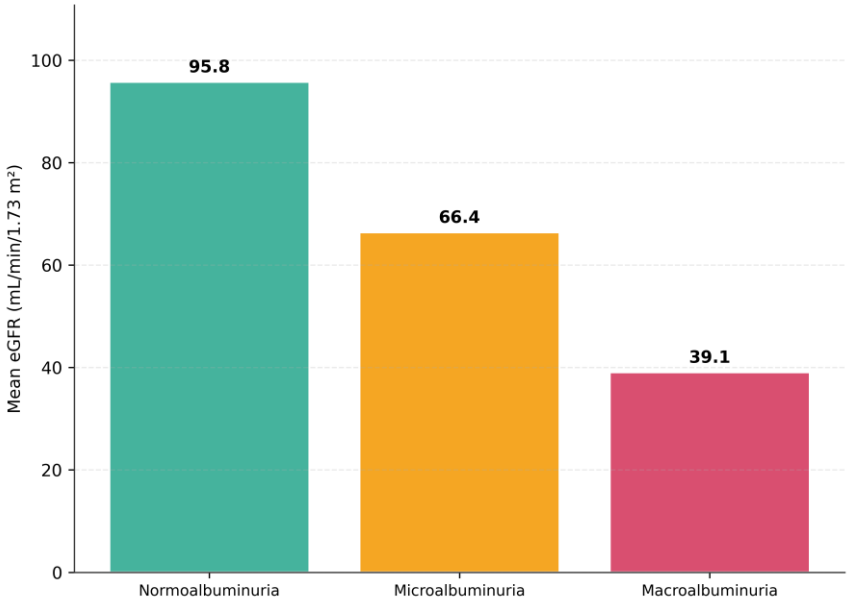

Figure 3. Mean eGFR across Albuminuria Categories

Bar chart shows mean eGFR; bold labels above bars indicate mean values in mL/min/1.73 m².

Categorical variables are shown in Table 3. Sex distribution did not differ significantly across albuminuria categories (p=0.229). Treatment pattern, ACE inhibitor or ARB use, smoking status, and eGFR band differed significantly across albuminuria groups (all p<0.001). The strong association with treatment category should be interpreted as a marker of disease severity and clinical escalation, not as evidence that insulin caused albuminuria.

Table 3. Categorical Variables across Albuminuria Categories

Variable	Category	Normoalbuminuria (N=29)	Microalbuminuria (N=39)	Macroalbuminuria (N=32)	P-Value
Sex	Male	13 (44.8)	17 (43.6)	20 (62.5)	0.229

	Female	16 (55.2)	22 (56.4)	12 (37.5)	
Treatment	Metformin only	21 (72.4)	1 (2.6)	0 (0.0)	<0.001
	OAD only	8 (27.6)	26 (66.7)	0 (0.0)	
	Insulin+OAD	0 (0.0)	9 (23.1)	11 (34.4)	
	Insulin	0 (0.0)	3 (7.7)	21 (65.6)	
ACEi/ARB use	No	27 (93.1)	1 (2.6)	0 (0.0)	<0.001
	Yes	2 (6.9)	38 (97.4)	32 (100.0)	
Smoking	Never	28 (96.6)	13 (33.3)	9 (28.1)	<0.001
	Former	1 (3.4)	17 (43.6)	9 (28.1)	
	Current	0 (0.0)	9 (23.1)	14 (43.8)	
eGFR band	≥90	23 (79.3)	0 (0.0)	0 (0.0)	<0.001
	60-89	6 (20.7)	28 (71.8)	0 (0.0)	
	30-59	0 (0.0)	11 (28.2)	30 (93.8)	
	<30	0 (0.0)	0 (0.0)	2 (6.2)	

Values are n (%) within each albuminuria category. p-values calculated using chi-square test.

When HbA1c CV was grouped into tertiles, macroalbuminuria concentrated in the highest variability tertile. In the high CV tertile, 28 of 34 patients had macroalbuminuria, whereas macroalbuminuria was absent in the low tertile. Figure 4 shows this shift across variability strata.

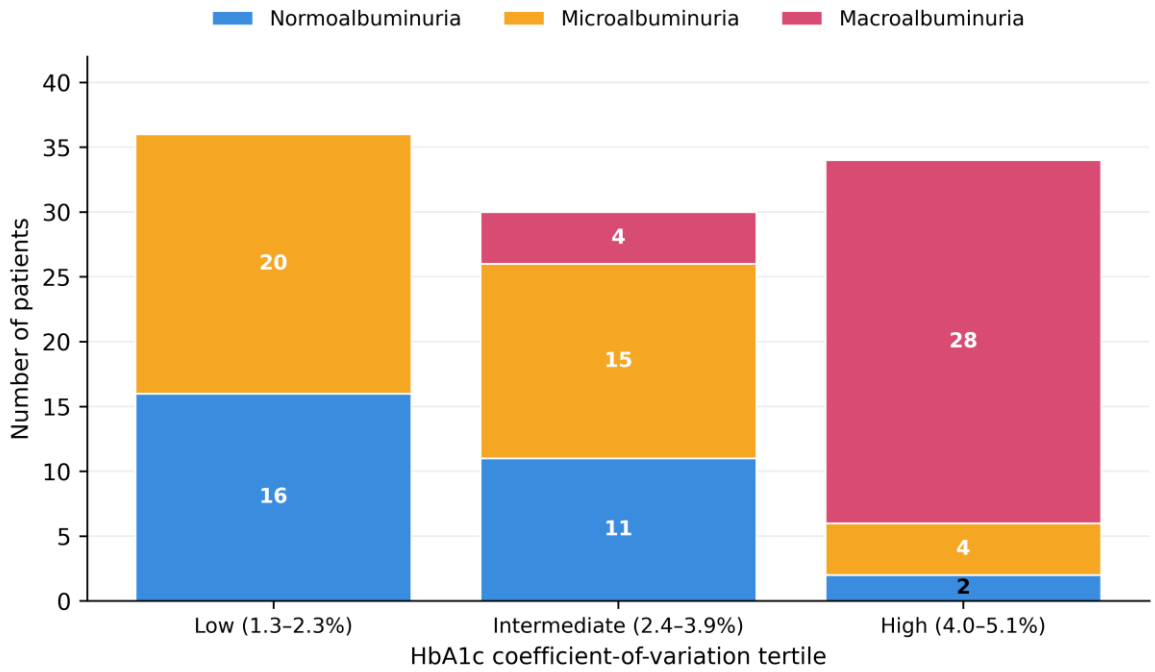

Figure 4. Albuminuria Categories across Hba1c Coefficient-of-Variation Tertiles

Stacked bars show patient counts in each albuminuria category; segment labels indicate counts.

To examine glycaemic variability more directly, patients were grouped into HbA1c CV tertiles. Mean UAER increased progressively from the low to high tertile, whereas mean eGFR declined. Macroalbuminuria was absent in the low CV tertile, present in 13.3% of the intermediate tertile, and concentrated in 82.4% of the high tertile (Table 4).

Table 4. Renal Profile across Hba1c Coefficient-of-Variation Tertiles

Variable	Low CV Tertile (N=36)	Intermediate CV Tertile (N=30)	High CV Tertile (N=34)	P-Value
UAER, mg/24 h	56.67 ± 41.79	151.83 ± 232.25	559.41 ± 270.71	<0.001
eGFR, mL/min/1.73 m²	79.94 ± 15.52	75.30 ± 22.47	43.50 ± 13.55	<0.001
Mean HbA1c, %	8.00 ± 0.80	8.33 ± 1.18	10.06 ± 0.85	<0.001
Systolic BP, mmHg	136.58 ± 9.53	138.83 ± 12.79	157.65 ± 9.25	<0.001
Diabetes duration, years	10.97 ± 3.83	12.07 ± 6.37	21.18 ± 4.62	<0.001
Normoalbuminuria	16 (44.4)	11 (36.7)	2 (5.9)	<0.001
Microalbuminuria	20 (55.6)	15 (50.0)	4 (11.8)	
Macroalbuminuria	0 (0.0)	4 (13.3)	28 (82.4)	

Values are mean ± SD or n (%). Tertile ranges: low 1.3–2.3%, intermediate 2.4–3.9%, and high 4.0–5.1%. P-values calculated using one-way analysis of variance for continuous variables and chi-square test for albuminuria category distribution.

DISCUSSION

This study found a clear graded relationship between worsening albuminuria and markers of long-term glycaemic exposure. The pattern was not subtle. Patients with macroalbuminuria had longer diabetes duration, higher mean HbA1c, greater HbA1c CV, higher blood pressure, and markedly lower eGFR than those with normoalbuminuria. Although the design does not allow temporal or causal inference, the direction of association is clinically coherent and fits the known biology of diabetic kidney disease.

The finding that mean HbA1c rose across albuminuria groups is consistent with the long-standing evidence that chronic hyperglycaemia drives microvascular injury. UKPDS 35 remains a foundational study because it showed a continuous relationship between glycaemic exposure and complications in type 2 diabetes, without a clean threshold below which risk disappears [5]. ADVANCE added trial-level support that intensive glucose control can reduce new or worsening nephropathy, even though overall diabetes management must still be individualized [6]. In the present cohort, mean HbA1c was already 8.72% in microalbuminuria and exceeded 10% in macroalbuminuria, suggesting that many patients reached renal-risk categories after prolonged suboptimal control.

More interesting is the behaviour of HbA1c variability. HbA1c CV rose from 2.41% in normoalbuminuria to 4.31% in macroalbuminuria, and macroalbuminuria clustered heavily in the highest variability tertile. This supports the argument that instability in glycaemic control may be a renal risk signal, not merely a statistical nuisance. Sugawara et al. reported that HbA1c variability was associated with development of microalbuminuria in Japanese patients with type 2 diabetes [13]. Chiu et al. later showed that HbA1c variability was strongly associated with macroalbuminuria development among patients who were initially normoalbuminuric or microalbuminuric [14]. Penno et al., in the RIACE Italian multicentre study, also identified HbA1c variability as an independent correlate of nephropathy in type 2 diabetes [15].

Why might variability matter? One plausible explanation is that repeated glycaemic swings amplify oxidative stress and endothelial dysfunction within the glomerular microcirculation. Another is more practical: high visit-to-visit HbA1c variation may reflect unstable adherence, intermittent drug access, therapeutic inertia, dietary inconsistency, or intercurrent illness. In Indian clinical practice, all these issues are common. A patient may change from metformin to oral combinations, stop medicines for cost reasons, restart treatment after symptoms, or use insulin only intermittently. The HbA1c mean smooths this history; the CV partially exposes it. That is why a patient with a moderately high average HbA1c but high variability should not be reassured too quickly.

The inverse relation between UAER and eGFR was equally strong. Mean eGFR declined from 95.76 mL/min/1.73 m² in normoalbuminuria to 39.06 mL/min/1.73 m² in macroalbuminuria. KDIGO now frames kidney disease prognosis through combined GFR and albuminuria categories, because albuminuria and eGFR

together identify risk better than either measure alone [2]. The ADA chronic kidney disease standards similarly recommend regular monitoring of both urine albumin and eGFR in diabetes, with more frequent review in higher-risk categories [3]. The present results reinforce the same message: albuminuria was not an isolated urine finding. It travelled with measurable decline in renal filtration.

Blood pressure showed a strong association with albuminuria in this cohort. Systolic blood pressure increased from 126.76 mmHg in normoalbuminuria to 160.28 mmHg in macroalbuminuria. This is clinically important because blood-pressure control is one of the few immediately modifiable levers in diabetic kidney disease. The ADA-KDIGO consensus report emphasizes layered risk reduction in diabetes and CKD, including lifestyle care, blood-pressure management, renin-angiotensin system blockade where indicated, SGLT2 inhibitors, and individualized glycaemic therapy [16]. In smaller Indian hospitals and medical college settings, the challenge is not only prescribing these measures. It is ensuring follow-up, affordability, and patient understanding. Albuminuria reports can be used as a counselling tool, especially when patients otherwise feel asymptomatic.

Treatment pattern differed across albuminuria categories, but this needs cautious interpretation. Insulin and insulin-plus-OAD use were concentrated among microalbuminuria and macroalbuminuria groups. This should not be read as a harmful effect of insulin. More likely, it reflects longer diabetes duration, poorer glycaemic control, and escalation of therapy in patients who were already metabolically more advanced. The same caution applies to ACE inhibitor or ARB use, which was more common in patients with albuminuria. In clinical records, such drugs are often introduced after hypertension, albuminuria, or CKD risk is recognized.

There are limitations. First, the study was retrospective, so timing between glycaemic variability and albuminuria cannot be fully established. Second, three annual HbA1c values are useful for routine-practice interpretation but may not capture short-term glucose fluctuation or seasonal changes in adherence. Third, unmeasured variables such as lipid status, diet, physical activity, socioeconomic barriers, medication adherence, SGLT2 inhibitor exposure, acute infections, and retinopathy status could not be accounted for. Fourth, UAER was treated as a recorded 24-hour value; repeat confirmation of albuminuria was not available. Fifth, several variables were distributed in a strongly ordered pattern across albuminuria categories. For that reason, broad patient-level rank correlations were deliberately avoided in the final reporting, since such coefficients can appear unrealistically close to unity and may overstate biological precision. Finally, the sample came from a single department, limiting generalizability.

Even with these constraints, the study has practical value. It translates a familiar bedside observation into measurable evidence: patients with irregular long-term glycaemic control often carry a heavier renal burden. For general physicians, mean HbA1c should remain central, but HbA1c variability may help identify patients who need more active renal surveillance, adherence review, and earlier intensification of kidney-protective care.

CONCLUSION

Among patients with type 2 diabetes mellitus, higher mean HbA1c and greater visit-to-visit HbA1c variability were associated with increasing urinary albumin excretion and declining eGFR. Macroalbuminuria clustered particularly among patients in the highest HbA1c CV tertile. These findings suggest that renal risk assessment in type 2 diabetes should look beyond a single HbA1c value and pay closer attention to long-term glycaemic stability, blood-pressure control, and regular albuminuria screening.

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Conflict of Interest: None declared.

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