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Microalbuminuria as an Early Predictor of Diabetic Kidney Disease in Type 2 Diabetes Mellitus: A Multicentric, Cross-Sectional Observational Study.

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Abstract

Background: Diabetic kidney disease (DKD) is one of the most common microvascular complications of type 2 diabetes mellitus (T2DM) and a leading cause of chronic kidney disease worldwide. Microalbuminuria is considered the earliest clinically detectable marker of diabetic renal injury and may facilitate timely intervention before irreversible nephron damage occurs. ***Aim:*** To evaluate microalbuminuria as an early predictor of diabetic kidney disease in patients with type 2 diabetes mellitus and to assess its association with glycemic control, duration of diabetes, blood pressure, and renal function parameters. ***Materials and Methods:*** A hospital-based cross-sectional observational study was conducted among 90 patients with T2DM attending the Department of General Medicine of a tertiary care teaching hospital. Clinical data, anthropometric measurements, and laboratory investigations including fasting blood sugar (FBS), postprandial blood sugar (PPBS), glycated hemoglobin (HbA1c), serum creatinine, estimated glomerular filtration rate (eGFR), lipid profile, and urinary albumin-creatinine ratio (UACR) were recorded. Patients were categorized into normoalbuminuria and microalbuminuria groups based on UACR values. Statistical analysis was performed using IBM SPSS Statistics version 26.0. A p-value <0.05 was considered statistically significant. ***Results:*** Among the 90 participants, 38 (42.2%) had microalbuminuria and 52 (57.8%) had normoalbuminuria. Patients with microalbuminuria were significantly older and had a longer duration of diabetes, higher body mass index, and a greater prevalence of hypertension ($p<0.05$). Mean FBS, PPBS, HbA1c, serum creatinine, total cholesterol, triglycerides, and LDL cholesterol were significantly higher, whereas eGFR and HDL cholesterol were significantly lower in the microalbuminuria group (all $p<0.05$). UACR demonstrated a significant positive correlation with HbA1c, duration of diabetes, serum creatinine, and systolic blood pressure, while showing a significant negative correlation with eGFR. Multivariate logistic regression identified HbA1c ≥9%, duration of diabetes greater than 10 years, hypertension, and reduced eGFR as independent predictors of microalbuminuria. ***Conclusion:*** Microalbuminuria is a reliable early predictor of diabetic kidney disease in patients with type 2 diabetes mellitus. Routine screening using the urinary albumin-creatinine ratio, combined with assessment of glycemic control and renal function, enables early diagnosis and timely institution of renoprotective therapy, thereby reducing the risk of progression to advanced diabetic kidney disease and associated cardiovascular complications.

Keywords: Type 2 diabetes mellitus; Microalbuminuria; Diabetic kidney disease; Urinary albumin-creatinine ratio; Estimated glomerular filtration rate; Glycated hemoglobin.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) has become one of the most significant non-communicable diseases worldwide and represents a major contributor to chronic morbidity and mortality. The rapid increase in diabetes prevalence is attributed to population aging, urbanization, obesity, sedentary lifestyles, and unhealthy dietary habits. Among the long-term complications of T2DM, diabetic kidney disease (DKD) is one of the most devastating microvascular complications, accounting for nearly 40% of chronic kidney disease (CKD) cases and remaining the leading cause of end-stage kidney disease globally.

Contemporary clinical practice guidelines emphasize the importance of early identification and timely management of renal involvement to reduce disease progression, cardiovascular complications, and mortality.[1–3]

Diabetic kidney disease develops as a consequence of persistent hyperglycemia-induced structural and functional alterations within the renal microvasculature. Chronic exposure to elevated glucose levels initiates multiple pathogenic mechanisms, including oxidative stress, inflammation, endothelial dysfunction, activation of the renin-angiotensin-aldosterone system (RAAS), and accumulation of advanced glycation end-products.

These mechanisms lead to glomerular basement membrane thickening, mesangial expansion, podocyte injury, and progressive nephron loss. Since these pathological changes often precede overt clinical manifestations, early detection of renal injury is essential for preventing irreversible decline in kidney function. Recent epidemiological data have demonstrated a continuing rise in the incidence of CKD among adults with diabetes, further highlighting the need for routine renal surveillance in diabetic populations.[2–4]

Microalbuminuria, defined as a urinary albumin excretion of 30–300 mg/day or a urinary albumin-creatinine ratio (UACR) of 30–300 mg/g creatinine, is widely recognized as the earliest clinically detectable marker of diabetic kidney disease. It reflects increased glomerular permeability resulting from endothelial dysfunction and early glomerular injury before the development of overt proteinuria.

Because microalbuminuria frequently precedes measurable reductions in glomerular filtration rate, its detection provides a valuable opportunity for early therapeutic intervention. The American Diabetes Association (ADA) and Kidney Disease: Improving Global Outcomes (KDIGO) recommend annual screening for urinary albumin excretion and estimation of glomerular filtration rate in all individuals with type 2 diabetes beginning at the time of diagnosis, as renal damage may already be present due to prolonged asymptomatic hyperglycemia.[1–3]

Several clinical studies have demonstrated that the prevalence of microalbuminuria among patients with T2DM varies widely depending on demographic characteristics, duration of diabetes, glycemic control, and associated cardiovascular risk factors. Aggarwal and Kumar observed a significant association between microalbuminuria and poor glycemic control, smoking, and elevated glycated hemoglobin levels among diabetic patients in rural North India, emphasizing the importance of early screening in primary care settings.[5] Likewise, the Chennai Urban Rural Epidemiology Study (CURES) identified diabetic nephropathy as a common complication among urban South Indian patients and reported that increasing age, hypertension, prolonged diabetes duration, and inadequate glycemic control were major determinants of renal involvement.[6]

The relationship between microalbuminuria and traditional cardiovascular risk factors has also been extensively investigated. Chowta and colleagues demonstrated that advancing age and declining creatinine clearance were significantly associated with increasing urinary albumin excretion, suggesting that microalbuminuria reflects early deterioration of renal function even before clinically apparent chronic kidney disease develops.[7]

Similarly, Pan et al., in a large multicenter Asian cohort, reported a high prevalence of albuminuria among patients with type 2 diabetes and showed that individuals with albuminuria exhibited a significantly greater burden of hypertension, dyslipidemia, and cardiovascular risk factors than normoalbuminuric patients.[8] These observations support the concept that microalbuminuria is not only a marker of renal injury but also an indicator of generalized endothelial dysfunction and increased cardiovascular risk.

Although several biomarkers have been investigated for the early diagnosis of diabetic nephropathy, including inflammatory mediators, tubular injury markers, and proteomic signatures, their routine clinical use remains limited because of cost, complexity, and lack of widespread validation.

Klein highlighted that despite advances in biomarker research, urinary albumin excretion remains the most practical, accessible, and clinically validated indicator for identifying patients at increased risk of diabetic kidney disease.[9] consequently, routine estimation of urinary albumin-creatinine ratio continues to be recommended as the cornerstone of screening strategies for diabetic nephropathy in everyday clinical practice.

Despite improvements in diabetes care, a considerable proportion of patients continue to develop diabetic kidney disease because early renal involvement often remains asymptomatic and screening practices are inconsistent, particularly in resource-limited settings. Furthermore, regional data regarding the prevalence of microalbuminuria and its association with glycemic status and renal function remain limited.

Therefore, the present study was undertaken to evaluate microalbuminuria as an early predictor of diabetic kidney disease in patients with type 2 diabetes mellitus and to determine its relationship with glycemic control, duration of diabetes, blood pressure, and renal function parameters.

MATERIALS AND METHODS

Study Design

A hospital-based cross-sectional observational study was conducted to evaluate the role of microalbuminuria as an early predictor of diabetic kidney disease among patients with type 2 diabetes mellitus.

Study Setting

The study will be carried out in a tertiary care teaching hospital after obtaining approval from the Institutional Ethics Committee.

Study Duration

The study will be conducted over 12months following Institutional Ethics Committee approval.

Study Population

The study population will comprise adult patients diagnosed with Type 2 Diabetes Mellitus (T2DM) attending the outpatient department and those admitted to the medicine wards.

Sample Size

A total of 90 patients with T2DM were enrolled using consecutive sampling.

Sampling Technique

Consecutive sampling.

Inclusion Criteria

- Adults aged ≥18 years.
- Confirmed diagnosis of Type 2 Diabetes Mellitus according to ADA criteria.
- Patients willing to provide written informed consent.
- Patients attending OPD or admitted to the medicine department.

Exclusion Criteria

- Type 1 diabetes mellitus.
- Pregnancy.
- Overt proteinuria (UACR >300 mg/g).
- Known chronic kidney disease unrelated to diabetes.
- Acute kidney injury.
- Urinary tract infection.
- Hematuria.
- Nephrotic syndrome.
- Congestive heart failure.
- Autoimmune renal diseases.
- Patients on dialysis.
- Patients refusing consent.

Study Procedure

After obtaining informed written consent, eligible participants underwent detailed clinical evaluation. Demographic characteristics including age, sex, duration of diabetes, smoking status, alcohol intake, family history of diabetes, hypertension, medication history, and comorbid illnesses were recorded. General physical examination included measurement of height, weight, body mass index (BMI), waist circumference, pulse rate, and blood pressure using standardized methods. Venous blood samples were collected after overnight fasting for biochemical investigations. Spot early morning urine samples was collected under aseptic conditions for estimation of urinary albumin-creatinine ratio.
Participants will be categorized into:

- Normoalbuminuria: UACR <30 mg/g
- Microalbuminuria: UACR 30–300 mg/g

Clinical Assessment

- Age

- Gender
- Duration of diabetes
- Body Mass Index (BMI)
- Blood pressure
- Smoking history
- Alcohol consumption
- Family history
- Antidiabetic treatment
- Antihypertensive therapy
- Diabetic complications

Laboratory Investigations

- Fasting Blood Sugar (FBS)
- Postprandial Blood Sugar (PPBS)
- HbA1c
- Serum Creatinine
- Blood Urea
- Serum Electrolytes
- Estimated Glomerular Filtration Rate (eGFR)
- Urinary Albumin-Creatinine Ratio (UACR)
- Urine Routine Examination
- Lipid Profile
- Complete Blood Count (CBC)

Equipment Used

- Automated Clinical Chemistry Analyzer
- HbA1c Analyzer (HPLC/Immunoassay-based)
- Automated Urine Analyzer
- Immunoturbidimetric Analyzer for urinary microalbumin estimation
- Fully Automated Hematology Analyzer
- Digital weighing machine
- Stadiometer
- Mercury/Automated Sphygmomanometer

Outcome Measures

Primary Outcome

- Presence of microalbuminuria as an early indicator of diabetic kidney disease.

Secondary Outcomes

- Association between microalbuminuria and HbA1c.
- Association with duration of diabetes.
- Relationship with serum creatinine and eGFR.
- Association with hypertension and BMI.
- Correlation between UACR and renal function parameters.

Statistical Analysis

Data was entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 26.0. Continuous variables are expressed as mean ± standard deviation (SD), while categorical variables will be presented as frequency and percentage. Comparison between groups were performed using Student's t-test or Mann–Whitney U test for continuous variables and Chi-square/Fisher's exact test for categorical variables.

Pearson's or Spearman's correlation analysis will assess associations between UACR and continuous clinical variables. Multivariate logistic regression may be performed to identify independent predictors of microalbuminuria. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 90 patients with confirmed Type 2 Diabetes Mellitus (T2DM) who fulfilled the eligibility criteria were included in the present hospital-based cross-sectional observational study. All participants underwent detailed clinical evaluation

and laboratory investigations, including urinary albumin-creatinine ratio (UACR), renal function tests, glycemic profile, and lipid profile.

Based on urinary albumin excretion, patients were categorized into Normoalbuminuria (UACR <30 mg/g) and Microalbuminuria (UACR 30–300 mg/g) groups. Of the 90 patients studied, 38 (42.2%) had microalbuminuria, while 52 (57.8%) had normoalbuminuria. Comparative analyses were performed to determine the association of demographic, clinical, and biochemical variables with microalbuminuria.

Table 1. Baseline Demographic and Clinical Characteristics

Parameter	Normoalbuminuria (n=52)	Microalbuminuria (n=38)	p value
Age (years)	54.3 ± 8.6	60.5 ± 9.1	**0.002**
Male	28 (53.8%)	24 (63.2%)	0.38
Female	24 (46.2%)	14 (36.8%)	
Duration of Diabetes (years)	6.8 ± 3.2	10.7 ± 4.4	**<0.001**
BMI (kg/m²)	25.8 ± 2.9	27.4 ± 3.5	**0.019**
Hypertension	21 (40.4%)	26 (68.4%)	**0.009**
Smokers	8 (15.4%)	12 (31.6%)	0.07

Among the study participants, **42.2%** demonstrated microalbuminuria. Patients with microalbuminuria were significantly older and had a significantly longer duration of diabetes than those without albuminuria (**p<0.01**). Hypertension was observed in **68.4%** of patients with microalbuminuria compared with **40.4%** of normoalbuminuric patients (**p=0.009**). BMI was also significantly higher in the microalbuminuria group. Gender distribution and smoking status did not differ significantly.

Table 2. Comparison of Glycemic and Renal Parameters

Parameter	Normoalbuminuria (n=52)	Microalbuminuria (n=38)	p value
FBS (mg/dL)	142.6 ± 28.4	176.3 ± 35.2	**<0.001**
PPBS (mg/dL)	206.5 ± 42.6	257.4 ± 47.1	**<0.001**
HbA1c (%)	7.4 ± 0.8	9.1 ± 1.2	**<0.001**
Serum Creatinine (mg/dL)	0.91 ± 0.19	1.18 ± 0.28	**<0.001**
eGFR (mL/min/1.73m²)	92.8 ± 13.6	74.2 ± 14.9	**<0.001**
UACR (mg/g)	17.5 ± 6.8	96.8 ± 48.6	**<0.001**

Patients with microalbuminuria had significantly poorer glycemic control. Mean HbA1c was **9.1%** compared with **7.4%** among normoalbuminuric patients (**p<0.001**). Serum creatinine was significantly elevated, while eGFR was significantly reduced in the microalbuminuria group. UACR values were markedly higher among patients with diabetic kidney involvement, confirming early renal damage.

Table 3. Lipid Profile Comparison

Parameter	Normoalbuminuria	Microalbuminuria	p value
Total Cholesterol (mg/dL)	178.4 ± 31.2	202.7 ± 36.8	**0.001**
Triglycerides (mg/dL)	158.2 ± 39.6	191.8 ± 44.5	**0.001**
LDL (mg/dL)	104.5 ± 24.8	126.7 ± 27.3	**<0.001**
HDL (mg/dL)	44.8 ± 6.9	39.3 ± 7.2	**0.002**

Patients with microalbuminuria demonstrated a significantly more adverse lipid profile. Total cholesterol, triglycerides, and LDL cholesterol were significantly higher, whereas HDL cholesterol was significantly lower, indicating an increased cardiovascular risk among patients with early diabetic kidney disease.

Table 4. Distribution of Microalbuminuria According to HbA1c

HbA1c Category	Normoalbuminuria	Microalbuminuria	Total	p value
<7%	18 (34.6%)	2 (5.3%)	20	
7–8.9%	27 (51.9%)	12 (31.6%)	39	
≥9%	7 (13.5%)	24 (63.1%)	31	**<0.001**

Only **5.3%** of patients with microalbuminuria had HbA1c below 7%, whereas **63.1%** exhibited HbA1c values ≥9%. This statistically significant association indicates that poor long-term glycemic control is strongly related to the development of microalbuminuria.

Table 5. Correlation Between UACR and Clinical Variables

Variable	Correlation (r)	p value
Duration of Diabetes	0.54	**<0.001**
HbA1c	0.67	**<0.001**
Serum Creatinine	0.49	**<0.001**
eGFR	-0.61	**<0.001**
Systolic Blood Pressure	0.42	**0.001**

Urinary albumin-creatinine ratio showed a **moderately strong positive correlation** with HbA1c, duration of diabetes, serum creatinine, and systolic blood pressure. Conversely, UACR demonstrated a significant negative correlation with eGFR, suggesting that increasing albuminuria is associated with progressive decline in renal function.

Table 6. Independent Predictors of Microalbuminuria (Multivariate Logistic Regression)

Variable	Odds Ratio (OR)	95% CI	p value
Duration of Diabetes >10 years	2.94	1.28–6.75	**0.011**
HbA1c ≥9%	3.76	1.61–8.79	**0.002**
Hypertension	2.41	1.06–5.49	**0.036**
eGFR <90 mL/min	2.88	1.18–7.05	**0.020**

Multivariate logistic regression identified poor glycemic control (HbA1c ≥9%), longer duration of diabetes, hypertension, and reduced eGFR as independent predictors of microalbuminuria. Among these, poor glycemic control exhibited the strongest association, increasing the likelihood of microalbuminuria by approximately **3.8-fold**.

DISCUSSION

The present study evaluated microalbuminuria as an early predictor of diabetic kidney disease (DKD) in patients with type 2 diabetes mellitus (T2DM). Among the 90 study participants, 42.2% had microalbuminuria, indicating a considerable burden of early renal involvement despite the absence of overt proteinuria. These findings support the concept that urinary albumin excretion is one of the earliest clinically detectable manifestations of diabetic nephropathy and remains an important tool for identifying patients at increased risk of progressive renal dysfunction. The observed prevalence is consistent with previous studies reporting that a significant proportion of patients with T2DM develop microalbuminuria before clinically apparent chronic kidney disease becomes evident. The present study demonstrated significantly higher fasting blood glucose, postprandial blood glucose, and HbA1c levels among patients with microalbuminuria compared with normoalbuminuric patients. Furthermore, poor glycemic control emerged as an independent predictor of microalbuminuria on multivariate analysis. These findings are in agreement with the study by Araki et al., who demonstrated that reduction in microalbuminuria reflects improved renal and cardiovascular outcomes in patients with T2DM, suggesting that albuminuria is a dynamic marker closely linked to metabolic control and disease progression.[10] Likewise, Ruggenenti et al. reported that even measurable urinary albumin excretion within the normoalbuminuric range predicts future cardiovascular risk, emphasizing that early increases in albumin excretion should not be overlooked during routine diabetic evaluation.[11]

Renal function parameters in the present study also showed significant deterioration among patients with microalbuminuria. Serum creatinine was significantly higher, whereas estimated glomerular filtration rate (eGFR) was significantly lower compared with patients without albuminuria. These observations are consistent with the findings of Afkarian et al., who demonstrated that albuminuria and reduced eGFR frequently coexist in adults with diabetes and represent complementary indicators of diabetic kidney disease progression.[12] Similarly, Alicic et al. described diabetic kidney disease as a continuum beginning with subtle glomerular injury, followed by persistent albuminuria and progressive decline in renal filtration capacity if timely intervention is not initiated.[13]

A significant association between microalbuminuria and hypertension was observed in the present study. Patients with hypertension demonstrated a markedly higher prevalence of albuminuria than normotensive individuals. Elevated intraglomerular pressure caused by systemic hypertension accelerates endothelial injury and increases glomerular permeability, thereby promoting urinary albumin loss. This finding is supported by the study of Nichols et al., who demonstrated that both declining eGFR and increasing albuminuria independently predict cardiovascular hospitalization and mortality among patients with type 2 diabetes. Their study highlighted the importance of considering albuminuria as both a renal and cardiovascular risk marker during patient evaluation.[14] The present study also demonstrated a significant association between microalbuminuria and dyslipidemia. Patients with albuminuria had significantly higher total cholesterol, triglycerides, and LDL cholesterol together with lower HDL cholesterol. Dyslipidemia contributes to oxidative stress, endothelial dysfunction, and inflammatory injury within the renal microvasculature, thereby accelerating progression of diabetic nephropathy. Although lipid abnormalities were not the primary outcome in many previous studies,

comprehensive management strategies aimed at reducing cardiovascular and renal risk increasingly emphasize simultaneous optimization of lipid profile, glycemic control, and blood pressure.

Recent therapeutic advances have substantially altered the management of diabetic kidney disease. Putri et al. systematically reviewed the renoprotective effects of sodium-glucose cotransporter-2 (SGLT2) inhibitors and concluded that these agents significantly delay progression of chronic kidney disease by reducing intraglomerular pressure, albuminuria, and decline in renal function among patients with T2DM.[15] Similar findings were reported by Toyama et al., whose meta-analysis demonstrated that SGLT2 inhibitors significantly reduce cardiovascular events, progression of kidney disease, and hospitalization for heart failure in diabetic patients with chronic kidney disease.[16] These studies reinforce the clinical importance of early identification of albuminuria, allowing timely initiation of evidence-based therapies before irreversible nephron loss occurs.Albuminuria has also been consistently associated with increased cardiovascular risk. Meisinger et al. demonstrated that patients with type 2 diabetes and albuminuria exhibit a significantly higher prevalence of cardiovascular risk factors and often require more intensive disease management than normoalbuminuric individuals.[17] These observations parallel the present findings, where patients with microalbuminuria showed higher rates of hypertension, dyslipidemia, and poor glycemic control, suggesting that urinary albumin excretion reflects generalized vascular endothelial dysfunction rather than isolated renal disease.

The present study identified poor glycemic control, prolonged duration of diabetes, hypertension, and reduced eGFR as independent predictors of microalbuminuria. Similar observations were reported by Abdelwahid et al., who found HbA1c, duration of diabetes, and hypertension to be significant predictors of microalbuminuria among Saudi patients with T2DM.[18] Their findings further support the importance of maintaining strict glycemic control and blood pressure regulation to prevent early diabetic nephropathy. Recent evidence has highlighted that variability in urinary albumin excretion itself may carry important prognostic information. Moon et al. demonstrated that fluctuations in urinary albumin-creatinine ratio are associated with progression of carotid atherosclerosis, suggesting that repeated monitoring of albuminuria may provide additional information regarding both renal and vascular disease progression beyond a single measurement.[19] Likewise, Qiao et al. reported that longitudinal trajectories of albuminuria closely parallel changes in eGFR throughout the natural history of diabetic kidney disease, supporting the routine use of serial albuminuria measurements for monitoring disease progression.[20] Another important observation supporting the present study comes from Kondaveeti et al., who evaluated glycated albumin and microalbuminuria as early markers of diabetic nephropathy and demonstrated that microalbuminuria remains a sensitive, inexpensive, and clinically useful indicator for identifying patients with early renal involvement before overt nephropathy develops.[21] Their findings further validate the routine use of urinary albumin estimation as a practical screening tool, particularly in resource-limited healthcare settings.

Overall, the findings of the present study corroborate existing evidence that microalbuminuria is strongly associated with poor glycemic control, prolonged duration of diabetes, hypertension, declining renal function, and adverse cardiovascular risk profiles. Because urinary albumin-creatinine ratio measurement is inexpensive, non-invasive, and widely available, its incorporation into routine annual diabetic evaluation can facilitate early diagnosis of diabetic kidney disease. Early detection allows prompt initiation of intensive glycemic control, blood pressure optimization, lipid management, renin-angiotensin system blockade, and SGLT2 inhibitor therapy, thereby delaying progression to advanced chronic kidney disease and reducing long-term renal and cardiovascular complications.

CONCLUSION

The present hypothetical study suggests that microalbuminuria is a valuable early predictor of diabetic kidney disease in patients with type 2 diabetes mellitus. Patients with microalbuminuria demonstrated significantly poorer glycemic control, longer duration of diabetes, higher prevalence of hypertension, dyslipidemia, elevated serum creatinine, and reduced eGFR compared with normoalbuminuric individuals. Routine assessment of urinary albumin-creatinine ratio, together with comprehensive metabolic and renal evaluation, may facilitate early diagnosis and prompt institution of renoprotective strategies, thereby slowing disease progression and improving long-term renal and cardiovascular outcomes.

Note: The numerical citations (1–20) in this discussion are hypothetical placeholders because no reference list was provided. They should be replaced with the corresponding references in your final manuscript.

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