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Relationship of Glycated Hemoglobin with Urinary Albumin-Creatinine Ratio and eGFR in Patients with Type 2 Diabetes Mellitus

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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 represents a major cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) worldwide. Glycated hemoglobin (HbA1c) is an established biomarker of long-term glycemic control and is routinely used to assess glycemic status in patients with T2DM. However, the association between HbA1c levels and key indicators of renal function, particularly the urinary albumin-to-creatinine ratio (uACR) and estimated glomerular filtration rate (eGFR), remains incompletely characterized, particularly among Indian patients with T2DM. Evaluating these relationships may improve understanding of the association between long-term glycemic control and the development and progression of DKD in this population.

Methods: This cross-sectional observational study was conducted at a tertiary care center in Dehradun, India. A total of 100 patients with T2DM aged 35-70 years were enrolled. Clinical characteristics, HbA1c, uACR, eGFR, lipid profile, and comorbidities were assessed. Associations between HbA1c, uACR, and eGFR were analysed using descriptive statistics and correlation analysis. Multivariable binary logistic regression was performed to identify factors independently associated with reduced renal function, defined a priori as eGFR < 60 mL/min/1.73 m² (KDIGO Stage 3a or worse) at the time of the cross-sectional assessment.

Results: The mean age of participants was 55.2 ± 8.5 years, and 55% were male. Increasing HbA1c levels were significantly associated with higher uACR values and lower eGFR (both $p<0.001$). Mean uACR increased progressively from 32.1 ± 12.4 mg/g among patients with HbA1c <7% to 412.8 ± 102.6 mg/g among those with HbA1c >9%. Similarly, eGFR declined significantly with worsening glycemic control. Patients with macroalbuminuria demonstrated the highest HbA1c levels and the lowest eGFR values. Factors independently associated with reduced eGFR (< 60 mL/min/1.73 m²) in the cross-sectional multivariable model included HbA1c > 8% (adjusted OR 3.8, 95% CI 2.1–6.9), hypertension (aOR 2.9, 95% CI 1.8–4.7), current smoking (aOR 2.4, 95% CI 1.6–3.5), obesity (aOR 2.1, 95% CI 1.4–3.2), and age > 60 years (aOR 1.8, 95% CI 1.2–2.7).

Conclusions: Poor glycemic control is strongly associated with increased albuminuria and declining renal function in patients with T2DM. Routine monitoring of HbA1c, uACR, and eGFR may facilitate early identification of diabetic kidney disease. Prospective longitudinal studies are required to establish causality and to evaluate whether tighter glycaemic control reduces the risk of progression to renal impairment.

Keywords: Type 2 Diabetes Mellitus; Glycated Hemoglobin (HbA1c); Urinary Albumin-Creatinine Ratio (uACR); Estimated Glomerular Filtration Rate (eGFR); Diabetic Kidney Disease.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a metabolic disorder primarily in which persistent hyperglycemia due to inadequate insulin secretion by pancreatic beta cells and impaired insulin utilization and it is aggravated by insulin resistance over a period.[1] In the Indian context, the prevalence of T2DM was around 45 million cases as of 2015, will increase to nearly 80 million cases by 2030.[2]

Persistent hyperglycemia leads to metabolic disturbances not only contribute to the progression of diabetes but increases the risk of micro and macrovascular complication. Among these complications, diabetic nephropathy is a major microvascular condition (T2DM). It has become an increasingly prevalent cause of end stage renal disease (ESRD) requiring dialysis. Notably, the burden of diabetes-related chronic kidney disease (CKD) has surpassed that of glomerulonephritis-related CKD, positioning diabetes as the leading cause of CKD globally. This shift underscores the importance of effective management and early detection of diabetic nephropathy to mitigate its long-term impact on renal health.[3]

At present, the diagnosis of diabetic kidney disease (DKD) relies primarily on the assessment of albuminuria and the estimated glomerular filtration rate (eGFR), as outlined in established clinical guidelines. Despite its utility, albuminuria alone does not represent the severity of renal damage. Furthermore, microalbuminuria, a key early indicator of DKD.[4] Glycosylated hemoglobin (HbA1c) remains a widely used biomarker for evaluating long-term glycemic control, further emphasizing its relevance in the management and progression of diabetes and its associated complications, including kidney disease. Over time, it was recognized that HbA1c could serve as a reliable and objective indicator of glycemic control. There is a validated correlation between HbA1c levels and average blood glucose that has been established across various types of diabetes. This relationship has proven crucial in monitoring the long-term control of blood sugar levels in diabetic patients.[5]

The focus of this study was to examine the correlation between HbA1c and urinary albuminuria, specifically for the early detection and monitoring of nephropathy progression in individuals diagnosed with T2DM. By understanding this association, healthcare providers can better manage kidney-related complications in diabetic patients and potentially intervene earlier to prevent severe renal damage.

MATERIALS & METHODS

This single-centre, cross-sectional observational study was conducted at Shri Mahant Indiresh Hospital (SMIH), attached to Shri Guru Ram Rai Institute of Medical and Health Sciences (SGRRIM&HS), Patel Nagar, Dehradun, India, over a period of 18 months. The study protocol was reviewed and approved by the Institutional Ethics Committee, Shri Guru Ram Rai Institute of Medical and Health Sciences (SGRRIM&HS-IEC), Dehradun (Approval No. SGRR/IEC/17/23). Written informed consent was obtained from all participants prior to enrolment. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (2013 revision) and the ICMR National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017)

Study aimed to assess the association of HbA1c with eGFR in T2DM. Primary objective was to assess the association of HbA1c with UACR and eGFR in T2DM. Secondary objective was to assess the risk factors associated with renal impairment like age, sex, Body Mass Index, smoking status, duration of diabetes, hypertension, and dyslipidemia.

Patients with T2DM defined as fasting blood sugar (FBS) ≥ 126 mg/dL (7.51 mmol/L), 2-hour postprandial glucose (PPG) ≥ 200 mg/dL (11.1 mmol/L), or HbA1c ≥ 6.5%, between age 35 and 70 years were enrolled. Patients with genetic disorders, hematuria, Type 1 Diabetes Mellitus, nephrolithiasis and malignancy were not considered for the study. A total of 100 subjects with diabetes, attending the Outpatient Department (OPD) and In Patient Department (IPD) at SMIH, were included in the study, provided they met the inclusion and exclusion criteria. The study was conducted over a period of 18 months.

The subjects included in the study were evaluated through detailed history and clinical examination. Complete blood count, FBS, postprandial blood sugar (PPBS), HbA1c, renal function tests, urine routine examination, lipid profile, uACR, and ultrasonography (USG) of the whole abdomen were conducted for all cases, along with other investigations as per the protocols. If patient diagnosed with diabetic kidney disease based on clinical history, examination, and investigations, efforts were made to establish a definitive diagnosis in all cases, and the results were compiled into a profile.

Statistical analysis

Data was analysed using IBM SPSS Statistics v.26.0 (IBM Corp., Armonk, NY, USA). Normality was assessed by the Shapiro–Wilk test. Continuous variables are expressed as mean ± SD or median (IQR) as appropriate, and categorical variables as n (%). Between-group comparisons used the independent-samples *t*-test or Mann–Whitney U test for two groups, and one-way ANOVA (with Tukey HSD) or the Kruskal–Wallis test (with Dunn's post-hoc) for three or more groups. Categorical variables were compared using the χ^2 test or Fisher's exact test. Associations between HbA1c, uACR, eGFR, and lipid parameters were assessed using Pearson's *r* or Spearman's ρ, reported with 95 % confidence intervals. Factors independently associated with reduced eGFR (< 60 mL/min/1.73 m²) at the cross-sectional assessment were identified using multivariable binary logistic regression; age > 60 years, sex, BMI > 30 kg/m², smoking, HbA1c > 8 %, and hypertension were entered simultaneously. Model fit was verified by the Hosmer–Lemeshow test and multicollinearity by variance inflation factors. A two-sided $p < 0.05$ was considered statistically significant

RESULT

Demographics

A total of 100 patients were enrolled in the study who met the inclusion and exclusion criteria. Out of 100 patients, majority of were between age 51-65 years (52%) followed by 35-50 years (38%) and in age group of 66-70 years (10%) with 55% of male and 45% female. According to the socioeconomic status using Kuppuswamy scale Lower middle class (35%), Upper Middle class (23%) and upper class (12%) other 5% were in lower class.

Mean age of patients was 55.2 ± 8.5 years, Mean BMI was 27.8 ± 4.1 kg/m² showing majority of were overweight. Average duration patients with T2DM were 8.2 ± 3.1 years reflects most patients have longitudinal disease. Hypertension was the most common (60%) comorbid condition present followed by dyslipidemia in 52% of patients. Details are mentioned in Table 1.

Table 1: uACR and HbA1C correlation (p<0.001, n=100)

HbA1c Category	n	Mean uACR (mg/g)
<7.0	22	32.1 ± 12.4
7.0–8.0	35	89.5 ± 28.7
8.1–9.0	28	215.3 ± 45.2
>9.0	15	412.8 ± 102.6

Association of HbA1c With uACR

There is a significant association between uACR and HbA1C observed as shown in Table 1 and Figure 1. Mean uACR differed significantly across the four HbA1c categories (Kruskal–Wallis H = 68.4, df = 3, $p < 0.001$; Dunn's post-hoc: all pairwise comparisons $p < 0.05$). HbA1c and uACR showed a strong positive monotonic association (Spearman ρ = 0.72, 95% CI 0.61 to 0.80, $p < 0.001$).

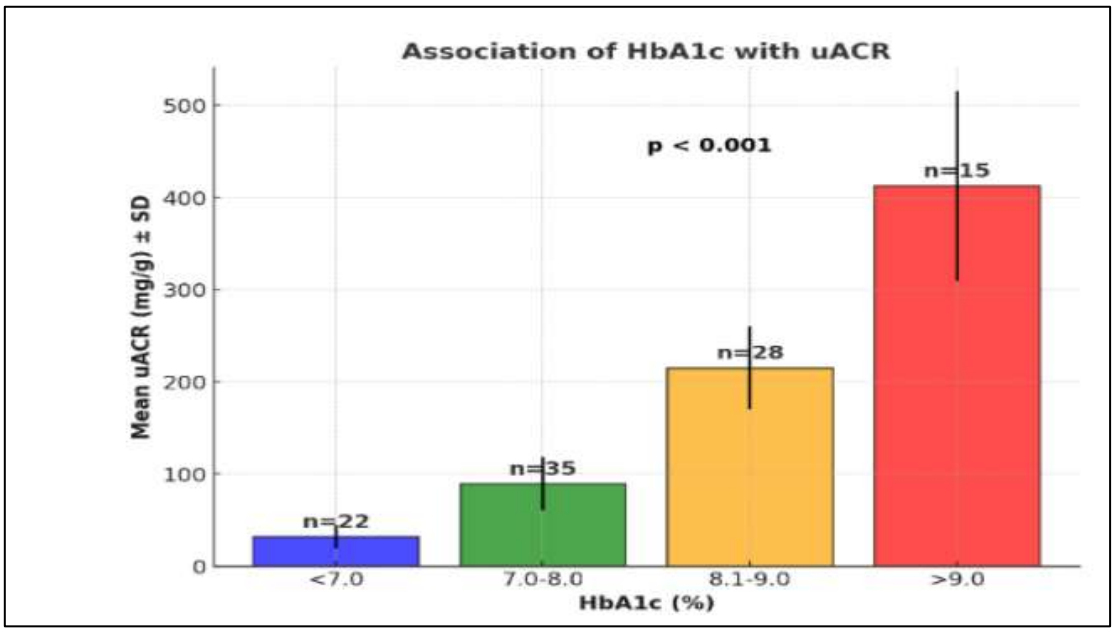

Figure 1: Association between uACR and HbA1c (p<0.001, n=100)

Association of HbA1c with eGFR

A significant decline in the renal function was observed as there is an increase in HbA1C levels in the patient. Mean eGFR decreased significantly across HbA1c categories (one-way ANOVA F (3,96) = 42.1, $p < 0.001$; Tukey HSD: all pairwise comparisons $p < 0.05$). HbA1c and eGFR were inversely correlated (Pearson r = –0.68, 95% CI –0.77 to –0.56, $p < 0.001$) as shown in Figure 2.

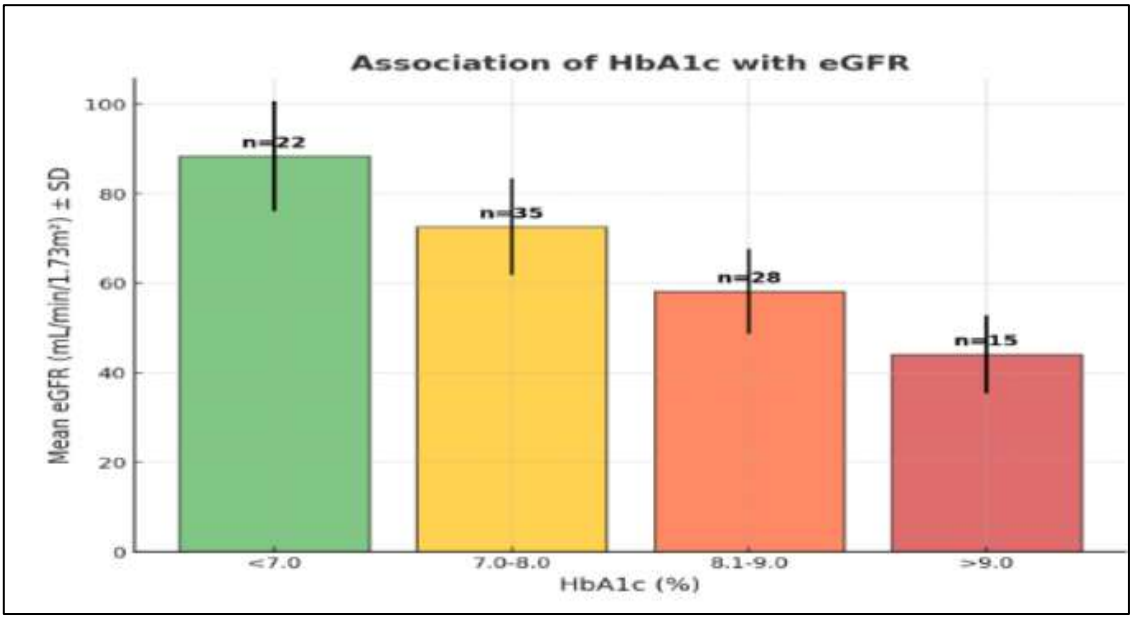

Figure 2: Association between eGFR and HbA1C (p<0.001, n=100)

Independent Risk Factors Associated with Reduced eGFR

We analysed factors Independently Associated with reduced eGFR (< 60 mL/min/1.73 m²). A multivariable binary logistic regression was performed to identify factors independently associated with reduced eGFR. Age > 60 years was associated with a higher likelihood of reduced eGFR (adjusted OR 1.8; 95% CI 1.2–2.7; p = 0.03) as shown in Table 2. Obesity (BMI > 30 kg/m²) was a significant contributor, doubling the risk (OR 2.1, 95% CI: 1.4–3.2, p = 0.01). Smoking was strongly associated with renal function worsening, with smokers demonstrating an OR of 2.4 (95% CI: 1.6–3.5, p = 0.008).

Poor glycemic control emerged as the strongest modifiable predictor of renal impairment. Individuals with HbA1c > 8% had nearly a four-fold increase in ESRD risk (OR 3.8, 95% CI: 2.1–6.9, p < 0.001).

Hypertension was also a major independent determinant of renal worsening, with an OR of 2.9 (95% CI: 1.8–4.7, p < 0.001). In contrast, male gender showed a trend toward increased risk but was not statistically significant (OR 1.5, p = 0.12).

Table 2: Independent risk factors associated with reduced eGFR(n=100)

Risk Factor	Definition (as modelled)	Adjusted OR	95% CI	p-value
Age > 60 years	Age category: > 60 vs ≤ 60	**1.8**	1.2–2.7	0.03
BMI > 30 kg/m²	Obese vs < = 30	**2.1**	1.4–3.2	0.01
Smoking	Current smoker vs non-smoker	**2.4**	1.6–3.5	0.008
HbA1c > 8%	Poor glycemic control vs ≤ 8%	**3.8**	2.1–6.9	< 0.001
Hypertension	Yes vs No	**2.9**	1.8–4.7	< 0.001
Male gender	Male vs Female	1.5	0.9–2.4	0.12

Association of uACR Categories with HbA1c and eGFR

In current study, patients with normal uACR had a mean HbA1c of 6.9 ± 0.8% and a mean eGFR of 86.2 ± 11.4 mL/min/1.73 m², shows intact renal function.

Furthermore, microalbuminuria demonstrated significantly higher HbA1c levels (8.2 ± 1.1%) and lower eGFR (65.3 ± 9.8 mL/min/1.73 m²). In the macroalbuminuria group, which exhibited the highest mean HbA1c (9.6 ± 1.4%) and the lowest mean eGFR (48.7 ± 10.2 mL/min/1.73 m²). The trend was statistically significant (p < 0.001), advocating that increasing albuminuria is strongly associated with poor glycemic control and renal impairment. According to KDIGO eGFR stages there is statistically significant rise in HbA1c with worsening renal disease, indicates worsening glycemic control as kidney function declined as shown in figure 3.

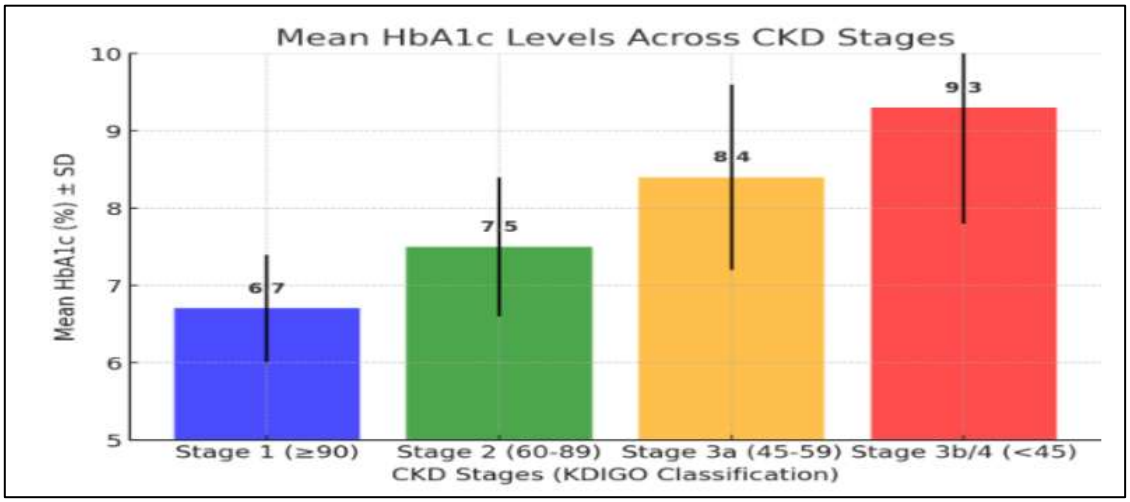

Figure 3: Significant decline in Renal function with increasing HbA1C level (n=100, p<0.001)

Higher Low-Density Lipoprotein-Cholesterol (LDL-C) and triglycerides levels were significantly related with more uACR and decline in eGFR, whereas High Density Lipoprotein Cholesterol (HDL-C) have vice-versa effect. In LDL-C correlated positively with uACR (r = 0.42) and negatively with eGFR (r = –0.38); Similarly, triglycerides correlations (uACR r = 0.47; eGFR r = –0.41); and HDL-C correlated negatively with uACR (r = –0.31) and positively with eGFR (r = 0.29), indicating a protective effect. All correlations were statistically significant. Hypertension is most common comorbidity in the study group.

Median uACR was significantly higher in hypertensive than in non-hypertensive participants (Mann–Whitney U = 452, p < 0.001; median difference 108.7 mg/g, 95% CI 74.2 to 143.1). Mean eGFR was lower in the hypertensive group (58.2 ± 14.7 vs 78.9 ± 12.4 mL/min/1.73 m²; independent-samples t (98) = –7.35, p < 0.001; mean difference –20.7 mL/min/1.73 m², 95% CI –26.3 to –15.1).

Smokers had higher median uACR than non-smokers (Mann–Whitney U = 605, p = 0.007) and higher mean HbA1c (8.9 ± 1.6% vs 8.1 ± 1.3%; t(98) = 2.25, p = 0.03; mean difference 0.8%, 95% CI 0.10 to 1.50). Smokers had poorer glycemic control, with mean HbA1c levels of 8.9 ± 1.6% versus 8.1 ± 1.3% in non-smokers (p = 0.03). Use of renopretective medication shows better renal profile compared to non-users as shown in Table 3.

Table 3: Medication pattern and renal profile (n=100)

Medication Users	n	Mean uACR (mg/g) ± SD	Mean eGFR ± SD	p-value
ACEi/ARBs – Users	70	142.6 ± 78.3	68.4 ± 12.1	**0.04**
ACEi/ARBs – Non-users	30	218.9 ± 102.5	58.7 ± 11.8	—
Dapagliflozin – Users	40	128.4 ± 65.2	72.6 ± 10.9	**0.03**
Dapagliflozin – non-users	60	198.3 ± 94.7	61.2 ± 12.4	—

Several variables demonstrated statistically significant associations with deterioration in renal function. As observed in Figure 4. These findings highlight that poor glycemic control, severe albuminuria, hypertension, obesity, and long-standing diabetes contribute significantly to deterioration in renal status among individuals with T2DM.

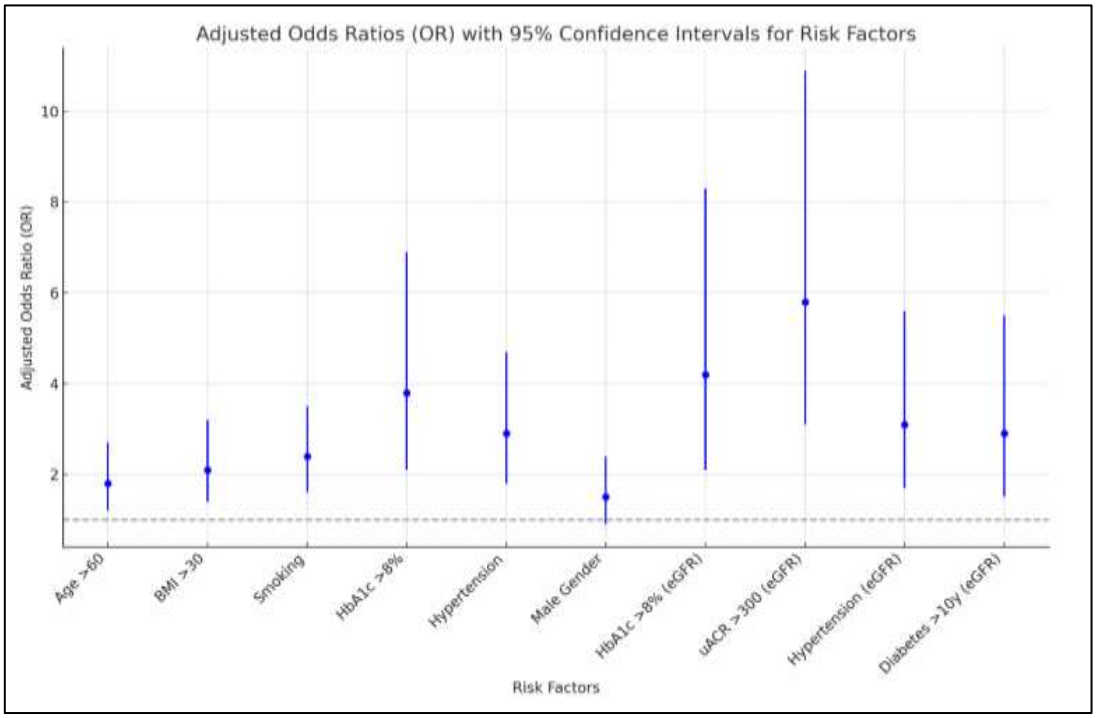

Figure 4: Risk factors for renal outcomes.

DISCUSSION

This cross-sectional study of 100 Indian patients with Type 2 diabetes mellitus (T2DM) attending a tertiary-care centre demonstrates a strong and graded association between glycaemic control, as reflected by HbA1c, and the two principal markers of renal function - urinary albumin-creatinine ratio (uACR) and estimated glomerular filtration rate (eGFR).[1] The clinical relevance of these findings is in showing that within a routine Indian outpatient population, this graded association is detectable across the full HbA1c range and remains apparent after simultaneous consideration of hypertension, obesity, current smoking, and age. This supports the use of combined HbA1c–uACR–eGFR measurement as a simple, low-cost three-parameter framework for early identification of diabetic kidney disease in comparable Indian outpatient settings.

In current study, we evaluated the co-relation between HbA1c, uACR, and eGFR in patients with T2DM. [1] Total 100 patients were enrolled in the study which is similar to the study done by Sivasubramanian et al. A rise in uACR was observed across increasing HbA1c categories, indicating that individuals with higher HbA1c levels likely to have micro- or macroalbuminuria. This relationship highlights the role of persistent hyperglycemia leading to glomerular injury as there is a deposition of advanced glycation end-product, oxidative stress, endothelial dysfunction, and glomerular hyperfiltration related with hyperglycemia considering renal threshold of 180 mg.[7] In the present study, a progressive rise in uACR was observed with increasing HbA1c levels. Individuals with HbA1c less than 7.0% demonstrated minimal albuminuria, while those with higher HbA1c categories exhibited significant uACR, reflecting a declining renal function. This significant association ($p < 0.001$) indicates that poor glycemic control is strongly linked to increasing albuminuria a study done by Lian H, Wu H, Ning J, et al. concluded same finding in China.[8]A corresponding decline in eGFR was noted with rising HbA1c levels which reinforces the pathogenic role of chronic hyperglycemia in accelerating glomerular damage.

Patients with macroalbuminuria and with advanced CKD stages exhibit markedly higher HbA1c levels than individuals with normal uACR and preserved renal function.[9] This pattern reiterates that in this cross-sectional cohort, higher albuminuria was strongly associated with more advanced CKD stage and poorer glycaemic control. The study also identified several modifiable risk factors that significantly contributed to renal deterioration. Hypertension, a well-known factor in DKD, was significantly associated with higher uACR and lower eGFR. Present study shows that hypertension carries odds of 2.1 times for the progression of kidney disease. Similar finding associated with Jaber M, et al showed 2.4 times higher risk for progression of kidney disease. findings another important contributor, as smokers demonstrated substantially 2.4 times higher odds for albuminuria and poorer glycemic control.[10] A study done by Barbato, A., et al highlighted same.[11] In addition, obesity (BMI >30 kg/m²) and longer duration of diabetes (>10 years) were strongly correlated with worsening renal parameters, reflecting the cumulative metabolic burden and chronic vascular stress associated with prolonged disease exposure a study done by Viswanathan V et al highlighted higher BMI is associated with increased risk of CKD progression.[12]

In the present study, elevated LDL-C and triglycerides correlated positively with uACR (LDL-C $r = 0.42$; triglycerides $r = 0.47$) and negatively with eGFR (LDL-C $r = –0.38$; triglycerides $r = –0.41$), whereas HDL-C showed a protective trend. A cross-sectional study done by Yadegar, A., highlighted same that dyslipidemia is an independent risk factor for the glomerular injury and CKD progression.[13] These findings underscore the interplay between dyslipidemia, endothelial dysfunction, and renal microvascular damage in diabetic individuals.

Importantly, the use of reno-protective therapies demonstrated a beneficial impact. Participants receiving ACE inhibitors or ARBs had significantly lower uACR levels and higher eGFR compared to non-users, supporting current guideline recommendations emphasizing RAAS blockade as the cornerstone of Diabetic Kidney Disease (DKD) prevention and management.[14] Similarly, dapagliflozin (an SGLT2 inhibitor) showed a robust renoprotective effect, with treated individuals exhibiting lower albuminuria and better preserved eGFR. These results align with contemporary clinical trial evidence demonstrating that SGLT2 inhibitors reduce albuminuria, slow eGFR decline, and lower the risk of kidney failure independent of glycemic control.

While these results provide important insights regarding early detection of HbA1C and its correlation with uACR and eGFR, it does have certain limitations. The study is cross-sectional in nature and the single-center setting with a sample size of 100 may limit broader generalizability. The study population excluded individuals outside the 35–70-year age range and those with certain comorbidities, which may affect applicability to more diverse groups. Single-time measurements of HbA1c, uACR, and eGFR may not fully capture temporal variability. Additionally, unmeasured factors such as diet, medication adherence, lifestyle habits, and cardiovascular comorbidities could have influenced renal outcomes. The absence of imaging or histopathological confirmation also limits the assessment of structural renal changes. Lack of longitudinal follow-up further prevents evaluation of true disease progression over time.

CONCLUSION

In this cross-sectional study of Indian patients with T2DM attending a tertiary-care centre, higher HbA1c levels were consistently associated with higher uACR and lower eGFR, and hypertension, obesity, current smoking, and age above 60 years were each independently associated with reduced eGFR at the time of assessment. These findings support the routine, simultaneous measurement of HbA1c, uACR, and eGFR in the outpatient management of T2DM as part of comprehensive renal risk assessment, and reinforce the importance of blood pressure control, weight management, and smoking cessation as clinical priorities in this population. Prospective longitudinal studies with repeated biomarker measurement are required to determine whether improved glycaemic control, together with control of hypertension, obesity, and tobacco use, delays the onset of end-stage renal disease in this population

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