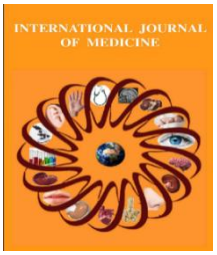

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Original Research

Correlation of Glycated Hemoglobin with Lipid Profile and Renal Function Parameters in Patients with Type 2 Diabetes Mellitus: An Observational Study

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Abstract

Background: Type 2 diabetes mellitus is frequently accompanied by atherogenic dyslipidaemia and renal injury. Glycated hemoglobin reflects sustained glycaemic exposure, but its relationship with routine lipid and renal indices requires evaluation in individual settings. **Objectives:** To determine the correlation of glycated hemoglobin with lipid profile and renal function parameters in adults with type 2 diabetes mellitus. **Methods:** This hospital-based cross-sectional study included 100 adults with type 2 diabetes mellitus at Government Medical College, Suryapet, Telangana, India, from April 2025 to March 2026. Glycated hemoglobin, lipid profile, serum urea, serum creatinine, estimated glomerular filtration rate, and urine albumin-to-creatinine ratio were measured. Participants were compared using a glycated hemoglobin threshold of 7.0%. Correlation and multivariable linear regression analyses were performed. **Results**: The mean age was 55.1 ± 10.8 years, and 58.0% were males. Mean glycated hemoglobin was 8.3 ± 1.8%; 72.0% had values of at least 7.0%. Poor glycaemic control was associated with adverse lipid values, higher serum urea, serum creatinine, and urine albumin-to-creatinine ratio, and lower estimated glomerular filtration rate. Glycated hemoglobin correlated positively with triglycerides (r=0.47), low-density lipoprotein cholesterol (r=0.39), serum creatinine (r=0.36), and urine albumin-to-creatinine ratio (ρ=0.48), and inversely with estimated glomerular filtration rate (r=−0.43); all p<0.001. Triglycerides, estimated glomerular filtration rate, urine albumin-to-creatinine ratio, and diabetes duration remained independently associated with glycated hemoglobin. **Conclusion**: Poor glycaemic control was closely linked to an adverse lipid pattern and early renal dysfunction. Concurrent assessment of glycated hemoglobin, lipid profile, estimated glomerular filtration rate, and albuminuria can strengthen cardiometabolic and renal risk stratification in type 2 diabetes mellitus.

Keywords: *albuminuria; diabetic kidney disease; dyslipidaemia; estimated glomerular filtration rate; glycated hemoglobin; type 2 diabetes mellitus.*

Introduction

Type 2 diabetes mellitus is a major metabolic disorder characterised by persistent hyperglycaemia arising from insulin resistance, progressive beta-cell dysfunction, and altered hepatic glucose metabolism. India carries a substantial and heterogeneous burden of diabetes and related cardiometabolic abnormalities. The nationwide ICMR-INDIAB study reported that diabetes, dyslipidaemia, hypertension, and obesity frequently coexist, creating a large population at risk of vascular and renal complications.1 Glycated hemoglobin (HbA1c) reflects average glycaemic exposure over the preceding two to three months and remains a central measure for monitoring long-term control. For many non-pregnant adults, an HbA1c target below 7.0% is recommended, although treatment goals require individualisation.2

Dyslipidaemia is a common metabolic accompaniment of type 2 diabetes mellitus. Insulin resistance increases adipose tissue lipolysis, hepatic free-fatty-acid delivery, very-low-density lipoprotein production, and triglyceride-rich remnant particles. It also promotes small dense low-density lipoprotein cholesterol and reduces high-density lipoprotein cholesterol. This atherogenic pattern contributes to the high cardiovascular risk observed in diabetes. Contemporary guidance therefore emphasises regular lipid assessment and integrated control of glucose, blood pressure, smoking, body weight, and lipid concentrations.3,7 HbA1c could have additional clinical value when it parallels adverse lipid changes, because one routinely obtained marker would then provide information about both glycaemic status and cardiometabolic risk.

Diabetic kidney disease is another important complication and is identified clinically through reduced estimated glomerular filtration rate (eGFR), increased urinary albumin excretion, or both. Albuminuria can precede a measurable decline in filtration and is independently associated with cardiovascular and kidney outcomes. Current recommendations support periodic assessment of serum creatinine-derived eGFR and urine albumin-to-creatinine ratio (UACR) in patients with type 2 diabetes mellitus.4,5 Chronic hyperglycaemia promotes advanced glycation, oxidative stress, endothelial dysfunction, glomerular hyperfiltration, and progressive structural renal damage. Consequently, higher HbA1c levels are biologically expected to accompany albuminuria and declining renal function, although the magnitude of these relationships differs across populations and stages of disease.

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Several studies have described associations between HbA1c and total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol.8-11 Other investigations have linked poor or variable glycaemic control with albuminuria and diabetic kidney disease.12,13 However, simultaneous assessment of lipid and renal parameters remains limited in many Indian tertiary-care settings. Local data are useful because treatment patterns, duration of diabetes, obesity, hypertension, dietary practices, and access to monitoring can modify these associations. The present study therefore aimed to evaluate the correlation of HbA1c with lipid profile and renal function parameters among adults with type 2 diabetes mellitus and to identify biochemical and clinical factors independently associated with HbA1c.

Materials and Methods

Study design and setting

A hospital-based cross-sectional observational study was conducted in the Department of General Medicine, Government Medical College and its associated teaching hospital, Suryapet, Telangana, India, from October 2024 to May 2025. Reporting followed the Strengthening the Reporting of Observational Studies in Epidemiology principles.

Study population.

Adults aged 18 years or older with a documented diagnosis of type 2 diabetes mellitus who attended the outpatient department or were admitted for routine clinical care were screened. Patients who provided written informed consent and had complete HbA1c, fasting lipid profile, serum urea, serum creatinine, eGFR, and UACR measurements were eligible. Exclusion criteria were type 1 or gestational diabetes, acute hyperglycaemic emergencies, acute kidney injury, primary or obstructive renal disease, nephrotic syndrome, active infection, decompensated liver disease, recent major surgery, and incomplete biochemical records. Consecutive eligible patients were enrolled to minimise selection based on investigator preference.

Sample size and recruitment.

The minimum sample size was estimated for detecting a correlation coefficient of 0.28 between HbA1c and a biochemical parameter, with a two-sided alpha level of 0.05 and 80% power. The calculated requirement was 98 participants and was rounded to 100. Of 106 patients assessed, six were excluded and 100 were analysed.

Data collection and laboratory assessment.

A structured form recorded age, sex, body mass index, diabetes duration, hypertension, smoking, family history of premature cardiovascular disease, and antidiabetic treatment. After an overnight fast of 8-12 hours, venous blood was collected. HbA1c was measured using a National Glycohemoglobin Standardization Program-aligned high-performance liquid chromatography method. Lipids, serum urea, and serum creatinine were measured using validated automated chemistry methods. The eGFR was calculated using the 2021 CKD-EPI creatinine equation.6 A first-morning spot urine sample was used to determine UACR. Albuminuria was classified as normal to mildly increased below 30 mg/g, moderately increased at 30-300 mg/g, and severely increased above 300 mg/g.4,5 Adequate glycaemic control was defined as HbA1c below 7.0%.2 Lipid abnormalities were classified using predefined laboratory thresholds consistent with contemporary recommendations.3,7

Statistical analysis.

Data were analysed using IBM SPSS Statistics, version 26.0. Continuous variables were summarised as mean ± standard deviation or median with interquartile range; categorical variables were reported as frequency and percentage. Independent-samples t-test or Mann-Whitney U test was used for continuous group comparisons, while chi-square test or Fisher's exact test was used for categorical variables. Pearson correlation assessed normally distributed variables; Spearman rank correlation was used for UACR. Multivariable linear regression was performed with HbA1c as the dependent variable. UACR was logarithmically transformed because of skewness. Age, sex, body mass index, diabetes duration, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, eGFR, and log-transformed UACR were entered into the model. Statistical significance was set at $p<0.05$.

Ethical considerations.

Necessary Permissions were obtained before starting the study. Written informed consent was obtained from every participant, and study data were anonymised before analysis.

Results

Participant recruitment and study population

During the study period, 106 patients with type 2 diabetes mellitus were assessed for eligibility. Six patients were excluded: three did not satisfy the eligibility criteria, two declined participation, and one had incomplete biochemical data. The remaining 100 patients were enrolled and included in the final analysis. Complete glycated hemoglobin, lipid profile, and renal function measurements were available for all participants.

The mean age of the study population was 55.1 ± 10.8 years, with a range of 32-78 years. Fifty-eight (58.0%) participants were males and 42 (42.0%) were females. The mean body mass index was 26.4 ± 4.1 kg/m². Thirty-one (31.0%) patients were obese, and 38 (38.0%) were overweight.

The median duration of type 2 diabetes mellitus was 7 years (interquartile range [IQR]: 4-11 years). Forty-seven (47.0%) patients had hypertension, 18 (18.0%) were current smokers, and 22 (22.0%) had a family history of premature cardiovascular disease. Oral antidiabetic drugs alone were used by 65 (65.0%) patients, whereas 35 (35.0%) were receiving insulin either alone or in combination with oral agents.

Glycaemic status, lipid profile, and renal function

The mean glycated hemoglobin level was 8.3 ± 1.8%. Twenty-eight (28.0%) patients had adequate glycaemic control, defined as HbA1c <7.0%, whereas 72 (72.0%) had HbA1c ≥7.0%. Among patients with inadequate glycaemic control, 44 (44.0%) had HbA1c values between 7.0% and 8.9%, while 28 (28.0%) had HbA1c ≥9.0%.

The mean total cholesterol level was 198.6 ± 42.1 mg/dL, mean triglyceride level was 176.4 ± 75.2 mg/dL, and mean low-density lipoprotein cholesterol was 123.5 ± 35.7 mg/dL. The mean high-density lipoprotein cholesterol level was 42.8 ± 10.2 mg/dL. According to the predefined lipid thresholds, elevated triglyceride levels were observed in 54 (54.0%) patients, elevated low-density lipoprotein cholesterol in 61 (61.0%), and reduced high-density lipoprotein cholesterol in 46 (46.0%).

The mean serum urea and creatinine levels were 31.6 ± 10.8 mg/dL and 1.05 ± 0.34 mg/dL, respectively. The mean estimated glomerular filtration rate was 83.7 ± 20.5 mL/min/1.73 m². Twelve (12.0%) patients had an estimated glomerular filtration rate below 60 mL/min/1.73 m². The median urine albumin-to-creatinine ratio was 46 mg/g (IQR: 18-128 mg/g). Normoalbuminuria was present in 55 (55.0%) patients, moderately increased albuminuria in 31 (31.0%), and severely increased albuminuria in 14 (14.0%). The overall clinical and biochemical profile is shown in Table 1.

Table 1. Clinical and biochemical characteristics of the study population

Characteristic	Total, n=100
Age, years, mean ± SD	55.1 ± 10.8
Male sex	58 (58.0)
Body mass index, kg/m², mean ± SD	26.4 ± 4.1
Overweight	38 (38.0)
Obesity	31 (31.0)
Duration of diabetes, years, median (IQR)	7 (4-11)
Hypertension	47 (47.0)
Current smoking	18 (18.0)
HbA1c, %, mean ± SD	8.3 ± 1.8
HbA1c <7.0%	28 (28.0)
HbA1c 7.0-8.9%	44 (44.0)
HbA1c ≥9.0%	28 (28.0)
Total cholesterol, mg/dL, mean ± SD	198.6 ± 42.1
Triglycerides, mg/dL, mean ± SD	176.4 ± 75.2
LDL cholesterol, mg/dL, mean ± SD	123.5 ± 35.7
HDL cholesterol, mg/dL, mean ± SD	42.8 ± 10.2
Serum urea, mg/dL, mean ± SD	31.6 ± 10.8
Serum creatinine, mg/dL, mean ± SD	1.05 ± 0.34
eGFR, mL/min/1.73 m², mean ± SD	83.7 ± 20.5
Urine albumin-to-creatinine ratio, mg/g, median (IQR)	46 (18-128)
eGFR <60 mL/min/1.73 m²	12 (12.0)
Moderately increased albuminuria	31 (31.0)
Severely increased albuminuria	14 (14.0)

Data are presented as number (percentage), mean ± standard deviation, or median (interquartile range). HbA1c: glycated hemoglobin; LDL: low-density lipoprotein; HDL: high-density lipoprotein; eGFR: estimated glomerular filtration rate.

Comparison according to glycaemic control

Patients with HbA1c ≥7.0% had significantly higher mean total cholesterol levels than those with HbA1c <7.0% (205.7 ± 43.0 versus 180.3 ± 34.5 mg/dL; p=0.003). Triglyceride levels were also markedly higher among patients with inadequate glycaemic control (191.5 ± 78.6 versus 137.5 ± 49.8 mg/dL; p<0.001).

Mean low-density lipoprotein cholesterol was significantly higher in the HbA1c ≥7.0% group than in the HbA1c <7.0% group (129.5 ± 36.3 versus 108.2 ± 29.1 mg/dL; p=0.003). Conversely, high-density lipoprotein cholesterol was lower among patients with inadequate glycaemic control (41.2 ± 10.2 versus 47.0 ± 9.0 mg/dL; p=0.007).

Patients with HbA1c ≥7.0% had higher serum urea and creatinine levels and lower estimated glomerular filtration rates. The median urine albumin-to-creatinine ratio was 62 mg/g (IQR: 25-154 mg/g) among patients with inadequate glycaemic control, compared with 19 mg/g (IQR: 10-44 mg/g) among those with adequate control (p<0.001). Albuminuria was present in 38 of 72 (52.8%) patients with HbA1c ≥7.0% and in 7 of 28 (25.0%) patients with HbA1c <7.0% (p=0.013). An eGFR below 60 mL/min/1.73 m² was observed in 11 (15.3%) patients with inadequate glycaemic control and in 1 (3.6%) patient with adequate control. Detailed group comparisons are presented in Table 2.

Table 2. Lipid and renal parameters according to glycaemic control

Parameter	HbA1c <7.0%, n=28	HbA1c ≥7.0%, n=72	p-value
HbA1c, %, mean ± SD	6.4 ± 0.4	9.0 ± 1.4	<0.001
Total cholesterol, mg/dL	180.3 ± 34.5	205.7 ± 43.0	0.003
Triglycerides, mg/dL	137.5 ± 49.8	191.5 ± 78.6	<0.001
LDL cholesterol, mg/dL	108.2 ± 29.1	129.5 ± 36.3	0.003
HDL cholesterol, mg/dL	47.0 ± 9.0	41.2 ± 10.2	0.007
Serum urea, mg/dL	27.6 ± 8.1	33.2 ± 11.3	0.007
Serum creatinine, mg/dL	0.91 ± 0.22	1.10 ± 0.36	0.002
eGFR, mL/min/1.73 m²	94.8 ± 17.2	79.4 ± 20.4	<0.001
Urine albumin-to-creatinine ratio, mg/g	19 (10-44)	62 (25-154)	<0.001
Albuminuria	7 (25.0)	38 (52.8)	0.013
eGFR <60 mL/min/1.73 m²	1 (3.6)	11 (15.3)	0.174

Continuous data are presented as mean ± standard deviation or median (interquartile range), and categorical data as number (percentage). The independent-samples t-test, Mann-Whitney U test, chi-square test, or Fisher's exact test was used as appropriate.

Correlation of HbA1c with lipid profile and renal function

Glycated hemoglobin showed a significant positive correlation with total cholesterol (r=0.34; p=0.001), triglycerides (r=0.47; p<0.001), and low-density lipoprotein cholesterol (r=0.39; p<0.001). A significant inverse correlation was observed between HbA1c and high-density lipoprotein cholesterol (r=−0.31; p=0.002). The strongest lipid association was between HbA1c and triglyceride concentration.

HbA1c was positively correlated with serum urea (r=0.29; p=0.003) and serum creatinine (r=0.36; p<0.001). A moderate inverse correlation was found between HbA1c and eGFR (r=−0.43; p<0.001). UACR demonstrated a significant positive correlation with HbA1c (Spearman's ρ=0.48; p<0.001). The complete correlation matrix is shown in Table 3.

Table 3. Correlation of HbA1c with lipid and renal parameters

Parameter	Correlation coefficient	p-value
Total cholesterol	0.34	0.001
Triglycerides	0.47	<0.001
LDL cholesterol	0.39	<0.001
HDL cholesterol	−0.31	0.002
Serum urea	0.29	0.003
Serum creatinine	0.36	<0.001
Estimated glomerular filtration rate	−0.43	<0.001
Urine albumin-to-creatinine ratio	0.48	<0.001

Pearson's correlation coefficient was used for normally distributed variables. Spearman's rank correlation coefficient was used for urine albumin-to-creatinine ratio because of its skewed distribution.

Multivariable analysis

A multivariable linear regression model was constructed with HbA1c as the dependent variable. Age, sex, body mass index, duration of diabetes, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, eGFR, and logarithmically transformed UACR were included as explanatory variables.

After adjustment, triglyceride level remained independently and positively associated with HbA1c (standardised β=0.29; p=0.002). eGFR was independently and inversely associated with HbA1c (standardised β=−0.25; p=0.006). UACR was positively associated with HbA1c (standardised β=0.24; p=0.008), while a longer duration of diabetes independently predicted higher HbA1c levels (standardised β=0.18; p=0.035). Low-density lipoprotein cholesterol showed a positive association that did not reach statistical significance (standardised β=0.15; p=0.082). Age, sex, body mass index, and high-density lipoprotein cholesterol were not independently associated with HbA1c. The model explained 38% of the variation in HbA1c (adjusted R^2=0.38; overall p<0.001) (Table 4).

Table 4. Multivariable linear regression analysis of factors associated with HbA1c

Variable	Standardised β coefficient	p-value
Age	0.07	0.384
Male sex	0.05	0.521
Body mass index	0.11	0.169
Duration of diabetes	0.18	0.035
Triglycerides	0.29	0.002
LDL cholesterol	0.15	0.082
HDL cholesterol	−0.10	0.214
Estimated glomerular filtration rate	−0.25	0.006
Log-transformed urine albumin-to-creatinine ratio	0.24	0.008

Dependent variable: HbA1c. Adjusted R^2=0.38; overall model p<0.001.

Discussion

This study demonstrated a clinically coherent relationship between chronic glycaemic exposure, dyslipidaemia, and renal impairment in adults with type 2 diabetes mellitus. Nearly three-quarters of participants had HbA1c values of at least 7.0%. Those with inadequate control had higher total cholesterol, triglycerides, low-density lipoprotein cholesterol, serum urea, serum creatinine, and UACR, together with lower high-density lipoprotein cholesterol and eGFR. The correlation pattern remained consistent after adjustment: triglycerides, eGFR, UACR, and duration of diabetes were independently associated with HbA1c. These observations support the concept that poor glycaemic control clusters with both macrovascular and microvascular risk markers rather than occurring as an isolated biochemical abnormality.

The positive correlations of HbA1c with total cholesterol, triglycerides, and low-density lipoprotein cholesterol, and its inverse relationship with high-density lipoprotein cholesterol, agree with earlier reports. Khan et al. observed direct associations of HbA1c with atherogenic lipids and an inverse association with high-density lipoprotein cholesterol.8 Hussain et al. similarly reported that HbA1c predicted hypercholesterolaemia, elevated low-density lipoprotein cholesterol, and triglyceride abnormalities.9 Naqvi et al. identified a significant independent relationship between HbA1c and triglycerides,10 while Sharahili et al. confirmed significant associations with cholesterol and triglyceride concentrations in primary-care patients.11 In the present analysis, triglycerides showed the strongest lipid correlation and remained significant in the regression model. Insulin resistance and relative insulin deficiency plausibly explain this pattern through increased free-fatty-acid flux, enhanced hepatic triglyceride synthesis, and reduced clearance of triglyceride-rich particles.

Renal findings were equally important. HbA1c correlated positively with serum creatinine and UACR and inversely with eGFR. Albuminuria was approximately twice as frequent in patients with HbA1c of at least 7.0% as in those below this threshold. Abdelwahid et al. reported that microalbuminuria was associated with poor glycaemic control and hypertension among patients with type 2 diabetes.12 Longitudinal evidence

from Hsu et al. further showed that HbA1c variability independently predicted the development of microalbuminuria.13 Persistent hyperglycaemia activates advanced glycation, oxidative stress, inflammatory signalling, and intraglomerular haemodynamic changes. These processes impair the filtration barrier, increase urinary albumin loss, and ultimately reduce filtration capacity. The independent associations of both UACR and eGFR with HbA1c indicate that albumin excretion and filtration provide complementary information.

The findings have practical relevance for routine diabetic care. HbA1c should not be interpreted only as a treatment target; a high value should prompt systematic review of lipid status, kidney function, albuminuria, blood pressure, treatment adherence, and cardiovascular risk. Current standards recommend integrated risk-factor management and regular eGFR and UACR screening.3-5 The UK Prospective Diabetes Study established that improved glycaemic control reduces microvascular complications,14 but glycaemic treatment alone does not address the full risk burden. Periodic combined testing can identify patients who require intensified lifestyle measures, lipid-lowering therapy, renoprotective treatment, medication review, and closer surveillance. Because the present study was conducted in a public tertiary-care setting, its results also underline the value of incorporating these accessible laboratory markers into structured follow-up pathways.

LIMITATIONS

The cross-sectional design prevented assessment of temporal sequence or causality. Participants were recruited from a single tertiary-care institution, which restricts generalisability to community populations. A single HbA1c, lipid profile, creatinine, and UACR measurement was analysed, so biological variability could not be examined. Residual confounding from diet, physical activity, lipid-lowering treatment, medication adherence, and diabetes complications remained possible despite multivariable adjustment.

Conclusion

Among adults with type 2 diabetes mellitus, higher HbA1c was associated with an atherogenic lipid profile, greater urinary albumin excretion, higher serum creatinine, and lower estimated glomerular filtration rate. Triglycerides, eGFR, UACR, and duration of diabetes retained independent relationships with HbA1c after adjustment. These findings support concurrent monitoring of glycaemic control, lipids, and renal indices during routine follow-up. Patients with persistently elevated HbA1c require comprehensive cardiovascular and kidney risk assessment rather than glucose-focused management alone. Early recognition of dyslipidaemia and albuminuria, followed by integrated treatment and regular surveillance, can improve risk stratification and guide timely preventive care across tertiary and primary clinical practice settings in diverse Indian and international populations.

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