RESEARCH PAPER

Glycated Haemoglobin and Renal Function in Patients with Diabetic Chronic Kidney Disease: A Single-Centre Prospective Observational Study

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Abstract

Background: Diabetic chronic kidney disease (CKD) is a leading cause of end-stage renal disease. Glycated haemoglobin (HbA1c) reflects long-term glycaemic control, but whether a single HbA1c value tracks renal function or short-term functional decline in patients with established diabetic CKD remains uncertain.

Objective: To examine the association between HbA1c and estimated glomerular filtration rate (eGFR), and between HbA1c and 120-day change in eGFR, in patients with diabetic CKD.

Methods: In this single-centre prospective observational study, 210 adults with known diabetes mellitus and CKD were assessed at baseline for HbA1c, fasting and postprandial glucose, renal function tests, abdominal ultrasonography and eGFR, and were re-assessed for renal function at 120 days. Associations were tested using Pearson/Spearman correlation, one-way ANOVA, the chi-square test, and multivariable linear regression, with two-tailed $p<0.05$ considered significant.

Results: Mean age was 49.0 ± 12.0 years; 127 (60.5%) were male. Mean HbA1c was 7.14 ± 1.34% and mean baseline eGFR 54.9 ± 22.8 mL/min/1.73 m². HbA1c did not correlate with baseline eGFR ($r = -0.05$; 95% CI −0.19 to 0.08; $p = 0.45$), and mean eGFR did not differ across HbA1c categories (ANOVA $F(5,204) = 0.62$; $p = 0.68$). HbA1c >7% was not associated with eGFR <60 mL/min/1.73 m² (OR 0.93; 95% CI 0.54–1.60; $p = 0.78$). eGFR declined modestly over 120 days (mean −8.2 ± 26.4 mL/min/1.73 m²), but this change was highly variable (90/210 patients (43%) improved) and was unrelated to HbA1c on univariable ($r = 0.05$; $p = 0.45$) and multivariable analysis (adjusted $\beta = -0.34$; $p = 0.59$).

Conclusion: In this cohort of patients with established diabetic CKD, a single HbA1c measurement was not associated with concurrent renal function or with short-term change in eGFR. HbA1c alone may be insufficient to stratify renal risk in established diabetic CKD, and these findings should be interpreted in light of the study's limitations.

Keywords: Diabetic nephropathies; Glycated haemoglobin A; Glomerular filtration rate; Diabetes mellitus, type 2; Renal insufficiency, chronic; Disease progression.

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.1. Introduction

Diabetes mellitus affects an estimated 537 million adults worldwide, and diabetic kidney disease develops in 30–40% of people with diabetes, accounting for a large share of end-stage renal disease and imposing a substantial burden on patients and health systems.[1,2] Progressive loss of glomerular filtration is central to this trajectory, making the identification of accessible prognostic markers clinically important.[3]

Glycated haemoglobin (HbA1c) reflects mean glycaemia over the preceding two to three months and is the standard measure of long-term glycaemic control,[4] and current guidelines define HbA1c-

based glycaemic targets on this basis.[5] Landmark trials such as the DCCT[6] and UKPDS[7] established that intensive glycaemic control reduces the incidence of microvascular complications, including new-onset nephropathy, in patients without established kidney disease.

Whether these findings extend to patients with already-established diabetic CKD is less certain. Once nephron loss is substantial, non-glycaemic drivers—haemodynamic injury, structural fibrosis, ageing and comorbidity—may dominate, and several studies have reported that HbA1c has limited prognostic value in advanced diabetic kidney disease.[8] Measures capturing glycaemic variability rather than a single HbA1c value have also been proposed as more informative for renal decline.[9]

Against this background, we examined, in a real-world cohort of patients with diabetic CKD spanning early to advanced stages, whether a single HbA1c measurement was associated with concurrent renal function (eGFR) and with short-term (120-day) change in renal function.

2. Materials and Methods

2.1 Study design and setting

This single-centre prospective observational study was conducted in the Department of General Medicine, in collaboration with the Nephrology and General Medicine outpatient departments, emergency room and inpatient wards, at Shri Sathya Sai Medical College and Research Institute, Sri Balaji Vidyapeeth, Tamil Nadu, India. Participants were assessed at baseline and renal function was re-assessed after 120 days. The study is reported in accordance with the STROBE statement for observational studies.

2.2 Participants

Adults of either sex with known diabetes mellitus and known chronic kidney disease were eligible. Patients were excluded if they had acute kidney injury (a >50% fall in GFR within three months), hypertensive nephropathy, renal cell carcinoma, hepatorenal syndrome, pyonephrosis or pyelonephritis, or were non-compliant with medication or follow-up.

2.3 Variables and measurements

The exposure of interest was HbA1c, categorised for descriptive analysis using American Diabetes Association thresholds. The primary outcomes were baseline eGFR and the change in eGFR at 120 days. Fasting and postprandial blood glucose, serum urea and creatinine, and abdominal ultrasonography (renal echogenicity) were recorded. HbA1c was measured by HPLC method, and eGFR was estimated using the CKD-EPI equation. CKD was staged by eGFR according to KDIGO criteria.[10]

2.4 Sample size

Assuming a 22% prevalence of type 2 diabetes among CKD patients and a 6% absolute precision, the required sample using $n = 4pq/L^2$ was 191; allowing for a 10% non-response rate gave a target of 210 participants.

2.5 Statistical analysis

Data were analysed using SPSS version 23. Continuous variables are presented as mean ± standard deviation and categorical variables as frequencies and percentages. The HbA1c–eGFR relationship was assessed using Pearson and Spearman correlation; mean eGFR across HbA1c categories using one-way ANOVA; the association between HbA1c >7% and eGFR <60 mL/min/1.73 m² using the chi-square test with an odds ratio and 95% confidence interval; and baseline versus 120-day eGFR using the paired t-test. A multivariable linear regression model examined 120-day eGFR change as a function of HbA1c, adjusting for baseline eGFR and age. A two-tailed $p<0.05$ was considered statistically significant.

3. Results

3.1 Participant characteristics

A total of 210 patients were studied. The mean age was 49.0 ± 12.0 years (range 20–70), and 127 (60.5%) were male. Baseline clinical and biochemical characteristics are shown in Table 1. Increased renal cortical echogenicity was present in 130 patients (61.9%). Mean serum urea was 47.9 ± 8.9 mg/dL and mean creatinine 1.77 ± 0.33 mg/dL.

Table 1. Baseline characteristics of the study population (n = 210).

Characteristic	Value
Age, years (mean ± SD)	49.0 ± 12.0
20–30 / 31–40 / 41–50, n	17 / 28 / 60
51–60 / >60, n	71 / 34
Male / Female, n (%)	127 (60.5) / 83 (39.5)
HbA1c, % (mean ± SD)	7.14 ± 1.34
Fasting blood glucose, mg/dL (mean ± SD)	115.9 ± 16.7
Postprandial blood glucose, mg/dL (mean ± SD)	172.3 ± 28.6
Serum urea, mg/dL (mean ± SD)	47.9 ± 8.9
Serum creatinine, mg/dL (mean ± SD)	1.77 ± 0.33
Baseline eGFR, mL/min/1.73 m² (mean ± SD)	54.9 ± 22.8
Increased renal echogenicity, n (%)	130 (61.9)

Oliguria / Lower-limb oedema, n	90 / 100
Facial puffiness / Breathlessness, n	85 / 120

3.2 HbA1c distribution and CKD stage

HbA1c ranged from 4.8% to 9.5%; 105 patients (50.0%) had HbA1c >7% (Table 2). By eGFR, most patients were in KDIGO stages G2–G4, with none in stage G5 (Table 3).

Table 2. Distribution of HbA1c (n = 210).

HbA1c category (%)	n	%
<5.7	35	16.7
5.7–6.1	19	9.0
6.2–6.9	47	22.4
7.0–7.9	38	18.1
8.0–8.9	50	23.8
≥9.0	21	10.0
Total	210	100.0

Table 3. KDIGO CKD stage by baseline eGFR (n = 210).

CKD stage (eGFR, mL/min/1.73 m²)	n	%
G1 (≥90)	20	9.5
G2 (60–89)	70	33.3
G3a (45–59)	31	14.8
G3b (30–44)	49	23.3
G4 (15–29)	40	19.0
G5 (<15)	0	0.0
Total	210	100.0

3.3 HbA1c and renal function

HbA1c showed no correlation with baseline eGFR (Pearson r = −0.05; 95% CI −0.19 to 0.08; p = 0.45; Spearman ρ = −0.05; p = 0.50; Figure 1). Mean eGFR was similar across all HbA1c categories (range 50.7–60.2 mL/min/1.73 m²), with no significant between-group difference (one-way ANOVA $F(5,204) = 0.62$; p = 0.68; Table 4, Figure 2). Patients with HbA1c >7% did not differ from those with HbA1c ≤7% in mean eGFR (53.7 ± 21.4 vs 56.1 ± 24.2; p = 0.46), and HbA1c >7% was not associated with eGFR <60 mL/min/1.73 m² (OR 0.93; 95% CI 0.54–1.60; $\chi^2 = 0.08$; p = 0.78; Table 5).

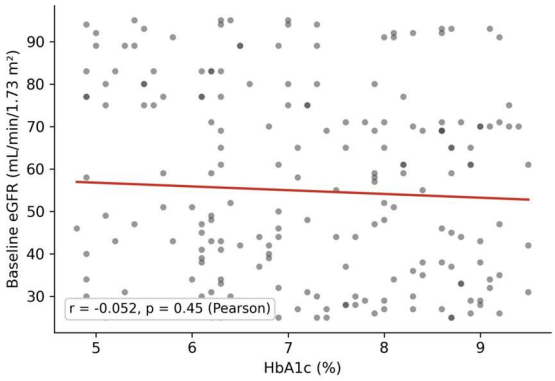

Figure 1. Scatter plot of HbA1c against baseline eGFR (n = 210). The fitted line is nearly flat, consistent with the absence of correlation.

Table 4. Mean baseline eGFR across HbA1c categories.

HbA1c (%)	n	Mean eGFR ± SD (mL/min/1.73 m²)
<5.7	35	60.2 ± 26.0
5.7–6.1	19	50.7 ± 20.7
6.2–6.9	47	52.6 ± 23.0
7.0–7.9	38	54.9 ± 23.0
8.0–8.9	50	55.4 ± 21.6
≥9.0	21	53.6 ± 21.7

One-way ANOVA: $F(5,204) = 0.62$, p = 0.68.

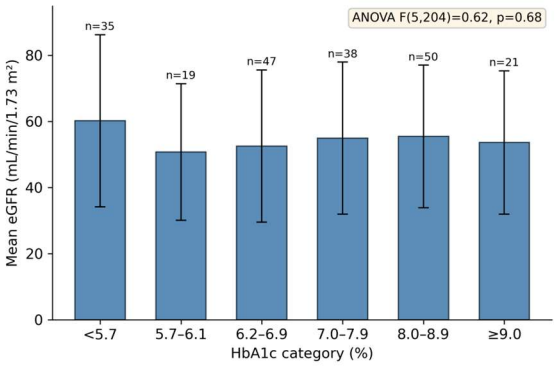

Figure 2. Mean baseline eGFR (± SD) by HbA1c category; differences across groups were not statistically significant.

3.4 Change in renal function at 120 days

Between baseline and 120 days, mean eGFR fell from 54.9 ± 22.8 to 46.7 ± 12.8 mL/min/1.73 m² (mean change −8.2 ± 26.4; paired t = 4.52; p < 0.001). This change was highly heterogeneous: 90 of 210 patients (43%) showed an increase in eGFR, and the interquartile range of change spanned −28.1 to +12.2 mL/min/1.73 m² (Figure 3). The 120-day change in eGFR was not associated with HbA1c on univariable analysis (r = 0.05; p = 0.45), nor after

adjustment for baseline eGFR and age (adjusted β = −0.34 per 1% HbA1c; p = 0.59); baseline eGFR (β = −1.02; p < 0.001) and age (β = −0.37; p < 0.001) were the only significant predictors (model R^2 = 0.79) (Table 5).

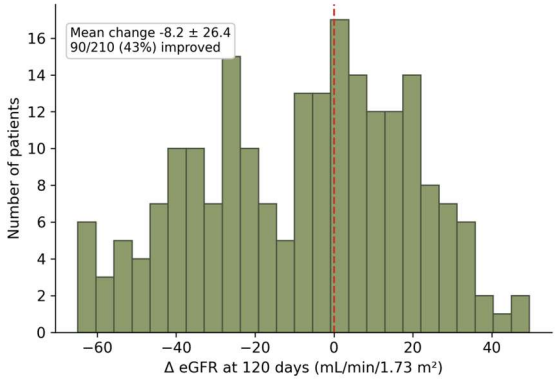

Figure 3. Distribution of 120-day change in eGFR. The wide spread and the proportion of patients improving indicate substantial measurement variability over the short follow-up.

Table 5. Association between HbA1c and renal outcomes.

Analysis	Estimate	95% CI	p
HbA1c vs baseline eGFR (Pearson r)	−0.05	−0.19 to 0.08	0.45
HbA1c category vs eGFR (ANOVA F)	0.62	—	0.68
HbA1c >7% vs eGFR <60 (OR)	0.93	0.54 to 1.60	0.78
HbA1c vs 120-day ΔeGFR (Pearson r)	0.05	—	0.45
HbA1c → 120-day ΔeGFR, adjusted (β)*	−0.34	—	0.59

**Multivariable linear regression adjusting for baseline eGFR and age (model R^2 = 0.79). OR, odds ratio; CI, confidence interval.*

4. Discussion

In this prospective cohort of 210 patients with established diabetic CKD, a single HbA1c measurement was not associated with concurrent renal function or with short-term change in eGFR. Mean eGFR was essentially flat across the full range of HbA1c, and glycaemic category did not predict either reduced eGFR or 120-day functional decline.

This null result is consistent with reports that HbA1c has limited prognostic value once diabetic kidney disease is established, where extensive nephron loss and non-glycaemic mechanisms may blunt the signal from any single glycaemic measurement.[8] It contrasts with the DCCT and UKPDS, which demonstrated benefit of glycaemic control for the incidence of new microvascular disease rather than progression within established CKD.[6,7] Our findings are also compatible with evidence that glycaemic variability, rather than a single HbA1c value, is more closely linked to renal decline;[9] variability was not captured here. Studies using derived glycaemic indices such as the glycation gap have likewise reported associations with renal function that a single HbA1c may not capture.[11] Notably, trial evidence that tighter glucose targets slow diabetic kidney disease is strongest for the prevention of incident disease rather than for progression within established CKD,[12] which is consistent with the pattern observed here.

Several factors may explain the absence of association. The cohort spanned early to advanced CKD, and mixing stages can obscure stage-specific effects. Albuminuria—a more sensitive marker of diabetic kidney injury than eGFR—was not measured. A single baseline HbA1c cannot characterise long-term glycaemic exposure. Finally, the observed 120-day change in eGFR was highly variable, with almost half of patients apparently improving; over such a short interval this most plausibly reflects biological and measurement variability (and regression to the mean, since baseline eGFR was the dominant predictor of change) rather than true disease progression. The short follow-up therefore limits any inference about progression.

Clinically, these data suggest that HbA1c alone is insufficient to stratify renal risk in patients who already have diabetic CKD, and that assessment should incorporate albuminuria, eGFR trajectory over longer periods, and other risk markers. This does not diminish the established value of glycaemic control for preventing incident diabetic kidney disease.

Strengths and limitations. Strengths include the prospective design, a real-world South Indian cohort spanning the CKD spectrum, and complete baseline data. Limitations are important: the single-centre design limits generalisability; albuminuria was not assessed; only one HbA1c value was available; the 120-day follow-up is short and the follow-up eGFR values were highly variable, precluding reliable assessment of progression; and clinical endpoints such as hospitalisation and mortality were not captured, so no survival or event analysis was possible. Residual confounding cannot be excluded.

5. Conclusion

In patients with established diabetic CKD, a single HbA1c measurement was not associated with renal function or with short-term change in eGFR. HbA1c alone appears insufficient for renal risk stratification in this setting. Larger multicentre studies with longer follow-up, serial HbA1c, and albuminuria are needed

to define the prognostic role of glycaemic control in established diabetic kidney disease.

6. Declaration

Patient Consent and Ethics

Patient consent: Written informed consent for publication was obtained from the patient ; a signed copy is available on request.

Ethics: This report adheres to the Declaration of Helsinki (2013 revision). IEC approval: SSSMCRI, Chengalpattu (Ref: IHEC-SSSMCRI/2026/.).

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All authors have reviewed the final version of the manuscript, approved it for submission, and agreed to be accountable for all aspects of the work.

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