

Association of ACR with Serum Creatinine in Diabetic Patients in a Secondary Hospital of Bangladesh

Afroza Azad,¹ KM Istiak Rohan,² Farah Imrana³

1. Associate Professor
Department of Clinical Biochemistry Lab
BIHS General Hospital
Dhaka, Bangladesh
2. Assistant Professor
Department of Endocrinology
Dhaka Medical College Hospital
Dhaka, Bangladesh
3. Associate Professor
Department of Pathology
BIHS General Hospital
Dhaka, Bangladesh

Correspondence to:

Afroza Azad
Associate Professor
Department of Clinical Biochemistry Lab
BIHS General Hospital, Dhaka, Bangladesh
Email: afrozaazad9@gmail.com



Submission Date : 25 Dec 2025
Accepted Date : 05 Feb 2026
Published Date : 30 March 2026
DOI: <https://doi.org/10.3329/jrPMC.v11i1.89951>

Abstract

Background:

Albuminuria and serum creatinine are key markers of diabetic kidney involvement, yet their relationship in routine secondary-care settings in Bangladesh is not well characterized.

Objective:

This study aimed to evaluate the association between albumin-to-creatinine ratio (ACR) and serum creatinine among diabetic patients.

Methods:

This cross-sectional study was conducted among 70 diabetic patients at Bangladesh Institute of Health Sciences (BIHS) General Hospital, Dhaka, from July 2024 to June 2025. Spot urine ACR, serum creatinine, fasting blood sugar, and post-breakfast blood sugar were analyzed using standard cut-offs and appropriate comparative tests.

Results:

Mean ACR was 655.75 ± 1023.99 mg/g, and mean serum creatinine was 1.32 ± 0.49 mg/dL. Moderately increased ACR was observed in 50.0% and severely increased ACR in 40.0%, while 44.3% had elevated creatinine (>1.3 mg/dL). Although the ACR category was not significantly associated with creatinine status ($p=0.067$), serum creatinine rose significantly across ACR categories ($p=0.001$). Glycaemic parameters showed no significant differences by creatinine status.

Conclusion:

Routine ACR screening alongside creatinine may improve early DKD detection in secondary care.

Keywords: Albumin-to-creatinine ratio, Serum creatinine, Diabetic kidney disease, Renal function

Citation: Azad A, Rohan KMI, Imrana F. Association of ACR with Serum Creatinine in Diabetic Patients in a Secondary Hospital of Bangladesh. *J Rang Med Col.* 2026 Mar; 11(1):45-50. doi: <https://doi.org/10.3329/jrPMC.v11i1.89951>

Introduction:

Diabetes mellitus represents a major global public health challenge, with prevalence rising sharply over recent decades, particularly in low and middle-income countries, driven by urbanization, lifestyle transitions, and population aging.¹ South Asia carries a disproportionate share of this burden, and Bangladesh is projected to experience one of the steepest increases in diabetes prevalence. A meta-analysis by Akhtar et al reported a pooled national prevalence of 7.8%, while subsequent studies have shown a wide range, from 4.5% to 35%, likely reflecting

heterogeneity in urbanization, healthcare access, and case detection.^{2,3} Despite improving diagnostic and treatment capacity in metropolitan areas, diabetic complications remain a substantial source of morbidity and mortality, increasing pressure on secondary and tertiary hospitals. Among chronic complications, diabetic kidney disease (DKD) is one of the most consequential, contributing substantially to chronic kidney disease (CKD) and end-stage renal disease (ESRD) worldwide, exceeding one-third of CKD cases and more than half of ESRD in some populations.^{4,5} The burden of DKD has increased over the last three decades, with the steepest growth in

resource-limited settings where structured screening and early intervention are often inconsistent.⁶ In Bangladesh, hospital-based evidence indicates diabetes is now a dominant driver of CKD and ESRD, and many patients present at advanced stages due to limited routine surveillance and restricted access to nephrology services.^{7,8} This late detection worsens renal outcomes and amplifies health-system costs. DKD pathogenesis reflects intertwined metabolic and hemodynamic mechanisms: persistent hyperglycemia promotes non-enzymatic protein glycation, oxidative stress, inflammatory signaling, and activation of the renin angiotensin aldosterone system, progressively disrupting glomerular structure and function.^{9,10} These processes lead to glomerular basement membrane thickening, mesangial expansion, and podocyte loss, increasing permeability to proteins, often remaining clinically silent until measurable biochemical changes appear.¹¹ Urinary albumin excretion is among the earliest detectable abnormalities, and the albumin-to-creatinine ratio (ACR) is the preferred quantitative marker because it corrects for urine dilution and enables reliable assessment from spot samples.¹² Guideline-based categories define ACR as normal to mildly increased (<30 mg/g), moderately increased (30–300 mg/g), and severely increased (>300 mg/g).¹³ Importantly, ACR elevation can precede declines in GFR by years, supporting its value for early risk stratification and treatment adjustment.¹⁴ Serum creatinine, in contrast, is a functional marker that typically rises only after substantial nephron loss, and it is influenced by factors such as muscle mass, diet, and medications, limiting sensitivity for early DKD.¹⁵⁻¹⁷ Clarifying how ACR relates to serum creatinine can illuminate DKD progression, including clinically relevant discordance patterns, and can guide pragmatic screening in secondary hospitals where comprehensive renal profiling may be constrained.^{14,18,19} Therefore, this study aims to investigate the association between albumin-to-creatinine ratio and serum creatinine among diabetic patients attending a secondary hospital in Bangladesh.

Methods:

This cross-sectional observational study was conducted in the Department of Clinical Biochemistry Laboratory, BIHS General Hospital,

Dhaka, Bangladesh, from July 2024 to June 2025. Seventy consecutive adult patients with diabetes mellitus (N=70) attending outpatient or inpatient services during routine care were enrolled to assess the association between urinary albumin-to-creatinine ratio (ACR) and serum creatinine as complementary markers of diabetic renal involvement. Eligible participants were aged ≥ 18 years and had complete laboratory data for spot urinary ACR (mg/g), serum creatinine (mg/dL), fasting blood sugar (FBS, mmol/L), and post-breakfast blood sugar (ABF, mmol/L). Spot urine samples were collected for albumin and creatinine estimation, and fasting venous blood was obtained for biochemical analysis in the hospital's central diagnostic laboratory. Patients were excluded if they had documented non-diabetic kidney disease (e.g., chronic glomerulonephritis, obstructive uropathy, structural renal abnormalities), acute urinary tract infection or hematuria at sampling, pregnancy, acute febrile illness, heart failure, severe dehydration, or other conditions likely to cause transient proteinuria, and if key laboratory variables were missing.

ACR was calculated as urinary albumin (mg) divided by urinary creatinine (g), categorized as <30, 30–300, and >300 mg/g. Serum creatinine was classified as normal (≤ 1.3 mg/dL) or elevated (>1.3 mg/dL). FBS (<7.0 vs ≥ 7.0 mmol/L) and ABF (<10.0 vs ≥ 10.0 mmol/L) were categorized using ADA-aligned thresholds. Data were analyzed in SPSS v26.0. Descriptive statistics were reported as mean \pm SD. Mann–Whitney U, one-way ANOVA, independent t-test, and chi-square tests were applied as appropriate, with $p < 0.05$ considered significant. IRB approval was obtained, and de-identified routine clinical data were analyzed with strict confidentiality.

Results:

Among 70 diabetic participants, the mean age was 56.17 ± 13.17 years (range: 20 to 93). Marked variability was observed in renal and glycemic measures, with mean ACR 655.75 ± 1023.99 mg/g (10 to 5275.7), mean serum creatinine 1.32 ± 0.49 mg/dL (0.5 to 2.86), mean fasting blood sugar 9.78 ± 3.95 mmol/L (3.7 to 24.6), and mean post-breakfast blood sugar 13.11 ± 5.48 mmol/L (6.05 to 35.2) (Table-I).

Table-I: Baseline characteristics among the participants (N=70)

Baseline characteristics	Mean±SD	Range
Age	56.17±13.17	20-93
ACR (mg/gm)	655.75±1023.99	10-5275.7
S. Creatinine (mg/dl)	1.32±0.49	0.5-2.86
Fasting Blood sugar (mmol/L)	9.78±3.95	3.7-24.6
After Breakfast Blood Sugar (mmol/L)	13.11±5.48	6.05-35.2

Half of the patients had moderately increased ACR (30 to 300 mg/g, 50.0%), while 40.0% had severely increased ACR (>300 mg/g); only 10.0% were in the normal or mildly increased category (<30 mg/g) (Table-II).

Serum creatinine was elevated (>1.3 mg/dL) in 44.29% of participants, and glycemic control was poor overall, with 75.71% having uncontrolled fasting glucose (≥ 7.0 mmol/L) and 70.0% having uncontrolled post-breakfast glucose (≥ 10.0 mmol/L) [Table 2]. Across ACR strata, elevated creatinine was more frequent in the severely increased ACR category (54.8%) compared with the moderately increased category (35.5%) and the normal or mildly increased category (9.7%), showing a borderline association ($p=0.067$) (Table-III).

Table-II: Distribution of patients according to variable categories (N=70)

Variable	no. (%)
Albumin-to-Creatinine Ratio Category	
Normal or mildly increased (<30 mg/g)	7(100.00)
Moderately increased (30–300 mg/g)	35(50.00)
Severely increased (>300 mg/g)	28(40.00)
Serum Creatinine Category	
Normal (≤ 1.3 mg/dL)	39(55.71)
Elevated (>1.3 mg/dL)	31(44.29)
Fasting Blood Sugar Category	
Controlled (<7.0 mmol/L)	17(24.29)
Uncontrolled (≥ 7.0 mmol/L)	53(75.71)
Post-breakfast Blood Sugar Category	
Controlled (<10.0 mmol/L)	21(30.00)
Uncontrolled (≥ 10.0 mmol/L)	49(70.00)

Mean serum creatinine differed significantly by ACR category, rising to 1.58 ± 0.53 mg/dL in the severely increased group versus 1.14 ± 0.37 mg/dL and 1.15 ± 0.40 mg/dL in the lower ACR groups (ANOVA $p=0.001$) (Table-IV).

Table-III: Distribution of serum creatinine status across acr categories (N=70)

ACR Category	Normal Creatinine (n=39) no. (%)	Elevated Creatinine (n=31) no. (%)	p-value
Normal / mildly increased (<30 mg/g)	4(10.3)	3(9.7)	0.067
Moderately increased (30–300 mg/g)	24(61.5)	11(35.5)	
Severely increased (>300 mg/g)	11(28.2)	17(54.8)	

Table-IV: Mean serum creatinine across ACR categories (N=70)

Albumin-to-Creatinine Ratio Category	Serum Creatinine (mg/dL) Mean±SD	p-value
Normal or mildly increased (<30 mg/g)	1.14±0.37	0.001
Moderately increased (30–300 mg/g)	1.15±0.40	
Severely increased (>300 mg/g)	1.58±0.53	

Table-V: Comparison of ACR and blood glucose parameters across serum creatinine categories (N=70)

Parameter	Normal Creatinine (n=39) no. (%)	Elevated Creatinine (n=31) no. (%)	p-value
ACR (mg/g)	517.52±833.17	829.67±1215.04	0.112a
Fasting Blood Sugar (mmol/L)	9.90±3.45	9.64±4.57	0.375b
Post-Breakfast Blood Sugar (mmol/L)	13.27±5.14	12.91±5.97	0.786b

a Mann–Whitney U test, b independent sample t-test

However, ACR, fasting glucose, and post-breakfast glucose did not differ significantly between normal and elevated creatinine groups ($p=0.112$, 0.375 , and 0.786 , respectively) (Table-V).

Discussion:

The present study assessed the relationship between urinary albumin-to-creatinine ratio (ACR) and serum creatinine among diabetic patients attending a secondary hospital in Bangladesh, and it demonstrates a substantial burden of renal involvement with wide heterogeneity in disease stage. The markedly elevated mean ACR (655.75 ± 1023.99 mg/g) with an extremely broad range indicates that many patients had clinically meaningful albuminuria, spanning from early microvascular injury to advanced nephropathy, a pattern consistent with the continuum model of diabetic kidney disease (DKD) described in prior work.^{7,20} In parallel, the mean serum creatinine of 1.32 ± 0.49 mg/dL, with nearly half of the cohort classified as having elevated creatinine, suggests that a considerable proportion were already experiencing functional decline, which is plausible in secondary-care settings where patients frequently present later in the disease course.²¹ The distribution across albuminuria categories further reinforces this interpretation: 50.0% had moderately increased ACR and 40.0% had severely increased ACR, aligning with regional reports indicating a high prevalence of albuminuria among diabetic patients in South Asia and Bangladesh.⁷ Importantly, severely increased albuminuria was more common among those with elevated creatinine than among those with normal creatinine, whereas most patients with moderately increased albuminuria still had creatinine within the reference range. This pattern supports the established concept that albuminuria is often detectable earlier than creatinine elevation, with creatinine rising later after substantial nephron loss, and it mirrors observations that ACR and creatinine capture different but overlapping dimensions of DKD progression.²⁰

Although the categorical association between ACR strata and creatinine status did not reach conventional statistical significance ($p=0.067$), the directional trend remained clinically coherent and comparable to prior reports noting only modest concordance between structural markers (albuminuria) and functional markers (creatinine) in diabetic nephropathy, particularly in

heterogeneous clinical populations.²¹ More compellingly, mean serum creatinine increased significantly across escalating ACR categories, with the highest creatinine observed in the severely increased ACR group ($p=0.001$). This dose-response pattern is consistent with evidence that higher albuminuria is associated with subsequent renal functional deterioration and worsening filtration indices in diabetes.^{22,23} Taken together, the results support a biologically plausible linkage between worsening albuminuria and declining renal function, while also underscoring that these markers may not progress in parallel for every individual, which has been described in longitudinal studies where albuminuria may fluctuate, regress, or dissociate from measured renal function trajectories.^{20,21} Glycaemic indices, including fasting and post-breakfast glucose, did not differ significantly between creatinine groups, suggesting that short-term glycaemic status alone did not explain the observed renal impairment in this cohort. This aligns with literature emphasizing that DKD progression reflects multiple drivers beyond glucose, including hemodynamic factors, oxidative stress, endothelial dysfunction, and coexisting cardiometabolic risk.²³ Overall, the study provides locally relevant evidence supporting the combined interpretation of ACR and serum creatinine for DKD detection and staging in Bangladeshi secondary-care practice, and it highlights the need for routine ACR screening alongside creatinine estimation to enable earlier identification of high-risk patients and timely risk-reduction interventions.^{7,20-23}

Limitations:

This study was conducted at a single secondary-care hospital with a relatively small sample size, which may limit generalizability to broader diabetic populations. The cross-sectional design also restricts the ability to infer causality or assess long-term renal progression in relation to albuminuria and creatinine dynamics.

Conclusion:

This study shows a high burden of albuminuria among diabetic patients in a Bangladeshi secondary hospital, with serum creatinine increasing significantly as ACR worsened, indicating progressive renal involvement. ACR and creatinine provided complementary information for DKD detection and risk stratification,

supporting routine screening even in resource-limited settings. Routine ACR testing should be incorporated into standard diabetic care at secondary hospitals to enable early detection of renal involvement. Patients with moderately or severely increased ACR should receive closer follow-up, even when serum creatinine is normal. Larger multicentre longitudinal studies, including eGFR and HbA1c, are recommended to strengthen risk prediction and clarify progression patterns of diabetic kidney disease.

References:

1. GBD 2021 Diabetes Collaborators. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. *Lancet*. 2023 Jul 15;402(10397):203-234. doi: 10.1016/S 0140-6736(23)01301-6.
2. Akhtar S, Nasir JA, Sarwar A, Nasr N, Javed A, Majeed R, et al. Prevalence of diabetes and pre-diabetes in Bangladesh: a systematic review and meta-analysis. *BMJ Open*. 2020 Sep 9;10(9):e036086. doi: 10.1136/bmjopen-2019-036086.
3. Chowdhury MAB, Islam M, Rahman J, Uddin MJ, Haque MR. Diabetes among adults in Bangladesh: changes in prevalence and risk factors between two cross-sectional surveys. *BMJ Open*. 2022 Aug 4;12(8):e055044. doi: 10.1136/bmjopen-2021-055044.
4. Ma X, Liu R, Xi X, Zhuo H, Gu Y. Global burden of chronic kidney disease due to diabetes mellitus, 1990-2021, and projections to 2050. *Front Endocrinol (Lausanne)*. 2025 Feb 21;16:1513008. doi: 10.3389/fendo. 2025.1513008.
5. Liu WY, Chen WY, Zhang JH, Targher G, Byrne CD, Misra A, et al. The global burden of diabetes-related chronic kidney disease from 1990 to 2021, with projections to 2036. doi:10.20517/mtod.2025.42
6. Hoogeveen EK. The Epidemiology of Diabetic Kidney Disease. *Kidney Dial*. 2022;2(3):433-44. doi:https://doi.org/10.3390/kidneydial2030038
7. Islam SMS, Salehin M, Zaman SB, Tansi T, Gupta RD, Barua L, Banik PC, Uddin R. Factors Associated with Chronic Kidney Disease in Patients with Type 2 Diabetes in Bangladesh. *Int J Environ Res Public Health*. 2021 Nov 23;18(23):12277. doi: 10.3390/ijerph182312277.
8. Mostafi M, Jabin M. Renal Disease in Bangladesh Perspective. *Bangla J Med*. 2023 Sep;34(3):180-185. doi:https://banglajol.info/index.php/ bjmed/ article/ view/68418
9. Poloni JAT, Rotta LN. Diabetic kidney disease: pathophysiological changes and urinalysis contribution to diagnosis—a narrative review. *J Lab Precis Med*.2022;7(3).doi: 10.21037/ jlp-21-20
10. Doumani G, Theofilis P, Vordoni A, Thymis V, Liapis G, Smirloglou D, et al. Diabetic Kidney Disease: From Pathophysiology to Regression of Albuminuria and Kidney Damage: Is It Possible? *Int J Mol Sci*. 2025 Aug 24;26(17):8224. doi: 10.3390/ijms 26178224.
11. Qazi M, Sawaf H, Ismail J, Qazi H, Vachharajani T. Pathophysiology of Diabetic Kidney Disease. *EMJ*. 2022 June 30;10(1): 102-113. doi:https://doi.org/ 10.33590/emjnephrol/22-00060
12. Christofides EA, Desai N. Optimal Early Diagnosis and Monitoring of Diabetic Kidney Disease in Type 2 Diabetes Mellitus: Addressing the Barriers to Albuminuria Testing. *J Prim Care Community Health*. 2021 Jan-Dec;12:21501327211003683. doi: 10.1177/21501327211003683.
13. Rūsimont G, Cavalier E, Radermecker RP, Delanaye P. Albuminuria in diabetic patients: how to measure it?—a narrative review. *J Lab Precis Med*. 2022 Jan 30;7(0). doi: 10.21037/ jlp-21-58
14. Selby NM, Taal MW. An updated overview of diabetic nephropathy: Diagnosis, prognosis, treatment goals and latest guidelines. *Diabetes Obes Metab*. 2020 Apr;22 Suppl 1:3-15. doi: 10.1111/dom.14007.
15. Gounden V, Bhatt H, Jialal I. Renal Function Tests. 2024 Jul 27. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. PMID: 29939598.
16. Bvila M, Mora S6nchez MG, Bernal Amador AS, Paniagua R. The Metabolism of Creatinine and Its Usefulness to Evaluate Kidney Function and Body Composition in Clinical Practice. *Biomolecules*. 2025 Jan 1;15(1):41. doi: 10.3390/biom15010041.
17. Bargnoux AS, Kuster N, Cavalier E, Pi6roni L, Souweine JS, Delanaye P, et al. Serum

- creatinine: advantages and pitfalls. *J Lab Precis Med.* 2018 Aug;3(0). doi: 10.21037/jlpm. 2018.08.01
18. Romero-Aroca P, Baget-Bernaldiz M, Navarro-Gil R, Moreno-Ribas A, Valls-Mateu A, Sagarra-Alamo R, et al. Glomerular Filtration Rate and/or Ratio of Urine Albumin to Creatinine as Markers for Diabetic Retinopathy: A Ten-Year Follow-Up Study. *J Diabetes Res.* 2018 Feb 26;2018:5637130. doi: 10.1155/2018/5637130.
 19. Zhou L, Gao Y, Li M, Cai X, Zhu Y, Han X, Ji L. Baseline Urine Albumin-to-Creatinine Ratio is Associated With Decline of Estimated Glomerular Filtration Rate in Patients Newly Diagnosed With Type 2 Diabetes Mellitus: An Observational 5-year Cohort Study. *Endocr Pract.* 2024 Feb;30(2):107-112. doi: 10.1016/j.eprac.2023.10.136.
 20. Norris KC, Smoyer KE, Rolland C, Van der Vaart J, Grubb EB. Albuminuria, serum creatinine, and estimated glomerular filtration rate as predictors of cardio-renal outcomes in patients with type 2 diabetes mellitus and kidney disease: a systematic literature review. *BMC Nephrol.* 2018 Feb 9;19(1):36. doi: 10.1186/s12882-018-0821-9.
 21. Sanyal M, Hossain MZ, Khan MAU, Rahman F, Ali MH. Correlation Between Serum Creatinine & Urine Albumin in Diabetic Nephropathy Patients. *J Dhaka Med Coll.* 2018; 27(2) : 141-147. doi:https://doi.org/10.3329/jdmc.v27i2.45826
 22. Lee JI, Kwon HS, Oh SJ, Lee JM, Chang SA, Cha BY, et al. Association of Spot Urine Albumin-to-Creatinine Ratio and 24 Hour-Collected Urine Albumin Excretion Rate in Patients with Type 2 Diabetes Mellitus. *Korean Diabetes J* 33:299~305, 2009. doi: 10.4093/kdj.2009.33.4.299
 23. Tangri N, Singh R, Chen Y, Betts KA, Farag YM, Beeman Set al. Change in urine albumin-to-creatinine ratio and clinical outcomes in patients with chronic kidney disease and type 2 diabetes. *BMJ Open Diabetes Res Care.* 2025 Oct 15;13(5):e004854. doi: 10.1136/bmjdr-2024-004854.