

Original Research

Received 07 June 2026

Revised 22 July 2026

Accepted 05 Aug 2026

Natural Resources for Human Health 2026, Vol. 6 (15s): 590–599

KEYWORDS:

contrast-induced nephropathy, type 2 diabetes mellitus, glycated hemoglobin, admission glucose, hyperglycemia, coronary angiography

Associations of Acute and Chronic Glycemic Measures with Contrast-Induced Nephropathy in Type 2 Diabetes Mellitus Undergoing Coronary Angiography

Andi Rahmat Hidayat^{1*}, Andi Makbul Aman¹, Haerani Rasyid², Syakib Bakri², Idar Mappangara³, Husaini Umar¹, Himawan Sanusi¹, Fabiola Maureen Shinta Adam¹, Khadijah Khairunnisa Hasyim², Achmad Fikry², Ilham Jaya Patellongi⁴

¹Division of Endocrine, Metabolic and Diabetes, Department of Internal Medicine, Faculty of Medicine, Hasanuddin University, Makassar 90245, Indonesia

²Division of Nephrology and Hypertension, Department of Internal Medicine, Faculty of Medicine, Hasanuddin University, Makassar 90245, Indonesia

³Division of Cardiology, Department of Internal Medicine, Faculty of Medicine, Hasanuddin University, Makassar 90245, Indonesia

⁴Department of Biostatistics, Faculty of Medicine, Hasanuddin University, Makassar 90245, Indonesia

ABSTRACT: Contrast-induced nephropathy (CIN) remains an important complication of coronary angiography, particularly in patients with type 2 diabetes mellitus (T2DM), yet the relative associations of chronic and acute glycemic measures with CIN remain incompletely understood. The present study compared the associations of glycated hemoglobin (HbA1c), admission blood glucose and pre-procedural blood glucose with CIN in patients with T2DM undergoing coronary angiography. A total of 200 participants were included, of whom 98 (49.0%) met the CIN definition. Higher HbA1c, admission glucose and pre-procedural glucose were observed in participants with CIN. In mutually adjusted logistic regression models adjusting for cardiorenal and procedural covariates, HbA1c remained associated with CIN per 1% increase (odds ratio [OR], 1.338; 95% confidence interval [CI], 1.023–1.750; $P=0.034$), as did admission glucose per 50 mg/dl increase (OR, 1.750; 95% CI, 1.113–2.753; $P=0.015$), whereas the linear association for pre-procedural glucose was attenuated (OR, 1.175; 95% CI, 0.772–1.788; $P=0.453$). Restricted cubic spline analysis identified significant nonlinear components for HbA1c and pre-procedural glucose. In the final flexible model, likelihood-ratio testing demonstrated independent global contributions from HbA1c ($P<0.001$), admission glucose ($P=0.008$) and pre-procedural glucose ($P=0.029$). These findings indicate that chronic and acute glycemic measures provide complementary information regarding CIN, with distinct exposure-response patterns that may be obscured by conventional linear modeling.

1. INTRODUCTION

Contrast-induced nephropathy (CIN), often discussed within the broader framework of contrast-associated acute kidney injury (CA-AKI), remains a clinically relevant complication after coronary angiography. A meta-analysis of 134 studies involving more than 1.2 million patients reported a pooled CA-AKI incidence of 12.8% [1]. Contemporary reviews continue to emphasize risk

stratification and prevention, particularly in patients with diabetes and chronic kidney disease, in whom susceptibility to post-contrast renal injury is increased [2,3].

Glycemic markers capture different time domains. HbA1c reflects average glycemia over approximately 2–3 months, whereas admission glucose is more responsive to acute metabolic and stress-related changes [4,5]. In patients with diabetes undergoing coronary angiography or PCI, higher HbA1c was independently associated with CIN [6]. Separately, higher preoperative fasting glucose was associated with increased CIN risk among patients with diabetes or prediabetes [7].

More recent studies have examined relative hyperglycemia using the stress hyperglycemia ratio (SHR), which integrates acute glucose with background HbA1c. SHR has been associated with AKI in patients with diabetes and acute myocardial infarction [8], while studies in broader coronary angiography or PCI populations have reported nonlinear associations between SHR and CI-AKI or CIN [9–11]. However, these studies largely evaluated individual markers or composite ratios rather than directly comparing HbA1c, admission glucose and pre-procedural glucose in established type 2 diabetes. The present study therefore compared these three glycemic measures and examined their independent associations with CIN while allowing for potential nonlinear exposure-response relationships.

2. MATERIALS AND METHODS

2.1 Study design and participants

The present single-center observational study was conducted at Dr. Wahidin Sudirohusodo Central General Hospital, Makassar, Indonesia. Consecutive inpatients aged ≥ 18 years with type 2 diabetes mellitus (T2DM) undergoing coronary angiography were recruited beginning in May 2026 and screened for eligibility.

T2DM was defined by a previous clinical diagnosis, current glucose-lowering treatment, or established diagnostic criteria for diabetes [12]. Exclusion criteria were intravascular contrast exposure or nephrotoxic medication within the preceding 2 weeks, severe hemodynamic instability or shock, malignancy, severe infection or sepsis, and conditions interfering with HbA1c interpretation, including altered erythrocyte turnover, hemoglobinopathies, recent blood transfusion or pregnancy.

Based on an anticipated CIN proportion of 20% [13], a 95% confidence level and 10% absolute precision, the minimum required sample size was 62 participants. During the study period, 200 consecutive eligible participants were enrolled and included in the analysis.

2.2 Clinical and procedural assessment

Baseline data included age, sex, diabetes duration, hypertension, body mass index (BMI), hemoglobin concentration, renal function and left ventricular ejection fraction (LVEF). BMI was calculated as weight divided by height squared (kg/m^2). Hypertension was defined as systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg, a previous diagnosis, or current antihypertensive treatment [14]. Blood pressure was measured after ≥ 5 min of seated rest, with two measurements obtained 1–2 min apart and averaged.

Diabetes duration was calculated from first diagnosis to the index admission. LVEF was obtained by echocardiography. Coronary angiography was performed using intra-arterial iodinated contrast media, and total contrast volume was obtained from the procedural record and analyzed continuously.

2.3 Assessment of glycemic exposure

HbA1c, admission blood glucose and pre-procedural blood glucose were evaluated as continuous glycemic measures. Admission glucose was random venous blood glucose measured at hospital presentation, whereas pre-procedural glucose was measured from venous blood within ≤ 4 h before coronary angiography; both were expressed in mg/dl.

HbA1c was measured in EDTA-anticoagulated whole blood using an enzymatic method on an ARCHITECT c4000 analyzer (Abbott Diagnostics; reagent no. 69353UQ11) and expressed as a percentage. Admission and pre-procedural blood glucose were measured using the glucose oxidase-peroxidase (GOD-PAP) method on a Randox Imola clinical chemistry analyzer (Randox Laboratories Ltd.). HbA1c reflects glycemic exposure over approximately the preceding 2–3 months [4,12]. All three glycemic measures were retained as continuous variables without data-derived categorization.

2.4 Renal assessment and CIN definition

Serum creatinine was measured using the Jaffe method on a Randox Imola clinical chemistry analyzer (Randox Laboratories Ltd.; reagent no. CR 3814) before contrast exposure and repeated 48–72 h after coronary angiography. Estimated glomerular filtration rate (eGFR) was calculated using the 2021 CKD-EPI creatinine equation and expressed in ml/min/1.73 m² [15]. Urinary albumin-to-creatinine ratio (uACR) was assessed from a spot urine sample using the reflective photometry method on a Sysmex UC-3500 analyzer (Sysmex Corporation; reagent no. AC 5094) and categorized as <30, 30–300 and >300 mg/g, corresponding to the A1, A2 and A3 albuminuria categories, respectively [16].

The primary outcome was contrast-induced nephropathy (CIN). Serum creatinine was repeated 48–72 h after coronary angiography. CIN was defined as an absolute increase in serum creatinine of ≥ 0.5 mg/dl or a relative increase of $\geq 25\%$ from baseline within 48–72 h after contrast exposure, in the absence of another evident cause of acute renal dysfunction [13,17].

2.5 Data management and statistical analysis

Data were entered into a coded electronic database and checked for completeness, range and internal consistency. No observations were missing for variables included in the primary analyses; therefore, no imputation was performed.

Continuous-variable distributions were assessed using the Shapiro-Wilk test and visual inspection. Approximately normally distributed variables are presented as mean $\pm$ standard deviation (SD) and compared using independent-samples t-tests; non-normally distributed variables are presented as median and interquartile range and compared using Mann-Whitney U tests. Categorical variables are presented as frequencies and percentages and compared using Pearson's χ^2 test.

Binary logistic regression was used to examine associations between glycemic measures and CIN. HbA1c was modeled per 1-percentage-point increase and admission and pre-procedural glucose per 50-mg/dl increase. Multivariable models adjusted for age, sex, diabetes duration, hypertension, baseline eGFR, uACR category, LVEF and total contrast volume. Baseline creatinine was not entered simultaneously with eGFR because eGFR was derived from serum creatinine and inclusion of both variables would introduce redundancy. Each glycemic measure was first evaluated separately and subsequently in a mutually adjusted model containing all three exposures. Covariates were entered simultaneously. Standardized ORs per 1-SD increase were also calculated, and collinearity was assessed using tolerance and variance inflation factors.

Linearity of each glycemic exposure with the log odds of CIN was assessed using Box-Tidwell transformation terms, with a Bonferroni-adjusted threshold of $P < 0.017$ for three tests. Exposures showing evidence of nonlinearity were modeled using restricted cubic splines with three knots at the 10th, 50th and 90th percentiles; other exposures were retained as linear terms. The median exposure value was used as the graphical reference, with adjusted ORs displayed on a logarithmic scale [18].

The final flexible model included all three glycemic exposures and the same clinical covariates. Global contributions of individual glycemic measures were assessed using likelihood-ratio tests comparing the full model with nested models excluding all terms for the corresponding exposure. For spline-modeled exposures, linear and nonlinear components were tested jointly. Model fit was assessed using the Hosmer-Lemeshow test and Nagelkerke's pseudo-R². All tests were two-sided, with $P < 0.05$ considered statistically significant except for the prespecified nonlinearity tests. Analyses were performed using IBM SPSS Statistics for Mac, version 30.0 (IBM Corp., Armonk, NY, USA).

3. RESULTS AND DISCUSSION

3.1 Participant characteristics

A total of 200 participants were analyzed, of whom 98 (49.0%) met the CIN criterion and 102 (51.0%) did not. All variables included in the primary analyses were complete (Table I).

Table I. Clinical and laboratory characteristics according to CIN status.

Characteristic	Overall (n=200)	Non-CIN (n=102)	CIN (n=98)	P-value
Age, years	57.36 $\pm$ 9.30	57.25 $\pm$ 9.17	57.47 $\pm$ 9.48	0.871
Male sex, n (%)	139 (69.5)	71 (69.6)	68 (69.4)	0.973
BMI, kg/m ²	25.34 (22.73–27.54)	25.49 (23.20–27.39)	24.73 (22.26–27.94)	0.404
Duration of diabetes, years	3.00 (0.52–7.75)	3.00 (1.00–5.00)	3.50 (0.06–10.00)	0.378

Hypertension, n (%)	134 (67.0)	61 (59.8)	73 (74.5)	0.027*
Hemoglobin, g/dl	13.41±1.83	13.44±1.76	13.37±1.92	0.774
HbA1c, %	7.90 (7.00–9.65)	7.55 (7.00–8.33)	8.95 (7.08–10.70)	<0.001*
Admission blood glucose, mg/dl	183.5 (146.0–234.8)	164.5 (137.8–206.5)	206.5 (160.5–284.5)	<0.001*
Pre-procedural blood glucose, mg/dl	156.5 (124.3–208.0)	146.5 (122.5–183.0)	179.0 (128.8–262.8)	<0.001*
Baseline creatinine, mg/dl	1.08 (0.80–1.28)	1.14 (1.01–1.33)	0.88 (0.66–1.25)	<0.001*
Baseline eGFR, ml/min/1.73 m ²	76.61 (59.32–97.78)	69.26 (56.11–81.11)	94.91 (64.54–107.13)	<0.001*
uACR category, n (%)				<0.001*
<30 mg/g	61 (30.5)	43 (42.2)	18 (18.4)	
30–300 mg/g	73 (36.5)	41 (40.2)	32 (32.7)	
>300 mg/g	66 (33.0)	18 (17.6)	48 (49.0)	
LVEF, %	45.33±8.94	47.68±8.43	42.88±8.84	<0.001*
Contrast volume, ml	150 (80–200)	140 (50–200)	150 (100–242.5)	0.007*

Data are presented as mean ± standard deviation, median (interquartile range), or n (%), as appropriate. P-values were calculated using the independent-samples t-test, Mann-Whitney U test or Pearson's χ^2 test, as appropriate. *P<0.05. BMI, body mass index; CIN, contrast-induced nephropathy; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; uACR, urinary albumin-to-creatinine ratio.

Compared with participants without CIN, those with CIN had higher HbA1c [8.95% (IQR, 7.08–10.70%) vs. 7.55% (IQR, 7.00–8.33%)], admission glucose [206.5 (160.5–284.5) vs. 164.5 (137.8–206.5) mg/dl] and pre-procedural glucose [179.0 (128.8–262.8) vs. 146.5 (122.5–183.0) mg/dl] (all P<0.001; Fig. 1). Hypertension (74.5 vs. 59.8%; P=0.027), severe albuminuria (uACR >300 mg/g; 49.0 vs. 17.6%; P<0.001) and contrast volume [150 (100–242.5) vs. 140 (50–200) ml; P=0.007] were also higher in the CIN group, whereas LVEF was lower (42.88±8.84 vs. 47.68±8.43%; P<0.001). Baseline creatinine was lower and eGFR higher in participants with CIN (both P<0.001). Age, sex, BMI, diabetes duration and hemoglobin concentration did not differ significantly between groups.

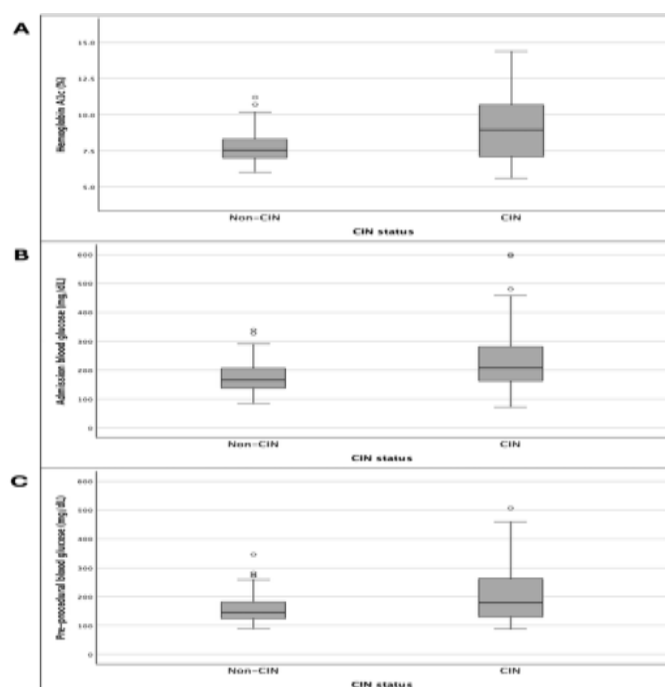


Figure 1. Distribution of glycemic measures according to CIN status. (A) Hemoglobin A1c. (B) Admission blood glucose. (C) Pre-procedural blood glucose.

3.2 Associations between glycemic measures and CIN

All three glycemic measures were associated with CIN in univariable and separately adjusted models (Table II). In the mutually adjusted model, HbA1c remained independently associated with CIN (OR per 1% increase, 1.338; 95% CI, 1.023–1.750; $P=0.034$), as did admission glucose (OR per 50-mg/dl increase, 1.750; 95% CI, 1.113–2.753; $P=0.015$). The linear association for pre-procedural glucose was attenuated (OR, 1.175; 95% CI, 0.772–1.788; $P=0.453$).

On a standardized scale, admission glucose showed the largest effect estimate (OR per 1-SD increase, 2.582; 95% CI, 1.198–5.562; $P=0.015$), followed by HbA1c (OR, 1.720; 95% CI, 1.043–2.837; $P=0.034$). The maximum variance inflation factor was 2.208, indicating no evidence of problematic multicollinearity.

Table II. Associations between glycemic measures and CIN.

Glycemic measure	Unadjusted OR (95% CI)	P-value	Adjusted OR (95% CI) ^a	P-value	Mutually adjusted OR (95% CI) ^b	P-value	Standardized OR (95% CI) ^c
HbA1c, per 1% increase	1.565 (1.299–1.886)	<0.001*	1.692 (1.308–2.188)	<0.001*	1.338 (1.023–1.750)	0.034*	1.720 (1.043–2.837)
Admission glucose, per 50 mg/dl increase	1.760 (1.387–2.234)	<0.001*	2.248 (1.616–3.128)	<0.001*	1.750 (1.113–2.753)	0.015*	2.582 (1.198–5.562)
Pre-procedural glucose, per 50 mg/dl increase	1.629 (1.285–2.064)	<0.001*	1.948 (1.438–2.640)	<0.001*	1.175 (0.772–1.788)	0.453	1.270 (0.681–2.369)

^aEach glycemic measure was modeled separately and adjusted for age, sex, duration of diabetes, hypertension, baseline eGFR, uACR category, LVEF and contrast volume. ^bAll three glycemic measures were entered simultaneously with the same covariates.

^cStandardized ORs represent a 1-SD increase: HbA1c, 1.86%; admission glucose, 84.72 mg/dl; and pre-procedural glucose, 74.23 mg/dl. P-values for the standardized estimates are identical to those of the corresponding mutually adjusted coefficients because standardization changes the scale but not the statistical test. * $P<0.05$. CI, confidence interval; CIN, contrast-induced nephropathy; OR, odds ratio; SD, standard deviation.

Nonlinear associations and global contribution of glycemic measures

Evidence of nonlinearity was observed for HbA1c ($P<0.001$) and pre-procedural glucose ($P=0.006$), but not admission glucose ($P=0.052$). HbA1c and pre-procedural glucose were therefore modeled using restricted cubic splines with knots at the 10th, 50th and 90th percentiles: 6.5, 7.9 and 11.2% for HbA1c and 108, 156.5 and 282 mg/dl for pre-procedural glucose. Nonlinear terms significantly improved model fit for both HbA1c ($\chi^2=19.575$; $P<0.001$) and pre-procedural glucose ($\chi^2=10.444$; $P=0.001$). Adjusted curves showed increasing estimated odds across the upper ranges of both exposures, with wider confidence intervals at the extremes (Fig. 2).

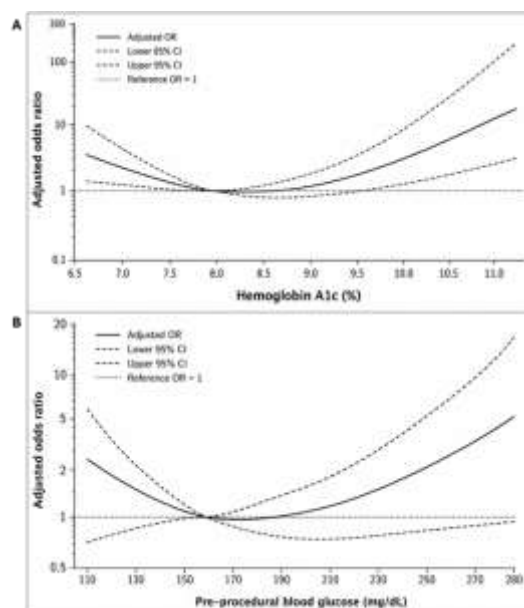


Figure 2. Adjusted nonlinear associations of glycemic measures with CIN. (A) Association between HbA1c and CIN, with 7.9% used as the reference value. (B) Association between pre-procedural blood glucose and CIN, with 156.5 mg/dl used as the reference value.

In the final flexible model, admission glucose remained independently associated with CIN (OR per 50-mg/dl increase, 1.995; 95% CI, 1.182–3.365; $P=0.010$). The final flexible model yielded a Nagelkerke R^2 of 0.709 and a Hosmer-Lemeshow $P=0.124$. Global likelihood-ratio tests showed independent contributions from HbA1c ($\chi^2=18.833$; $df=2$; $P<0.001$), admission glucose ($\chi^2=7.141$; $df=1$; $P=0.008$) and pre-procedural glucose ($\chi^2=7.089$; $df=2$; $P=0.029$) (Table III).

Table III. Global contribution of glycemic measures in the flexible multivariable model.

Glycemic component removed	Reduced-model –2 log likelihood	LR χ^2 vs. full model	df	P-value
HbA1c	144.240	18.833	2	<0.001*
Admission blood glucose	132.548	7.141	1	0.008*
Pre-procedural blood glucose	132.496	7.089	2	0.029*

The –2 log likelihood of the full flexible model was 125.407. HbA1c and pre-procedural blood glucose were represented by linear and restricted cubic spline terms; therefore, their global contributions were evaluated using 2 degrees of freedom. Admission blood glucose was modeled linearly and evaluated using 1 degree of freedom. The full model additionally included age, sex, duration of diabetes, hypertension, baseline eGFR, uACR category, LVEF and contrast volume. * $P<0.05$. LR, likelihood ratio.

3.5 Discussion

The present study directly compared chronic and acute glycemic measures in patients with type 2 diabetes mellitus undergoing coronary angiography. CIN was identified in 98 of 200 participants (49.0%). In mutually adjusted linear models, HbA1c and admission glucose remained independently associated with CIN, whereas the linear coefficient for pre-procedural glucose was attenuated. Flexible modeling refined this interpretation: HbA1c and pre-procedural glucose showed significant nonlinear components, and global likelihood-ratio tests supported independent contributions from all three glycemic measures. A meta-analysis of >1.2 million patients reported a pooled CA-AKI incidence of 12.8%, while rates in selected high-risk coronary populations can exceed 50% depending on clinical profile and the definition applied [1,19]. Diabetes is associated with CA-AKI, particularly when chronic kidney disease coexists [20].

The HbA1c finding is consistent with Zhang et al. (2021), who reported an independent association between higher HbA1c and CIN among 670 patients with diabetes undergoing coronary arteriography or PCI [6]. Our analysis extends that observation by retaining HbA1c as a continuous exposure and allowing a nonlinear association. Chronic hyperglycemia is linked to mitochondrial oxidative stress, inflammation, fibrosis, and altered glomerular autoregulation in diabetic kidney disease [21]. These abnormalities may increase renal vulnerability to a peri-contrast insult involving vasoconstriction, medullary hypoxia, oxidative stress, inflammation, and tubular toxicity [22].

Admission glucose showed a different pattern: its association was approximately linear and persisted after simultaneous adjustment for the other glycemic measures and cardiorenal covariates. Higher preoperative fasting glucose has been associated with CIN in patients with diabetes or prediabetes [7], and relative stress hyperglycemia has been associated with AKI in patients with diabetes and acute myocardial infarction [8]. Acute hyperglycemia is shaped by neurohormonal and inflammatory stress responses and therefore represents a physiologic domain distinct from HbA1c [5]. These observations do not establish that short-term glucose lowering prevents CIN, but support admission glucose as complementary peri-procedural risk information.

The pre-procedural glucose result illustrates why functional form matters. A conventional mutually adjusted linear coefficient was not significant, yet pre-procedural glucose remained significant when its linear and spline components were tested jointly. Similar complexity has been reported for the stress hyperglycemia ratio (SHR). Shan et al. (2023) found a J-shaped association between fasting SHR and CI-AKI after coronary angiography or PCI [9], Li et al. (2024) reported a reverse J-shaped SHR-CA-AKI association in a large coronary angiography cohort [10], and Zhao et al. (2025) observed a J-shaped relationship in acute coronary syndrome patients undergoing PCI [11]. Although these studies evaluated relative hyperglycemia rather than raw pre-procedural glucose, they similarly suggest that glycemia-related renal risk may not be fully characterized by a strictly linear specification.

Several nonglycemic characteristics also differed according to CIN status. Severe albuminuria was more frequent and LVEF lower in participants with CIN. Microalbuminuria has been associated with post-angiography CIN in acute coronary syndrome [23], while lower ejection fraction and greater heart-failure severity have been associated with CI-AKI after PCI [24]. PCI data also identify diabetes, chronic kidney disease, heart failure, hypertension, and hemodynamic or procedural factors as correlates of CA-AKI [25]. Notably, participants with CIN had lower baseline creatinine and higher eGFR. Because the endpoint incorporated a $\geq 25\%$ relative creatinine increase, baseline creatinine may partly influence the probability of meeting this criterion; this finding should not be interpreted as evidence that higher eGFR increases CIN susceptibility. The excess of severe albuminuria also emphasizes that eGFR and albuminuria capture different dimensions of kidney risk [16]. Prevention remains multifactorial, including appropriate volume management, avoidance of unnecessary nephrotoxins, and minimization of contrast exposure [3].

Strengths include evaluation of three glycemic time domains in the same T2DM cohort, preservation of continuous information, and explicit assessment of nonlinearity. Limitations include the single-center observational design, limited precision at the extremes of the spline curves, and reliance on serum creatinine as the renal endpoint. Admission and pre-procedural glucose were single measurements susceptible to acute illness and differences in fasting status; pre-procedural glucose may additionally have been influenced by glucose-lowering treatment administered after admission. The findings should be interpreted as associations rather than causal effects. Among the glycemic measures, HbA1c showed the strongest global contribution to model fit; admission glucose retained an approximately linear association, whereas pre-procedural glucose contributed through a nonlinear relationship. These findings support evaluating the functional form of continuous glycemic exposures when examining their associations with CIN [18].

4. CONCLUSION

In patients with T2DM undergoing coronary angiography, chronic and acute glycemic measures were independently associated with CIN, although their exposure-response patterns differed. HbA1c and pre-procedural glucose showed significant nonlinear associations, whereas admission glucose demonstrated an approximately linear relationship and remained independently associated with CIN after adjustment for other glycemic, cardiorenal, and procedural factors. These findings suggest that HbA1c, admission glucose, and pre-procedural glucose provide complementary information regarding CIN risk and that conventional linear modeling may not fully capture the associations of glycemic measures with renal outcomes. Given the

observational, single-center design, these results should be interpreted as associations rather than causal effects and warrant validation in larger prospective multicenter studies.

ACKNOWLEDGEMENTS

The authors thank the study participants and the clinical, laboratory, and cardiac catheterization staff at Dr. Wahidin Sudirohusodo Central General Hospital for their support during participant recruitment, clinical assessment, laboratory testing, follow-up, and data collection.

AUTHOR CONTRIBUTIONS

ARH contributed to the conceptualization and methodology of the study, participant recruitment and data collection, data curation, formal analysis, visualization, project administration, and preparation of the original manuscript draft. AMA, HR and SB contributed to study conceptualization, methodology, supervision, validation, interpretation of the findings, and critical revision of the manuscript. IM, HU, HS, FMSA, KKH and AF contributed to methodological evaluation, validation, interpretation of the findings, and critical revision of the manuscript for important intellectual content. IJP contributed to statistical methodology, formal analysis, validation, interpretation of the results, visualization, and critical revision of the manuscript. All authors contributed to the final interpretation of the findings, read and approved the final version of the manuscript, and agreed to be accountable for all aspects of the work.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

This research received no external funding.

ETHICS STATEMENT

The study was approved by the Research Ethics Committee of the Faculty of Medicine, Hasanuddin University (approval no. 606/UN4.6.4.5.31/PP36/2026) and was conducted in accordance with the principles of the Declaration of Helsinki. Permission to conduct the study was obtained from the Clinical Research Unit of Dr. Wahidin Sudirohusodo Central General Hospital (no. DP.04.03/D.XVIII.13/211/2026). Written informed consent was obtained from all participants prior to enrollment.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional restrictions.

REFERENCES

- [1] Lun, Z., Liu, L., Chen, G., Ying, M., Liu, J., Wang, B., et al., 2021. The global incidence and mortality of contrast-associated acute kidney injury following coronary angiography: A meta-analysis of 1.2 million patients. *Journal of Nephrology* 34, 1479–1489.
- [2] Tea, I., Gilani, S., Kleiman, N.S., 2022. When prevention is truly better than cure: Contrast-associated acute kidney injury in percutaneous coronary intervention. *Methodist DeBakey Cardiovascular Journal* 18, 73–85.
- [3] Pranata, R., Wahyudi, D.P., 2024. Prevention of contrast-induced nephropathy in patients undergoing percutaneous coronary intervention. *Current Cardiology Reviews* 20, e241023222628.
- [4] American Diabetes Association Professional Practice Committee for Diabetes, 2026. 6. Glycemic goals, hypoglycemia, and hyperglycemic crises: Standards of care in diabetes—2026. *Diabetes Care* 49(Suppl. 1), S132–S149.
- [5] Li, M., Chen, G., Feng, Y., He, X., 2021. Stress-induced hyperglycemia in the context of acute coronary syndrome: Definitions, interventions, and underlying mechanisms. *Frontiers in Cardiovascular Medicine* 8, 676892.

- [6] Zhang, H., Fu, H., Fu, X., Zhang, J., Zhang, P., Yang, S., et al., 2021. Glycosylated hemoglobin levels and the risk for contrast-induced nephropathy in diabetic patients undergoing coronary arteriography/percutaneous coronary intervention. *BMC Nephrology* 22, 206.
- [7] Zhang, P., Fu, H., Liu, J., Liu, X., Yang, S., Guo, Z., et al., 2022. Preoperative fasting blood glucose levels and the risk of contrast-induced nephropathy in patients with diabetes and pre-diabetes undergoing coronary arteriography or percutaneous coronary intervention: A cross-sectional study. *Angiology* 73, 660–667.
- [8] Gao, S., Liu, Q., Chen, H., Yu, M., Li, H., 2021. Predictive value of stress hyperglycemia ratio for the occurrence of acute kidney injury in acute myocardial infarction patients with diabetes. *BMC Cardiovascular Disorders* 21, 157.
- [9] Shan, Y., Lin, M., Gu, F., Ying, S., Bao, X., Zhu, Q., et al., 2023. Association between fasting stress hyperglycemia ratio and contrast-induced acute kidney injury in coronary angiography patients: A cross-sectional study. *Frontiers in Endocrinology* 14, 1300373.
- [10] Li, Y., Zhang, L., Liu, W., Deng, J., Liu, J., Zhou, Y., et al., 2024. The impact of the stress hyperglycemia ratio on the risk of contrast-associated acute kidney injury in patients undergoing coronary angiography: A large real-world cohort study. *Diabetology & Metabolic Syndrome* 16, 107.
- [11] Zhao, Y., Zhao, Y., Wang, S., Fan, Z., Wang, Y., Liu, F., et al., 2025. Association between stress hyperglycemia ratio and contrast-induced nephropathy in ACS patients undergoing PCI: A retrospective cohort study from the MIMIC-IV database. *BMC Cardiovascular Disorders* 25, 135.
- [12] American Diabetes Association Professional Practice Committee for Diabetes, 2026. 2. Diagnosis and classification of diabetes: Standards of care in diabetes—2026. *Diabetes Care* 49(Suppl. 1), S27–S49.
- [13] Mehran, R., Nikolsky, E., 2006. Contrast-induced nephropathy: Definition, epidemiology, and patients at risk. *Kidney International Supplement* 100, S11–S15.
- [14] McEvoy, J.W., McCarthy, C.P., Bruno, R.M., Brouwers, S., Canavan, M.D., Ceconi, C., et al., 2024. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension: Developed by the task force on the management of elevated blood pressure and hypertension of the European Society of Cardiology (ESC) and endorsed by the European Society of Endocrinology (ESE) and the European Stroke Organisation (ESO). *European Heart Journal* 45, 3912–4018.
- [15] Inker, L.A., Eneanya, N.D., Coresh, J., Tighiouart, H., Wang, D., Sang, Y., et al., 2021. New creatinine- and cystatin C-based equations to estimate GFR without race. *New England Journal of Medicine* 385, 1737–1749.
- [16] Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group, 2024. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney International* 105(4S), S117–S314.
- [17] McCullough, P.A., Choi, J.P., Feghali, G.A., Schussler, J.M., Stoler, R.M., Vallabahn, R.C., et al., 2016. Contrast-induced acute kidney injury. *Journal of the American College of Cardiology* 68, 1465–1473.
- [18] Discacciati, A., Palazzolo, M.G., Park, J.G., Melloni, G.E.M., Murphy, S.A., Bellavia, A., 2025. Estimating and presenting non-linear associations with restricted cubic splines. *International Journal of Epidemiology* 54, dyaf088.
- [19] Jones, D.A., Beirne, A.M., Kelham, M., Wynne, L., Andiapien, M., Rathod, K.S., et al., 2024. Inorganic nitrate benefits contrast-induced nephropathy after coronary angiography for acute coronary syndromes: The NITRATE-CIN trial. *European Heart Journal* 45, 1647–1658.
- [20] Liu, L., Liang, Y., Li, H., Lun, Z., Ying, M., Chen, S., et al., 2021. Association between diabetes mellitus and contrast-associated acute kidney injury: A systematic review and meta-analysis of 1.1 million contrast exposure patients. *Nephron* 145, 451–461.
- [21] Ricciardi, C.A., Gnudi, L., 2021. Kidney disease in diabetes: From mechanisms to clinical presentation and treatment strategies. *Metabolism* 124, 154890.
- [22] Anjanappa, P., Kularia, S., Shrestha, Y., 2026. Contrast-induced acute kidney injury: From pathophysiology to risk prediction and prevention strategies. *Journal of Basic and Clinical Physiology and Pharmacology* 37, 201–212.

- [23] Korolov, Y., Nogués, I., Gambarte, M.J., Donato, M.S., Ciambone, M.G., Novo, F., et al., 2021. Microalbuminuria predicts contrast-induced nephropathy in patients with acute coronary syndrome. *REC: Interventional Cardiology* 3, 21–25.
- [24] Xu, T., Lin, M., Shen, X., Wang, M., Zhang, W., Zhao, L., et al., 2021. Association of the classification and severity of heart failure with the incidence of contrast-induced acute kidney injury. *Scientific Reports* 11, 15348.
- [25] Mohebi, R., Karimi Galougahi, K., Garcia, J.J., Horst, J., Ben-Yehuda, O., Radhakrishnan, J., et al., 2022. Long-term clinical impact of contrast-associated acute kidney injury following PCI: An ADAPT-DES substudy. *JACC: Cardiovascular Interventions* 15, 753–766.