


# A Comparison Between Two Different Definitions of Contrast-Associated Acute Kidney Injury for Long-Term Mortality in Patients with Chronic Kidney Disease Undergoing Coronary Angiography

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**Background:** Contrast-associated acute kidney injury (CA-AKI) is a prevalent complication following coronary angiography (CAG). However, there is ongoing controversy surrounding its precise definition. Although previous studies have demonstrated the successful application of appropriate definitions in managing high-risk CA-AKI patients, there remains limited research on the association between different definitions and prognosis specifically in patients with chronic kidney disease (CKD).

**Methods:** A total of 4197 CKD patients undergoing coronary angiography (CAG) were included in this study. Two definitions of contrast-associated acute kidney injury (CA-AKI) were used: CA-AKI<sub>A</sub>, which was defined as an increase of  $\geq 0.5$  mg/dL or  $>25\%$  in serum creatinine (SCr) from baseline within 72 hours after CAG, and CA-AKI<sub>B</sub>, which was defined as an increase of  $\geq 0.3$  mg/dL or  $>50\%$  in SCr from baseline within 48 hours after CAG. Cox regression analysis was employed to assess the association between these two definitions and long-term mortality. Additionally, population attributable risks (PARs) were calculated to evaluate the impact of CA-AKI definitions on long-term prognosis.

**Results:** During the median follow-up period of 4.70 (2.50–7.78) years, the overall long-term mortality was 23.6%, and the long-term mortality in patients with CA-AKI according to both CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub> criteria were 33.5% and 33.8%, respectively. We found that CA-AKI<sub>A</sub> (HR: 1.45, 95% CI: 1.23–1.70,  $p < 0.001$ ) and CA-AKI<sub>B</sub> (HR: 1.44, 95% CI: 1.23–1.69,  $p < 0.001$ ) were associated with long-term mortality. The PARs were the highest for CA-AKI<sub>A</sub> (5.87%), followed by CA-AKI<sub>B</sub> (5.70%).

**Conclusion:** Contrast-associated acute kidney injury (CA-AKI) is a frequently observed complication in CKD patients undergoing coronary angiography (CAG), and both definitions of CA-AKI are significantly correlated with a poor long-term prognosis. Consequently, in the clinical management of CKD patients, it is crucial to prioritize CA-AKI, irrespective of the specific CA-AKI definition used.

**Keywords:** contrast-associated acute kidney injury, chronic kidney disease, definitions, long-term mortality, population attributable risks

## Introduction

With the aging population, the incidence of coronary heart disease increases. Contrast-associated acute kidney injury (CA-AKI) is a common complication of coronary angiography (CAG) and is associated with a poor prognosis. Previous studies have shown that the incidence of CA-AKI is between 5–20%, with high rates of rehospitalization, dialysis, and mortality.<sup>1–4</sup> However, the diversity in the definition of CA-AKI makes it challenging for clinicians to effectively

recognize and address this issue in clinical practice. Different definitions of CA-AKI have varying incidence rates and prognoses.<sup>5</sup> For instance, the Acute Kidney Injury Network (AKIN) criteria have demonstrated better efficacy in predicting long-term mortality in patients with ST-elevation myocardial infarction (STEMI).<sup>6</sup> For diabetic patients, the definition of CA-AKI<sub>A</sub> (an increase in serum creatinine levels by 0.3 mg/dl or 50% within 72 hours after the procedure) is more suitable as it is associated with poorer clinical outcomes and higher population attributable risks (PARs).<sup>7</sup> Furthermore, the European Society of Urogenital Radiology definition of CA-AKI can better identify high-risk patients with poor prognosis at an early stage in elderly and heart failure patients.<sup>8,9</sup> It is well known that chronic kidney disease (CKD) is a risk factor for CA-AKI. CA-AKI patients with CKD have a 36% all-cause mortality rate and an increased risk of renal dialysis treatment as their estimated glomerular filtration rate (eGFR) decreases.<sup>10–12</sup> However, few studies have explored the applicable definition of CA-AKI in patients with CKD. Therefore, this article aims to evaluate the association between different definitions of CA-AKI and long-term mortality in CKD patients after coronary angiography.

## Methods

### Study Design and Population

This study is a single-center retrospective observational study conducted at Dongguan TCM Hospital. Between January 2015 and January 2023, a total of 4197 consecutive patients, aged 18 years or older, who were diagnosed with CKD, underwent coronary angiography and were included in the study. All patients underwent percutaneous coronary intervention (PCI) and coronary angiography (CAG) following standard guidelines. Exclusion criteria for the study included pregnancy, lactation, recent intravascular administration of contrast media within 7 days before or 3 days after the operation, non-use of low osmotic pressure contrast media, cardiovascular surgery or intravascular repair, end-stage renal disease or kidney replacement, creatinine deficiency before or after the operation, malignant tumor, and non-use of isotonic saline for hydration. The study protocol was designed following the principles outlined in the Declaration of Helsinki and received approval from the Ethics Committee of Dongguan TCM Hospital (approval number: PJ (2023) 31).

### Endpoint and Definitions

The primary outcome in this article was long-term all-cause mortality. Our study calculated the estimated glomerular filtration rate (eGFR) by applying the Modification of Diet in Renal Disease (MDRD) formula. The definition of CKD was  $eGFR \leq 60 \text{ mL/min/1.73 m}^2$ .<sup>13,14</sup> CA-AKI<sub>A</sub> was defined as an increase  $\geq 0.5 \text{ mg/dL}$  or  $>25\%$  in SCr from baseline within 72 hours after CAG, and CA-AKI<sub>B</sub> was defined as an increase  $\geq 0.3 \text{ mg/dL}$  or  $>50\%$  in serum creatinine (SCr) from baseline within 48 hours after CAG.<sup>15,16</sup> Serum creatinine (SCr) concentrations were measured on days 1, 2, and 3 in all patients before and after surgery. Serum creatinine (SCr) from the day before surgery was used as baseline creatinine. Chronic heart failure (CHF) was defined as Killip classes II–IV or New York Heart Association (NYHA) classes III–IV.<sup>10</sup> Anemia was confirmed by gender and hematocrit (male's hematocrit below 39% and female's hematocrit below 36%). Diabetes was defined as a previous diagnosis of diabetes or an HbA1c level  $\geq 6.5$ .

### Statistical Analysis

Continuous variables were reported as mean  $\pm$  standard deviation (SD) or median  $\pm$  interquartile range (IQR). To compare differences between two independent samples (with and without CA-AKI) using different definitions, Wilcoxon rank-sum tests or Student's t-tests were employed. Categorical variables, expressed as counts (percentages), were analyzed using the Chi-square test or Fisher's exact test to assess differences. In the initial step, univariate logistic regression models were utilized to identify predictors of CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub>. The results of this analysis are presented as hazard ratios with corresponding 95% confidence intervals (CIs). Additionally, multivariate logistic regression analysis was performed. Cumulative mortality rates based on various definitions were calculated using Kaplan-Meier analysis, and differences between survival curves were assessed using the Log rank test. A multivariate Cox regression model adjusting for other risk factors such as age, diabetes, CHF, and stroke was used to examine the relationship between CA-AKI and long-term mortality. The adjusted risk factors in the univariate Cox regression analysis

were selected based on previous studies and clinical significance. Two separate multivariate Cox proportional hazard regression models were applied for each definition of CA-AKI. Population attributable risk (PAR) was computed using the equation:  $PAR = P(HR-1)/[1+P(HR-1)]$ , where p represents the incidence of CA-AKI according to different definitions in our database. The standard error of the PAR was estimated using the delta method. A two-sided probability value less than 0.05 was considered statistically significant. All data analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC) and R software (version 4.0.0; R Core Team, Vienna, Austria).

## Result

### Patient Characteristics

A total of 4197 patients who received coronary angiography were ultimately included in the analysis. The baseline characteristics were shown in Table 1. The overall mean age was  $67.56 \pm 10.20$  years, and 1141 (27.2%) patients were older than 75 years. There were 1009 (24.0%) patients with CHF, 2885 (68.7%) patients with hypertension, and 876 (20.9%) patients with AMI. A total of 2492 (59.4%) patients underwent PCI.

### Incidence of CA-AKI by the CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub> Criteria

According to the contrast-induced acute kidney injury (CA-AKI<sub>A</sub>) criteria, CA-AKI occurred in 582 patients (13.9%), while according to the CA-AKI<sub>B</sub> criteria, CA-AKI occurred in 577 patients (13.7%). However, regardless of the definition of contrast-induced acute kidney injury, patients with CA-AKI were older, had reduced left ventricular ejection fraction (LVEF), and had more underlying diseases. The usage of beta-blockers and statins was lower in patients with

**Table 1** Baseline Characteristics

Variables	Total (n = 4197)	CA-AKI <sub>A</sub>			CA-AKI <sub>B</sub>		
		Yes (n=582)	No (n=3615)	P value	Yes (n=577)	No (n=3620)	P value
Age, y	67.56 ± 10.20	67.90 ± 10.53	67.50 ± 10.15	0.380	68.41 ± 10.46	67.42 ± 10.15	0.031
Age ≥ 75, n (%)	1141 (27.2)	177 (30.4)	964 (26.7)	0.067	184 (31.9)	957 (26.4)	0.007
Male sex, n (%)	2884 (68.7)	358 (61.5)	2526 (69.9)	<0.001	389 (67.4)	2495 (68.9)	0.499
CHF, n (%)	1009 (24.0)	221 (38.0)	788 (21.8)	<0.001	217 (37.6)	792 (21.9)	<0.001
CAD, n (%)	3226 (76.9)	407 (69.9)	2819 (78.0)	<0.001	433 (75.0)	2793 (77.2)	0.287
Stroke, n (%)	400 (9.5)	56 (9.6)	344 (9.5)	0.996	64 (11.1)	336 (9.3)	0.194
LVEF, %	54.56 ± 14.35	52.41 ± 14.60	54.92 ± 14.27	0.001	51.62 ± 14.67	55.03 ± 14.24	<0.001
Hypertension, n (%)	2885 (68.7)	368 (63.2)	2517 (69.6)	0.002	289 (67.4)	2496 (69.0)	0.491
Anemia, n (%)	2283 (54.7)	386 (66.4)	1897 (52.8)	<0.001	391 (67.9)	1892 (52.6)	<0.001
AMI, n (%)	876 (20.9)	136 (23.4)	740 (20.5)	0.123	132 (22.9)	744 (20.6)	0.222
eGFR mL/min/1.73 m <sup>2</sup>	42.86 ± 14.29	39.96 ± 16.02	43.33 ± 13.94	<0.001	38.32 ± 15.58	43.58 ± 13.94	<0.001
LDL-C, mmol/L	2.74 ± 0.96	2.72 ± 0.93	2.75 ± 0.97	0.617	2.73 ± 0.95	2.74 ± 0.97	0.658
HDL-C, mmol/L	0.97 ± 0.27	0.97 ± 0.27	0.96 ± 0.27	0.397	0.98 ± 0.27	0.96 ± 0.27	0.319
HS-CRP, mg/L	20.43 ± 36.75	35.23 ± 47.23	18.68 ± 34.91	<0.001	34.79 ± 45.17	18.68 ± 35.21	<0.001
SCR, μmol/L	174.39 ± 140.15	195.70 ± 169.09	170.96 ± 134.62	<0.001	202.15 ± 164.75	169.97 ± 135.32	<0.001
Hemoglobin, g/L	123.47 ± 21.50	116.56 ± 22.23	124.59 ± 21.17	0.001	116.23 ± 22.29	124.63 ± 21.14	<0.001
Hypoalbuminemia, n (%)	2313 (56.6)	360 (63.2)	1953 (55.6)	0.001	372 (66.3)	1941 (55.1)	<0.001
Serum albumin, g/L	33.90 ± 4.84	32.96 ± 5.41	34.06 ± 4.73	<0.001	32.65 ± 5.29	34.10 ± 4.47	<0.001
Cholesterol, mmol/L	4.48 ± 1.22	4.44 ± 1.20	4.49 ± 1.22	0.386	4.45 ± 1.20	4.49 ± 1.22	0.495
Beta-blocker, n (%)	2892 (72.9)	315 (63.4)	2577 (74.2)	<0.001	335 (68.2)	2557 (73.5)	0.016
Statins, n (%)	3211 (80.9)	336 (67.6)	2875 (82.8)	<0.001	365 (74.3)	2846 (81.8)	<0.001
PCI, n (%)	2492 (59.4)	309 (53.1)	2183 (60.4)	0.001	330 (57.2)	2162 (59.7)	0.270
Death, n (%)	991 (23.6)	195 (33.5)	796 (22.0)	<0.001	195 (33.8)	796 (22.0)	<0.001

**Notes:** CA-AKI<sub>A</sub>, which was defined as an increase of  $\geq 0.5$  mg/dL or  $>25\%$  in serum creatinine from baseline within 72 hours after coronary angiography. CA-AKI<sub>B</sub>, which was defined as an increase of  $\geq 0.3$  mg/dL or  $>50\%$  in serum creatinine from baseline within 48 hours after coronary angiography.

**Abbreviations:** CHF, chronic heart failure; CAD, coronary artery disease; LVEF, left ventricular ejection fraction; AMI, acute myocardial infarction; eGFR, estimated glomerular filtration rate; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; HS-CRP, high-sensitivity C-reactive protein; SCR, serum creatinine; PCI, percutaneous coronary intervention.

CA-AKI<sub>A</sub> or CA-AKI<sub>B</sub>. According to the CA-AKI<sub>B</sub> criteria, no significant association was found between hypertension and coronary artery disease (CAD) in patients with CA-AKI compared to non-CA-AKI patients.

## Incidence and Predictors of Long-term Mortality

During a median follow-up period of 4.70 (2.50–7.78) years, the overall long-term mortality rate was 23.6%. Specifically, among patients with CA-AKI according to both CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub> criteria, the long-term mortality rates were 33.5% and 33.8%, respectively. Regardless of the definition of CA-AKI, Kaplan-Meier curves showed that the long-term prognosis of patients with CA-AKI was worse than that of those without CA-AKI according to both CA-AKI definitions (log-rank  $p < 0.01$ ; Figure 1).

Table 2 and Figure 2 present the results of univariate and multivariate analyses of risk factors for long-term mortality. The analyses revealed several independent predictive factors associated with long-term mortality, including age, PCI, DM, CHF, anemia, and CA-AKI. Notably, CA-AKI was associated with an increased risk of long-term mortality. Both CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub> increased the risk of long-term mortality by approximately 1.45 times (95% CI 1.23–1.70 and 95% CI 1.23–1.69, respectively).

## PARs of CA-AKI by CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub> Criteria

Among the two definitions of CA-AKI, the prevalence of CA-AKI<sub>A</sub> was 13.9%, while the prevalence of CA-AKI<sub>B</sub> was 13.7%. The population attributable risk (PAR) for CA-AKI<sub>A</sub> was 5.87% (95% CI 3.09–8.85%), and for CA-AKI<sub>B</sub> it was 5.70% (95% CI 3.07–8.66%) (Figure 2).

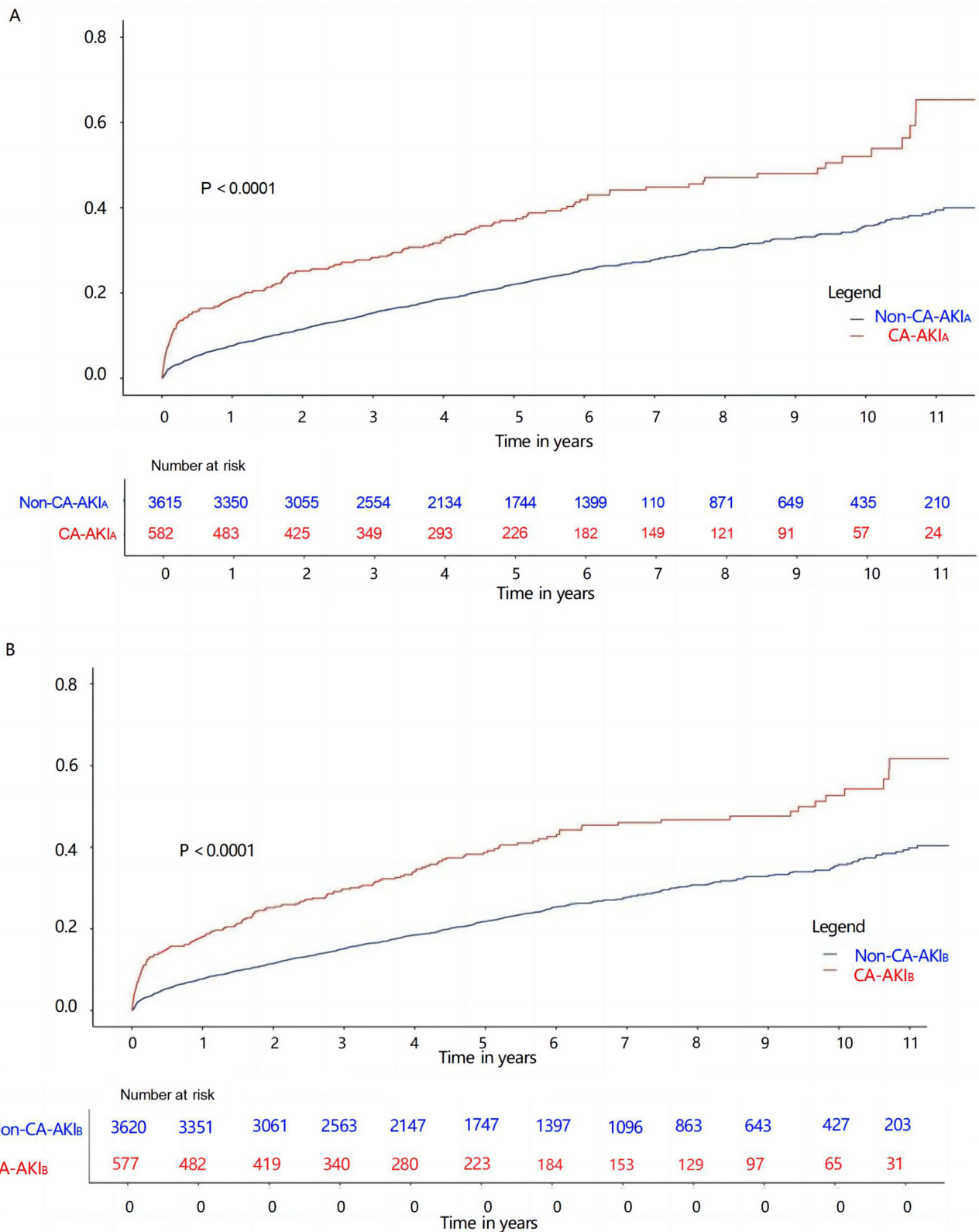
## Discussion

We are the first to investigate the relationship between the definition of CA-AKI and long-term mortality in CKD patients. Our findings demonstrate that regardless of the definition used, CA-AKI is associated with increased long-term mortality. Additionally, our study identifies age, PCI, DM, CHF, and anemia as independent risk factors for long-term mortality in CKD patients undergoing CAG.

The incidence of CA-AKI<sub>A</sub> in our study was slightly lower than that of CA-AKI<sub>B</sub>, as well as their respective mortality rates. However, our study observed a higher incidence compared to previous reports, which can be attributed to the inclusion of CKD patients.<sup>17,18</sup> CKD is a known significant risk factor for CA-AKI and is often incorporated into prediction models.<sup>19,20</sup> Furthermore, different definitions of CA-AKI exhibit variations in incidence and mortality, a finding also supported by Wang et al's study,<sup>8</sup> affirming our results.

Additionally, our study shows that CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub> increase the risk of long-term mortality by approximately 1.45 times and 1.44 times, respectively. This aligns with previous research indicating that different definitions of CA-AKI yield varying risks. Centola et al discovered that different definitions of CA-AKI lead to different long-term prognostic risks among 402 STEMI patients.<sup>6</sup> They found that CA-AKI defined by the AKIN standard increased the risk of long-term mortality by 9.70 times, which is higher than our results, likely due to disparate follow-up durations. In our study, the median follow-up period was 4.7 years, while Centola's study followed patients for only 12 months. Similarly, He et al identified different definitions of CA-AKI as having distinct long-term prognoses in PCI patients, with AKI defined by AKIN criteria increasing the risk of death by 2.2 times,<sup>9</sup> closely resembling our findings. Wang et al conducted similar studies in patients with AMI, HF, and diabetes, further corroborating our results,<sup>7,8,21</sup> adding credibility to our findings.

Furthermore, our study reveals that the population attributable risks (PAR) of CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub> are 5.87% and 5.70%, respectively. However, Wang et al's study found that the PAR of CA-AKI<sub>A</sub> (an increase  $\geq 0.5$  mg/dl or  $>25\%$  in SCr) was higher than that of CA-AKI<sub>B</sub> (an increase of  $\geq 0.3$  mg/dl or  $>50\%$  in SCr),<sup>8</sup> which differs from our results. The discrepancy can be attributed to the inclusion of CHF patients in Wang's analysis, while our study focused on CKD patients. Moreover, previous studies have demonstrated that in patients with diabetes, CA-AKI<sub>A</sub> (an increase of  $\geq 3$  mg/dl or  $>50\%$  in SCr) has a higher PAR. This indicates that the same definition of CA-AKI can yield varying PARs in different patient populations, underscoring the importance of tailoring the definition for each patient.<sup>7</sup> In our study, different definitions of CA-AKI resulted in similar PARs, emphasizing the need to prevent and treat CA-AKI in CKD patients. CA-AKI may be a significant risk factor for long-term mortality rather than an intermediary factor, aligning with



**Figure 1** Kaplan-Meier curves for the long-term prognosis of patients with CA-AKI and without CA-AKI. **(A)** Kaplan-Meier for the long-term prognosis of patients with CA-AKI<sub>a</sub> and without CA-AKI<sub>a</sub>. **(B)** Kaplan-Meier for the long-term prognosis of patients with CA-AKI<sub>b</sub> and without CA-AKI<sub>b</sub>. **Notes:** CA-AKI<sub>a</sub>, which was defined as an increase of  $\geq 0.5$  mg/dL or  $>25\%$  in serum creatinine from baseline within 72 hours after coronary angiography. CA-AKI<sub>b</sub>, which was defined as an increase of  $\geq 0.3$  mg/dL or  $>50\%$  in serum creatinine from baseline within 48 hours after coronary angiography.

**Table 2** Univariable Analysis of Risk Factors for Long-Term Mortality

Variables	Univariable Analysis		
	HR	95% CI	P value
Age	1.02	1.01–1.02	<0.001
Age≥75	1.41	1.23–1.61	<0.001
Male	1.07	0.94–1.23	0.309
CHF	2.14	1.88–2.45	<0.001
CAD	1.15	0.99–1.34	0.073
Stroke	1.30	1.07–1.59	0.009
Hypertension	0.93	0.82–1.06	0.302
Anemia	1.59	1.40–1.81	<0.001
AMI	1.34	1.15–1.55	<0.001
eGFR	0.98	0.97–0.98	<0.001
LDL-C	0.96	0.90–1.03	0.257
HDL-C	0.77	0.55–0.89	0.004
Hypoalbuminemia	1.62	1.41–1.86	<0.001
Cholesterol	1.09	0.94–1.27	0.275
Beta-blocker	0.94	0.81–1.09	0.440
Statins	0.98	0.82–1.16	0.771
PCI	0.86	0.76–0.97	0.016
CA-AKI <sub>A</sub>	1.73	1.48–2.03	<0.001
CA-AKI <sub>B</sub>	1.74	1.49–2.03	<0.001

**Notes:** CA-AKI<sub>A</sub>, which was defined as an increase of  $\geq 0.5$  mg/dL or  $>25\%$  in serum creatinine from baseline within 72 hours after coronary angiography. CA-AKI<sub>B</sub>, which was defined as an increase of  $\geq 0.3$  mg/dL or  $>50\%$  in serum creatinine from baseline within 48 hours after coronary angiography.

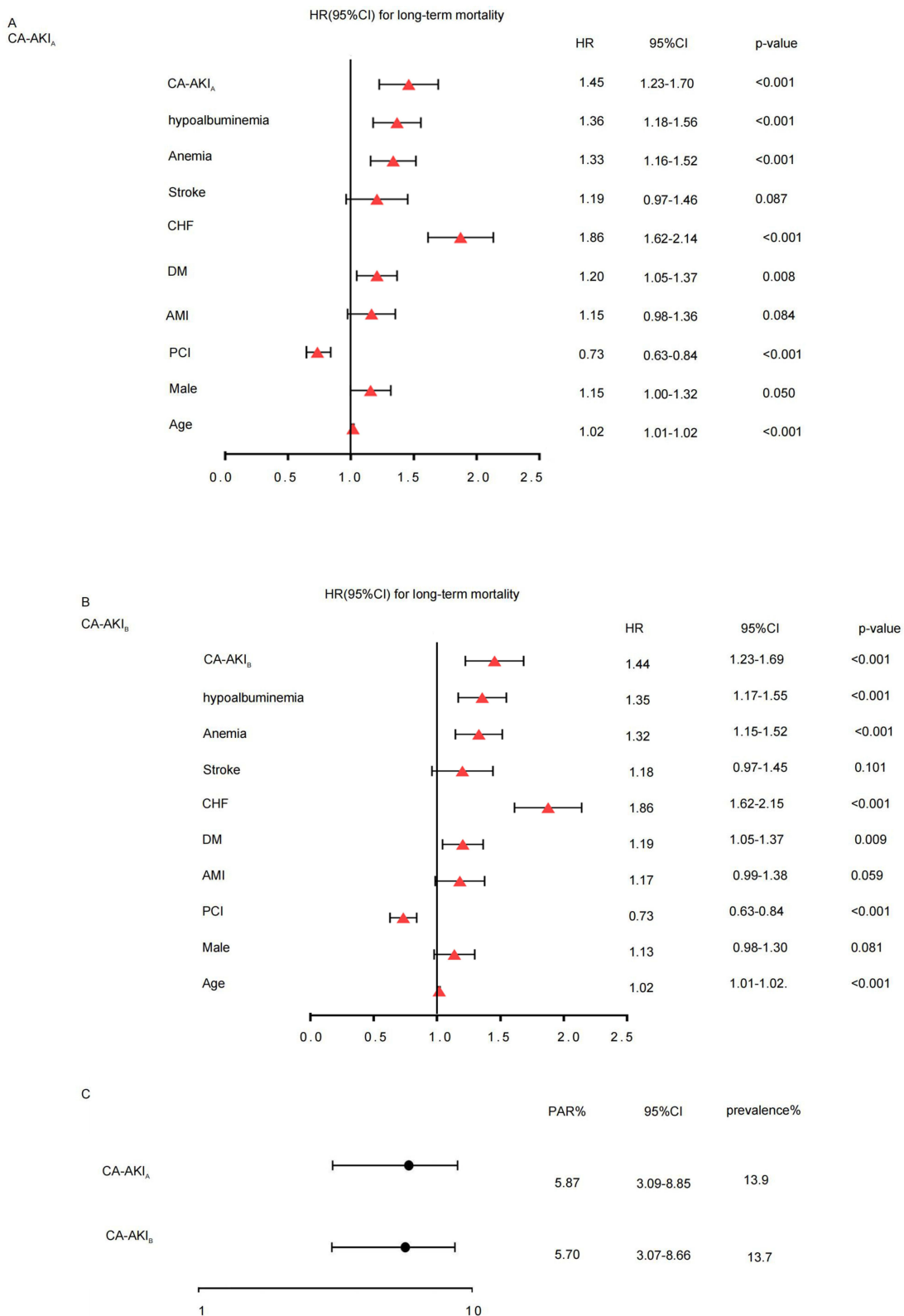
**Abbreviations:** CHF, chronic heart failure; CAD, coronary artery disease; AMI, acute myocardial infarction; eGFR, estimated glomerular filtration rate; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; PCI, percutaneous coronary intervention.

Mehran's recently described viewpoint.<sup>22</sup> Lassnigg et al's research also supports this perspective, finding that even small fluctuations in serum creatinine are significantly associated with death.<sup>23</sup>

The pathogenesis of CA-AKI primarily involves hemodynamic changes in the kidney, the toxic effect of contrast agents on renal tubular cells, and oxidative stress. CKD reduces renal cell tolerance to toxins, increasing the risk of CA-AKI. Consequently, our findings highlight the need to prioritize attention and treatment for CA-AKI in CKD patients. While current guidelines recommend evaluating CA-AKI risk and administering hydration therapy to high-risk populations before CAG,<sup>24</sup> there is no mention of CA-AKI prevention specifically for CKD patients. Recent studies have identified that preoperative simplified rapid hydration can effectively reduce the risk of CA-AKI in CKD patients.<sup>25</sup>

## Limitations

Several limitations were identified in this study. Firstly, the study solely focused on patients with CKD, and therefore, caution should be exercised when extrapolating the findings to the general population. Secondly, the study utilized a single-center retrospective cohort design, potentially limiting its representativeness. However, the inclusion of over 4000 CKD patients enhanced the reliability of the results. Thirdly, the study's sample size constraints restricted the number of variables available for adjustment in the multivariable analysis. It is important to note that long-term mortality is a complex outcome influenced by multiple factors. Fourthly, for acute patients, because the hospital stay is shorter, failure to complete the preoperative creatinine test may affect the applicability of the results.



**Figure 2** HR(95% CI) for long-term mortality. (A) HR(95% CI) for long-term mortality of CA-AKI<sub>A</sub>. (B) HR(95% CI) for long-term mortality of CA-AKI<sub>B</sub>. (C) The comparison of CA-AKI<sub>A</sub> and CA-AKI<sub>B</sub>.

**Notes:** CA-AKI<sub>A</sub>, which was defined as an increase of  $\geq 0.5$  mg/dL or  $>25\%$  in serum creatinine from baseline within 72 hours after coronary angiography. CA-AKI<sub>B</sub>, which was defined as an increase of  $\geq 0.3$  mg/dL or  $>50\%$  in serum creatinine from baseline within 48 hours after coronary angiography.

**Abbreviation:** PAR, the population attributable risk.

## Conclusion

Contrast-induced acute kidney injury (CA-AKI) is a frequently observed complication in CKD patients undergoing coronary angiography (CAG), and both CA-AKI definitions are linked to an unfavorable prognosis. Therefore, in the clinical practice of CKD patients, it is crucial to give heightened attention to CA-AKI, irrespective of the specific definition utilized.

## Abbreviations

CA-AKI, Contrast-associated acute kidney injury; CAG, Complication following coronary angiography; CKD, Chronic kidney disease; AKIN, Acute Kidney Injury Network; PARs, Population attributable risks; eGFR, Estimated glomerular filtration rate; PCI, Percutaneous coronary intervention; MDRD, Modification of Diet in Renal Disease; CHF, Chronic heart failure; NYHA, New York Heart Association.

## Data Sharing Statement

The dataset generated for this study is not publicly available, but is available from the corresponding author upon reasonable request.

## Ethics Approval and Consent to Participate

The studies involving human participants were reviewed and approved by The Ethics Committee of Dongguan Traditional Chinese Medicine Hospital (approval number: PJ (2023) 31). Prior to the study, all participants had given informed consent and signed written informed consent.

## Consent for Publication

All authors have given consent for publication.

## Acknowledgments

We thank all subjects and colleagues for participating in the study.

## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

## Funding

The study was sponsored by the Dongguan social development technology project (Grant Nos. 20231800935442).

## Disclosure

The authors declare that they have no competing interests in this work.

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