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ORIGINAL RESEARCH

Total cholesterol as an independent risk factor for contrast-induced acute kidney injury in patients undergoing coronary angiography

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Abstract

Background: Identifying high-risk patients based on risk factors is crucial for targeted prevention and management strategies. This study investigated the relationship between total cholesterol (TC) and contrast-induced acute kidney injury (CI-AKI). **Methods**: This multicenter retrospective study analyzed patients undergoing coronary angiography at five tertiary hospitals in China between January 2007 and December 2021. Univariate and multivariate logistic regression models were employed to investigate the association between total cholesterol levels and contrast-induced acute kidney injury (CI-AKI). Based on international diagnostic criteria, CI-AKI was defined as either an absolute elevation of serum creatinine (SCr) $\geq$0.5 mg/dL or a relative increase $\geq$25% from baseline values within 48–72 hours following intravenous contrast administration. **Results**: A total of 38,938 patients (mean age 63.1 $\pm$ 11.4 years, 71.6% male) were included in this study. The analysis revealed a progressive elevation in CI-AKI incidence across cholesterol quartiles, with rates of 10.7% (Q1), 10.7% (Q2), 12.2% (Q3) and 13.6% (Q4). Among 4587 documented CI-AKI cases (11.7% prevalence), serum total cholesterol concentrations were significantly elevated compared to non-CI-AKI counterparts (4.7 $\pm$ 1.2 *vs.* 4.6 $\pm$ 1.1 mmol/L, $p < 0.001$). Multivariable adjustment confirmed total cholesterol as an independent predictor for CI-AKI development (odds ratio (OR), 1.103, 95% confidence interval (CI), 1.072–1.135, $p < 0.001$). **Conclusions**: The measurement of plasma TC concentration in patients undergoing Coronary Angiography (CAG) may serve as a valuable tool for identifying individuals at risk of CI-AKI, thus representing a novel and significant risk factor for CI-AKI.

Keywords

Contrast-induced acute kidney injury; Total cholesterol; Coronary angiography

1. Introduction

Contrast-induced acute kidney injury (CI-AKI) represents a clinically significant complication of coronary angiography, with incidence rates documented up to 12.8% in observational studies [1]. It is a leading cause of hospital-acquired renal failure, ranking as the third most common cause, and it affects 1%–2% of patients who lack predisposing risk factors [2]. As diagnostic imaging techniques and minimally invasive interventions increasingly rely on contrast agents for clinical assessments, CI-AKI has emerged as a significant contributor to iatrogenic renal impairment in modern healthcare practice [3]. CI-AKI is associated with increased risk of requiring hospital dialysis, developing long-term renal failure and increased mortality, with reported rates of 7%–31%. Although increasing emphasis on the prevention of CI-AKI has reduced its prevalence, it remains a significant cause of AKI in hospitalised patients. Given the lack of effective AKI treatments, the early and accurate prediction of CI-AKI is critical. Therefore, precise stratification, identification and pre-emptive management of high-risk CI-AKI patients are crucial.

Although previous studies have validated a series of risk stratification models that include patient-related and surgical factors, with the most classical being the Mehran score of eight variables in the 2004 study by Mehran *et al.* [4], few studies have explored the connection between TC and CI-AKI. Chronic inflammation exacerbates endothelial dysfunction and oxidative stress, which are key contributors to renal injury. A deterioration in renal function is associated with increased inflammatory burden, as evidenced by elevated markers such as interleukin-6 (IL-6) and C-reactive protein in patients with AKI. Therefore, the investigation of the role of TC in CI-AKI is both clinically and mechanistically justified. Although

the pathophysiology of CI-AKI remains largely unknown, we speculate that TC might be linked to CI-AKI through the mechanism contributing to the development of CI-AKI and aim to explore the value of TC for predicting CI-AKI.

2. Methods

2.1 Study design and population

The CIN-II investigation (Cardiorenal Improvement II, NCT05050877) constitutes a multicenter retrospective cohort analysis encompassing five tertiary care centers in China, implemented between January 2007 and December 2021. Patients undergoing coronary angiography (CAG) due to clinical indications were included in this study. The inclusion criteria were as follows: (1) undergoing CAG, (2) availability of lipid data at baseline and (3) records of serum creatinine (SCr) levels at baseline and within 48–72 hours after contrast exposure. Meanwhile, exclusion criteria were as follows: (1) presence of severe heart valve disease or planned heart surgery (*e.g.*, heart valve replacement or coronary artery bypass surgery) and (2) preoperative estimated glomerular filtration rate (eGFR) less than 15 mL/min/1.73 m^2 or existing long-term haemodialysis. In total, 38,938 patients were enrolled in this study. This multicenter protocol obtained institutional review board clearance through Guangdong Provincial People's Hospital Ethics Committee (GDREC2019-555H-2) with subsequent approvals from collaborating institutions, adhering to Declaration of Helsinki standards. Comprehensive data de-identification protocols were implemented to preserve participant confidentiality.

2.2 Data collection

Patient longitudinal records were systematically acquired through institutionally standardized electronic health record platforms deployed at all study sites. The dataset encompassed comprehensive clinical variables spanning demographic profiles, historical diagnoses, biochemical parameters, therapeutic interventions and operative records. These parameters were methodologically curated within a structured repository compliant with FAIR (Findable; Accessible; Interoperable; Reusable) principles, enabling analytical processing via R statistical environment (v4.3.1, R Foundation for Statistical Computing, Austria). Two senior cardiologists were responsible for data quality control and regular database validation. Standardized serum creatinine (SCr) quantification was performed institution-wide using Beckman Coulter AU5800 platforms (Beckman Coulter, Brea, CA, USA), with baseline assessments at hospitalization and repeat measurements 48–72 hours post-contrast administration, employing Jaffe's kinetic colorimetric methodology. Additional clinical indicators, such as routine blood findings, serum glucose and lipid levels, were also measured before surgery. Preprocedural echocardiographic assessment of left ventricular systolic performance was standardized across all cases prior to coronary angiography. Diagnostic or interventional coronary procedures (CAG/PCI) were executed in alignment with contemporary interventional cardiology guidelines and evidence-based procedural protocols.

2.3 Outcomes and definitions

The primary endpoint of the study was the incidence of CI-AKI, which is defined as an absolute increase in SCr by 0.5 mg/dL or 25% over baseline values within 48–72 h after exposure to contrast medium (CM) [5]. The secondary endpoints included long-term all-cause mortality after admission. Additionally, congestive heart failure (CHF) was classified as New York Heart Association class >2 or Killip class >1. Acute myocardial infarction (AMI), type 2 diabetes, and hypertension (HT) were diagnosed based on the codes of the International Classification of Diseases, 10th Revision (ICD-10).

2.4 Statistical analysis

The population was categorised into four groups based on TC quartiles. Q1 (TC <3.77 mmol/L), Q2 (TC 3.77–4.50 mmol/L), Q3 (TC 4.50–5.29 mmol/L) and Q4 groups (TC ≥ 5.30 mmol/L). The results of descriptive analysis for continuous variables are shown as mean $\pm$ standard deviation or median and interquartile range, according to the distribution. Proportions were used to describe categorical variables. Continuous variables were analysed using analysis of variance (ANOVA), while categorical variables were assessed using Pearson's chi-squared test. Kaplan-Meier curves were employed to visualize time-to-event data (Fig. 1), and differences between groups were assessed by the log-rank test. Univariate and multivariate logistic regression models were used to analyse the relationship between TC level and CI-AKI, with OR and 95% CI being calculated. A variance inflation factor (VIF) of 5 or higher was considered indicative of multicollinearity among independent variables. The nonlinear association was further evaluated using restricted cubic splines in logistic regression models. A two-tailed p-value of < 0.05 was considered statistically significant. All statistical analyses were performed using R software (version 4.3.1; R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1 Baseline characteristics

A total of 38,938 patients undergoing PCI were analysed in this study (mean age 63.1 $\pm$ 11.4 years; mean TC level 4.6 $\pm$ 1.1 mmol/L). With increasing TC levels, several trends were observed: the average age gradually decreased, the proportion of males also decreased and the proportions of patients with hypertension, likewise decreased. There were also statistical differences among non-smokers, current smokers and former smokers. Conversely, the levels of white blood cell (WBC) and eGFR increased, while contrast medium volume decreased, Additionally, the proportion of patients undergoing PCI increased (Table 1).

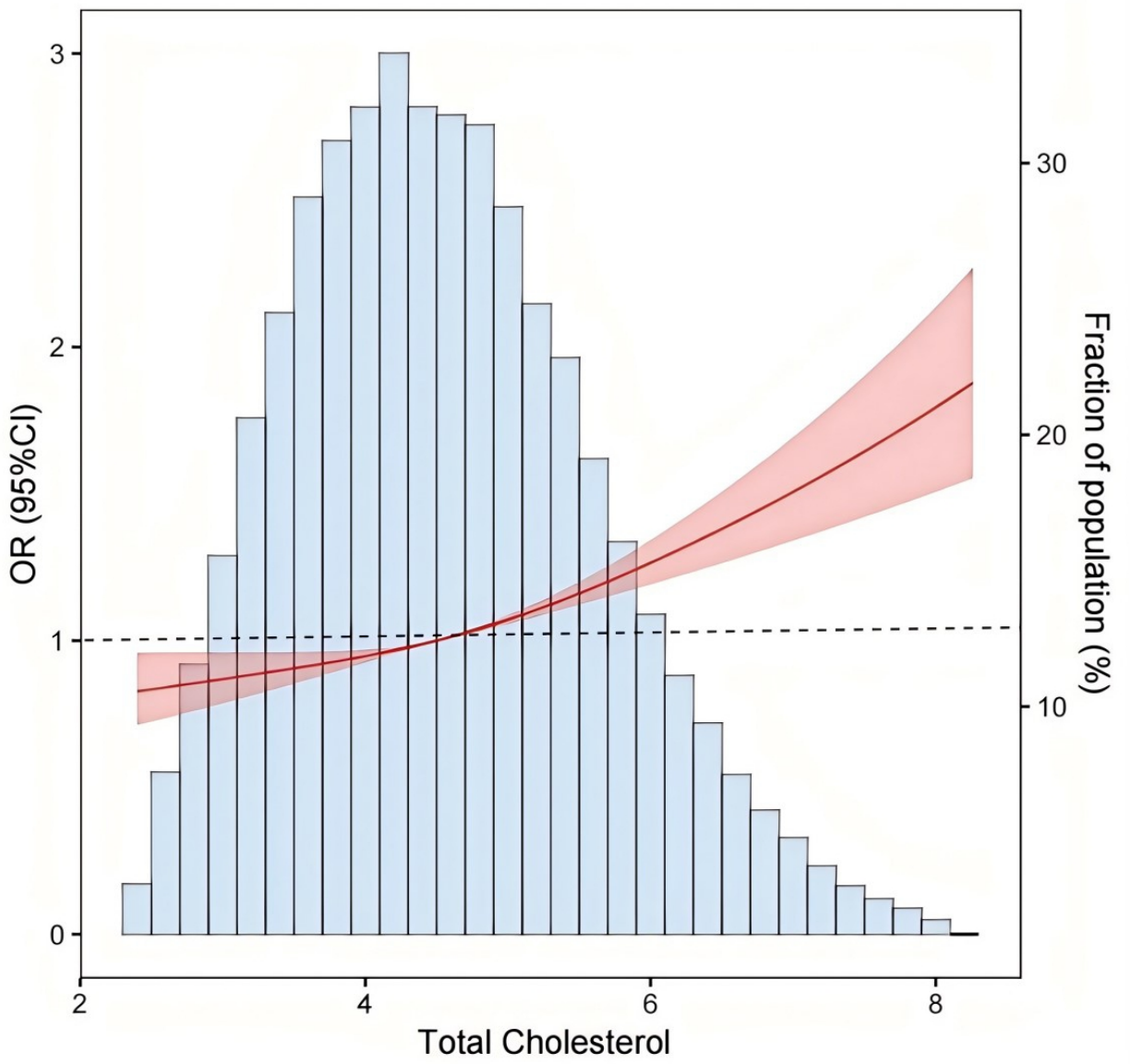

FIGURE 1. Nonlinear association between admission TC level and CI-AKI in patients undergoing percutaneous coronary intervention. OR: odds ratio; CI: confidence interval.

TABLE 1. Baseline characteristics according to TC level categories.

Characteristics	All (n = 38,938)	Q1 (n = 9813)	Q2 (n = 9883)	Q3 (n = 9627)	Q4 (n = 9615)	*p*	SMD
Age (yr)	63.1 (11.4)	64.3 (11.6)	63.5 (11.2)	63.0 (11.3)	61.7 (11.4)	<0.0001	0.1218
Male (%)	27,898 (71.6)	7770 (79.2)	7212 (73.0)	6703 (69.6)	6213 (64.6)	<0.0001	0.1761
Clinical profile (%)							
Diabetes mellitus	12,988 (33.4)	3587 (36.6)	3207 (32.4)	3043 (31.6)	3151 (32.8)	<0.0001	0.0534
Hypertension	21,041 (54.4)	5657 (58.0)	5377 (54.8)	5120 (53.6)	4887 (51.2)	<0.0001	0.0722
CHF	7417 (19.2)	1960 (20.1)	1890 (19.3)	1803 (18.9)	1764 (18.5)	0.0318	0.0220
AMI	10,976 (28.4)	2179 (22.3)	2623 (26.7)	2814 (29.5)	3360 (35.2)	<0.0001	0.1535
Smoke (%)							
Never	17,597 (63.0)	4241 (61.1)	4398 (62.2)	4313 (62.7)	4645 (66.0)		
Current	7223 (25.9)	1741 (25.1)	1837 (26.0)	1881 (27.4)	1764 (25.1)	<0.0001	0.0973
Former	3110 (11.1)	964 (13.9)	837 (11.8)	680 (9.9)	629 (8.9)		

TABLE 1. Continued.

Characteristics	All (n = 38,938)	Q1 (n = 9813)	Q2 (n = 9883)	Q3 (n = 9627)	Q4 (n = 9615)	*p*	SMD
Laboratory data							
WBC (10^9/L)	9.1 (45.8)	8.3 (13.5)	8.7 (17.3)	9.6 (56.4)	10.0 (69.2)	0.0444	0.0247
HGB (g/L)	133.4 (17.9)	129.1 (18.9)	132.4 (17.4)	134.9 (17.0)	137.1 (17.3)	<0.0001	0.2481
TC (mmol/L)	4.6 (1.1)	3.3 (0.3)	4.1 (0.2)	4.9 (0.2)	6.1 (0.7)	<0.0001	3.9597
LDL-C (mmol/L)	2.9 (1.0)	1.9 (0.4)	2.6 (0.4)	3.1 (0.5)	4.1 (0.8)	<0.0001	2.2284
HDL-C (mmol/L)	1.1 (0.3)	0.9 (0.3)	1.0 (0.3)	1.1 (0.3)	1.2 (0.3)	<0.0001	0.4838
SCr (mmol/L)	0.9 (0.8, 1.2)	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)	0.9 (0.8, 1.1)	0.9 (0.8, 1.1)	<0.0001	0.1064
URIC (umol/L)	393.5 (119.2)	395.1 (123.2)	391.5 (120.8)	393.7 (117.4)	393.9 (115.1)	0.2396	0.0154
eGFR (mL/min/ 1.73 m^2)	78.0 (25.9)	76.0 (27.8)	77.6 (25.3)	78.5 (25.0)	79.9 (25.1)	<0.0001	0.0798
CMV (mL)	121.0 (113.3)	123.7 (84.7)	122.0 (114.0)	119.1 (83.1)	119.0 (155.9)	0.0238	0.0270
Procedural and medication (%)							
IABP	591 (1.5)	193 (2.0)	139 (1.4)	118 (1.2)	141 (1.5)	0.0002	0.0305
Statins	31,627 (86.8)	7886 (86.4)	7998 (86.0)	7683 (85.3)	8060 (89.4)	<0.0001	0.0634
β blocker	27,143 (74.5)	6908 (75.7)	6904 (74.3)	6628 (73.6)	6703 (74.3)	0.0114	0.0244
ACEI/ARB	23,770 (65.2)	6078 (66.6)	6106 (65.7)	5876 (65.2)	5710 (63.3)	<0.0001	0.0358
CCB	9869 (27.1)	2530 (27.7)	2502 (26.9)	2499 (27.7)	2338 (25.9)	0.0181	0.0235
PCI	23,265 (59.7)	5714 (58.2)	5839 (59.1)	5660 (58.8)	6052 (62.9)	<0.0001	0.0493

AMI: acute myocardial infarction; CHF: congestive heart failure; PCI: percutaneous interventions; WBC: white blood cell; HGB: hemoglobin; TC: total cholesterol; LDL-C: low-density lipoprotein cholesterol; HDL-C: High-density lipoprotein cholesterol; SCr: serum creatinine; eGFR: estimated glomerular filtration rate; CMV: contrast medium volume; IABP: intra-aortic balloon pump; ACEI: angiotensin converting enzyme inhibitor; ARB: angiotensin receptor blocker; CCB: calcium channel blocker; SMD: standardized mean difference; URIC: uric acid.

3.2 CI-AKI and all-cause mortality risk analysis

CI-AKI was observed in a total of 4587 patients. During a mean 5.32-year follow-up, 6494 patients died, of which 1105 were in the CI-AKI group and 5389 were in the group without CI-AKI. Time-to-event curves showed that patients with CI-AKI experienced an increased risk of all-cause mortality compared with those without CI-AKI. Furthermore, Time-to-event curves also demonstrated that CI-AKI was significantly associated with a higher risk of death (Fig. 2).

3.3 TC and CI-AKI relationship analysis

The incidence of CI-AKI was 10.7% (n = 1050) in the Q1 group, 10.7% (n = 1055) in the Q2 group, and 12.2% (n = 1171) in the Q3 group, while the incidence significantly increased to 13.6% (n = 1311) in the Q4 group ($p < 0.0001$) (Fig. 3). Furthermore, a restricted cubic splines (RCS) model with multiple adjustments was conducted to visualise the relationship between TC and CI-AKI incidence (Fig. 1). We observed that the risk of CI-AKI increased with increasing TC levels. Associated with the risk of CI-AKI after complete adjustment for known confounders, the Q4 and Q3 groups with higher TC levels showed gradual increases in risk rates compared with the Q1 group (Q3: OR, 1.122; 95% CI, 1.024–1.229, $p = 0.014$; Q4: OR, 1.244; 95% CI, 1.137–1.362, $p < 0.001$) (Table 2).

Univariate and multivariate regression analyses were performed to determine the relationship between TC and CI-AKI. In univariate logistic regression analysis, TC was significantly associated with CI-AKI (OR, 1.121; 95% CI, 1.091–1.152, $p < 0.001$). After adjustment, Model 2 and Model 3 also showed similar results (adjusted model 2: OR, 1.112; 95% CI, 1.082–1.144, $p < 0.001$; adjusted model 3: OR, 1.103; 95% CI, 1.072–1.135, $p < 0.001$) (Table 3).

4. Discussion

To the best of our knowledge, this is the first large, multicentre study to explore the relationship between TC and the risk of CI-AKI after CAG. This work highlights TC as an independent risk factor and shows that the elevation of its levels significantly affects CI-AKI. Thus, monitoring TC could play a crucial role in preventing CI-AKI in CAG patients.

As coronary intervention technology becomes more widely used in disease management, cases of contrast-induced nephropathy are on the rise. CI-AKI is associated with prolonged renal impairment, the need for dialysis, and higher rates of all-cause mortality. Therefore, early identification of high-risk CI-AKI patients after CAG is critical for prevention and prognosis.

Risk stratification for contrast-induced AKI is essential in both clinical and research settings, prompting the creation of multiple risk assessment tools [6, 7]. A recent study on AKI after PCI revealed the following PCI key findings: the

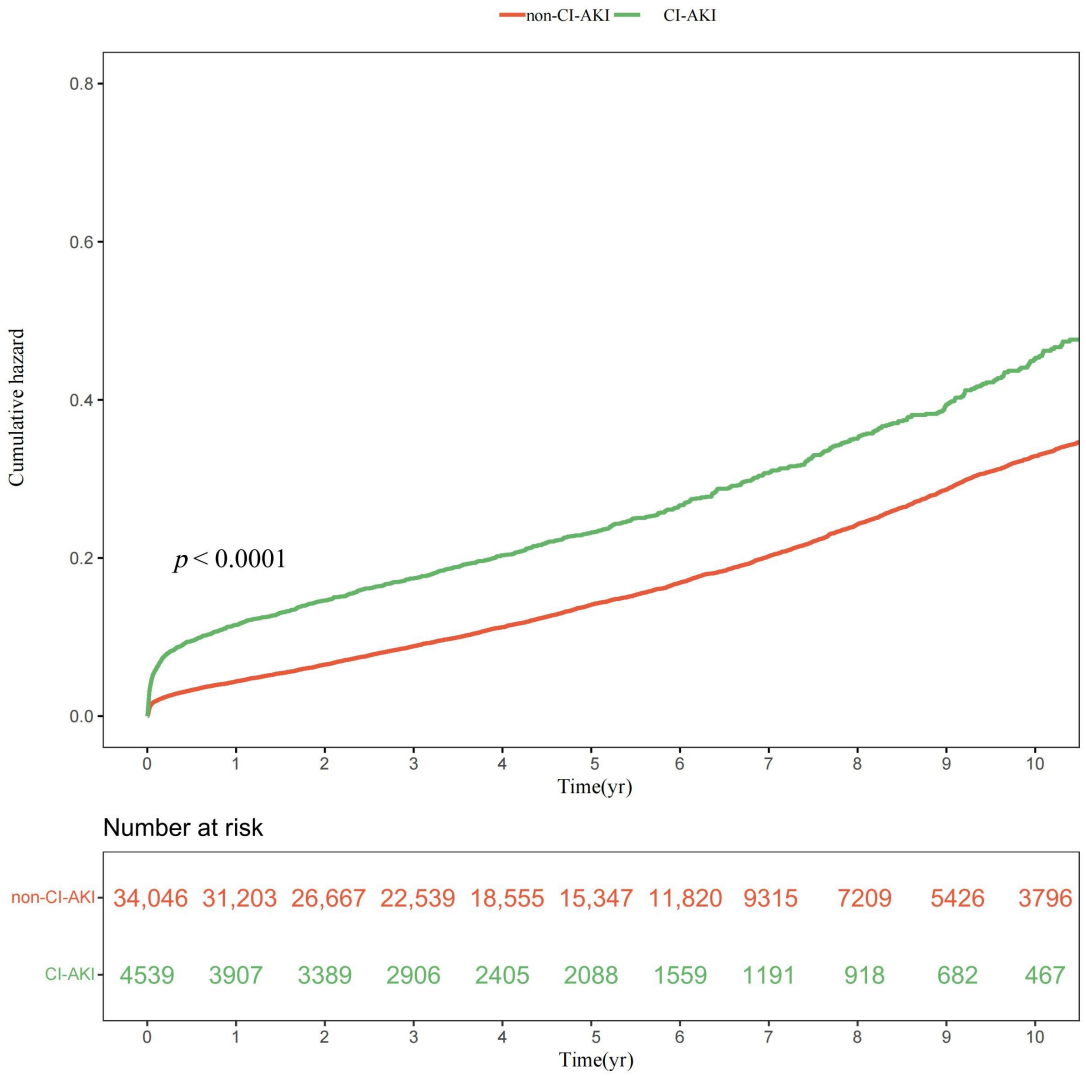

FIGURE 2. Kaplan–Meier curves of all-cause mortality. CI-AKI: contrast-induced acute kidney injury.

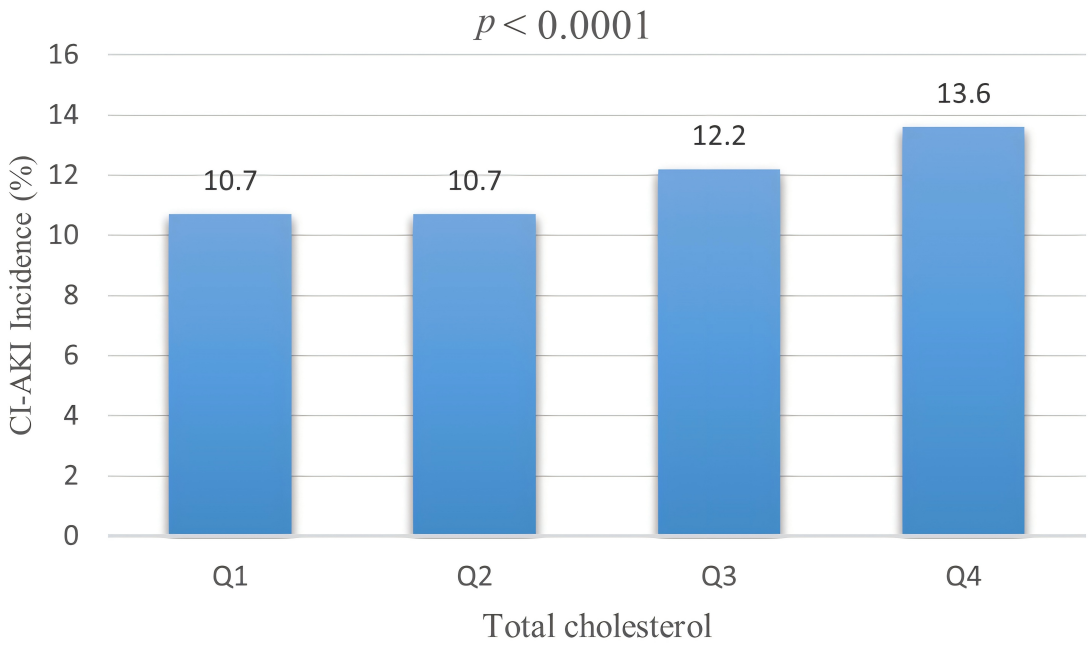

FIGURE 3. Incidence of CI-AKI in the four groups. CI-AKI: contrast-induced acute kidney injury.

TABLE 2. Multivariate logistic regression model of TC quartile level and CI-AKI.

Predictor variable	Model 1			Model 2			Model 3		
	OR	95% Confidence interval	*p*	OR	95% Confidence interval	*p*	OR	95% Confidence interval	*p*
Q1	-	-	-	-	-	-	-	-	-
Q2	0.997	0.911–1.092	0.954	0.982	0.897–1.076	0.699	0.970	0.884–1.064	0.521
Q3	1.156	1.058–1.263	0.001	1.131	1.034–1.236	0.007	1.122	1.024–1.229	0.014
Q4	1.318	1.208–1.437	<0.001	1.281	1.173–1.399	<0.001	1.244	1.137–1.362	<0.001

Model 1: unadjusted; Model 2: adjusted for Age, Male; Model 3: adjusted for Age, Male, Hypertension, Diabetes mellitus, CHF, AMI, SCr and IABP. OR: odds ratio.

TABLE 3. Multivariate logistic analysis of CI-AKI risk indicators.

Predictor variable	OR	95% Confidence interval	*p*
Model 1			
TC	1.121	1.091–1.152	<0.001
Model 2			
TC	1.112	1.082–1.144	<0.001
Age	1.009	1.007–1.012	<0.001
Male	0.724	0.677–0.774	<0.001
Model 3			
TC	1.103	1.072–1.135	<0.001
Age	1.011	1.008–1.014	<0.001
Male	0.843	0.784–0.908	<0.001
Diabetes mellitus	1.136	1.063–1.215	<0.001
Hypertension	0.903	0.845–0.964	0.002
CHF	2.103	1.951–2.265	<0.001
AMI	1.158	1.078–1.243	<0.001
SCr	0.443	0.400–0.489	<0.001
IABP	3.843	3.204–4.600	<0.001

Unadjusted and adjusted OR and 95% CIs for the primary endpoint (CI-AKI) of TC. Model 1: unadjusted; Model 2: adjusted for Age, Male, Smoke, Hypertension, Diabetes mellitus, CHF and AMI; Model 3: adjusted for Age, Male, Hypertension, Diabetes mellitus, CHF, AMI, SCr and IABP. OR: odds ratio; TC: total cholesterol; CHF: congestive heart failure; AMI: Acute myocardial infarction; SCr: serum creatinine; IABP: intra-aortic balloon pump.

incidence of AKI slightly decreased over the last decade, with significant variation depending on the diagnostic criteria used. An updated risk score incorporating eight clinical variables (Mehran risk score) was developed to predict contrast-associated AKI. The addition of procedural characteristics slightly improved the predictive accuracy of the score [8]. However, existing risk scores, which incorporate factors such as type and volume of contrast medium and comprehensive medical histories, are often impractical in clinical settings. Clinicians require simpler, more sensitive and readily accessible biomarkers to enhance the preoperative identification of CI-AKI. TC, which can be easily measured using widely available and cost-effective methods, has been independently linked to myocardial infarction and ischaemic heart disease. Despite its clinical relevance, the association between TC and CI-AKI remains underexplored, suggesting a potential area for further investigation and application in clinical practice.

Previous studies on factors associated with CI-AKI have revealed no significant differences in total cholesterol (TC) levels between patients with and without CI-AKI [9]. However, most investigations did not document TC levels in these patient groups [10–13]. In contrast, some studies have confirmed that TC levels in patients with CI-AKI are higher than those in patients without CI-AKI (194 ± 49 *vs.* 179 ± 49 mg/dL, $p = 0.006$) [14], which is similar to our results. Nevertheless, the role of TC as a risk factor for CI-AKI has not been explored.

The link between total cholesterol and the development of CI-AKI is intricate, possibly involving direct toxic effects and renal medulla ischemia and hypoxia. First, hyperosmolar contrast material causes renal ischaemia by narrowing renal vessels and reducing renal blood flow. Simultaneously, the contrast agent can also cause the shrinkage and deformation of

red blood cells in renal blood flow and increase blood viscosity, which leads to the slowing and stasis of renal blood flow, causing renal hypoxic injury. This state of ischaemia and hypoxia can further damage renal tubular epithelial cells, which can, in turn, impair renal function. In addition, hyperosmolar contrast medium can also cause renal medullary ischaemia and hypoxia, which may be due to the intense constriction of renal medullary blood vessels caused by this medium. Renal medullary ischaemia and hypoxia can also lead to damage to renal tubular epithelial cells, which further damages renal function [15, 16]. Recent studies have shown that elevated cholesterol levels may induce the activation of NOD-like receptor family pyrin domain containing 3 (NLRP3) inflammasome and subsequent secretion of interleukin-1 beta (IL-1β) and Interleukin-18 (IL-18), which aggravate kidney injury and apoptosis [17–19]. Additionally, hypercholesterolemia may facilitate the progression of kidney disease primarily through renal atherosclerosis, which can cause renal ischemia and subsequently impair renal function. Elevated cholesterol levels may also alter glomerular charge selectivity and affect the permeability of renal filtration membranes, further impacting renal function [20].

In our study, an increase in TC was associated with an increased risk of CI-AKI after CAG, especially under conditions with high TC. Therefore, clinicians should monitor serum TC levels in patients with AKI to evaluate susceptibility to CI-AKI, identify high-risk patients, and implement suitable preventive measures.

5. Limitations

This study has several limitations. First, changes in kidney function after discharge were not monitored. Second, while SCr is commonly used to define CI-AKI, it has limitations as a renal function marker. Moreover, cystatin C, a more sensitive biomarker that exhibits elevation earlier after CI-AKI than SCr does, was not evaluated, potentially leading to the incidence of CI-AKI being underestimated. Third, only a single TC measurement at admission was used, preventing the assessment of TC level changes and their impact on CI-AKI and hospital outcomes. Fourth, the precise mechanism linking TC and CI-AKI remains unclear and requires further investigation. Fifth, the study included patients over a 15-year period (2007–2021). During this time, advancements in contrast media technology, procedural techniques (*e.g.*, reduced contrast volume protocols) and evolving guidelines for CI-AKI prevention (*e.g.*, hydration strategies, statin use) may have influenced both CI-AKI incidence and risk factor associations. The analysis did not adjust for temporal trends, potentially introducing variability that could affect the generalizability of findings to contemporary clinical practice. Finally, unmeasured confounders such as contrast agent osmolality, procedural duration, and intraoperative hemodynamic fluctuations were not analysed. These factors may independently affect the risk of CI-AKI and should be addressed in future investigations. While we adjusted for statin use, variations in dosage and treatment duration may influence the association between TC levels and CI-AKI, warranting further investigation.

6. Conclusions

The findings suggest that the prognosis of CI-AKI patients is worse compared to those without CL-AKI, and an increase in TC levels may be significantly associated with a higher risk of CI-AKI in patients undergoing CAG. TC has emerged as an independent risk factor for CI-AKI. These results underscore the clinical relevance of TC as a modifiable risk factor, making serum TC a valuable clinical test for assessing the risk of CI-AKI and adverse hospital outcomes before CAG.

AVAILABILITY OF DATA AND MATERIALS

The datasets generated and/or analyzed during this study are accessible from the corresponding author upon reasonable request.

AUTHOR CONTRIBUTIONS

CYW—was responsible for writing and statistics. JL—reviewed and revised the article. XZL and HZH—provided suggestions for the article. YBH—also offered suggestions. JLW and JMC—were in charge of data collection. MIK—was responsible for writing. YL, LX and JYC—reviewed and revised the article.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval for this study was obtained from the Guangdong Provincial People's Hospital Ethics Committee (Approval No. GDREC2019-555H-2), and all collaborating institutions secured independent approvals from their local ethics review boards. The research adhered to the Declaration of Helsinki, and all data were anonymised before use. As the study involved retrospective cases without additional interventions and all patient information was desensitised, informed consent was not required. The requirement for informed consent was waived by Guangdong Provincial People's Hospital, Shenzhen People's Hospital, Yangjiang People's Hospital, Maoming People's Hospital, and Longyan People's Hospital.

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CONFLICT OF INTEREST

The author declares no conflict of interest.

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