Journal of Inflammation Research

Dovepress
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ORIGINAL RESEARCH

Fibrinogen/Albumin Ratio is Associated with the Occurrence of Contrast-Induced Acute Kidney Injury in Patients with Congestive Heart Failure

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Purpose: Patients with congestive heart failure (CHF) are associated with an elevated risk of mortality and poor prognosis. Contrast-induced acute kidney injury (CI-AKI), a common complication in CHF patients undergoing contrast-enhanced procedures, exacerbates renal dysfunction and contributes to adverse outcomes. However, the relationship between the preoperative fibrinogen/albumin ratio (FAR) and the risk of CI-AKI or all-cause mortality in CHF remains unclear. This study analyzed the correlation of FAR with the risk of CI-AKI and all-cause mortality in patients with CHF.

Patients and Methods: In this retrospective observational study, CHF patients undergoing coronary angiography (CAG) were enrolled and grouped according to their FAR quartiles. The association between FAR and clinical outcomes was assessed using the multivariate logistic regression and restricted cubic spline (RCS) analyses.

Results: This study included 7,235 CHF patients with a mean age of 65.8 ± 11.7 years. Among these, 2,100 were female (29.0%), and 1,094 (15.1%) experienced CI-AKI. FAR showed a non-linear relationship with CI-AKI ($p < 0.001$). The risk of CI-AKI was significantly higher with increasing FAR. After adjusting for all the potential confounding variables, the risk of CI-AKI was highest in patients with FAR >0.150 (OR = 1.572, 95% CI 1.237–2.004, $p < 0.001$). Multivariate COX proportional risk model showed that the risk of all-cause mortality was highest in CHF patients with FAR > 0.150 (HR = 1.20, 95% CI 1.04–1.38, $p = 0.014$).

Conclusion: FAR is an independent risk factor for the occurrence of CI-AKI in patients with CHF.

Keywords: fibrinogen/albumin ratio, FAR, contrast-induced acute kidney injury, CI-AKI, congestive heart failure, CHF, biomarker, prognosis

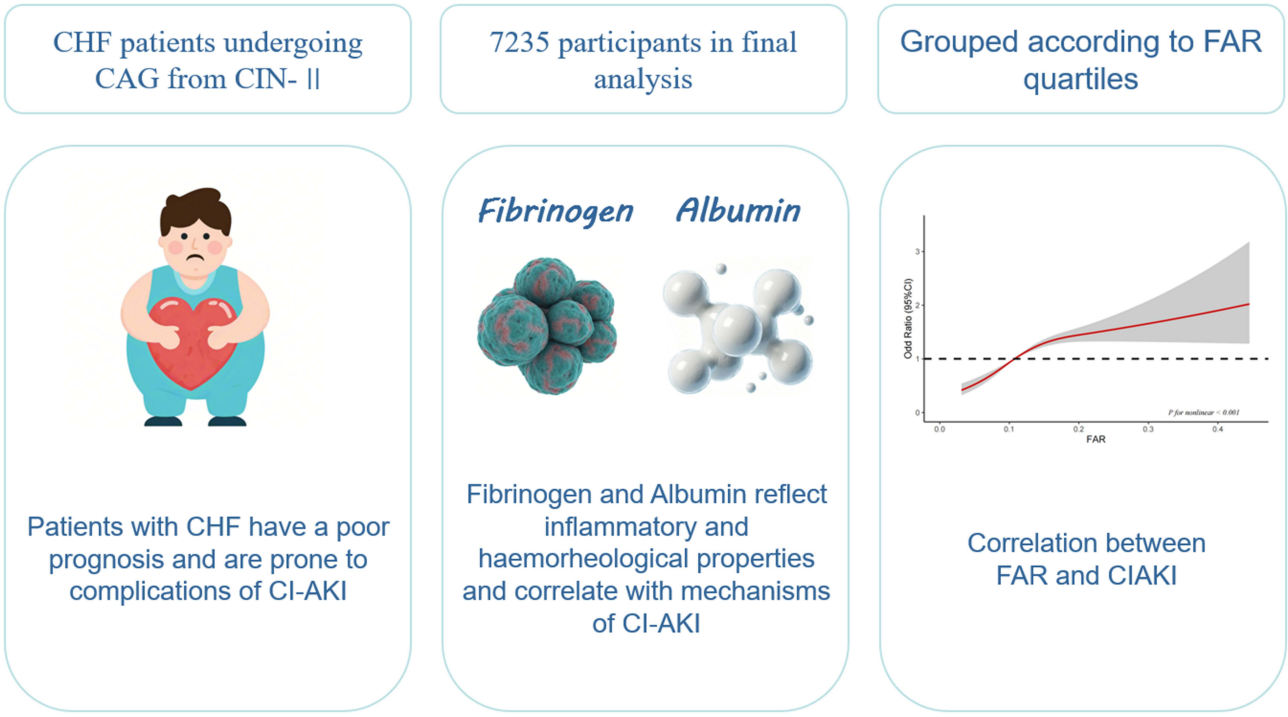
Introduction

Patients with congestive heart failure (CHF) are associated with high mortality rates and poor prognosis.[1] Coronary angiography (CAG) is a valuable diagnostic tool for assessing the underlying cause of CHF but is associated with a high risk of contrast-induced kidney injury (CI-AKI), which leads to adverse outcomes such as chronic kidney disease, heart disease, stroke, and even death, thereby significantly impacting their survival outcomes.[2]

Mehran score is the most common CI-AKI risk assessment tool for evaluating eight risk factors such as hypotension, CHF, anemia, diabetes mellitus, advanced age, renal insufficiency, iodinated contrast dosage, and intra-aortic balloon pump (IABP) use.[3–6] Inflammation also plays a key role in the development of CI-AKI.[7] Severe inflammation causes microcirculatory disturbances, thereby reducing renal blood flow and oxygen supply, and increasing the risk of CI-AKI.[8] Systemic inflammation is a common pathological feature of acute and chronic heart failure.[9] The Canakinumab Anti-inflammatory Thrombosis Outcomes Study (CANTOS) trial results demonstrated that inflammation was a key player in the pathogenesis of heart failure.[10]

Received: 4 December 2024
Accepted: 10 April 2025
Published: 17 April 2025

Graphical Abstract



Previous studies have demonstrated that CI-AKI is closely associated with the systemic immune-inflammation index, high-sensitive C-reactive protein (hs-CRP), procalcitonin and other inflammatory indicators.[11–15] Although these factors have significant predictive value in assessing CI-AKI, they are costly and difficult to obtain and disseminate clinically. Therefore, there is an urgent need to identify simple inflammatory biomarkers for predicting CI-AKI with high accuracy. The fibrinogen/albumin ratio (FAR) has emerged as a promising biomarker of inflammatory response. Previous research studies have demonstrated an association between FAR and CI-AKI. In patients with non-ST elevation acute coronary syndrome (NSTE-ACS) who have undergone drug-eluting stent (DES) implantation, elevated preoperative FAR is strongly linked with the subsequent development of CI-AKI and has a higher predictive value than the Mehran score (AUC 0.702 vs 0.645).[16] Patients undergoing elective percutaneous coronary intervention (PCI) have also shown similar results.[17]

The relationship between FAR and CI-AKI remains unclear in patients with CHF. Therefore, this study assessed the correlation of FAR with the risk of CI-AKI and all-cause mortality in patients with CHF.

Materials and Methods

Study Population

This study used data from the Cardiorenal Improvement Nt II (CIN-II, NCT05050877) trial, a large multicenter study performed at five large tertiary hospitals. The CIN-II study data included information on hospitalizations of patients who underwent CAG from 2007 to 2020. This study included CHF patients classified as New York Heart Association class > II or Killip class > I.[18] The exclusion criteria were as follows: (1) patients under the age of 18 years; (2) patients with malignant tumors; (3) patients with sepsis; (4) patients with cirrhosis and liver failure; and (5) patients with missing or abnormal preoperative or postoperative serum creatinine (SCr) and FAR data. Finally, 7235 patients were included in this study (Figure 1).

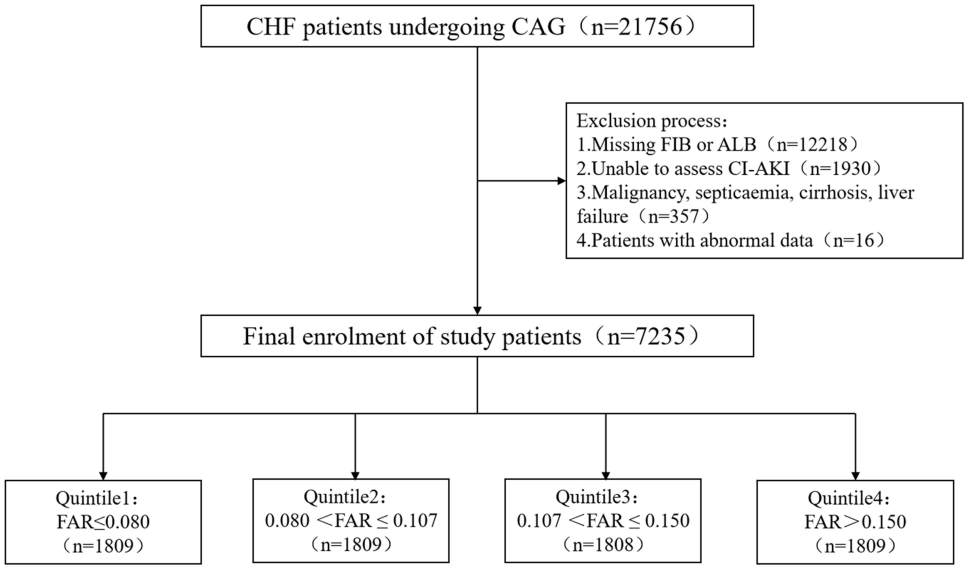


Figure 1 Study flowchart.

Data Collection

The clinical data of the included patients was extracted from the electronic clinical management system (ECMS) of each participating hospital. The baseline data included demographic characteristics, laboratory test results, and history of comorbidities and medication use. The blood samples at baseline were collected early in the morning from all patients. The baseline SCr value was measured at hospital admission and compared with the highest value observed within a 48-h period following the CAG procedure. FAR was defined as the ratio of fibrinogen levels to albumin levels. The estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[19] The Mehran score was calculated for each patient individually and was based on the risk factors outlined in the Mehran risk score model described previously.[6]

Outcomes and Definitions

The primary endpoint was CI-AKI, and the secondary endpoint was all-cause mortality. CI-AKI was defined as a rise in SCr of ≥0.3 mg/dl (≥26.5 μmol/L) or ≥1.5 times the baseline within 48–72 h of iodine contrast administration according to the 2012 Kidney Disease: Improving Global Outcomes (KDIGO) guidelines.[20] Hypertension was defined as a systolic blood pressure of 140 mmHg or greater and/or a diastolic blood pressure of 90 mmHg or greater.[21] Diabetes mellitus was defined as the recent use of insulin or antidiabetic medication, a fasting blood glucose value exceeding 7.0 mmol/L, a random blood glucose value exceeding 11.1 mmol/L, or glycated hemoglobin exceeding 6.5%.[22] Chronic kidney disease (CKD) was defined as eGFR <60 mL/min/1.73 m^2. Hypotension was defined as a systolic blood pressure of less than 80 mmHg for a minimum of one hour that necessitated pharmacologic or arterial intra-arterial positive inotropic support. Anemia was defined as a baseline hematocrit level of less than 39% in males and less than 36% in females. IABP is defined as the use of intra-aortic balloon pump support 24 h prior to CAG.[6]

Statistical Analysis

The patients were classified into four groups based on their FAR levels. Descriptive statistics were expressed as mean ± standard deviation (SD), median, or quartiles, whereas categorical variables were expressed as ratios. Normally distributed descriptive data were compared using the independent samples *t*-test or ANOVA. Non-normally distributed data were analyzed using the Kruskal–Wallis test. Categorical variables were compared between groups using the chi-square test. Restricted cubic spline analysis (RCS) was used to investigate the relationship between FAR and the incidence of CI-AKI. The risk factors included in the Mehran score were incorporated into a multifactorial logistic

regression model and analyzed. The following 3 multivariate regression models were evaluated: (1) model 1 was adjusted for age and gender; (2) model 2 was adjusted for IABP, diabetes mellitus, eGFR and anemia in addition to factors adjusted in model 1; and (3) model 3 was adjusted for contrast dosage in addition to factors adjusted in model 2. Subsequently, the adjusted odds ratios (OR) and 95% confidence intervals (CI) were calculated. A multifactorial COX proportional risk model was used to analyze the relationship between FAR and the incidence of all-cause mortality. The results were expressed as risk ratios (HR) and their 95% CI. Cumulative incidence of CI-AKI and all-cause mortality was calculated using the Kaplan-Meier plots and the Log rank test. The receiver operating characteristic (ROC) curves and corresponding areas under the curve (AUC) values were used to compare the predictive values of FAR and other related metrics for CI-AKI. The statistical analysis was performed using the R software (version 4.3.4). All the statistical tests were based on a two-sided alternative hypothesis and a P value of less than 0.05 was considered as statistically significant.

Results

Baseline Characteristics

The baseline characteristics of the study population, grouped by the FAR quartile levels, are presented in Table 1. This study included 7,235 patients with CHF. Among these, 2100 (29.0%) were females and the remaining 5135 (71%) were males. The mean age of the study population was 65.8 ± 11.7 years. Furthermore, we identified 3807 (52.6%) patients

Table 1 Baseline Characteristics of the Study Subjects

Characteristics	All (n=7235)	FAR Quartiles				P value
		Q1 (n=1809)	Q2 (n=1809)	Q3 (n=1808)	Q4 (n=1809)	
Age (year)	65.8 (11.7)	64.4 (12.5)	65.5 (11.7)	66.9 (11.5)	66.3 (11.1)	<0.001
Female, n (%)	2100 (29.0)	550 (30.4)	590 (32.6)	543 (30.0)	417 (23.1)	<0.001
Smoke, n (%)	1972 (37.9)	429 (36.7)	458 (36.1)	492 (36.4)	593 (41.8)	0.006
Medical history, n (%)						
Atrial fibrillation	1098 (15.2)	361 (20.0)	330 (18.2)	231 (12.8)	176 (9.7)	<0.001
Hypertension	3807 (52.6)	825 (45.6)	908 (50.2)	1018 (56.3)	1056 (58.4)	<0.001
Diabetes Mellitus	2943 (40.7)	566 (31.3)	661 (36.5)	782 (43.3)	934 (51.6)	<0.001
Chronic Kidney Disease	2979 (41.2)	488 (27.0)	658 (36.4)	845 (46.7)	988 (54.6)	<0.001
Stroke	635 (8.8)	113 (6.2)	164 (9.1)	167 (9.2)	191 (10.6)	<0.001
Laboratory parameters						
HGB (g/L)	128.4 (21.5)	135.7 (19.3)	132.1 (20.2)	126.6 (20.4)	119.3 (22.5)	<0.001
Neutrophil (10^9/L)	6.9 (4.0)	6.7 (4.2)	6.4 (3.8)	6.7 (3.7)	8.0 (4.1)	<0.001
Lymphocyte (10^9/L)	1.7 (0.8)	1.8 (0.9)	1.7 (0.8)	1.6 (0.7)	1.5 (0.7)	<0.001
Monocyte (10^9/L)	0.7 (0.4)	0.6 (0.3)	0.6 (0.3)	0.7 (0.3)	0.9 (0.4)	<0.001
SCr (μmol/L)	1.4 (1.3)	1.1 (0.7)	1.3 (1.1)	1.4 (1.3)	1.8 (1.7)	<0.001
Fibrinogen (g/L)	4.2 (1.6)	2.6 (0.5)	3.5 (0.4)	4.4 (0.6)	6.3 (1.3)	<0.001
ALB (g/L)	35.8 (5.4)	40.1 (4.3)	37.5 (4.0)	35.1 (3.9)	30.7 (4.4)	<0.001
eGFR	66.2 (28.1)	75.5 (23.4)	68.8 (26.8)	63.6 (28.2)	56.9 (30.3)	<0.001
Medication use, n (%)						
ACEI/ARB	4081 (62.3)	984 (61.3)	1017 (62.7)	1044 (62.7)	1036 (62.4)	0.848
Spironolactone	3476 (53.0)	852 (53.1)	912 (56.2)	845 (50.7)	867 (52.2)	0.014
β-Blockers	4920 (75.1)	1174 (73.2)	1195 (73.6)	1261 (75.7)	1290 (77.7)	0.011
Statins	5471 (83.5)	1327 (82.7)	1285 (79.2)	1380 (82.8)	1479 (89.0)	<0.001
SGLT-2	53 (0.8)	16 (1.0)	12 (0.7)	15 (0.9)	10 (0.6)	0.6
Procedural characteristics						
PCI	4693 (64.9)	1008 (55.7)	1049 (58.0)	1196 (66.2)	1440 (79.6)	<0.001
Mehran Score	13.7 (4.4)	11.7 (3.7)	12.5 (3.9)	13.9 (4.3)	15.1 (4.6)	<0.001

Abbreviations: HGB, Hemoglobin; SCr, serum creatinine; SGLT2, sodium-dependent glucose transporters 2; PCI, percutaneous coronary intervention.

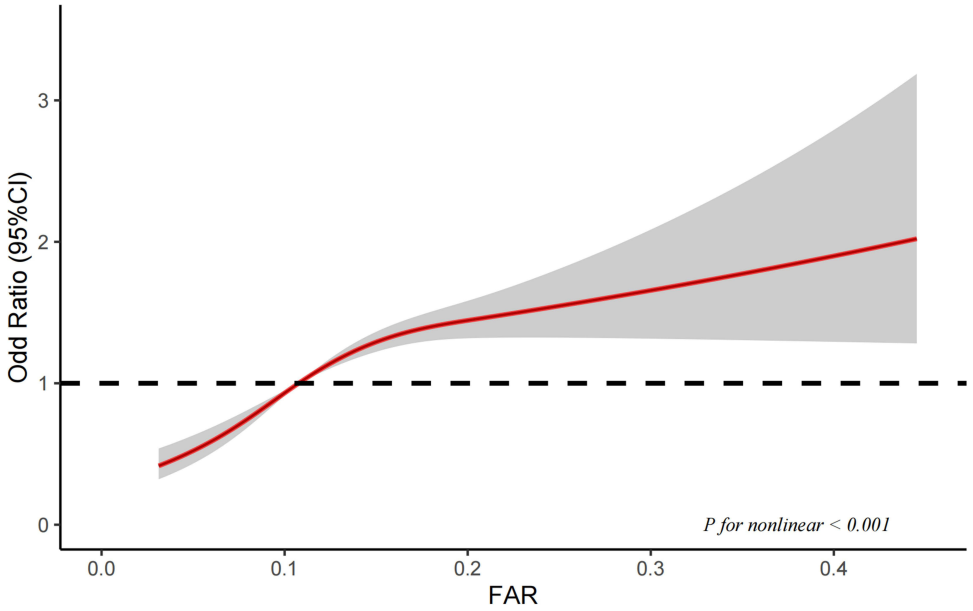


Figure 2 RCS curves for the correlation between FAR and CI-AKI.

with hypertension, 2943 (40.7%) patients with diabetes mellitus, 2979 (41.2%) patients with CKD, 635 (8.8%) patients with a history of stroke, and 1098 (15.2%) patients with atrial fibrillation (AF). Patients with a higher FAR demonstrated increased rates of hypertension, diabetes mellitus, CKD, stroke, elevated baseline SCr levels, increased rates of PCI, and high Mehran scores. Conversely, a lower proportion of patients with a higher FAR showed a previous history of AF and lower eGFR and hemoglobin (HGB) levels at admission.

Correlation Between FAR and CI-AKI in CHF Patients

During this study, 1,094 CHF patients (15.1%) who underwent CAG developed CI-AKI. The RCS curves for the risk of CI-AKI across different quartiles of FAR are shown in Figure 2. The RCS curve analysis demonstrated a nonlinear relationship between FAR and CI-AKI after adjusting for confounding variables ($p < 0.001$), and patients with a higher FAR were associated with an increased risk of CI-AKI.

A multifactorial logistic regression model was used to evaluate the independent correlation between FAR and CI-AKI in patients with CHF. Patients in the 4th quartile of FAR exhibited the highest risk of CI-AKI after adjusting for all the relevant variables (OR = 1.572, 95% CI 1.237–2.004, $p < 0.001$). In model 3, the risk of CI-AKI was significantly higher for patients in Q2 versus patients in Q1 (OR = 1.406, 95% CI 1.108–1.791, p=0.005) but the risk of CI-AKI was comparable between patients in Q3 and Q1 (p=0.069) (Table 2). ROC curve analysis results showed that the AUC values of FAR, fibrinogen, albumin, neutrophil/lymphocyte ratio (NLR), and monocyte/lymphocyte ratio (MLR) for predicting

Table 2 Multivariate Logistic Regression Analyses of the Relationship Between Different FAR Quartiles and CI-AKI

FAR	Model 1		Model 2		Model 3	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Q1 (<0.080)	–	–	–	–	–	–
Q2 (0.080–0.107)	1.646 (1.341–2.025)	<0.001	1.450 (1.176–1.791)	0.001	1.406 (1.108–1.791)	0.005
Q3 (0.107–0.150)	1.827 (1.493–2.241)	<0.001	1.404 (1.139–1.736)	0.002	1.251 (0.984–1.595)	0.069
Q4 (>0.150)	2.554 (2.103–3.114)	<0.001	1.627 (1.318–2.013)	<0.001	1.572 (1.237–2.004)	<0.001

Notes: Model 1: adjusted for age and gender.Model 2: adjusted for IABP, diabetes, eGFR, anemia, and factors adjusted in Model 1.Model 3: adjusted for contrast dosage and factors adjusted in Model 2.
Abbreviation: FAR, Fibrinogen/albumin ratio.

https://doi.org/10.2147/JIR.S507160

Table 3 Relationship Between FAR Quartiles and All-Cause Mortality of CHF Patients

FAR	Unadjusted		Adjusted*	
	HR (95% CI)	P value	HR (95% CI)	P value
Q1 (<0.080)	-	-	-	-
Q2 (0.080–0.107)	1.19 (1.03–1.37)	<0.001	1.06 (0.91–1.22)	0.455
Q3 (0.107–0.150)	1.54 (1.34–1.76)	<0.001	1.19 (1.04–1.38)	0.013
Q4 (>0.150)	1.70 (1.49–1.95)	<0.001	1.20 (1.04–1.38)	0.014

Notes:: *Adjusted for age, gender, IABP, diabetes, eGFR and anemia.

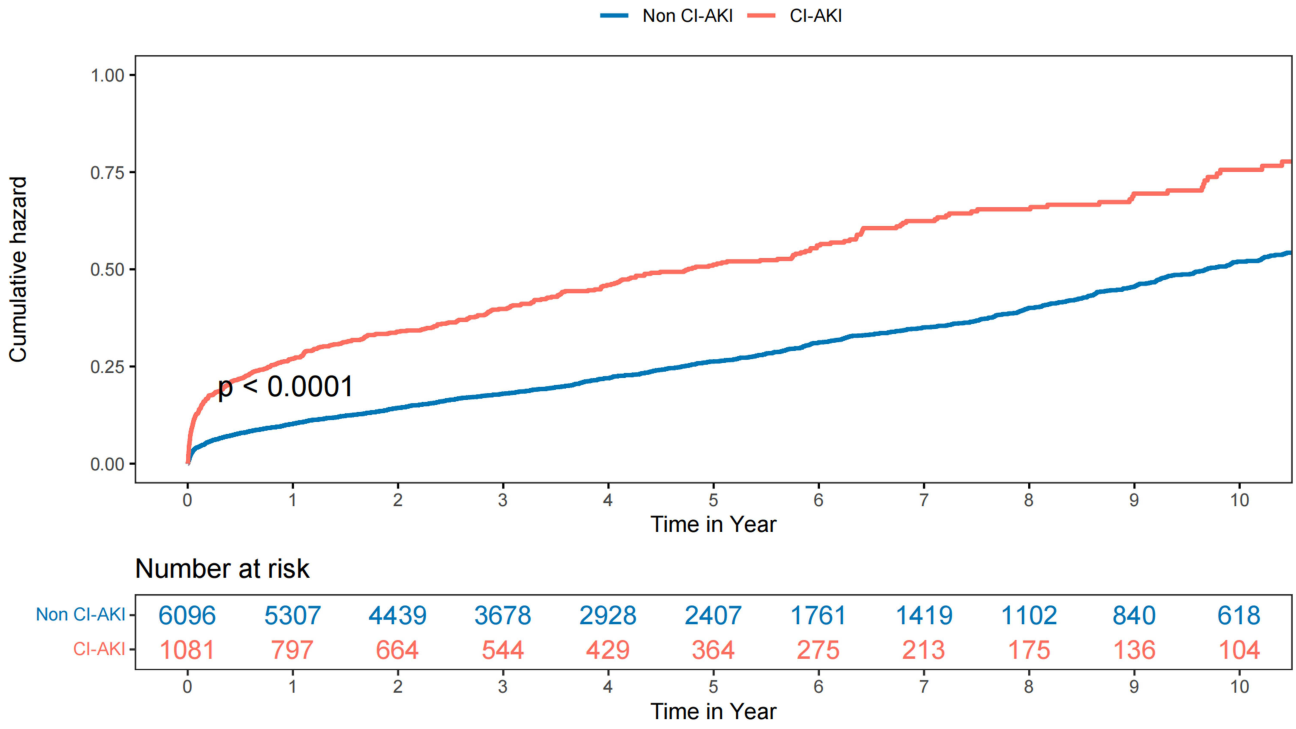


Figure 3 Kaplan-Meier curves show the cumulative risk of all-cause mortality in study subjects.

CI-AKI were 0.589, 0.578, 0.578, 0.566, and 0.587, respectively (Supplementary Figure 1). The cut-off value of FAR on admission to predict CI-AKI in CHF population was 0.090, with 77.2% sensitivity and 36.4% specificity.

Association of FAR and CI-AKI on All-Cause Mortality in CHF Patients

During a median follow-up of 3.71 years, 2011 (28.0%) participants died. The association between FAR and all-cause mortality in different FAR subgroups based on the COX proportional risk model is shown in Table 3. The data was adjusted for age, gender, IABP, diabetes, eGFR, and anemia. The risk of all-cause mortality was 20% higher for CHF patients with FAR >0.150 (Q4 group) compared to patients with FAR <0.080 (Q1 group) (95% CI 1.04–1.38; P = 0.014). Kaplan-Meier curves demonstrated that CHF patients with CI-AKI exhibited a significantly higher risk of all-cause mortality compared to those without CI-AKI (Figure 3**; Log rank test P value <0.0001)**.

Discussion

This study investigated the correlation between FAR and CI-AKI in patients with CHF. Our data showed that the prevalence of CI-AKI was 15.1% among CHF patients who underwent CAG. Furthermore, we demonstrated an independent and nonlinear positive correlation between FAR and the risk of CI-AKI in the CHF patients. The Cox

proportional risk model showed that FAR was an independent predictor of all-cause mortality. The risk of all-cause mortality was highest among CHF patients with a FAR > 0.150. Furthermore, Kaplan-Meier survival curves showed that the long-term all-cause mortality rates were higher for the CHF patients who developed CI-AKI compared to those who did not develop CI-AKI.

Fibrinogen, a key player in the coagulation cascade, also regulates inflammatory response by promoting leukocyte transmigration and enhancing the function of leukocyte effectors within damaged tissues.[23,24] Fibrinogen levels are significantly elevated during inflammation. Furthermore, activation of endothelial cells by fibrinogen enhances the duration of vascular inflammation and promotes platelet aggregation and vascular leakage.[25] Furthermore, fibrinogen stimulates the expression of proinflammatory cytokines such as interleukin-1β (IL-1 β) and tumor necrosis factor-α (TNF- α) from mononuclear cells such as monocytes.[26] This demonstrates that fibrinogen is a promising marker of inflammation. These data also suggested that inflammation played a key role in the pathogenesis of CI-AKI.[27] Consequently, fibrinogen is a reliable marker to estimate the risk of CI-AKI. Fibrinogen, a long-lasting plasma acute-phase reactant, also induces endothelial dysfunction.[28] Elevated levels of fibrinogen affect blood viscosity, leading to endothelial shear stress damage, impaired microcirculatory blood flow, inadequate tissue perfusion, and hypoxic conditions in the renal medulla.[29,30] Fibrinogen causes significant renal fibrosis and dysfunction by stimulating the proliferation of renal fibroblasts.[31] The onset of acute kidney injury significantly increases renal fibrinogen levels.[32] Furthermore, urinary fibrinogen levels were significantly elevated at two hours after angiography.[33] Elevated fibrinogen levels are an independent risk factor for CI-AKI in patients with acute coronary syndrome undergoing PCI.[34]

Albumin is the most abundant protein in human plasma. It accounts for about 50% of total plasma proteins and plays a significant role in ligand binding, transport of substances, prevention of fluid extravasation, protection of capillary membranes, anticoagulation, and anti-platelet aggregation.[35] Oxidative stress plays a key role in contrast-induced kidney injury. Elevated levels of free oxygen radicals exacerbate the vasoconstrictive effects of contrast media and leads to aggravation of renal ischemia.[36] Furthermore, elevated generation of reactive oxygen species because of renal ischemia increases the vasoconstrictive effects of contrast media, thereby leading to a vicious cycle.[37] Serum albumin demonstrates significant antioxidant properties and has been shown to scavenge more than 70% of oxygen radicals in the blood plasma.[38] At physiological concentrations, albumin selectively inhibited TNF-α-induced expression of vascular cell adhesion molecule 1 (VCAM1), monocyte adhesion, and activation of human endothelial cell nuclear factor κB (NF-kB).[39] Therefore, albumin acts as an anti-inflammatory protein. Furthermore, serum albumin levels are negatively correlated with inflammatory response because the albumin levels are reduced when the inflammatory burden in the body increases. During the inflammatory response, albumin biosynthesis is inhibited by pro-inflammatory mediators such as interleukin-6, interleukin-1 and tumor necrosis factor; moreover, inflammation promotes catabolism of albumin, thereby further decreasing its levels.[40,41] Albumin is a potent inhibitor of platelet activation and aggregation. Low albumin levels inhibit the physiological fibrinolytic system. This reduces the spontaneous lysis of thrombi and enhances the progression of thrombotic disease.[42] Furthermore, the bioavailability of prostacyclin (PGI2), a potent inhibitor of platelet aggregation, is regulated by albumin levels, and may cause microcirculatory disturbances.[43] Reduced albumin levels alter blood viscosity and impair endothelial function.[44] Albumin suppressed inflammation-induced endothelial cell apoptosis by inhibiting the expression of cell adhesion molecules. However, low levels of albumin trigger endothelial dysfunction by selectively inhibiting TNF α-induced expression of VCAM1 in the human aortic endothelial cells.[39,45] Furthermore, albumin levels indicate the nutritional status of the organism. Low levels of albumin indicate malnutrition in human patients. Hypoproteinemia is highly prevalent in patients with heart failure and is associated with an increased risk of cardiovascular diseases, including coronary artery disease and heart failure, and poorer prognosis of these patients.[46,47] For example, Karki et al reported that in-hospital mortality was two-fold higher in heart failure patients with hypoalbuminemia than in those without hypoalbuminemia.[48]

Cugno et al demonstrated that chronic heart failure lead to a hypercoagulable state, which subsequently played an important role in disease progression.[49] Furthermore, Iglesias et al reported that the thrombotic-inflammatory phenotype was significantly associated with CHF and was an independent risk factor of short-term mortality.[50] In CHF patients, hyperactivation of the renin-angiotensin-aldosterone system (RAAS) and the sympathetic nervous system (SNS) are compensatory mechanisms to maintain circulatory homeostasis. However, they significantly increase circulating blood

volume through sodium and water retention and reduce plasma albumin concentration.[51] During this process, overexpression of bioactive molecules can adversely affect heart function and blood circulation. This leads to further activation of the inflammatory signaling pathways, which increase or decrease the levels of fibrinogen and albumin, which are positive and negative acute phase response proteins, respectively.[52]

The pathogenesis of CI-AKI remains uncertain. Inflammation, oxidative stress, direct tubular toxicity, endothelial dysfunction, and decreased renal blood flow are potential contributing factors for CI-AKI.[53] Fibrinogen and albumin are two proteins that possess both inflammatory and hemorheological properties and are also involved in biological pathways that regulate development of CI-AKI. Albumin inhibits the activity of fibrinogen, thereby hindering its CI-AKI-promoting functions.[54] Therefore, FAR is a useful risk index that integrates both fibrinogen and albumin and provides a more sensitive and robust estimation of the inflammatory state, blood viscosity, and thrombogenicity.[17]

FAR is estimated according to the plasma fibrinogen and albumin levels, and is strongly associated with cardiovascular events. FAR correlated with the severity of coronary artery lesions in patients with stable coronary artery disease[55] and in patients with ST-segment elevation myocardial infarction (STEMI).[56] FAR also shows significant predictive value in patients with NSTE-ACS and enhances risk stratification.[57] Furthermore, FAR was a potential prognostic indicator for patients with NSTE-ACS who underwent PCI.[58] Xu et al reported that FAR was an independent risk factor for 90-day all-cause mortality, 1-year all-cause mortality, and length of hospital stay in patients with heart failure.[59] Yang et al conducted a 750-day median follow-up of 916 patients with CHF and found that FAR was an independent predictor of all-cause death in CHF patients, regardless of the heart failure subtype.[51] Faruk et al demonstrated that FAR was a significant predictor of contrast nephropathy (CIN) development in patients undergoing carotid arteriography.[60] You et al analyzed the clinical data of 565 patients who underwent PCI in an emergency setting and demonstrated a strong correlation between pre-procedural FAR and the incidence of CI-AKI.[61] However, to the best of our knowledge, none of the studies so far have focused on the relationship between preoperative FAR and CI-AKI in patients with CHF.

This study validates the predictive value of FAR for CI-AKI in patients with CHF. Chronic inflammation, microcirculatory dysfunction, and malnutrition (common in heart failure) may synergistically increase the susceptibility to contrast-induced renal injury. FAR not only reflects systemic inflammation (via elevated fibrinogen), but also indirectly indicates the presence of oxidative stress and endothelial dysfunction associated with hypoalbuminemia. This dual mechanism highlights the unique predictive advantage of FAR in this population. Therefore, addition of FAR may provide a more comprehensive approach to CI-AKI risk stratification.

This study has several limitations. Firstly, this study was a retrospective study that may have inherent bias. Therefore, large-scale prospective studies are necessary to confirm our results. Secondly, this study did not evaluate indicators of blood viscosity and well-defined biomarkers of CI-AKI such as cystatin C, NGAL, and kidney injury molecule 1 (KIM-1). Thirdly, this study did not analyze dynamic changes in FAR. Therefore, further studies are required to validate the impact of dynamic alterations in FAR on the long-term renal function in CHF patients.

Conclusion

Our study demonstrated that FAR was an independent risk factor for CI-AKI in patients with CHF undergoing CAG. Therefore, prior assessment of FAR can assist clinicians to identify the high-risk patients and implement management strategies to prevent CI-AKI.

Data Sharing Statement

The data presented in this study are derived from a registry study that was conducted at the Guangdong Provincial People's Hospital. The original data is not accessible to the public because of stringent patient privacy and ethical protection requirements. However, upon reasonable request, the corresponding author can provide a comprehensive overview of the study methodology, including pooled analysis results in the form of statistical tables, graphs, and charts.

Ethics Statement

The Ethics Committee of the Guangdong Provincial People's Hospital approved this study (Approval no. GDREC2019-555H-2). This study was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent

was not required as this was a retrospective study. All the patient information was extracted from the ECMS, and private patient information was hidden.

Acknowledgments

We thank Jiyan Chen, Director of the CIN-II program, and all the patients who participated in this study.

Author Contributions

All authors significantly contributed towards study conception, study design, execution, acquisition of data, analysis and interpretation. All authors took part in drafting, revising, or critically reviewing the article. All authors approved the final draft for publication and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that they have no known competing commercial interests or personal relationships that could have influenced the research findings reported in this paper.

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