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ORIGINAL RESEARCH

Predictive Significance of the Monocyte-to-High-Density Lipoprotein Cholesterol Ratio in Post-Percutaneous Coronary Intervention Contrast-Induced Nephropathy Among Patients with Coronary Artery Disease

Aijie Lin[1,*], Yanchun Peng[2,*], Jiayi Shao[1], Liangwan Chen[3], Yanjuan Lin[2,4]

[1]School of Nursing, Fujian Medical University, Fuzhou, Fujian, People's Republic of China; [2]Department of Nursing, Fujian Medical University Union Hospital, Fuzhou, Fujian, People's Republic of China; [3]Department of Cardiac Surgery, Fujian Medical University Union Hospital, Fuzhou, Fujian, People's Republic of China; [4]Department of Cardiac Surgery Nursing, Fujian Medical University Union Hospital, Fuzhou, Fujian, People's Republic of China

*These authors contributed equally to this work

Correspondence: Liangwan Chen, Department of Cardiac Surgery, Fujian Medical University Union Hospital, No. 29 Xinquan Road, Fuzhou, Fujian, 350001, People's Republic of China, Email fjxhlwc@163.com; Yanjuan Lin, Department of Nursing, Fujian Medical University Union Hospital, No. 29 Xinquan Road, Fuzhou, Fujian, 350001, People's Republic of China, Email fjxhyjl@163.com

Purpose: This study aimed to examine the connection between monocyte-to-high-density lipoprotein cholesterol ratio (MHR) and contrast-induced nephropathy (CIN) and to introduce a novel perspective to early clinical diagnosis of CIN.

Patients and Methods: This single-center bidirectional cohort study included 1771 patients with coronary artery disease (CAD) who underwent elective percutaneous coronary intervention (PCI) from January 2017 to July 2022 at the Fujian Medical University Union Hospital. The study collected data retrospectively through an electronic medical record system with postoperative follow-up. The primary endpoint was CIN, and secondary endpoints were mortality and readmission within two years after PCI, along with total days of hospitalization. Patients were divided into three groups based on preoperative MHR (Group 1: MHR <0.42; Group 2: MHR ≥0.42 and <0.64; Group 3: MHR ≥0.64). The study used regression analyses to investigate the relationship between MHR and CIN rate and postoperative mortality, and demonstrated the cumulative incidence of postoperative fatal events in the three groups of patients by Kaplan-Meier curves, and also analyzed the predictive value of MHR and other inflammatory indicators on the risk of CIN by using receiver operating characteristic (ROC) curves.

Results: Compared to Group 1, Groups 2 and 3 had a higher incidence of CIN, higher mortality, and longer hospital stays (P <0.001). Survival analysis showed significant differences in long-term mortality among the three groups (Log rank test, P = 0.032). Multivariate regression analysis showed that for every 1-unit increase in MHR, the risk of CIN increased 8.567-fold (95% CI: 4.291–17.102, P <0.001) and the risk of death increased 3.080-fold (95% CI: 1.423–6.666, P = 0.004). ROC analysis showed that MHR had a good predictive ability for CIN (AUC: 0.713, 95% CI: 0.666–0.759, P <0.001).

Conclusion: MHR is significantly linked to CIN and 2-year mortality following PCI in CAD patients, serving as a CIN risk assessment tool to assist clinics in early identification of high-risk patients and optimization of treatment strategies.

Keywords: monocyte-to-high-density lipoprotein cholesterol ratio, inflammation, contrast-induced nephropathy, coronary artery disease, cardiovascular diseases, percutaneous coronary intervention

Introduction

From 1990 to 2019, the global incidence of cardiovascular diseases increased from 270 million to 520 million, with coronary artery disease (CAD) ranking as the second most prevalent condition.[1] Percutaneous coronary intervention

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(PCI) is the predominant therapeutic approach for CAD. In 2022, 1.376 million PCI procedures were conducted in China, markedly enhancing the quality of life of those with CAD.[2,3] The PCI treatment process necessitates the administration of contrast agent to visualize the lesion area. Contrast-induced nephropathy (CIN), a prevalent complication following PCI, has an incidence rate ranging from 3.3% to 13.3%,[4–6] can raise the risk of major adverse cardiovascular events (MACE) by 1.8 times, augment mortality by over 10 times,[7] and result in more healthcare expenditures.[8] The prompt identification of risk factors linked to CIN and the prevention of its onset are essential for improving patient outcomes post-PCI.

The pathogenesis of CIN remains incompletely understood; however, research indicates that it is primarily associated with oxidative stress and inflammatory responses.[9] Recent research findings have established a significant correlation between inflammatory indicators derived from clinical laboratory parameters, including the neutrophil-to-lymphocyte ratio (NLR), neutrophil-to-high-density lipoprotein cholesterol ratio (NHR), and platelet-to-lymphocyte ratio (PLR), with CIN and other inflammation-related diseases.[10–12] From the levels of monocytes and high-density lipoprotein cholesterol (HDL-C) in routine blood tests, a new inflammatory indicator called the monocyte-to-high-density lipoprotein cholesterol ratio (MHR) is created. This ratio indicates the body's pro-inflammatory and anti-inflammatory equilibrium.[13] MHR has been used to forecast adverse outcomes, including all-cause mortality and MACE, and is associated with arterial plaque formation and the severity of atherosclerosis due to the accessibility and cost-effectiveness of the data.[10,14–16] Recent findings suggest MHR may be an important predictor of CIN after PCI. A study involving 647 patients with acute coronary syndromes (ACS) identified preoperative MHR level as an independent risk factor for CIN, elevating the risk of CIN by a factor of 1.09.[13] Similar results were noted in the study of Sağ.[17] However, the above articles focused on myocardial infarction (MI) patients. They had relatively small sample sizes, and the studies' follow-up period was limited to the hospitalization period to 6 months post-procedure, which did not adequately reflect the potential of MHR to forecast CIN and other long-term prognostic outcomes after PCI. Most current research focuses solely on a single sign and does not directly compare the prognostic usefulness of MHR with other inflammation indicators. Consequently, augmenting the sample size and incorporating non-MI CAD patients is imperative to examine the relationship between MHR and post-PCI CIN comprehensively. Furthermore, comparing MHR with other inflammatory indicators will elucidate whether it possesses more excellent predictive capability for CIN.

In conclusion, this study sought to expand the inclusion of sample size to assess the predictive capacity of MHR for CIN following PCI in patients with CAD. By comparing the diagnostic efficacy of MHR with inflammatory markers such as NLR, NHR and PLR, it was determined to explore whether MHR has significant advantages in the prediction of CIN, providing new insights and methods for the early clinical diagnosis and prevention of CIN.

Materials and Methods

Study Participants

This was a single-center bidirectional cohort study, encompassing patients with CAD aged 18 years or older who attended the Fujian Medical University Union Hospital for elective PCI between January 2017 and July 2022 to construct a dynamic cohort. MHR was calculated using monocyte versus HDL-C levels measured at admission. Based on the tertiles of MHR, patients were divided into three groups (Group 1: MHR <0.42; Group 2: MHR ≥0.42, <0.64; Group 3: ≥0.64). Exclusion criteria include: (1) severe hepatic and renal insufficiency, with severe hepatic insufficiency defined as serum alanine aminotransferase >2 times the upper limit of the reference value, and severe renal insufficiency defined as an estimated glomerular filtration rate (eGFR) of <15 mL/min/1.73 m^2 or the need to undergo prolonged dialysis; (2) hyperthyroidism, severe infection, concurrent malignancy, immune system diseases, or hematological disorders (combined with moderate-to-severe anemia with hemoglobin [Hb] <90 g/L, leucocytosis or leukopenia [peripheral blood leucocyte count consistently above 10.0×10^9/L or consistently below 4.0×10^9/L], thrombocytosis or thrombocytopenia [platelet count consistently above 300×10^9/L or consistently below 100×10^9/L]); (3) exposure to or allergy to contrast agent within 7 days prior to PCI; (4) administration of nephrotoxic drugs within 48 hours before the procedure; (5) absence of any data from a complete blood count or preoperative or postoperative serum creatinine (SCr) within 48 hours following the procedure; and (6) patients receiving specific preoperative prophylactic interventions (e.g., hydration therapy or the need to take non-steroidal mineralocorticoid receptor antagonists [ns-MRAs]). An entire group of 1,771

patients were ultimately incorporated into the study. The approval was obtained from the Medical Ethics Committee of Fujian Medical University Union Hospital (2023KY032). All enrolled patients volunteered and provided informed consent.

Data Collection

Patients' demographics, interventions, lab results, and length of stay in the hospital were retrieved from their electronic medical records. The blood collection method for determining biochemical indicators in this study involved instructing patients to fast after 8 PM on the night of admission. Each patient was required to fast for at least 10 hours before blood samples were taken, which were taken the following morning between 6 and 8 AM. At least two seasoned interventional cardiologists oversaw each patient's care while they were in the hospital, and they followed all applicable protocols for PCI and perioperative management.

Endpoints and Follow-Up

The primary endpoint of the study was CIN, which was defined as a rise of SCr of 0.3 mg/dL or more or 50% within 48 hours after PCI, according to the Acute Kidney Injury Network.[18] Secondary endpoints were mortality and readmission within two years after PCI, along with total days of hospitalization. The study constructed a retrospective cohort upfront to collect the occurrence of CIN through an electronic medical record system. Postoperative deaths and readmissions were collected by nurses with a license to practice and master's degree nursing students through outpatient and telephone follow-up visits every four months after PCI. Follow-up time was calculated from the starting point of observation to death, loss of visit, or the termination of two years postoperatively.

Definitions

Smoking was defined as the daily intake of more than one cigarette, sustained for a period surpassing six months.[19] Drinking was described as the consumption of at least one alcoholic beverage per week, with a total intake of 50 mL or more, maintained for a duration surpassing six months.[20] Repeated in-hospital blood pressure readings of 140 mmHg or higher for systolic and 90 mmHg or higher for diastolic, or the use of anti-hypertensive medication in conjunction with self-reported hypertension, were considered to be hypertension.[21] Diabetes mellitus (DM) was characterized by asymptomatic individuals exhibiting fasting blood glucose (FBG) levels of ≥7.0 mmol/L on multiple occasions or postprandial blood glucose levels of ≥11.1 mmol/L two hours after an oral glucose tolerance test; alternatively, glycated hemoglobin levels of ≥6.5%; or the presence of typical hyperglycemic symptoms or a hyperglycemic crisis with a random blood glucose ≥11.1 mmol/L; or a prior diagnosis of DM necessitating the use of oral antidiabetic medications or insulin.[22] Hyperlipidemia is identified when cholesterol levels meet the following criteria: total cholesterol (TC) ≥6.21 mmol/L, triglycerides (TG) ≥2.26 mmol/L, low-density lipoprotein cholesterol (LDL-C) ≥4.14 mmol/L, or HDL-C <1.04 mmol/L.[23] The definition of anemia was a Hb level below 130 g/L in adult males and 120 g/L in adult females.[24] ICD-10 criteria were used to define CAD, chronic kidney disease (CKD), acute myocardial infarction (AMI), heart failure (HF), atrial fibrillation (AF), and stroke.[25]

The formulas for calculating the various inflammatory indicators involved in this study are as follows:

1. MHR = monocyte count (10^9/L) / HDL-C (mmol/L).
2. NLR = neutrophil count (10^9/L) / lymphocyte count (10^9/L).
3. NHR = neutrophil count (10^9/L) / HDL-C (mmol/L).
4. PLR = platelet count (10^9/L) / lymphocyte count (10^9/L).

Statistical Analysis

Continuous variables in patients' baseline data were presented as mean ± standard deviation (for normal distribution) or median and interquartile range (for skewed distribution). In contrast, categorical variables were represented as counts and percentages. For normally distributed data, we used the ANOVA test; for skewed data, we used the Mann–Whitney *U*-test; and for categorical variables, we used either the chi-square test or Fisher's exact test. These statistical methods were applied to identify differences among the three groups. The factors associated with CIN risk were investigated using logistic regression

analysis. Age, white blood cell (WBC), left ventricular ejection fraction (LVEF), platelet (PLT), and SCr were corrected in Model I to remove potential confounding variables. Model II corrected for Model I with the number of stents placed, contrast dose, and infarcted vessel status. Model III adjusts for CKD, DM, and AMI based on Model II. We evaluated the diagnostic efficacy of four inflammatory indicators, including MHR, using receiver operating characteristic (ROC) curves and subsequently analyzed the predictive power of MHR for CIN compared to other inflammatory indicators. The study also used Cox proportional hazards regression analysis and expressed the association between preoperative MHR and death within two years after PCI by hazard ratio (HR) and 95% confidence interval (CI). Model I was adjusted for age, LVEF, and SCr. Model II was corrected for the number of stents placed and infarcted vessels. Model III adjusted CKD and DM based on Model II. The Kaplan-Meier and Log rank tests were used for survival analysis on three groups. The study used SPSS 27.0 (SPSS Inc., Chicago, IL, USA) for data analysis and GraphPad Prism 8.3 (GraphPad Software, CA, USA) to visualize the results. A significance level of P <0.05 was considered to be different.

Results

Baseline Characteristics of the Study Population

The inclusion of the study population is shown in Figure 1. The medical records of 1,950 patients with CAD who underwent elective PCI from January 2017 to July 2022 were reviewed. Based on the excluded criteria, 153 patients were omitted from the study. Additionally, 90 patients were lost to follow-up throughout the two years, leaving a final analysis of 1,771 patients (93.9%).

The median MHR for all participants was 0.51 (0.36, 0.69). Table 1 delineates the baseline features of patients in each group, categorized by their MHR levels. The average age was 63.7 ± 0.2 years, with 357 (20.2%) female participants. Compared to Group 1, which exhibited lower MHR levels, Groups 2 and 3 comprised older patients, a higher proportion of males, elevated BMI, a more significant number of individuals who smoked and drank, and lower LVEF (P <0.05). In addition, the combination of CKD, hyperlipidemia, AMI, and HF was more significant in Group 2 and Group 3 (P <0.05). Regarding inflammatory indicators, Group 2 and Group 3, with higher MHR, had higher NLR and NHR and lower PLR. The three groups were more evenly matched in terms of drug use and contrast dose (P >0.05).

Differences in Clinical Outcomes of Patients in Different MHR Level Groups

In this study, 113 patients developed CIN in the postoperative period (6.4%), and 69 patients died during the two-year follow-up (3.9%), with a mean length of hospitalization of 8.0 ± 0.1 days and 818 readmissions (46.2%) (Table 2).

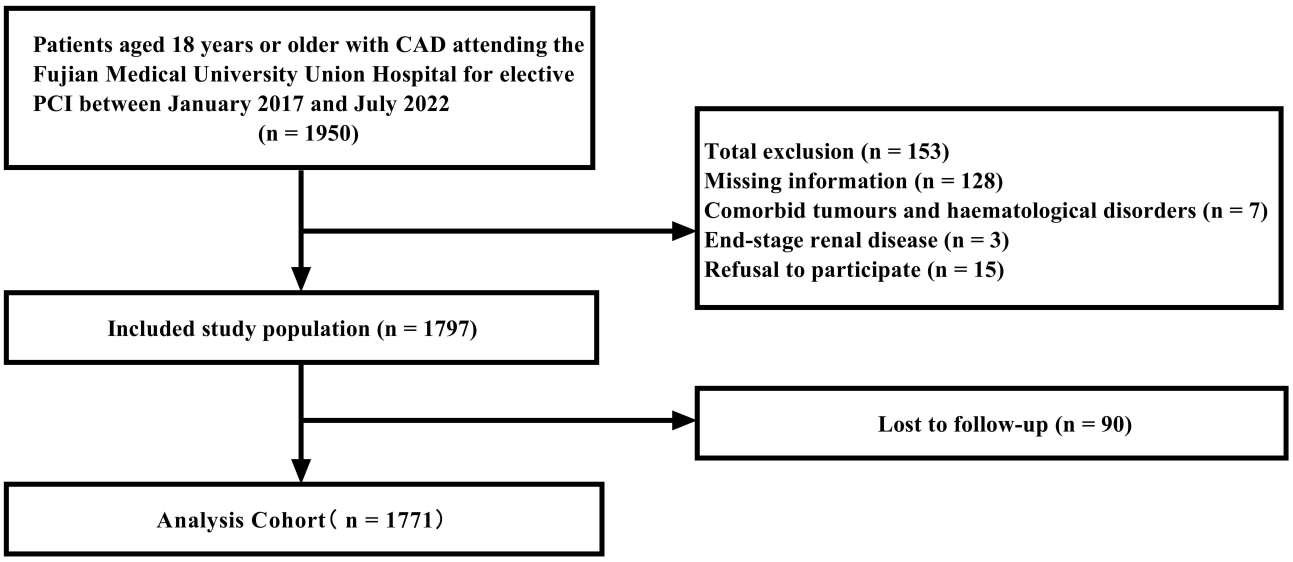

Figure 1 Patients included in the study.
Abbreviations: CAD, coronary artery disease; PCI, percutaneous coronary intervention.

Table 1 Baseline Characteristics of Patients

Variables	Group 1 (n = 604)	Group 2 (n = 597)	Group 3 (n = 570)	*P* value
Demographics				
Age (years)	65.2 ± 0.4	64.5 ± 0.4	61.3 ± 0.4	<0.001*
Male	408 (67.5%)	490 (82.1%)	516 (90.5%)	<0.001*
BMI (kg/m^2)	23.53 (21.63, 25.46)	23.74 (22.13, 26.94)	23.70 (22.32, 25.17)	0.049*
SBP, mean ± SD (mmHg)	133.33 ± 0.78	131.52 ± 0.78	127.47 ± 0.84	<0.001*
Smoking	171 (28.3%)	222 (37.2%)	305 (53.5%)	<0.001*
Drinking	37 (6.1%)	48 (8.0%)	60 (10.5%)	0.023*
Comorbidities (n%)				
Hypertension	396 (65.6%)	391 (65.5%)	350 (61.4%)	0.239
DM	216 (35.8%)	232 (38.9%)	234 (41.1%)	0.173
CKD	24 (4.0%)	42 (7.0%)	72 (12.6%)	<0.001*
Hyperlipidemia	191 (31.6%)	238 (39.90%)	267 (46.8%)	<0.001*
Anemia	42 (7.0%)	61 (10.2%)	54 (9.5%)	0.114
AF	39 (6.5%)	37 (6.2%)	44 (7.7%)	0.544
AMI	185 (30.6%)	271 (45.4%)	370 (64.9%)	<0.001*
Stroke	63 (10.4%)	54 (9.0%)	64 (11.2%)	0.459
HF	8 (1.3%)	10 (1.7%)	19 (3.3%)	0.038*
Medication				
Diuretics	94 (15.6%)	93 (15.6%)	92 (16.1%)	0.954
CCB	162 (26.8%)	155 (26.0%)	144 (25.3%)	0.830
ACEI/ARB/ARNI	200 (33.1%)	208 (34.8%)	185 (32.5%)	0.670
β-blockers	327 (54.1%)	320 (53.6%)	311 (54.6%)	0.947
Stains	500 (82.8%)	496 (83.1%)	457 (80.2%)	0.366
Antiplatelet	522 (86.4%)	497 (83.2%)	489 (85.8%)	0.264
Procedure performed				
Contrast dose (g)	80.32 ± 1.75	79.62 ± 1.50	81.43 ± 1.34	0.711
Number of stents	1.45 ± 0.03	1.62 ± 0.04	1.66 ± 0.04	<0.001*
Culprit artery				
RCA	513 (84.9%)	507 (84.9%)	503 (88.2%)	0.171
LMCA	86 (14.3%)	119 (20.4%)	321 (18.1%)	0.009*
LAD	569 (94.2%)	564 (94.5%)	537 (94.2%)	0.975
LCX	425 (70.4%)	457 (76.5%)	444 (77.9%)	0.006*
Number of infarcted vessels	2.63 ± 0.03	2.76 ± 0.03	2.81 ± 0.03	<0.001*
Laboratory examination				
WBC (× 10^9/L)	5.93 (5.04, 6.94)	7.10 (8.47, 6.05)	9.03 (7.48, 11.25)	<0.001*
PLT (× 10^9/L)	201.00 (180.25, 238.75)	222.00 (190.00, 261.00)	222.50 (185.00, 266.25)	<0.001*
Hb (g/L)	135.00 (124.00, 146.00)	137.00 (125.00, 147.00)	136.00 (123.00, 146.00)	0.390
TG (mmol/L)	1.23 (0.92, 1.74)	1.46 (1.08, 2.07)	1.65 (1.18, 2.43)	<0.001*
TC (mmol/L)	4.30 (3.52, 5.19)	4.06 (3.36, 5.02)	4.12 (3.34, 4.92)	<0.001*
HDL-C (mmol/L)	1.19 (1.03, 1.35)	0.96 (0.85, 1.09)	0.83 (0.72, 0.96)	<0.001*
LDL-C (mmol/L)	2.72 (2.02, 3.62)	2.59 (1.98, 3.50)	2.64 (1.99, 3.42)	0.219
FBG (mmol/L)	5.40 (4.87, 6.52)	5.44 (4.80, 6.88)	5.76 (4.90, 7.50)	0.002*
SCr (mg/dL)	0.85 (0.74, 0.97)	0.89 (0.76,1.05)	0.93 (0.80,1.11)	<0.001*
LVEF (%)	64.7 (58.9, 70.5)	60.3 (52.3, 68.2)	55.3(48.5, 64.2)	<0.001*
NLR	2.25 (1.60, 3.06)	2.37 (1.76, 3.52)	3.17 (2.11, 4.60)	<0.001*
NHR	3.02 (2.25, 3.82)	4.55 (3.74, 5.68)	6.99 (5.47, 9.15)	<0.001*
PLR	124.78 (99.25, 150.67)	123.39 (97.89, 153.25)	118.94 (93.74, 146.25)	0.089

Notes: *P<0.05.

Abbreviations: ACEIs, angiotensin-converting enzyme inhibitor; AF, atrial fibrillation; AMI, acute myocardial infarction; ARBs, angiotensin receptor blockers; ARNI, angiotensin receptor neprilysin inhibitor; BMI, body mass index; CCB, calcium channel blocker; CKD, chronic kidney disease; DM, diabetes mellitus; FBG, fasting blood glucose; Hb, hemoglobin; HDL-C, high-density lipoprotein cholesterol; HF, heart failure; LAD, left anterior descending; LCX, left circumflex; LDL-C, low-density lipoprotein cholesterol; LMCA, left main coronary artery; LVEF, left ventricular ejection fraction; NHR, neutrophil-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; PLT, platelet; RCA, right coronary artery; SBP, systolic blood pressure; SCr, serum creatinine; SD, standard deviation; TC, total cholesterol; TG, triglyceride; WBC, white blood cell.

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Table 2 Clinical Outcomes Among the Three Groups

Variables	Group 1 (n = 604)	Group 2 (n = 597)	Group 3 (n = 570)	*P* value
CIN	11 (1.8%)	38 (6.4%)	64 (11.2%)	<0.001*
Mortality	17 (2.8%)	20 (3.4%)	32 (5.6%)	0.032*
Length of hospitalization	7.2 ± 0.2	7.8 ± 0.2	9.0 ± 0.3	<0.001*
Readmission	288 (47.7%)	276 (46.2%)	254 (44.6%)	0.563

Notes: *P<0.05.
Abbreviation: CIN, contrast-induced nephropathy.

Median MHR levels were elevated in patients who experienced CIN or mortality following PCI, showing a significant difference from those who did not (0.49 vs 0.67, P <0.001; 0.50 vs 0.62, P = 0.024) (Figure 2). Group 2 and Group 3 exhibited higher rates of CIN incidence, mortality, and extended hospital stays than Group 1 (P <0.001). All three groups had similar readmission rates (P = 0.563) (Table 2). The long-term mortality rates varied significantly among the groups, as seen by the Kaplan-Meier survival analysis curves (Log rank test, P = 0.032) (Figure 3).

The Predictive Capacity of MHR for Post-PCI CIN and Death Within Two Years of Follow-Up in CAD Patients

To identify potential causes of CIN following PCI, the research employed univariate logistic regression analysis (Table 3). We constructed three logistic regression models to investigate MHR's predictive power further. In Model I, following adjustments for demographic variables and laboratory markers, the risk of CIN dramatically escalated with each 1-unit rise in MHR, exhibiting an approximate 7.97-fold increase (OR: 7.966, 95% CI: 4.070–15.591, P <0.001). After controlling for confounding variables and certain co-morbidities in Model II, Model III found similar results (OR: 8.567, 95% CI: 4.291–17.102, P <0.001). In all three models, the risk of CIN in Groups 2 and 3 patients exceeded that of Group 1 by more than two times. The study examined the association between MHR and mortality within two years post-PCI through Cox proportional hazards regression analysis. After correction, all three models revealed a significant trend of increased mortality risk associated with higher MHR levels. Patients in Group 3 had more than a two-fold risk of death two years after surgery compared with Group 1. However, no disparity in mortality risk was observed in Group 2 (Table 4).

Comparison with Inflammatory Indicators

Figure 4 shows the ROC curve analysis for predicting CIN. Among the four inflammatory indicators, the AUCs of MHR, NLR, and NHR were above 0.7 (0.713, 0.705, and 0.727, respectively), suggesting a good predictive value for CIN. We found no statistically significant differences between the three AUCs (P >0.05). MHR had a higher sensitivity (83.2%) but lower specificity (51.7%) for predicting CIN compared to the others (Table 5).

Discussion

This study evaluated the ability to predict MHR for CIN and mortality within two years post-PCI in CAD patients. It also compared its value for prediction against other inflammatory indicators. This area has not been extensively researched before. The findings suggested that patients presenting elevated MHR levels were at an increased risk of developing CIN following PCI. This association remains even after adjusting for potential confounders. Significantly higher mortality within two years after interventional treatment was also observed in the study in both groups of patients with higher MHR levels. In ROC curves analysis, MHR displayed a moderate predictive ability for CIN relative to other inflammatory indicators. This indicates the significance of MHR in the prognostic evaluation of CAD patients receiving elective PCI.

In interventional cardiology, the prevalence of contrast agent has rendered CIN a clinically important issue. It is the third leading cause of acute kidney injury (AKI) that occurs in hospitals[26] and is strongly associated with negative short- and long-term clinical outcomes.[13,17] Active prevention and risk prediction are still the mainstays since there are no suitable clinical treatments to lower the occurrence of CIN. While many factors contribute to CIN's complicated

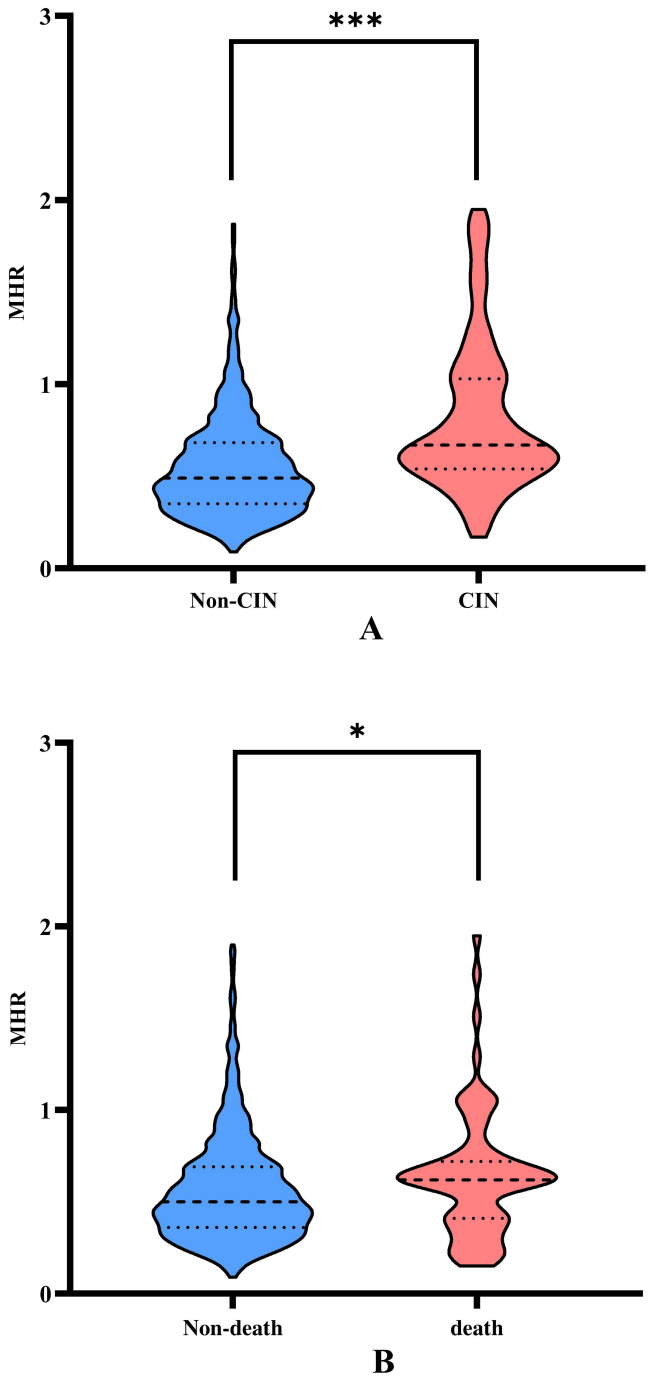

Figure 2 Relationship between clinical outcomes and MHR.
Notes: (**A**) Comparison of preoperative MHR levels in patients who developed CIN versus those who did not develop CIN. (**B**) Comparison of preoperative MHR levels in dead and non-dead patients. *P <0.05, ***P <0.001.
Abbreviations: MHR, monocyte-to-high-density lipoprotein cholesterol ratio.

pathophysiology, mounting evidence points to the inflammatory response as one of the critical culprits. Pro-inflammatory cytokines and inflammation are closely related. Research conducted by Kwasa et al indicated that patients exhibiting elevated CRP levels (indicative of an inflammatory state) faced an increased relative risk of developing CIN, implying a possible association between inflammation and CIN.[27] The observed situation may result from elevated reactive oxygen species caused by osmotic stress and nephrotoxicity from the contrast agent, alongside endogenous molecules released by injured renal tubular epithelial cells that activate the kidney's resident phagocytic receptor, initiating an inflammatory cascade that ultimately leads to renal injury.[28–30] Current research trends emphasize the identification of potential

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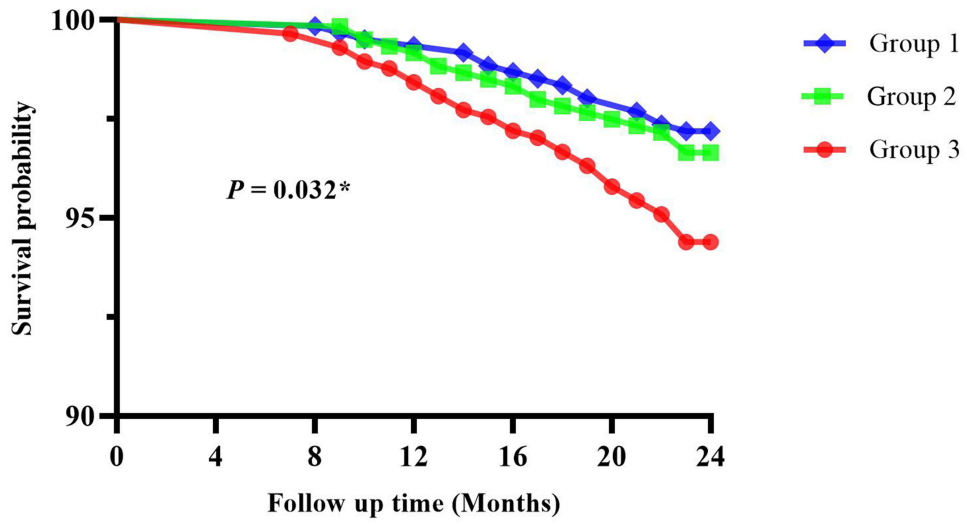

Figure 3 Mortality between patients in three groups.
Notes: *P <0.05.

biomarkers for predicting and diagnosing the onset of CIN, with specific markers linked to the inflammatory response; for instance, baseline levels of interleukin-18 and tumor necrosis factor-α serve as the most reliable predictors of CIN progression.[31] Nonetheless, identifying these markers is both time-intensive and expensive, indicating a continued necessity to investigate more efficient and dependable predictive instruments to inform clinical practice.

MHR represents the ratio of monocytes to HDL-C and serves as a novel biomarker of the interplay between inflammation and lipid metabolism in the body. This metric robustly predicts all types of adverse outcomes post PCI.[10,16] The findings of this study indicate that MHR levels in patients with CAD are positively correlated with the risk of developing CIN post-PCI and

Table 3 Univariate Analysis of Predictors of CIN and Death Within Two years of PCI in Patients

Variables	CIN		Mortality	
	OR (95% CI)	*P* value	HR (95% CI)	*P* value
Age (years)	1.067 (1.044, 1.090)	<0.001*	1.159 (1.123, 1.196)	<0.001*
Male	1.356 (0.808, 2.277)	0.248	0.905 (0.511, 1.604)	0.905
BMI (kg/m^2)	1.021 (0.958, 1.089)	0.519	0.951 (0.881, 1.027)	0.200
SBP, (mean ± SD)	0.996 (0.986, 1.006)	0.400	0.998 (0.984, 1.009)	0.565
Smoking	1.387 (0.946, 2.034)	0.093	0.664 (0.398, 1.109)	0.118
Drinking	1.095 (0.559, 2.146)	0.791	1.068 (0.462, 2.467)	0.878
CKD	3.006 (1.805, 5.006)	<0.001*	4.034 (2.333, 6.976)	<0.001*
DM	2.198 (1.495, 3.231)	<0.001*	1.750 (1.091, 2.806)	0.020*
Hyperlipidemia	0.908 (0.612, 1.347)	0.632	0.764 (0.463, 1.261)	0.293
AMI	1.942 (1.313, 2.874)	<0.001*	1.501 (0.933, 2.416)	0.094
HF	2.352 (0.899, 6.159)	0.081	2.189 (0.688, 6.962)	0.184
Number of stents	1.199 (0.985, 1.461)	0.071	1.270 (1.010, 1.597)	0.041*
Number of infarcted vessels	1.187 (0.929, 1.517)	0.170	1.742 (1.252, 2.426)	0.001*
Contrast dose	1.005 (1.001, 1.009)	0.009*	1.003 (0.998, 1.007)	0.243
WBC	1.157 (1.102, 1.215)	<0.001*	1.029 (0.956, 1.108)	0.448
PLT	1.003 (1.001, 1.006)	0.015*	0.996 (0.992, 1.000)	0.050
TG	1.006 (0.929, 1.088)	0.887	0.796 (0.605, 1.047)	0.103
TC	1.012 (0.879, 1.166)	0.879	0.851 (0.693, 1.045)	0.124

(*Continued*)

Table 3 (Continued).

Variables	CIN		Mortality	
	OR (95% CI)	*P* value	HR (95% CI)	*P* value
HDL-C	0.305 (0.137, 0.677)	0.004*	0.978 (0.422, 2.269)	0.959
FBG	1.039 (0.981, 1.101)	0.187	1.038 (0.971, 1.110)	0.269
SCr	2.177 (1.667, 2.843)	<0.001*	1.367 (1.125, 1.660)	0.002*
LVEF	0.962 (0.948, 0.976)	<0.001*	0.973 (0.955, 0.991)	0.003*
MHR	7.978 (4.875, 13.056)	<0.001*	2.124 (1.085, 4.158)	0.028*
NLR	1.240 (1.151, 1.336)	<0.001*	1.043 (0.946, 1.149)	0.398
NHR	1.193 (1.138, 1.251)	<0.001*	1.016 (0.943, 1.095)	0.671
PLR	1.003 (1.001, 1.006)	0.006*	0.999 (0.995, 1.004)	0.816

Notes: *P <0.05.

Abbreviations: AMI, acute myocardial infarction; BMI, body mass index; CI, confidence interval; CIN, contrast-induced nephropathy; CKD, chronic kidney disease; DM, diabetes mellitus; FBG, fasting blood glucose; Hb, hemoglobin; HDL-C, high-density lipoprotein cholesterol; HF, heart failure; HR, hazard ratio; LVEF, left ventricular ejection fraction; NHR, neutrophil-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; OR, odds ratio; PCI, percutaneous coronary intervention; PLR, platelet-to-lymphocyte ratio; PLT, platelet; SBP, systolic blood pressure; SCr, serum creatinine; SD, standard deviation; TC, total cholesterol; TG, triglyceride; WBC, white blood cell.

Table 4 Multivariate Analysis for Predictors of CIN and Death Within Two years of PCI in Patients

	CIN		Mortality	
	OR (95% CI)	*P* value	HR (95% CI)	*P* value
Model I				
MHR (per unit increase)	7.966 (4.070, 15.591)	<0.001*	3.943 (1.869, 8.317)	<0.001*
Tertile 1	Ref		Ref	
Tertile 2	2.901 (1.397, 6.022)	0.004*	0.897 (0.452, 1.780)	0.756
Tertile 3	3.773 (1.647, 8.640)	0.002*	2.270 (1.221, 4.220)	0.010*
P for trend		<0.001*		<0.001*
Model II				
MHR (per unit increase)	8.203 (4.169, 16.139)	<0.001*	3.554 (1.663, 7.593)	0.001*
Tertile 1	Ref		Ref	
Tertile 2	2.912 (1.392, 6.092)	0.005*	0.858 (0.425, 1.732)	0.669
Tertile 3	3.795 (1.641, 8.777)	0.002*	2.316 (1.242, 4.320)	0.008*
P for trend		<0.001*		0.002*
Model III				
MHR (per unit increase)	8.567 (4.291, 17.102)	<0.001*	3.080 (1.423, 6.666)	0.004*
Tertile 1	Ref		Ref	
Tertile 2	2.909 (1.381, 6.129)	0.005*	0.830 (0.413, 1.666)	0.600
Tertile 3	3.984 (1.683, 9.435)	0.002*	2.274 (1.213, 4.265)	0.010*
P for trend		<0.001*		0.003*

Notes: *P <0.05. Model I: adjusted age, WBC, LVEF, PLT, and SCr. Model II: Model I + the number of stents, contrast dose, and the number of infarcted vessels. Model III: Model II + CKD, DM, and AMI.Mortality Model I: adjusted age, LVEF, and SCr. Model II: Model I + number of stents and number of infarcted vessels. Model III: Model II + CKD and DM.

Abbreviations: AMI, acute myocardial infarction; CI, confidence interval; CIN, contrast-induced nephropathy; CKD, chronic kidney disease; DM, diabetes mellitus; HR, hazard ratio; LVEF, left ventricular ejection fraction; OR, odds ratio; PCI, percutaneous coronary intervention; PLT, platelet; SCr, serum creatinine; WBC, white blood cell.

mortality within two years, with preoperative MHR typically elevated in patients exhibiting poorer prognoses. This is consistent with the outcomes of prior research. A Turkish study identified elevated preoperative MHR as an independent risk factor for the onset of CIN following PCI in patients with ACS. However, no correlation with mortality within six months post-surgery was noted.[13] The correlation of MHR with CIN, cardiovascular disease, and other poor prognoses can be

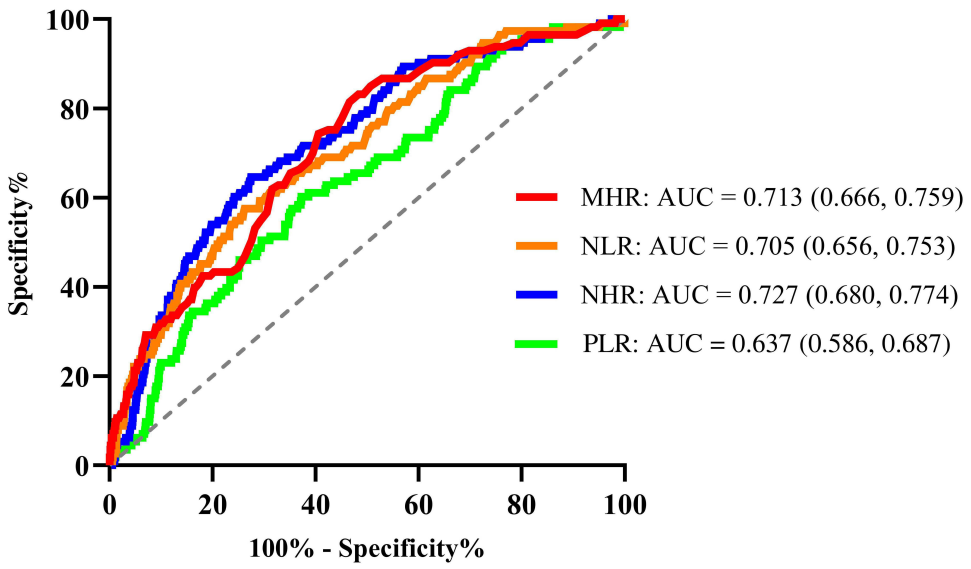

Figure 4 Receiver operating characteristic curves of MHR, NLR, NHR, and PLR for CIN.
Abbreviations: AUC, area under the curve; CIN, contrast-induced nephropathy; NHR, neutrophil-to-high-density lipoprotein cholesterol ratio; NLR, neutrophil-to-lymphocyte ratio; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; PLR, platelet-to-lymphocyte ratio.

explained by the biological function of monocytes and HDL-C. Firstly, monocytes are a significant source of pro-inflammatory substances that bind to adhesion molecules on the damaged vascular endothelium, initiating atherosclerosis and releasing pro-inflammatory and pro-oxidant cytokines.[32] In addition, monocytes have extensive phenotypic diversity, which are divided into functionally distinct subpopulations. Nonclassical and interferon-responsive monocytes may be more closely associated with increased inflammation in the atherosclerotic process than other subtypes.[33] Julla et al investigated the relationship between circulating monocytes and the severity of CAD in patients with T2DM using transcriptomics and metabolomics. Their findings demonstrated a positive correlation between coronary artery calcification scores and monocyte counts and proportions, suggesting that monocytes may act as a predictive indicator for assessing the risk of cardiovascular events in T2DM patients.[34] HDL-C represents the ability of high-density lipoprotein (HDL) to transport cholesterol, and HDL has anti-atherosclerotic properties. It inhibits the process of atherosclerosis by promoting cholesterol efflux, reducing the increase in myeloid cells and leukopoiesis, and thereby inhibiting the process of atherosclerosis.[35] Disruptions in lipid metabolism, characterized by increased triglyceride, heightened trim, oxidized low-density lipoprotein, and diminished HDL-C, are commonly observed in the initial phases of renal illness.[36] A cohort study involving 2,168 patients indicates that low levels of HDL-C are significantly linked to the progression of CKD and represent a key factor affecting its advancement.[37] Chen et al conducted a stratified Mendelian randomization analysis using data from the UK Biobank, demonstrating that HDL-C levels below 50 mg/dL significantly and negatively correlated with the risk of cardiovascular diseases.[38] Guo et al discovered that HDL-C-related indicators had strong predictive power for the occurrence of MACE during hospitalization following PCI.[10] It is noteworthy that high MHR levels were significantly associated with the risk of

Table 5 Predictive Value of MHR, NLR, NHR, and PLR for Developing CIN After PCI in Patients

Variables	AUC	95 CI%	Cutoff value	*P* value	Sensitivity	Specificity	Youden index	ΔAUC	*Pvs MHR*
MHR	0.713	(0.666, 0.759)	0.505	<0.001*	0.832	0.517	0.349	—	—
NLR	0.705	(0.656, 0.753)	3.465	<0.001*	0.575	0.736	0.313	0.009	0.759
NHR	0.727	(0.680, 0.774)	5.945	<0.001*	0.646	0.725	0.371	−0.014	0.489
PLR	0.637	(0.586, 0.687)	133.695	<0.001*	0.602	0.630	0.232	0.077	0.023*

Notes: *P <0.05.
Abbreviations: AUC, area under the curve; CI, confidence interval; CIN, contrast-induced nephropathy; NHR, neutrophil-to-high-density lipoprotein cholesterol ratio; NLR, neutrophil-to-lymphocyte ratio; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; PCI, percutaneous coronary intervention; PLR, platelet-to-lymphocyte ratio; ΔAUC: differences in AUC between MHR and other metrics; Pvs MHR, P-value for AUC comparison with MHR.

death within two years after PCI compared with low levels of MHR in this study. However, similar results were not observed in the comparison of the medium and low-level MHR groups. This may be due to the fact that inflammation and metabolic disturbances in the medium-level MHR group had not yet reached the threshold to significantly affect the risk of death. Further exploration is required regarding the MHR threshold that elevates the mortality risk.

Current studies have demonstrated that NHR and NLR are important for CIN prognosis.[10,12] Therefore, we further compared the predictive efficacy of MHR with other inflammatory indicators such as NLR, NHR, and PLR for the occurrence of CIN after PCI using ROC curve analysis. The data showed that the predictive efficacy of MHR was similar to that of NHR and NLR. These results may be attributable to the fact that neutrophils are also associated with the key pathophysiological basis of CAD, namely atherosclerosis, and their mediated inflammatory response accelerates the formation and rupture of atherosclerotic plaques.[32,39] Indicators derived by combining various laboratory parameters can effectively assess the likelihood of poor prognosis in patients after PCI, providing new ideas for clinical risk stratification. There are many risk-scoring tools available for predicting the occurrence of CIN.[40] However, these risk-scoring tools are less likely to incorporate biomarkers other than SCr. The study found that scoring tools incorporating routine blood and basic metabolic parameters are more potent in predicting adverse outcomes in CAD patients than the widely utilized Mehran score.[41] Therefore, combining novel indices of biomarkers such as MHR with traditional risk scoring tools is expected to significantly improve the predictive efficacy of existing tools.

However, in the present study we found that although the accuracy of MHR in predicting CIN was high, the specificity was lower than other similar studies.[13] The composition of the research population might account for this discrepancy. Unlike prior studies that have only included patients with MI, ours has a broader cohort of CAD patients that includes both MI and non-MI individuals. Patients with MI usually have a more severe inflammatory response and oxidative stress state, with more significant changes in MHR, thus showing higher specificity in predicting CIN. Conversely, non-MI patients may exhibit less pronounced alterations in MHR compared to MI patients owing to a less inflammatory response. Mixing the two categories of patients for analysis may dilute the specificity of MHR for CIN prediction. Consequently, subsequent subgroup studies may be conducted to investigate the disparities in the predictive capacity of MHR for CIN between infarcted and non-infarcted patients.

Despite advancements in vascular interventional techniques, CIN continues to pose a significant issue for patients with CAD. Its potential impact on patient prognosis warrants careful attention in clinical practice. There is a lack of research on the correlation between MHR and CIN after PCI. The results of this study support the idea that MHR levels are positively associated with the risk of developing CIN and that the potential of MHR in predicting CIN may be greater compared to predicting death after PCI. In addition, MHR is a simple, cost-effective, and easy-to-measure indicator for clinical staff compared to traditional CIN risk factors such as glomerular filtration rate. This study has certain limitations in several aspects. First, this is an observational single-center study with Chinese subjects. Whether there are differences in the predictive ability of MHR for CIN in different ethnic groups and the causal relationship between MHR and CIN needs to be further explored. Second, inflammation indicators fluctuate over time, and a solitary measurement may not accurately represent trends. Future high-quality longitudinal studies are essential to dynamically assess changes in MHR during the perioperative period of PCI and further analyze its association with adverse outcomes, such as CIN. In addition, some inflammatory markers (e.g., erythrocyte sedimentation rate [ESR] and high-sensitivity c-reactive protein [hs-CRP]) were lacking in the patients of this study, and future studies should include more inflammatory markers to assess their relevance and prognostic value in CAD. This study failed to include recent cases due to the limitation of the follow-up time requirement, which may affect the timeliness of the results. Follow-up studies will further update the data to improve the representativeness and reference value of the results.

Conclusions

This study shows that MHR independently forecasts the occurrence of CIN and mortality within two years in patients with CAD undergoing elective PCI, demonstrating strong predictive efficacy. This result suggests that clinical staff may consider the impact of MHR levels on outcomes when performing preoperative risk assessment for patients with CAD. In the future, we can develop more prediction models in conjunction with MHR to improve the application potential of prediction tools and provide more comprehensive support for clinical decision-making.

Abbreviations

ACEIs, angiotensin-converting enzyme inhibitor; ACS, acute coronary syndromes; AF, atrial fibrillation; AKI, acute kidney injury; AMI, acute myocardial infarction; ARBs, angiotensin receptor blockers; ARNI, angiotensin receptor neprilysin inhibitor; AUC, area under the curve; BMI, body mass index; CAD, coronary artery disease; CCB, calcium channel blocker; CI, confidence interval; CKD, chronic kidney disease; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; ESR, erythrocyte sedimentation rate; FBG, fasting blood glucose; Hb, hemoglobin; HDL, high-density lipoprotein; HDL-C, high-density lipoprotein cholesterol; HF, heart failure; HR, hazard ratio; hs-CRP, high-sensitivity c-reactive protein; LAD, left anterior descending; LCX, left circumflex; LDL-C, low-density lipoprotein cholesterol; LMCA, left main coronary artery; LVEF, left ventricular ejection fraction; MACE, major adverse cardiovascular events; MI, myocardial infarction; NHR, neutrophil-to-lymphocyte ratio; NLR, neutrophil-to-lymphocyte ratio; ns-MRAs, non-steroidal mineralocorticoid receptor antagonists; OR, odds ratio; PCI, percutaneous coronary intervention; PLR, platelet-to-lymphocyte ratio; PLT, platelet; RCA, right coronary artery; SBP, systolic blood pressure; SCr, serum creatinine; SD, standard deviation; TC, total cholesterol; TG, triglyceride; WBC, white blood cell.

Data Sharing Statement

Data supporting the results of this study are available from Fujian Medical University Union Hospital, but the availability of these data is limited and they are used under license for the current study and therefore not publicly available. However, data are available to the authors upon reasonable request and with permission from Fujian Medical University Union Hospital.

Ethics Approval and Informed Consent

The Ethics Committee of Fujian Medical University Union Hospital accepted the study protocol and informed consent procedure. All procedures adhered to the Declaration of Helsinki. All participants provided informed written consent for publication without revealing personally identifiable information.

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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.

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Disclosure

The authors report no conflicts of interest in this work.

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