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High-Sensitivity C-Reactive Protein to High-Density Lipoprotein Cholesterol Ratio and the Risk of Contrast-Induced Acute Kidney Injury in Patients Undergoing Percutaneous Coronary Intervention

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Abstract

Background: High-sensitivity C-reactive protein to high-density lipoprotein Cholesterol Ratio (CHR) is a new biomarker, which is related to the incidence rate of Coronary Artery Disease (CAD). The purpose of this study was to investigate the association between CHR and Contrast-Induced Acute Kidney Injury (CI-AKI).

Methods: In this retrospective cross-sectional study, 10,917 patients underwent PCI. Diagnose CI-AKI according to KIDIGO standard. Univariate and multivariable logistic regression analysis were used to determine the relationship between CHR and CI-AKI, and further draw the Receiver Operating Characteristic (ROC) curve of subjects to evaluate the clinical diagnostic performance of CHR on CI-AKI.

Results: 1,037 patients (9.50%) developed CI-AKI after PCI. The subjects were 64.1 ± 11.1 years old, 2,511 were females (23.0%). Multivariate logistic regression analysis showed that the increase of CHR level was associated with the increase of CI-AKI inciden ce rate ([Q4 vs. Q1]: Odds Ratio (OR) = 1.89, 95% Confidence Interval (CI) [1.42 to 2.54], P<0.001). Restrictive cubic spline analysis showed that CHR and CI-AKI were linear relationship. ROC analysis confirmed that CHR was a good predictor of CI-AKI (area under ROC curve =0.606, 95% CI [0.588 to 0.624]).

Conclusions: High CHR level is closely related to the increase of CI-AKI incidence, indicating that CHR may be an independent risk factor of CI-AKI.

Keywords: contrast-induced acute kidney injury; high-sensitivity C-reactive protein; high-density lipoprotein cholesterol.

Summary at a Glance

High-sensitivity C-reactive protein to high-density lipoprotein Cholesterol Ratio (CHR) is a new laboratory parameter, which is related to inflammation. High CHR level is closely related to the increase of contrast-induced acute kidney injury (CI-AKI) incidence, indicating that CHR may be an independent risk factor of CI-AKI.

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Introduction

Contrast-Induced Acute Kidney Injury (CI-AKI) is one of the serious complications after Coronary Angiography (CAG) or Percutaneous Coronary Intervention (PCI) [1]. It is now considered to be the third common cause of hospital acquired AKI after renal perfusion reduction and drug induced renal function damage [2]. It is related to prolonged hospitalization, increased medical costs and increased mortality [3,4], especially in patients undergoing PCI, which has caused a heavy burden on patients' economy, health and spirit. Previous studies have suggested that the original factors such as renal dysfunction (estimated glomerular filtration rate [eGFR] <60 mL/min/1.73m^2), heart failure, coronary heart disease, Diabetes Mellitus (DM), old age, anemia, ejection fraction and so on can increase the incidence of CI-AKI [5-7]. However, the pathogenesis of CI-AKI has not been clearly clarified [8], and there is no effective treatment. Therefore, it is very important to screen patients with high risk factors and take timely preventive strategies to reduce the occurrence and poor prognosis of CI-AKI.

At present, several risk factors and risk scores have been established for the occurrence and prognosis of CI-AKI patients [6,9,10], among which Mehran et al proposed a risk score including eight variables to predict the occurrence of CI-AKI after PCI. However, these prediction models include the type and number of CM, and a complete medical history, which is not convenient in clinical practice. Recently, the occurrence and prognosis of some inflammatory indicators in CI-AKI patients have also been proved [11-14].

High-sensitivity C-reactive protein to high-density lipoprotein Cholesterol Ratio (CHR) is a new laboratory parameter, which is related to inflammation. Recent studies have shown that CHR is not only closely related to the existence and severity of CAD, but also an independent predictor of severe CAD [15]. However, the association between CHR and CI-AKI risk remains uncertain. Therefore, this retrospective cross-sectional study was designed to explore the relationship between CHR and CI-AKI in CAD patients undergoing PCI.

Materials and Methods

Study Population

This is a multicenter retrospective study, with data from the Cardiorenal Improvement II (CIN-II) Study (ClinicalTrials.gov NCT05050877). The CIN-II Study recruited patients who underwent coronary angiography (CAG) and/or Percutaneous Coronary Intervention (PCI) treatment at five regional central tertiary teaching hospitals in China. 29,333 CAD patients who undergoing PCI between 2010 and 2020 were included.

The following patients met the inclusion criteria: (1) Patients over 18 years old who undergoing PCI treatment; (2) Patients who recorded Scr at baseline and within 48 hours after Contrast Medium (CM) exposure.Patients combined with conditions as follow were excluded: (1) hypersensitivity to CM; (2) estimated Glomerular Filtration Rate (eGFR) <15 mL/min/1.73m^2; (3) severe liver and kidney dysfunction or serious infectious diseases; (4) give iodine CM within 7 days; (5) have taken nephrotoxic drugs within 14 days; (6) malignant tumor; (7) patients with incomplete clinical data. Finally, 10,917 patients were included in this study. The study followed the Helsinki Declaration and was approved by the Ethics Committee of Guangdong Provincial People's Hospital (No. GDREC2019-555H-2).

Data Collection and Grouping

All demographic characteristics and clinical data of patients were obtained from the Hospital Information System, including age, gender, height, weight, complications, past medical history, hematological parameters, medication and PCI related data. All hematological parameters were measured by clinical laboratory technicians in the hospital using standard automatic biochemical analyzer, and the results were recorded. CHR is calculated using the following formula: CHR=log [hypersensitive C-reactive protein (mg/L) / high-density lipoprotein cholesterol (mmol/L)] [15].

Participants were assigned to four quartiles as follows: Q1 (n=2,730, CHR ≤ 0.24), Q2 (n=2,729, 0.24 < CHR ≤ 1.41), Q3 (n=2,729, 1.41<CHR ≤ 2.67), and Q4 (n=2,729, CHR > 2.67).

Definition and Follow-up

All patients were measured at baseline at admission and within 48 hours after PCI. If the patient had more than one postoperative creatinine measurement within 48 hours after PCI, the highest serum creatinine value was used for calculation. According to the new CI-AKI diagnostic criteria proposed by the research team "Kidney Disease: Improving Global Outcomes (KDIGO)", the study was used: within 48 hours after CM exposure, the Scr increased by ≥ 26.5 μmol/L (0.3 mg/dL) compared with the baseline value ; or the Scr increased to ≥1.5 times baseline within the previous 7 day [16].

PCI treatment is performed by experienced interventional cardiologists using standard technology according to standard clinical practice. Patients undergoing elective surgery were hydrated with 0.9% sodium chloride solution at a rate of 1mL/ (kg · h) before and 6-12 h after surgery, and the rate of severe cardiac insufficiency (left ventricular ejection fraction <40% or pulmonary edema) was halved. All clinical drugs are used according to the patient's condition.

The primary outcome of this study was the occurrence of CI-AKI, which was defined based on preoperative and postoperative serum creatinine levels. All participants were followed up through outpatient visits, hospital records or telephone interviews.

Statistical Analysis

Data were presented as the mean with Standard Deviation (SD) or median with Interquartile range (IQR) for continuous variables and quantity and frequency (%) for categorical variables. Categorical variables were compared using Pearson chi-squared test, and continuous variables using t-test. Univariable and multivari able logistic regression was used to test the association between CHR and CI-AKI. Restricted cubic splines were used to reveal the association between CHR as a continuous variable and the OR for CI-AKI.

Receiver Operator Characteristic (ROC) analysis, by using R package "ROCit" was set to determine the optimal cut-off point of CHR, and evaluate the predictive performance of CHR on CI-AKI. Finally, Exploratory analysis was performed among prespecified subgroups. All analyses were performed by R software (version 4.1.2; R Foundation for Statistical Computing, Vienna, Austria). A two-sided P-value<0.05 indicated the significance for all analyses.

Results

Baseline Characteristics of CHR Quartiles

The study included 10,917 patients who undergoing PCI. The average age of the study population was 64.1 ± 11.1 years; 2,511 were females (23.0%); A total of 1,037 patients (9.50%) developed CI-AKI (Table 1). The patient data were divided into quartiles according to CHR index: Q1: CHR ≤ 0.24; Q2: 0.24 < CHR ≤ 1.41; Q3: 1.41 < CHR ≤ 2.67; Q4: CHR > 2.67. The incidence rate of CI-AKI increased significantly with the increase of CHR level. Only 5.8% of patients in Q1 had CI-AKI, while the incidence rate of CI-AKI in Q4 was as high as 14.1%. The incidence of CI-AKI in Q2 (8.4%) and Q3 (9.7%) was significantly higher (P for trend<0.001). Compared with the Q1, patients in the Q4 were older, had higher levels of neutrophil, WBC, FPG, HbA1c and LDL-C, had higher incidence of DM, CHF and anemia, and had lower levels of eGFR and LVEF. In the group with higher CHR index, the proportion of patients were more likely to use the drug diuretics (P<0.001).

The Association Between CHR and the Occurrence of CI-AKI

Univariate and multivariate logistic regression analysis was conducted to determine the relationship between CHR and CI-AKI risk (Table 2). Model 1 shows that, compared to the reference, the increase of CHR level is related to the increase of CI-AKI risk, especially in Q2, Q3 and Q4 ([Q2 vs. Q1]: OR=1.51, 95% CI [1.22 to 1.86], P<0.001; [Q3 vs. Q1]: OR=1.76, 95% CI [1.43 to 2.17], P<0.001; [Q4 vs. Q1]: OR=2.69, 95% CI [2.22 to 3.28], P<0.001). After further adjustment and full adjustment, Model 2 and Model 3 also showed similar results (P for trends<0.001). In addition, a Restricted Cubic Spline (RCS) model with multiple adjustments was performed to visualize the relationship between CHR and CI-AKI incidence rate (Figure 1). The results revealed a potential linear relationship. With the increase of CHR level, the incidence of CI-AKI increased, which was similar to the results of logical regression (P for non-linear=0.314).

The Assessment of Predictive Ability of CHR for CI-AKI

We further drew the Receiver Operating Characteristic (ROC) curve to evaluate the clinical diagnostic performance of CHR and Mehran score on CI-AKI (Figure 2). The optimal cut-off value was 1.855, its AUC was 0.606 (95% CI [0.588 to 0.624]), the corresponding sensitivity was 61.3%, and the specificity was 55.4%, showing satisfactory diagnostic performance. The predictive value of CHR was similar to that of the Mehran score for CI-AKI (The AUC of CHR vs. Mehran score; 0.606 vs. 0.684; P<0.001).

Subgroup Analysis of CHR with CI-AKI Among PCI Patients

Further subgroup analysis was conducted according to age (<65 or ≥65 years), gender (male or female), hypertension, congestive heart failure and diabetes (Figure 3). The higher the CHR level, the higher the CI-AKI risk. Most subgroup analysis results are consistent with the overall group results. It is worth noting that CHR is an independent predictor of CI-AKI in the non-CHF subgroup, but not in the CHF subgroup. Regardless of the status of diabetes, the increase of CHR level is related to CI-AKI.

Discussion

In this study, we mainly discussed the predictive value of the new laboratory parameter CHR on the occurrence of CI-AKI after PCI. The results showed that 1,037 cases of CI-AKI occurred in 10,917 patients, the incidence rate was 9.50%, which was basically consistent with the results of similar studies at home and abroad [17,18]. Patients with higher baseline CHR levels were more likely to develop CI-AKI than those with lower CHR levels. Multivariate Logistic regression analysis showed that CHR level was an independent risk factor for CI-AKI undergoing PCI. ROC analysis showed that CHR had a good predictive effect on the occurrence of CI-AKI.

At present, many studies have shown that hs-CRP or HDL-C has independent predictive value for CI-AKI. Liu et al [19] prospectively observed 165 patients with STEMI after PCI and found that hs-CRP >16.10 mg/L was an important and independent predictor of CI-AKI. Zhang et al [20] had retrospectively studied 1,452 STEMI patients undergoing PCI, and obtained similar results. In the Park HS study [21], they enrolled 1,592 patients receiving PCI treatment from multiple centers, and found that low HDL-C levels were associated with an increased risk of CI-AKI and long-term mortality. The latest research shows that CHR is closely related to the occurrence of CAD [15]. However, there are no study on the relationship between CHR and CI-AKI. In this study, the results showed that CHR was an independent risk factor for CI-AKI in patients after PCI. The results of multiple logistic regression analysis showed that the risk of CI-AKI in patients with higher CHR level was 1.89 times higher than that in patients with lower CHR level. Therefore, this comprehensive indicator is conducive to identifying high-risk groups, taking preventive measures in advance, preventing the occurrence of CI-AKI through individual precise treatment.

The pathophysiological mechanism of CI-AKI is complex [8], which has not been clearly clarified so far. The mainstream theory is the direct and indirect effects of contrast agents on renal function and the disorder of hemodynamics [22]. In direct mechanism, CM has toxic effect on renal tubular epithelial cells, leading to loss of function, apoptosis and necrosis; The indirect mechanism is related to vascular endothelial cells, nitric oxide, prostaglandins and other vasoactive substances mediated vascular injury. In addition, the relatively low oxygen partial pressure of extrarenal medulla, coupled with the increased metabolic demand, makes medullary abnormalities vulnerable to the hemodynamic impact of CM. Current studies have shown that inflammatory factors play a central role in the occurrence of CI-AKI [23]. High levels of hs-CRP are related to endothelial dysfunction, which leads to vascular damage, decreased renal blood flow and deterioration of renal function [24]; at the same time, the increase of hs-CRP level is related to the decrease of nitric oxide production, which is also the reason for CI-AKI. On the contrary, HDL-C has a protective effect on atherosclerosis, which can reverse cholesterol transport, anti-inflammatory and antioxidant effects [25]. Theoretically, the increase of hs-CRP count and the decrease of HDL-C level may further accelerate atherosclerosis and increase the risk of adverse cardiovascular events. Recently, the correlation between CHR and CAD has been shown [15], but the relationship with CI-AKI has not been reported. In this study, our study showed for the first time that elevated CHR level is a reliable predictor of CI-AKI after primary PCI.

The occurrence of CI-AKI is closely related to some chronic basic diseases and age of patients. At present, studies at home and abroad show that old age (>75 years old), primary renal insufficiency, DM, CHF, the type and dosage of CM used and other factors are all risk factors for CI-AKI. Old age (>75 years old) is an independent risk factor for CI-AKI, which may be related to the fact that the elderly patients are often accompanied by chronic

diseases such as HT, DM and CAD, which is likely to lead to renal ischemia, thus causing the decline of glomerular filtration rate. Marenzi G [26] found that among 208 AMI patients undergoing primary PCI, the incidence of CI-AKI in elderly patients (>75 years old) reached 36%; Mehran R et al [18] studied 8,357 patients undergoing PCI and found that the incidence of CI-AKI in elderly patients (≥75 years old) was 21.8%. In this study, the incidence of CI-AKI in elderly patients undergoing PCI treatment was up to 29.3%, which is similar to previous results. Therefore, in some high-risk patients with chronic diseases, it is of great significance to carry out risk stratification and early intervention for patients through some simple and rapid identification of biomarkers of CI-AKI.

At present, there is no exact and effective method for the treatment of CI-AKI, but there are many studies on the effectiveness of the prevention of CI-AKI [2,27,28], including intravenous dilation with isotonic saline or sodium bicarbonate before PCI, anti-oxidation treatment with N-acetylcysteine or ascorbic acid [29], and the use of low osmolality or isotonic CM [28]. In addition, more and more studies have shown that statins can reduce the risk of CI-AKI [30-32], mainly through improving endothelial function, maintaining the production of nitric oxide, reducing inflammation, oxidative stress and platelet adhesion. Even if taken before PCI, statins can also reduce the risk of renal failure and CI-AKI in CAD patients. However, not all patients can start these preventive treatments in time. Moroni F et al [33] have shown that the beginning of intravenous infusion before surgery is associated with a lower incidence of CI-AKI, but patients with heart failure symptoms may not be able to fully replenish water. In this study, the risk of CI-AKI in CHF patients increased significantly, up to 45.4%. It is important to identify this high-risk group in time. It is worth noting that our further subgroup analysis shows that, for the CHF patient population, the predictive indicator of CHR is not significantly related to it, which may be caused by the high inflammatory indicators of CHF patients themselves. The specific situation needs further exploration.

Limitation

This study also had some limitations. First of all, this study only detected the baseline concentration of hs-CRP before surgery, and did not monitor the changes of inflammatory biomarkers during the study.

Secondly, this study only included some commonly used inflammatory markers, other inflammatory indicators, such as interleukin-1, tumor necrosis factor, neutrophil gelatinase associated lipocalin, were not routinely detected in this center, so it could not make a comparative analysis. Finally, many patients who did not recheck the blood SCr value within 48 hours after surgery were excluded from the screening of patients, which may cause selection bias due to the lack of data.

Conclusions

In conclusion, our study shows that among CAD patients undergoing PCI, patients with high CHR levels have a higher CI-AKI probability than those with low CHR levels. CHR has potentially predictive value for CI-AKI and may play an independent role in risk stratification in clinical practice.

Author Statements

Ethics Approval and Consent to Participate

The study protocol was approved by Guangdong Provincial People's Hospital ethics committee, and all participating sites received institutional review board approval from their own ethics committees. The study was performed according to the declaration of Helsinki. Informed consent was not required for this study by the Guangdong Provincial People's Hospital Ethics Committee.

Consent for Publication

Not Applicable in this section.

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declared no potential conflicts of interest with respect to the research, authorship of this article.

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Authors' Contributions

Research idea and study design: YL, LXD, HC, QBX; Data acquisition: JRD, XZL, KDH, LGT; Data analysis/interpretation: KDH, SQC, JL, YL; Statistical analysis: JRD, ZLL; Supervision and mentorship: YL, YL, SQC. Each author contributed important intellectual content during manuscript drafting or revision and accepts accountability for the overall work by ensuring that questions pertaining to the accuracy or integrity of any portion of the work are appropriately investigated and resolved.

Acknowledgments

Additional Contributions: Dr Liu had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. The study protocol was approved by Guangdong Provincial People's Hospital ethics committee, and all participating sites received institutional review board approval from their own ethics committees. The study was performed according to the declaration of Helsinki. Informed consent was not required for this study by the Guangdong Provincial People's Hospital Ethics Committee.

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