Research Article

C-Reactive Protein Level Predicts Cardiovascular Risk in Chinese Young Female Population

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Background. C-reactive protein (CRP) is one of the most common oxidative indexes affected by many diseases. In recent years, there have been many studies on CRP, but the relationship between CRP levels and the cardiovascular risk in the Chinese young female population is still unclear. The purpose of this work is to explore the predictive value of CRP for the cardiovascular risk in the Chinese young female population.

Methods. The study is conducted by 1:1 case-control to retrospectively analyze 420 young women with acute coronary syndrome (ACS group) who underwent percutaneous coronary intervention (PCI) and 420 young women (control group) who underwent coronary angiography (CAG) to exclude coronary heart disease from January 2007 to December 2016. All patients are divided into three subgroups according to CRP values: subgroup 1: CRP < 1.0 mg/L (n = 402); subgroup 2: 1.0 mg/L ≤ CRP ≤ 3.0 mg/L (n = 303); subgroup 3: CRP > 3.0 mg/L (n = 135). The levels of CRP were observed in the two groups and three subgroups.

Results. A total of 840 patients were analyzed. The mean duration of follow-up was 66.37 ± 30.06 months. The results showed that the level of CRP in the ACS group was significantly higher than that in the control group (1.30 ± 1.70 vs. 3.33 ± 5.92, respectively, p < 0.001), and patients with higher CRP levels were associated with a significantly increased rate of major adverse cardiovascular events (MACE) (7.0% vs. 8.9% vs. 19.30%, respectively, p < 0.05). After adjustment for baseline covariates, CRP level was still an independent predictor for the incidence of MACE, either as a continuous variable or as a categorical variable. There was a significantly higher rate of all-cause mortality and myocardial infarction in patients with higher CRP values during follow-up. Conclusions. The research results show that high CRP is associated with increased risk of ACS in the Chinese young female population. Risk stratification with CRP as an adjunct to predict clinical risk factors might be useful in the Chinese young female population.

1. Introduction

Coronary heart disease (CHD) has become one of the top leading causes of death among Chinese adults [1]. The cardiovascular (especially CHD) morbidity and mortality have been rising in women from 1990 to 2020 [2–4].急性冠状动脉综合征 (ACS) is a serious event for CHD. Among men and women with ACS, there are important dissimilarities in clinical presentation, cardiovascular risk factors and receiving medical care and unfavorable outcomes after percutaneous coronary intervention (PCI) is performed [5, 6]. Compared with men, women with ACS are receiving too little attention at present and have a higher risk of death and rate of major adverse cardiac events (MACE) following PCI [7, 8]. On the pathogenesis of ACS, previous studies have suggested that various inflammatory-related factors including oxidative stress may lead to vascular endothelial damage, which plays an important role in the process of atherosclerosis and makes important contributors to the development of ACS [9, 10]. As a marker of systemic inflammation, C-reactive protein (CRP) has been proven to be associated with increased relative risks of cardiovascular events in ACS patients [11]. However, thus far, no data are available regarding the relationship between CRP levels...
and the incidence of ACS in the Chinese young female population. In the present study, we evaluated the predictive value of CRP levels for the risk of ACS and clinical outcomes of long-term follow-up in the Chinese young female population.

2. Materials and Methods

2.1. Patients. The study was conducted by 1:1 case-control to retrospectively analyze 420 young women with ACS (ACS group) who underwent PCI and 420 young women (control group) who underwent coronary angiography (CAG) to exclude CHD by propensity score matching from January 2007 to December 2016. All patients were divided into three subgroups according to risk stratification from the American Heart Association (AHA): subgroup 1 (low risk): CRP < 1.0 mg/L (n = 402); subgroup 2 (average risk): 1.0 mg/L ≤ CRP ≤ 3.0 mg/L (n = 303); and subgroup 3 (high risk): CRP > 3.0 mg/L (n = 135). The levels of serum CRP were observed in the two groups and three subgroups. Baseline characteristics of the enrolled patients were recorded in detail, including age, gender, body mass index (BMI), smoking history, hypertension, diabetes, the level of CRP, and other biomarkers. Exclusion criteria included the following: including congestive heart failure (CHF), myocardialopathy, congenital heart disease, history of aorta surgery, rheumatic heart disease (RHD), severe valve disease, pulmonary or connective tissue disease, active cancer, infective endocarditis, and chronic kidney disease. All the patients were recruited from the Chinese population, and all subjects signed the informed consent. All patients with ACS took prescribed dual antiplatelet therapy, including aspirin (300 mg loading dose, followed by 100 mg once daily) and clopidogrel (300 mg loading dose, followed by 75 mg once daily) for at least 12 months after PCI regardless of drug-eluting stent (DES) type. Individual medical management decisions during hospitalization were exclusively decided by their responsible interventional cardiologists and physicians. Clinical follow-up was performed by telephone contact or outpatient clinical visits. The date of follow-up was ended in December 2018. The primary endpoints for this study were MACE, which included composite occurrence of cardiac death, nonfatal myocardial infarction, and target vessel revascularization (TVR). The follow-up time range was from 11 months to 146 months. The mean duration of follow-up was 66.37 ± 30.06 months.

2.2. Statistical Analysis. The sample size of this study was calculated by using Power Analysis and Sample Size software (PASS, v 11.0.10, developed by NCSS, LLC, Kaysville, Utah, USA). The data statistical analyses were performed using the Statistical Package for Social Sciences software (SPSS, version 20.0, SPSS Inc. Chicago, IL, USA). All data of cases were inputted into the computer software database. The continuous variables with normal distributions were expressed as mean ± standard deviation. The comparisons between groups were performed using the independent Student’s t-test. The counting data were expressed as a percentage (%), and the chi-square (χ²) test was used for comparison between groups. The association of CRP and MACE was investigated by multivariable Cox proportional-hazard models adjusted for all potential confounding factors. The test level was set as a double-tail test α = 0.05, p < 0.05 was statistically significant, and p < 0.01 was statistically very significant.

3. Results and Analysis

3.1. Baseline Characteristics in ACS Group and Control Group Are Shown in Table 1. There is no significant difference in age between the two groups (p > 0.05). Compared with the control group, the proportion of overweight, smoking, hypertension, diabetes, and hypercholesterolemia in the ACS groups is significantly higher (p < 0.05). The level of CRP in the ACS group is significantly higher than that in the control group (1.30 ± 1.70 vs. 3.33 ± 5.92, respectively, p < 0.001).

3.2. Bivariate Logistic Regression Analysis of the Relationship between CRP and ACS Is Shown in Table 2. The result suggests that CRP was an independent predictor of ACS (OR = 1.233, p < 0.05).

3.3. Baseline Characteristics in Each Subgroup Are Shown in Table 3. There was no significant difference in age, HB, and Hcy in the three subgroups (p > 0.05). Overweight, smoking,

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**Table 1: Baseline characteristics of each group.**

|                  | ACS group (420) | Control group (420) | t/x² | p value |
|------------------|----------------|--------------------|------|---------|
| Age              | 40.64 ± 4.14   | 40.77 ± 3.86       | 0.353| 0.742   |
| Overweight       | 265 (63.10)    | 70 (16.67)         | 188.804| ≤0.001 |
| Smoking          | 27 (6.43)      | 12 (2.86)          | 6.050 | 0.014   |
| Hypertension     | 207 (49.29)    | 49 (11.67)         | 140.262| ≤0.001 |
| Diabetes         | 97 (23.10)     | 17 (4.05)          | 46.956| ≤0.001  |
| Hypercholesterolemia | 35 (8.33) | 8 (1.90)          | 17.868| ≤0.001  |
| CRP              | 3.33 ± 5.92    | 1.30 ± 1.70        | 6.761 | ≤0.001  |

**Table 2: Bivariate logistic regression analysis of the relationship between CRP and ACS.**

| B     | Wald   | p      | OR     | 95% CI     |
|-------|--------|--------|--------|------------|
| CRP   | 0.209  | 33.697 | ≤0.001 | 1.233      |
|       |        |        |        | 1.15-1.32  |
CRP is an acute inflammatory response protein induced by cytokines and is secreted by the liver and activated macrophages in atherosclerotic plaques [12]. CRP also plays a direct regulatory role in the process of atherosclerosis, which is related to cytokine release, smooth muscle cell migration, extracellular matrix remodeling, endothelial dysfunction, and the activation of circulating monocytes [13, 14]. Elevated preprocedural CRP is associated with an increased risk for CI-AKI in patients undergoing PCI [15]. CRP is closely related to ACS. CRP promotes the formation of unstable atherosclerotic plaques and triggers the rupture of vulnerable plaques, leading to coronary thrombosis and occurrence of ACS and MACE [16, 17]. Studies have shown that the elevated CRP is closely related to coronary artery events and may be to predict future adverse cardiac events. There was significant difference in serum CRP level between the coronary artery disease (CAD) group and the non-CAD group. The serum CRP level in acute myocardial infarction (AMI), unstable angina, and stable angina pectoris is higher than that in non-CAD patients. It is suggested that CRP is related to the severity of CAD [18]. It is not only an

hypertension, diabetes, hypercholesterolemia, unstable angina, double coronary lesions, three coronary lesions, total stent length, SCr, Uric, LDL, HDL, TC, and TG in subgroup 3 were significantly higher than those in subgroup 1 and subgroup 2 (p < 0.05).

3.4. Long-Term Clinical Outcomes during Follow-Up Are Shown in Table 4. Patients with higher CRP levels (CRP > 3 mg/L) were associated with a significantly increased rate of MACE. Cox regression model analysis illustrated that there were significant increases in MACE in patients with higher CRP during the follow-up (see Figure 1).

4. Discussion

CRP is an acute inflammatory response protein induced by cytokines and is secreted by the liver and activated macrophages in atherosclerotic plaques [12]. CRP also plays a direct regulatory role in the process of atherosclerosis, which is related to cytokine release, smooth muscle cell migration, extracellular matrix remodeling, endothelial dysfunction, and the activation of circulating monocytes [13, 14]. Elevated preprocedural CRP is associated with an increased risk for CI-AKI in patients undergoing PCI [15]. CRP is closely related to ACS. CRP promotes the formation of unstable atherosclerotic plaques and triggers the rupture of vulnerable plaques, leading to coronary thrombosis and occurrence of ACS and MACE [16, 17]. Studies have shown that the elevated CRP is closely related to coronary artery events and may be to predict future adverse cardiac events. There was significant difference in serum CRP level between the coronary artery disease (CAD) group and the non-CAD group. The serum CRP level in acute myocardial infarction (AMI), unstable angina, and stable angina pectoris is higher than that in non-CAD patients. It is suggested that CRP is related to the severity of CAD [18]. It is not only an

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Table 3: Baseline characteristics of each subgroup.

| Subgroup 1: CRP < 1 mg/L | Subgroup 2: 1.0 mg/L ≤ CRP ≤ 3.0 mg/L | Subgroup 3: CRP > 3.0 mg/L | F/X^2 | p value |
|--------------------------|--------------------------------------|-----------------------------|-------|--------|
| Number                   | 402                                  | 303                         | 135   |        |
| Age (years old)          | 40.83 ± 3.57                         | 40.98 ± 3.99                | 40.41 ± 4.61 | 1.010 | 0.365 |
| Overweight n (%)         | 134 (33.3)                           | 114 (37.3)                  | 87 (64.4) | 41.935 | ≤0.001 |
| Smoking, n (%)           | 18 (4.48)                            | 14 (4.62)                   | 7 (5.19) | 0.118 | 0.943 |
| Hypertension, n (%)      | 108 (26.87)                          | 84 (27.72)                  | 64 (47.41) | 21.846 | ≤0.001 |
| Diabetes, n (%)          | 44 (10.95)                           | 38 (12.54)                  | 32 (23.70) | 14.486 | 0.001 |
| Hypercholesterolemia, n (%) | 12 (2.99)           | 20 (6.60)                   | 11 (8.15) | 7.761 | 0.02 |
| Unstable angina, n (%)   | 154 (38.31)                          | 91 (30.03)                  | 64 (47.41) | 12.652 | 0.002 |
| Double coronary lesions, n (%) | 24 (5.97) | 23 (7.59)                   | 22 (16.30) | 14.567 | 0.001 |
| Three coronary lesions, n (%) | 5 (1.24)   | 2 (0.66)                    | 5 (3.70) | 6.323 | 0.042 |
| Total stent length (mm)  | 10.39 ± 12.06                        | 10.07 ± 12.87               | 20.16 ± 10.80 | 41.751 | ≤0.001 |
| SCr (μmol/L)             | 61.38 ± 14.63                        | 59.49 ± 11.03               | 65.26 ± 26.05 | 6.086 | 0.002 |
| Uric (μmol/L)            | 269.56 ± 71.24                       | 289.61 ± 72.77              | 296.73 ± 88.36 | 18.119 | ≤0.001 |
| HB (g/L)                 | 128.14 ± 13.09                       | 130.53 ± 13.67              | 126.23 ± 15.39 | 0.147 | 0.701 |
| Hcy (μmol/L)             | 9.53 ± 4.87                          | 8.84 ± 3.94                 | 9.91 ± 5.51 | 0.669 | 0.414 |
| LDL (mmol/L)             | 2.40 ± 0.81                          | 2.56 ± 0.83                 | 2.78 ± 1.21 | 22.904 | ≤0.001 |
| HDL (mmol/L)             | 1.19 ± 0.30                          | 1.22 ± 0.35                 | 1.05 ± 0.31 | 10.028 | 0.002 |
| TC (mmol/L)              | 4.10 ± 1.08                          | 4.50 ± 1.12                 | 4.51 ± 1.28 | 21.039 | ≤0.001 |
| TG (mmol/L)              | 1.44 ± 0.87                          | 1.73 ± 1.49                 | 1.78 ± 1.16 | 13.081 | ≤0.001 |

Note: SCr: serum creatinine; HB: hemoglobin; Hcy: homocysteine; Uric: serum uric acid; HDL: high-density lipoprotein; LDL: low-density lipoprotein; TC: total cholesterol; TG: triglycerides.

Table 4: Clinical outcomes during follow-up.

| Subgroup 1: CRP < 1.0 mg/L | Subgroup 2: 1.0 mg/L ≤ CRP ≤ 3.0 mg/L | Subgroup 3: CRP > 3.0 mg/L | X^2 | p value |
|---------------------------|--------------------------------------|-----------------------------|-----|--------|
| Death, n (%)              | 3 (0.75)                             | 1 (0.33)                    | 4 (2.96) | 7.210 | 0.027 |
| MI, n (%)                 | 5 (1.24)                             | 0 (0.00)                    | 4 (2.96) | 7.931 | 0.019 |
| TVR, n (%)                | 26 (6.47)                            | 26 (8.58)                   | 22 (16.30) | 12.166 | 0.002 |
| MACE, n (%)               | 28 (6.97)                            | 27 (8.91)                   | 26 (19.26) | 17.832 | ≤0.001 |

Note: MI: myocardial infarction; TVR: target vessel revascularization; MACE: major adverse cardiac events.
Our research indicates that high CRP is associated with increased risk of ACS in the Chinese young female population. Risk stratification with CRP as an adjunct to predict clinical risk factors might be useful in the Chinese young female population.

5. Conclusions

Data Availability

The data used to support the findings of this study are available from the corresponding authors upon request.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Authors’ Contributions

Conceptualization was handled by Tongku Liu. Data curation and analysis were worked on by Ruifang Liu, Fangxing Xu, and Tongku Liu. Funding acquisition and resources were secured by Yujie Zhou and Tongku Liu. Investigation was conducted by Qian Ma, Yujie Zhou, and Tongku Liu. Writing (original draft) was conducted by Ruifang Liu and Tongku Liu. Writing (review and editing) was taken care of by Ruifang Liu, Tongku Liu, Qian Ma, and Yujie Zhou.

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