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Original Article

# C-reactive protein positively correlates with metabolic syndrome in coronary artery disease patients

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ABSTRACT

*Objective:* C-reactive protein (CRP) is an independent risk factor for coronary artery disease (CAD). Metabolic syndrome has been associated with an increased risk of CAD, as well as increased mortality from CAD. The aim of this study is to investigate the relationship between CRP and metabolic syndrome among CAD patients.

*Materials and Methods:* Fasting blood samples were obtained from 112 CAD patients. Metabolic syndrome and its components were defined using the diagnostic criteria from the International Diabetes Federation.

*Results:* Using Taiwanese individuals, a total of 56 CAD patients (50%) had metabolic syndrome. Fasting CRP levels positively correlated with metabolic syndrome ($p = 0.014$). Univariate linear regression analysis showed that body weight ($p = 0.014$), body mass index ($p < 0.001$), and body fat mass ($p < 0.001$) were positively correlated with serum CRP levels, while high-density lipoprotein cholesterol (HDL-C) ($p = 0.049$) was negatively correlated with fasting serum CRP levels in CAD patients. Multivariate forward stepwise linear regression analysis of the significant variables showed that body fat mass ($\beta = 0.530$, R square = 0.289, $p < 0.001$) and HDL-C ($\beta = -0.171$, R square = 0.029, $p = 0.034$) were independent predictors of serum CRP levels in CAD patients.

*Conclusion:* CRP level positively correlated with metabolic syndrome among Taiwanese CAD patients. Body fat mass and HDL-C were independent predictors of serum CRP levels in CAD patients.



## 1. Introduction

C-reactive protein (CRP) is a hepatic derived pentraxin that plays a key role in the innate immune response and is an independent risk factor for coronary artery disease (CAD) [1,2]. Metabolic syndrome is a significant risk factor for cardiovascular disease and mortality in the general population [3,4]. It is also independently associated with CAD [5]. CAD patients with metabolic syndrome show an increased risk of cardiovascular morbidity after follow-up [6]. Recent studies have shown that a high level of CRP is associated with metabolic syndrome among European individuals [7]. The aim of this study was to investigate the relationship between serum CRP level and the metabolic syndrome among CAD patients in Taiwan.

## 2. Materials and methods

### 2.1. Patients

Between June 2008 and December 2008, 112 CAD consecutive clinic outpatients at a medical center in Hualien, eastern Taiwan (80 males and 32 females), who were defined as individuals having as > 50% stenosis in any segment by coronary angiography, were enrolled into this study. The Protection of Human Subjects Institutional Review Board of Tzu Chi University and Hospital approved this study. Patients were excluded if they had any acute infection, acute myocardial infarction, acute coronary syndrome, pulmonary edema, heart failure at the time of blood sampling or if they refused to provide informed consent for the study.

### 2.2. Anthropometric analysis

Body weight was measured in light clothing and without shoes to the nearest half kilogram. Height was measured to the nearest

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half centimeter. Waist circumference was measured to the nearest half centimeter at the shortest point below the lower rib margin and the iliac crest. The body mass index (BMI) was calculated as weight (kilograms) divided by height squared (meters). Bioimpedance measurements of fat mass were performed at the bedside, using the standard tetrapolar whole body (hand-foot) technique and a single-frequency (50 kHz) analyzer (Biodynamic-450, Biodynamics Corporation, Seattle, WA, USA). Measurements were carried out by the same operator and the fat mass was analyzed using the specific formulae provided by the manufacturer [8].

### 2.3. Biochemical investigations

Fasting blood samples taken from each subject were immediately centrifuged at 3000 g for 10 minutes for the biochemical studies. The serum was stored at 4°C for biochemical examination within 1 hour after collection. Serum levels of creatinine (Cre), fasting glucose, total cholesterol (TCH), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), albumin, globulin, and C-reactive protein (CRP) were measured using an autoanalyzer (COBAS Integra 800, Roche Diagnostics, Basel, Switzerland).

### 2.4. Metabolic syndrome and its components

The prevalence of metabolic syndrome was defined using the International Diabetes Federation definition [9]. People were classified as having metabolic syndrome if they had central (abdominal) obesity with a waist circumference ≥90 cm (men) or ≥80 cm (women) (Chinese criteria), and had two or more of the following additional criteria: fasting serum glucose ≥110 mg/dL, triglycerides ≥150 mg/dL, HDL-C level <40 mg/dL in men or <50 mg/dL in women, or blood pressure ≥130/85 mm Hg. The use of antihypertensive medication was considered as equivalent to high blood pressure in this analysis. Type 2 diabetes was determined according to World Health Organization criteria [10]. A person was regarded as diabetic if either the fasting plasma glucose ≥126 mg/dL, or the 2 hour glucose during an oral glucose tolerance test was ≥200 mg/dL, or if he/she was using diabetes medication (oral or insulin).

### 2.5. Statistical analysis

Data are expressed as means ± standard deviation (SD) and tested for normal distribution by Kolmogorov-Smirnov statistics. Categorical variables were analyzed by the Chi-square test. Comparisons between patients were performed using the Student *t* test (two tailed) for normally distributed data, or the Mann-Whitney U test for parameters that presented with non-normal distribution (fasting glucose, CRP, and Cre). Clinical variables that correlated with CRP in CAD patients were evaluated by univariate linear regression analyses. Variables that were significantly associated with CRP in CAD patients were tested for independency by multivariate forward stepwise regression analysis. Data were analyzed using SPSS for Windows (version 13.0; SPSS Inc., Chicago, IL, USA). A $p$ value <0.05 was considered statistically significant.

## 3. Results

The clinical and laboratory characteristics of the CAD patients are presented in Table 1 and Table 2. Medical histories included: diabetes ($n$ = 43; 36.7%), hypertension ($n$ = 90; 80.4%), and hyperlipidemia ($n$ = 71; 63.4%). The use of drugs included: angiotensin receptor blocker (ARB; $n$ = 39; 34.8%), angiotensin-converting enzyme inhibitor (ACEI; $n$ = 33; 29.5%), calcium channel blocker (CCB; $n$ = 24; 21.4%), β-blocker (n = 64; 57.1%), thiazide ($n$ = 29; 25.9%), fibrate ($n$ = 8; 7.1%), aspirin ($n$ = 68; 60.7%), clopidogrel ($n$ = 35; 31.3%), sulfonylurea ($n$ = 24; 21.4%), metformin ($n$ = 22; 19.6%), peroxisome proliferator-activated receptor-γ (PPAR-γ) agonist ($n$ = 5; 4.5%) and statin ($n$ = 65; 58.0%).

The clinical characteristics and fasting serum CRP levels of the 112 CAD patients are presented in Table 2. A total of 56 patients (50.0%) had metabolic syndrome. CAD patients with metabolic syndrome had a higher CRP level ($p$ = 0.014). CRP levels did not differ statistically by gender distribution, diabetes, hypertension, hyperlipidemia, ARB, ACEI or CCB. Neither did they differ by β-blocker, thiazide, statin, fibrate, sulfonylurea, metformin or PPAR-γ drugs use. Multivariate forward stepwise linear regression analysis of the clinical characteristics and fasting serum CRP levels also showed that metabolic syndrome (β = 0.334, R square = 0.112, $p$ < 0.001) was positively correlated with serum CRP levels in CAD patients (data not shown).

The univariate linear analysis of fasting serum CRP levels in CAD patients is presented in Table 3. Body weight ($p$ = 0.014), body mass index ($p$ < 0.001), and body fat mass ($p$ < 0.001) were positively correlated with serum CRP levels, while HDL-C ($p$ = 0.049) was negatively correlated with fasting serum CRP levels in CAD patients.

Multivariate forward stepwise linear regression analysis of the significant variables showed that body fat mass (β = 0.530, R square = 0.289, $p$ < 0.001) and HDL-C (β = −0.171, R square = 0.029, $p$ = 0.034) were the independent predictors of serum CRP levels in CAD patients (Table 4).

**Table 1**
Clinical and analytical characteristics of 112 coronary artery disease patients.

| Item | Parameter | | Parameter | |
|---|---|---|---|---|
| Anthropometric findings | Height (cm) | 160.34 ± 15.68 | Waist circumference (cm) | 91.47 ± 9.80 |
| | Body weight (kg) | 67.65 ± 11.23 | Body fat mass (%) | 27.95 ± 6.83 |
| | Body mass index (kg/m$^2$) | 25.84 ± 3.58 | Age (y) | 68.37 ± 11.33 |
| | Gender (male/female) | 80/32 | | |
| Biochemical findings | Albumin (g/dL) | 4.42 ± 0.34 | Globulin (g/dL) | 3.08 ± 0.43 |
| | Triglyceride (mg/dL) | 145.00 ± 70.93 | Total cholesterol (mg/dL) | 167.05 ± 35.55 |
| | Fasting glucose (mg/dL) | 122.62 ± 44.35 | HDL-C (mg/dL) | 46.43 ± 12.44 |
| | CRP (mg/dL) | 0.39 ± 0.39 | LDL-C (mg/dL) | 105.63 ± 31.57 |
| | Creatinine (mg/dL) | 1.15 ± 0.97 | | |

Data are expressed as means ± SDs or proportion.
ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; CRP = C-reactive protein; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol.

**Table 2**
Clinical characteristics and fasting serum C-reactive protein levels of 112 coronary artery disease patients.

| Characteristic | | Number (%) | CRP (mg/dL) | *p* |
|---|---|---|---|---|
| Gender | Male | 80 (71.4) | 0.38 ± 0.36 | 0.819 |
| | Female | 32 (28.6) | 0.40 ± 0.41 | |
| Diabetes | No | 69 (61.6) | 0.39 ± 0.36 | 0.713 |
| | Yes | 43 (38.4) | 0.40 ± 0.44 | |
| Hypertension | No | 22 (19.6) | 0.31 ± 0.23 | 0.886 |
| | Yes | 90 (80.4) | 0.41 ± 0.42 | |
| Hyperlipidemia | No | 41 (36.6) | 0.42 ± 0.40 | 0.625 |
| | Yes | 71 (63.4) | 0.38 ± 0.39 | |
| Metabolic syndrome | No | 56 (50.0) | 0.26 ± 0.21 | 0.014* |
| | Yes | 56 (50.0) | 0.52 ± 0.48 | |
| ARB | No | 73 (65.2) | 0.35 ± 0.36 | 0.127 |
| | Yes | 39 (34.8) | 0.47 ± 0.44 | |
| ACE inhibitors | No | 79 (70.5) | 0.42 ± 0.41 | 0.202 |
| | Yes | 33 (29.5) | 0.33 ± 0.32 | |
| CCB | No | 88 (78.6) | 0.36 ± 0.38 | 0.073 |
| | Yes | 24 (21.4) | 0.52 ± 0.42 | |
| β-blocker | No | 48 (42.9) | 0.35 ± 0.37 | 0.171 |
| | Yes | 64 (57.1) | 0.43 ± 0.40 | |
| Thiazide | No | 83 (74.1) | 0.39 ± 0.34 | 0.269 |
| | Yes | 29 (25.9) | 0.41 ± 0.38 | |
| Statin | No | 47 (42.0) | 0.41 ± 0.38 | 0.269 |
| | Yes | 65 (58.0) | 0.38 ± 0.40 | |
| Fibrate | No | 104 (92.9) | 0.39 ± 0.39 | 0.968 |
| | Yes | 8 (7.1) | 0.38 ± 0.37 | |
| Sulfonylurea | No | 88 (78.6) | 0.40 ± 0.39 | 0.498 |
| | Yes | 24 (21.4) | 0.38 ± 0.41 | |
| Metformin | No | 90 (80.4) | 0.41 ± 0.39 | 0.201 |
| | Yes | 22 (19.6) | 0.33 ± 0.39 | |
| PPAR-γ agonist | No | 107 (95.5) | 0.40 ± 0.40 | 0.257 |
| | Yes | 5 (4.5) | 0.18 ± 0.10 | |

*$p < 0.05$ was considered statistically significant after performing the Mann-Whitney U test.
ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; PPAR-γ = peroxisome proliferator-activated receptor-γ.

## 4. Discussion

The results of our study showed that the CRP level was positively associated with metabolic syndrome in CAD patients. Body fat mass and HDL-C were the independent predictors of serum CRP levels in CAD patients. Metabolic syndrome is a constellation of physical and laboratory abnormalities including hypertension, hyperglycemia, hyperlipidemia, and abdominal obesity [9]. Metabolic syndrome constitutes a major health problem in the West, and is estimated to affect at least 20% of the adult population [11]. Recent studies have also demonstrated that metabolic syndrome is a significant risk factor for cardiovascular disease and mortality in the general population [3,4]. The overall prevalence of the metabolic syndrome in CAD patients has been found to be between 37.5% and 65.5% by various studies [2,6,12,13]. The prevalence of metabolic syndrome in our study was 50%, which is a similar prevalence to that reported by other studies of CAD patients [6,13].

CRP is an acute phase marker whose blood levels depend on interleukin-6 and other inflammatory proteins that stimulate its production by hepatocytes, lymphocytes, alveolar macrophages and monocyte-derived macrophages in atherosclerotic plaques [14]. CRP can induce a number of activities at tissue and cell levels involved in the processes of atherosclerosis and thrombosis [15]. CRP has been shown to impair insulin signalling, contribute to atherothrombosis, and is associated with insulin resistance, adiposity and metabolic syndrome [16]. Beyond traditional established cardiovascular risk factors, CRP has been shown to be crucial throughout atherosclerosis from endothelial dysfunction to plaque rupture and thrombosis [17]. Recent studies have found that CRP is an independent risk factor for CAD [1,2]. High CRP levels have been related to the risk factors for dyslipidemia, hypertension, diabetes mellitus, and obesity [18]. CRP interacted multiplicatively with apolipoprotein B and other variables associated with metabolic syndrome [19]. Elevated CRP levels have been associated with metabolic syndrome among European individuals [7]. In a similar finding for Taiwanese individuals, our study noted that CAD patients with metabolic syndrome had higher fasting serum CRP levels.

CRP concentration was found to be positively correlated with body weight, negatively correlated with fasting concentration of triglycerides and showed no correlation with LDL-C or total cholesterol in a Czech population [20]. CRP concentration has been directly related to the volume of visceral fat determined by magnetic resonance imaging [21]. Abdominal adiposity resulted in significantly elevated CRP values among non-obese people [22]. A higher BMI, as well as central obesity, have been shown to be independently associated with higher levels of CRP in Taiwan [23]. Higher amounts of visceral adipose tissue have been associated with CRP and this may lead to subclinical left ventricular diastolic dysfunction in otherwise-healthy subjects [24]. Higher CRP levels were also observed in subjects with a high BMI and a high waist circumference among European CAD patients [7]. However, one study noted that, in the lower tertile of BMI, patients with CAD had a markedly higher CRP concentration compared to control subjects [25]. Our study found that, among CAD patients, body weight, BMI, and body fat mass were positively correlated with the fasting serum CRP levels, while HDL-C was negatively correlated with the fasting serum CRP levels. CRP concentration was not correlated with LDL-C level, waist circumference, triglyceride level, fasting glucose level or total cholesterol level in our study. The multivariate forward stepwise linear regression analysis of the significant variables showed that body fat mass and HDL-C were independent predictors of fasting serum CRP levels in our study. Further studies are

**Table 3**
Correlation between fasting serum C-reactive protein levels and clinical variables in 112 coronary artery disease patients by univariate linear regression analyses.

| Items | Beta | *p* |
|---|---|---|
| Age (y) | −0.057 | 0.430 |
| Height (cm) | −0.124 | 0.191 |
| Body weight (kg) | 0.232 | 0.014* |
| Waist circumference (cm) | 0.164 | 0.084 |
| Body mass index (BMI; kg/m$^2$) | 0.396 | <0.001* |
| Body fat mass (%) | 0.537 | <0.001* |
| Albumin (g/dL) | 0.054 | 0.568 |
| Globulin (g/dL) | 0.062 | 0.517 |
| Total cholesterol (mg/dL) | 0.021 | 0.828 |
| Triglyceride (mg/dL) | 0.118 | 0.217 |
| HDL-C (mg/dL) | −0.187 | 0.049* |
| LDL-C (mg/dL) | 0.012 | 0.904 |
| Fasting glucose (mg/dL) | 0.178 | 0.060 |
| Creatinine (mg/dL) | −0.041 | 0.667 |

*$p < 0.05$ was considered statistically significant after univariate linear analyses.
HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol.

**Table 4**
Multivariate stepwise linear regression analysis of body weight, body mass index, body fat mass, and HDL-C level: correlation with fasting serum C-reactive protein levels in the 112 coronary artery disease patients.

| Items | Beta | R square | R square change | *p* |
|---|---|---|---|---|
| Body fat mass (%) | 0.530 | 0.289 | 0.289 | <0.001* |
| HDL-C (mg/dL) | −0.171 | 0.318 | 0.029 | 0.034* |

*$p < 0.05$ was considered statistically significant in the multivariate stepwise linear regression analysis.
HDL-C = high-density lipoprotein cholesterol.

required to elucidate the relationship between CRP and metabolic syndrome components in CAD patients.

Pharmacological interventions have been shown to influence serum CRP levels in humans. Statins can reduce serum CRP level, independent of the type and dose of statin used [26]. PPAR-α activation by fibrates also impairs pro-inflammatory cytokine-signaling pathways in the liver resulting in modulation of the acute phase response reaction via mechanisms independent of changes in lipoprotein levels [27]. PPAR-γ agonist treatment results in decreased plasma levels of CRP in both obese and type 2 diabetic patients [28]. In hypertension patients, β-blocker, angiotensin-converting enzyme inhibitor and angiotensin receptor blocker monotherapy have been associated with lower CRP levels than in those taking a diuretic monotherapy [29]. Our results did not show a relationship between the use of statins, PPAR-γ agonists or other drugs (ARB, ACEI, CCB, fibrate or thiazide diuretic) and serum CRP among our CAD patients.

There are some limitations to our study. Firstly, this study is a cross-sectional design. Therefore, our findings need to be further investigated using long-term prospective studies before a causal relationship between serum CRP and metabolic syndrome in CAD patients can be established. Secondly, serum insulin or insulin resistance were not measured in this study. Thirdly, there is no control group in the study. Another limitation is that this study did not check other inflammatory markers, such as TNF-α, IL-6, and IL-12. Finally, the sample size is small and the power of this study is only 0.75 (analysis by G-Power 3.0.1), so more patients are needed to support our findings. Therefore, further studies are also needed to investigate the association of metabolic syndrome and serum CRP levels in patients with or without CAD.

In summary, we found a positive association between circulating fasting CRP levels and metabolic syndrome among CAD patients. Body fat mass and HDL-C were the independent predictors of serum CRP levels among Taiwanese CAD patients.

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