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Serum levels of C1q/TNF-related protein-1 (CTRP1) and CTRP3 in patients with coronary artery disease

Gholamreza Namazi[1,2*], Maryam Mehranian[2], Alireza Mafi[2], Razieh Salami[2], Fariba Raygan[3]

1- Research Center for Biochemistry and Nutrition in Metabolic Diseases, Kashan University of Medical Sciences, Kashan, Iran
2- Department of Clinical Biochemistry, School of Medicine, Kashan University of Medical Sciences, Kashan, Iran
3- Department of Cardiology, School of Medicine, Kashan University of Medical Sciences, Kashan, Iran

Correspondence:

Gholamreza Namazi;
Department of Clinical Biochemistry, Faculty of Medicine, Kashan University of Medical Sciences, Qotb –e Ravandi Blvd, Kashan, Iran;
Email: namazi-gh@kaums.ac.ir

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Abstract

BACKGROUND: Research suggests that C1q/TNF-related proteins (CTRPs) play a role in inflammation and may impact coronary artery disease (CAD). This study aimed to measure their levels in CAD patients and assess their correlation with disease severity.

METHODS: This study involved 88 individuals (50 men and 38 women) who underwent coronary angiography. Among them, 47 patients had significant coronary obstruction and were selected as the case group, and 41 individuals without significant obstruction were considered as the control group. Serum levels of CTRP1 and CTRP3 were quantified using the ELISA method, whereas other parameters were assessed by standard laboratory techniques. Multiple logistic regression analysis was used to examine the association between CTRP1 and CTRP3 levels with CAD.

RESULTS: The mean age of CAD patients was 62.1 ± 11.5 years. Thirty-one patients were male and sixteen were female. The control group comprised nineteen men and twenty-two women, with a mean age of 62.3 ± 12.7 years.

The concentrations of CTRP1 and CTRP3 proteins were significantly higher in individuals with CAD relative to controls. Except for serum HDL levels, which were lower in the CAD group than in the control group, there were no significant differences in the demographic or clinical features between the studied groups. A direct and significant association was discovered between the amounts of assessed adipokines and the severity of CAD.

CONCLUSION: A greater concentration of both CTRP1 and CTRP3 was strongly associated with the presence and severity of CAD, indicating that these adipocytokines may serve as markers for CAD.

Keywords: C1q-TNF-related Protein-1; CTRP1; C1q-TNF-Related Protein-3; CTRP3, Coronary Artery Disease

Introduction

Adipose tissue, functioning as a crucial endocrine organ, secretes diverse physiologically active proteins known as adipokines, which significantly contribute to the pathophysiology of obesity, diabetes, hypertension, and cardiovascular illnesses. One of the most important of these adipokines that has received significant attention in recent years is the C1q/TNF-related protein (CTRP) superfamily, which consists of 15 members, including CTRP1 to CTRP15[1]. These members not only modulate the signalling of hormones associated with carbohydrate and lipid metabolism but also significantly influence tissue inflammation levels[2].

CTRP1 is an adipokine primarily expressed in adipose tissue cells. Increased levels of CTRP1 in the bloodstream are associated with insulin resistance, cardiac failure, and hypertension[3–5].

CTRP3, a recently identified adipokine exhibiting sequence homology with adiponectin, plays a role in the regulation of metabolism and cardiovascular function. CTRP3 has been identified as a modulator of metabolism, endocrine system, and immunological processes[6–8].

Limited studies have examined the role of CTRPs in CAD patients, yielding inconsistent findings, especially for CTRP3 levels in the blood of individuals with CAD. To our knowledge, in one of the published studies[9], the concentration of CTRP3 in CAD patients was reported to be elevated compared to the control group, whereas in a few other studies, it was observed to be diminished[10–12]. Furthermore, only one study has employed the Gensini index to assess the severity of coronary artery disease (CAD), while the others relied on the count of occluded coronary arteries, which is not a precise metric[13,14]. In addition, the Gensini scoring system is not a perfect system, and contemporary scholars advocate for the utilization of alternative modified systems[15].

The primary objective of the present study was to assess the blood concentrations of CTRP1 and CTRP3 in individuals with CAD and to compare these levels with those in individuals without CAD. The correlation between the concentrations of these two adipocytokines and the existence and severity of the disease (measured by the modified Gensini index) was also examined.

Materials and Methods

Study participants and their characteristics

This case-control study involved 88 individuals (50 men and 38 women) who were referred to the angiography department of Shahid Beheshti Hospital in Kashan for chest pain. Among them, 47 patients had obstruction of more than 50% in at least one of the main coronary arteries and were selected as the case group, and 41 individuals without significant obstruction were considered as the control group.The sample size was calculated using a power analysis based on preliminary data obtained in our centre. Assuming a mean difference of 1.3 ng/mL in CTRP1 levels between groups, an SD of 2.2 ng/mL, alpha = 0.05, and power = 80%, the required minimum sample size was 39 subjects per group.

Clinical characteristics of the patients, including common risk factors for cardiovascular diseases, were recorded. To prevent individual errors, all measurements were taken by one person, and blood pressure was measured by a skilled physician after 15 minutes of rest. Patients who had a history of recent myocardial infarction (MI), blood disorders, metabolic or hormonal disorders, inflammatory diseases, or malignancy were excluded from the study.

The research was conducted in accordance with the Declaration of Helsinki, and written informed consent was acquired from all participants.

Angiographic analysis

Coronary angiography was performed for all patients using the Judkins technique. Two blinded cardiac surgeons analysed all the angiograms and calculated the modified Gensini score[16].

Biochemical analysis

After collecting all the samples, the serum of the study participants was sent to the clinical

laboratory to examine fasting blood glucose levels and lipid profiles. For each patient, the sample was thawed only once before biochemical analyses, and the relevant tests were conducted. The measurement of fasting blood glucose and lipid profile was carried out using diagnostic kits from Pars Azmoon Company (Iran) and the BT-3000 auto-analyser (Italy).

The serum concentrations of CTRP1 and CTRP3 were quantified utilising the sandwich ELISA technique and appropriate commercial kits produced by ZellBio GmbH, Wurttemberg, Germany.

Statistical analysis

Qualitative data were presented as percentages. Following the assessment of data distribution normality by the Kolmogorov–Smirnov test, the normally distributed parameters were expressed as the mean ± standard deviation (SD).

The independent sample t-test was used to assess differences between two groups, and a chi-square test was applied for categorical variables. Single and multiple logistic regression analyses were utilised to examine the relationship between adipokine levels and other risk factors associated with cardiovascular diseases in relation to the presence of coronary artery disease (CAD). In the single analysis, CAD was designated as the outcome variable, and the relationships between demographic, clinical, and biochemical variables were analysed sequentially in relation to CAD. Variables demonstrating a significant association with CAD in the single analysis were incorporated into the multivariate analysis. Data analysis was conducted using SPSS statistical software (Version 20), and a p-value < 0.05 was considered statistically significant.

Results

Table 1 shows the clinical, demographic, and laboratory characteristics of participants. We studied 88 individuals, including 41 patients with CAD in the case group and 47 individuals without CAD in the control group. The mean age of CAD patients was 62.1 ± 11.5 years. Thirty-one patients were male and sixteen were female.

Table 1. Clinical, demographic, and laboratory characteristics of participants with and without coronary artery disease

	Total Patients (N=88)	Control patients (N=41)	Patients with CAD (N=47)	P-Value
Age (years)	62.2 ± 12.0	62.3 ± 12.7	62.1 ± 11.5	0.990
Gender n (%)				
Male	50 (56.8)	19 (46.3)	31 (66)	0.064
Female	38 (43.2)	22 (53.7)	16 (34)	
BMI	26.8 ± 4.5	27.55 ± 4.99	26.07 ± 3.92	0.132
WHR	0.91 ± 0.05	0.89 ± 0.05	0.93 ± 0.03	0.125
SBP (mm /Hg)	129.1 ± 15.9	127.2 ± 17.4	130.7 ± 14.4	0.300
DBP (mm/ Hg)	80.2 ± 9.9	78.9 ± 9.9	81.3 ± 9.8	0.255
TG (mg/dL)	147.2 ± 61.4	145.3 ± 49.7	148.9 ± 71.4	0.208
Total cholesterol (mg/dL)	179.6 ± 42.1	185.2 ± 40.5	174.7 ± 43.3	0.242
HDL-C (mg/dL)	57.1 ± 11.7	60.9 ± 10.8	53.9 ± 11.6	0.004
LDL-C (mg/dL)	96.7 ± 23.8	100.6 ± 21.9	93.4 ± 25.1	0.158
CRRP1	5.3 ± 2.4	4.26 ± 2.03	6.20 ± 2.40	<0.001
CTRP3	13.7 ± 5.4	11.19 ± 3.07	15.97 ± 5.96	<0.001

Quantitative variables were expressed as mean ± SD and qualitative variables were expressed as frequency (percent). BMI: Body mass index; CTRP1: C1q-TNF-related protein-1; CTRP3: C1q-TNF-related protein-3; DBP: Diastolic blood pressure; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; SBP: Systolic blood pressure; TG: Triglyceride; WHR: Waist hip ratio.

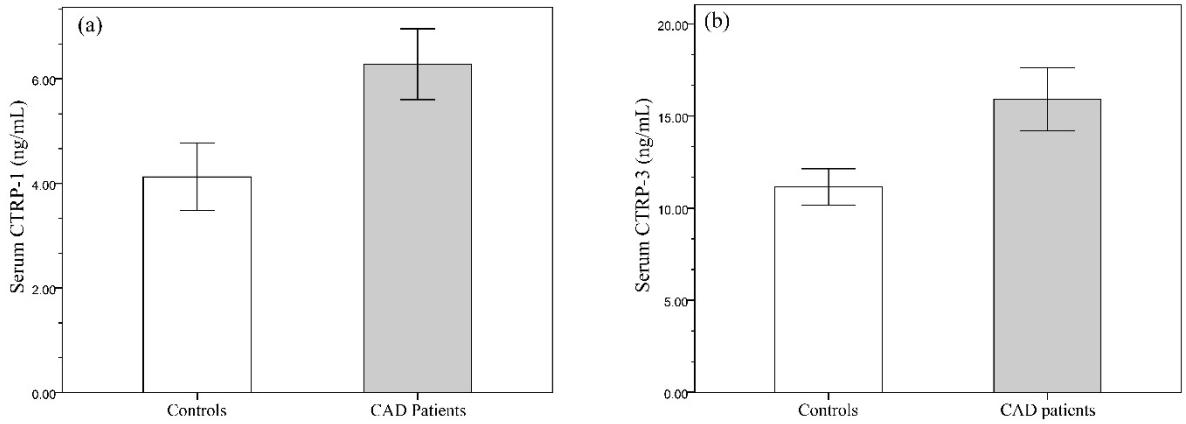

Figure 1. Serum levels of CTRP1 (a) and CTRP3 (b) in studied groups.

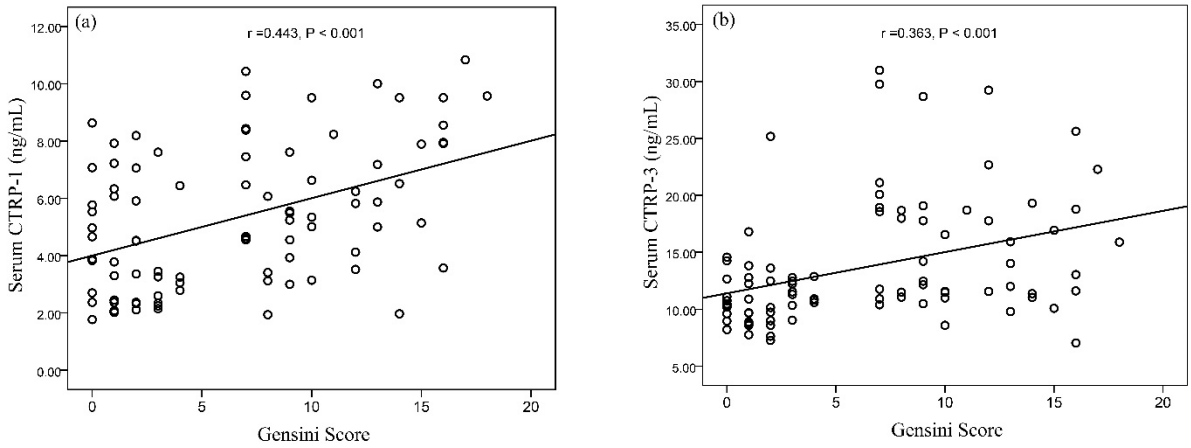

Figure 2. Correlation of serum levels of CTRP1 (a) and CTRP3 (b) with CAD severity in studied groups.

The control group comprised nineteen men and twenty-two women, with a mean age of 62.3 ± 12.7 years.

There were no significant differences in demographic or clinical characteristics between the two study groups, with the exception of serum HDL levels, which were lower in the patient group than in the control. As shown in Figure 1a, serum levels of CTRP1 were significantly higher in patients (6.20 ± 2.40) compared to the control group (4.26 ± 2.03, $p < 0.001$). As seen in Figure 1b, the serum level of CTRP3 was also significantly higher in the patient group (15.97 ± 5.96) compared to the control group (11.19 ± 3.07, $p < 0.001$).

Figures 2a and 2b respectively show the relationship between serum levels of CTRP1 and CTRP3 with the modified Gensini index. As seen in Figure 2a, there is a direct and statistically significant correlation between serum levels of CTRP1 and the severity of coronary artery stenosis ($r = 0.443$, $p < 0.001$). Figure 2b also shows a direct and significant relationship between serum levels of CTRP3 and the severity of coronary artery stenosis ($r = 0.363$, $p < 0.001$).

To ensure that the relationship between CTRP concentration and disease severity is not altered by confounding factors, a multiple linear regression test was used, and it was determined that this relationship is not significantly affected by demographic, clinical, and biochemical variables (Table 2). In addition, the examination of the relationship between adipokine levels and demographic and clinical variables showed that the level of CTRP3 is directly related to WHR and inversely related to HDL levels (Table 3).

Table 2. Multiple linear regression analysis between the modified Gensini score and demographic, clinical, and biochemical variables.

Variables	β (95% CI)	P-Value
Age	0.45 (-0.8–3.25)	0.671
Gender	0.25 (-0.4-0.224)	0.056
BMI	0.12 (-0.05-0.152)	0.912
WHR	0.22 (-0.52–0.110)	0.074
SBP	0.06 (-0.270–0.150)	0.607
DBP	0.04 (-0.17–0.34)	0.743
TG	0.09 (-0.15–0.20)	0.392
Total cholesterol	0.19 (-0.17–0.145)	0.532
HDL-C	0.14 (-0.83-3.77)	0.205
LDL-C	-0.09 (-0.560–0.890)	0.750
CRRP1	0.27 (0.216–0.946)	0.009
CTRP3	0.23 (0.190–0.860)	0.029

BMI: Body mass index; CTRP1: C1q-TNF-related protein-1; CTRP3: C1q-TNF-related protein-3; DBP: Diastolic blood pressure; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; SBP: Systolic blood pressure; TG: Triglyceride; WHR: Waist hip ratio.

Table 3. Correlation of CTRP1 and 3 with demographic, clinical and biochemical variables in all participants.

Variables	CTRP-1		CTRP-3	
	r	P-Value	r	P-Value
Age	-0.02	0.986	0.173	0.108
BMI	-0.099	0.361	-.0170	0.113
WHR	0.151	0.159	0.218	0.041
SBP	0.017	0.872	-0.022	0.836
DBP	-0.21	0.844	0.120	0.276
TG	0.089	0.412	0.042	0.699
Total cholesterol	-0.001	0.994	-0.148	0.169
HDL-C	-0.036	0.740	-0.330	0.002
LDL-C	0.027	0.801	-0.188	0.079
CTRP-1	-	-	0.330	0.002
CTRP-3	0.330	0.002	-	-

BMI: Body mass index; CTRP1: C1q-TNF-related protein-1; CTRP3: C1q-TNF-related protein-3; DBP: Diastolic blood pressure; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; SBP: Systolic blood pressure; TG: Triglyceride; WHR: Waist hip ratio.

Although there was a significant correlation between the levels of CTRP1 and CTRP3, no relationship was observed between CTRP1 and any of the demographic or clinical variables.

To determine the association between adipokines and CAD, single and multiple logistic regression analyses were performed between the study groups. All variables listed in Table 4 were regarded as independent. Single and multiple regression analyses showed that CTRP1, CTRP2, and HDL were significantly associated with CAD.

Discussion

The primary finding of this study was the elevated levels of CTRP1 and CTRP3 in patients with CAD relative to non-patients, alongside a direct and significant correlation with the presence and

Table 4. Association of adipokine levels and other risk factors with the presence of CAD.

	Total Patients (N=88)	Control patients (N=41)	Patients with CAD (N=47)	P-Value
Age (years)	62.2 ± 12.0	62.3 ± 12.7	62.1 ± 11.5	0.990
Gender n (%)				
Male	50 (56.8)	19 (46.3)	31 (66)	0.064
Female	38 (43.2)	22 (53.7)	16 (34)	
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WHR	0.91 ± 0.05	0.89 ± 0.05	0.93 ± 0.03	0.125
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TG (mg/dL)	147.2 ± 61.4	145.3 ± 49.7	148.9 ± 71.4	0.208
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HDL-C (mg/dL)	57.1 ± 11.7	60.9 ± 10.8	53.9 ± 11.6	0.004
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CRRP1	5.3 ± 2.4	4.26 ± 2.03	6.20 ± 2.40	<0.001
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Quantitative variables were expressed as mean ± SD and qualitative variables were expressed as frequency (percent). BMI: Body mass index; CTRP1: C1q-TNF-related protein-1; CTRP3: C1q-TNF-related protein-3; DBP: Diastolic blood pressure; HDL-C: High-density lipoprotein-cholesterol; LDL-C: Low-density lipoprotein-cholesterol; SBP: Systolic blood pressure; TG: Triglyceride; WHR: Waist hip ratio.

severity of CAD. Choi KM and colleagues first reported the levels of CTRP3 in patients with stable and unstable coronary artery disease in 2014[11]. They showed that the serum level of CTRP3 in both the stable and unstable groups was lower compared to the control group. In the same year, another study conducted by Fadaei and colleagues reported lower levels of CTRP3 in patients with CAD compared to the control group. About a year later, in 2017, Wang and colleagues, contrary to the findings of the two previous studies, showed that serum CTRP3 levels increase in patients with CAD[9]. In another recent study, the serum level of CTRP3 in both stable and unstable patient groups was reported to be lower compared to the control group[12]. These contradictions may be explained by the hypothesis that a defensive response to metabolic stresses or resistance to CTRP3 action results in elevated serum CTRP3 concentrations in CAD patients.

To our knowledge, in contrast to CTRP3, all research conducted so far on serum CTRP1 levels in patients with CAD has indicated elevated blood levels in these patients, regardless of whether they had stable or unstable CAD, in comparison to control subjects[17–21]. Consistent with these investigations, the current research also revealed that the concentration of CTRP3 was elevated in persons with CAD relative to the control group.

Evidence from various studies indicates that the involvement of CTRP1 in CAD and the process of atherosclerosis may occur through the induction of mild chronic inflammation. CTRP1 stimulates the expression of adhesion molecules and the synthesis of inflammatory cytokines via the NF-κB and MAPK pathways, enhancing leukocyte adherence to endothelial cells[22]. These data suggest that CTRP1 may play a role in the advancement of thrombosis, a critical element in the onset and worsening of CAD[23].

Additionally, in the present study, a direct correlation between the level of CTRP3 and WHR was observed. This may be due to the higher amount of visceral fat tissue in individuals with abdominal obesity, which produces more CTRP3 compared to subcutaneous fat tissue[24].

Despite published studies aiming to elucidate

the correlation between plasma levels of CTRPs and the severity of coronary artery stenosis, most have failed to employ suitable indices for evaluating stenosis severity. In fact, it would have been better to use the modified Gensini score in these studies, which is recognised as the preferred method for determining the severity of stenosis.

The current investigation encounters some limitations that require attention. Initially, we examined a comparatively limited sample; hence, our findings require validation in a more extensive statistical cohort. Secondly, the study was conducted on a population of Iranian nationality, so its conclusions cannot be applied to other societies. Ultimately, although intravascular ultrasonography (IVUS) is more precise than angiography, we were unable to utilize IVUS due to cost constraints[25,26].

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Conflict of interests
The authors declare no conflict of interest.

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Author's Contributions
Study Conception or Design: GN, MM
Data Acquisition: MM, AM, RS, FR
Data Analysis or Interpretation: GN
Manuscript Drafting: GN, MM
Critical Manuscript Revision: GN
All authors have approved the final manuscript and are responsible for all aspects of the work.

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