



# Correlation between Plasma Adiponectin Levels and the Presence and Severity of Coronary Artery Disease

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## Abstract

**Background:** The existing evidence suggests that plasma adiponectin concentrations can be indicative of the presence and severity of coronary artery disease (CAD). However, the results of the studies conducted hitherto on this subject are inconsistent. We sought to investigate the possible correlation between plasma adiponectin levels and the presence and severity of CAD in patients undergoing non-urgent coronary angiography.

**Methods:** In 399 consecutive patients undergoing non-urgent coronary angiography for CAD survey, plasma adiponectin, triglyceride, total cholesterol, high-density lipoprotein and low-density lipoprotein cholesterol, and fasting blood sugar levels were measured and demographic characteristics such as age, sex, Body Mass Index, diabetes mellitus history, systemic hypertension history, and family history of CAD were collected. According to the angiography results, the patients were divided into two groups of CAD and non-CAD. The severity of coronary atherosclerosis in the CAD group was defined using the Gensini score system.

**Results:** Average age was  $61.4 \pm 9.94$  years in the CAD group and  $57.9 \pm 10.75$  years in the non-CAD group. Also, 73.7% of the CAD group and 55.4% of the non-CAD group were male. Totally, 278 (69.7%) patients were found to have CAD. Patients without CAD did not have higher mean plasma adiponectin concentrations than did those with CAD ( $13.38 \pm 11.96$  vs.  $14.95 \pm 14.11$  mcg/ml;  $p$  value = 0.896). After adjustment for CAD conventional risk factors, plasma adiponectin levels still were not associated with CAD. No association was found between plasma adiponectin levels and the Gensini score. Furthermore, in contrast to the fairly strong correlation previously reported, there was no correlation between adiponectin levels and conventional CAD risk factors.

**Conclusion:** We could not observe any relationship between plasma adiponectin concentrations and the presence or severity of CAD in patients undergoing coronary angiography.

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**Keywords:** Adiponectin • Coronary artery disease • Risk factors • Coronary angiography

## Introduction

Adiponectin (ARCP 30, AdipoQ, apM1 or GBP28),

secreted by the adipose tissue, is a 247-amino acid peptide which was discovered in 1995.<sup>1, 2</sup> The circulating level of this peptide ranges from 5 to 30  $\mu$ g/ml, accounting for

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about 0.01% of total plasma protein, three times higher than the concentrations of most other adipose tissue-derived hormones.<sup>3</sup> Adiponectin has gained particular interest on account of its relation with insulin sensitivity, atherosclerosis, and inflammation.<sup>4</sup> Several experimental studies have reported the anti-atherogenic and anti-inflammatory effects of adiponectin.<sup>5,6</sup>

In human studies, not only were plasma adiponectin concentrations lower in patients with coronary artery disease (CAD) than in age-matched and body mass index (BMI)-matched controls,<sup>7</sup> but also these levels were inversely allied to other traditional cardiovascular risk factors such as diabetes, blood pressure, total and low-density lipoprotein (LDL) cholesterol, and triglyceride levels.<sup>8,9</sup> An association has also been observed between hypoadiponectinemia and early CAD onset and multiple atherosclerotic lesions in coronary arteries.<sup>10</sup> It seems, therefore, that plasma adiponectin levels predict CAD atherosclerotic burden, even after adjustment for the effect of its predictors.<sup>11</sup>

Recent findings suggest that high adiponectin concentrations are an independent predictor of mortality in chronic heart failure patients, chronic kidney patients, and elderly patients, who are at high risk for cardiovascular events.<sup>11-15</sup> Furthermore, plasma adiponectin concentrations both tend to show variable levels in people of different countries and races<sup>16-19</sup> and tend to be higher in women,<sup>20</sup> although there is evidence of a significant correlation between adiponectin and the extent of CAD in men.<sup>21</sup> In contrast, there have also been studies unable to demonstrate a good association between low levels of adiponectin and an increased risk for CAD.<sup>22,23</sup>

Accordingly, given that the clinical importance of hypoadiponectinemia in CAD has yet to be fully elucidated, we aimed to investigate whether plasma adiponectin concentrations could be a marker for the presence and severity of coronary atherosclerosis in these patients.

## Methods

This cross-sectional study recruited 399 patients admitted to the clinics of Tehran Heart Center, who had symptoms or signs of CAD or objective evidence of myocardial ischemia and were candidates for elective coronary angiography. Patients with acute coronary syndromes, valvular heart disease, renal and hepatic dysfunction, systolic heart failure, and any systemic illness were excluded. This study was approved by our institutional Review Board for Protecting Human Rights.

Pre-angiography data included demographic characteristics, past medical history, and history of cardiac risk factors. As a hospital routine, a 10-cc venous volume of fasting blood sample was taken on the morning prior to coronary angiography for the analysis of the following

parameters using standard techniques after immediate centrifugation: glucose; triglycerides; total cholesterol; high-density lipoprotein (HDL); and LDL cholesterol. Total adiponectin levels were measured using a commercially available immunoassay method, human adiponectin ELISA kit (Bio Vendor, Modřice, Czech; Cat. No. RD 195023100). The study population's serum samples were kept at -80 °C before use.

The BMI was expressed as kilograms per meter square (kg/m<sup>2</sup>). The definitions of the CAD risk factors and angiographic procedures in our center have been previously published.<sup>24</sup>

Coronary angiography was performed via the Judkins technique through the femoral artery access. The patients were categorized into two groups based on their diagnostic angiograms: patients with at least one stenosis >50% in a major epicardial vessel comprised the CAD group, and those with <50% stenosis formed the non-CAD group.

The Gensini score was employed to quantify the severity of CAD in the CAD group and was defined according to stenosis severity as one point for <25% stenosis, two points for 26 to 50% stenosis, four points for 51 to 75% stenosis, eight points for 76 to 90% stenosis, and 32 points for total occlusion. The calculated scores were thereafter multiplied according to factors that define the importance of a stenosis site.<sup>25</sup>

Stenosis severity was estimated visually in at least two orthogonal views by two expert cardiologists.

The results are presented as mean ± SD (standard deviation) for the quantitative variables, and are summarized by absolute frequencies and percentages for the categorical variables. The continuous variables were compared using the Student t-test or nonparametric Mann-Whitney U test whenever the data did not appear to have normal distributions, while the categorical variables were compared using the chi-square or Fisher exact test, as appropriate.

The association between the continuous variables was assessed using the Spearman correlation coefficient.

For the statistical analysis, the statistical software SPSS version 15.0 for Windows (SPSS Inc., Chicago, IL) was used. All *p* values were 2-tailed, with statistical significance defined by a *p* value ≤ 0.05.

## Results

Of the 399 study patients, 278 patients comprised the CAD group and 121 patients were classified as the non-CAD group (control subjects). The baseline characteristics of the two groups are depicted in Table 1. According to Table 1, in comparison with the controls, the CAD group patients were older and were more likely to be a male and smoker. Comparisons between the serum LDL and HDL levels as well as the Gensini score showed statistically significant differences between the CAD and control groups (*p* value <

Table 1. Levels of adiponectin and other basic characteristics in the case and control groups\*

Variables	Cases with abnormal angiography (n=278)	Controls (n=121)	P value
Age (y)	61.41±9.94	57.92±10.75	0.002
Sex (Male)	205 (73.7)	67 (55.4)	0.001
BMI (kg/m <sup>2</sup> )	27.45±4.97	28.13±4.63	0.092
Family history	93 (33.5)	39 (32.2)	0.812
Hypertension	116 (41.7)	50 (41.3)	0.940
Diabetes mellitus	73 (26.3)	24 (19.8)	0.169
Cigarette smoker	63 (22.7)	15 (12.4)	0.017
MI history	118 (42.4)	13 (10.7)	0.001
Dyslipidemia	187 (67.3)	76 (62.8)	0.388
Drug history			
Thrombolytic agent	31 (11.2)	3 (2.5)	0.004
ASA	253 (91.0)	105 (86.8)	0.201
ACEIs	105 (37.8)	29 (24.0)	0.007
B-blocker	229 (82.4)	94 (77.7)	0.273
Nitrate	231 (83.1)	83 (68.6)	0.001
Calcium channel blocker	70 (25.2)	16 (13.2)	0.008
Antihyperlipidemics	85 (30.6)	25 (20.7)	0.042
Adiponectin (mcg/ml)	14.63±13.99	14.14±12.44	0.896
Ejection fraction (%)	49.41±11.02	55.97±7.26	0.001
Triglyceride (mg/dl)	187.52±134.50	187.63±93.35	0.238
Total cholesterol (mg/dl)	185.40±47.18	179.78±40.11	0.254
Low-density lipoprotein (mg/dl)	109.50±40.29	97.36±39.97	0.010
High-density lipoprotein (mg/dl)	40.52±10.82	45.22±10.19	0.001
Gensini score	65.08±46.33	2.21±3.42	0.001

\*Data are presented as mean ± SD or n (%)

BMI, Body Mass Index ; MI, Myocardial infarction; ASA, Acetylsalicylic acid; ACEIs, Angiotensin-converting enzyme inhibitors

Table 2. Bivariate Spearman correlation of serum adiponectin with metabolic and cardiovascular indices

Variables	Correlation with Adiponectin (r)	P value
Total cholesterol (mg/dl)	0.010	0.841
Triglyceride (mg/dl)	-0.090	0.071
High-density lipoprotein (mg/dl)	0.020	0.692
Low-density lipoprotein (mg/dl)	0.045	0.374
Fasting blood sugar (mg/dl)	-0.028	0.588
Body Mass Index (kg/m <sup>2</sup> )	-0.050	0.322
Ejection fraction (%)	-0.064	0.202
Creatinine (mg/dl)	-0.092	0.071

0.010, p value < 0.001, and p value < 0.001, respectively). Also, previous use of thrombolytic agents, angiotensin-converting enzyme inhibitors (ACEIs), nitrates, calcium-channel blockers, and antihyperlipidemic agents was higher in the CAD patients.

Plasma adiponectin levels were similar between the cases and controls (Table 1). Comparison of adiponectin concentrations between the men and women revealed no significant differences. The bivariate Spearman correlation

coefficient test was used to determine the correlation between serum adiponectin concentrations and the BMI and biochemical variables between the two groups (Table 2), and the results demonstrated a non-significant correlation between adiponectin levels and the BMI, cardiac ejection fraction, and serum creatinine level. Moreover, no correlation was found between serum adiponectin levels and total cholesterol (TC), triglyceride (TG), HDL levels, and LDL levels. Adiponectin had no linear correlation with the



Table 3. Logistic regression model using the Enter technique for adjusting the results

	P value	Odds ratio	Lower confidence interval	Upper confidence interval
Adiponectin	0.582	1.005	0.986	1.025
Diabetes mellitus	0.006	2.277	1.259	4.115
Age	< 0.001	1.045	1.021	1.070
sex	< 0.001	2.762	1.644	4.641
High-density lipoprotein	< 0.001	0.963	0.943	0.983
Low-density lipoprotein	0.004	1.009	1.003	1.014
Cigarette smoking	0.101	1.728	0.899	3.323
Constant	0.031	0.130		

Gensini score in the CAD group (Figure 1).

After adjustment for age, gender, cigarette smoking, LDL, HDL, and diabetes mellitus (DM) (factors that could be potentially confounders), the association between adiponectin concentrations and the presence of CAD was non-significant (Table 3).

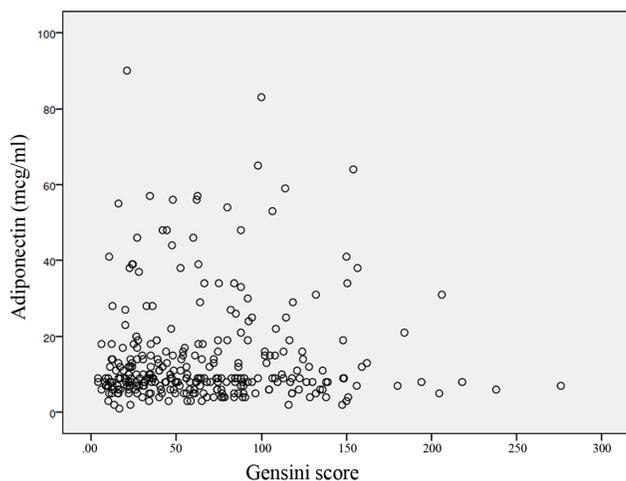


Figure 1. Adiponectin and Gensini score correlation in the coronary artery disease group

## Discussion

The current research demonstrated that the mean serum level of adiponectin was not significantly different between groups with and without angiographic findings of CAD, even after adjustment for other related factors. Importantly, no evidence of significant association was found between the plasma levels of adiponectin and the patients' demographics, CAD risk factors, and cardiovascular indices.

In line with the available experimental evidence in terms of supporting the vasoprotective effect of adiponectin, several clinical investigations have reported reduced circulating adiponectin levels in patients with CAD.<sup>4, 10, 26, 27</sup> However, the associations reported between adiponectin levels and risk of CAD in these studies are moderate. In contrast, there

are some studies that have not been able to demonstrate any correlation between low levels of adiponectin and an increased risk for CAD.<sup>22, 23, 28, 29</sup> In a study conducted by Sattar et al.,<sup>28</sup> 589 incident cases of CAD and 1,231 controls were compared and also a meta-analysis on six previous studies was performed. Nevertheless, no association was detected between adiponectin levels and cardiovascular disease risk. The most recent case-control study, on 1,035 CAD patients and 1,920 age- and gender-matched controls, after a mean 7.7 years of follow-up, did not suggest that measurement of adiponectin levels was useful to refine cardiovascular disease risk assessment where the traditional risk factors are considered.<sup>30</sup>

A relationship was reported between obesity and serum adiponectin concentrations by Mamaghani et al.,<sup>31</sup> who reported that adiponectin concentrations in overweight and obese non-diabetic women were lower than in normal weight subjects. In contrast, in the Sattar et al.<sup>28</sup> study on two groups with significantly different BMI values, the mean values of the plasma concentrations of adiponectin did not differ significantly.

Our findings chime in with the results obtained from the Mozafari et al. study,<sup>32</sup> demonstrating that the serum levels of adiponectin were not significantly different between patients with and without CAD as confirmed by angiography. According to the authors, the participants in their study had indication for coronary angiography by obvious clinical findings. It is, therefore, prudent to consider that patients with normal coronary angiography may have involvement of subepicardial or small vessels, which cannot be found on coronary angiography. This may explain why the levels of serum adiponectin were similar in both groups, but significantly lower than those in the normal population, as specified by the diagnostic kit.

The reasons for the differences between the varieties of evidence are unknown. One of the probable suggestions would be partly attributed to the differences in the study power, the estimating difference which has been considered significant, and variations in the general plans of the studies. Secondly, it is possible that adiponectin also may affect other unknown biological factors which play an opposite role in CAD by modulating them such as insulin resistance

or dyslipidemia. Thirdly, a variety of confounding factors could be responsible for the variation in the results of the studies. For example, inflammatory phase mediators such as C-reactive protein (CRP) could be mentioned in this regard. Fourthly, compared with the general population, the prevalence of CAD risk factors was higher in both of our study groups; consequently, the CAD risk factor prevalence was not totally different between the CAD and non-CAD groups. (Only age, sex, history of smoking, previous myocardial infarction, HDL levels, and LDL levels had statistically significant differences between the two groups.) Although the age distribution between the CAD group and the non-CAD group showed statistically significant differences between the two groups ( $p$  value = 0.002), the mean value for age was higher than that in the normal population: this could explain the similarity in terms of the CAD risk factor profile between the CAD and non-CAD groups. And finally, the differences between the study results could be due to the estimation of different molecular forms of adiponectin.<sup>28</sup>

To the best of our knowledge, there is a paucity of research on plasma adiponectin levels in the Iranian race.<sup>31,32</sup> What is more, various mean levels of serum adiponectin have been reported in these studies and the employment of different study designs precludes a comparison between their results. Mamaghani et al.<sup>30</sup> included 157 non-diabetic women with different grades of obesity and reported mean values of serum adiponectin of  $25.55 \pm 6.1 \mu\text{g/mL}$  and  $19.55 \pm 4.9 \mu\text{g/mL}$  in the normal and grade III obese women ( $\text{BMI} \geq 40 \text{ kg/m}^2$ ), respectively. In contrast, the Mozafari et al. study,<sup>32</sup> which encompassed stable angina patients candidated for coronary angiography, revealed that the serum levels of adiponectin were lower than the minimum levels specified by the kit ( $10.1 \mu\text{g/mL}$  vs.  $14 \mu\text{g/mL}$ , respectively). The results of the present study on Iranian samples showed no statistically significant differences in terms of the mean value of plasma adiponectin concentrations between the male and females groups.

## Conclusion

In keeping with the results of several available studies, we could not find any association between adiponectin concentrations and the presence or severity of CAD. Further studies taking probable mediators into consideration are required to shed more light on this issue.

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