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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Relationship between serum adiponectin levels and calcific aortic valve disease

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Abstract

Background: Adiponectin, an adipose tissue derived cytokine, is known to have antiatherogenic and anti-inflammatory effects on endothelial cells and macrophages. Calcific aortic valve disease has a similar physiopathology to atherosclerosis.

Aim: To investigate the relationship between adiponectin and calcific aortic valve disease.

Methods: The study group consisted of 58 patients with calcific aortic stenosis and 24 healthy controls. Aortic stenosis patients were divided into three groups according to their valvular areas: mild (n = 11), moderate (n = 25), and severe (n = 22). Serum adiponectin levels and other biochemical parameters were measured.

Results: The aortic stenosis and control group were similar in terms of age, gender and cardiovascular risk factors. Adiponectin median values did not differ significantly between two groups (2.19 μg/mL [1.43–3.18], 1.79 μg/mL [1.34–3.42] aortic stenosis and control group, respectively; p = 0.7). Aortic stenosis patients were divided into three groups according to their valvular area as mild, moderate and severe. There were no differences when we compared adiponectin levels among those groups (mild: 2.10 μg/mL [1.47–3.31], moderate: 2.13 μg/mL [1.44–2.91], severe: 2.65 μg/mL [1.28–3.43]; p = 0.67). Age (r = 0.26, p = 0.045) and aspartate aminotransferase (r = 0.28, p = 0.04) had positive correlations with adiponectin; while white blood cell count (r = –0.32, p = 0.015), fasting blood glucose (r = –0.29, p = 0.03), haemoglobin (r = –0.27, p = 0.04) and triglyceride levels (r = –0.41, p = 0.002) had negative correlations.

Conclusions: In our study, we did not find a relationship between adiponectin levels and calcific aortic valve disease.

Key words: aortic stenosis, coronary artery disease, adiponectin, calcific aortic disease

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INTRODUCTION

Calcific valvular aortic stenosis (AS) is a chronic disease that has slowly progressing course. Previously, it had been thought that calcific AS was generated from tearing of the leaflets and passive calcium deposition over degenerated valves. However, recent findings have suggested that it has an active physiopathology similar to atherosclerosis which includes lipoprotein accumulation, chronic inflammation and calcification. As calcific aortic disease and atherosclerosis have similar risk factors, and there are correlations between coronary heart disease and the severity of the aortic calcification, their pathogenesis is thought to be common [1]. Adiponectin is an adipocytokine that is known to have an important role in hyperglycaemia, dyslipidaemia and inflammatory processes. Though adiponectin's definite physiologic role has not been clearly elucidated, this cytokine has been shown to have antiatherogenic and anti-inflammatory effects on endothelial cells and macrophages [2]. Besides, lower levels of adiponectin have been demonstrated in patients with atherosclerosis. In the literature, data regarding adiponectin levels in calcific AS is scarce. In this study, we aimed to determine adiponectin levels in patients with calcific AS at various stages of the disease.

METHODS

Study patients

Patients admitted to Türkiye Yüksek Ihtisas Hospital Cardiology and Cardiovascular Surgery departments between November 2009 and March 2010 and diagnosed with calcific AS were included in our study. Patients with AS that was caused by any other aetiology including rheumatic heart disease or bi-

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cuspid aorta, patients with decompensated heart failure (HF), rheumatologic mitral or tricuspid valve pathology, acute or inflammatory disease were excluded from the study. Fifty-eight consecutive patients were included. There were 30 male and 28 female patients in the AS group, whereas the control group consisted of 13 males and 11 females. Twenty four subjects of similar age and gender profile without valvular disease were included as control group. All patients were questioned about having hypertension, diabetes mellitus, hyperlipidaemia, coronary artery disease (CAD), and HF. The study was approved by the Institutional Research Ethics Committee and informed consent was obtained from each patient.

Echocardiography

All patients underwent echocardiography by GE-Vingmed Vivid 7 System. Left ventricular ejection fraction was measured by using modified Simpson method. Continuous wave Doppler was used to detect aortic valvular velocities and gradients. Aortic valvular areas (AVA) were calculated by using continuity equation. AVA > 1.5 cm^2 were classified as mild, AVA between 1.0–1.5 cm^2 as moderate, and AVA < 1.0 cm^2 as severe AS.

Biochemical analyses

After 12 hours of fasting, venous blood samples were obtained from all patients. Plasma concentrations of total cholesterol, high density lipoprotein cholesterol, triglyceride, glucose, aspartate aminotransferase, creatinine, C-reactive protein (CRP), and adiponectin levels were measured and complete blood count parameters were calculated. Low density lipoprotein cholesterol levels were calculated using the Frieadwald formula. Plasma concentrations of adiponectin were measured by solid phase enzyme linked immunosorbent assay (ELISA) with a commercially available kit (RayBio® Human Adiponectin/Acrp30 ELISA Kit).

Statistical analysis

All analyses were performed by SPSS version 17 (Chicago, IL, USA) software. Parametric variables were presented as median with an inter-quartile range and they were compared with Mann Whitney U or Kruskall Wallis tests. Non parametric variables were presented as n (%) and compared with χ^2 test. The parameters that may interact with adiponectin were analysed by Spearman's correlation analysis. A two-tailed p value < 0.05 was considered to be statistically significant.

RESULTS

Demographic and clinical characteristics of study subjects with blood analyses are summarised in Table 1. Age, gender, history of hypertension, diabetes mellitus, hyperlipidaemia, CAD and HF did not differ between groups. Median left ven-

Table 1. Demographic and clinical characteristics of the study subjects with biochemical blood analyses

	All patients (n = 82)	Control (n = 24)	AS (n = 58)	P
Age [years]	71.0 (64.8–75.0)	71.0 (67.3–73.0)	70.5 (61.8–75.0)	0.599
Gender [F/M]	47.6%/52.4%	45.8%/54.2%	48.3%/51.7%	1
Body mass index [kg/m^2]	27.0 (24.1–30.3)	27.1 (24.7–29.9)	27.0 (24.0–31.2)	0.72
Waist circumference [cm]	95.5 (87.0–102.3)	94.5 (91.0–103.8)	96.0 (86.8–102.0)	0.88
Hypertension	63.40%	70.80%	60.30%	0.454
Diabetes mellitus	19.50%	25%	17.20%	0.541
Hyperlipidaemia	54.90%	50%	56.90%	0.63
Coronary artery disease	42.70%	29.20%	48.30%	0.143
Heart failure	6.10%	0%	8.60%	0.315
LVEF [%]	60.0 (58.0–65.0)	64.0 (60.0–65.0)	60.0 (55.0–62.0)	0.002*
Adiponectin [μg/mL]	2.12 (1.41–3.23)	1.79 (1.34–3.42)	2.19 (1.43–3.18)	0.737
CRP [mg/L]	0.45 (0.27–0.93)	0.5 (0.2–0.8)	0.43 (0.29–1.03)	0.726
Total cholestrol [mg/dL]	189.0 (154.0–228.0)	187.0 (158.0–213.0)	192.0 (149.0–231.0)	0.904
LDL [mg/dL]	116.0 (87.0–149.0)	118.0 (88.0–144.0)	114.0 (87.0–149.0)	0.95
HDL [mg/dL]	44.0 (35.0–51.0)	45.0 (35.0–50.0)	43.0 (34.0–50.0)	0.95
Triglyceride [mg/dL]	120.0 (89.0–174.0)	128.0 (99.0–186.0)	116.0 (80.0–171.0)	0.257
Glucose [mg/dL]	10.0 (95.0–116.0)	104.0 (95.0–116.0)	102.0 (95.0–116.0)	0.94
AST [U/L]	21.0 (18.0–27.0)	20.0 (17.0–25.0)	21.0 (18.0–28.0)	0.29

*Statistically significant; Range was expressed as 25^{th}–75^{th} percentile for parametric variables; AS — aortic stenosis; AST — aspartate aminotransferase; CRP — C-reactive protein; F/M — female/male; HDL — high density lipoprotein; LDL — low density lipoprotein; LVEF — left ventricular ejection fraction

tricular ejection fraction was 60% (55–62%) in the AS group, while it was 64% (60–65%) in the control group (p = 0.002). Adiponectin levels did not differ between groups (Fig. 1).

The AS group was divided into three groups according to their valvular area. Eleven patients were classified as mild, 25 patients as moderate, and 22 patients as severe AS. Clinical and demographic characteristics with adiponectin levels of these subgroups are shown in Table 2. Hypertension was more frequent in the mild AS group (p = 0.006). Adiponectin levels did not differ between subgroups (Fig. 2).

Age (r = 0.26, p = 0.045) and aspartate aminotransferase (r = 0.28, p = 0.04) were positively correlated with adiponectin levels, whereas white blood cell count (r = –0.32, p = 0.015), fasting blood glucose (r = –0.29, p = 0.03), triglyceride (r = –0.41, p = 0.002), and haemoglobin (r = –0.27, p = 0.04) were inversely correlated with adiponectin levels.

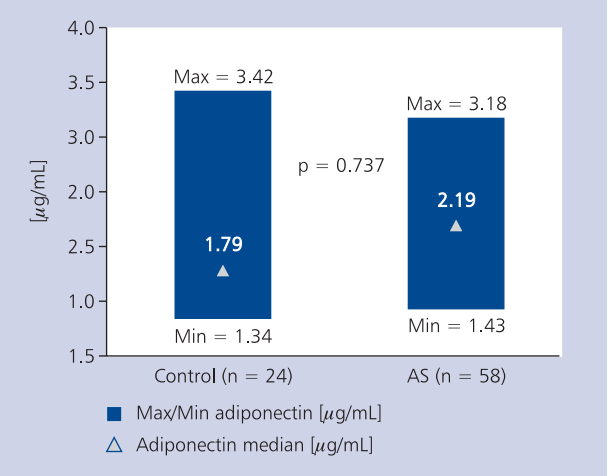

Figure 1. Adiponectin levels in aortic stenosis (AS) patients and control group; Min — minimum; Max — maximum

Table 2. Clinical, echocardiographic, and demographic characteristics and biochemical blood analyses of the subgroups

	Aortic stenosis (n = 58)			P
	Mild (n = 11)	Moderate (n = 25)	Severe (n = 22)	
Age [years]	69.0 (65.0–73.0)	71.0 (59.5–75.5)	70.5 (63.8–74.3)	0.988
Gender [F/M]	45.5%/54.5%	40.0%/60.0%	59.1%/40.9%	0.417
Body mass index [kg/m^2]	28.7 (27.5–29.7)	26.2 (24.0–31.2)	25.8 (22.2–33.2)	0.288
Waist circumference [cm]	100.0 (94.0–102.0)	97.0 (88.5–104.0)	92.5 (76.8–101.5)	0.252
Hypertension	90.9%	68%	36.4%	0.006*
Diabetes mellitus	36.4%	12%	13.6%	0.174
Hyperlipidaemia	54.5%	56%	59.1%	0.963
Coronary artery disease	36.4%	56%	45.5%	0.524
Heart failure	0%	4%	18.2%	0.118
LVEF [%]	60.0 (55.0–60.0)	60.0 (58.0–64.5)	60.0 (53.0–60.0)	0.295
Adiponectin [μg/mL]	2.10 (1.47–3.31)	2.13 (1.44–2.91)	2.65 (1.28–3.43)	0.671
CRP [mg/L]	0.51 (0.28–1.46)	0.42 (0.30–0.93)	0.43 (0.17–1.32)	0.985
Total cholestrol [mg/dL]	160.5 (142.0–213.3)	196.0 (156.3–225.0)	215.0 (150.5–235.0)	0.574
LDL [mg/dL]	106.5 (78.0–145.8)	110.0 (86.3–140.5)	132.0 (97.0–153.5)	0.601
HDL [mg/dL]	44.0 (38.3–50.3)	42.5 (30.0–49.8)	42.0 (34.5–53.0)	0.759
Triglyceride [mg/dL]	97.0 (69.5–130.5)	133.0 (93.5–207.8)	112.0 (78.5–190.0)	0.604
Glucose [mg/dL]	117.5 (92.5–144.8)	103.0 (95.5–112.0)	101.0 (94.5–112.5)	0.516
AST [U/L]	21.5 (16.5–28.0)	21.0 (18.0–24.0)	23.0 (18.0–47.5)	0.443
AVA [cm^2]	1.70 (1.56–1.90)	1.15 (1.10–1.32)	0.81 (0.70–0.90)	0.000*
Aortic regurgitation:				0.439
Severe	0%	0%	9.1%	
Moderate	27.3%	28%	31.8%	
Mild	63.6%	72%	54.5%	
None	9.1%	0%	4.5%	

*Statistically significant; Range was expressed as 25th–75th percentile for parametric variables; AST — aspartate aminotransferase; AVA — aortic valvular area; CRP — C-reactive protein; F/M — female/male; HDL — high density lipoprotein; LDL — low density lipoprotein; LVEF — left ventricular ejection fraction

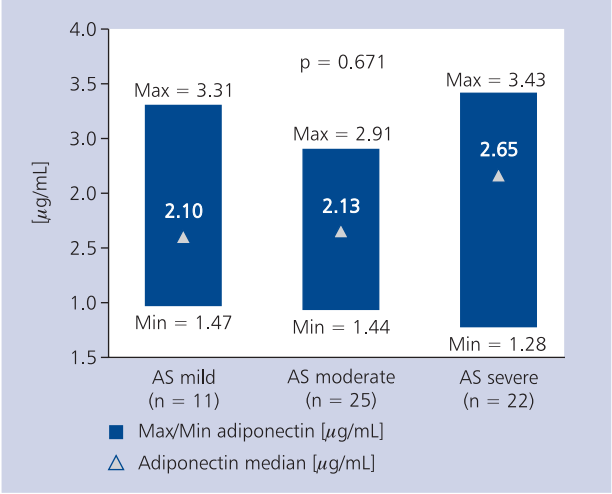

Figure 2. Adiponectin levels in aortic stenosis (AS) group according to severity of the valvular area; Min — minimum; Max — maximum

DISCUSSION

Atherosclerosis is a chronic inflammatory process that is characterised by endothelial dysfunction, vascular inflammation, and accumulation of lipids and inflammatory cells through the intima layer. Like atherosclerosis, in calcific AS, chronic process is triggered by endothelial injury over the mechanical stress regions of the aortic valve. Degeneration of the endothelium is seen in the earlier phases, whereas inflammatory cell infiltration, lipid accumulation, thickening of the fibrosa with collagen and calcium deposition occur during the following stages [1]. Adiponectin is an important cytokine secreted by adipose tissue. It is reduced in certain metabolic conditions such as diabetes mellitus, insulin resistance, metabolic syndrome and obesity [3–6]. It has been revealed that adiponectin has anti-inflammatory and antiatherosclerotic effects on endothelial cells and macrophages [2, 7–9]. The levels of adiponectin have been found to be lower in patients with CAD [10–13].

Although lower levels of adiponectin were expected in patients with AS (as in patients with atherosclerosis), serum levels of adiponectin did not differ significantly between patients with degenerative AS and the control group. Cote et al. [14] evaluated valvular inflammation and oxidative stress parameters in patients with AS. They showed a positive correlation between adiponectin levels and tumour necrosing factor-alpha, and a negative correlation with interleukin-6. They found that higher oxidative stress and activation of the renin angiotensin system contribute to higher valvular inflammation in AS, but they did not evaluate the effect of adiponectin during that process. A recent study by Kolasa-Trela et al. [15] did not find a significant difference in adiponectin levels between AS patients and controls, as in our study.

Although several studies have claimed lower levels of adiponectin in CAD, other studies have not [16]. Interestingly, Nakamura et al. [11] found decreased adiponectin levels in patients with acute coronary syndrome; while they did not find a difference in patients with stable angina pectoris. In another study, calcium score and severity of the CAD were not found to be related with adiponectin levels [17]. Also in a recent work investigating adiponectin levels within various plaque compositions by multidetector computed tomography, it has been found that patients with non-calcified plaques had significantly lower levels of adiponectin. However, in the same study, adiponectin levels were not associated with calcified plaques [18]. By using intravascular ultrasound, Marso et al. [19] demonstrated that lipid formed vulnerable plaques and intimal thickening were related with lower serum adiponectin levels. Those studies have suggested that adiponectin is associated with an antiatherogenic role in the early stages of disease (vulnerable plaque) but its role decreases as the disease progresses (calcified plaque). There are studies claiming that adiponectin inhibits proatherogenic endothelial activation in the early phases of atherosclerosis, while its levels rise in line with the inflammatory cytokines during the chronic stages [20–22]. Furthermore, inflammation, lipotoxicity and oxidative stress are thought to increase adiponectin expression [20, 23]. As it has anti-inflammatory and antioxidative properties, adiponectin levels may rise in such conditions in a compensatory way [24, 25].

This data gives rise to the thought that adiponectin is prominently active in the earlier stages of atherosclerosis with its anti-inflammatory effects, which probably was the main cause of observing similar adiponectin levels between groups in our study. Also in AS patients, with the progression of the disease, calcification becomes more prominent which resembles plaque stabilisation in atherosclerosis. Likewise, the medications used to treat atherosclerosis have not been found to be effective in AS [26, 27]. The histological appearance of aortic sclerosis, the antecedent lesions of calcific AS, looks like vulnerable atherosclerotic plaques when the calcification has not been dominant yet. Patients with aortic sclerosis have common cardiovascular risk factors and endothelial dysfunction signs [28]. Theoretically, adiponectin levels should be low in aortic sclerosis. The vast majority of our study patients consisted of moderate and severe AS, that may have affect the results.

In our study, we observed a negative correlation between adiponectin and triglyceride levels which was in accordance with recent studies [29, 30]. There was a positive correlation between age and adiponectin levels. Increased adiponectin levels in the elderly have been shown to be related to higher mortality which was thought to have occurred in response to homeostatic dysregulation [11, 27, 31, 32]. Adiponectin has effects on glucose metabolism, such as augmenting insulin sensitivity or reducing plasma glucose levels [33]. We also observed an adverse relation between fasting blood glucose and adiponectin levels. In our research, there was a negative

correlation between white blood cell count and adiponectin. Likewise, it was revealed that adiponectin has inhibitory effects on granulocyte-monocyte precursors, which supports its anti-inflammatory properties [34]. Our findings did not detect a correlation between adiponectin and CRP levels, whereas previous studies indicated a negative correlation between these two variables, suggesting lower adiponectin levels promoting the inflammatory process [35, 36].

Limitations of the study

We did not perform dobutamine stress echocardiography to patients with ventricular systolic dysfunction; this may have led to a miscalculation of the aortic gradients in those patients. CAD, diabetes mellitus, metabolic syndrome, HF, and renal failure may interfere with adiponectin levels. Patients who had these characteristics were not excluded.

CONCLUSIONS

We did not find a relationship between adiponectin levels and calcific aortic valve disease. In fact, we found that adiponectin levels were associated with some metabolic parameters.

Conflict of interest: *none declared*

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Związek między stężeniem adiponektyny w surowicy i zwapnieniowym zwężeniem zastawki aortalnej

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Streszczenie

Wstęp: Adiponektyna, cytokina produkowana przez tkankę tłuszczową, ma działanie przeciwmiażdżycowe i przeciwzapalne w stosunku do komórek śródbłonka i makrofagów. Zwapnienie zastawki aorty ma podobny patomechanizm jak miażdżyca.

Cel: Celem niniejszej pracy było zbadanie zależności między stężeniem adiponektyny a stopniem zwapnieniowego zwężenia aorty.

Metody: Do badania włączono 58 chorych ze zwapnieniowym zwężeniem aorty i 24 zdrowe osoby stanowiące grupę kontrolną. Chorych ze stenozą aortalną podzielono na 3 grupy w zależności od stopnia zwężenia: osoby z łagodnym (n = 11), umiarkowanym (25) i ciężkim (n = 22) zwężeniem zastawki aortalnej. U wszystkich uczestników badania zmierzono stężenie adiponektyny w surowicy oraz inne parametry biochemiczne.

Wyniki: Rozkład wieku, płci i czynników ryzyka sercowo-naczyniowego był podobny w grupie ze stenozą aortalną i w grupie kontrolnej. Porównanie mediany stężenia adiponektyny również nie wykazało istotnej różnicy między grupami [odpowiednio 2,19 μg/ml (1,43–3,18); 1,79 μg/ml (1,34–3,42); p = 0,7]. Chorych ze stenozą aortalną podzielono na trzy grupy — z lekkim, umiarkowanym lub ciężkim zwężeniem — w zależności od pola powierzchni ujścia zastawki. Nie stwierdzono różnic, porównując stężenia adiponektyny w tych trzech grupach [łagodne zwężenie: 2,10 μg/ml (1,47–3,31), umiarkowane: 2,13 μg/ml (1,44–2,91), ciężkie: 2,65 μg/ml (1,28–3,43); p = 0,67]. Wiek (r = 0,26; p = 0,045) i stężenie aminotransferazy asparaginianowej (r = 0,28; p = 0,04) korelowały dodatnio ze stężeniem adiponektyny, natomiast w przypadku liczby krwinek białych (r = –0,32; p = 0,015), glikemii na czczo (r = –0,29; p = 0,03) oraz stężenia hemoglobiny (r = –0,27; p = 0,04) i triglicerydów (r = –0,41; p = 0,002) wykazano korelację ujemną.

Wnioski: W niniejszym badaniu nie wykazano związku stężenia adiponektyny z zwapnieniowym zwężeniem aorty ani ze stopniem zwężenia.

Słowa kluczowe: stenoza aortalna, choroba wieńcowa, adiponektyna, zwapnieniowe zwężenie zastawki aortalnej

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