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CLINICAL STUDY

The relationship between epicardial adipose tissue thickness and coronary artery disease progress

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ABSTRACT

BACKGROUND: The relationship between epicardial adipose tissue and inflammatory events has been shown in many studies. Because it is an inflammatory process in coronary progression, it is aimed to examine the relationship between coronary artery disease progression and epicardial adipose tissue thickness.

MATERIALS AND METHODS: Our research was conducted with 50 patients (33 men, 17 women) who underwent planned or emergency coronary angiography, by evaluating the coronary artery disease progression from the coronary angiography images together with the echocardiographic epicardial adipose tissue thickness measurement. Patients were examined in two groups according to their tissue thickness, 17 patients with less than 0.55 cm were defined as group 1 and 33 patients with ≥ 0.55 were determined as group 2.

RESULTS: There was no significant difference between the groups in terms of gender, diabetes, age, hypertension. In addition, a significant relationship was found with epicardial adipose tissue thickness (> 0.5 cm), ejection fraction and smoking in the group with coronary progression. Patients without stenotic changes were found to be statistically significantly lower $p < 0.005$.

CONCLUSION: An independent relationship was found between epicardial adipose tissue and coronary artery progression. In the light of these findings, it can be concluded that epicardial adipose tissue residue is effective in the development of coronary artery stenosis and calcific-atherosclerotic changes in the coronary arteries. In the light of the information obtained, a positive correlation was determined between epicardial adipose tissue thickness and coronary artery disease *(Tab. 3, Fig. 2, Ref. 15)*. Text in PDF *www.elis.sk*

KEY WORDS: coronary artery disease, epicardial adipose tissue, progression.

Introduction

Atherosclerosis causing CAD is a chronic process. It may present with a stable or unstable clinical picture after a silent period. There are many risk factors that trigger exacerbation periods called Acute Coronary Syndrome. It is known that visceral adipose tissue (VYD) plays a key role in the evaluation of obesity as a risk factor, especially in patients with metabolic syndrome. Epicardial adipose tissue (EAT), which is accepted as the equivalent of visceral adipose tissue, locally effects the morphology and functions of the heart with the proatherogenic and proinflammatory cytokines it secretes. The relationship between EAT and coronary artery disease has been shown in many studies. (1–2) Various imaging methods are used to measure the amount of EAT (3–4). Transthoracic echocardiography (TTE) is the preferred method for EAT measurement due to its advantages such as easy accessibility, cheapness, no radiation exposure, and simultaneous acquisition of other cardiac parameters.

Epicardial Adipose Tissue (EAT), known as localized cardiac adipose tissue, which develops from brown adipose tissue, which has the same embryological origin as the mesenteric and omental visceral adipose tissue surrounding the internal organs, is accepted as the equivalent of VYD. EAT is defined as an endocrine and inflammatory organ that affects the morphology and functions of the heart locally with the proatherogenic and proinflammatory cytokines it secretes (5). There is no separating layer between the epicardial adipose tissue and the underlying coronary arteries and myocardium. As EAT increases, it surrounds the coronary arteries and acts on the vessel wall with the proatherogenic and proinflammatory cytokines it secretes, directly or through the vasa vasorum. Thus, it plays an active role in the process of coronary artery disease and metabolic syndrome (6, 7). The mean thickness is 5.3 ± 1.6 mm when measured perpendiculary to the RV free wall. It constitutes approximately 1 % of the total body fat mass (8). Vertical thickness of epicardial fat; compatible with total epicardial fat volume. It is easy to measure, shows indirect visceral adiposity and is an important method in the assessment of cardiovascular risk (8).

Epicardial adipose tissue thickness is measured on the right ventricular free wall from both parasternal long and short axis views. Imaging limitations are used to ensure that epicardial fat thickness is not measured obliquely (9).

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The aim of this study is to reveal the relationship between epicardial adipose tissue thickness detected in routine echocardiography during hospitalization and coronary artery disease progression in patients admitted to the hospital with previously known coronary artery disease. In a previous study, it was shown that the thickness of the epicardial adipose tissue is proportional to the coronary artery disease and its severity. However, there is no study in the literature examining epicardial adipose tissue thickness and coronary artery progression. Our research will be the first study on this subject.

Materials and methods

Study population

The study was conducted with 50 patients (33 males, 17 females) admitted to the state hospital with the diagnosis of ACS without a previous history of CAD. The patients were evaluated in two groups according to their echocardiographic epicardial adipose tissue thicknesses, 17 patients less than 0.55 cm in group 1 and 33 patients greater than or equal to ≥ 0.55 in group 2 (p = 0 .005). Cardiovascular risk factors such as age, gender, class, mean heart rate and arterial blood pressure, HT, DM, smoking, etc., were recorded after the admission of the patients.

The patients will be evaluated echocardiographically with the mindray DC-8 EXP Echocardiography device and 2.5 MHz echocardiography probe in the cardiology outpatient clinic of our hospital. The EAT thickness was measured using two-dimensional echocardiographic method in both the systole and diastole phases of the heart in the parasternal long axis image, and from the place where the heart is widest, from the hyperechoic area consistent with the remaining EAT density between the right ventricular free wall and the pericardium, where the perpendicular line drawn with the aortic annulus as a reference passes.

The progression of coronary artery disease was investigated by looking at the latest angiography images and previous angiography images of the patients. Coronary angiography was performed by an experienced interventional cardiologist (> 75 cases per year) femoral percutaneous route using the Judkins technique on a SIEMENS angiography device. Coronary arteries were visualized in the right and left oblique planes with cranial and caudal angles at 12 frames per second (12 fps). Iopromide (Ultravist-370) was used as the opaque material. It has been digitally recorded on CDs in the DICOMR standard. Coronary flow rates were determined by the TIMI frame count method.

Patients with a history of rheumatic valve disease or cardiomyopathy (hypertrophic, dilated, restrictive) known as anamnesis or echocardiographic finding, with insufficient echogenicity echocardiographically, with chronic renal failure, without previous coronary angiography or with unknown previous coronary angiography images were excluded from the study.

Coronary artery progression scores (LM, LAD, Cx, RCA) were calculated to evaluate the extent and severity of CAD. Epicardial adipose tissue thickness and mediastinal adipose tissue thickness were measured by echocardiography.

Tab. 1. Distribution of the descriptive statistics.

		n (%)
Gender	Male	36 (72.0)
	Female	14 (28.0)
Age	Mean±SD	61.64±13.67
	Median (Min–Max)	61 (35–93)
Coronary artery disease Progression	No	22 (44.0)
	Yes	28 (56.0)
Diabetes Mellitus	No	42 (84.0)
	Yes	8 (16.0)
Hypertension	No	15 (30.0)
	Yes	35 (70.0)
Smoking	No	33 (66.0)
	Yes	17 (34.0)
Location of intervention	Left femoral artery	1 (2.0)
	Right femoral artery	47 (94.0)
	Left radial artery	2 (4.0)

Tab. 2. Distribution of Progression Site and Severity.

		n (%)
Progression Site	CX	5 (10.0)
	CX and LAD	3 (6.0)
	D1-Saphena	1 (2.0)
	Diagonal	2 (4.0)
	LAD	7 (14.0)
	LAD-Saphena	1 (2.0)
	RCA	7 (14.0)
	RCA and CX	1 (2.0)
	RCA and LAD	1 (2.0)

Tab. 3. Comparison of descriptive characteristics according to the progression of coronary artery disease.

	Progression of coronary artery disease		p
	No (n=22)	Yes (n=28)	
Gender (male)	14 (63.6)	22 (78.6)	0.343
Age	61.14±11.35	62.04±15.45	0.820
Epicardial fat tissue thickness	0.55±0.08	0.62±0.09	e0.011*
Ejection fraction	52.95±7.35	49.29±6.77	0.014*
Diabetes Mellitus	3 (13.6)	5 (17.9)	1.000
Hypertension	14 (63.6)	21 (75.0)	0.384
Smoking	2 (9.1)	15 (53.6)	0.001**
BUN	26±1.6	28±1	0.472
Creatinin	0.96±0.08	0.88±0.1	0.712
Hemogram	12.3±2.3	13.4±1.8	0.329
WBC	7.5±1.8	8.8±1.1	0.193
HDL	41±9	46±11	0.060
LDL	124±32	130±24	0.230
Total cholesterol	166±62	187±49	0.200
Triglycerides	135±16	159±12	0.262

Statistical analysis

The data of the individuals participating in the study were recorded in the previously prepared study forms. Then, in order to perform the analyzes to be used in the study, the data were collected with SPSS v. 22.0 software for Windows (SPSS Inc. Chicago, Illinois, USA) saved in the database. Descriptive statistical methods (mean, standard deviation, median, frequency, percentage, minimum, maximum) were used to evaluate the study data. The conformity of the data to the normal distribution was evaluated

with the Kolmogorov-Smirnov and Shapiro-Wilk test. Kruskal-Wallis analysis of variance and Mann-Whitney U test were used for data that did not fit the nominal distribution. Data were presented as numbers, percentages, and arithmetic mean±standard deviation. The Pearson chi-square test, Fisher's exact test and the Fisher-Freeman-Halton exact test were used for comparison of the qualitative data. Significance level was accepted as $p < 005$.

Results

The study was conducted in a total of 50 participants in the Research and Training Hospital between June and December 2021. Among the participants, 14 (28 %) were female and 35 (72 %) were male. The ages of the cases ranged between 35 and 93, and the mean age was calculated as 61.64 ± 13.67.

Among the participants, 56 % (n = 28) had coronary artery disease progression.

DM and HT were observed in 16 % (n = 8) and 70 % (n = 35) of the participants, respectively.

Among the cases, 34 % (n = 17) were smokers and contrast nephropathy had developed in 2 % (n = 1).

Among the participants, 92 % (n = 46) were compliant to medication.

Location of intervention was left femoral artery in 2 % (n = 1), right femoral artery in 94 % (n = 47) and left radial artery in 4 % (n = 2).

Among the participants with coronary artery progression, the progression site was CX in 10 % (n = 5), CX and LAD in 6 % (n = 3), D1-Saphena in 2 % (n = 1), Diagonal in 4 % (n = 2), LAD in 14 % (n = 7) and LAD- Saphena in 2 % (n = 1). Among the cases, 14 % (n = 7) were RCA, 2 % (n = 1) were RCA and CX, and 2 % (n = 1) were RCA and LAD.

The progression rates of the cases varied between 70 and 100 %, and the mean rate was 93.58 ± 7.76 %.

No statistically significant difference was observed in the ages or genders of the participants according to coronary artery disease progression ($p > 0.05$).

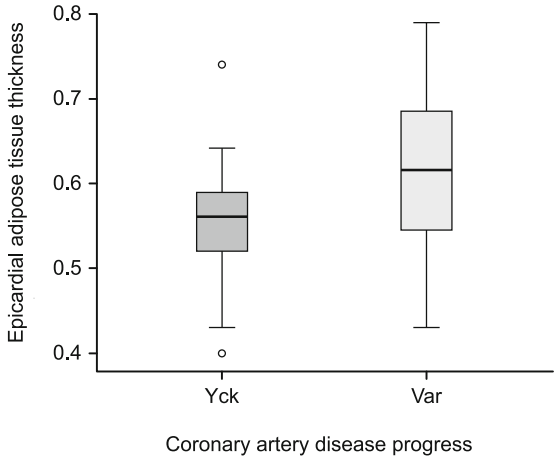

Fig. 1. Distribution of epicardial fat tissue thickness according to coronary artery disease progression.

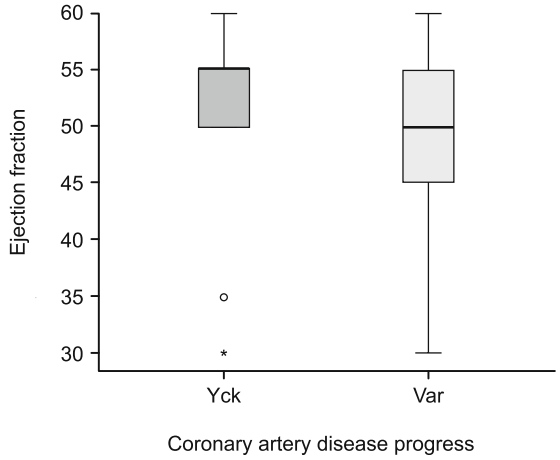

Fig. 2. Distribution of ejection fractions according to coronary artery disease progression.

It was observed that the epicardial fat tissue thickness of the cases with coronary artery disease progression was statistically significantly higher compared to those with no progression ($p = 0.011$; $p < 0.05$).

The Ejection fraction of the cases with coronary artery disease progression was observed to be significantly lower compared to those with no progression ($p = 0.014$; $p < 0.05$).

The rate of smoking among cases with coronary artery disease progression was observed to be significantly higher compared to those without progression ($p = 0.001$; $p < 0.01$).

No significant difference was observed in DM, HT, contrast nephropathy development or compliance to medication according to coronary artery disease progression ($p > 0.05$).

Discussion

In the light of the information obtained, a positive correlation was determined between epicardial adipose tissue thickness and coronary artery disease and metabolic syndrome. Epicardial adipose tissue is a metabolically active organ that produces many active molecules and significantly affects cardiac functions. Adipose tissue, which has been seen as an energy store for a long time, has been accepted as an important endocrine organ of the body in recent years. Adipose tissue has biological activities related to energy metabolism, neuroendocrine function and immune functions. Both deficiency and excess of adipose tissue have important metabolic and endocrinological consequences. Currently, the relationship between the biochemical local and systemic effects of epicardial adipose tissue and cardiovascular and metabolic complications has been investigated. However, it has been tried to prove whether epicardial adipose tissue can be a predictor of certain cardiovascular and metabolic complications rather than its contribution to treatment.

Studies have reported that adipokine changes, which cause pro-inflammatory properties in epicardial adipose tissue, play a critical role in the development of coronary atherosclerosis and coronary artery disease (10, 11, 12). Studies on epicardial adipose

tissue suggest that epicardial adipose tissue is anatomically and clinically related to heart morphology and function. It has been suggested that epicardial adipose tissue can provide valuable information in the evaluation of the risk, extent and activity of coronary artery disease, and therefore, echocardiography and tomography methods have been used to show the epicardial adipose tissue.

When we look at the literature, there are controversial opinions in various studies that BMI may also have an effect on the relationship between CAD and epicardial adipose tissue. Yong et al and Gorter et al. BMI was added to the parameters in the studies performed by BMI, and as a result, when high-weight patients were excluded, it was reported that the amount of pericardial adipose tissue was significantly higher in those with atherosclerotic changes than in those without (13). However, in another study comparing abdominal adipose tissue area and epicardial adipose tissue volume, it was thought that the increase in epicardial adipose tissue was associated with the development of coronary calcium plaque independently of abdominal visceral adipose tissue (14). These findings suggest that the effect of BMI on the increase in epicardial adipose tissue and the development of CAD should be supported by further studies.

JeongJW. et al. in their study of 202 patients found a significant correlation between EAT thickness measured by echocardiography and the severity of coronary artery disease, and suggested that it could be used in risk stratification in patients with coronary artery disease (15).

Considering the results of this study, it is an expected result that the EAT thickness is higher in elderly and obese individuals. In our study, there was no significant difference between the groups in terms of cholesterol value. However, smoking was more common in the group with higher EAT thickness. This finding was interpreted as the cardiovascular risk of patients in the group with increased EAT thickness is higher.

In many studies, it has been shown that inflammatory markesr mRNA expression, IL-1, IL-6, MCP-1, TNF levels increase in the epicardial adipose tissue of patients who underwent CABG (6). They play an important role in the formation and development of atherosclerosis with local and systemic effects. The increased presence of inflammatory cells in epicardial adipose tissue may reflect a similar response to inflammatory infiltrates found in the adventitia and perivascular regions adjacent to advanced atherosclerotic lesions.

Conclusion

An independent relationship was found between epicardial adipose tissue and coronary artery progression. In the light of these findings, it can be concluded that epicardial adipose tissue residue is effective in the development of coronary artery stenosis and calcific-atherosclerotic changes in the coronary arteries. In addition, it is thought that a single cross-sectional area measurement from the level of the left main coronary artery compared to the right femoral can be a good alternative to total epicardial adipose tissue volume calculations, since it can be measured more easily in calculating the amount of epicardial adipose tissue.

Limitations

The primary limitation of the study is that it was limited to patients with a history of CAD who came to Karaman State Hospital in 2020.

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