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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The effect of epicardial adipose tissue thickness on left ventricular diastolic functions in patients with normal coronary arteries

Mustafa Topuz[1], Ali Dogan[2]

[1]Adana Numune Training and Research Hospital, Adana, Cukurova, Turkey
[2]Erciyes University, Medicine Faculty, Kayseri, Talas, Turkey

Abstract

Background and aim: The aim of this study is to evaluate the effect of epicardial adipose tissue (EAT) thickness on left ventricular diastolic functions in patients with normal coronary arteries (NCA) proven by angiography.

Methods: We selected study patients who were referred to coronary angiography due to typical chest pain or atypical chest pain with a positive pre-test result for coronary artery disease (CAD). After coronary angiography, 85 patients with significant coronary lesion (≥ 50% stenosis) served as the CAD group, 82 patients with non-significant coronary lesion (< 50% stenosis) and/or a coronary plaque served as the non-significant CAD group, and 83 patients with NCA served as the NCA group. All patients underwent transthoracic echocardiographic examination to measure EAT thickness and left ventricular diastolic properties.

Results: Gensini score, total cholesterol, and C-reactive protein were significantly higher in the CAD group compared to the non-significant CAD group and the NCA group. The average of EAT thickness was 7.3 ± 2.4 mm in all groups. It was 5.8 ± 2.3 mm in the NCA group, 6.4 ± 2.4 mm in the non-significant CAD group, and 7.8 ± 2.2 mm in the CAD group ($p < 0.001$). In correlation analyses, average EAT thickness was significantly correlated with E/e' ratio in the NCA group. In multivariate analysis, average of EAT thickness was significantly associated with left ventricular diastolic dysfunction in subjects with NCA (OR 1.019, 95% CI 1.012–1.027, $p < 0.001$).

Conclusions: Based on our findings, EAT thickness may have an effect on left ventricular diastolic functions. This independent relationship showed us the clinical importance of measuring of EAT thickness.

Key words: epicardial adipose tissue, diastolic function, coronary artery disease

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INTRODUCTION

Diastolic dysfunction is defined as an increase in resistance of one or both ventricle/s [1]. Approximately 40% of patients presenting with heart failure (HF) have a normal systolic function and clinical symptoms linked to left ventricular (LV) diastolic dysfunction (LVDD) [2]. LVDD can increase the risk of HF and cardiovascular (CV) mortality, so it is clinically beneficial that diastolic functions can be measured for every patient when undergoing routine echocardiographic examination, even if the patient is asymptomatic or at preclinical stage [3].

Body fat tissue produces a wide range of molecules and acts like a highly complex endocrine organ that has a wide variety of local and systemic effects. A visceral adiposity of epicardial regions, known as epicardial adipose tissue (EAT), has an effect on the CV system as well as abdominal external fat tissue [4]. Previous studies have shown an association between EAT and traditional CV risk factors, structural heart disease, coronary artery plaque burden, major adverse CV events, non-dipper status, and increased left atrial size [5–8].

Although the association of EAT thickness with LVDD has been studied, there is no specific study in current literature regarding the association of EAT thickness with LV diastolic function in patients with normal coronary arteries (NCA) proven by coronary angiography. The aim of this study is to investigate the possible relation of EAT thickness with LVDD.

Address for correspondence:
Dr. Mustafa Topuz, Adana Numune Training and Research Hospital, Adana, Cukurova, Postal Code: 01150, Turkey, tel: +905066743544, fax: +903223550150, e-mail: topuzm46@gmail.com; mtpuz@hotmail.com

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METHODS

Patient population

We selected study patients who were referred to coronary angiography due to typical chest pain or atypical chest pain with a positive pre-test result for coronary artery disease (CAD). Eighty-five patients with significant coronary lesion (≥ 50% stenosis) served as the CAD group, 82 patients with non-significant coronary lesion and/or presence of a plaque (< 50% stenosis) served as the non-critical CAD group, and 83 patients with NCA served as the NCA group. All patients underwent transthoracic echocardiographic (TTE) examination to measure the average of EAT thickness and LV diastolic properties. The standard treatments were as prescribed by international guidelines. Informed consent was obtained from each patient about the research. The study protocol was approved by the local Ethics Committee.

Patients with LV ejection fraction (LVEF) ≤ 50%, diabetes mellitus, stroke, acute or chronic renal failure, history of acute infection within the previous seven days, acute or chronic hepatic failure, presence of any chronic inflammatory and autoimmune disease, and any known malignancy were excluded from the study. Also, patients with poor echogenicity, presence of pericardial effusion, calcified pericardium, pericardial thickness above 5 mm, severe valvular stenosis, or insufficiency were excluded from the study.

Coronary angiography

In all patients coronary angiography was performed using the standard poses Philips Medical Systems Integris H 5000 or Toshiba® Infinix CC-i monoplan cardiac angiography using 6 F diagnostic catheters with standard Judkins technique via the left or right femoral approach. All observations were performed by two interventional cardiologists who were blinded to the study patients.

The degree of coronary artery stenosis was estimated using Gensini scores, which were computed by assigning a severity score to each coronary segment according to the degree of luminal narrowing and geographic importance [9]. For the Gensini score, the most severe stenosis in each of the eight coronary segments, namely left anterior descending artery, main diagonal branch, first septal perforator, left circumflex artery, obtuse marginal and posterolateral vessels, right coronary artery, and main descending branch, was graded from 1 to 4 (1% to 49% lumen diameter reduction, 1 point; 50% to 74% stenosis, 2 points; 75% to 99% stenosis, 3 points; 100% occlusion, 4 points). The total score could range between 0 and 32.

Echocardiography

All TTE examinations were performed by two experienced cardiologists, who were blinded to the study patients demographic and laboratory parameters, using a 1.5–3.4 MHz probe with Vivid-7S echocardiography device. Study patients were examined in left lateral decubitus by M-mode, two-dimensional echocardiography, and tissue Doppler imaging.

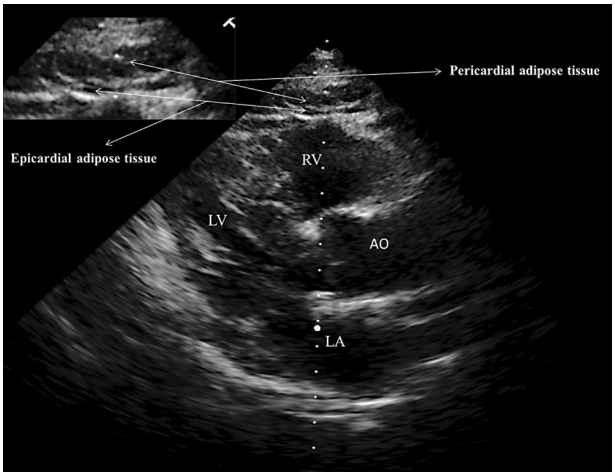

Figure 1. Measurement of epicardial fat thickness by echocardiography. Epicardial adipose tissue, identified as an echo-free space between the outer myocardium and visceral pericardium from the parasternal long-axis view on two-dimensional echocardiography, was measured perpendicularly in front of the right ventricular free wall at end-diastole; AO — aorta; LV — left ventricle; LA — left atrium; RV — right ventricle

Measurements of EAT thickness were measured perpendicularly in front of the right ventricular (RV) free wall at end-diastole because this area is known to have the thickest EAT layer. All measurements were acquired using the long parasternal axis, and at end-diastole over three cardiac cycles in the left lateral decubitus position [10, 11]. To standardise the measuring axis, we used the aortic annulus as an anatomical reference. EAT was identified as the echocardiographically free space between the outer wall of the myocardium and the visceral layer of the pericardium. A still image of EAT thickness was zoomed for better visualisation and accurate measurement (Fig. 1). The measurement was performed at a point on the free wall of the RV along the midline of the ultrasound beam, perpendicular to the aortic annulus. The average value comprising three cardiac cycles of each echocardiographic view was used for the statistical analysis. Inter-observer correlation coefficients were 0.91, whereas intra-observer correlation coefficients were 0.94 (the intraobserver mean absolute difference in measurements of EAT was 0.2 ± 0.3 mm and coefficient of variation was 1.5%).

Left ventricular ejection fraction was measured by bi-plane Simpson's method. Maximum and minimum left atrial (LA) volumes were measured by the method of discs from the apical four-chamber view at end-systole and end-diastole. End-systolic measurements were obtained from the frame preceding the mitral valve opening. As diastolic parameters, early (E) and late mitral inflow (A) velocities, lateral and medial

mitral annular velocities (e', Am), isovolumic relaxation time (IVRT), and deceleration time were measured according to guidelines by the American Society of Echocardiography [3].

Statistical analysis

Descriptive analyses were presented as mean ± standard deviation, and categorical variables were expressed as percentages. The variables were investigated using visual (histograms and probability plots) and analytical methods (Kolmogorov-Smirnov test) to determine whether or not they were normally distributed. Mean values of the groups were compared by ANOVA test. Pearsons and Sperman correlations were used to evaluate the correlations of the parameters with LV diastolic function. Using regression analysis, other possible confounding covariables were adjusted. Statistical significance was defined as $p < 0.05$. At the end of the study, for statistical analysis the SPSS statistical software program (version 15.0, SPSS, USA) was used.

RESULTS

The baseline characteristics of the groups are summarised in Table 1. Gensini score, total cholesterol, and C-reactive protein (CRP) were significantly higher in the CAD group compared to the non-significant CAD group and the NCA group. The average EAT thickness was 7.3 ± 2.4 mm in all study patients and was found to be higher in the CAD group than in the non-significant CAD group and NCA groups (respectively; 7.8 ± 2.2 mm, 6.4 ± 2.4 mm, and 5.8 ± 2.3 mm, $p < 0.001$).

In correlation analysis the average EAT thickness was significantly correlated with Gensini score, LA dimension, and echocardiographic parameters associated with LVDD in all patients (Table 2). Also EAT had significant correlations between CRP ($r = 0.287$, $p = 0.021$), Gensini score ($r = 301$, $p = 0.017$), and body mass index (BMI; $r = 0.263$, $p = 0.041$), as demographic and laboratory parameters; LA diameter ($r = 0.297$, $p = 0.019$), E/A ratio ($r = 0.324$, $p = 0.01$), E/e' ratio ($r = 0.387$, $p < 0.001$), and IVRT ($r = 0.271$, $p = 0.027$) as echocardiographic parameters in the CAD groups (in both the CAD group and the non-significant CAD group).

The average EAT thickness was higher in patients with LVDD than in those without LVDD (5.1 ± 2.0 mm vs. 3.3 ± 1.8 mm, $p = 0.001$). Correlation analysis was performed to evaluate the association of various parameters in the NCA group with E/e' ratio. The average EAT thickness and patient age were significantly correlated with E/e' ratio (Table 3).

We defined LVDD in 35 patients in the NCA group according to transmitral Doppler findings (conventional early transmitral velocity [E] < conventional late transmitral velocity [A]), LA volume ≥ 30 mL/m^2 and tissue Doppler findings (conventional early transmitral velocity [E] / tissue Doppler early diastolic mitral annular velocity [e'] ≥ 13). Univariate and multivariate logistic regression analysis were performed including age, presence of hypertension, BMI, LA diameter, E/A ratio, E/e' ratio, IVRT, and EAT thickness, as shown in Table 4. After adjusting for the covariables, EAT was still significantly associated with LVDD in subjects with NCA (OR 1.019, 95% CI 1.012–1.027, $p < 0.001$).

DISCUSSION

We revealed that echocardiographically measured EAT thickness is related significantly with LVDD in NCA patients. To the best of our knowledge, this is the first study documenting this association, which was conducted by coronary angiography.

We aimed to define the possible role of EAT thickness as a risk factor for developing LVDD in patients with normal coronary arteries. As shown in our study, increased EAT may be independently associated with LVDD in subjects with NCA. LVDD is clinically important and related to poor outcome. Patients with LVDD with normal systolic function and no symptoms of HF are defined as preclinical diastolic dysfunction. It is clinically important that preclinical diastolic dysfunction has been shown to be associated with development of HF and is predictive of all-cause mortality [12]. In the epidemiology section, the development of preclinical diastolic dysfunction to advanced HF was found to be associated with established CV risk factors such as increasing age, CAD, hyperlipidaemia, metabolic syndrome, peripheral vascular disease, and diabetes [13]. Early detection of these risk factors is essential to decrease patient morbidity and mortality because LVDD increases the risk of HF and CV mortality even if patients were in asymptomatic course or preclinical stage. Also, advanced LVDD is associated with reduced quality of life and structural abnormalities that reflect increased CV risk [14]. It is also important to determine these factors because there is no definitive treatment for LVDD. The determination of these risk factors, including EAT thickness, may prevent development of preclinical diastolic dysfunction. Our findings implicate the clinical importance of measuring EAT thickness. Patients with thicker EAT seem to have a higher risk of LVDD and a worse prognosis, even if they have normal coronary arteries. The best treatment of a thicker EAT is prevention of the metabolic abnormalities that can lead to increased EAT thickness because there is currently no medical treatment option for lowering EAT thickness. In fact, as a visceral adiposity, EAT thickness may be closely associated with the development of metabolic abnormalities. For example, weight reduction in obesity is associated with a decrease in EAT thickness.

Epicardial adipose tissue is an organ that gives paracrine control and an active role in development of an adverse CV situation by producing many bioactive molecules. The embryological source of EAT is similar to abdominal visceral adiposity. Local and systemic effects of EAT on biochemical, CV, and metabolic complications is the subject of current research. There is strong evidence that EAT, especially presented around the coronary arteries, can lead to the development of atherosclerosis by affecting the coronary vascular wall, as

Table 1. Baseline characteristics of the study patients (n = 250)

Parameters	NCA group (n = 82)	Non-significant CAD group (n = 83)	CAD group (n = 85)	p
Age [years]	59.4 ± 11.3	61 ± 11.7	61.7 ± 12.3	0.421
Gender (male/female)	63/20	66/16	61/24	0.742
BMI [kg/m^2]	27.9 ± 4.8	28.3 ± 5.1	29.2 ± 5.0	0.312
Hypertension	25	37	41	0.034
Smoking [%]	64.7	69.5	71.1	0.108
Gensini score	0	36.4 ± 8.8	74 ± 24.9	< 0.001
Glucose [mg/dL]	93 ± 12	99 ± 17	99 ± 14	0.096
LDL [mg/dL]	128 ± 35	138 ± 33	145 ± 36	0.056
HDL [mg/dL]	45 ± 14	47 ± 11	45 ± 9	0.614
TG [mg/dL]	165 ± 89	171 ± 123	186 ± 109	0.611
Total cholesterol [mg/dL]	205 ± 44	219 ± 35	227 ± 41	0.029
Creatinine [mg/dL]	0.86 ± 0.23	0.85 ± 0.13	0.81 ± 0.10	0.146
CRP [mg/dL]	0.38 ± 0.41	0.46 ± 0.42	0.72 ± 0.57	0.002
EAT thickness [mm]	5.8 ± 2.3	6.4 ± 2.4	7.8 ± 2.2	< 0.001
SBP [mm Hg]	146.2 ± 14.7	146.3 ± 14.4	148.1 ± 14.2	0.452
DBP [mm Hg]	86.1 ± 8.3	84.5 ± 7.4	85.1 ± 8.1	0.751
Echocardiography				
LVEF [%]	59.6 ± 6.7	58.8 ± 6.4	57.1 ± 6.1	0.645
LVEDD [mm]	45.4 ± 9.1	46.6 ± 4.1	47.8 ± 4.3	0.275
LVESD [mm]	27.1 ± 4.3	29.2 ± 6.2	30.4 ± 5.7	0.321
IVSD [mm]	11.7 ± 1.1	12.2 ± 1.5	13.7 ± 2.4	< 0.001
PWD [mm]	10.4 ± 1.3	11.1 ± 1.2	12.5 ± 1.5	< 0.001
Doppler echocardiography				
Mitral E velocity [cm/s]	72.4 ± 15.1	70.1 ± 18.4	63.2 ± 16.2	0.064
Mitral A velocity [cm/s]	73.2 ± 16.4	80.7 ± 18.6	87.2 ± 16.3	0.002
E/A ratio	1.04 ± 0.35	0.90 ± 0.34	0.73 ± 0.19	< 0.001
DT [ms]	204 ± 32	232 ± 58	234 ± 38	0.061
IVRT [ms]	81 ± 12	93 ± 17	101 ± 18	0.001
LA diameter [mm]	35.7 ± 3.1	37.3 ± 4.5	40.1 ± 3.2	< 0.001
LA volume [mm^3]:				
Maximum	53 ± 12	58 ± 16	67 ± 17	0.001
Minimum	21 ± 6	26 ± 10	31 ± 11	0.011
Tissue Doppler parameters				
Sm [cm/s]: (L/S)	9.3 ± 1.9/7.9 ± 1.1	10.0 ± 2.6/7.9 ± 1.5	8.7 ± 1.7/7.1 ± 1.4	015/0.020
e' [cm/s]: (L/S)	11.1 ± 2.9 /7.9 ± 1.5	9.1 ± 2.9 /7.9 ± 1.1	7.8 ± 2.6/ 7.1 ± 1.4	0.025/0.020
Am [cm/s]: (L/S)	10.1 ± 2.6/9.8 ± 2.3	11.1 ± 3.0/10.0 ± 2.2	10.5 ± 2.8/9.9 ± 2.1	0.375/0.941
e'/Am ratio: (L/S)	1.1 ± 0.5/0.9 ± 0.4	1.0 ± 0.4/0.7 ± 0.2	0.9 ± 0.4/0.6 ± 0.2	0.032/0.001
E/e' ratio: (L/S)	6.8 ± 1.7/9.2 ± 2.7	7.6 ± 2.6/11.0 ± 2.7	8.0 ± 2.5/10.7 ± 2.9	0.162/0.044

A — atrial (late) peak mitral velocity obtained from pulsed Doppler; Am — late diastolic mitral annular velocity obtained from tissue Doppler; BMI — body mass index; CAD — coronary artery disease; CRP — C-reactive protein; DBP — diastolic blood pressure; DT — deceleration time; E — early peak mitral velocity obtained from pulsed Doppler; e' — early diastolic mitral annular velocity obtained from tissue Doppler; EAT — epicardial adipose tissue; HDL — high density lipoprotein; IVSD — interventricular septal diameter; IVRT — isovolumetric relaxation time; LA — left atrium; L/S — lateral/septal; LDL — low density lipoprotein; LVEDD — left ventricular end diastolic diameter; LVESD — left ventricular end systolic diameter; LVEF — left ventricular ejection fraction; NCA — normal coronary artery; TG — triglyceride; SBP — systolic blood pressure; PWD — posterior wall diameter; p < 0.05 was accepted as significant

Table 2. The correlation analysis of epicardial adipose tissue thickness with study parameters in all study patients

	Correlation coefficient	p
Age	0.249	0.044
Waist circumference	0.132	0.174
Body mass index	0.297	0.003
C-reactive protein	0.155	0.103
LA diameter	0.371	< 0.001
E/A ratio	–0.274	0.041
Lateral e' velocity	– 0.306	0.024
Lateral Am velocity	–0105	0.221
E/e' ratio	0.418	< 0.001
IVRT	0.216	0.021
Deceleration time	0.142	0.125
Gensini score	0.389	< 0.001

A — atrial (late) peak mitral velocity obtained from pulsed Doppler; Am — late diastolic mitral annular velocity obtained from tissue Doppler; E — early peak mitral velocity obtained from pulsed Doppler; e' — early diastolic mitral annular velocity obtained from tissue Doppler; IVRT — isovolumic relaxation time; $p < 0.05$ was accepted as significant

Table 3. The correlation analysis of study parameters with E/e' ratio in patients with normal coronary arteries

	Correlation coefficient	p
Age	0.324	0.002
Waist circumference	0.153	0.280
Body mass index	0.123	0.352
LA diameter	0.259	0.021
E/A ratio	–0.431	< 0.001
EAT thickness	0.306	0.013
IVRT	0.208	0.079
Deceleration time	0.147	0.271

A — atrial (late) peak mitral velocity obtained from pulsed Doppler; Am — late diastolic mitral annular velocity obtained from tissue Doppler; E — early peak mitral velocity obtained from pulsed Doppler; e' — early diastolic mitral annular velocity obtained from tissue Doppler; EAT — epicardial adipose tissue; LA — left atrium; IVRT — isovolumic relaxation time; $p < 0.05$ was accepted as significant

well as destabilisation of existing atherosclerotic lesions [15]. In fact, Mazurek et al. [7] showed that 18-fluorodeoxyglucose (FDG) uptake of periventricular adipose tissue of the coronary territory was related to plaque composition and corresponded to necrotic core rate and plaque burden. In addition to atherosclerosis, EAT has a close anatomical and functional relationship with myocardium is also associated with both structural and ultra-structural changes in myocardium. For example, Kırış et al. [8] suggested that EAT may be associated with ventricular premature beats, which is associated with structural heart disease. In fact, the authors showed that patients with frequent ventricular premature beats had increased EAT thickness compared to control subjects. They found that patients with thicker EAT had increased LV mass index and suggested that EAT could lead to ventricular arrhythmia via structural changes such as fibrotic process in myocardium [8].

According to our results, EAT thickness may have a direct effect on LV diastology after adjusting another factors and excluding coronary ischaemia. In summary, association of EAT and LVDD, as a structural heart disease, is well known too. There are a lot of studies evaluating the association of EAT and LVDD. Park et al. [16] showed that EAT thickness is associated with LVDD in patients with metabolic syndrome, who are free of known CAD, although it is well known that the presence of metabolic syndrome has also been linked with impaired LVDD [17]. Cavalcante reported that epicardial fat volume has an independent correlation with impaired diastolic function after accounting for associated comorbidities in patients without clinical signs and symptoms of heart and metabolic disorders [6]. Cetin et al. [18] found that echocardiographically measured EAT thickness was significantly related with LVDD and increased LA dimensions. In another study, Börekci et al. [19] investigated the relationship between EAT and tissue Doppler myocardial performance index (TD-MPI), which incorporates both systolic and diastolic LV function, in newly diagnosed hypertension patients. Lin et al. [20] also showed that EAT is significantly independently associated with LVDD in patients undergoing peritoneal dialysis. Although the main limitation of all of the above studies is that the authors did not objectively assess the presence of CAD in their study populations, we well know that the association of CAD with LVDD was studied before, and it was shown that CAD is an independent risk factor for LVDD [21]. Also, LVDD is frequently seen in patients with documented CAD, with subclinical atherosclerosis, and even abnormal coronary artery calcium score as recently demonstrated [22, 23]. Our study results were in line with previous these studies showing that EAT thickness is significantly associated with LV diastolic function parameters. In contrast to all of the abovementioned studies, which demonstrated the association of EAT with LVDD, our study was conducted by coronary angiography to evaluate CAD, which is the most important factor for LVDD, as we explained above, and further classified for the angiographic severity with the Gensini scoring system. To the best of our knowledge, no studies to date have investigated the effect of EAT on LV diastolic properties in a CAD-free population who were diagnosed with coronary angiography. In this respect, our results may provide more objective data to the current literature.

Table 4. Univariate and multivariate logistic regression analysis showing parameters associated with left ventricular diastolic dysfunction in the normal coronary arteries group

	Univariate		Multivariate	
	OR (95% CI)	p	OR (95% CI)	p
Age [year]	1.401 (1.149–1.642)	0.18	0.993 (0.979–1.007)	0.302
Hypertension	3.561 (1.494–8.487)	0.004	2.694 (1.070–9.789)	0.106
LA diameter [mm]	1.143 (1.074–1.178)	0.041	1.037 (1.007–1.068)	0.015
E/A ratio	1.075 (1.025–1.099)	0.027	1.175 (1.006–1.371)	0.041
E/e' ratio	1.090 (1.037–1.146)	0.011	1.090 (1.037–1.146)	< 0.001
IVRT [ms]	1.021 (0.998–1.037)	0.031	1.017 (0.998–1.037)	0.085
EAT thickness [mm]	1.013 (1.007–1.039)	0.017	1.019 (1.012–1.027)	< 0.001

A — atrial (late) peak mitral velocity obtained from pulsed Doppler; CI — confidence interval; E — early peak mitral velocity obtained from pulsed Doppler; e' — early diastolic mitral annular velocity obtained from tissue Doppler; EAT — epicardial adipose tissue; IVRT — isovolumic relaxation time; LA — left atrium; OR — odds ratio; $p < 0.05$ was accepted as significant

Our findings may be explained by some possible mechanisms. One of them is impaired coronary flow reserve. EAT is associated with poor coronary flow reserve even in patients with angiographically normal coronary arteries [24]. Indeed, a strong relationship between impaired endothelial coronary flow reserve and IVRT time and LVDD was shown in two recent studies in uncomplicated hypertension and type 1 diabetic patients free of CAD [25, 26]. This association is not surprising because coronary flow occurs predominantly during diastole. EAT's anatomic proximity to the heart without fascial protection and possible local interaction via paracrine or vasocrine effect is also suggested as another possible mechanism by which EAT may cause LVDD. Pathophysiological research about LVDD implicates an active fibrotic process [27]. As a source of inflammatory mediators such as tumour necrosis factor-alpha, interleukin-1b, and interleukin-6, EAT can lead pathological changes within myocardium including myocardial fibrosis and the perivascular area, which may cause diastolic dysfunction via impaired myocardial stiffness [15, 28].

Limitations of the study

There are several limitations in our study. First, the measurement of epicardial fat tissue by echocardiography can vary depending on the experience of the cardiologist. On the other hand, TTE has a good correlation with abdominal adiposity measured by computed tomography and magnetic resonance imaging, and also it is radiation-free and free from contrast agent-related side effects [10]. Second, the assessment of additional biomarkers such the inflammatory mediators, which were associated with EAT, may help strengthen our findings and aid in the interpretation of our results.

For the severity of CAD, we used the Gensini score, a validated scoring system to demonstrate angiographic severity of CAD, instead of SYNTAX score. Although SYNTAX score could be used for this purpose, it has been used frequently to risk-stratify patients with complex CAD undergoing percutaneous coronary intervention. Also, it was previously implied that The SYNTAX and Gensini score in combination with clinical variables could be used to predict the CV prognosis during a long-term follow-up of up to eight years in CAD patients and there is a significant positive correlation between them [29]. Moreover, physicians can include all coronary lesions, which amounts to even less than 50% in the Gensini scoring system, although it is an older scoring system than SYNTAX. This may increase the sensitivity of the statistical results and provide more objective classification of the study patients with CAD. Also, we classified 50% coronary lesions as significant due to the study design, also to homogenise the CAD groups because it was previously demonstrated that patients with 70% coronary lesion had more severe LV dysfunction than patients with 50% lesion [30]. Finally, we did not measure patients' coronary flow reserve and/or LV pressure invasively. Further and larger studies are needed to explain the effect of the EAT mechanism on LV diastolic properties.

CONCLUSIONS

In current study, we aimed to investigate the association between EAT thickness and LV diastolic functions in patients with normal coronary arteries. Although the underlying mechanism is not fully understood yet, increased EAT thickness consistently showed independent correlation with impaired LVDD parameters in the current study. According to our study results, we may classify EAT thickness as an independent factor for development of LVDD. The role of EAT thickness should be kept in mind when physicians encounter a patient having clinically low output findings and normal LV systolic function.

Conflict of interest: *none declared*

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Wpływ grubości nasierdziowej tkanki tłuszczowej na czynność rozkurczową lewej komory u chorych z prawidłowymi tętnicami wieńcowymi

Mustafa Topuz[1], Ali Dogan[2]

[1]Adana Numune Training and Research Hospital, Adana, Cukurova, Turcja
[2]Erciyes University, Medicine Faculty, Kayseri, Talas, Turcja

Streszczenie

Wstęp i cel: Niniejsze badanie przeprowadzono w celu oceny wpływu grubości nasierdziowej tkanki tłuszczowej (EAT) na czynność rozkurczową lewej komory u chorych, u których w angiografii stwierdzono prawidłowe tętnice wieńcowe (NCA).

Metody: Do badania włączono pacjentów skierowanych na koronarografię z powodu typowego bólu w klatce piersiowej lub nietypowego bólu w klatce piersiowej w połączeniu z dodatnim wynikiem wstępnego badania w kierunku choroby wieńcowej (CAD). Po koronarografii chorych podzielono na trzy grupy: grupę z CAD obejmującą 85 osób z istotnymi zmianami w tętnicach wieńcowych (≥ 50-procentowe zwężenie), grupę z nieistotną CAD utworzoną z 82 osób z nieistotnymi zwężeniami w tętnicach wieńcowych (< 50-procentowe zwężenie) i/lub blaszkami miażdżycowymi oraz grupę NCA, w której znajdowało się 83 osób z prawidłowym obrazem tętnic wieńcowych. U wszystkich chorych wykonano echokardiografię przezklatkową w celu zmierzenia grubości EAT i parametrów czynności rozkurczowej lewej komory.

Wyniki: Wskaźnik Gensiniego, stężenie cholesterolu całkowitego i stężenie białka C-reaktywnego były istotnie wyższe w grupie CAD niż w grupach z nieistotną CAD i NCA. Średnia grubość EAT we wszystkich grupach wynosiła 7,3 ± 2,4 mm. Dla poszczególnych grup grubości EAT wynosiły: 5,8 ± 2,3 mm w grupie NCA, 6,4 ± 2,4 mm w grupie z nieistotną CAD i 7,8 ± 2,2 mm w grupie CAD ($p < 0,001$). Wykazano znamienną korelację średniej grubości EAT ze współczynnikiem E/e' w grupie NCA. W analizie wieloczynnikowej średnia grubość EAT była istotnie związana z dysfunkcją rozkurczową lewej komory u pacjentów z NCA (OR 1,019; 95% CI 1,012–1,027; $p < 0,001$).

Wnioski: Uzyskane wyniki dowodzą, że grubość EAT może wpływać na czynność rozkurczową lewej komory. Ta niezależna relacja wskazuje na ważne implikacje kliniczne pomiaru grubości EAT.

Słowa kluczowe: nasierdziowa tkanka tłuszczowa, czynność rozkurczowa, choroba wieńcowa

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Adres do korespondencji:
Dr. Mustafa Topuz, Adana Numune Training and Research Hospital, Adana, Cukurova, Postal Code: 01150, Turkey, tel: +905066743544, fax: +903223550150, e-mail: topuzm46@gmail.com; mtpuz@hotmail.com

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