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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Relationship between epicardial fat and coronary microvascular dysfunction

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Abstract

Background: Coronary microvascular dysfunction (CMVD) is often suggested in patients with typical chest pain and abnormal stress test results that are indicative of myocardial ischaemia, in whom coronary angiography fails to show fixed stenosis in epicardial coronary arteries. The efficacy of pharmacologic treatment on clinical outcome remains to be determined.

Aim: To determine the relationship between CMVD and epicardial fat thickness (EFT).

Methods: A case-controlled study was conducted on 124 patients, aged 40–91 years. A matched set of 62 symptomatic and 62 asymptomatic patients underwent an exercise electrocardiogram and transthoracic echocardiography. Coronary angiography was performed in patients with abnormal exercise test results. Patients without coronary artery disease were considered for the study. EFT was measured by transthoracic echocardiography in all patients.

Results: EFT was significantly higher in patients with positive exercise test results (but normal epicardial coronary arteries) compared to patients with negative exercise test results ($p < 0.001$). Additionally, the HDL-cholesterol level was significantly lower in patients with positive exercise test results (but normal epicardial coronary arteries) compared to patients with negative exercise test results ($p < 0.0001$).

Conclusions: Patients with increased EFT are at an increased risk for developing angina, recurrent hospitalisation and adverse outcomes, even with normal epicardial coronary arteries.

Key words: coronary atherosclerosis, microcirculation, epicardial fat thickness, echocardiography

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INTRODUCTION

Epicardial coronary artery stenosis is usually responsible for myocardial ischaemia. In the past 30 years, studies have shown that abnormalities in coronary microcirculation may also cause myocardial ischaemia [1]. Coronary microvascular dysfunction (CMVD) is often suggested in patients with typical chest pain and abnormal stress test results indicative of myocardial ischaemia, in whom coronary angiography fails to show fixed stenosis in epicardial coronary arteries [2]. It seems that a sparse distribution of myocardial ischaemia, sufficient to produce exercise electrocardiogram (ECG) changes, may not result in visible contractile abnormalities due to normal function of the surrounding myocardial tissue. Additionally, ischaemic metabolites released into the coronary sinus may be undetected because of dilution of the flow from normal myocardial tissue [1]. Common cardiovascular risk factors (e.g. hypertension, hyperlipidaemia, diabetes mellitus and smoking) are known causes of CMVD [3].

Several mechanisms have been proposed for its pathophysiology, including smooth muscle cell hypertrophy, impairment of smooth muscle cell relaxation, and enhanced vasoconstrictor activity in coronary microcirculation [1]. About 20–30% of patients have progressive worsening of symptoms, which significantly impairs quality of life [4]. In patients with non-ST-elevation myocardial infarction, acute coronary syndrome and normal coronary arteries, a one-year

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rate of death from myocardial infarction was 1.2%, and a recurrence of unstable angina was 8.4% [5]. On the other hand, visceral adipose tissue secretes several pro-inflammatory and pro-atherogenic cytokines [6].

Epicardial adipose tissue (EAT), located beneath the visceral pericardium, is a particular variety of visceral fat [7]. Epicardial fat is usually found in the atrioventricular and interventricular grooves. As the amount of epicardial fat increases, it fills the space between the two ventricles and covers the entire epicardial surface [8]. The physiological, biochemical, and biomolecular properties of EAT and the possible paracrine reactions have been reported in previous studies [7]. Studies have shown the EAT may be a stronger risk factor for coronary artery disease (CAD) than adipose tissue located in other parts of the body [9]. Additionally, there is a robust association between epicardial fat volume and coronary artery calcification [10].

The purpose of this study was to investigate the relationship between epicardial fat (measured by transthoracic echocardiography [TTE]) and objective ischaemia (detected by an exercise ECG) in symptomatic patients with normal epicardial coronary arteries (measured by coronary angiography) after the consideration and exclusion of confounding factors.

METHODS

Patient population

A case-controlled study consisting of 124 individuals was conducted. Sixty-two patients with exertional chest pain and objective ischaemia (detected by an exercise ECG), who underwent coronary angiography with suspicion of CAD and had normal epicardial coronary arteries (measured by coronary angiography), and 62 age- and sex-matched asymptomatic volunteers without ischaemia (detected by an exercise ECG) were included in the study. Patients were referred to the Fatemeh Zahra Teaching Hospital between 2011 and 2012. The study was performed according to the guidelines of the Helsinki Declaration and was approved by the ethics committee of the hospital. Written informed consent was obtained from all participants.

Clinical data collection, monitoring and definitions

Echocardiography was performed on all patients using a Vivid S5 (GE Healthcare, Wauwatosa, WI, USA) 1–3 MHz transducer. Maximum epicardial fat thickness (EFT) was measured using two-dimensional TTE with the long axis view of the right ventricular free wall, perpendicular to the aortic annulus, as an anatomic landmark. Epicardial fat often appears as a hyperechoic space between the outer wall of the myocardium and the visceral layer of the pericardium (Fig. 1). It should be measured at end-systole because it is compressed during diastole [8]. All images were stored on a hard disc for better offline measurements, and the

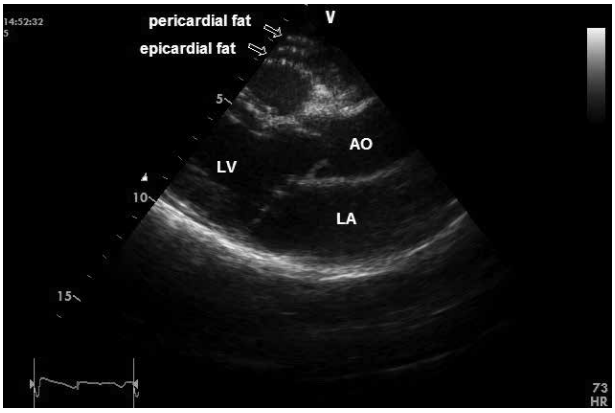

Figure 1. Parasternal long-axis transthoracic two-dimensional echocardiographic images of epicardial fat thickness; Ao — aorta; LA — left atrium; LV — left ventricle

results were confirmed by two echocardiographers who were blinded to the patient's clinical information (one echocardiographer for each patient). The average values of three cardiac cycles from each echocardiographic view were determined. Patients with poor echo window for determining EFT were excluded from study.

To assess the reproducibility of the echocardiographic measurements, EFT was measured in 20 randomly selected patients (according to systematic sampling method) and inter-observer correlation coefficients were calculated by two echocardiographers. In the same group of patients, echocardiographic measurements were repeated one day later to calculate intra-observer correlation coefficients. Inter-observer correlation coefficient was 0.93 and intra-observer correlation coefficient was 0.95. After performing echocardiography, all patients underwent an exercise stress test. A standard Bruce protocol was employed using a h/p/Cosmos Sports & Medical GmbH (Am Sportplatz 8DE 83365, Nussdorf-Traunstein, Germany). During the exercise test, the PQ junction was chosen as the isoelectric point. The development of 0.1 mV (1 mm) or more of flat (< 1 mV/s) J point depression, measured from the PQ junction (depressed 0.1 mV or more than 80 ms after the J point), in three consecutive beats, with a stable baseline, was considered to be an abnormal response.

Normal response (absence of flat ≥ 0.1 mV J point depression in three consecutive beats) was included only after performing ≥ 7 metabolic equivalent (MET) workloads [11].

Patients in the case group were selected from 750 patients who underwent coronary angiography due to an abnormal exercise ECG result and had normal coronary arteries (measured by coronary angiography). CAD was defined as stenosis ≥ 20% in any coronary artery.

Each stenosis can be assigned to the corresponding stenosis degree by applying quantitative coronary angiography. Stenoses [12] are classified as mild (20–50%), moderate (51–70%), severe (71–95%) and occluded (> 95%), respec-

tively. Therefore, we considered normal coronary arteries as less than 20% stenosis in each epicardial coronary artery.

Coronary angiography was performed using a cardiac angiography system (Siemens AG, Medical Solutions, Erlangen, Germany). Patients in the control group were asymptomatic volunteers with a normal exercise ECG result who did not undergo coronary angiography because of the absence of symptoms and no ischaemia, as measured by the exercise test. Patients with anaemia, cardiomyopathy or a decreased left ventricular ejection fraction, digitalis use, hyperglycaemia, hyperventilation, hypokalemia, intraventricular conduction disturbance, left ventricular hypertrophy, mitral valve prolapse, pre excitation syndrome, significant valvular heart disease, severe hypertension, severe hypoxia, or supraventricular tachyarrhythmia were excluded from this study, due to their confounding effects on exercise test results (non-coronary causes of ST-segment depression and false positive exercise test results) [13].

Failure to complete the exercise test (fewer than seven METs) before the appearance of diagnostic ST-depression was another exclusion criterion. Patients with fever, immune deficiency and autoimmune disorders were also excluded from the study. Blood samples were obtained during fasting, and levels of plasma glucose, total cholesterol (TC), high and low density lipoprotein cholesterol (HDL-C, LDL-C), and triglycerides (TG) were measured. Study subjects were designated as having fasting blood glucose (FBS) < 100 mg/dL, FBS ≥ 100 mg/dL, LDL < 100 mg/dL, LDL ≥ 100 mg/dL, TC < 200 mg/dL, TC ≥ 200 mg/dL, TG < 150 mg/dL, or TG ≥ 150 mg/dL [14].

Systolic and diastolic blood pressure was measured after 5 min of rest. Height and weight were measured, and body mass index (BMI) was calculated as body weight divided by height squared. Hypertension was defined as a systolic blood pressure ≥ 140 mm Hg, a diastolic blood pressure ≥ 90 mm Hg [15] or requiring antihypertensive medication. Diabetes mellitus was defined according to the criteria of the American Diabetes Association [16] or requiring insulin or oral hypoglycaemic drugs. A family history of CAD was defined as having a first degree relative (male < 55 years or female < 65 years) with a history of myocardial infarction, coronary revascularisation, or sudden death [17]. A history of smoking was determined by a face-to-face questionnaire.

Statistical analysis

Continuous variables are expressed as the mean ± standard deviation. A t-test was used to assess differences among groups, and categorical variables were compared with a χ^2 test. A p value < 0.05 was considered statistically significant and a 95% confidence interval (CI) was used. Well-known CAD risk factors were included in a conditional logistic regression model of CAD. All statistical calculations were performed using SPSS/PASW (Predictive Analytics SoftWare) Statistics 18 software (SPSS Inc., Chicago, IL, USA). Sample size was determined by a previous study [10] and with the following statistical formula:

$$n = \frac{(z_{1-\frac{\alpha}{2}} + z_{1-\beta})^2 \times (\sigma_1^2 + \sigma_2^2)}{(\mu_1 - \mu_2)^2} = 60, \quad in \quad each \quad groups$$

$$z_{1-\frac{\alpha}{2}} = 1.96, \quad z_{1-\beta} = 1.28, \quad \alpha = 0.05, \quad \beta = 0.1$$

$$\sigma_1^2 = 5.95, \quad \sigma_2^2 = 95.45$$

$$\mu_1 = 5.03, \quad \mu_2 = 9.24$$

RESULTS

Baseline clinical characteristics of the study population are presented in Table 1. Sixty-two separate patients were included in both the negative and positive exercise tolerance test results (there were four missed dates).

Demographic profile, common cardiovascular risk factors, BMI and EFT results of the study groups (categorised by exercise test results) are presented in Tables 2 and 3. The mean age of subjects with negative exercise test results was 52.11 ± 7.83 years, and the mean age of subjects with positive exercise test results (but normal epicardial coronary arteries) was 52.26 ± 7.22 years (p ≥ 0.916). According

Table 1. Baseline clinical characteristics of the study population

	N	Minimum	Maximum	Mean	Standard deviation
Age	127	40	91	52.31	7.712
Triglyceride	128	50	1204	199.19	149.155
Cholesterol	127	107	422	203.57	51.151
High-density lipoprotein	121	19	89	46.25	10.306
Low-density lipoprotein	121	57	224	111.93	31.127
Fasting blood glucose	128	65	353	116.51	46.620
Epicardial fat	128	1.00	10.00	5.6016	1.77138
Body mass index	127	19.81	43.85	27.0346	4.21977
Missed exercise test data	4				

Table 2. Measures of epicardial fat and other known cardiovascular risk factors of subjects by study group

	Negative exercise test			Positive exercise test		
	N	Mean	Standard deviation	N	Mean	Standard deviation
Epicardial fat	62	5.0274	2.08548	62	6.1097	1.17260
Age	61	52.11	7.836	62	52.26	7.225
Triglyceride	62	197.05	104.461	62	204.74	187.345
Cholesterol	61	209.07	48.576	62	199.66	53.423
High-density lipoprotein	55	50.51	10.686	62	42.42	8.446
Low-density lipoprotein	55	115.56	33.825	62	110.06	28.866
Fasting blood glucose	62	111.90	37.008	62	122.21	55.233
Body mass index	61	28.8399	4.68103	62	25.3425	2.95410

Table 3. Percentage of common cardiovascular risk factors of subjects by study group

	Negative exercise test	Positive exercise test (normal epicardial arteries)	Total	P
Women	48 (77.4%)	44 (70%)	92 (74%)	≥ 0.412
Men	14 (22.6%)	18 (30%)	32 (26%)	
Smoking	5 (45.5%)	6 (54.5%)	11 (100%)	≥ 0.752
Family history	25 (54.3%)	21 (45.7%)	46 (100%)	≥ 0.457
Diabetes mellitus	19 (61.3%)	12 (38.7%)	31 (100%)	≥ 0.147
Hypertension	28 (47.5%)	31 (52.5%)	59 (100%)	≥ 0.590
Hypercholesterolaemia ≥ 200 mg/dL	32 (53.3%)	28 (46.7%)	60 (100%)	≥ 0.418
Hypertriglyceridaemia ≥ 150 mg/dL	41 (54.7%)	34 (45.3%)	75 (100%)	≥ 0.199
Low-density lipoprotein ≥ 100 mg/dL	32 (52.5%)	29 (47.5%)	61 (100%)	≥ 0.218
Fasting blood glucose ≥ 100 mg/dL	34 (48.6%)	36 (51.4%)	70 (100%)	≥ 0.717

Table 4. Use of logistic regression analysis to predict abnormal exercise test

	B	SE	Wald	df	Sig.	Exp (B)	Exp (B) for 95% CI	
	Lower	Upper	Lower	Upper	Lower	Upper	Lower	Upper
High-density lipoprotein	–0.110	0.030	13.456	1	0.000	0.896	0.845	0.950
Epicardial fat	0.470	150.0	9.865	1	0.002	1.600	1.193	2.146

CI — confidence interval; Exp (B) — exponential (B); Sig. — significant; SE — standard error

to the results of statistical testing, there was no significant difference between the two groups for common cardiovascular risk factors such as diabetes mellitus, hypertension, history of smoking, family history and hyperlipidaemia. The difference between the exercise test results and EFT means was assessed by an independent t-test. EFT was significantly higher in patients with positive exercise test results (but normal epicardial coronary arteries) compared to patients with negative exercise test results (6.1 ± 1.17 vs. 5 ± 2.08 mm, $p < 0.001$). Logistic regression analysis confirmed this correlation (odds ratio [OR] 1.6, 95% CI 1.21–2.12) (Table 4, Fig. 2).

Additionally, an independent t-test showed that HDL-C level was significantly lower in patients with positive exercise test results (but normal epicardial coronary arteries) compared to patients with negative exercise test results (42.42 vs. 50.51 mg/dL, $p < 0.0001$). Logistic regression analysis confirmed this negative correlation (OR 0.898, 95% CI 0.85–0.948) (Table 4, Fig. 3). BMI was also significantly lower in patients with positive exercise test results (25.3 ± 2.95 kg/m^2) compared to patients with negative exercise test results (28.83 ± 4.68 kg/m^2, $p < 0.0001$).

The association between exercise test results and variables related to cardiovascular risk factors was assessed

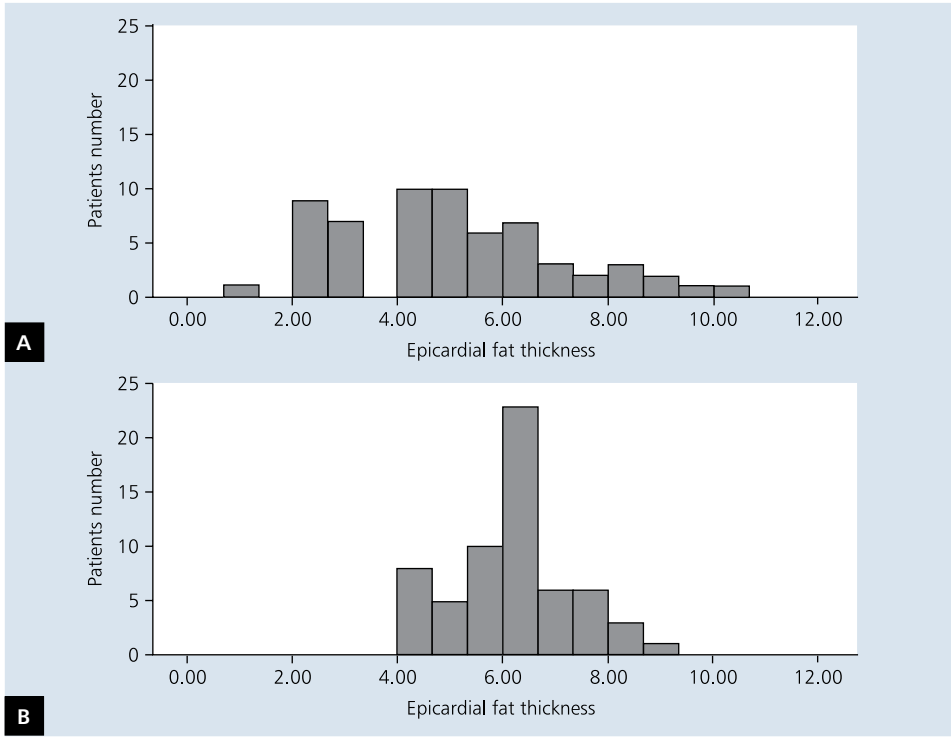

Figure 2. Distribution diagram for epicardial fat thickness by study group; **A**. Normal exercise test; **B**. Abnormal exercise test

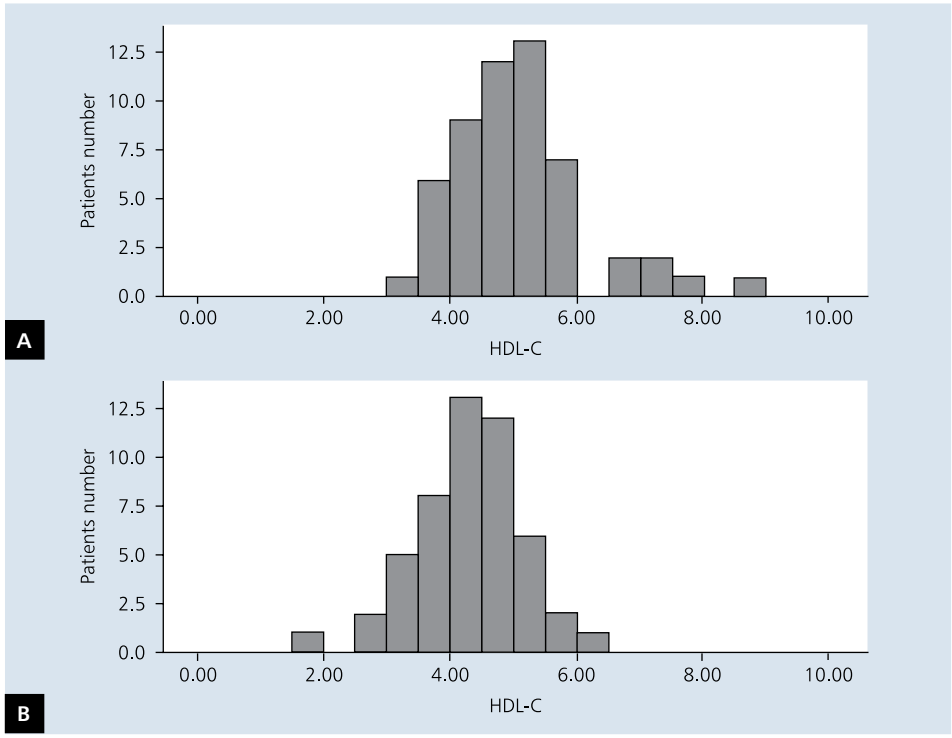

Figure 3. Distribution diagram for high-density lipoprotein-cholesterol (HDL-C) by study group; **A**. Normal exercise test; **B**. Abnormal exercise test

by a χ^2 test. No significant correlation was found for sex ($p \geq 0.412$), family history of CAD ($p \geq 0.457$), smoking ($p \geq 0.752$), diabetes mellitus ($p \geq 0.147$), hypertension ($p \geq 0.59$), hypertriglyceridaemia ($p \geq 0.199$), hypercholesterolaemia ($p \geq 0.418$), FBS ≥ 100 mg/dL ($p \geq 0.717$), or LDL-C ≥ 100 mg/dL ($p \geq 0.218$). Furthermore, an independent t-test did not show significant correlations between exercise test results and FBS level ($p \geq 0.225$), LDL-C level ($p \geq 0.349$), TC level ($p \geq 0.309$), TG level ($p \geq 0.778$) and age ($p \geq 0.916$).

DISCUSSION

Until recently, magnetic resonance imaging (MRI) was known as the gold standard for the measurement of EFT. Iacobellis et al. [8] reported the echocardiographical measurement of epicardial fat for the first time. They showed an excellent correlation between echocardiographical EFT and MRI epicardial fat measurements. Echocardiographically measured epicardial fat may provide a highly reliable index of true visceral fat content, without probable confounding effects of increased subcutaneous abdominal fat.

Echocardiographic EFT varies from a minimum of 1 mm to a maximum of 23 mm. This variability is probably due to a variation in abdominal visceral fat distribution [8]. In our study, this range is from a minimum of 1 mm to a maximum of 10 mm. Natale et al. [18] set 7 mm as the upper limit of EFT on the basis of mean value of EFT in 58 normal volunteers. This was consistent with our study (average EFT in our study was 5.6 mm).

In 2013, Nabati et al. [19] evaluated the relationship between EFT (measured by TTE) and coronary atherosclerosis in 143 patients. Patients were divided into two groups, one group with EFT equal to or more than 7 mm and another group with EFT less than 7 mm. EFT was significantly correlated with the existence and severity of CAD ($p < 0.001$). In 2012, Erdogan et al. [20] evaluated the relationship between EFT (measured by TTE) and isolated slow coronary flow (SCF) in 66 individuals with normal coronary arteries (measured by coronary angiography). EFT was significantly increased in the SCF group compared to the normal coronary flow group ($p < 0.001$). Several mechanisms have been proposed for SCF, including small vessel disease, microvascular dysfunction and endothelial dysfunction.

In 2009, Sade et al. [21] evaluated the relationship between EFT (measured by TTE) and coronary flow reserve (CFR) in 68 women with chest pain and normal coronary arteries (measured by coronary angiography). Forty percent had reduced CFR, suggesting microvascular dysfunction. Menopause, hypertension and abnormal stress test results were significantly more prevalent, and EFT was significantly increased in women with microvascular dysfunction ($p < 0.0001$).

CMVD is present in approximately one half of women with chest pain in the absence of obstructive CAD and cannot be predicted by risk factors for atherosclerosis and hormone levels [22]. This is an explanation for the higher percentage of women in our study.

In the present study, the difference between EFT in symptomatic patients with objective ischaemia (determined by stress test results) but normal epicardial coronary arteries and matched asymptomatic patients without ischaemia (determined by stress test results) was only 1.1 mm. For determining whether EFT is a risk factor for having CMVD, conditional logistic regression analysis was performed and OR was calculated. CMVD was also 1.6 times more prevalent in persons with thicker epicardial adipose tissue compared to persons without it (95% CI 1.21–2.12).

To the best of our knowledge, this is the first study evaluating the relationship between ischaemia (determined by a non-invasive test) and increased EFT in patients with normal epicardial arteries. Typical chest pain and abnormal stress test results without non-coronary causes of ST-segment depression in patients with normal epicardial coronary arteries suggest CMVD [1]. Its aetiology is diverse and may be due to endothelial reactivity, low endogenous oestrogen levels, coagulopathies and abnormal inflammatory reaction. The prognosis is worse in women with various risk factors. Therefore, aggressive risk factor modification is recommended in these patients [23].

One powerful modifiable risk factor is low HDL-C. Patients with low HDL-C levels (≤ 35 mg/dL) were at a three times greater risk for CAD. CAD risk decreased by 2–3% for every 1 mg/dL increase in HDL-C [24]. The primary protective mechanism of HDL-C is reverse cholesterol transport. Additionally, HDLs have antioxidant activity and beneficial effects on endothelial functions. Again, to the best of our knowledge, our study demonstrated for the first time a significant negative correlation between abnormal stress test results and HDL-C levels in patients with normal epicardial coronary arteries.

Additionally, in our study, patients with ischaemic stress test results and normal epicardial coronary arteries had lower BMIs compared to asymptomatic non-ischaemic patients. Regarding reverse correlation between BMI and EFT, it is probably due to a higher incidence of significant epicardial CAD in patients with higher BMI which led to exclusion of them from the study.

Thus, it seems that a positive exercise test in patients with higher BMI is more probably due to significant CAD compared to patients with lower BMI. This finding eliminates BMI as a confounding risk factor in producing ischaemia in exercise test results and confirms the high power of epicardial fat alone, independent of BMI, in predicting CMVD.

Limitations of the study

Our study was case-controlled. Therefore, symptom alteration with EFT variation in individual patients could not be measured. Also, there were more women than men in the study,

and women are known to have more false positive results on exercise ECG studies, and all of the positive treadmill tests are not from CMVD. In the current study, patients with fever, immune deficiency and autoimmune disorders were excluded. This exclusion was due to their confounding effects on EFT or exercise test results. So, the role of inflammatory particles could not be determined.

CONCLUSIONS

MRI has been known as the gold standard for the measurement of EFT. TTE is a good alternative due to its lower cost, easy accessibility and bedside performance. We used conventional coronary angiography as a tool for excluding CAD. This was due to the relative low positive predictive value of computed tomography (CT) angiography.

The presence of a significant stenosis on CT coronary angiography frequently is not confirmed on conventional coronary angiography [25]. This discrepancy has been attributed to the inferior spatial and temporal resolution of CT angiography when compared to conventional coronary angiography [25]. Our study demonstrated that patients with increased EFT are at increased risk for developing angina, recurrent hospitalisation and adverse outcomes even with normal epicardial coronary arteries. Additionally, it seems that modifying HDL-C may be a useful option in decreasing symptoms in resistant patients, in whom coronary angiography has revealed normal epicardial coronary arteries.

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Conflict of interest: *none declared*

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Zależność między nasierdziową tkanką tłuszczową a dysfunkcją mikrokrążenia wieńcowego

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Streszczenie

Wstęp: U chorych z typowymi bólami w klatce piersiowej i nieprawidłowym wynikiem próby wysiłkowej wskazującymi na niedokrwienie mięśnia sercowego, u których angiografia wieńcowa nie wykazuje stałego zwężenia w nasierdziowych tętnicach wieńcowych, często sugeruje się występowanie dysfunkcji mikrokrążenia wieńcowego (CMVD). Nie ustalono dotychczas, czy farmakoterapia jest skuteczna w przypadku tego zaburzenia.

Cel: Celem badania było określenie zależności miedzy CMVD a grubością nasierdziowej tkanki tłuszczowej (EFT).

Metody: Przeprowadzono badanie kliniczno-kontrolne z udziałem 124 chorych w wieku 40–91 lat. Odpowiednio dobraną grupę 62 chorych z objawami niedokrwiennymi i 62 pacjentów bez objawów poddano wysiłkowemu badaniu elektrokardiograficznemu oraz echokardiografii przezprzełykowej. U chorych z nieprawidłowymi wynikami próby wysiłkowej wykonano angiografię wieńcową. Pacjenci bez choroby wieńcowej mogli zostać włączeni do badania. U wszystkich osób zmierzono EFT za pomocą echokardiografii przezprzełykowej.

Wyniki: U pacjentów z dodatnim wynikiem próby wysiłkowej (lecz prawidłowym obrazem nasierdziowych tętnic wieńcowych) EFT była istotnie większa niż u osób z ujemnym wynikiem próby wysiłkowej ($p < 0,001$). Ponadto u chorych z dodatnim wynikiem próby wysiłkowej (lecz prawidłowym obrazem nasierdziowych tętnic wieńcowych) stwierdzono istotnie niższe stężenia cholesterolu w porównaniu z wynikami uzyskanymi u osób z ujemnym wynikiem próby wysiłkowej ($p < 0,0001$).

Wnioski: U chorych z grubszą warstwą nasierdziowej tkanki tłuszczowej występuje zwiększone ryzyko dławicy, ponownych hospitalizacji i niekorzystnych zdarzeń, nawet jeśli obraz nasierdziowych tętnic wieńcowych w koronarografii jest prawidłowy.

Słowa kluczowe: miażdżyca tętnic wieńcowych, mikrokrążenie, grubość nasierdziowej tkanki tłuszczowej, echokardiografia

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