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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The impact of diabetes on the association between epicardial fat thickness and extent and complexity of coronary artery disease in patients with non-ST elevation myocardial infarction

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Abstract

Background: Epicardial fat thickness (EFT) is associated with increased cardio metabolic risk. Recent studies have suggested that there is a strong relationship between diabetes and EFT. Although the relationship between EFT and coronary artery disease (CAD) is well known, the possible impact of diabetes on the relationship between EFT and extent and complexity of CAD was not fully investigated.

Aim: We aimed to investigate the relationship between EFT and extent and complexity of CAD in patients with non-ST elevation myocardial infarction (NSTEMI) with and without diabetes.

Methods: We prospectively included 454 patients with NSTEMI (mean age: 61.8 ± 10.4 years) in the present study. Patients were classified into two groups according to their diabetes status (diabetic group and non-diabetic group). EFT was measured by transthoracic echocardiography on the right ventricle in individuals having the left lateral decubitus position. SYNTAX score was used to define the extent and complexity of CAD. High-sensitivity C-reactive protein (hs-CRP) and other biochemical markers were measured in all participants.

Results: Diabetic patients had higher EFT values compared with non-diabetics ($p < 0.05$). EFT is independently associated with diabetes, SYNTAX score, and hs-CRP in all patients ($p < 0.05$, for all). When patients were divided into two groups, as diabetic and non-diabetic, the association between EFT and SYNTAX score was stronger in diabetic patients compared with non-diabetics ($r = 0.635$; $p < 0.001$ vs. $r = 0.179$; $p = 0.003$).

Conclusions: Epicardial fat thickness is associated with SYNTAX score in both diabetic and non-diabetic patients. Furthermore, there is a stricter relationship between EFT and SYNTAX score in diabetic patients.

Key words: epicardial fat thickness, SYNTAX, diabetes, non-ST elevation myocardial infarction, C-reactive protein

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INTRODUCTION

Epicardial adipose tissue (EAT), the visceral fat depot of the heart, is a metabolically active organ and reflects intra-abdominal and myocardial fat and correlates with metabolic syndrome, coronary artery disease (CAD), and type 2 diabetes mellitus (T2DM) [1–4]. EAT stimulates paracrine actions that include increased production of reactive oxygen species, and atherogenic and inflammatory cytokines that can lead to myocardial inflammation and dysfunction as well as left ventricular hypertrophy and CAD [5]. It is well known that

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epicardial fat thickness (EFT) is associated with both the presence and the severity of CAD in patients with stable and unstable CAD [4, 6–9]. Moreover, it has been reported that patients with increased EFT are at increased risk for developing angina, recurrent hospitalisation, and adverse outcomes [10].

It is well known that patients with T2DM are at increased risk of cardiovascular disease due to accelerated atherosclerosis. Previous studies have reported that increased EAT is associated with T2DM as well as CAD [2–4, 11]. Moreover, EAT has been demonstrated to be associated with fasting glucose, insulin resistance, and adiponectin in patients with T2DM [12–14]. In a recent study, Iacobellis et al. [15] reported that patients with type 1 diabetes have higher EAT values than non-diabetic subjects.

Although the relationship between EFT with T2DM and CAD is well known, the possible impact of diabetes on the association between EFT and the extent and complexity of CAD has not been fully investigated. The main purpose of our study is to investigate the relationship between EFT and extent and complexity of CAD indicated by SYNTAX score in patients with non-ST elevation myocardial infarction (NSTEMI) with and without diabetes.

METHODS

Study population

The population of this study consists of 454 consecutive patients (299 males and 155 females; mean age 61.8 ± 10.4 years) admitted to the Cardiology Clinic of the Adana Numune Training and Research Hospital with NSTEMI between January 2014 and August 2014. Patients are diagnosed as NSTEMI when there is an elevation in the troponin-I levels (more than 1.0 ng/mL in any sample during the first 12 h post-admission), with or without ST/T changes in the electrocardiography, in the presence of features of unstable angina pectoris [16]. Patients who had coronary lesions with a diameter stenosis of ≥ 50%, in vessels of size ≥ 1.5 mm, were included. Patients also underwent transthoracic echocardiography before the coronary angiography.

Patients with a previous history of CAD, stable and unstable angina, ST elevation myocardial infarction, typical signs and symptoms of congestive heart failure, left ventricular (LV) systolic dysfunction (ejection fraction [EF] < 55%), severe valvular heart disease, myocardial or pericardial disease, neoplastic disease, recent major surgical procedures, and liver or kidney disease (eGFR < 60) were excluded from the study. Moreover, patients with poor image quality in the echocardiographic examination were also excluded. The Institutional Ethics Committee approved the study protocol and each participant provided written informed consent.

After assessment of detailed medical history and a complete physical examination, the baseline characteristics including age, sex, hypertension, hyperlipidaemia, T2DM, smoking status, family history of CAD, body mass index (BMI), and previous medications were recorded for all patients. T2DM was diagnosed according to American Diabetes Association criteria [17].

Blood measurements

After 12 h of fasting, venous blood samples were collected from all participants to measure fasting plasma glucose, uric acid, lipid profile, and serum creatinine with an auto-analyser. Highly-sensitive C-reactive protein (hs-CRP) was measured with an auto-analyser (Aeroset) using a commercial spectrophotometric kit (Scil Diagnostics GmbH, Viernheim, Germany). Highly-sensitive cardiac troponin I (hs-cTnI) was measured using a fourth-generation assay on an Elecsys 2010/cobas e 411 instrument (Roche Diagnostics, Mannheim, Germany).

Measurement of EFT

Standard parasternal long-axis and short-axis views from two-dimensional images were used for measurement of EFT on the free wall of the right ventricle while patients were in left lateral decubitus position. Images from standard parasternal long- and short-axis views were digitally stored and reviewed by a single echocardiographer blinded to the clinical data. EFT obtained by echocardiography is generally determined as the relatively echo-lucent space between the outer wall of the myocardium and the visceral layer of pericardium; and is measured perpendicularly on the free wall of the right ventricle at end-systole in three cardiac cycles [18]. The average value of three cardiac cycles from each echocardiographic view was determined. Intra-observer variability of EFT measurement was evaluated from 50 randomly selected patients. Selected subjects underwent echocardiography done by the same echocardiographer one week later. The coefficient of intra-observer variation was 2.4%.

Extent and complexity of CAD (SYNTAX score) and angiographic analysis

Coronary lesions leading to a diameter stenosis of ≥ 50% in vessels of ≥ 1.5 mm were scored separately and added together to provide the cumulative SYNTAX score, which was prospectively calculated using the SYNTAX score algorithm on the baseline diagnostic angiogram [19]. Two experienced interventional cardiologists analysed the SYNTAX score; the opinion of a third analyst was obtained, and the final judgment was made by consensus in cases of disagreement. Analysts who were blinded to procedural data and clinical outcome calculated the final score using individual lesion scores.

Statistical analysis

Statistical analysis was carried out using SPSS 17.0 for Windows (SPSS Inc., Chicago, Illinois, USA). Data are expressed as mean value ± standard deviation. Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Categorical variables were compared using the χ^2 test. Comparisons of continuous variables between the two groups were

Table 1. Comparison of baseline, clinical, and laboratory findings between the groups

Variables	Diabetic group (n = 186)	Non-diabetic group (n = 268)	p
Baseline findings			
Age [years]	62.5 ± 9.6	61.2 ± 10.9	0.165
Gender [male]	117 (62.9%)	182 (67.9%)	0.157
BMI [kg/m^2]	28.6 ± 4.4	27.6 ± 4.2	0.013
Hypertension	134 (72.0%)	114 (42.5%)	< 0.001
Current smoking	68 (36.6%)	117 (43.7%)	0.078
Hyperlipidaemia	104 (55.9%)	81 (30.2%)	< 0.001
Family history	54 (29.4%)	59 (22%)	0.056
SBP [mm Hg]	125.9 ± 28.4	119.5 ± 19.1	0.005
DBP [mm Hg]	77.0 ± 13.3	75.7 ± 12.9	0.306
Laboratory findings			
Glucose [mg/dL]	179.6 ± 86.5	105.2 ± 18.1	< 0.001
HbA1c [%]	7.7 ± 2.2	5.4 ± 0.3	< 0.001
Total cholesterol [mg/dL]	197.8 ± 46.5	191.0 ± 44.7	0.116
Triglyceride [mg/dL]	187.2 ± 96.4	155.7 ± 92.5	< 0.001
HDL [mg/dL]	39.1 ± 9.2	41.2 ± 12.8	0.064
LDL [mg/dL]	129.3 ± 38.9	127.0 ± 35.8	0.432
Creatinine [mg/dL]	1.0 ± 0.8	0.9 ± 0.6	0.039
Uric acid [mg/dL]	5.6 ± 1.4	5.4 ± 1.5	0.300
Hs-CRP [mg/dL]	0.97 ± 0.4	0.72 ± 0.4	< 0.001
Peak troponin T [ng/dL]	568.2 ± 274.0	478.6 ± 333.9	0.153
WBC [×1000/μL]	9.6 ± 2.3	9.2 ± 2.8	0.148
Haemoglobin [mg/dL]	13.3 ± 1.9	13.7 ± 1.8	0.017
Echocardiography			
LAD [mm]	38.0 ± 3.5	37.8 ± 4.8	0.770
LVID [mm]	46.6 ± 4.7	46.9 ± 4.0	0.497
EF [%]	54.0 ± 10.7	55.7 ± 6.7	0.049
EFT [mm]	6.5 ± 0.7	5.3 ± 1.0	< 0.001
Angiography			
SYNTAX score	22.9 ± 7.9	14.6 ± 5.7	< 0.001
Previous medications			
ACE-I use	52 (28%)	70 (26.1%)	0.371
ARB use	42 (22.6%)	46 (17.2%)	0.095
Beta-blocker use	11 (5.9%)	23 (8.6%)	0.190
Statin use	34 (18.3%)	44 (16.4%)	0.347

Data are presented as mean ± standard deviation or number (percentage); BMI — body mass index; SBP — systolic blood pressure; DBP — diastolic blood pressure; HbA1c — haemoglobin A1c; HDL — high-density lipoprotein; LDL — low-density lipoprotein; hs-CRP — highly-sensitive C-reactive protein; WBC — white blood cell; LAD — left atrial diameter; LVID — left ventricle internal diameter; EF — ejection fraction; EFT — epicardial fat thickness; ACE-I — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor blocker

performed using the independent samples t-test**.** Multiple linear regression analysis was used to determine the independent relationships of EFT. All significant parameters in the univariate analysis were selected in the multivariate model. A p-value of < 0.05 was considered significant.

RESULTS

The mean EFT values of diabetes and non-diabetes groups were 6.5 ± 0.7 mm and 5.3 ± 1.0 mm, respectively (Table 1).

Patients with diabetes had higher frequency of hypertension and hyperlipidaemia, BMI and systolic blood pressure,

Table 2. Bivariate and multivariate relationships of epicardial fat thickness in all patients group

Variables	Pearson correlation coefficient	p	Standardised regression coefficients*	p
Age [years]	0.145	0.002	0.008	0.824
BMI [kg/m^2]	0.206	< 0.001	0.129	< 0.001
Hypertension	0.242	< 0.001	–0.004	0.921
Hyperlipidaemia	0.117	0.012	–0.037	0.290
Diabetes	0.596	< 0.001	0.384	< 0.001
Glucose	0.297	< 0.001	–0.004	0.936
HbA1c	0.321	< 0.001	–0.037	0.447
Uric acid	0.111	0.018	–0.002	0.953
Hs-CRP	0.488	< 0.001	0.279	< 0.001
Peak troponin	0.131	0.005	0.033	0.320
SYNTAX score	0.559	< 0.001	0.298	< 0.001

*Multiple linear regression analysis. Abbreviations as in Table 1

Table 3. Bivariate and multivariate relationships of epicardial fat thickness in non-diabetic patients

Variables	Pearson correlation coefficient	p	Standardised regression coefficients	p
BMI	0.158	0.009	0.152	0.004
Hs-CRP	0.484	< 0.001	0.449	< 0.001
SYNTAX score	0.179	0.003	0.136	0.012
Total cholesterol	0.150	0.014	0.094	0.081

Abbreviations as Table 1

glucose level, HbA1c, triglyceride, creatinine, and hs-CRP levels, and had lower haemoglobin levels compared with non-diabetic patients ($p < 0.05$ for all).

Moreover, EFT and SYNTAX score values were higher and LVEF values were lower in diabetic patients compared with non-diabetic patients ($p < 0.05$ for all).

Relationships of EFT in all patients

Epicardial fat thickness is associated with age ($r = 0.151$, $p = 0.001$), BMI ($r = 0.200$, $p < 0.001$), hypertension ($r = 0.270$, $p < 0.001$), hyperlipidaemia ($r = 0.119$, $p = 0.011$), diabetes ($r = 0.575$, $p < 0.001$), glucose ($r = 0.290$, $p < 0.001$), HbA1c ($r = 0.310$, $p < 0.001$), uric acid ($r = 0.094$, $p = 0.046$), hs-CRP ($r = 0.503$, $p < 0.001$), peak troponin I ($r = 0.134$, $p = 0.034$), and SYNTAX score ($r = 0.594$, $p < 0.001$) in bivariate analysis.

In the cohort of all patients, EFT is independently associated with BMI ($\beta = 0.119$, $p < 0.001$), diabetes ($\beta = 0.321$, $p < 0.001$), hs-CRP ($\beta = 0.299$, $p < 0.001$), and SYNTAX score ($\beta = 0.355$, $p < 0.001$) in multiple linear regression analysis (Table 2).

Relationships of EFT in the non-diabetic group

Epicardial fat thickness is associated with BMI ($r = 0.148$, $p = 0.015$), hs-CRP ($r = 0.516$, $p < 0.001$), SYNTAX score ($r = 0.296$, $p < 0.001$), and total cholesterol level ($r = 0.169$, $p = 0.005$) in bivariate analysis.

Epicardial fat thickness is independently associated with BMI ($\beta = 0.150$, $p = 0.004$), hs-CRP ($\beta = 0.461$, $p < 0.001$), and SYNTAX score ($\beta = 0.200$, $p < 0.001$) in multiple linear regression analysis (Table 3).

Relationships of EFT in the diabetic group

Epicardial fat thickness is associated with age ($r = 0.280$, $p < 0.001$), BMI ($r = 0.199$, $p = 0.007$), hypertension ($r = 0.237$, $p = 0.001$), peak troponin-I level ($r = 0.152$, $p = 0.038$), hs-CRP ($r = 0.289$, $p < 0.001$), and SYNTAX score ($r = 0.635$, $p < 0.001$) in bivariate analysis.

Multiple linear regression analysis showed that EFT is associated with BMI ($\beta = 0.150$, $p = 0.008$), hs-CRP ($\beta = 0.201$, $p < 0.001$), and SYNTAX score ($\beta = 0.633$, $p < 0.001$) (Table 4).

Table 4. Bivariate and multivariate relationships of epicardial fat thickness in diabetic patients

Variables	Pearson correlation coefficient	p	Standardised regression coefficients	p
Age	0.280	< 0.001	0.030	0.616
BMI	0.199	0.007	0.150	0.008
Hypertension	0.238	0.001	-0.090	0.148
Peak troponin I	0.152	0.038	0.068	0.210
Hs-CRP	0.289	< 0.001	0.201	< 0.001
SYNTAX score	0.635	< 0.001	0.633	< 0.001

Abbreviations as in Table 1

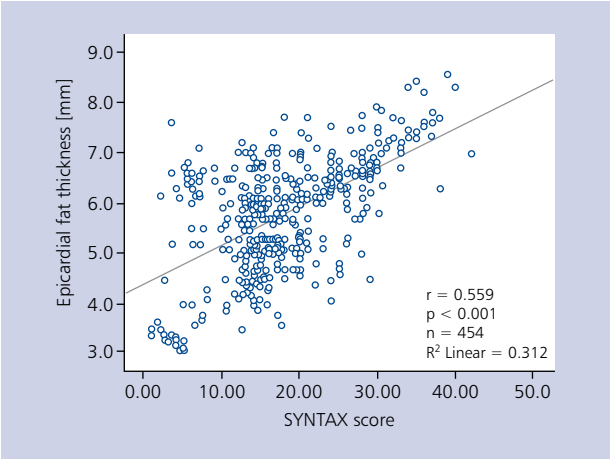

Figure 1. Relationship between epicardial fat thickness and SYNTAX score in all patients

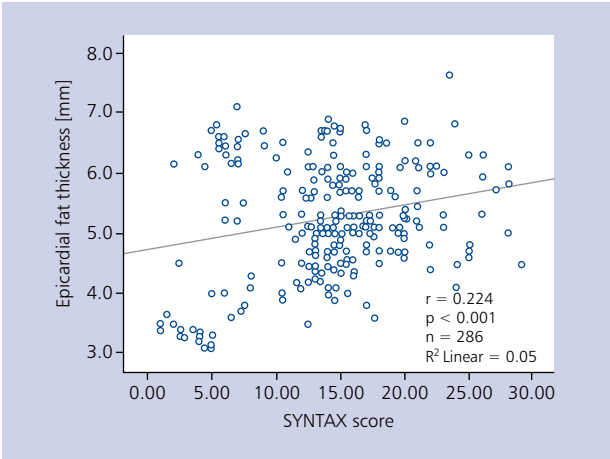

Figure 2. Relationship between epicardial fat thickness and SYNTAX score in non-diabetic patients

The relationships of EFT with SYNTAX score in all patients, non-diabetic patients and diabetic patients are demonstrated in Figures 1, 2, and 3.

DISCUSSION

According to the results of our study, NSTEMI patients with diabetes had higher EFT values compared with non-diabetic patients. We also demonstrated that the relationship between EFT and SYNTAX score in NSTEMI patients with diabetes was stronger than for the non-diabetic NSTEMI patients.

Epicardial adipose tissue has recently been reported to be a new risk factor, and to actively contribute, in metabolic and cardiovascular diseases [2]. In the present study, we showed that NSTEMI patients with diabetes have higher EFT values compared with non-diabetic NSTEMI patients. Our results are consistent with previous studies [2–4, 11]. In several studies, it has been demonstrated that both EFT and EAT volume were increased in T2DM patients [3, 4]. Also, in a recent study, Iacobellis et al. [15] reported that T1DM patients had higher EFT values compared with non-diabetic subjects. On the other hand, the accumulation of epicardial fat helps to reduce

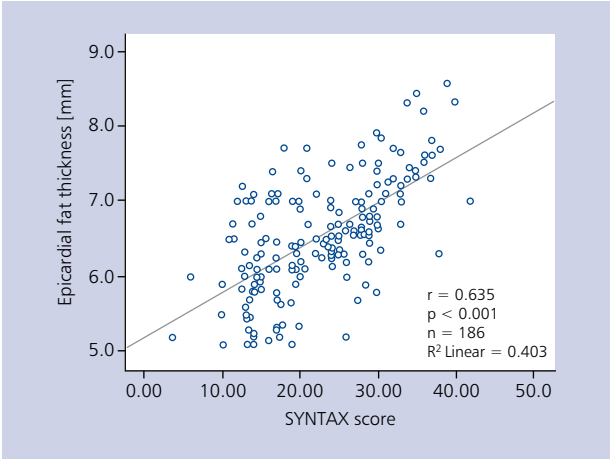

Figure 3. Relationship between epicardial fat thickness and SYNTAX score in diabetic patients

insulin sensitivity and adiponectin, which causes increased inflammation in adipose tissue, activating the expression and secretion of pro-inflammatory cytokines and contributing to

the development of T2DM [2, 12–14]. In a previous study, the ability of EFT in predicting glucose abnormalities was assessed [13]. In that study, subjects were categorised as having normal fasting glucose and impaired fasting glucose (fasting glucose between 100 mg/dL and 126 mg/dL). EFT was found to be significantly higher in patients with impaired fasting glucose and correlated strongly with the level of fasting glucose [13]. Insulin resistance is one of the major components of T2DM. It has been shown in obese subjects that EFT was associated with indices of insulin sensitivity assessed by euglycaemic hyperinsulinaemic clamp [20]. We did not investigate the mechanism of increased EFT in T2DM patients in the present study. However, it was suggested that increases of EAT in T2DM patients may be related to impaired insulin sensitivity and the lipid metabolism pathway [4].

Epicardial fat thickness is an independent predictor of coronary atherosclerosis. In several previous studies it was suggested that EAT might be a contributing factor in the pathogenesis of CAD [1, 4–9]. Moreover, it is well known that EFT is associated with the severity of CAD and with potentially the most dangerous types of atherosclerotic plaques [4, 5, 7–9, 11]. EFT is also shown to be related with unstable plaques (high necrotic core rate and burden plaque) rather than stable plaque (fibrous plaque) in patients with NSTEMI [21]. EAT releases numerous inflammatory cytokines and affects the coronary arteries as a paracrine organ, given the anatomical contiguity between the two structures without any intervening fascial barriers [2, 5]. An increase in EAT thickness causes activation of the production of reactive oxygen species, atherogenic and inflammatory cytokines [1]. In concordance with our findings, it has been demonstrated that EFT was strongly associated with CAD in combination with adipocytokine imbalance, macrophage polarisation, and inflammatory cell infiltration, independent of overall adiposity [22, 23]. All of these mechanisms may explain the relationship between EFT and coronary atherosclerosis and its severity.

Although the relationship between EFT and CAD is well known, the impact of T2DM on the relationship between EFT and the extent and complexity of CAD has not been fully investigated in patients with ACS. In a recent study, Groves et al. [11] reported that after adjustment for coronary artery calcium score in asymptomatic subjects with and without T2DM, EAT volume was an independent predictor of CAD, and an increasing volume of EAT was associated with the increasing severity of CAD [11]. However, in that study [11], the authors did not investigate the relationship between EAT and the extent and complexity of CAD in patients with and without diabetes. In the present study, we demonstrated the relation between EFT and SYNTAX score, which was stronger in NSTEMI patients with diabetes compared with non-diabetic NSTEMI patients. The exact mechanism of this powerful relationship in diabetic patients is not fully understood. However, the association between EFT and the pathogenesis of diabetic coronary atherosclerosis was shown in previous studies [4]. Although in patients with T2DM functional pericardial fat tissue may play a protector role over the myocardium or endothelium, EAT may lead to impairment in the contraction of cardiomyocytes. The close anatomical relationship between EAT, a biologically active adipokine-secreting tissue, and the coronary arteries, suggests that EAT make a contribution in the pathogenesis of diabetic coronary atherosclerosis. It has been suggested that the proteins secreted by EAT, such as alfa-1-glycoprotein, might play a crucial role in the connection between T2DM and the complexity of coronary lesions in patients with CAD [24].

In the present study, we also showed that EFT is associated with low-grade systemic inflammation. Similar findings between EFT and hs-CRP were reported in some recent studies [9]. It has been demonstrated that increased EFT was associated with systemic inflammatory response rather than general risk factors and body fat composition [25]. In our study hs-CRP values were found to be higher than in other studies, but our study population had clinical factors such as diabetes mellitus and acute coronary syndromes, which increase hs-CRP. We may speculate that high hs-CRP values are associated with low-grade systemic inflammation and increased EFT.

Limitations of the study

In the present study, echocardiographic EFT measurements were used for comparison. Although EFT is better visualised on high-speed computed tomography and magnetic resonance imaging, it is not practical to use these methods for its routine assessment. Number of patients with hyperlipidaemia, hypertension and BMI measurements were not shown to be homogenous between diabetic and non-diabetic groups. Diabetes mellitus and acute coronary syndromes can affect hs-CRP values, which can cause heterogeneity of inflammatory status between the groups. The fact that echocardiography is routinely performed in high-risk cardiac patients means that this objective measure could be readily available at no extra cost.

CONCLUSIONS

Epicardial fat thickness may be associated with the extent and complexity of CAD in both diabetic and non-diabetic NSTEMI patients. However, there is a stricter relationship between EFT with the extent and complexity of CAD in diabetic patients.

Conflict of interest: *none declared*

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Wpływ cukrzycy na związek między grubością nasierdziowej tkanki tłuszczowej a rozległością i złożonością choroby wieńcowej u pacjentów z zawałem serca bez uniesienia odcinka ST

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Streszczenie

Wstęp: Grubość nasierdziowej tkanki tłuszczowej (EFT) wiąże się ze zwiększonym ryzykiem chorób sercowych i metabolicznych. Najnowsze badania sugerują, że istnieje silna zależność miedzy cukrzycą a EFT. Chociaż znany jest związek EFT z chorobą wieńcową (CAD), to potencjalny wpływ cukrzycy na zależność między EFT a rozległością i złożonością CAD nie został dokładnie zbadany.

Cel: Celem niniejszej pracy była ocena zależności między EFT a rozległością i złożonością CAD u pacjentów z zawałem serca bez uniesienia odcinka ST (NSTEMI) z cukrzycą i bez cukrzycy.

Metody: Do badania włączono prospektywnie 454 chorych z NSTEMI (średnia wieku: 61,8 ± 10,4 roku). Pacjentów podzielono na dwie grupy w zależności od obecności cukrzycy (grupa chorych na cukrzycę i grupa osób bez cukrzycy). Grubość nasierdziowej tkanki tłuszczowej w rejonie prawej komory określono za pomocą echokardiografii przezklatkowej w pozycji leżącej na lewym boku. Do określenia rozległości i złożoności CAD użyto wskaźnika SYNTAX. U wszystkich uczestników zmierzono stężenie białka C-reaktywnego (hs-CRP) i innych markerów biochemicznych.

Wyniki: U chorych na cukrzycę wartości EFT były wyższe niż u osób bez tego schorzenia ($p < 0,05$). U wszystkich pacjentów EFT była niezależnie związana z obecnością cukrzycy, wskaźnikiem SYNTAX i stężeniem hs-CRP ($p < 0,05$, dla wszystkich porównań). Po podzieleniu uczestników na dwie grupy w zależności od obecności cukrzycy związek między EFT a wskaźnikiem SYNTAX był silniejszy w grupie chorych na cukrzycę niż u osób bez tego schorzenia ($r = 0,635$; $p < 0,001$ *vs.* $r = 0,179$; $p = 0,003$).

Wnioski: Grubość nasierdziowej tkanki tłuszczowej wiąże się ze wskaźnikiem SYNTAX zarówno u chorych na cukrzycę, jak i u osób bez cukrzycy. Związek między EFT a wskaźnikiem SYNTAX jest silniejszy u chorych na cukrzycę.

Słowa kluczowe: grubość nasierdziowej tkanki tłuszczowej, SYNTAX, cukrzyca, zawał serca bez uniesienia odcinka ST, białko C-reaktywne

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