ORIGINAL ARTICLE

Increased epicardial adipose tissue in patients with slow coronary flow phenomenon

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Abstract

Background: Slow coronary flow (SCF) is an angiographic finding characterised by delayed opacification of epicardial coronary arteries without obstructive coronary disease. Epicardial adipose tissue (EAT), localised beneath the visceral pericardium, is a metabolically active endocrine and paracrine organ with possible interactions within the heart. EAT and low-grade inflammation play major roles in the atherosclerotic vascular processes and may be important in other coronary pathologies such as SCF.

Aim: To investigate whether EAT and C-reactive protein (CRP) are increased in patients with isolated SCF compared to normal subjects.

Methods: The present study was cross-sectional and observational, consisting of 66 individuals who underwent coronary angiography with a suspicion of coronary artery disease and who had angiographically normal coronary arteries of varying coronary flow rates. The relationship between EAT, CRP and SCF phenomenon was investigated. Thirty-three patients with isolated SCF (mean age: 56 ± 10 years) and 33 age- and gender-matched control participants with normal coronary flow (NCF), but without SCF, (mean age: 55 ± 10 years) were included in the study.

Results: EAT thickness was significantly increased in the SCF group compared to the NCF group (7.1 ± 2.7 vs. 4.7 ± 1.9 mm, $p < 0.001$). Body mass index (BMI, $p < 0.001$) and the percentage of isolated SCF ($p = 0.002$) were significantly higher in patients with increased EAT thickness. CRP was not related to SCF. When we performed multiple logistic regression analysis, only increased EAT thickness was related to the presence of SCF (OR 1.720, 95% CI 1.175–2.516, $p = 0.005$) independent of BMI and CRP.

Conclusions: This study revealed, for the first time, a significant increase in EAT thickness in patients with SCF compared to NCF. We believe that further studies are needed to clarify the role of adipose tissue in patients with SCF.

Key words: slow coronary flow, epicardial fat pad, epicardial adipose tissue, C-reactive protein, echocardiography, coronary angiography

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INTRODUCTION

Slow coronary flow (SCF) is an angiographic finding characterised by delayed opacification of epicardial coronary arteries without obstructive coronary disease [1]. SCF is a relatively common angiographic finding, with a reported incidence of 1% in patients undergoing coronary angiography for the suspicion of coronary artery disease (CAD) [2]. Since the first description in 1972 by Tambe et al. [1], only a limited number of studies have focused on SCF. Therefore, the precise pathophysiological mechanisms and the clinical importance of SCF are not yet fully understood. Several mechanisms have been proposed for the SCF phenomenon, including small vessel disease, microvascular vasomotor dysfunction, diffuse atherosclerosis, and endothelial dysfunction [3–5]. Occlusive

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disease of the small coronary arteries, which may be a form of early-phase atherosclerosis, has also been suggested as a cause [6].

Visceral adipose tissue, a metabolically active endocrine and paracrine organ, secretes many pro-inflammatory and pro-atherogenic cytokines [7]. Epicardial adipose tissue (EAT), localised beneath the visceral pericardium, is a particular variety of visceral fat depot, which is more closely related to visceral fat than to total body fat.

The physiological, biochemical, and biomolecular properties of EAT and the possible paracrine interactions within the heart have been described in previous studies [8, 9]. EAT exists mainly in the atrioventricular and interventricular groove along the major coronary arteries and branches, to a lesser extent in the atrium, right ventricle and the left ventricular free wall, and shows extension to the apex [10]. The embryological origin of EAT is similar to intra-abdominal visceral adipose tissue [11].

Previous studies have indicated EAT to be a stronger risk factor for CAD than adipose tissues located in other parts of the body, and EAT may play an important role in the development of CAD [9, 12–14].

The aim of this study was to investigate whether epicardial fat pad tissue and inflammation are increased in patients with SCF compared to normal subjects.

METHODS

Patient population and study protocol

The present study was cross-sectional and observational, consisting of 66 individuals who underwent coronary angiography with the suspicion of CAD and had angiographically normal coronary arteries of varying coronary flow rates. The relationship between EAT, C-reactive protein (CRP) and the SCF phenomenon was investigated. The cases were selected from the 1,283 patients who underwent coronary angiography at Rize Education and Research Hospital between January and December 2011. Thirty-three patients with isolated SCF (mean age: 56 ± 10 years) and 33 age- and gender-matched control participants with normal coronary flow (NCF) (mean age: 55 ± 10 years), were included in the study. The control group was selected in a consecutive manner from recently catheterised patients during the study period. The coronary angiograms were evaluated for SCF by two experienced interventional cardiologists who were totally blind to the study. Patients with concomitant coronary artery stenosis were excluded.

All patients had chest pain or angina equivalent symptoms with either positive treadmill test or myocardial perfusion study. Clinical characteristics, which consisted of multiple descriptors from each patient's history and physical examination, were collected by physicians from the cardiology clinic of each patient at the time of cardiac catheterisation and were stored in the database of the coronary angiography laboratory at our institution.

Coronary angiography and determination of SCF

Standard selective coronary angiography with at least four views of the left coronary system and two views of the right coronary artery were performed using the Judkins technique and 6-French right and left heart catheters without the use of nitroglycerin. Coronary angiograms were recorded in right and left oblique planes using cranial and caudal angulations, at a rate of 30 frames/s. During coronary angiography, iopromide (Ultravist 370, Schering AG, Berlin, Germany) was used as the contrast agent in all patients and control participants.

The patients were assessed for the presence of SCF during coronary angiography and coronary flow rates were quantified by the thrombolysis in myocardial infarction (TIMI) frame count method (TFC). For objective quantification of the coronary flow, two independent observers blinded to the clinical data of the study participants assessed the coronary flow in coronary arteries using TFC [15]. This method establishes the number of cine frames, recorded at 30 frames/s, required for the contrast to reach standard distal coronary landmarks in the left anterior descending (LAD), left circumflex (LCx) and right coronary arteries (RCA). Predefined distal landmarks are the distal bifurcation for the LAD, commonly referred to as the 'pitchfork' or 'whale's tail', the distal bifurcation of the segment with the longest total distance for the LCx, and the first branch of the posterolateral artery for the RCA. The standard mean values for normal visualisation of coronary arteries are described as 36.2 ± 2.6 frames for LAD, 22.2 ± ± 4.1 frames for LCx and 20.4 ± 3 frames for RCA. As the LAD is usually longer than the other major coronary arteries, the TFC for this vessel is often higher. Therefore, the TFC for LAD is divided by 1.7 to obtain the corrected TFC. The standard corrected mean value (cTFC) for LAD coronary artery is 21.1 ± 1.5 frames. All participants with a TFC greater than the two standard deviations of the previously published range for the particular vessel were considered to have SCF [15]. The mean TFC for each patient and control participant was calculated by dividing the sum of the TFC of the corrected LAD, LCx and RCA by three.

Routine measurements

Blood samples were drawn by venipuncture to measure routine blood chemistry parameters after fasting for at least eight hours before coronary angiography. Fasting blood glucose, serum creatinine, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and triglyceride levels were recorded. Glucose, creatinine, and lipid profile were determined using standard methods. Serum CRP levels were evaluated using the nephelometric method.

Echocardiography

All patients underwent a complete transthoracic examination including two-dimensional, colour flow and pulsed Doppler, tissue Doppler imaging as well as epicardial fat thickness

Table 1. Baseline characteristics of the study population

Parameters	NCF (n = 33)	SCF (n = 33)	P
Age [years]	55 ± 10	56 ± 10	NS
Gender (male)	46%	46%	NS
Body mass index [kg/m^2]	28 ± 5	32 ± 5	0.002
Hypertension	47%	63%	NS
Diabetes mellitus	19%	25%	NS
Smoking	38%	34%	NS
Hyperlipidaemia	47%	66%	NS
Family history of CAD	26%	31%	NS
Glucose [mg/dL]	106 ± 30	111 ± 51	NS
Creatinine [mg/dL]	0.79 ± 0.14	0.87 ± 0.20	NS
Total cholesterol [mg/dL]	190 ± 34	197 ± 39	NS
LDL [mg/dL]	117 ± 27	126 ± 35	NS
HDL [mg/dL]	44 ± 10	43 ± 12	NS
Triglyceride [mg/dL]	152 ± 108	143 ± 65	NS
C-reactive protein [mg/dL]	0.51 ± 0.38	0.48 ± 0.31	NS
LVEF [%]	65 ± 6	66 ± 9	NS
Epicardial fat pad thickness [mm]	4.7 ± 1.9	7.1 ± 2.7	< 0.001
TIMI frame count measurements:			
LAD	30 ± 7	62 ± 31	< 0.001
LAD (*corrected*)	18 ± 4	36 ± 18	< 0.001
LCx	23 ± 6	31 ± 14	0.005
RCA	22 ± 6	44 ± 25	< 0.001
Mean	21 ± 4	37 ± 13	< 0.001
Medications:			
Acetylsalicylic acid	10 (30%)	15 (46%)	NS
Beta blocker	10 (30%)	7 (21%)	NS
ACEI/ARB	10 (30%)	14 (42%)	NS
Statin	6 (18%)	7 (21%)	NS
Oral anti diabetic	3 (9%)	3 (9%)	NS
Calcium channel blocker	0 (0%)	2 (6%)	NS

NCF — normal coronary flow; SCF — slow coronary flow phenomenon; CAD — coronary artery disease; HDL — high-density lipoprotein; LDL — low-density lipoprotein; LVEF — left ventricular ejection fraction; LAD — left anterior descending artery; LCx — circumflex artery; RCA — right coronary artery; ACEI — angiotensin converting enzyme inhibitor; ARB — angiotensin II receptor blocker

measurement with a GE-Vingmed Vivid S5 (GE-Vingmed Ultrasound AS, Horten, Norway) using a 2.5–3.5 MHz transducer. All examinations were performed by an experienced cardiologist, blind to the patient's clinical information. The intra-observer correlation coefficient was 0.96.

Epicardial fat thickness was evaluated on the free wall of the right ventricle from the parasternal long-axis view, using the aortic annulus as an anatomic reference. Epicardial fat thickness, identified as an echo-free space between the myocardium and visceral pericardium on two-dimensional echocardiography, was measured perpendicularly, ahead of the right ventricular free wall, at the end of diastole, for three cardiac cycles [16]. The measurement was performed at a point on the free wall of the right ventricle along the midline of the ultrasound beam, perpendicular to the aortic annulus. The average value comprising three cardiac cycles of each echocardiographical view was used for the statistical analysis.

Statistical analysis

Continuous variables were given as mean ± SD; categorical variables were defined as percentages. Data was tested for normal distribution using the Kolmogorov-Smirnov test. The χ^2 test was used for the univariate analysis of the categorical variables. All tests of significance were two-tailed. Mean values were compared by ANOVA among different groups. Logistic regression with the Enter method was used for multivariate analysis. Statistical significance was defined as $p < 0.05$. SPSS statistical software (SPSS for Windows, version 15.0, Inc., Chicago, IL, USA) was used for all statistical calculations.

RESULTS

The clinical characteristics of the study population are detailed in Table 1. There were no statistically significant differences between the two groups with respect to age, gender, pre-

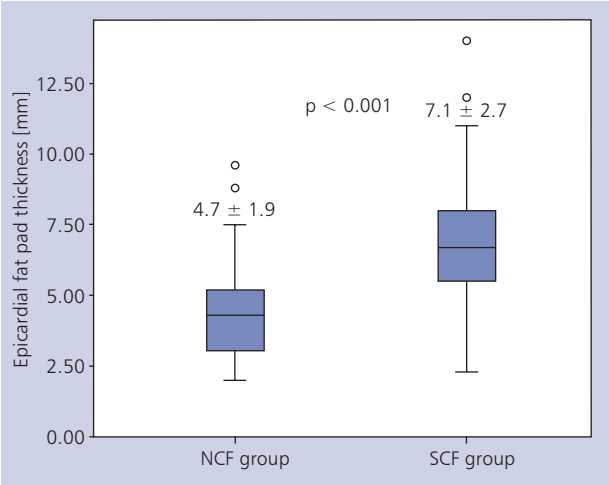

Figure 1. Epicardial fat pad thickness between slow coronary flow (SCF) and normal coronary flow (NCF) groups; values are given as mean ± SD

sence of hypertension or diabetes mellitus, and smoking habit ($p > 0.05$).

EAT thickness was significantly elevated in the SCF group compared to the NCF group (7.1 ± 2.7 vs. 4.7 ± 1.9 mm, $p < 0.001$) (Fig. 1). Body mass index (BMI, $p < 0.001$) and the percentage of isolated SCF ($p = 0.002$) were significantly higher in patients with increased EAT thickness (Table 2). CRP was not related to SCF. EAT correlated with mean TFC value ($r = 0.338$, $p = 0.006$). When we performed multiple logistic regression analysis, only increased EAT thickness was related to the presence of SCF (OR 1.720, 95% CI 1.175–2.516, $p = 0.005$) independent of BMI and CRP (Table 3).

DISCUSSION

In the present study, we revealed significantly higher EAT thickness in patients with SCF compared to patients with angiographically normal coronary arteries. To the best of our knowledge, this is the first report demonstrating the relationship of SCF with increased EAT.

Although the exact pathophysiological mechanism of SCF has not been unequivocally determined, several mechanisms have been suggested as being involved in the development of SCF.

Table 3. Use of logistic regression analysis to predict slow coronary flow phenomenon

Independent variables	P†	Odds ratio (95% CI)
Epicardial fat pad thickness [mm]	0.005	1.720 (1.175–2.516)
C-reactive protein [mg/dL]	0.268	0.327 (0.045–2.367)
Body mass index [kg/m²]	0.134	1.146 (0.959–1.370)
Constant	0.268	0.001
R^2	0.422	

†Logistic regression with the Enter method was used for multivariate analysis; CI — confidence interval

The first hypothesis, that small vessel dysfunction contributes to the pathogenesis of SCF, was proposed by Tambe et al. [1] and confirmed by Mangieri et al. [17], who demonstrated microvascular abnormalities in patients with SCF in a histopathological study. In addition, Kurtoglu et al. [18] reported an improvement in microvascular tone and coronary flow with microvascular vasodilators, suggesting a functional increase in microvascular resistance.

In contrast, intravascular ultrasound studies have identified epicardial CAD as a pathophysiological factor for SCF, as well as microvessel disease [3, 19, 20]. Abnormal slow flow pattern in coronary arteries has been found to be a manifestation of diffuse atherosclerotic disease due to endothelial injury without creating an angiographically visible coronary lesion [20]; therefore, SCF may be an early manifestation of diffuse atherosclerosis involving both the microvascular system and epicardial coronary arteries [3].

These observations suggest that a pathophysiologically relevant interaction exists between the SCF phenomenon and endothelial dysfunction.

Obesity is an important risk factor for atherosclerotic cardiovascular disease. Visceral adipose tissue, the fat deposited around the internal organs, rather than total body adiposity, might act as a marker for cardiovascular disease [21, 22]. EAT is a true visceral fat tissue. Previous reports have indicated a strong correlation between epicardial adipose tissue and abdominal fat deposits. This finding was justified through the common embryogenesis pathway; that is, epicardial fat and intraabdominal fat seem to be originally brown adipose tissue in infancy. This adipose depot is now recognised as

Table 2. Association of epicardial fat tissue thickness with slow coronary flow phenomenon, C-reactive protein and body mass index

	Epicardial fat tissue thickness			
	< 4 mm	4–7 mm	> 7 mm	P
Slow coronary flow phenomenon	18%	53%	77%	0.002
C-reactive protein [mg/dL]	0.38 ± 0.24	0.52 ± 0.39	0.56 ± 0.34	0.317
Body mass index [kg/m²]	26 ± 4	31 ± 4	34 ± 6	< 0.001

a source of variable bioactive molecules, such as adiponectin [23], tumour necrosis factor-alpha, monocyte chemotactic factor-1, interleukin-1 beta, interleukin-6 [24] and inflammatory cytokines, which might affect the coronary artery.

Until recently, magnetic resonance imaging (MRI) had been accepted as the gold standard for measuring epicardial fat thickness. In 2003, Iacobellis et al. [16] reported the echocardiographical measurement of epicardial fat for the first time. They showed an excellent correlation between echocardiographical epicardial fat thickness and MRI abdominal fat and epicardial fat measurements. Echocardiographically measured epicardial fat may provide a highly reliable index of true visceral fat content, avoiding the possible confounding effect of increased subcutaneous abdominal fat [8].

The precise pathophysiological mechanism of the SCF phenomenon remains uncertain. Small vessel abnormality and dysfunction have been implicated in its pathogenesis [1]. Mangieri et al. [17] described histopathological findings from the left ventricular endomyocardial biopsy specimens in a group of ten patients with SCF, without any other cardiac or systemic diseases. Examinations showed evidence of small vessel abnormality such as endothelial thickening due to cell oedema, capillary damage, and reduced luminal diameter of the small vessels. Additionally, inflammation [25], platelet function disorder [26], and imbalance of vasoactive substances [27] have also been implicated in the pathogenesis of the SCF phenomenon. Serum paraoxonase (PON), a high-density lipoprotein bound antioxidant enzyme, prevents atherosclerosis and endothelial dysfunction.

Yildiz et al. [28] reported an independent association between serum PON activity and the mean TIMI TFC; this suggests that reduced serum PON activity may be a biochemical marker of SCF. Enli et al. [29] demonstrated significantly increased serum malondialdehyde and erythrocyte superoxide dismutase and decreased erythrocyte-reduced glutathione levels in patients with SCF compared to patients with normal coronary flow. These findings indicate that free radical damage may play a role in the pathogenesis of SCF.

In our study, an interesting finding is that CRP was not related to SCF. In this aspect, the known relation between CAD and CRP is different and the possible role of CRP on CAD appears to be invalid for SCF. Even though SCF has been related to the inflammatory process, a recent study comparing SCF patients to normal coronary angiograms also found similar CRP levels [30]. Additionally, serum CRP level is not a highly specific inflammatory marker, and therefore may not represent a possible correlation with the low local inflammatory state in EAT.

There is a tendency to believe that SCF is a coronary artery abnormality caused by some systemic factors rather than a simple variation of atherosclerosis. In our study, there may be two explanations for the possible role of increased epicardial tissue in SCF. Firstly, there may be an active local paracrine role or passive thermogenic effect in this process or systemic endocrine effects on vasculature. Alternatively, it may be an innocent by-stander, which increases passively due to a systemic factor inducing both SCF and increased fat pad. Systemic influence thesis, at least for CRP, is not valid in our study, since CRP values were similar between SCF and NCF groups.

We think that our study, by demonstrating a significant correlation between epicardial fat and SCF for the first time, has clarified some gaps in the pathogenesis of SCF and strengthened the notion that EAT may be involved in different aspects of coronary pathologies.

The findings of the present study may support the notion that increased EAT may lead to the SCF phenomenon without visible atherosclerosis. We can speculate that existing vascular wall abnormality may be activated by increased EAT. Therefore, increased fat accumulation may have a critical role in this entity. Bioactive molecules from the pericoronary tissues may alter arterial homeostasis. EAT also releases factors that might profoundly modulate vascular function.

Limitations of the study

Our study has several limitations. First, the study population was relatively small. A larger study population would provide a higher statistical power. The main limitation of our study is the observational nature, which does not explain the exact mechanism of the relationship between increased EAT and SCF. In the current study, the patients did not undergo intravascular ultrasonography (IVUS) to detect atherosclerotic changes in the coronary arteries. Hence, the coexistence of non-obstructive CAD in patients with 'isolated' SCF cannot be established absolutely. Nevertheless, in clinical practice, isolated SCF patients do not undergo IVUS routinely and SCF is usually diagnosed with visual assessment of coronary angiography. MRI is currently the gold standard diagnostic method for measuring epicardial fat thickness. Not using MRI in our research is a study limitation. Although epicardial fat is readily visualised on high-speed computed tomography (CT) and MRI, the widespread use of these methods for the assessment of EAT is impractical. Echocardiography provides an objective, noninvasive, readily available method and is certainly less expensive than MRI or CT for measuring epicardial fat. It may be worth investigating inflammatory cytokines other than CRP to clarify possible causative mediators.

CONCLUSIONS

To the best of our knowledge, this is the first study to demonstrate a significant increase in epicardial adipose tissue in patients with SCF. We believe that further studies are needed to clarify the role of adipose tissue in SCF. Specific roles of adipose tissue could provide new treatment modalities in clinical cardiovascular medicine.

Conflict of interest: *none declared*

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Zwiększona ilość tkanki tłuszczowej nasierdziowej u osób ze zwolnionym przepływem wieńcowym

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Streszczenie

Wstęp: Zwolniony przepływ wieńcowy (SCF) jest nieprawidłowością stwierdzaną w koronarografii, charakteryzującą się opóźnionym zacienieniem tętnic nasierdziowych, bez obecności obstrukcyjnej choroby tętnic wieńcowych. Nasierdziowa tkanka tłuszczowa (EAT), znajdująca się pod blaszką trzewną osierdzia, jest narządem metabolicznym o aktywności endokrynnej i parakrynnej, mogącym wpływać na serce. Zapalenie o niewielkim nasileniu i EAT odgrywają istotną rolę w procesie powstawania miażdżycowych zmian naczyniowych i mogą mieć istotne znaczenie w innych patologiach wieńcowych, np. w SCF.

Cel: Celem badania było ustalenie, czy u pacjentów z izolowanym SCF wartości EAT i białka C-reaktywnego (CRP) są obniżone w porównaniu z wartościami obserwowanymi u osób zdrowych.

Metody: Do badania, mającego przekrojowy i obserwacyjny charakter, włączono 66 pacjentów poddanych koronarografii z powodu podejrzenia choroby wieńcowej, u których stwierdzono angiograficznie prawidłowe tętnice wieńcowe i zróżnicowane prędkości przepływu. Przeanalizowano zależności między wartościami EAT i CRP oraz występowaniem SCF. Do badania włączono 33 osoby z izolowanym SCF (średni wiek: 56 ± 10 lat) i 33 osoby z prawidłowym przepływem wieńcowym (NCF) dopasowane pod względem płci i wieku, które stanowiły grupę kontrolną (średni wiek: 55 ± 10 lat).

Wyniki: Grubość EAT była istotnie zwiększona w grupie z SCF w porównaniu z osobami z NCF (7,1 ± 2,7 *v.* 4,7 ± 1,9 mm; $p < 0,001$). Wskaźnik masy ciała ($p < 0,001$) i odsetek pacjentów z izolowanym SCF ($p = 0,002$) były istotnie wyższe w grupie osób ze zwiększoną grubością EAT. Nie stwierdzono zależności między CRP i SCF. W wieloczynnikowej analizie regresji logistycznej wykazano, że jedynym parametrem związanym z obecnością SCR była zwiększona grubość EAT (OR 1,720; 95% CI 1,175–2,516; $p = 0,005$), niezależnie od wartości CRP i wskaźnika masy ciała.

Wnioski: W niniejszym badaniu po raz pierwszy wykazano, że u pacjentów z SCF grubość EAT jest istotnie zwiększona w porównaniu z osobami z NCF. Należy przeprowadzić dalsze badania w celu wyjaśnienia roli tkanki tłuszczowej u chorych z SCF.

Słowa kluczowe: zwolniony przepływ wieńcowy, tłuszcz nasierdziowy, tkanka tłuszczowa nasierdziowa, białko C-reaktywne, echokardiografia, koronarografia

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