

# Inflammatory activity of pericoronary adipose tissue may affect plaque composition in patients with acute coronary syndrome without persistent ST-segment elevation: preliminary results

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## Abstract

**Background:** The extravascular expression of inflammatory mediators may adversely influence coronary lesion formation and plaque stability through outside-to-inside signalling. It has been shown that the maximal standardised uptake value (SUV) of 18-fluorodeoxyglucose detected by positron emission tomography (PET/CT) is proportional to macrophage density.

**Aim:** To investigate whether the inflammatory activity of pericoronary adipose tissue (PVAT) may influence plaque composition in acute coronary syndrome without persistent ST-segment elevation (NSTEMI-ACS) patients.

**Methods:** In a prospective study, 36 coronary arteries (LM, RCA, LCX, LAD) were investigated in non-diabetic patients with a low or intermediate risk of NSTEMI-ACS (GRACE  $\leq$  140). SUV was measured in fat surrounding coronary arteries on the sections corresponding to proximal and medial segments (Siemens biograph 64-PET/CT system). Additionally, SUV was measured in subcutaneous fat (SC), visceral thoracic fat (VS), and epicardial fat over the right ventricle (EPI). Virtual histology intravascular ultrasound (VH-IVUS) was performed to assess plaque composition (Volcano, USA). PET/CT sections were further examined in segments corresponding to coronary plaques.

**Results:** PVAT SUV in NSTEMI-ACS patients was significantly greater than in other fat locations (LM SUV: 1.60; RCA SUV: 1.54; LCX SUV: 1.94; LAD SUV: 2.37 vs. SC SUV: 0.57; VS SUV: 0.77; EPI SUV: 0.98;  $p < 0.001$ ; ANOVA). PVAT SUV positively correlated with plaque burden ( $r = 0.49$ ,  $p < 0.05$ ) and necrotic core plaque rate ( $r = 0.68$ ,  $p < 0.05$ ), and negatively correlated with fibrous plaque rate ( $r = -0.52$ ,  $p < 0.05$ ).

**Conclusions:** The inflammatory activity of PVAT reflected by SUV is greater than in subcutaneous, visceral thoracic, or epicardial tissue in NSTEMI-ACS patients; PVAT SUV correlates with the plaque burden and necrotic core component of coronary plaque.

**Key words:** pericoronary adipose tissue, inflammation, NSTEMI-ACS, PET/CT, VH-IVUS

Kardiol Pol 2014; 72, 5: 410–416

## INTRODUCTION

It has been demonstrated that inflammation plays a central role in the development and progression of atherosclerosis [1]. There are inflammatory mediators, such as interleukin-6, monocyte chemoattractant protein-1, tumour necrosis factor- $\alpha$ , and vascular cell adhesion molecule 1, expressed in the coronary arteries of diabetic subjects [2]. In a series of

histological analysis, Virmani et al. [3] proved that lesions with a necrotic core rich in macrophages are mostly responsible for acute coronary vessel occlusion.

There have been a growing number of publications dealing with the role of adipose tissue in the pathogenesis of atherosclerosis. There are also differences in the expression of inflammatory mediators between visceral and subcutaneous adipose tissues [4].

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Received: 05.05.2013

Accepted: 29.10.2013

Available as AoP: 07.11.2013

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In patients with advanced coronary artery disease, who were sent for elective coronary by-pass grafting, it has been shown on mRNA (RT-reversed transcriptase) and protein (ELISA) levels that epicardial adipose tissue releases robust amounts of inflammatory markers compared to subcutaneous fat [5].

In histological studies, there have been found inflammatory infiltrations in epicardial fat in the areas near coronary lesions. On the other hand, they were not present in subcutaneous tissue. Infiltrations in epicardial fat mostly consisted of T-lymphocytes, mast cells and macrophages (CD3+, tryptase, CD68+, accordingly).

In 2008, Gorter et al. [6] presented potential mechanisms of the 'outside-to-inside' contribution in the development and destabilisation of atherosclerotic plaque. Erdogan et al. [7] postulated that coronary slow flow phenomenon may be related to the amount of epicardial adipose tissue measured in transthoracic echocardiography. On the other hand, a large analysis of the Framingham population of 5,200 patients confirmed that the amount of epicardial adipose tissue correlates with visceral thoracic, abdominal adipose tissue, left ventricular mass and the size of the left atrium [8]. Perivascular adipose tissue (PVAT) was previously evaluated in patients with myocardial infarction. It was recorded that its thickness was doubled compared to healthy volunteers. In addition, PVAT thickness correlated to patients' age, body mass index (BMI), serum glucose and triglycerides and maximal troponin I concentration. PVAT thickness surrounding left anterior descending artery (LAD) positively correlated to vessel stenosis (quantitative coronary analysis) [9]. It is postulated that the quality rather than the quantity of perivascular tissue is a key factor for atherosclerosis [10]. It has been previously stated that for the clinical importance of PVAT, its proinflammatory potential should be evaluated [11].

*In vivo*, this is made possible by introducing a maximal 18-fluorodeoxyglucose (FDG) uptake measurement in positron emission tomography (PET). Because of its low resolution for clinical application, PET is combined with simultaneous 64-row computed tomography (PET/CT). Its adoption has allowed the identification of unstable plaques in animal models [12].

Davies et al. [13] summarised the use of PET/CT in localising atherosclerotic plaques in humans. Such a non-invasive technique is a promising tool in the evaluation and quantification of unstable lesions in peripheral vessels: carotid arteries and aorta arteries [13, 14]. Ogawa et al. [15] proposed using the FDG uptake measured by PET/CT for monitoring the therapeutic effects of probucol — an anti-inflammatory drug in aortic plaque stabilisation. Tawakol et al. [16] established a link between FDG-PET accumulation and carotid arteries macrophage infiltrations.

In this study, we sought to investigate the proinflammatory potential of PVAT and its influence on coronary arteries in patients admitted with acute coronary syndrome using PET/CT technique.

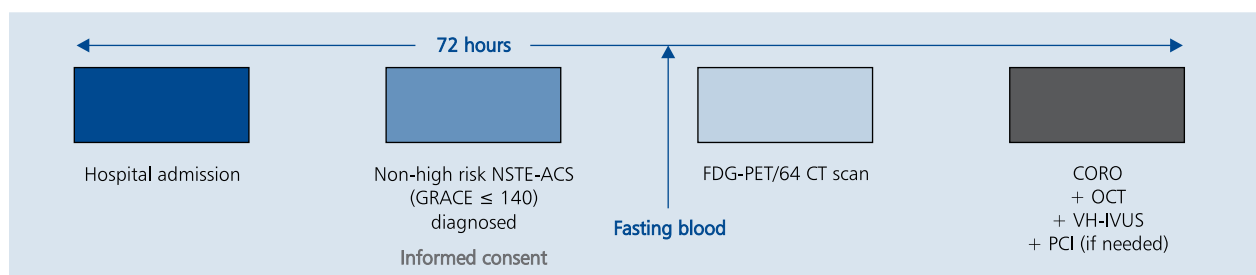
## METHODS

### *Study design, inclusion and exclusion criteria*

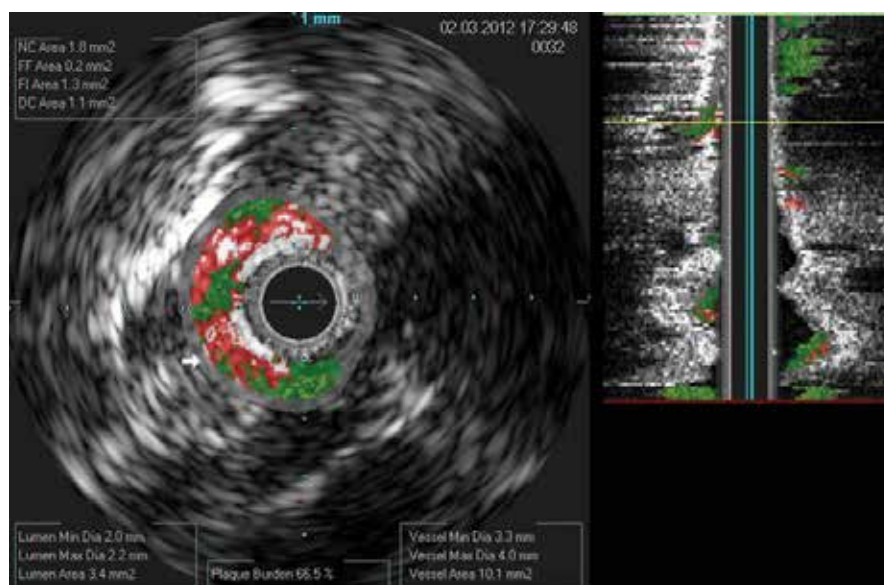
Consecutive patients admitted to our institution with acute coronary syndrome without persistent ST-segment elevation (NSTEMI-ACS) were selected. Patients with persistent chest pain, recurrent symptoms at rest, hypotension, tachycardia, and heart failure upon presentation were initially excluded. Patients were further evaluated at our coronary care unit with repeated 12-lead ECG and blood markers of myocardial necrosis (troponin I and CK-MB mass). Patients with prior myocardial infarction, coronary artery intervention (PCI), bypass grafting, renal insufficiency (eGFR < 60 mL/min/1.73 m<sup>2</sup>), history of diabetes mellitus, confirmed reduced left ventricular function (ejection fraction < 40%), permanent anticoagulation therapy, or any known inflammatory conditions were excluded from the study.

An individual risk of mortality was assessed by calculating an ischaemic risk (GRACE score). Patients with an intermediate risk of NSTEMI-ACS (GRACE 109–140) were consented. We also included patients with a low mortality risk (GRACE ≤ 108), in whom invasive strategy was indicated due to a relevant rise in troponin or dynamic ST- or T-wave changes [17, 18].

On admission, aspirin was given to all patients at an initial loading dose of 150–300 mg, and at a maintenance dose of 75–100 mg daily, as well as a 600-mg loading dose of clopidogrel (Fig. 1).



**Figure 1.** Study design; NSTEMI-ACS — acute coronary syndrome without persistent ST-segment elevation; FDG-PET/64 CT — positron emission tomography with simultaneous 64-row computed tomography; CORO — coronary angiography; OCT — optical coherence tomography; PCI — percutaneous coronary intervention; VH-IVUS — virtual histology intravascular ultrasound



**Figure 2.** Representative virtual histology intravascular ultrasound image from a medial segment of left anterior descending artery; NC — necrotic core; FF — fibro-fatty; FI — fibrous tissue; DC — dense calcium

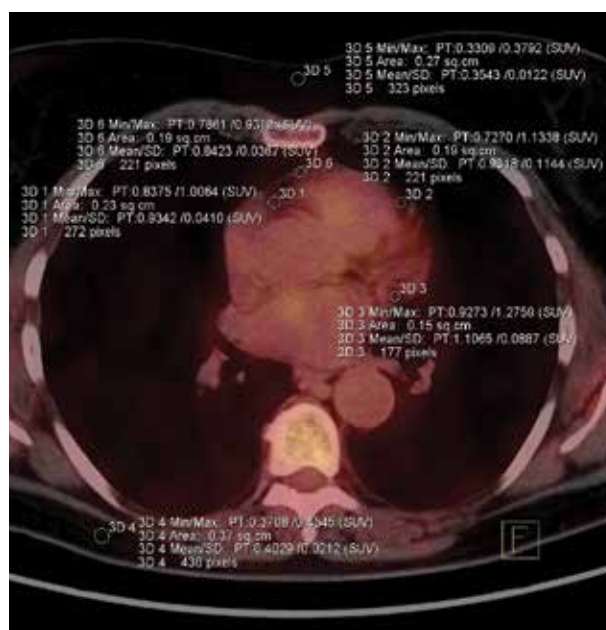
### *PET, IVUS, and VH-IVUS*

Within 24 h of admission, all included patients underwent FDG-PET/CT (Siemens biograph 64-PET/CT system). Within 72 h of presentation, all patients were sent to the catheterisation laboratory for invasive examination and treatment.

For the preliminary analysis, 36 coronary arteries, i.e. left main (LM), LAD, left circumflex (LCX), and right coronary artery (RCA) were investigated in nine patients.

In all examined vessels, grey scale intravascular ultrasound (IVUS) and virtual histology IVUS (VH-IVUS) were performed during routine coronarography. A 20 MHz, 3.2-French IVUS imaging catheter (Eagle Eye, In-Vision Gold/Platinum, Volcano Corp., USA) was advanced to the distal vessel area. Automated pullback was used at a speed of 0.5 mm/s. VH-IVUS quantitative analysis was performed across the entire examined artery.

Plaque composition was classified into four categories by Volcano software: calcified, fibrous, fibro-fatty, or necrotic core (Fig. 2). Due to a relatively low number of observations, for this preliminary analysis we decided to evaluate atheroma morphology recorded by VH-IVUS from proximal and medial segments of the vessel. Next, we correlated VH-IVUS recordings in the area reach of atherosclerotic plaque and maximal lumen narrowing with PET/CT sections perpendicular to the vessel, in segments corresponding to localised lesion, confirmed by simultaneous 64-row CT (maximal lumen narrowing lesion). Grey scale measurements, as well as remodelling index calculations based on the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies [19] will be performed after collecting complete data from the entire cohort.



**Figure 3.** Representative FDG-PET scan showing samples collection from subcutaneous fat, visceral thoracic fat, epicardial fat over right ventricle and perivascular adipose tissue

FDG uptake (standardised uptake value — SUV) was measured in fat surrounding the coronary arteries on the sections corresponding to coronary plaques in proximal and medial segments of LM, LAD, LCX, and RCA.

Additionally, SUV was measured in subcutaneous fat (SC), visceral thoracic fat (VS) and epicardial fat over the right ventricle (EPI) (Fig. 3).

Mean of three measurements taken by two researchers blinded to results with inter- and intra-observer variability < 5%.

Total PVAT SUV was calculated as the sum of all four locations (LM, LAD, LCX, RCA) of local PVAT SUV.

**Statistical analysis**

Statistica v. 10 software was used for statistical analysis. Kolmogorov-Smirnov test was used to check normal distribution of continuous variables, which were further presented as mean ± standard error (SE) or median depending on distribution. Comparisons between groups of variables was conducted with analysis of variance (ANOVA). Relationships between local (LM, LAD, LCX, RCA), total PVAT SUV, demographic, clinical variables and plaque composition percentages: necrotic, fibrous and plaque burden were determined by Spearman’s rank-correlation test and was expressed as Spearman’s rank-correlation coefficient. P-values lower than 0.05 were considered to be statistically significant.

**RESULTS**

SUV in fat surrounding all coronary arteries (PVAT SUV) was significantly greater than SUV in other fat locations: LM SUV: 1.60 ± 0.05; RCA SUV: 1.54 ± 0.10; LCX SUV: 1.94 ± 0.14; LAD SUV: 2.37 ± 0.15 vs. SC SUV: 0.57 ± 0.03; VS SUV: 0.77 ± 0.03; EPI SUV: 0.98 ± 0.04, p < 0.001 (ANOVA) (Table 1).

Total PVAT SUV was not related to gender, age, BMI, or serum glucose.

On the other hand, PVAT SUV of the given coronary territory was related to plaque composition. PVAT in all four coronary locations corresponded to necrotic core rate and plaque burden (Table 2). Total PVAT SUV positively correlated to necrotic core rate (Fig. 4), as well as plaque burden percentage (Fig. 5). It also negatively correlated to fibrous plaque rate (Fig. 6).

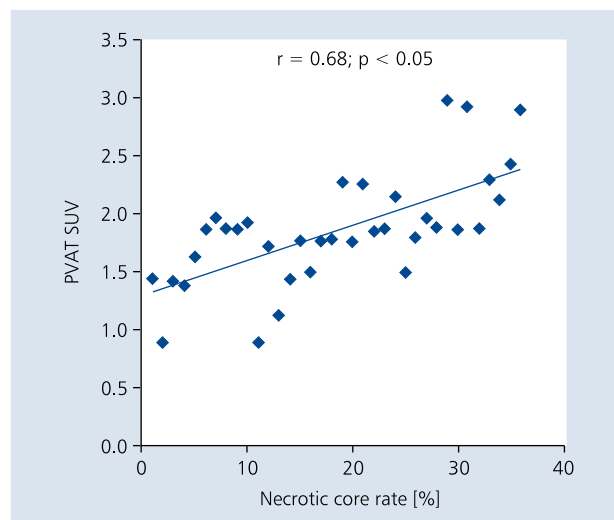
**DISCUSSION**

This preliminary study in a group of patients presenting with NSTE-ACS showed that pericoronary SUV was significantly greater than glucose uptake in subcutaneous, intrathoracic

**Table 1.** Clinical characteristics

Age (mean ± SE) [years]	68.8 ± 2.6
Male	66.7%
BMI (mean ± SE) [kg/m <sup>2</sup> ]	25.2 ± 1.2
Risk factors:	
Prior MI	0%
Hypertension	77.8%
Diabetes	0%
Dyslipidaemia	55.6%
Smoking	77.8%
Family history	44.5%
Max Tnl [ng/mL]	2.7 ± 0.6
Max CK-MB mass [ng/mL]	6.8 ± 0.9
Serum glucose [mg/dL]	125.5 ± 6.6

BMI — body mass index; MI — myocardial infarction; Tnl — troponin I; CK-MB mass — creatine kinase-MB mass assay

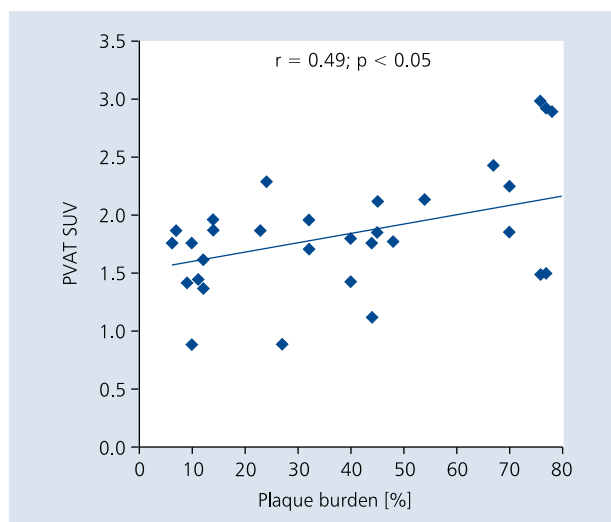


**Figure 4.** Relationship between total pericoronary adipose tissue standardised uptake value (PVAT SUV) and necrotic core rate (Spearman’s rank-correlation)

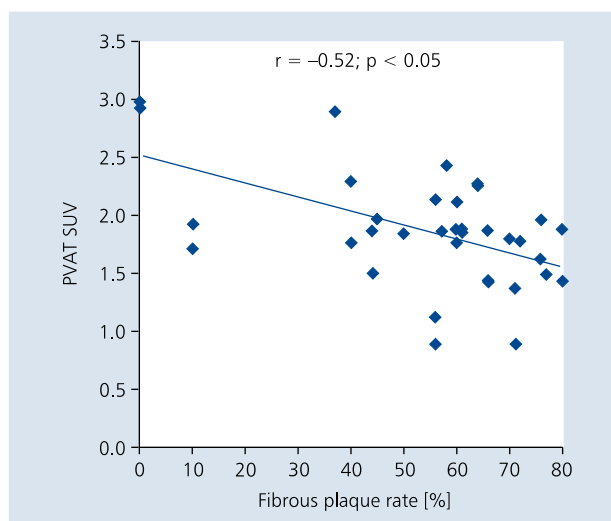
**Table 2.** Relationships between local (LM, LAD, LCX, RCA) SUV in fat and plaque composition percentages: necrotic, fibrous and plaque burden (Spearman’s rank-correlation)

PVAT SUV	Necrotic core	Fibrous plaque	Plaque burden
LM	0.69%; p < 0.05	NS	0.40%; p < 0.05
LAD	0.67%; p < 0.05	-0.87%; p < 0.05	0.32%; p < 0.05
LCX	0.77%; p < 0.05	-0.25%; p < 0.05	0.39%; p < 0.05
RCA	0.53%; p < 0.05	-0.40%; p < 0.05	0.26%; p < 0.05

LM — left main coronary artery; LAD — left anterior descending coronary artery; LCX — left circumflex coronary artery; RCA — right coronary artery; PVAT — pericoronary adipose tissue; SUV — standardised uptake value



**Figure 5.** Relationship between total pericoronary adipose tissue standardised uptake value (PVAT SUV) and plaque burden (Spearman's rank-correlation)



**Figure 6.** Relationship between total pericoronary adipose tissue standardised uptake value (PVAT SUV) and fibrous plaque rate (Spearman's rank-correlation)

(non-epicardial) and epicardial fat locations. This may represent the augmented proinflammatory activity of PVAT compared to other adipose tissues. The proinflammatory activity of epicardial adipose tissue has previously been reported in a number of studies. Inflammatory cytokines were isolated from epicardial tissue culture of patients with multivascular coronary artery disease [4, 5]. Inflammatory cells, mainly macrophages and T lymphocytes, were localised in perivascular tissue, as well as in other fat locations of severely obese patients [5, 20]. To the best of our knowledge, to date there

have been no publications specifically oriented on pericoronary adipose tissue inflammatory activity.

In our clinical model, the proinflammatory activity of PVAT corresponded with plaque composition examined by VH-IVUS. We hypothesise that a higher level of local inflammatory burden in perivascular adipose tissue may lead to necrotic core formation and result in plaque destabilisation. On the other hand, a high percentage of fibrotic tissue is a result of low inflammatory activity.

The elucidation of pathophysiological mechanisms leading to coronary plaque formation and destabilisation may bring new potential targets for therapeutic interventions.

### Limitations of the study

These results are an initial analysis of a preliminary study. The authors are going to follow the presented model in a group of 50 patients with NSTEMI-ACS to perform univariate and multivariate analysis. For the same reasons, at this stage the authors decided not to analyse grey scale IVUS measurements or remodelling index calculations. Due to technical difficulties in the co-localisation of coronary plaque areas with corresponding CT-PET sections, some selection bias cannot be entirely excluded. In addition, regardless of the multiple repeats with an excellent inter- and intra-observer variability, it is not possible to entirely rule out an impact of PET SUV of the left ventricle on the PVAT SUV measurements. Finally, patients with NSTEMI-ACS constitute a very heterogeneous group, with a highly variable extent of coronary plaque formation and differing prognoses. This may have influenced a relatively broad range of VH-IVUS findings.

### CONCLUSIONS

In NSTEMI-ACS patients, pericoronary SUV is greater than in subcutaneous, visceral (intrathoracic), epicardial adipose tissue, and is independent of gender, age, BMI, or serum glucose. The inflammatory activity of PVAT reflected by SUV correlates with the necrotic core component of coronary plaque and plaque burden in patients with NSTEMI-ACS. The proinflammatory activity of PVAT in patients with NSTEMI-ACS may contribute to plaque formation, vessel narrowing and plaque rupture, which supports the 'outside-to-inside' hypothesis.

### Acknowledgements

This study was supported by an unrestricted grant from the Polish Ministry of Science and Higher Education (No. N402 383538).

**Conflict of interest:** none declared

### References

1. Libby P. Inflammation in atherosclerosis. *Nature* 2002; 420: 868–874.
2. Zhang L, Zalewski A, Liu Y et al. Diabetes-Induced Oxidative Stress and Low-Grade Inflammation in Porcine Coronary Arteries. *Circulation*, 2003; 108: 472–478.

3. Virmani R, Burke AP, Farb A, Kolodgie FD. Pathology of the vulnerable plaque. *J Am Coll Cardiol*, 2006; 47: C13–C18.
4. Fried SK, Bunkin DA, Greenberg AS. Omental and subcutaneous adipose tissues of obese subjects release interleukin-6: depot difference and regulation by glucocorticoid. *J Clin Endocrinol Metab*, 1998; 83: 847–850.
5. Mazurek T, Zhang L, Zalewski A et al. Human epicardial adipose tissue is a source of inflammatory mediators. *Circulation*, 2003; 108: 2460–2466.
6. Gorter PM, van Lindert AS, de Vos AM et al. Quantification of epicardial and peri-coronary fat using cardiac computed tomography; reproducibility and relation with obesity and metabolic syndrome in patients suspected of coronary artery disease. *Atherosclerosis*, 2008; 197: 896–903.
7. Erdogan T, Canga A, Kocaman SA et al. Increased epicardial adipose tissue in patients with slow coronary flow phenomenon. *Kardiol Pol*, 2012; 70: 903–909.
8. Fox CS, Gona P, Hoffmann U et al. Pericardial fat, intrathoracic fat, and measures of left ventricular structure and function. The Framingham Heart Study. *Circulation*, 2009; 119: 1586–1591.
9. Mazurek T, Nijveldt R, Yudkin JS et al. Peri-coronary epicardial adipose tissue affects coronary atherosclerosis in patients with acute myocardial infarction. *Circulation*, 2008; 118: S580–S581.
10. Yudkin JS, Eringa E, Stehouwer CDA. „Vasocrine” signaling from perivascular fat: a mechanism linking insulin resistance to vascular disease. *Lancet*, 2005; 365: 1817–1820.
11. Mazurek T. Proinflammatory capacity of adipose tissue — a new insights in the pathophysiology of atherosclerosis. *Kardiol Pol*, 2009; 67: 1119–1124.
12. Aziz K, Berger K, Claycombe K, et al. Noninvasive detection and localization of vulnerable plaque and arterial thrombosis with computed tomography angiography/positron emission tomography. *Circulation*, 2008; 117: 2061–2070.
13. Davies JR, Rudd JH, Weissberg PL, Narula J. Radionuclide imaging for the detection of inflammation in vulnerable plaques. *J Am Coll Cardiol*, 2006; 47: C57–C68.
14. Tahara N, Kai H, Nakaura H et al. The prevalence of inflammation in carotid atherosclerosis: analysis with fluorodeoxyglucose-positron emission tomography. *Eur Heart J*, 2007; 28: 2243–2248.
15. Ogawa M, Magata Y, Kato T et al. Application of 18F-FDG PET for monitoring the therapeutic effect of antiinflammatory drugs on stabilization of vulnerable atherosclerotic plaques. *J Nucl Med*, 2006; 47: 1845–1850.
16. Tawakol A, Migrino RQ, Hoffmann U et al. Noninvasive in vivo measurement of vascular inflammation with F-18 fluorodeoxyglucose positron emission tomography. *J Nucl Cardiol*, 2005; 12: 294–301.
17. Hamm CW, Agewall S, Bax J et al. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J*, 2011; 32: 2999–3054.
18. Montalescot G, Van de Werf F, Gulba DC et al. Stenting and glycoprotein IIb/IIIa inhibition in patients with acute myocardial infarction undergoing percutaneous coronary intervention: findings from the global registry of acute coronary events (GRACE). *Catheter Cardiovasc Interv*, 2003; 60: 360–367.
19. Mintz GS, Nissen SE, Anderson WD et al. American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies (IVUS). A report of the American College of Cardiology Task Force on Clinical Expert Consensus Documents. *J Am Coll Cardiol*, 2001; 37: 1478–1492.
20. Bigornia SJ, Farb MG, Mott MM et al. Relation of depot-specific adipose inflammation to insulin resistance in human obesity. *Nutr Diab*, 2012; 2: 1–6.

# Aktywność zapalna okołonaczyniowej tkanki tłuszczowej może wpływać na skład blaszki miażdżycowej u pacjentów z ostrym zespołem wieńcowym bez przetrwałego uniesienia odcinka ST: wyniki wstępne

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## Streszczenie

**Wstęp:** Pozanaczyniowa ekspresja mediatorów zapalnych może niekorzystnie wpływać na powstawanie i stabilność blaszki miażdżycowej w mechanizmie oddziaływania z zewnątrz do wewnątrz. Wykazano także, że maksymalna wartość znormalizowana wychwytu (SUV) 18-fluorodeoksyglukozy (FDG) mierzona za pomocą pozytonowej tomografii emisyjnej (PET/CT) jest proporcjonalna do gęstości makrofagów.

**Cel:** Celem niniejszej pracy było zbadanie, czy aktywność zapalna okołowieńcowej tkanki tłuszczowej (PVAT) może wpływać na skład blaszek miażdżycowych u chorych z ostrym zespołem wieńcowym bez przetrwałego uniesienia odcinka ST (NSTEMI-ACS).

**Metody:** W badaniu prospektywnym poddano analizie 36 tętnic wieńcowych (LM, RCA, LCX, LAD) u pacjentów z NSTEMI-ACS, bez wywiadu cukrzycy, z niskim lub umiarkowanym ryzykiem zgonu (GRACE  $\leq$  140). SUV mierzono w tkance tłuszczowej otaczającej tętnicę wieńcową w przekrojach odpowiadających proksymalnym i środkowym odcinkom tętnic (Siemens biograph 64-PET/CT system). Ponadto mierzono SUV w podskórnej (SC), trzewnej-wewnątrzpiersiowej (VS) i nasierdziowej tkance tłuszczowej (EPI). Morfologię zmian oceniano za pomocą ultrasonografii wewnątrzwieńcowej (IVUS) z wirtualną histologią (VH-IVUS) (Volcano, USA). Przekroje PET/CT były następnie analizowane w segmentach, w których zlokalizowano blaszki miażdżycowe.

**Wyniki:** PVAT SUV u pacjentów z NSTEMI-ACS była istotnie wyższa niż w innych lokalizacjach tkanki tłuszczowej (LM SUV: 1,60; RCA SUV: 1,54; LCX SUV: 1,94; LAD SUV: 2,37 vs. SC SUV: 0,57; VS SUV: 0,77; EPI SUV: 0,98;  $p < 0,001$ ; ANOVA). PVAT SUV korelowała pozytywnie z wielkością blaszki miażdżycowej ( $r = 0,49$ ;  $p < 0,05$ ), jądra martwicze ( $r = 0,68$ ;  $p < 0,05$ ), a negatywnie — ze stopniem zwłóknienia blaszki ( $r = -0,52$ ;  $p < 0,05$ ).

**Wnioski:** U pacjentów z NSTEMI-ACS aktywność zapalna okołowieńcowej tkanki tłuszczowej oceniana za pomocą pomiaru wychwytu 18-FDG jest większa niż w tkance tłuszczowej podskórnej, trzewnej-wewnątrzpiersiowej i nasierdziowej; PVAT SUV koreluje z wielkością blaszki i wielkością jądra miażdżystego blaszki miażdżycowej.

**Słowa kluczowe:** okołowieńcowa tkanka tłuszczowa, zapalenie, NSTEMI-ACS, PET/CT, VH-IVUS

Kardiologia 2014; 72, 5: 410–416

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Praca wpłynęła: 05.05.2013 r.

Zaakceptowana do druku: 29.10.2013 r.

Data publikacji AoP: 07.11.2013 r.