Novel atherogenesis markers for identification of patients with a multivessel coronary artery disease

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Abstract

Background: Patients with advanced coronary artery disease (CAD) have an unfovourable prognosis. Therefore, early identification of this high-risk group is important.

Aim: To asses the utility of clinical, electrographic and echocardiographic parameters, supported by novel atherogenesis markers, to identify patients with triple vessel coronary artery disease (CAD).

Methods: The study group comprised 37 patients (29 males, mean age 64±8 years) suffering from multivessel CAD and a control group of 16 patients (8 males, mean age 60±10 years), in whom – despite typical stenocardial symptoms, positive exercise stress test and segmental contractility disturbances – coronary angiography did not reveal any haemodynamically significant CAD. Apart from coronary angiography, each patient had additionally an entire test panel performed assessing both the disease severity and the presence of other systemic dysfunction. Mean Gensini score in the study group was 91.9±43.8, including proximal Gensini score 52.6±45.6 and distal one 39.4±29.7.

Results: Patients with triple vessel disease had a long history of angina (mean 84 months), of whom 30 (81%) experienced at least Q-wave myocardial infarction (MI). ECG changes typical for ischaemia were observed more often than in the control group. Also in patients with triple vessel disease echocardiography showed more escalated segmental contractility disorders, and left ventricular ejection fraction in this group was significantly lower than in the control group (44 vs. 55%, p <0.001). There were significant differences between CAD patients and control groups with respect to serum levels of: adiponectin (10.5±4.2 vs. 17.6±3 µg/ml, p=0.001), resistin (13.7±6.1 vs. 7.2±2.4 ng/ml, p=0.007), TNF-α (4.2±2.9 vs. 2.1±1.1 pg/ml, p=0.02) and IL-8 (18.4±4.1 vs. 12.2±4.1 pg/ml, p=0.008). Significant differences were also noted in lipid profile (total cholesterol: 201±47.1 vs. 183±18 mg/dl, NS; HDL cholesterol: 45±8.5 vs. 54±11 mg/dl, p=0.005; LDL cholesterol: 126.1±46.9 vs. 102±29 mg/dl, p=0.004), NT-proBNP [516 (174-1426) vs. 187 (39-573) pg/ml, p=0.02] and fasting blood glucose levels (97±14 vs. 94±11 mg/dl, p=0.03). Significantly lower serum adiponectin levels were observed in men and tobacco smokers.

Conclusions: Medical history, supported by interpretation of selected, routine imaging studies and novel biochemical markers, such as adiponectin, resistin, TNF-α, IL-8 or NT-proBNP, seem to be the key factors when assessing the risk of presence of advanced coronary artery atherosclerosis.

Key words: multivessel coronary artery disease, adiponectin

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Introduction

Cardiovascular diseases, including coronary artery disease (CAD) as one of the most widespread, are the main causes of death in developed countries [1]. Over the last several dozen years great progress in cardiology has been made. Nevertheless, global cardiovascular mortality is still very high and in 2002 reached more than 7 mln [1, 2]. The development of interventional cardiology has led to much better access to invasive diagnostics of ischaemic heart disease over the last few years. As a result, far beyond expectations, there was a widespread diagnosis of advanced stage of atherosclerosis with simultaneous worsening of flow in all major coronary arteries, regarded as a separate nosological unit – multivessel coronary artery disease (MCAD) [3]. The definition of MCAD was formed based on angiographic findings as subcritical or critical

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(crosssection area decreased by >75%) stenosis of at least two of three main major coronary arteries supplying the myocardium [1, 4]. According to various sources, it has been estimated that it even makes up to 50% of all cases of CAD [1, 2]. The severity of atherosclerosis and numerous complications with simultaneous lack of clear classification systems and diagnostic criteria bring about an exceptionally high mortality rate in this population, which in 5-year follow-up ranges from 10 to even 60% depending on advancement of atherosclerotic lesions as well as other risk factors [1-3, 5].

Considering the unfavourable clinical course and poor prognosis in this group of patients based on interpretation of some diagnostic tests, including physical examination, it would be very useful for every clinician to be able to initially qualify patients into the group of high risk of severe CAD. The crucial aim of current cardiological research today is finding markers which with high specificity would allow for risk stratification of CAD depending on atherosclerosis severity hindering flow in epicardial arteries.

The aim of this study was to determine clinical characteristics extended by determination of new atherogenesis markers in patients with the most severe MCAD disqualified from intervention due to severity of coronary atherosclerosis.

Methods

Study group

The study involved a group of 37 patients hospitalised at our site in 2007 and meeting the following criteria:

1. Coronary artery disease with ≥75% stenosis in three main coronary arteries supplying the myocardium confirmed on coronary angiography. The diameter of stenosis of the left main coronary artery could not exceed 50%.
2. Disqualification from surgical and percutaneous treatment of CAD for technical or clinical reasons or a lack of consent for such management.
3. No congenital heart defects.
4. Absence of significant diseases of other organs.

A control group consisted of 16 patients with a history of typical, exercise-induced stenocardial disorders, clinically and electrographically positive exercise stress test, segmental contractility disturbances on echocardiography, with CAD confirmed on angiography but with non-significant coronary artery lumen narrowing (<50%). The presence of any other diseases was an exclusion criterion in this patient group.

Following discharge from the Department, all patients remained under the care of the Cardiology Outpatient Clinic and were treated according to the current guidelines of the Polish Cardiac Society. Each patient enrolled in the study was subject to consultation by an experienced cardiologist and cardiac surgeon during which the decision concerning disqualification from surgical treatment of CAD was made. Patients included in the study signed an informed consent form to participate; the study was also approved by the Bioethics Committee at the Medical University in Lodz.

Biochemical tests

All patients had additional tests performed in order to evaluate disease severity and possible dysfunctions of other organs. The following were evaluated: complete blood count, complete lipid profile, fasting blood glucose (and oral glucose tolerance test with blood glucose measurement after 2 hours in patients without previous carbohydrate metabolism disorders), CKMB, CK, urea, creatinine, hepatic transferases, and NT-proBNP levels. Apart from standard biochemical parameters, we examined new markers such as adiponectin, resistin, TNF-α, and IL-8, which are useful in an assessment of pathophysiological process severity promoting atherogenesis.

ECG, echocardiography, and exercise stress test

Transthoracic echocardiography, ECG at rest and electrocardiographic exercise test were performed in all patients. The Athens QRS score was used for analysis of exercise test based on changes in QRS wave amplitudes in the leads aVF and V5 in resting and post-exercise ECG.

Coronary angiography

Based on the coronary angiographic results a semi-quantitative analysis was made of severity of atherosclerotic changes and expressed as Gensini score [involving vessel division into proximal segments, referring to LM, p-LAD, p-LCx, p-RCA (proximal Gensini score) and distal ones, for the remaining parts of the standard artery division (distal Gensini score)] – Figure 1.

Statistical analysis

Kolmogorov-Smirnov test was used for determining normality of distribution of analysed variables. Constant variables showing normal distribution are presented as means ± standard deviations, whereas constant variables of distribution different from normal and ordinal variables are expressed as medians (25^{th}-75^{th} percentile). Variance analysis and the Wilcoxon non-parametric test were applied to compare the differences between presence of particular features in patient groups. The relationship between constant variables of normal distribution was analysed using linear Pearson's correlation analysis. If at least one variable showed other than normal distribution or was of ordinal type, Spearman rank correlation analysis was applied. An assessment of the relationship between adiponectin, resistin, IL-8 and TNF-α and categorical variables was based on evaluating mean levels of adiponectin, resistin, IL-8 and TNF-alpha in subgroups

GENSINI SCORE = sum of (stenosis severity index × lesion location index)

Lesions location – point value:

LM:	5	d-LCx:	1
p-LAD:	2.5	OM:	1
m-LAD:	1.5	p-RCA:	1
d-LAD:	1	m-RCA:	1
1st Dx:	1	d-RCA:	1
2nd Dx:	0.5	PD:	1
p-LCx:	2.5	PL:	1

Stenosis severity – point value:

25%:	1	90%:	8
50%:	2	99%:	16
75%:	4	100%:	32

Figure 1. Principle of computing Gensini score

LM – left main coronary artery, LAD – left anterior descending branch, Dx – diagonal branch, LCx – circumflex branch, OM – obtuse marginal branch, RCA – right coronary artery, PD – posterior descending branch, PL – postero-lateral branch, p – proximal, m – middle, d – distal

chosen by different realisations of categorical variables. In this case, Mann-Whitney test was used depending on normality of data distribution in subgroups. The results were considered statistically significant if p value was <0.05.

Results

Patients with MCAD had a long history of angina (mean 84 months) and the majority of them had previous MI. The distribution of CAD risk factors in the study group revealed no significant differences in comparison with the control group. In the study group the detection of obliterative atherosclerosis of lower limbs was more common. Detailed data are shown in Table I.

All patients with MCAD had ischaemic changes on ECG. The most frequent pathological Q waves, negative T waves and ST depression were observed. The only difference between analysed groups was the frequency of electrical potential loss on ECG. In patients with MCAD echocardiography showed much more aggravated segmental contractility disorders, and left ventricular ejection fraction was significantly lower than in controls (Table II).

Exercise test was more often positive in the MCAD patients (p=0.03); and the work was lower (5.7 vs. 7.6 METS, p=0.04). The Athens QRS score was significantly lower in comparison with the control group (p=0.01) (Table III).

Mean Gensini score in MCAD patients was 91.9±43.8, including proximal Gensini score of 52.6±45.6 and distal one of 39.4±29.7.

There were significant differences in adiponectin, resistin, TNF-α and IL-8 serum levels between the groups. Significant differences were also observed for lipid profile, NT-proBNP and fasting blood glucose. Detailed data are presented in Table IV.

In addition, a negative correlation between the adiponectin and both triglyceride (r=–0.48; p=0.004) and serum NT-proBNP levels (r=–0.38; p=0.024) was documented in the MCAD patients. There was a positive correlation between IL-8 and C-reactive protein (r=0.36; p=0.033) as well as TNF-α and NT-proBNP (r=0.35; p=0.035) levels. Serum adiponectin concentration was significantly lower both in males and tobacco smokers (p=0.006 and p=0.002, respectively). Adiponectin levels remained significantly different in both groups after matching to gender proportions, but only in the male part of the group (8.2 vs. 10.4 μg/ml, p=0.02).

Discussion

Initial group selection of patients with high risk of severe MCAD, made on the basis of interpretation of some biochemical tests and imaging exams, would allow for much earlier qualification for invasive procedures, thus limiting the frequency of numerous cardiovascular complications in this patient group. Medical history together with interpretation of some selected routine imaging studies appears to be the key aspect of management in this case. In light of the above, novel biochemical markers, such as adiponectin, resistin, TNF-α and IL-8, have brought promising results.

Studies conducted so far have focused mainly on characteristics, prognosis, and comparison of various therapeutic strategies in patients with CAD, and not on distinguishing its specific forms. A great deal of general rules along with a constantly increasing number of prognostic factors were identified and new management standards were compiled as well. The necessity of a different approach to patients with diabetes and renal failure as well as the requirement for distinction of CAD variants depending on severity of CAD made the previous diagnostic and therapeutic management insufficient in many cases [6-11].

Understanding of mechanisms underlying the atherosclerotic process, for a pathologist being a form of inflammatory response to factors damaging the vessel wall,

Table I. Selected demographic data of patients with multivessel CAD and control group

	MCAD N=37	Control group N=16	p
Males (%)	20 (79)	8 (50)	0.036
Age [years]	64±9	60±10	NS
Duration of angina [months, mean ± SD]	82.5±83.7	6±5	<0.001
History of myocardial infarction (%)	30 (81)	0	<0.01
NYHA class	2 (2-3)	1 (0-2)	<0.001
CCS class	2 (2-3)	2 (1-2)	NS
Renal failure (%)	0	2 (17)	0.011
Arterial hypertension (%)	36 (97)	13 (81)	NS
Diabetes mellitus (%)	16 (43)	6 (37)	NS
Impaired glucose tolerance (%)	17 (46)	4 (25)	NS
Obesity (%)	16 (43)	5 (31)	NS
BMI [mean ± SD]	28.8±4	29±3	NS
Smoking (%)	10 (27)	2 (12)	NS
Positive family history (%)	6 (16.2)	1 (6)	NS
Atrial fibrillation (%)	4 (10.8)	0	NS
Atherosclerosis of peripheral arteries (%)	10 (27)	0	0.02
Stroke (%)	2 (5.4)	0	NS

Table II. Summary of echocardiography and resting electrocardiogram results in patients with multivessel CAD and control groups

	MCAD N=37	Control group N=16	p
Heart rate [bpm, mean ± SD]	71.9±11.5	75±17	NS
ST elevation (%)	4 (11)	0	NS
ST depression (%)	15 (40.5)	4 (25)	NS
Negative T waves (%)	21 (57)	9 (56)	NS
Pathological Q wave (%)	23 (62)	0	<0.001
LBBB (%)	1 (3)	0	NS
LAH (%)	2 (5.4)	1 (6)	NS
RBBB (%)	1 (3)	1 (6)	NS
QRS width [mm, mean, range]	90 (80-95)	83 (72-93)	NS
Non-corrected QT [ms, mean ± SD]	387±37	363±33	0.018
Left ventricular diastolic diameter [mm, mean ± SD]	52±8	46±3	0.01
Left ventricular systolic diameter [mm, mean ± SD]	39±9	30±6	0.001
EF [%, mean ± SD]	44±12	55±5	<0.001
Right ventricular diameter [mm, mean ± SD]	26±3	26±3	NS
Left atrium diameter [mm, mean ± SD]	43±5	40±4	NS
Aorta diameter [mm, mean ± SD]	34±4	34±3	NS

Abbreviations: LBBB – left bundle branch block, RBBB – right bundle branch block, LAH – left anterior hemiblock, EF – ejection fraction

made it possible to identify many markers of inflammatory response crucial in atherogenesis. Apart from white blood count, which is the easiest test but at the same time the least sensitive and specific diagnostic parameter, the following factors can be named: levels of pro- and anti-inflammatory cytokines (IL-1, IL-6, IL-8, TNF-α), adhesive molecules, oxidised lipoproteins, C-reactive protein (CRP) and amyloid A [12, 13].

A new, recently revealed inflammatory marker, closely connected with ischaemic heart disease, is the fat cell protein product resistin. This molecule takes part in atherogenesis through complex pathophysiological

Table III. Characteristics of the multivessel CAD and control groups regarding exercise test result

	MCAD N=24	Control group N=16	p
Workload [METS, mean ± SD]	5.7±2.5	7.6±3	0.04
Significant ST depression (%)	19 (79)	5 (42)	NS
Significant ST elevation (%)	1 (4)	0	NS
Athens QRS score [mean ± SD]	–2.3±–1.5	1.1±1	0.01
Positive test (%)	20 (83)	3 (25)	0.03
Non-diagnostic test (%)	2 (8)	2 (17)	NS
Negative test (%)	2 (8)	7 (58)	NS

Table IV. Selected laboratory test results in the multivessel CAD and control groups. Means are given ± SD except for NT-pro-BNP where mean and range are given

	MCAD N=37	Control group N=16	p
Adiponectin [μg/mg]	10.5±4.2	17.6±3	0.001
Resistin [ng/ml]	13.7±6.1	7.2±2.4	0.007
IL-8 [ng/ml]	18.4±4.1	12.2±4.1	0.008
TNF-α [ng/ml]	4.2±2.9	2.1±1.1	0.02
Haemoglobin [g/dl]	14.8±1.6	14.6±1	NS
WBC [K/μl]	7.8±1.9	7.2±1	NS
PLT [K/μl]	255.7±68.2	239±48	NS
Total cholesterol [mg/dl]	201±47.1	183±18	NS
HDL-cholesterol [mg/dl]	45±8.5	54±11	0.005
LDL-cholesterol [mg/dl]	126.1±46.9	102±29	0.004
Triglycerides	150±43	161±59	NS
CRP [mg/dl]	5.3±4.7	1.7±1.2	0.01
Urea [mg/dl]	35.8±9.9	34±17	NS
Creatinine [mg/dl]	0.9±0.2	0.89±0.17	NS
Uric acid [mg/dl]	6.1±1.2	5.4±1.3	0.02
GFR [ml/min/1.73 m^2]	96.9±28.8	97±43	NS
NT-proBNP [ng/ml]	516 (174-1426)	187 (39-573)	0.02
Fasting blood glucose [mg/dl]	97±14	94±11	0.03
Blood glucose after 2 hours [mg/dl]	186±51.7	143±45	0.02
HBA_{1C} [%]	6.5±2.1	6.17±1	NS
Fibrinogen [mg/dl]	388.1±74.6	376±89	NS

Abbreviations: IL – interleukini, TNF – tumor necrosis factor, WBC – withe blood coles, PLT – platelets, CRP – C-reactive protein, GFR – glomerular filtration rate, HBA_{1C} – glycasylated haemoglobin

pathways of the inflammatory response which finally lead to activation of endothelial cells and smooth muscle proliferation [14-16]. Additionally, resistin mRNA was found to be important in the inflammatory response of cells constituting atherosclerotic plaque [17]. All the studies showed elevated serum resistin level in CAD patients, while its level indicates the severity of the inflammatory response connected with atherogenesis. The reports published so far on the relationship between resistin levels and progression, severity and prognosis of patients with CAD in most cases confirmed, usefulness of resistin in the routine diagnostic process. Patients with CAD have significantly higher levels of serum resistin, especially those with early CAD manifestation [18, 19]. Reilly et al. demonstrated an association between resistin level and the degree of coronary artery calcification ('calcium score') computed based on interpretation of imaging from computed tomography performed in asymptomatic patients [20]. Based on angiographic evaluation, Ohmeri et al. came to the conclusion that there is a correlation between the this adpocin level and the number of stenoses in coronary arteries [21]. Wenlan et al. also documented significantly different concentrations of resistin in stable and unstable CAD, thus confirming the role of resistin in

risk stratification of atherosclerotic plaque destabilisation in CAD patients [22]. On the other hand, Pilz et al. did not show any correlation between resistin level and severity of atherosclerosis in a group of 1100 patients, though high level of this adopcin was a strong and independent predictor of non-fatal cardiovascular events in this group [23]. Our results are similar. Serum resistin concentrations were significantly higher in patients with multivessel CAD as compared to controls. The lack of relationship between the studied parameters and atherosclerosis severity in the coronary artery expressed as Gensini score can be explained by very advanced, quite homogeneous lesions hindering blood flow in coronary arteries.

Another substance recently detected and found useful in risk stratification of multivessel CAD is adiponectin, which takes part in carbohydrate homeostasis, and has strong insulin-sensitising, anti-atherosclerotic and anti-inflammatory action. This fat cell protein product acts by means of two types of receptors dependent on G protein (AdipoR1 and AdipoR2) localised mainly in skeletal muscles and liver [24, 25]. These receptors were also found in atherosclerotic plaques and their expression is induced by peroxisome proliferator-activated receptors [26]. Adiponectin increases sensitivity to insulin and it may be brought about by stimulation of fatty acid oxidation, which is the result of growth of protein kinase activation being activated by AMP and PPAR-α [27]. It's anti-inflammatory effect is due to reduction of pro-inflammatory cytokine production (TNF-α and IL-8), caused by nuclear factor kappa B inhibition [25, 26]. The anti-atherosclerotic effect of adiponectin is additionally strengthened by stimulation of expression and activity of nitric oxide synthase [27, 28]. Pleiotropy of adipokine activity is the reason for performing a number of clinical studies investigating its usefulness in everyday medical practice. Many studies have reaffirmed a strong relation between low serum level of adiponectin and development of metabolic syndrome or type 2 diabetes [29-31].

The usefulness of the adiponectin analysis in risk stratification of CAD development in healthy individuals raises much more doubts. A meta-analysis performed using seven large prospective trials including over 1300 patients showed no usefulness of adiponectin for the assessment of risk of CAD development (OR 0.84; 95% CI 0.7-1.01) [32]. Adiponectin plays a completely different role as a marker reflecting severity of CAD in patients with known CAD. According to Otsuka et al. in a group of 207 patients with confirmed CAD a level of adiponectin lower than 4 µg/ml is a strong and independent predictor of severe atherosclerosis of coronary arteries (OR 2.14; p=0.027) [33]. Miłosz D et al. drew similar conclusions – mainly hypoadiponectinaemia and exacerbation of inflammatory process, expressed by CRP and sVCAM-1 concentrations, were responsible for greater escalation of atherogenesis in coronary arteries [34]. Some recent publications expressed doubts on the potential usefulness of adipokin in long-term risk assessment in patients with CAD [35].

Patients with multivessel CAD included in our study had significantly lower serum adiponectin concentration as opposed to the control group. Previous reports are consistent with our findings with respect to lower adiponectin concentration in males, tobacco smokers and those with impaired carbohydrate metabolism, although we studied a smaller group of patients then in other reports.

What seems to be of interest is a wide dissemination of carbohydrate metabolism disorders of various severity in patients with CAD. Diabetes is a well-known risk factor of CAD and its presence significantly promotes atherogenesis. Impaired carbohydrate metabolism accelerates the formation of lipid deposits in arterial walls; this phenomenon is of systemic nature and to varying extent affects all vessels in the body [8, 9]. In our study only three patients had normal carbohydrate homeostasis defined as normal fasting blood glucose and 2 hours after oral glucose tolerance test using 75 g of glucose, which makes the assesment of relationship between diabetes and MCAD difficult in our study.

Among interpretations of additional tests, non-invasive CAD diagnostics using a treadmill test requires a separate explanation. The specificity of such a test in patients with MCAD is higher than in the population with isolated narrowing of epicardial artery, reaching 85-90% (1,36), which roughly corresponds with our results. The presence of ischaemic ST depression in the posterior and lateral leads (II, III, aVF, V_5-V_6) observed in all patients with positive test result (n=20) is noticeable. It is interesting to point out that such an exercise test result was also typical for patients with dominant myocardial perfusion defect within the region supplied by the left anterior descending branch. The Athens QRS score also turned out to be a very useful parameter for analysis of the exercise test result. Its value is independent of exercise-induced ischaemic ECG changes, thus showing great usefulness in interpretation of non-diagnostic exercise test results [37, 38]. An analysis of QRS complex in both the MCAD and control groups revealed significant differences.

In conclusion, MCAD is a serious health problem and because of a lack of clear classification systems and clear diagnostic criteria has still relatively very poor prognosis. Bearing all that in mind, the ability of each clinician to perform risk stratification of atherosclerosis severity in coronary arteries is very important. The key element of the management strategy seems to be medical history broadened with interpretation of selected, routine imaging studies and novel biochemical markers such as adiponectin, resistin, pro-inflammatory cytokines or NT-proBNP.

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Stężenia wybranych adipokin w surowicy osób z wielonaczyniową chorobą wieńcową

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Streszczenie

Wstęp: Z powodu bardzo niekorzystnego rokowania wczesne kwalifikowanie chorych do grupy o wysokim ryzyku rozpoznania zaawansowanego stadium miażdżycy w tętnicach wieńcowych ma duże znaczenie kliniczne.

Cel: Charakterystyka kliniczna, elektrograficzna i echokardiograficzna, poszerzona o oznaczenie nowatorskich markerów aterogenezy, chorych z trójnaczyniowym wariantem choroby wieńcowej.

Metody: Do badania włączono grupę 37 chorych (29 mężczyzn, średni wiek 64±8 lat) z wielonaczyniową chorobą wieńcową oraz grupę kontrolną 16 chorych (8 mężczyzn, średni wiek 60±10 lat), u których pomimo typowych dolegliwości stenokardialnych, dodatniego wyniku testu wysiłkowego i odcinkowych zaburzeń kurczliwości nie stwierdzano w badaniu angiograficznym istotnej hemodynamicznie miażdżycy tętnic wieńcowych. U wszystkich chorych wykonano cały zestaw badań dodatkowych [oprócz badania angiograficznego – 29 mężczyzn (78%), średni wiek w grupie 64±8 lat] oceniających stopień zaawansowania choroby i obecność innych dysfunkcji narządowych. Średnia wartość wskaźnika *Gensini score* w grupie badanej wyniosła 91,9±43,8, w tym *Gensini score* proksymalny 52,6±45,6 i dystalny 39,4±29,7.

Wyniki: Osoby z trójnaczyniowym wariantem choroby wieńcowej miały długi wywiad dolegliwości wieńcowych (średnio 84 miesiące), 30 (81%) spośród nich przebyło w przeszłości co najmniej jeden pełnościenny zawał mięśnia sercowego. W EKG częściej stwierdzano u nich zmiany typowe dla niedokrwienia, w badaniu echokardiograficznym – znacznie bardziej nasilone odcinkowe zaburzenia kurczliwości, frakcja wyrzutowa lewej komory była istotnie niższa w zestawieniu z grupą kontrolną (44 *vs* 55%, p <0,001). Chorzy w grupie badanej i kontrolnej mieli znamiennie różne wartości stężenia adiponektyny (10,5±4,2 *vs* 17,6±3 µg/ml, p=0,001), rezystyny (13,7±6,1 *vs* 7,2±2,4 ng/ml, p=0,007), TNF-α (4,2±2,9 *vs* 2,1±1,1 pg/ml, p=0,02) oraz IL-8 (18,4±4,1 *vs* 12,2±4,1 pg/ml, p=0,008) w surowicy. Istotne statystycznie różnice dotyczyły również lipidogramu (cholesterol całkowity 201±47,1 *vs* 183±18 mg/dl, NS; cholesterol HDL 45±8,5 *vs* 54±11 mg/dl, p=0,005; cholesterol LDL 126,1±46,9 *vs* 102±29 mg/dl, p=0,004), NT-proBNP [516 (174–1426) *vs* 187 (39–573) pg/ml, p=0,02] oraz wartości glikemii na czczo (97±14 *vs* 94±11 mg/dl, p=0,03). Mężczyźni oraz osoby palące tytoń mieli istotnie niższe stężenia adiponektyny w surowicy.

Wnioski: Wydaje się, że głównym elementem oceny ryzyka obecności zaawansowanego stadium miażdżycy w tętnicach wieńcowych jest wywiad chorobowy, poszerzony o interpretację wybranych, rutynowo wykonywanych badań obrazowych oraz nowatorskich markerów biochemicznych, jak adiponektyna, rezystyna, TNF-α, IL-8 czy NT-proBNP.

Słowa kluczowe: wielonaczyniowy, choroba wieńcowa, charakterystyka

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