Diminished serum paraoxonase activity in patients with coronary artery calcification

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Abstract

Background: Previous studies have shown an association between paraoxonase (PON) activity and the presence and severity of coronary atherosclerosis.

Aim: To demonstrate any association between serum PON activity and the presence and severity of coronary artery calcification (CAC).

Methods: A total of 156 consecutive patients having the suspicion of coronary atherosclerosis or needing risk stratification for cardiovascular events were included in the present study. Peripheral venous blood samples of all participants to measure serum PON activity were collected before undergoing multidetector computed tomography, which was used to determine the presence and quantity of CAC.

Results: Serum PON-1 levels were lower in the CAC group compared to the no CAC group (60 [35–96] U/L vs. 291 [230–371] U/L, respectively, p < 0.001). There was a significant negative correlation between total CAC score and PON ($r^2 = 0.335$, $p < 0.001$). In multivariate analysis, the significant and independent predictors of the presence of CAC were male sex, high-sensitive C-reactive protein, and PON. Similarly, increased PON was significantly and independently associated with freedom from CAC. In receiver operating characteristics analysis, PON level < 197 U/L had 87% sensitivity, 91% specificity, 93% positive predictive value, and 85% negative predictive value in predicting CAC.

Conclusions: Diminished serum PON activity is significantly and independently associated with the presence and severity of CAC, and vice versa.

Key words: coronary calcification, multidetector computed tomography, paraoxonase

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INTRODUCTION

An increased level of high density lipoprotein (HDL) has been reported to be associated with a decreased risk for coronary artery disease. This protective effect of HDL against atherosclerosis has been at least partly attributed to enzymes associated with HDL. One of these enzymes is paraoxonase (PON). Studies have shown that HDL-associated PON-1 inhibits lipid peroxidation and degrades biologically active oxidised lipids in low-density lipoprotein (LDL) [1]. PON-1 is recruited with breakdown of lipid peroxides before their accumulation on LDL [2].

The presence and quantity of coronary artery calcium (CAC) detected by computed tomography (CT) correlates with the overall magnitude of the atherosclerotic plaque burden and with the development of subsequent adverse coronary events [3–7]. It has been shown that its increased levels, measured as either percentiles (CAC > 75th percentile for age and sex matched) or absolute scores (i.e. > 100 or > 400)
are associated with adverse events, and because of that in several guidelines the high CAC score has been proposed as a marker to identify individuals requiring aggressive preventive management [8–10]. It has been also demonstrated that CAC improved risk prediction after taking into account Framingham risk score in four racial or ethnic groups [11].

METHODS

Study population

All patients admitted to our department of cardiology were possible candidates for inclusion in the study. However, among them, patients who were scheduled for multidetector CT as a screening tool for cardiovascular risk assessment of low-intermediate likelihood persons (coronary CT has higher accuracy values when low-likelihood persons are subjected) and patients who underwent coronary CT after a non-diagnostic, non-conclusive stress test to determine coronary atherosclerosis, were candidates for inclusion in the study. A total of 156 consecutive patients (mean age 57 ± 10 years; range 36–87 years; 98 [63%] male and 58 [37%] female) were recruited. Venous blood samples were obtained from all study participants for the measurement of laboratory parameters before multidetector CT. Exclusion criteria included the following: known coronary artery disease; left ventricular dysfunction (left ventricular ejection fraction [LVEF] < 50%) and hypertrophy (defined as increased left ventricular mass index calculated echocardiographically; > 95 g/m² for women and > 115 g/m² for men); unstable ischaemic conditions (unstable angina pectoris and myocardial infarction); rhythms other than sinus; significant valvular heart disease; current systemic inflammatory conditions; pregnancy; renal or hepatic dysfunction (creatinine > 2.5 mg/dL, AST and ALT > 2 times upper limit of normal, respectively); psychiatric abnormalities requiring drug use; thyroid abnormalities; any drug use affecting measured laboratory parameters including statins, fibrates (any drug for dyslipidaemia), furosemide, antidepressants, anticonvulsants, vitamin supplements, acetylsalicylic acid, non-steroidal anti-inflammatory drugs, antibiotics, alcohol, and refusal to take part in the study. The only permitted drugs and comorbidities were beta-blockers, calcium channel blockers, angiotensin converting enzyme inhibitors/angiotensin receptor blockers, hypertension and diabetes. Written consent was obtained from all patients and our local ethical committee approved the study.

Measurement of PON activity

Blood samples were obtained following an overnight fasting period before coronary CT. The serum was separated from the cells by centrifugation at 3,000 r/min for 10 min, and stored at –78°C until measurement of PON activity.

Serum PON activities were measured using the synthetic substrate paraoxon (diethyl-p-nitrophenol, PS610, SUPELCO, USA). The rate of paraoxon hydrolysis was measured by monitoring the increase of absorbency at 415 nm at 37°C. The amount of generated p-nitrophenol was calculated from the molar absorptivity coefficient at pH 8, which was 16,900/M/cm [12]. PON activity was expressed as U/L serum. The most widely available and used method for measuring PON-1 activity is the continuous recording spectrophotometer [13–18].

Other laboratory data

Fasting peripheral venous blood samples were also obtained from all patients in the study for the measurement of total cholesterol, LDL cholesterol, HDL cholesterol, triglyceride, and glucose levels. Blood samples were centrifuged and plasma was obtained. Total cholesterol, HDL cholesterol, triglyceride, and glucose levels were measured enzymatically with an auto analyser. Measurement of LDL cholesterol level was done through application of Friedewald’s formula.

Multidetector computed tomography

Computed tomography images were obtained and the quantity of CAC was measured with the 64-slice technique (Toshiba Aquilion 64 scanner, Toshiba Medical, Tochigi, Japan). Slices of 3 mm-thickness were acquired. The score according to the algorithm suggested by Agatston et al. [19] was used by one radiologist blinded to the study protocol. On the basis of the electrocardiography tracing, the software automatically selected a reduced set of diastolic images from each cardiac cycle. All pixels with a density > 130 HU were automatically highlighted in colour on the images. The radiologist assigned one of four locations to each calcified plaque: left main, left anterior descending, circumflex, or right coronary artery. The Agatston method determines the density of the highest density pixel in each plaque and applies a weighting factor to each plaque dependent on the peak density in the plaque: density [HU] of 130–199 = weight of 1; density of 200–299 = weight of 2; density of 300–399 = weight of 3; and density of ≥ 400 = weight of 4. The score for each plaque equals the plaque area X weighting factor X increment/slice width. The score for the entire specimen equals the sum of the scores for each plaque. The original Agatston method used a slice thickness of 3 mm.

Statistical analysis

For an 80% statistical power, a minimum sample size per group (two-tailed hypothesis, two-tailed α of 0.05) of 64 (total of 128) is required. Therefore, the statistical power of our study was 87.3%. Data was analysed with SPSS software version 15.0 for Windows (SPSS Inc., Chicago, IL, USA). Categorical variables were presented as frequency and percentage. The χ² test and Fisher’s exact test were used to compare categorical variables. The Kolmogorov-Smirnov test was used to assess the distribution of continuous variables. Student’s t-test was used for variables with normal distribution and the values
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were presented as mean ± standard deviation. Continuous variables without normal distribution were analysed using Mann-Whitney U test and the obtained values were presented as median (50th) and interquantile (25th and 75th) ranges; ACE-I/ARB — angiotensin converting enzyme inhibitor/angiotensin receptor blocker; BP — blood pressure; CAC — coronary artery calcium; HDL — high density lipoprotein; LDL — low density lipoprotein; LVEF — left ventricular ejection fraction.

### RESULTS

#### Baseline characteristics

Baseline demographic, clinical and laboratory characteristics of the two study groups are outlined in Table 1. There was a male predominance in the CAC group compared to the no CAC group. The CAC group was also older than the no CAC group. The prevalence of hypertension was more frequent in the CAC group compared to the no CAC group, although the prevalence of diabetes was similar. Diastolic blood pressure level was lower in the CAC group than in the no CAC group, although no systolic blood pressure level difference was present. Echocardiographically determined diastolic dysfunction was more prevalent in the CAC group compared to the no CAC group. LVEF measured by CT was lower in the CAC group than in the no CAC group. Laboratory parameters including total cholesterol, LDL cholesterol and HDL cholesterol were higher in the no CAC group compared to the CAC group.

#### Main laboratory parameters

The main objective laboratory data of the study is presented in Table 2. Accordingly, PON levels were lower and hsCRP levels were higher in the CAC group compared to the no CAC group. However, in the diabetic subgroup there was no significant difference regarding serum PON-1 activity between the CAC and no CAC groups (115 [90–198] U/L vs. 187 [86–208] U/L).

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Table 1. Baseline demographic, clinical and laboratory parameters of the two study groups

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>CAC group (n = 86)</th>
<th>No CAC group (n = 70)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>66 (77%)</td>
<td>32 (46%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Age [years]</td>
<td>60 ± 10</td>
<td>53 ± 8</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>20 (23%)</td>
<td>12 (17%)</td>
<td>0.427</td>
</tr>
<tr>
<td>Hypertension</td>
<td>64 (74%)</td>
<td>36 (51%)</td>
<td>0.004</td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
<td>62 (72%)</td>
<td>54 (77%)</td>
<td>0.581</td>
</tr>
<tr>
<td>Smoking</td>
<td>22 (26%)</td>
<td>18 (26%)</td>
<td>0.985</td>
</tr>
<tr>
<td>Systolic BP [mm Hg]*</td>
<td>120 (117–127)</td>
<td>124 (117–130)</td>
<td>0.314</td>
</tr>
<tr>
<td>Diastolic BP [mm Hg]</td>
<td>72 ± 10</td>
<td>75 ± 9</td>
<td>0.046</td>
</tr>
<tr>
<td>Pulse pressure [mm Hg]*</td>
<td>50 (45–57)</td>
<td>50 (45–53)</td>
<td>0.364</td>
</tr>
<tr>
<td>Heart rate [bpm]*</td>
<td>70 (65–79)</td>
<td>70 (66–80)</td>
<td>0.384</td>
</tr>
<tr>
<td>Beta-blocker use</td>
<td>5 (6%)</td>
<td>4 (6%)</td>
<td>0.979</td>
</tr>
<tr>
<td>Calcium channel blocker use</td>
<td>9 (11%)</td>
<td>5 (7%)</td>
<td>0.470</td>
</tr>
<tr>
<td>ACE-I/ARB use</td>
<td>50 (58%)</td>
<td>27 (39%)</td>
<td>0.015</td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>72 (84%)</td>
<td>42 (60%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Tomographic LVEF*</td>
<td>64 (54–67)</td>
<td>67 (63–70)</td>
<td>0.003</td>
</tr>
<tr>
<td>Fasting plasma glucose [mg/dL]*</td>
<td>99 (93–122)</td>
<td>97 (89–110)</td>
<td>0.214</td>
</tr>
<tr>
<td>Total cholesterol [mg/dL]</td>
<td>189 ± 45</td>
<td>208 ± 38</td>
<td>0.008</td>
</tr>
<tr>
<td>LDL cholesterol [mg/dL]</td>
<td>111 ± 40</td>
<td>125 ± 37</td>
<td>0.025</td>
</tr>
<tr>
<td>HDL cholesterol [mg/dL]*</td>
<td>42 (37–52)</td>
<td>44 (40–57)</td>
<td>0.048</td>
</tr>
<tr>
<td>Triglyceride [mg/dL]*</td>
<td>151 (105–195)</td>
<td>137 (107–187)</td>
<td>0.957</td>
</tr>
<tr>
<td>Lipid hydroxide [nmol]*</td>
<td>11.79 (4.84–20.63)</td>
<td>10.32 (5.47–14.74)</td>
<td>0.194</td>
</tr>
<tr>
<td>Aryl esterase [U/L]</td>
<td>202 ± 27</td>
<td>208 ± 27</td>
<td>0.144</td>
</tr>
<tr>
<td>Total thiol [μL]</td>
<td>518 ± 125</td>
<td>570 ± 81</td>
<td>0.004</td>
</tr>
</tbody>
</table>

*Values were presented as median (50th) and interquartile (25th and 75th) ranges; ACE-I/ARB — angiotensin converting enzyme inhibitor/angiotensin receptor blocker; BP — blood pressure; CAC — coronary artery calcium; HDL — high density lipoprotein; LDL — low density lipoprotein; LVEF — left ventricular ejection fraction.
respectively, \( p = 0.436 \) and no significant correlation was found between PON-1 and CAC \( (r^2 = 0.095, \ p = 0.085) \).

According to measured and then calculated Agatston score, 86 (55%) patients had a score \( \leq 10 \), 20 (13%) had a score of 11–100, 20 (13%) had a score of 101–400, 14 (9%) had a score of 401–1,000, and 16 (10%) patients had a score of \( > 1,000 \).

### Univariate and multivariate analyses

In univariate analysis of demographic, clinical and laboratory parameters, male sex, age, hypertension, pulse pressure, LVEF, diastolic dysfunction, and laboratory parameters including total cholesterol and LDL cholesterol, lipid hydroxide, total thiol, PON, and hsCRP were found to be significant predictors of the presence of CAC (Table 3). Low total cholesterol and LDL cholesterol predict coronary calcium by univariate analysis. This may be a consequence of total cholesterol and LDL cholesterol being used to select patients for CAC screening.

In multivariate analysis, however, after the addition of variables of male sex, age, hypertension, diastolic and pulse pressures, LVEF, diastolic dysfunction, and laboratory parameters including total cholesterol and LDL cholesterol, lipid hydroxide, total thiol, PON, and hsCRP, the significant and independent predictors of the presence of CAC were male sex, hsCRP and PON (Table 4).

In addition, in both univariate (OR 1.018; 95% CI 1.013–1.024; \( p < 0.001 \)) and multivariate (OR 1.012; 95% CI 1.004–1.019; \( p = 0.002 \)) analyses, serum PON increment (for each 1 U/L) was found to be a significant and independent predictor of the absence of CAC (CAC = 0).

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**Table 2. Main laboratory parameters between the two study groups**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>CAC group (n = 86)</th>
<th>No CAC group (n = 70)</th>
<th>( \chi^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paraoxonase [U/L]*</td>
<td>60 (35–96)</td>
<td>291 (230–371)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>High-sensitive C-reactive protein [mg/L]*</td>
<td>3.73 (1.88–5.07)</td>
<td>0.97 (0.56–1.48)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

*Values were presented as median (50th) and interquartile (25th and 75th) ranges; CAC — coronary artery calcium

**Table 3. Univariate analyses for the risk of coronary artery calcium**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
<th>( \chi^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>3.922</td>
<td>1.972–7.813</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Age [year]</td>
<td>1.090</td>
<td>1.047–1.135</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>2.747</td>
<td>1.401–5.405</td>
<td>0.003</td>
</tr>
<tr>
<td>Diastolic blood pressure [mm Hg]</td>
<td>0.966</td>
<td>0.933–1.000</td>
<td>0.049</td>
</tr>
<tr>
<td>Pulse pressure [mm Hg]</td>
<td>1.035</td>
<td>0.994–1.077</td>
<td>0.096</td>
</tr>
<tr>
<td>ACE-I/ARB use</td>
<td>2.212</td>
<td>1.161–4.213</td>
<td>0.016</td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>3.425</td>
<td>1.626–7.246</td>
<td>0.001</td>
</tr>
<tr>
<td>Tomographic LVEF [%]</td>
<td>0.923</td>
<td>0.871–0.978</td>
<td>0.006</td>
</tr>
<tr>
<td>Total cholesterol [mg/dL]</td>
<td>0.989</td>
<td>0.982–0.997</td>
<td>0.010</td>
</tr>
<tr>
<td>LDL cholesterol [mg/dL]</td>
<td>0.990</td>
<td>0.982–0.999</td>
<td>0.028</td>
</tr>
<tr>
<td>Lipid hydroxide [nmol]</td>
<td>1.030</td>
<td>0.998–1.063</td>
<td>0.067</td>
</tr>
<tr>
<td>Total thiol [( \mu L )]</td>
<td>0.995</td>
<td>0.992–0.999</td>
<td>0.005</td>
</tr>
<tr>
<td>Paraoxonase [U/L]</td>
<td>0.982</td>
<td>0.977–0.987</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>High-sensitive C-reactive protein [mg/L]</td>
<td>3.995</td>
<td>2.484–6.427</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

ACE-I/ARB — angiotensin converting enzyme inhibitor/angiotensin receptor blocker; LDL — low density lipoprotein; LVEF — left ventricular ejection fraction

**Table 4. Multivariate analyses for the risk of coronary artery calcium**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>4.673</td>
<td>1.080–20.408</td>
</tr>
<tr>
<td>High-sensitive C-reactive protein [mg/L]</td>
<td>2.044</td>
<td>1.056–3.954</td>
</tr>
<tr>
<td>Paraoxonase [U/L]</td>
<td>0.988</td>
<td>0.981–0.996</td>
</tr>
</tbody>
</table>
Sensitivity, specificity, positive and negative predictive values analyses

In ROC analysis, PON level < 197 U/L had 87% sensitivity, 91% specificity, 93% positive predictive value, and 85% negative predictive value in predicting CAC (area under the curve 0.077; 95% CI 0.034–0.119) and hsCRP level > 1.78 mg/dL had 78% sensitivity, 94% specificity, 94% positive predictive value, and 78% negative predictive value in predicting CAC (area under the curve 0.869; 95% CI 0.808–0.929) (Fig. 3).

If three independent parameters of male sex, hsCRP > 1.78 mg/dL and PON < 197 U/L were included in the model, the sensitivity, specificity, positive predictive value, and negative predictive value were 64%, 100%, 100%, and 69%, respectively.

Risk estimate of CAC and no CAC for binary value (197 U/L) of PON

Lastly, 75 patients had a PON level < 197 U/L in the CAC group (11 patients with ≥ 197 U/L) compared to six patients in the no CAC group (64 patients with ≥ 197 U/L) (87%...
vs. 9%, p < 0.001). PON level < 197 U/L increased the risk of presence of CAC (or ≥ 197 U/L increased the predictive value for freedom from CAC) by 73 times (OR 72.727; 95% CI 25.472–207.652; p < 0.001).

DISCUSSION

We have demonstrated for the first time that there was a notable decrease in PON activity in patients with CAC when compared to control subjects without CAC, and vice versa. We further demonstrated a significant and independent association between PON activity and the presence/severity of CAC.

There are three known PON subtypes: PON-1, PON-2 and PON-3. The most abundant, PON-1, is primarily synthesised in the liver and transported to the HDL in the plasma [20]. PON-1 activity is a readily detectable and measurable process in human plasma. Most reported HDL protective effects have been believed to be mediated by PON-1. PON-1 is by far the most abundant of all PONs within the vasculature. Oxidative modification of LDL plays a major role in the pathogenesis of atherosclerosis [21], and PON-1 directly protects LDL from oxidative modification [22]. In other words, HDL-associated PON-1 prevents lipid peroxidation and inhibits biologically active oxidised lipids in the LDL [1]. Specifically, the genes related to PON have been demonstrated to be associated with measures of arterial calcification. These PON genes have been shown to be associated with calcified plaques in both the carotid and the coronary arteries [23].

In addition, total plasma thiols have been the focus of great interest because previous studies have reported that even a mild degree of hyperhomocysteinaemia was associated with an elevated risk of stenotic vascular disease [24–28]. There is conflicting data regarding the pathophysiological mechanism behind the vascular injury.

Previous studies have shown that PON-1 polymorphism genotypes were associated with increased carotid intima-media thickness in various populations [29, 30]. It has been suggested that this serum marker can be used as an additional risk factor for subclinical atherosclerosis.

Similarly to the study by Kaya et al. [31], a lower serum PON activity in patients with CAC compared to no CAC, and a significantly negative correlation between CAC quantity and PON activity, were found in our study. However, unlike Kaya et al. [31], we used multidetector CT to determine the presence and quantity of CAC.

In a study performed by Kopp et al. [32], multi-detector CT has yielded better information for the detection and quantification of CAC than electron-beam CT, with low inter-observation variability. Because the volumetric method with isotropic interpolation has improved the reproducibility of CAC score, this has resulted in a statistically significant improvement over the standard calcium scoring method [33]. An observational study in regard to mortality data of approximately 10,000 asymptomatic persons has demonstrated that with increasing calcium scores, five-year survival from all-cause mortality worsens [34].

Lastly, Mackness et al. [35] demonstrated that there was no association between PON-1 activity and CAC in patients with type 2 diabetes. Similarly, we found no significant difference regarding serum PON-1 activity between CAC and no CAC groups, and no correlation between serum PON-1 activity and CAC in diabetic patients in our study.

Limitations of the study

Some important limitations should be mentioned about the current study: firstly, this was a cross-sectional case-control study which has inherent disadvantages including no randomisation, blindness and follow-up outcome data. Secondly, the sample size of the study population was small, which means that the results have to be confirmed by a larger study. Lastly, because serum PON levels were only measured at a single time-point we were unable to examine the variability and prognostic value of level changes over time or the impact of different therapeutic strategies such as drugs or interventional procedures on serum PON activity.

CONCLUSIONS

In patients with CAC, decreased serum activity levels of the antioxidant HDL-associated enzyme PON-1 was significantly and independently associated with the presence and severity of CAC. This serum marker can be used in predicting which patients might be at risk for increased and severe CAC presence. In addition, patients requiring more preventive measures such as more aggressive antiatherosclerotic drug
regimens or interventional procedures can be determined. In addition, with increased PON levels one can predict with a high ratio that the patient with low-intermediate likelihood cardiovascular risk is free from CAC.

Conflict of interest: none declared

References
Zmniejszona aktywność paraoksonazy w surowicy u chorych ze zwapnieniem tętnic wieńcowych

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Streszczenie

Wstęp: Wcześniej badania wykazały związek między aktywnością paraoksonazy (PON) a występowaniem i nasileniem zwapnień w tętnicach wieńcowych (CAC).

Cel: Celem badania było wykazanie zależności między aktywnością PON w surowicy a występowaniem i nasileniem CAC.

Metody: Do badania włączono 156 kolejnych pacjentów z podejrzeniem miażdżycy tętnic wieńcowych lub wymagających stratyfikacji ryzyka zdarzeń sercowo-naczyniowych. Od wszystkich uczestników badania pobrano próbki obwodowej krwi żylnej w celu wykonania pomiaru aktywności PON w surowicy przed badaniem metodą multidetektorowej tomografii komputerowej, które zastosowano do wykrycia obecności zwapnień i ich oceny ilościowej.

Wyniki: Stężenia PON-1 w surowicy były niższe w grupie z CAC niż u osób, u których nie stwierdzono CAC (odpowiednio 60 [35–96] j./l i 291 [230–371] j./l; p < 0,001). Wykazano istotną ujemną korelację między całkowitą punktacją w skali oceny CAC oraz PON ($r^2 = 0,335$; $p < 0,001$). W analizie wieloczynnikowej silnymi i niezależnymi czynnikami predykcyjnymi występowania CAC były: płeć męska, stężenia PON i białka C-reaktywnego mierzonego metodą wysokoczułą. Zwiększone aktywność PON była istotnie i niezależnie związana z brakiem zmian typu CAC. W analizie krzywych ROC stężenie PON wynoszące poniżej 197 j./l pozwalało prognozować wystąpienie CAC, przy czym oznaczenie tego parametru cechowało się 87-procentową czułością, 91-procentową swoistością, wartością predykcyjną dodatnią — 93% i wartością predykcyjną ujemną — 85%.

Wnioski: Zmniejszona aktywność PON w surowicy była istotnie i niezależnie związana z obecnością oraz nasileniem CAC i odwrotnie — mała aktywność CAC korelowała z brakiem CAC.

Słowa kluczowe: zwapnienie tętnic wieńcowych, multidetektorowa tomografia komputerowa, paraoksonaza

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