Prediction models for different plaque morphology in non-significantly stenosed regions of saphenous vein grafts assessed with optical coherence tomography

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Abstract

Introduction: Coronary artery bypass grafting (CABG) is a method of choice in treatment of diffuse coronary artery disease (CAD), although it has some limitations such as late saphenous vein graft (SVG) patency loss, which occurs in one fifth of all conduits at 5 years. Since atherosclerosis in SVG has diffuse characteristics, it appears that significantly and non-significantly stenosed lesions may have an equal impact on worse prognosis.

Aim: To assess non-significant lesions of SVG by the use of optical coherence tomography (OCT) and investigate the clinical and laboratory findings with the potential impact on plaque composition.

Material and methods: Twenty-nine patients with 43 non-significant lesions were enrolled in the study. All variables were assessed using uni- and multivariable logistic regression analysis with each plaque morphology as a dependent variable. Odds ratio (OR) and 95% confidence interval (CI) were computed.

Results: Plaque rupture (PRT) was independently associated with age (OR = 1.49, 95% CI: 1.09–2.04, p = 0.015) and lower rates of high-density lipoproteins (HDL) cholesterol (OR = 0.67, 95% CI: 0.49–0.92, p = 0.016). Intimal tearing or rupture (ITR) was related to reduced GFR (OR = 0.52, 95% CI: 0.38–0.72, p = 0.0004). Lipid-rich plaque (LRP) was associated with raised platelet count (PLT) (OR = 1.51, 95% CI: 1.16–1.96, p = 0.004) and increased frequency of smoking (OR = 1.45, 95% CI: 1.12–1.89, p = 0.007).

Conclusions: Atherosclerosis of SVG is not restricted to significantly stenosed lesions. Plaque composition is independently associated with different types of clinical and laboratory findings, mostly recognized as risk factors of CAD.

Key words: optical coherence tomography, coronary artery disease, saphenous vein graft coronary artery bypass grafting.

Summary

The current report focuses on optical coherence tomography imaging of the saphenous vein grafts (SVG) atherosclerosis in non-significantly stenosed regions of the vessel wall. It demonstrates that specific clinical features and laboratory findings, as assessed by multivariate logistic regression analysis, have potential impact on plaque composition in these regions of the SVG.

Introduction

Coronary artery bypass grafting (CABG) is considered a method of choice in treatment of diffuse coronary artery disease (CAD) according to the current guidelines of revascularization and the reports from the SYNTAX trial [1–3]. The Medicare Provider and Analysis Review (MedPAR) database reported nearly 2.5 million CABGs performed between 1991 and 2005 in the United States (US) [4]. Despite the observed decline in the number of operations, the annual rate of CABGs dropped from 1742...
in the years 2001–2002 to 1261 in the years 2005–2006, and the number of these procedures in the years 2007–2008 was still 1081 per million adults in the US (average data from 2 years) [5]. The trend of a reduced frequency of CABG is related to the advances in interventional cardiology which have been made in recent years by the introduction of drug-eluting stents (DES), advanced percutaneous coronary intervention (PCI) techniques (rotablation, bifurcation dedicated stents, mechanical circulatory devices) and innovations in pharmacological treatment [6–8]. Nevertheless, CABG is still needed in a vast amount of patients not suitable for PCI. It is noteworthy that CABG poses many limitations, mainly related to the SVG late patency rate. Approximately 20% of saphenous vein grafts (SVGs) lose their patency at 5-year follow-up [9, 10], which is mostly caused by accelerated atherosclerosis. Risk factors of accelerated atherosclerosis are similar to those for native coronary atherosclerosis, but their relation to the SVG plaque composition was not described previously. Moreover, non-significant lesions are frequently associated with progression of the narrowing, which can manifest as acute coronary syndrome (ACS) [11, 12].

Aim

Therefore, the aim of the present study was to identify the variables with a potential impact on the SVG plaque type as assessed by optical coherence tomography (OCT) imaging.

Material and methods

Study population

Twenty-nine patients hospitalized in the Upper Silesia Medical Center between June 2013 and March 2016 were enrolled in the OCTOPUS registry [13, 14]. The study complies with the Declaration of Helsinki and was accepted by the local ethical committee. Each patient gave his informed written consent prior to enrollment. Inclusion criteria were as follows: CABG prior to intervention (SVG use mandatory), acute coronary syndrome, coronary artery disease with evidence of active ischemia in non-invasive testing. Exclusion criteria were as follows: lack of consent, ST-segment elevation myocardial infarction, less than 18 years of age, severe valvular insufficiency, contrast allergy, location of the lesion preventing safe examination. Lesions were defined as significant if they were involved in the initial manifestation of ACS assessed on the basis of clinical and non-invasive testing, and/or were quantitatively (QCA) assessed as 50% stenosed or more. The other lesions were considered as non-significant and assigned for further analysis.

OCT procedure and imaging technique

The St Jude Ilumien Optis Medical system was used for OCT Imaging. The OCT Dragonfly catheter was advanced through a guiding catheter over a 0.014’ guide-wire into the SVG via a 6 Fr left radial or femoral approach. The OCT probe was positioned 5 mm distal to the lesion submitted to analysis. All OCT images were acquired using automatic pullback triggered by the hand injection of contrast flush. All patients were adequately heparinized with the activated clotting time (ACT) > 300 s. The OCT image analysis was performed by an independent core laboratory at Krakow Cardiovascular Research Institute (www.KCRI.org). In case of a conflict of opinions the analyzed frame was excluded from the analysis. OCT analysis scrutinized serial images of the vessel at every 1 mm cross section (CS) for both significant and non-significant de novo SVG lesions. Cross-sectional area (CSA) and vessel lumen diameter were measured at every 1 mm in order to acquire the smallest values for both parameters, which were defined as minimal lumen diameter (MLD) and minimal CSA. They were assessed for both types of lesions. The OCT reference lumen area and reference diameter were estimated at the site of the largest CSA within the analyzed SVG for both de novo SVG lesions and non-significant lesions. Percentage lumen diameter and area stenosis were defined as the relative decrease in luminal diameter and CSA of the target lesion compared to the reference lumen diameter and CSA [15].

OCT image analysis

Tissue was classified as lipid for signal-poor regions with diffuse borders and high signal attenuation, homogeneous for signal-rich regions, calcified for signal-poor regions with sharp edges, and heterogeneous for poor signal regions without signal attenuation. The length of an arc of lipid and calcium that occupied the vessel wall circumference was measured and expressed in degrees [16, 17]. The thickness of the fibrous cap that covered the lipid core was measured in the thinnest part of a signal-rich zone that separated the lipid content from the vessel lumen (µm). The fibrous cap thickness was a mean value of three measurements. The OCT-defined thin-cap fibroatheroma (TCFA) was defined as a lipid-rich plaque (LRP) with fibrous cap thickness < 65 µm. Also, the presence of luminal thrombus, plaque rupture (PRT), intimal tear or rupture (ITR), friable tissue (FRB), calcified plaque (CAL) and the presence of venous valves were noted during the OCT analysis. An intimal tear was defined as a micro-cavity between the SVG lumen and its media, an intimal rupture as a micro-cavity of intima connected with the SVG lumen, tissue friability as a signal-free zone overlaid with signal-rich tissue inside the SVG wall [18]. Different types of SVG lesions are depicted in Figure 1. Offline OCT image analysis was performed using CAAS Intravascular 2.0 (Pie Medical Imaging BV). The quantitative OCT analysis was performed by three observers (GK, EP and TR). If a consensus could not be reached, the lesion was removed from...
Figure 1. Different types of lesions in saphenous vein grafts – morphology specified in the text: A – fibrotic lesion, B – calcified lesion, C – lipid-rich plaque, D – thin-cap fibroatheroma, E – intimal tearing, F – intimal rupture, G – friable tissue, H – plaque rupture
the analysis. The intraobserver variability for OCT quantitative measurements was presented previously [19].

**Statistical analysis**

Categorical variables are presented as counts and proportions, and the comparisons were performed using the \( \chi^2 \) test with Yates correction. The continuous variables are presented as the median and 25th to 75th percentile and mean ± SD. Linear variables with normal distribution were compared using Student’s \( t \)-test. Variables with abnormal distribution were compared using the Mann-Whitney \( U \) test. All variables were assessed using univariable logistic regression analysis with each plaque morphology as a dependent variable. The odds ratio (OR) and 95% confidence interval (CI) were computed. The variables fulfilling the Akaike information criterion (AIC) described previously [20, 21] with \( p < 0.1 \) were included in the primary multivariable logistic regression models of each plaque morphology occurrence. Additional adjustments for potential confounders were performed by the backward stepwise method, which enabled construction of the final multivariable logistic regression models of each plaque morphology occurrence. Differences between the values were considered statistically significant if \( p < 0.05 \). Analyses were performed using Statistica 10 with the medical package (StatSoft Inc.).

**Results**

Twenty-nine patients with 32 de novo SVG significant and 43 non-significant lesions were included in the study. The data for clinical characteristics were analyzed on a per patient basis and the data on plaque morphology were analyzed per lesion. The number of non-significant is greater than significant lesions because in eleven cases the plaque composition was complex and presented more than one pathology. It necessitated the examination of an additional region of the vessel wall, which resulted in an increased number of analyzed regions. Percutaneous coronary intervention was performed in 22 of the de novo SVG lesions. For the patient characteristics and OCT-derived data please refer to Tables I and II respectively.

All the analyzed variables were included in the univariable logistic regression analysis of each plaque occurrence – the data are depicted in Tables III and IV. The variables fulfilling the AIC (marked with an asterisk) were included in the multivariable logistic regression models. The obtained data from the multivariable logistic regression analysis, after exclusion of confounding factors (final models), were as follows: PRT was independently associated with age (OR = 1.49, 95% CI: 1.09–2.04, \( p = 0.015 \)) and lower serum concentration of high-density lipoprotein (HDL) cholesterol (OR = 0.67, 95% CI: 0.49–0.92, \( p = 0.016 \)). Intimal tear or rupture was related to reduced GFR (OR = 0.52, 95% CI: 0.38–0.72, SD – standard deviation, IQR – interquartile range, CABG – coronary artery bypass grafting, LIMA-LAD – left internal mammary artery to left anterior descending artery, ARB – angiotensin II receptor blocker, ACEI – angiotensin-converting-enzyme inhibitor, LDL – low-density lipoproteins, HDL – high-density lipoproteins, GFR – glomerular filtration rate.

**Table I. Patients’ characteristics (n = 29)**

| Clinical data | Results |
|---------------|---------|
| Male, n (%)   | 24 (83) |
| Age, mean ± SD| 69.07 ±7.56 |
| Body mass index, median (IQR) [kg/m²] | 28.5 (26–32) |
| Non-ST elevated myocardial infarction, n (%) | 1 (3) |
| Unstable angina, n (%) | 10 (35) |
| Stable angina, n (%) | 18 (62) |
| Risk factors: | |
| Hypertension, n (%) | 26 (90) |
| Current smoking, n (%) | 13 (45) |
| Hyperlipidemia, n (%) | 25 (86) |
| Diabetes, n (%) | 2 (7) |
| Time from CABG, median (IQR) [months] | 143 (100–212) |
| Number of saphenous vein grafts, n (%): | |
| 1 | 4 (14) |
| 2 | 18 (62) |
| 3 | 7 (24) |
| Arterial graft (LIMA-LAD), n (%) | 26 (90) |
| Pharmacological therapy, n (%): | |
| β-Adrenergic antagonist | 25 (86) |
| Calcium channel antagonist | 4 (14) |
| Aspirin | 28 (97) |
| Thienopyridine | 2 (7) |
| Statin | 29 (100) |
| ARB/ACEI | 20 (69) |
| Other lipid-lowering therapy | 6 (21) |
| Insulin | 2 (7) |
| Oral antidiabetics | 5 (17) |
| Laboratory results: | |
| GFR, median (IQR) [ml/min/1.73 m²] | 71 (53–88) |
| White blood cells, median (IQR) [× 10³/μl] | 6.32 (5.69–7.24) |
| Platelets, median (IQR) [× 10³/μl] | 184 (161–228) |
| Hemoglobin, median (IQR) [mg/dl] | 14.08 (12.90–15.22) |
| Total cholesterol, mean ± SD [mg/dl] | 162.29 ±58.52 |
| LDL cholesterol, median (IQR) [mg/dl] | 78 (68–98) |
| Triglyceride, median (IQR) [mg/dl] | 132 (103–157) |
| HDL cholesterol, median (IQR) [mg/dl] | 41 (32–48) |

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Lipid-rich plaque was associated with raised platelet count (PLT) \((OR = 1.51, 95\% CI: 1.16–1.96, p = 0.004)\) and increased frequency of smoking \((OR = 1.45, 95\% CI: 1.12–1.89, p = 0.007)\). The data are depicted in Figure 2.

On the other hand, FIB was independently associated with increased body surface area (BSA) \((OR = 1.62, 95\% CI: 1.25–2.10, p = 0.001)\), decreased frequency of smoking \((OR = 0.65, 95\% CI: 0.50–0.84, p = 0.003)\) and hypertension \((OR = 0.73, 95\% CI: 0.56–0.95, p = 0.024)\). Calcified plaque was related to decreased serum concentration of total cholesterol (TCH) \((OR = 0.73, 95\% CI: 0.54–0.99, p = 0.049)\) and more white blood cells (WBC) \((OR = 1.39, 95\% CI: 1.03–1.89, p = 0.036)\). Friable tissue was related to increased frequency of smoking \((OR = 1.61, 95\% CI: 1.17–2.22, p = 0.006)\). The data are depicted in Figure 3.

**Discussion**

According to our best knowledge, we are the first to address the issue of the hypothetical impact of clinical and laboratory findings on different plaque morphologies assessed using OCT in non-significant lesions of SVGs. The novelties in the study are the highly selected group of CABG patients with SVGs and the use of advanced statistical techniques to construct a best matching prediction model of each plaque morphology. Several imaging modalities including coronary angiography (CAG) and intravascular ultrasound (IVUS) have shown no differences in plaque composition of native vessels and laboratory findings on different plaque morphologies that decreased HDL cholesterol is independently associated with the incidence of PRT \((OR = 0.67, 95\% CI: 0.39)\). These data are in agreement with our findings that decreased HDL cholesterol is independently associated with the incidence of PRT \((OR = 0.67, 95\% CI: 0.39)\).

### Table II. Optical coherence tomography derived data concerning plaque characteristics

| Optical coherence tomography findings | Non-significantly stenosed lesions \((n = 43)\) |
|--------------------------------------|-----------------------------------------------|
| Region of interest, median (IQR) \([\text{mm}]\) | 112 (8.0–13.2) |
| Ref. lumen CSA, median (IQR) \([\text{mm}^2]\) | 7.5 (5.6–8.7) |
| Ref. mean lumen diameter, median (IQR) \([\text{mm}]\) | 3.1 (2.7–3.3) |
| Minimal lesion lumen CSA \([\text{mm}^2]\) | NA |
| Minimal lumen diameter, median (IQR) \([\text{mm}]\) | 2.8 (2.5–3.1) |
| Area stenosis, median (IQR) \([\%]\) | 15.0 (13.0–17.0) |
| Diameter stenosis, median (IQR) \([\%]\) | 0.0 (0.0–5.0) |
| Maximal lipid arc, median (IQR) \([\%]\) | 140.0 (125–155) |
| Maximal calcification arc, median (IQR) \([\%]\) | 94.0 (75–120) |
| Plaque calcification, \(n\) \((\%)\) | 16 (37) |
| TCFA, \(n\) \((\%)\) | 0 (0) |
| Thrombus, \(n\) \((\%)\) | 0 (0) |
| Heterogeneous tissue, \(n\) \((\%)\) | 4 (9) |
| Plaque rupture, \(n\) \((\%)\) | 4 (9) |
| Lipid-rich plaque, \(n\) \((\%)\) | 15 (35) |
| Dissection, \(n\) \((\%)\) | 0 (0) |
| Intimal tearing, \(n\) \((\%)\) | 2 (5) |
| Intimal rupture, \(n\) \((\%)\) | 3 (7) |
| Tissue friability, \(n\) \((\%)\) | 2 (5) |
| Plaque within the SVG valve, \(n\) \((\%)\) | 0 (0) |

CSA – cross sectional area, IQR – interquartile range, NA – not applicable, Ref – reference, TCFA – thin-cap fibroatheroma, SVG – saphenous vein graft.

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The progression of intermediate SVG lesions into severely stenosed lesions during a median of 35 months of follow-up was also reported by Abdel-Karim et al. [11]. These findings are in line with our results which revealed that smoking was an independent predictor of FRB and LRP occurrence.

### Study limitations

We cannot conclude definitively whether this advanced technology could contribute to clinical practice in this demanding group of patients because we did not correlate the OCT findings with the clinical endpoints. Moreover, the number of study participants is relatively small and the investigation was performed in a single center. However, the researchers did not interfere with the management process at any stage. It is noteworthy that, since OCT is an invasive procedure, there exists a theoretical possibility of iatrogenic damage of the vessel wall, which might have influenced the results. Consideration of these limitations is crucial for the interpretation of the findings.

### Table III. Univariable logistic regression analysis of multiple determinants on each plaque morphology

| Parameter                  | PRT (n = 4) | ITR (n = 5) | LRP (n = 15) |
|----------------------------|-------------|-------------|--------------|
|                            | Odds ratio (95% CI), p-value | Odds ratio (95% CI), p-value | Odds ratio (95% CI), p-value |
| EEM vol. [mm]              | 0.91 (0.66–1.24), 0.531 | 0.92 (0.67–1.25), 0.586 | 1.25 (0.92–1.69), 0.153 |
| Lumen vol. [mm]            | 0.94 (0.69–1.28), 0.692 | 0.92 (0.68–1.26), 0.617 | 1.23 (0.91–1.67), 0.178 |
| Min. av. lum. diam [mm]    | 1.19 (0.87–1.61), 0.276 | 1.12 (0.82–1.53), 0.471 | 1.14 (0.84–1.55), 0.401 |
| Min. lum. area [mm²]       | 1.18 (0.87–1.61), 0.289 | 1.11 (0.81–1.51), 0.519 | 1.13 (0.83–1.55), 0.423 |
| Min. lum. diam. [mm]       | 1.13 (0.83–1.55), 0.419 | 1.03 (0.75–1.41), 0.857 | 1.22 (0.90–1.66), 0.197 |
| Plaque vol. [mm]           | 0.82 (0.61–1.12), 0.209 | 0.91 (0.67–1.24), 0.536 | 1.26 (0.93–1.71), 0.130 |
| Stenosis EEM [%]           | 0.93 (0.68–1.27), 0.640 | 0.91 (0.66–1.24), 0.530 | 0.99 (0.72–1.35), 0.928 |
| Stenosis length [mm]       | 0.77 (0.57–1.05), 0.096* | 0.85 (0.62–1.16), 0.297 | 1.24 (0.91–1.68), 0.170 |
| Stenosis reference [%]     | 0.86 (0.63–1.17), 0.332 | 0.83 (0.61–1.13), 0.240 | 1.23 (0.90–1.67), 0.187 |
| Surf msk. TLP [mm²]        | 0.85 (0.62–1.15), 0.284 | 0.89 (0.65–1.21), 0.450 | 1.27 (0.94–1.72), 0.125 |
| Age [years]                | 1.31 (0.97–1.79), 0.084* | 1.37 (1.01–1.85), 0.047* | 1.20 (0.88–1.65), 0.250 |
| Body surface area [m²]     | 0.79 (0.56–1.13), 0.196 | 1.35 (0.95–1.90), 0.094* | 0.93 (0.65–1.33), 0.676 |
| BMI [kg/m²]                | 0.80 (0.56–1.13), 0.201 | 0.93 (0.65–1.33), 0.690 | 1.00 (0.70–1.44), 0.983 |
| LVEF [%]                   | 0.94 (0.69–1.29), 0.714 | 0.58 (0.45–0.76), < 0.001* | 1.12 (0.82–1.53), 0.457 |
| Troponin [ng/l]            | 0.86 (0.60–1.24), 0.415 | 1.42 (1.01–1.99), 0.051* | 1.21 (0.84–1.73), 0.298 |
| HGB [mg/dl]                | 0.91 (0.66–1.25), 0.563 | 0.95 (0.69–1.31), 0.773 | 0.94 (0.68–1.29), 0.694 |
| WBC [10⁹/μl]               | 1.11 (0.81–1.53), 0.519 | 1.08 (0.78–1.49), 0.632 | 1.04 (0.76–1.43), 0.802 |
| PLT [10⁹/μl]               | 1.12 (0.82–1.54), 0.466 | 0.94 (0.68–1.30), 0.712 | 1.59 (1.20–2.11), 0.002* |
| TCH [mg/dl]                | 0.93 (0.66–1.30), 0.668 | 0.63 (0.47–0.85), 0.004* | 1.13 (0.81–1.58), 0.473 |
| TG [mg/dl]                 | 1.18 (0.84–1.65), 0.337 | 0.74 (0.53–1.02), 0.070* | 0.97 (0.69–1.37), 0.858 |
| LDL [mg/dl]                | 0.92 (0.66–1.29), 0.621 | 0.84 (0.60–1.17), 0.308 | 1.08 (0.77–1.51), 0.646 |
| HDL [mg/dl]                | 1.86 (1.35–2.57), 0.058* | 0.96 (0.68–1.34), 0.801 | 0.98 (0.70–1.37), 0.897 |
| Creatinine [mg/dl]         | 1.12 (0.81–1.54), 0.498 | 1.28 (0.93–1.75), 0.127 | 0.86 (0.62–1.18), 0.346 |
| GFR [ml/min/1.73 m²]       | 0.94 (0.68–1.30), 0.702 | 0.57 (0.44–0.75), < 0.001* | 1.14 (0.82–1.57), 0.428 |
| Male                       | 0.95 (0.69–1.30), 0.737 | 0.99 (0.72–1.35), 0.934 | 0.76 (0.56–1.02), 0.072* |
| Diabetes                   | 0.85 (0.62–1.15), 0.283 | 1.07 (0.78–1.46), 0.683 | 1.03 (0.76–1.41), 0.840 |
| Hypertension               | 1.14 (0.83–1.55), 0.410 | 1.16 (0.85–1.58), 0.350 | 1.34 (1.00–1.81), 0.055* |
| Current smoking            | 0.90 (0.66–1.23), 0.513 | 1.14 (0.84–1.56), 0.393 | 1.55 (1.17–2.05), 0.003* |

PRT – plaque rupture, ITR – intimal tearing or rupture, LRP – lipid-rich plaque, EEM – external elastic membrane, TLP – total lumen perimeter, BMI – body mass index, LVEF – left ventricular ejection fraction, HGB – hemoglobin, WBC – white blood cells, PLT – platelets, TCH – total cholesterol, TG – triglycerides, LDL – low-density lipoproteins, HDL – high-density lipoproteins, GFR – glomerular filtration rate.
Table IV. Univariable logistic regression analysis of multiple determinants on each plaque morphology

| Parameter                  | FIB (n = 27)         | CAL (n = 16)         | FRB (n = 2)         |
|----------------------------|----------------------|----------------------|---------------------|
|                            | Odds ratio (95% CI), p-value | Odds ratio (95% CI), p-value | Odds ratio (95% CI), p-value |
| EEM vol. [mm]              | 1.01 (0.74–1.38), 0.960 | 0.93 (0.68–1.27), 0.655 | 0.83 (0.61–1.13), 0.225 |
| Lumen vol. [mm]            | 0.98 (0.72–1.34), 0.885 | 0.96 (0.70–1.31), 0.800 | 0.84 (0.62–1.15), 0.275 |
| Min. av. lum. diam [mm]    | 0.91 (0.67–1.25), 0.558 | 1.23 (0.91–1.67), 0.181 | 0.95 (0.69–1.30), 0.738 |
| Min. lum. area [mm²]       | 0.94 (0.69–1.28), 0.673 | 1.19 (0.87–1.61), 0.274 | 0.95 (0.69–1.29), 0.733 |
| Min. lum. diam. [mm]       | 0.89 (0.65–1.21), 0.448 | 1.18 (0.86–1.60), 0.299 | 0.94 (0.69–1.28), 0.689 |
| Plaque vol. [mm]           | 1.10 (0.81–1.51), 0.529 | 0.86 (0.63–1.17), 0.328 | 0.80 (0.59–1.08), 0.150 |
| Stenosis EEM [%]           | 1.19 (0.88–1.62), 0.257 | 0.86 (0.63–1.16), 0.316 | 0.87 (0.64–1.18), 0.359 |
| Stenosis length [mm]       | 1.05 (0.77–1.43), 0.759 | 0.81 (0.60–1.10), 0.176 | 0.84 (0.62–1.14), 0.263 |
| Stenosis reference [%]     | 0.98 (0.72–1.34), 0.898 | 0.79 (0.58–1.07), 0.134 | 0.98 (0.72–1.34), 0.883 |
| Surf msr. TLP [mm²]        | 1.02 (0.75–1.39), 0.897 | 0.90 (0.66–1.22), 0.484 | 0.82 (0.61–1.12), 0.211 |
| Age [years]                | 0.93 (0.68–1.28), 0.648 | 1.28 (0.94–1.74), 0.122 | 1.16 (0.85–1.59), 0.352 |
| Body surface area [m²]     | 1.53 (1.11–2.12), 0.014* | 0.95 (0.66–1.36), 0.764 | 0.81 (0.57–1.16), 0.248 |
| BMI [kg/m²]                | 1.28 (0.91–1.82), 0.161 | 0.77 (0.54–1.08), 0.133 | 0.89 (0.62–1.27), 0.520 |
| LVEF [%]                   | 0.85 (0.62–1.16), 0.300 | 0.91 (0.66–1.24), 0.534 | 1.05 (0.77–1.43), 0.755 |
| Troponin [ng/l]            | 0.80 (0.56–1.15), 0.230 | 1.13 (0.78–1.62), 0.512 | 1.36 (0.96–1.93), 0.083* |
| HGB [mg/dl]                | 1.06 (0.77–1.46), 0.708 | 0.81 (0.59–1.11), 0.184 | 0.91 (0.66–1.26), 0.575 |
| WBC [10³/μl]               | 0.94 (0.68–1.29), 0.697 | 1.35 (0.99–1.83), 0.057* | 0.96 (0.70–1.32), 0.795 |
| PLT [10³/μl]               | 0.75 (0.55–1.02), 0.067* | 0.95 (0.69–1.30), 0.739 | 0.94 (0.68–1.30), 0.704 |
| TCH [mg/dl]                | 0.78 (0.56–1.09), 0.147 | 0.71 (0.52–0.98), 0.040* | 1.08 (0.77–1.52), 0.634 |
| TG [mg/dl]                 | 0.66 (0.48–0.91), 0.013* | 1.01 (0.72–1.43), 0.939 | 1.15 (0.82–1.61), 0.423 |
| LDL [mg/dl]                | 0.80 (0.58–1.12), 0.195 | 0.87 (0.63–1.22), 0.428 | 1.04 (0.74–1.45), 0.833 |
| HDL [mg/dl]                | 1.35 (0.98–1.86), 0.072* | 0.68 (0.49–0.92), 0.016* | 1.00 (0.71–1.40), 0.980 |
| Creatinine [mg/dl]         | 1.21 (0.88–1.67), 0.230 | 1.02 (0.74–1.41), 0.911 | 0.85 (0.62–1.17), 0.315 |
| GFR [ml/min/1.73 m²]       | 0.90 (0.65–1.24), 0.510 | 0.83 (0.61–1.15), 0.260 | 1.00 (0.72–1.38), 0.996 |
| Male                       | 1.64 (1.25–2.16), 0.001* | 0.70 (0.36–1.36), 0.985 | 0.63 (0.48–0.83), 0.002* |
| Diabetes                   | 0.84 (0.62–1.14), 0.263 | 1.08 (0.79–1.48), 0.618 | 1.24 (0.91–1.68), 0.165 |
| Hypertension               | 0.73 (0.55–0.99), 0.043* | 1.36 (1.01–1.83), 0.043* | 1.09 (0.80–1.49), 0.571 |
| Current smoking            | 0.66 (0.50–0.88), 0.006* | 0.92 (0.68–1.26), 0.606 | 1.36 (1.01–1.83), 0.043* |

FIB – fibrotic plaque, CAL – calcified plaque, FRB – friable tissue, other abbreviations as in Table III.

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Conflict of interest
The authors declare no conflict of interest.

ing these facts, we believe further studies are warranted in this field.

Conclusions
Clinical and laboratory findings have an impact on plaque composition of non-significant lesions assessed with OCT. Saphenous vein graft endothelial pathology of non-significant lesions is associated with risk factors of CAD such as smoking, impaired renal function, elderly age, decreased HDL and raised PLT.
Figure 2. Logistic regression primary models of OCT-based prediction for different plaque types

HDL – high-density lipoproteins, GFR – glomerular filtration rate, LVEF – left ventricular ejection fraction.
### Table 1. Logistic regression secondary models of OCT-based prediction for different plaque types

| Plaque morphology       | Parameter                      | OR (95% CI)     | P-value |
|-------------------------|--------------------------------|-----------------|---------|
| Plaque rupture:         | HDL [mg/dl]                    | 0.67 (0.49–0.92) | 0.016   |
|                         | Age [years]                    | 1.49 (1.09–2.04) | 0.015   |
| Intimal tearing or rupture: | GFR [ml/min/1.73 m²]          | 0.52 (0.38–0.72) | <0.001  |
| Lipid rich plaque:      | Platelets [× 10³/μl]           | 1.51 (1.16–1.96) | 0.004   |
|                         | Smoking                        | 1.45 (1.12–1.89) | 0.007   |
| Fibrotic tissue:        | Hypertension                   | 0.73 (0.56–0.95) | 0.024   |
|                         | Smoking                        | 0.65 (0.50–0.84) | 0.003   |
|                         | Body surface area [m²]         | 1.62 (1.25–2.10) | 0.001   |
| Calcified plaque:       | Total cholesterol [mg/dl]      | 0.73 (0.54–0.99) | 0.049   |
|                         | White blood count [× 10³/μl]   | 1.39 (1.03–1.89) | 0.036   |
| Friable tissue:         | Smoking                        | 1.61 (1.17–2.22) | 0.006   |

Figure 3. Logistic regression secondary models of OCT-based prediction for different plaque types

HDL – high-density lipoproteins, GFR – glomerular filtration rate.

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