Saphenous graft atherosclerosis as assessed by optical coherence tomography data for stenotic and non-stenotic lesions from the OCTOPUS registry

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Abstract

Introduction: Coronary artery bypass grafting (CABG), although widely used for a long time in diffuse coronary artery disease (CAD), has serious limitations associated with graft aging and its degeneration.

Aim: The relationship between saphenous vein graft (SVG) plaque morphology assessed by optical coherence tomography (OCT) and clinical findings has not been elucidated yet.

Material and methods: We compared the morphology of SVG in stenotic vs. non-stenotic lesions using OCT imaging in 29 patients hospitalized in our center within the OCTOPUS registry.

Results: Stenotic lesions were characterized by higher incidence of thin-cap fibroatheroma (TCFA) (33% vs. 0%, p = 0.0048), thrombus (28% vs. 0%, p = 0.0008), lipid-rich plaque (LRP) (75% vs. 35%, p = 0.0013) and plaque within the SVG valve (19% vs. 0%, p = 0.0114) as compared to non-stenotic lesions. Patients with intimal tearing or rupture (ITR) were older (75.8% vs. 68.9 years, p = 0.047) and had lower left ventricular ejection fraction (LVEF) (32.0% vs. 49.7%, p = 0.001) and glomerular filtration rate (GFR) (36.0 vs. 73.6 ml/min/1.73 m^2, p = 0.010). Patients with calcified lesions vs. those without had lower high-density lipoprotein (HDL) cholesterol (33.2 vs. 44.1 mg/dl, p = 0.018), similarly to those with ruptured plaque vs. those without (28.3 vs. 41.7 mg/dl, p = 0.047).

Conclusions: Presence of ITR was associated with advanced age, decreased LVEF and renal insufficiency. Decreased concentration of HDL was associated with higher occurrence of calcified and ruptured plaque.

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

Introduction

Coronary artery bypass grafting (CABG) is widely applied to treat diffuse coronary artery disease [1]. It offers a significant reduction in mortality at 5-year follow-up compared to medical treatment only (10.2% vs. 15.8%; p = 0.0001) [2]. 88–95% of arterial conduits remain patent at ten or more years after surgery; thus their utilization is a preferred clinical modality. Unfavorably, their use has serious limitations (restrictions in the use of radial artery, mammary artery harvesting may result in sternal dehiscence and/or mediastinitis), particularly in obese and diabetic patients [3–6].

In contrast, only 32–71% of saphenous vein grafts (SVG) maintain patency at ten or more years [7–12]. As the re-do CABG has two- to four-fold increased mortality, percutaneous coronary intervention (PCI) within the SVG and/or native vessel remains the method of choice in the treatment of these cases [13, 14]. Percutaneous coronary intervention for SVG is associated with higher incidence of periprocedural myocardial infarction [15]. There exists a paradigm that atherosclerotic plaque localized in the venous conduits consists of friable tissue being prone to release its debris and cause distal embolization during PCI [16–19].

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There is a paucity of data concerning SVG plaque burden and tissue type. The majority of these observations were made before the introduction of optical coherence tomography (OCT) to the clinical setting. Hence, the question arises whether there is a significant difference between the stenotic and non-stenotic regions of the SVG as assessed by OCT imaging.

Aim

Therefore, the aim of the study was to compare the morphology of SVG in stenotic vs. non-stenotic lesions imaged by OCT, and to present these differences in relation to clinical settings.

Material and methods

Twenty-nine patients hospitalized in the Upper Silesia Medical Center between June 2013 and March 2016 were included in the OCTOPUS registry [20, 21]. Each patient gave informed written content, and the study complied with the Declaration of Helsinki and was accepted by the local ethical committee.

Inclusion criteria were: CABG prior to intervention and coronary artery disease with evidence of active ischemia in non-invasive testing or acute coronary disease. Exclusion criteria were: lack of consent or less than 18 years of age or severe valvular insufficiency or contrast allergy or localization of the lesion preventing safe examination or ST-elevated myocardial infarction.

Optical coherence tomography imaging technique

The lesion was defined as stenotic when it caused 50% stenosis as assessed. Otherwise, it was recognized as non-stenotic. The non-stenotic segments of the vessel were assigned for further analysis. 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' guidewire into the SVG via the 6 Fr left radial or femoral approach. The OCT probe was positioned 5 mm distal to the region 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 > 300 s.

Optical coherence tomography image analysis

The OCT image analysis was performed by an independent core laboratory at Krakow Cardiovascular Research Institute (www.KCRI.org). In the case of a conflict of opinions the analyzed frame was excluded from the analysis. The OCT region of interest (ROI) was defined as the lesion length limited by areas without atheroma or neointimal hyperplasia. The OCT analysis scrutinized serial cross-sectional images of the vessel at 1 mm intervals for both stenotic and non-stenotic *de novo* SVG lesions. Cross-sectional area (CSA), and vessel lumen diameter were measured every 1 mm. The smallest values for both parameters were defined as the minimal lumen diameter (MLD) of the minimal CSA and 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-stenotic 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.

Tissue was classified as homogeneous for signal-rich regions, lipid for signal-poor regions with diffuse borders and high signal attenuation, 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 [22, 23]. The maximal lipid arc and calcium arc were measured. 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 the mean value of three measurements. OCT defined thin-cap fibroatheroma (TCFA) as a lipid-rich plaque (LRP) with fibrous cap thickness < 65 μm. Also, the presence of plaque rupture (PRT), luminal thrombus, intimal tear or rupture (ITR), tissue friability (FRB) and venous valves was noted during the OCT analysis. An intimal tear was defined as a micro-cavity between the SVG lumen and its media, intimal rupture as a micro-cavity of the intima connected with the SVG lumen, tissue friability as a signal-free zone overlaid with signal-rich tissue inside the SVG wall [24]. Offline OCT image analysis was performed using CAAS Intravascular 2.0 (Pie Medical Imaging BV), and results of intraobserver variability for standard protocols were presented previously [25]. See Figure 1 for different types of plaque morphologies.

Statistical analysis

Distributions of the examined parameters were analyzed using the Shapiro-Wilk test. Categorical variables were expressed as *n* and percentage. Continuous variables were expressed as the mean ± standard deviation (SD) or as the median and the 25th and 75th percentiles (interquartile range). Linear variables with normal distribution were compared using Student's *t*-test. Variables with abnormal distribution were compared using the Mann-Whitney *U* test. Categorical variables of abnormal distribution were compared using the χ^2 test with Yates' correction. Differences between the values were considered statistically significant if $p < 0.05$. Analyses were

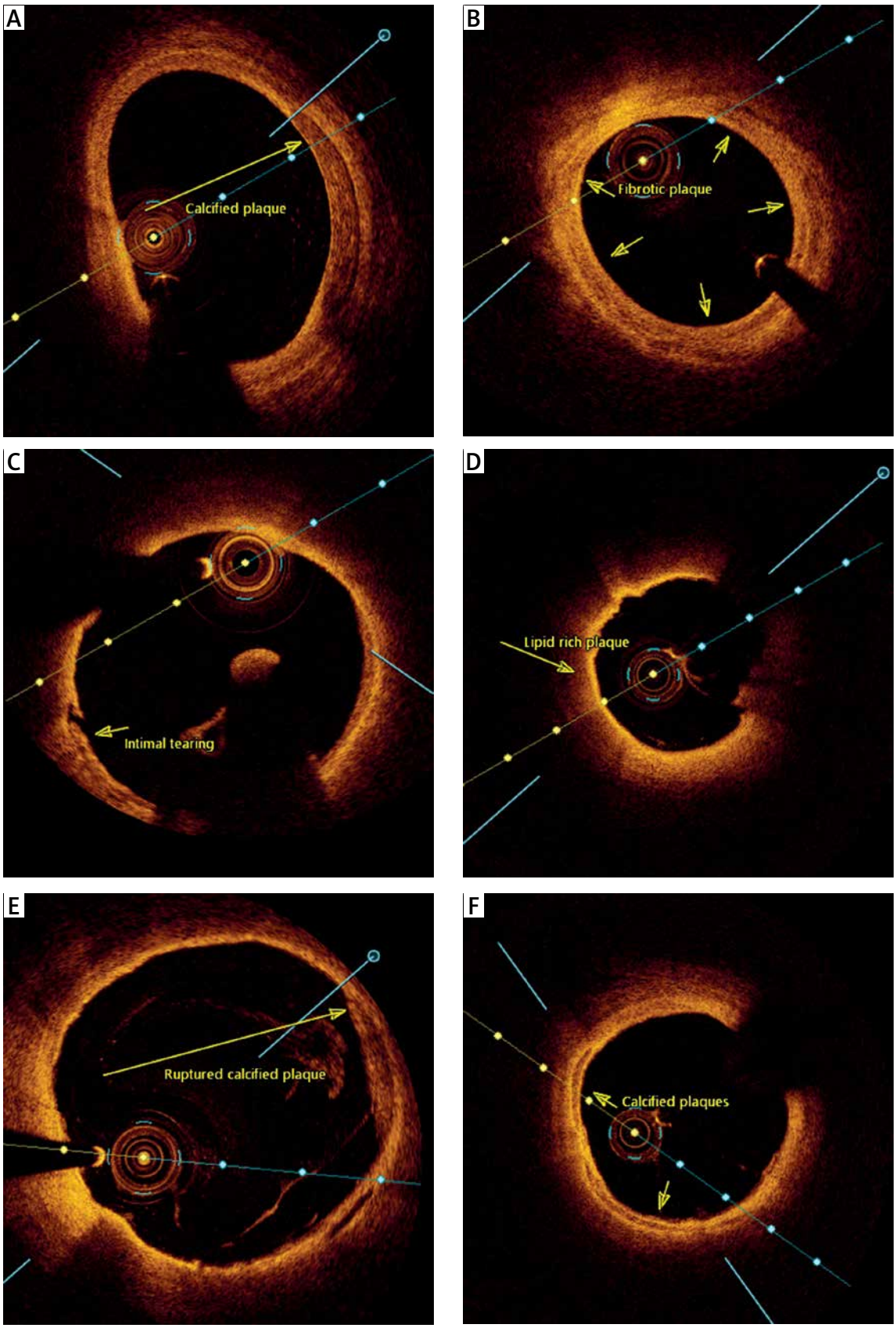

Figure 1. Different types of plaque morphologies. **A–F** – show different types of plaque morphologies: **A** – calcified plaque, **B** – fibrotic plaque, **C** – intimal tearing, **D** – lipid-rich plaque, **E** – ruptured calcified plaque, **F** – calcified plaques

Table I. Patient characteristics (n = 29)

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

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

performed using Statistica 10 with the medical package (StatSoft Inc.).

Results

Patients' characteristics

Twenty-nine patients with 32 *de novo* SVG stenotic and 43 non-stenotic lesions were included in the study. The data for clinical characteristics were depicted on a per patient basis, and the data for plaque morphology were analyzed on a per lesion basis. Percutaneous coronary intervention was performed in 22 of the *de novo* SVG lesions. The study population consisted of 24 males, mean age 69.07 ±7.56. Mean duration from CABG to the index procedure was 143 (100–212) months. Eighteen (62%) patients presented with stable CAD and 11 (38%) with acute coronary syndrome (ACS). Among ACS patients 10 presented unstable angina symptoms and 1 suffered from non-ST segment elevated myocardial infarction (NSTEMI). For patients' characteristics consisting of clinical data, pharmacological therapy and laboratory findings, see Table I.

Data derived from optical coherence tomography analysis of saphenous vein grafts

As shown in Table II, stenotic vs. non-stenotic lesions were characterized by a raised plaque burden expressed by lowered MLD (1.88 vs. 2.83 mm), increased area stenosis (61.00% vs. 15.05%), diameter stenosis (37.33% vs. 3.0%) and maximal lipid arc (269° vs. 97°); $p < 0.001$ for all. Furthermore, stenotic lesions had a higher incidence of TCFA (33% vs. 0%, $p = 0.0048$), thrombus (28% vs. 0%, $p = 0.0008$), LRP (75% vs. 35%, $p = 0.0013$), plaque within the SVG valve (19% vs. 0%, $p = 0.0114$) and decreased minimal cap thickness (80 vs. 139 µm, $p < 0.001$).

Patients with fibrotic (FIB) tissue were mostly men (26 vs. 9, $p = 0.004$) with higher body surface area (BSA) (2.0 vs. 1.9 m^2, $p = 0.014$) and increased serum creatinine concentration (1.1 vs. 1.0 mg/dl, $p = 0.028$). Moreover, this group of patients was characterized by a positive lipid profile consisting of significantly decreased triglycerides (TG) (116.0 vs. 164.4 mg/dl, $p = 0.013$), low-density lipoprotein (LDL) cholesterol (85.1 vs. 101.2, $p = 0.05$) and elevated high-density lipoprotein (HDL) cholesterol (43.2 vs. 34.8, $p = 0.06$), although neither of the last two p-values reached statistical significance. Patients with fibrotic tissue were less frequently current smokers (0 vs. 25%, $p = 0.029$). On the other hand, patients diagnosed with LRP had higher concentration of platelets (231.9 vs. 182.3 × 10^3/µl, $p = 0.008$) and were smokers (27% vs. 0%, $p = 0.020$). Data are presented in Table III.

As presented in Table IV, patients with calcified lesions (CAL) had decreased HDL cholesterol (33.2 vs. 44.1 mg/dl, $p = 0.018$), similarly to those with ruptured plaque (PRT) (28.3 vs. 41.7 mg/dl, $p = 0.047$).

Patients with intimal tearing or rupture (ITR) were older (75.8 vs. 68.9 years, p = 0.047), had significantly impaired systolic function with reduced left ventricular ejection fraction (LVEF) (32.0% vs. 49.7%, p = 0.001), decreased GFR (36.0 vs. 73.6 ml/min/1.73 m^2, p = 0.010) and total cholesterol (TCH) (93.4 vs. 161.3 mg/dl, p = 0.033). Patients with diagnosed ITR had raised cardiac troponin concentrations both before and after the procedure (0.6 vs. 0.2 ng/l, p = 0.05 for both) of borderline significance. Data are presented in Table V.

Discussion

According to our best knowledge, there is a lack of systematic comparison between stenotic vs. non-stenotic lesions assessed by OCT; thus, we encountered serious difficulties in addressing the issue in the previously published papers. Our observations concerning stenotic lesions are in line with the work of Davlouros *et al.* [24] with the exception that the ACS in our group of patients occurred in the minority of cases (11 ACS vs. 18 patients with stable angina), hence TCFA, PRT and ITR were considerably less frequent. What might be a novelty in the current research is that only TCFA, LRP, thrombus and plaque within the valve had a higher incidence rate in stenotic lesions compared to non-stenotic ones. In contrast, presence of PRT, ITR and FRB did not differ significantly. Adlam *et al.* [26] evaluated sixteen SVGs in asymptomatic patients 3 years after cardiac surgery and reported that the rates of TCFA and thrombus were 37.5% and 25% respectively. These data are consistent with our results – incidence of TCFA and thrombus were 33% and 28% respectively. Considering the significant imbalance between the time from CABG in Adlam's (3 years) and our (12 years) group of patients, it is tempting to speculate that the thrombus and TCFA formation accelerates in a non-linear way and a major impact on its occurrence is exerted by the quality of conduit tissue and periprocedural surgical conditions. Burgmaier *et al.* reported the relationship between plaque vulnerability and the left ventricle dilatation assessed by cardiac magnetic resonance

Table II. Comparison of stenotic vs. non-stenotic lesions in saphenous vein grafts (SVG)

Parameter	Stenotic lesions (n = 32)	Non-stenotic lesions (n = 43)	P-value
Region of interest [mm]	12.45 ±4.99	10.71 ±3.85	0.25
Reference lumen CSA [mm^2]	7.41 (IQR: 4.38–9.38)	7.56 (IQR: 5.60–8.70)	0.43
Reference mean lumen diameter [mm]	3.03 ±0.73	3.06 ±0.46	0.49
Minimal lesion lumen CSA, median (IQR) [mm^2]	2.71 (1.34–4.19)	NA	NA
Minimal lumen diameter [mm]	1.88 ±0.65	2.83 ±0.45	< 0.001
Area stenosis, median (IQR) (%)	61.00 (42.72–77.63)	15.05 (13.0–17.0)	< 0.001
Diameter stenosis (%)	37.33 ±17.25	3.0 ±4.82	< 0.001
Minimal cap thickness, median (IQR) [μm]	80 (60–101)	139 (125–155)	< 0.001
Maximal lipid arc, median (IQR) [°]	269 (163–317)	97 (75–120)	< 0.001
Maximal calcification arc [°]	86.89 ±54.19	112 ±51.6	0.11
Plaque calcification, n (%)	14 (44)	16 (37)	0.74
Thin-cap fibroatheroma, n (%)	7 (33)	0 (0)	0.0048
Thrombus, n (%)	9 (28)	0 (0)	0.0008
Heterogeneous tissue, n (%)	2 (6)	4 (9)	0.96
Plaque rupture, n (%)	4 (12.5)	4 (9)	0.95
Lipid-rich plaque, n (%)	24 (75)	15 (35)	0.0013
Dissection, n (%)	1 (3)	0 (0)	0.88
Intimal tearing, n (%)	2 (6)	2 (5)	0.83
Intimal rupture, n (%)	2 (6)	3 (7)	0.73
Tissue friability, n (%)	6 (19)	2 (5)	0.11
Plaque within the SVG valve, n (%)	6 (19)	0 (0)	0.0114

CSA – cross sectional area, IQR – interquartile range, NA – not applicable, SVG – saphenous vein graft; insignificant p values were rounded up to two decimal places.

imaging (CMR) in patients with type two diabetes [27]. We observed significant deterioration in the left ventricle (LV) systolic function in patients diagnosed with ITR (LVEF was 32.0% vs. 49.7%, $p = 0.001$). Moreover, this group of patients exhibited impaired renal function (GFR 36.0 vs. 73.6 ml/min/1.73 m^2, $p = 0.010$). These data are in line with the previous results of Burgmaier *et al.*, although some important differences should be addressed. First of all, we assessed the patients in a real-world setting; hence LVEF evaluation was performed by the use of ultrasound imaging and the penetration of HF is considerable (21% of patients with LVEF ≤ 35%). Secondly, despite

Table III. Clinical and imaging findings depending on tissue type according to OCT imaging

Parameter	FIB-0 (*n* = 16) Mean ± SD	FIB-1 (*n* = 27) Mean ± SD	*P*-value	LRP-0 (*n* = 28) Mean ± SD	LRP-1 (*n* = 15) Mean ± SD	*P*-value
EEM volume [mm]	123.4 ±53.3	124.3 ±57.7	0.96	115.1 ±55.8	140.6 ±52.7	0.15
Lumen volume [mm]	92.9 ±40.4	90.9 ±44.8	0.71	85.2 ±43.2	103.7 ±40.5	0.09
Min. av. lumen diameter [mm]	3.1 ±0.6	3.0 ±0.4	0.56	3.0 ±0.4	3.1 ±0.5	0.40
Min. lumen area [mm^2]	7.8 ±2.8	7.4 ±2.1	0.95	7.3 ±2.1	8.0 ±2.7	0.62
Min. lumen diameter [mm]	2.9 ±0.6	2.8 ±0.4	0.45	2.8 ±0.4	2.9 ±0.5	0.20
Plaque volume [mm]	30.4 ±14.2	33.3 ±14.5	0.58	29.8 ±14.0	36.8 ±14.2	0.13
Stenosis EEM [%]	14.5 ±2.5	15.4 ±2.3	0.26	15.1 ±2.3	15.0 ±2.7	0.93
Stenosis length [mm]	10.5 ±3.9	10.8 ±3.9	0.76	10.1 ±3.6	11.8 ±4.1	0.17
Stenosis reference (%)	3.1 ±6.5	2.9 ±3.6	0.19	2.3 ±3.6	4.3 ±6.5	0.42
Total lumen perimeter [mm^2]	109.8 ±41.1	111.5 ±43.8	0.90	103.6 ±41.6	124.5 ±41.6	0.13
Age [years]	70.4 ±5.5	69.3 ±8.4	0.65	68.7 ±6.4	71.5 ±8.7	0.25
Body surface area [m^2]	1.9 ±0.2	2.0 ±0.1	0.014	2.0 ±0.2	2.0 ±0.2	0.68
Body mass index [kg/m^2]	28.0 ±2.8	29.6 ±3.4	0.16	28.9 ±3.8	29.0 ±2.3	0.98
LVEF (%)	49.9 ±8.9	46.3 ±11.6	0.36	46.7 ±11.5	49.3 ±9.2	0.64
Troponin before [ng/l]	0.4 ±0.5	0.2 ±0.4	0.22	0.2 ±0.4	0.4 ±0.5	0.29
Troponin after [ng/l]	0.4 ±0.5	0.2 ±0.4	0.22	0.2 ±0.4	0.4 ±0.5	0.29
HGB [mg/dl]	15.0 ±7.3	15.9 ±7.1	0.71	15.9 ±7.1	15.0 ±7.1	0.69
WBC [× 10^3/µl]	7.1 ±1.8	6.9 ±1.6	0.54	6.9 ±1.4	7.1 ±2.0	0.97
PLT [× 10^3/µl]	221.1 ±65.8	189.7 ±40.9	0.19	182.3 ±31.9	231.9 ±65.5	0.008
TCH [mg/dl]	168.6 ±44.4	143.3 ±52.2	0.08	147.7 ±52.2	160.4 ±47.9	0.76
TG [mg/dl]	164.4 ±33.1	116.0 ±61.0	0.013	134.8 ±60.4	131.2 ±53.3	0.86
LDL [mg/dl]	101.2 ±37.2	85.1 ±34.3	0.05	88.7 ±33.0	94.5 ±41.3	0.67
HDL [mg/dl]	34.8 ±9.1	43.2 ±14.7	0.06	40.5 ±14.7	39.8 ±11.5	0.81
Creatinine [mg/dl]	1.0 ±0.4	1.1 ±0.4	0.028	1.1 ±0.3	1.0 ±0.3	0.13
GFR [ml/min/1.73 m^2]	72.4 ±17.9	67.3 ±24.7	0.77	66.8 ±24.6	72.9 ±18.7	0.50
Male, *n* (%)	9 (56)	26 (96)	0.004	25 (89)	10 (67)	0.16
Diabetes, *n* (%)	10 (63)	12 (44)	0.41	14 (50)	8 (53)	0.91
Hypertension, *n* (%)	16 (100)	21 (78)	0.12	22 (79)	15 (100)	0.14
Current smoking, *n* (%)	4 (25)	0 (0)	0.029	0 (0)	4 (27)	0.020

FIB-0/1 – fibrotic tissue absent/present, LRP-0/1 – lipid-rich plaque absent/present, EEM – external elastic membrane, min. – minimal, av. – average, LVEF – left ventricular ejection fraction, HGB – hemoglobin, WBC – white blood cells, PLT – platelets, TG – triglyceride, TCH – total cholesterol, LDL – low-density lipoprotein cholesterol, HDL – high-density lipoprotein cholesterol, GFR – glomerular filtration rate; insignificant p-values were rounded up to two decimal places, significant p-values were rounded up to three decimal places.

the fact that patients with diabetes are prone to glucose fluctuations which are associated with vulnerable plaque formation [28], PRT prevalence in the dilated LV group of patients was higher but without statistical significance (22.7% vs. 8.5%, p = 0.083). Notably, although many efforts aiming to improve long-term efficacy of SVG have been made throughout the years, interesting theoretical assumptions have not necessarily had a positive impact on clinical practice [29]. Last but not least, to date nothing is known about the relationship between clinical characteristics and SVG plaque morphology assessed by OCT in patients previously submitted to CABG. Therefore

Table IV. Clinical and imaging findings depending on tissue type according to OCT imaging

Parameter	CAL-0 (n = 27) Mean ± SD	CAL-1 (n = 16) Mean ± SD	P-value	PRT-0 (n = 39) Mean ± SD	PRT-1 (n = 4) Mean ± SD	P-value
EEM volume [mm]	126.9 ±57.5	119.0 ±53.4	0.66	125.7 ±57.5	107.2 ±28.2	0.53
Lumen volume [mm]	93.0 ±43.5	89.5 ±42.6	0.94	92.5 ±44.2	83.5 ±26.1	0.90
Min. av. lumen diameter [mm]	3.0 ±0.5	3.2 ±0.4	0.18	3.0 ±0.4	3.3 ±0.6	0.28
Min. lumen area [mm^2]	7.3 ±2.3	8.1 ±2.3	0.30	7.4 ±2.2	8.8 ±3.2	0.42
Min. lumen diameter [mm]	2.8 ±0.4	2.9 ±0.5	0.30	2.8 ±0.4	3.0 ±0.7	0.42
Plaque volume [mm]	33.9 ±15.0	29.4 ±13.0	0.41	33.1 ±14.7	23.6 ±2.3	0.17
Stenosis EEM [%]	15.3 ±2.3	14.6 ±2.6	0.32	15.1 ±2.4	14.5 ±2.9	0.64
Stenosis length [mm]	11.3 ±3.8	9.7 ±3.9	0.18	11.0 ±3.9	7.6 ±1.0	0.10
Stenosis reference (%)	3.9 ±5.7	1.6 ±2.4	0.19	3.2 ±5.0	0.8 ±1.5	0.25
Total lumen perimeter [mm^2]	114.4 ±42.0	104.9 ±43.5	0.48	113.1 ±43.6	89.1 ±15.7	0.28
Age [years]	68.3 ±8.6	71.9 ±4.1	0.12	69.1 ±7.4	75.8 ±2.5	0.08
Body surface area [m^2]	2.0 ±0.2	2.0 ±0.2	0.76	2.0 ±0.2	1.9 ±0.1	0.20
Body mass index [kg/m^2]	29.6 ±3.1	27.9 ±3.2	0.13	29.2 ±3.2	26.7 ±1.5	0.20
LVEF (%)	48.4 ±10.2	46.3 ±11.6	0.47	47.8 ±10.7	45.8 ±12.1	0.59
Troponin before [ng/l]	0.2 ±0.4	0.3 ±0.5	0.50	0.3 ±0.4	0.0 ±0.0	0.41
Troponin after [ng/l]	0.2 ±0.4	0.3 ±0.5	0.50	0.3 ±0.4	0.0 ±0.0	0.41
HGB [mg/dl]	16.6 ±8.5	13.5 ±1.2	0.18	15.8 ±7.4	13.6 ±1.1	0.56
WBC [× 10^3/µl]	6.6 ±1.2	7.6 ±2.2	0.19	6.9 ±1.5	7.5 ±2.6	0.88
PLT [× 10^3/µl]	202.4 ±42.0	196.6 ±69.4	0.13	198.4 ±48.1	218.8 ±89.7	0.91
TCH [mg/dl]	164.6 ±45.3	129.2 ±53.2	0.21	153.4 ±52.9	141.8 ±23.8	0.57
TG [mg/dl]	132.9 ±64.6	134.5 ±43.4	0.94	130.2 ±57.4	159.8 ±55.9	0.34
LDL [mg/dl]	94.2 ±42.2	84.3 ±18.4	0.90	91.8 ±37.1	82.3 ±21.3	0.86
HDL [mg/dl]	44.1 ±13.3	33.2 ±11.0	0.018	41.7 ±13.3	28.3 ±9.1	0.047
Creatinine [mg/dl]	1.0 ±0.3	1.1 ±0.4	0.87	1.0 ±0.3	1.2 ±0.6	0.89
GFR [ml/min/1.73 m^2]	71.9 ±17.4	63.4 ±30.0	0.44	69.4 ±22.8	64.8 ±24.1	0.60
Male, n (%)	22 (81)	13 (81)	0.70	32 (82)	3 (75)	0.74
Diabetes, n (%)	13 (48)	9 (56)	0.84	21 (54)	1 (25)	0.57
Hypertension, n (%)	21 (78)	16 (100)	0.16	33 (85)	4 (100)	0.93
Current smoking, n (%)	3 (11)	1 (6)	0.99	4 (10)	0 (0)	0.82

CAL-0/1 – calcified lesion absent/present, PRT-0/1 – plaque rupture absent/present, EEM – external elastic membrane, min. – minimal, av. – average, LVEF – left ventricular ejection fraction, HGB – hemoglobin, WBC – white blood cells, PLT – platelets, TG – triglyceride, TCH – total cholesterol, LDL – low-density lipoprotein cholesterol, HDL – high-density lipoprotein cholesterol, GFR – glomerular filtration rate; insignificant p-values were rounded up to two decimal places, significant p-values were rounded up to three decimal places.

we suggest initiating a randomized control trial of SVGs after cardiac surgery to address the issue of OCT-derived plaque morphology with respect to hypothetical clinical benefit in this therapeutically demanding group of patients.

This is a preliminary study that enrolled a relatively small number of patients. As it was performed in one center, although the researchers did not interfere with the management process, there exists a possibility of selection bias. Despite the fact that it was recently widely discussed and considered insignificant, since OCT is an invasive procedure there exists a theoretical possibility of iatrogenic damage of the vessel wall which might have influenced the results.

Table V. Clinical and imaging findings depending on tissue type according to OCT imaging

Parameter	ITR-0 (*n* = 38) Mean ± SD	ITR-1 (*n* = 5) Mean ± SD	*P*-value	FRB-0 (*n* = 41) Mean ± SD	FRB-1 (*n* = 2) Mean ± SD	*P*-value
EEM volume [mm]	125.7 ±54.1	111.1 ±70.6	0.59	126.2 ±55.8	77.1 ±10.6	0.23
Lumen volume [mm]	92.9 ±42.0	82.5 ±52.0	0.52	93.3 ±43.1	59.1 ±9.8	0.12
Min. av. lumen diameter [mm]	3.0 ±0.5	3.2 ±0.2	0.47	3.1 ±0.5	3.0 ±0.6	0.74
Min. lumen area [mm^2]	7.5 ±2.5	8.2 ±0.8	0.23	7.6 ±2.3	7.0 ±3.1	0.89
Min. lumen diameter [mm]	2.8 ±0.5	2.9 ±0.3	0.86	2.8 ±0.4	2.7 ±0.6	0.69
Plaque volume [mm]	32.8 ±13.9	28.5 ±18.8	0.36	33.0 ±14.3	17.9 ±0.8	0.12
Stenosis EEM [%]	15.1 ±2.5	14.4 ±1.1	0.53	15.1 ±2.4	13.5 ±0.7	0.36
Stenosis length [mm]	10.9 ±3.6	9.0 ±5.8	0.30	10.9 ±3.8	7.7 ±3.5	0.26
Stenosis reference (%)	3.3 ±5.0	0.6 ±1.3	0.14	3.0 ±4.9	2.5 ±3.5	1.00
Total lumen perimeter [mm^2]	112.7 ±39.9	97.2 ±61.7	0.45	112.7 ±42.5	74.0 ±11.6	0.21
Age [years]	68.9 ±7.2	75.8 ±5.7	0.047	69.5 ±7.5	74.5 ±2.1	0.35
Body surface area [m^2]	2.0 ±0.2	2.1 ±0.1	0.09	2.0 ±0.2	1.8 ±0.1	0.25
Body mass index [kg/m^2]	29.0 ±3.4	28.4 ±2.2	0.69	29.0 ±3.2	27.5 ±3.5	0.52
LVEF (%)	49.7 ±9.4	32.0 ±6.7	0.001	47.5 ±10.7	50.0 ±14.1	0.62
Troponin before [ng/l]	0.2 ±0.4	0.6 ±0.5	0.05	0.2 ±0.4	1.0 ±0.0	1.00
Troponin after [ng/l]	0.2 ±0.4	0.6 ±0.5	0.05	0.2 ±0.4	1.0 ±0.0	1.00
HGB [mg/dl]	15.7 ±7.5	14.7 ±1.6	0.77	15.7 ±7.2	12.8 ±1.3	0.58
WBC [× 10^3/µl]	6.9 ±1.6	7.3 ±1.7	0.69	7.0 ±1.7	6.7 ±0.0	0.57
PLT [× 10^3/µl]	201.6 ±55.3	192.2 ±18.4	0.84	201.1 ±52.9	186.5 ±48.8	0.61
TCH [mg/dl]	161.3 ±41.6	93.4 ±67.4	0.033	151.2 ±51.7	169.0 ±8.5	0.27
TG [mg/dl]	140.4 ±55.2	90.6 ±55.5	0.07	131.6 ±58.2	165.5 ±21.9	0.42
LDL [mg/dl]	93.1 ±37.6	75.4 ±12.1	0.27	90.4 ±36.7	96.0 ±4.2	0.33
HDL [mg/dl]	40.5 ±13.0	38.8 ±18.2	0.76	40.3 ±13.8	40.0 ±8.5	0.89
Creatinine [mg/dl]	1.0 ±0.3	1.2 ±0.2	0.16	1.1 ±0.3	0.8 ±0.0	0.31
GFR [ml/min/1.73 m^2]	73.6 ±17.4	36.0 ±29.8	0.010	68.9 ±23.2	69.0 ±4.2	0.64
Male, *n* (%)	31 (82)	4 (80)	0.60	35 (85)	0 (0)	0.036
Diabetes, *n* (%)	19 (50)	3 (60)	0.96	20 (49)	2 (100)	0.49
Hypertension, *n* (%)	32 (84)	5 (100)	0.79	35 (85)	2 (100)	0.64
Current smoking, *n* (%)	3 (8)	1 (20)	0.95	3 (7)	1 (50)	0.43

ITR-0/1 – intimal tearing or rupture absent/present, FRB-0/1 – tissue friability absent/present, EEM – external elastic membrane, min. – minimal, av. – average, LVEF – left ventricular ejection fraction, HGB – hemoglobin, WBC – white blood cells, PLT – platelets, TG – triglyceride, TCH – total cholesterol, LDL – low-density lipoprotein cholesterol, HDL – high-density lipoprotein cholesterol, GFR – glomerular filtration rate; insignificant p-values were rounded up to two decimal places, significant p-values were rounded up to three decimal places.

Conclusions

Stenotic lesions of the SVG had a higher incidence of LRP, TCFA, thrombus, and plaque within the valve compared to non-stenotic ones. Presence of ITR was associated with advanced age, deteriorated systolic function of the left ventricle and renal insufficiency. Decreased concentration of HDL was associated with higher occurrence of calcified and ruptured plaque.

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Conflict of interest

The authors declare no conflict of interest.

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