Original paper

Optical coherence tomography versus intravascular ultrasound for culprit lesion assessment in patients with acute myocardial infarction

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Abstract

Introduction: In patients with acute myocardial infarction (AMI) undergoing primary percutaneous coronary intervention (PCI) the implanted stent may not fully cover the whole intravascular ultrasound (IVUS)-derived thin-cap fibroatheroma (TCFA) related to the culprit lesion (CL).

Aim: Whether this phenomenon is more pronounced when optical coherence tomography (OCT) assessment of the CL is performed is not known.

Material and methods: Thus, we aimed to assess CLs in 40 patients with AMI treated with PCI, using VH (virtual histology)-IVUS and OCT before and after intervention. The results were blinded to the operator and PCI was done under angiography guidance.

Results: Uncovered lipid-rich plaques were identified in the stent reference segments of 23 (57.5%) patients: in 13 (32.5%) of them in the distal reference segment and in 19 (47.5%) of them in the proximal reference segment. In 9 of them (22.5%) lipid plaques were found in both reference segments. In 36 (90%) patients OCT confirmed lipid plaques identified as VH-derived TCFA by VH-IVUS in the reference segments of the stented segment. However, OCT confirmed that only in 2 (5%) patients were uncovered lipid plaques true TCFA as defined by histology. Comparing IVUS and OCT qualitative characteristics of the stented segments OCT detected more thrombus protrusions and proximal and distal stent edge dissections compared to IVUS (92.5 vs. 55%, $p = 0.001$; 20% vs. 7.5%, $p = 0.03$ and 25% vs. 5%, $p < 0.001$, respectively).

Conclusions: Due to its superior resolution, OCT identifies TCFA more precisely. OCT more often shows remaining problems related to stent implantation than IVUS after angiographically guided PCI.

Key words: percutaneous coronary intervention, acute myocardial infarction, optical coherence tomography, intravascular ultrasound, thin-cap fibroatheroma, culprit lesion.

Summary

In patients with acute myocardial infarction (AMI) undergoing primary percutaneous coronary intervention (PCI) the implanted stent may not fully cover the whole intravascular ultrasound (VH-IVUS)-derived thin-cap fibroatheroma (VH-TCFA) related to the culprit lesion (CL). Whether this phenomenon is more pronounced when optical coherence tomography (OCT) assessment of the CL is performed is not known. Thus we aimed to assess CLs in 40 patients with AMI treated with PCI, using VH-IVUS and OCT before and after intervention. Due to its superior resolution, OCT identifies thin-cap fibroatheroma more precisely. OCT more often shows remaining problems related to stent implantation than IVUS after angiographically guided PCI.

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Introduction

Atherosclerotic plaque rupture resulting in thrombus formation most commonly leads to acute coronary artery occlusion resulting in acute myocardial infarction (AMI) [1, 2]. However, postmortem assessments revealed that the occlusion is mainly composed of a thrombus and the plaque rupture is located proximal or distal to the site of occlusion and is not always lumen compromising [3, 4]. Therefore, in the case of treating the culprit lesion with stenting, incomplete stent coverage of the true culprit lesion (culprit of the culprit lesion (CL) or the site of plaque rupture) may occur, when only the occlusion or the minimum lumen area (MLA) site is treated under angiographic guidance. This could be one of the possible mechanisms responsible for future cardiac events.

Using intravascular ultrasound (IVUS), it was previously shown that the stent does not fully cover the whole virtual histology (VH)-IVUS-derived thin-cap fibroatheroma (VH-TCFA) related to the CL in patients with AMI undergoing primary percutaneous coronary intervention (PCI) with the optimal angiographic result [5, 6]. With its greater resolution, optical coherence tomography (OCT) allows even more precise visualization and quantification of TCFA than VH-IVUS [7].

Aim

We aimed to compare OCT and VH-IVUS assessment of the CL after primary PCI in patients with AMI.

Material and methods

The present study was a two-center, prospective, observational registry. The study protocol was approved by the Institutional Review Boards and conformed to the statute of the Declaration of Helsinki. All patients provided written informed consent before enrollment. Patients aged > 18 years with uncomplicated AMI (ST- and non-ST segment elevation myocardial infarction, 20 subjects in each group) within 12 h after onset of symptoms qualifying for emergent PCI were eligible. Patients were not eligible if angiography showed left main coronary artery stenosis of > 50%, baseline thrombolysis in myocardial infarction (TIMI) flow < 3 in the infarct-related artery (IRA) (restoration of TIMI-3 flow either spontaneously, after wire insertion, aspiration thrombectomy or predilatation with a 2.0 mm balloon was allowed), or if the coronary anatomy of the culprit vessel was inappropriate for IVUS and OCT assessment or stent implantation. CLs were de novo, non-ostial, and without heavy calcification in the proximal or middle segment of the patent IRA with a reference vessel diameter of ≥ 2.5 mm by visual estimation. VH-IVUS and OCT pullbacks were conducted before stent implantation. VH-IVUS derived TCFA was defined as a focal necrotic core-rich lesion without evident overlying fibrous tissue. OCT-derived TCFA was defined as lipid-rich plaque (lipid plaque with a lipid arc > 90°) with a fibrous cap < 65 µm. Stent length and diameter selection was based on angiography alone and was followed by direct stent implantation with post-dilation if required to achieve the optimal angiographic result (residual angiographic diameter stenosis of < 20% and TIMI flow 3). After finishing the procedure, VH-IVUS and OCT pullbacks were repeated. Operators performing PCI were blinded to VH-IVUS and OCT findings and therefore these findings did not impact PCI, which was carried out according to the standard practice of the center. IVUS pullbacks were performed with the Volcano S5 system and the Eagle-Eye Gold catheter (Volcano Corporation, Rancho Cordova, CA, USA). The automated pullback was performed at the speed of 0.5 mm/s. The scan area was from 10 mm distal to the CL to the aorto-ostial junction. All pullbacks were stored on a compact disc for off-line analysis. Radiofrequency backscatter data were collected simultaneously and triggered by the R-wave peak of the patient's electrocardiogram using a dedicated IVUS console (Rancho Cordova, California). The region of interest was defined in each vessel as the stented lesion plus 10 mm proximal and distal to the edges of the stent. Each region of interest imaged by IVUS and VH-IVUS was analyzed by 2 different analysts to address inter- and intraobserver variability. Planar and volumetric IVUS and VH-IVUS analyses were performed according to established standards [8, 9]. The IVUS analysis was performed using echoPlaque 4 software (INDEC Medical Systems, Santa Clara, California). The VH-IVUS analysis was performed using pcVH 2.2 and qVH software (Rancho Cordova, California) for tissue characterization and advanced analysis, respectively. IVUS and VH-IVUS data were analyzed by 2 independent analysts blinded to the clinical data and procedural information, and all analyses were reviewed by a single independent reviewer. Overall, inter- and intraobserver variability for TCFA detection showed a good intraclass correlation (κ = 0.933 and 0.894, respectively). Two OCT pullbacks were performed. The first pullback was for the assessment of the CL and its reference segments with TIMI grade 3 flow in IRA and the administration of 250 µg of intracoronary nitroglycerine. The second pullback was performed after stent implantation for the assessment of the stented segment and its reference segments. OCT pullbacks were performed with the OCT Ilumien system and the DragonFly OCT catheter (St. Jude Medical, St. Paul, MN, USA). For effective clearance of blood from the imaging field, angiographic contrast medium was injected with an automated power injector. Specifically, injection of 14 ml of contrast at a rate of 4 ml/s and the pressure of 400 PSI sufficed to achieve imaging time of 2–3 s consistently in all the major coronary arteries. Pullback speed was 20 mm/s. The scan area was 5.4 cm. All pullbacks were stored on a compact disc for off-line analysis. The analysis of OCT pullbacks

was performed in an independent core laboratory by two experienced analysts blinded to clinical data, IVUS and angiographic images. The intra- and interobserver variability showed a good correlation (κ = 0.912). The analysis was performed using the Ilumien off-line analysis workstation software (St. Jude Medical, St. Paul, MN, USA). The analysis was performed following the Consensus Standards for Acquisition, Measurement, and Reporting of Intravascular Optical Coherence Tomography Studies [9]. Inter- and intraobserver variability for OCT-derived TCFA detection showed a good intraclass correlation (κ = 0.912 and 0.889, respectively). Off-line qualitative and quantitative coronary angiographic (QCA) analysis was performed according to the well-established protocol [10, 11]. QCA analysis was performed using the Sanders Data Systems QCAPlus software (Palo Alto, California) by an experienced analyst blinded to clinical data and procedural information.

Statistical analysis

Statistical analysis was performed using SPSS Statistics for Windows v17.0 software (SPSS Inc., Chicago, IL, USA). Continuous variables are expressed as mean ± standard deviation (SD) or median (interquartile range), as appropriate. The categorical data were compared using Fisher's exact test or the χ^2 test. Normally distributed data were compared using Student's *t*-test and non-normally distributed data using the Mann-Whitney test. A *p*-value < 0.05 was considered statistically significant.

Results

Forty patients were eligible, and 40 CLs were analyzed in this study. Baseline clinical characteristics and procedural data are shown in Table I. Final TIMI grade 3 flow was achieved in all lesions. No death, reinfarction, or repeat interventions were reported during in-hospital, at 30-day, and at 1-year follow-up. Results of quantitative and qualitative coronary angiography assessment before stent implantation and after PCI are presented in Table II. Detailed IVUS and OCT lesion characteristics are presented in Tables III and IV. Uncovered lipid plaques were identified in the stent reference segments of 23 (57.5%) patients, in 13 (32.5%) of them in the distal reference segment and in 19 (47.5%) of them in the proximal reference segment (in 9 (22.5%) of these patients lipid plaques were found in both reference segments). In 36 (90%) patients, OCT confirmed lipid plaques identified as VH-TCFA by VH-IVUS in the reference segments of the stented segment of the IRA. However, OCT confirmed that in only 2 (5%) patients with AMI uncovered lipid plaques were OCT-derived TCFA. MLA was lower when assessed with OCT (1.63 ±1.6 vs. 2.78 ±1.03 mm^2, p = 0.03). Plaque rupture proximal to MLA was diagnosed more frequently with OCT (45% vs. 27.5%, p = 0.02). Distance of plaque rupture from MLA was lower when assessed with OCT (4.61 ±4.06 vs. 7.19 ±6.85 mm, p = 0.01).

Comparing IVUS and OCT qualitative characteristics of the stented segments OCT detected more thrombus protrusions and proximal and distal stent edge dissections compared to IVUS (92.5% vs. 55%, p = 0.001; 20% vs. 7.5%, p = 0.03 and 25% vs. 5%, p < 0.001, respectively). Table V depicts detailed data of plaque types in stent reference segments as identified by VH-IVUS compared to OCT and the OCT cap thickness in lipid plaques in 40 patients with AMI.

Discussion

In the present study, IVUS VH-IVUS and OCT images, blinded to the operator, were used to assess *in vivo* longitudinal distribution of culprit lesion plaque components before PCI and to evaluate stent coverage of these

Table I. Baseline characteristics and procedural data (n = 40)

Variable	Results
Age, mean ± SD [years]	58 ±12
Men	21 (52.5%)
Arterial hypertension	12 (30%)
Hypercholesterolemia	10 (40%)
Current smoking	4 (10%)
Diabetes mellitus	5 (12%)
Peripheral artery disease	0
Chronic renal failure	1 (2.5%)
Previous coronary artery bypass grafting	0
Previous stroke	2 (5%)
Positive family history of coronary artery disease	5 (12%)
Previous myocardial infarction	0
Previous percutaneous coronary intervention	1 (2.5%)
Infarct-related artery:	
Left anterior descending artery	16 (40%)
Left circumflex artery	9 (22.5%)
Right coronary artery	15 (37.5%)
Thrombus aspiration due to baseline TIMI flow < 3	17 (42.5%)
Use of glycoprotein IIb/IIIa inhibitor	3 (7.5%)
Balloon predilatation	16 (40%)
Number of deployed stents, mean ± SD	1.49 ±0.65
Stent post-dilatation	19 (47.5%)
Bare metal stent	12 (30%)

SD – standard deviation.

Table II. Quantitative and qualitative coronary angiography prior to and after stent implantation (n = 40)

Variable	Results
Prior to stenting:	
Quantitative coronary angiography:	
Lesion length, mean ± SD [mm]	15.17 ±6.5
Reference vessel diameter, mean ± SD [mm]	3.14 ±0.5
Minimum lumen diameter, mean ± SD [mm]	0.51 ±0.47
Diameter stenosis (%), mean ± SD	84 ±22
Qualitative coronary angiography:	
TIMI flow 3	9 (22.5%)
TIMI flow 2	15 (37.5%)
TIMI flow 1	4 (10%)
TIMI flow 0	12 (30%)
Angiographic presence of thrombus	24 (60%)
TIMI thrombus grade 0	13 (32.5%)
TIMI thrombus grade 1	3 (7.5%)
TIMI thrombus grade 2	3 (7.5%)
TIMI thrombus grade 3	5 (12.5%)
TIMI thrombus grade 4	6 (15%)
TIMI thrombus grade 5	10 (25%)
Post stenting:	
Quantitative coronary angiography:	
Stent length, mean ± SD [mm]	23.54 ±6.17
Stent diameter, mean ± SD [mm]	3.47 ±0.57
Proximal reference lumen diameter, mean ± SD [mm]	3.59 ±0.53
Distal reference lumen diameter, mean ± SD [mm]	3.08 ±0.46
Minimum lumen diameter, mean ± SD [mm]	3.0 ±0.46
Diameter stenosis (%), mean ± SD	7 ±4
Stent-length to lesion-length ratio	1.55
Qualitative coronary angiography:	
TIMI 3 flow	36 (90%)
TIMI 2 flow	4 (10%)
Angiographic presence of thrombus	1 (2.5%)
Residual dissection	3 (7.5%)
Angiographic signs of spasm	0
Angiographic signs of distal embolisation	1 (2.5%)
Angiographic complications after IVUS/OCT	0

IVUS – intravascular ultrasound, OCT – optical coherent tomography, SD – standard deviation, TIMI – thrombolysis in myocardial infarction.

components after angiography-guided emergent PCI as well as stent strut assessment in patients with AMI. The main conclusions were as follows: (1) OCT confirmed that in only 5% of patients with AMI uncovered lipid plaques were true TCFA as defined by histology; (2) in 57% of the angiography-guided PCIs in patients presenting with AMI, stent placement missed coverage of the whole length of the culprit-related VH-TCFA; and (3) OCT detected more malappositions, thrombus protrusions and proximal and distal stent edge dissections than IVUS. The culprit-of-the-culprit concept highlights that minimal lumen area site (MLA) (or angiographic CL) is very rarely the site of greatest instability (rather the site of greatest thrombus accumulation) while the site of maximum necrotic core or the site of VH-TCFA, the locations with the largest amount of necrotic tissue or the areas of greatest instability, are located a few millimeters proximal to or distal from the MLA [3, 12–14]. In our previous study longitudinal geographical miss (GM) was confirmed in 35% of patients with non-ST elevation myocardial infarction (NSTEMI) and in 50% of patients with ST elevation myocardial infarction (STEMI) [5, 6]. This means that in angiographically guided stent implantation stent length and/or longitudinal positioning was not accurate enough to cover fully all VH-TCFA in the treated segment of the IRA. In our current study we confirmed the same phenomenon in patients with acute coronary syndrome who again underwent angiographically guided PPCI with stent implantation. Uncovered VH-TCFAs were identified in stent reference segments of more than half of AMI patients, in one third of them in the distal reference segment and in almost half of them in the proximal reference segment (in 9 (22.5%) of these patients lipid plaques were found in both reference segments). Uncovered VH-TCFAs might cause problems and worsen the long-term outcomes for patients with AMI. The long-term mortality is reported at over 10%; in patients with NSTEMI it is twice as high in a 4-year follow-up period [15]. In this respect Stone *et al.* showed in their Predictors of Response to Cardiac Resynchronization Therapy (PROSPECT) trial a positive correlation of VH-TCFA occurrence and later non-restenotic and total major adverse cardiac events [16]. Calvert *et al.* emphasized the biological importance of uncovered VH-TCFAs and the high risk of adverse events associated with them [17]. Furthermore, Sakurai *et al.* and Liu *et al.* showed that an uncovered stent edge plaque burden > 50% was an independent predictor of marginal restenosis after DES implantation [18, 19]. Also, plaque disruption in the reference segments of the treated segment of IRA within 2 mm of the stent edge has been demonstrated to correlate with fatal late stent thrombosis [20]. These studies confirm the importance of the identification of vulnerable plaques in the treated segments of IRAs in patients with AMI. Compared to IVUS, OCT proved to be more accurate in the detection of MLA and plaque rupture sites (PRS). Compared to the study conducted by Toutouzas *et al.* we identified fewer PRS in both groups. However, the results are consistent with their conclusions that in many patients with AMI (especially STEMI) PR occurs in sites other than the MLA [21]. Although IVUS consistently showed hypoechoic, throm-

Table III. Quantitative and qualitative ultrasound and optical coherence tomography characteristics

Variable	IVUS data	OCT data	*P*-value
Culprit lesion:			
Lesion length, mean ± SD [mm]	24.2 ±10.28	22.52 ±9.18	0.53
Minimum lumen diameter, mean ± SD [mm]	1.61 ±0.25	1.14 ±0.55	0.21
Minimum lumen area, mean ± SD [mm^2]	2.78 ±1.03	1.63 ±1.6	0.03
Maximum plaque burden, mean ± SD (%)	80.18 ±7.94	NA	–
Vessel area at minimum lumen area, mean ± SD [mm^2]	14.05 ±5.61	NA	–
Distal reference segment:			
Minimum lumen diameter, mean ± SD [mm]	2.26 ±0.65	2.25 ±0.89	0.65
Maximum lumen area, mean ± SD [mm^2]	8.29 ±3.8	7.52 ±4.06	0.72
Minimum lumen area, mean ± SD [mm^2]	5.01 ±2.91	4.91 ±2.08	0.38
Proximal reference segment:			
Minimum lumen diameter, mean ± SD [mm]	2.58 ±0.58	2.74 ±0.76	0.45
Maximum lumen area, mean ± SD [mm^2]	11.64 ±4.05	11.33 ±5.32	0.59
Minimum lumen area, mean ± SD [mm^2]	6.94 ±2.81	7.58 ±3.92	0.72
Qualitative assessment of culprit lesion:			
Presence of MLA	38 (95%)	39 (97.5%)	0.82
Presence of plaque rupture	15 (37.5%)	18 (45%)	0.49
Presence of thrombus	NA	31 (77.5%)	–
Presence of thrombus at MLA	NA	30 (75%)	–
Thrombus length, mean ± SD [mm]	NA	6.9 ±4.02	–
Plaque rupture proximal to MLA	11 (27.5%)	18 (45%)	0.02
Plaque rupture distal to MLA	4 (10%)	0	0.06
Distance of plaque rupture from MLA, mean ± SD [mm]	7.19 ±6.85	4.61 ±4.06	0.01
Distance of maximal necrotic core from MLA, mean ± SD [mm]	5.14 ±4.39	NA	–
Maximal necrotic core proximal to MLA	27(67.5%)	NA	–
Maximal necrotic core distal to MLA	11(27.5%)	NA	–
Most unstable plaque type in the culprit lesion:			
Lipid	NA	30 (75%)	–
Fibrous	NA	6 (15%)	–
Calcific	NA	2 (5%)	–
Minimum cap thickness, mean ± SD [µm]	NA	121 ±47	–
Plaque type at the plaque rupture:			
Lipid	NA	11 (27.5%)	–
Empty cavity	NA	9 (22.5%)	–
Fibrous	NA	0	–

bus-like structures moving in the lumen in the regions of CLs, which were observed consistently on VH-IVUS as yellow-greenish masses, there are no official criteria for the IVUS detection of thrombus. OCT is a tool that can not only identify thrombi, but also differentiate between the white and the red types [9]. Since OCT is capable of identifying and quantifying thrombus, it is a surprise that no proper thrombus scoring system has been established yet and cannot be found in the literature. In our study we analyzed in how many consecutive OCT frames and their quadrants the thrombus was identified. IVUS with its relatively low resolution is also not the best imaging modality for the detection of these phenomena as well as minor stent edge dissections or stent malappositions. Kubo *et al*. showed that OCT has a better potential for the detection of stent edge dissections (40% vs. 16%, $p = 0.005$), tissue protrusions (58% vs. 20%, $p < 0.001$) and stent malappositions (47% vs. 8%, $p < 0.001$) after stent implantation compared to IVUS [22]. Considering the fact that any kind of GM is associated with an increased risk for target vessel revascularization and myocardial infarction at 1 year as described earlier, stent

Table IV. Stented segment ultrasound and optical coherent tomography characteristics

Variable	IVUS data	OCT data	*P*-value
Quantitative characteristics of the stented segment:			
Stent length, mean ± SD [mm]	24.64 ±9.89	24.12 ±9.79	0.84
Minimum lumen area, mean ± SD [mm^2]	6.94 ±6.59	6.57 ±6.65	0.79
Maximum stent area, mean ± SD [mm^2]	10.93 ±4.07	11.71 ±3.99	0.62
Minimum stent area, mean ± SD [mm^2]	7.02 ±2.42	6.9 ±2.14	0.87
Malapposed struts, mean ± SD	22.56 ±32.4	33.13 ±45.3	0.06
Maximum malapposition distance, mean ± SD [mm]	–	0.4 ±0.28	–
Malapposition	9 (22.5%)	12 (30%)	0.16
Thrombus protrusion	22 (55%)	37 (92.5%)	0.001
Proximal edge dissection	3 (7.5%)	8 (20%)	0.03
Distal edge dissection	2 (5%)	10 (25%)	< 0.001
Proximal reference segment plaque type:			
Healthy vessel	NA	1 (2.5%)	–
Fibrous	NA	9 (22.5%)	–
Calcific	NA	3 (7.5%)	–
Lipidic	NA	19 (47.5%)	–
Cap thickness if lipidic, mean ± SD [µm]	NA	171 ±82	–
Distal reference segment plaque type:			
Healthy vessel	NA	3 (7.5%)	–
Fibrous	NA	17 (42.5%)	–
Calcific	NA	3 (7.5%)	–
Lipidic	NA	13 (32.5%)	–
Cap thickness if lipidic, mean ± SD [µm]	NA	143 ±65	

IVUS – intravascular ultrasound, OCT – optical coherent tomography.

malappositions in AMI patients undergoing angiographically guided PPCI are a serious problem. Thrombus protrusions were identified by OCT in 92% of AMI patients. The clinical implications of thrombus protrusion, tissue prolapse, stent malappositions and stent edge dissections – usually small features – remain unclear [23]. However, the results of our study suggest another and a more important issue, which can be due to the superior resolution of OCT may be answered in the future and can impact the way we understood VH-TCFAs and their significance in guiding primary PCI. As mentioned earlier, different investigators have confirmed a positive correlation of VH-TCFA occurrence and later non-restenotic and total major adverse cardiac events [16, 18, 19]. The problem is the discrepancy between the definition of histologically derived TCFA, which includes: large lipid pool, a thin fibrous cap (≤ 65 µm) and activated macrophages near the fibrous cap on one hand and the definition of VH-TCFA on the other hand, which is limited by the relatively low resolution of this imaging modality (150–250 µm) and includes: ≥ 40% PB, consisting of ≥ 10% NC found in three consecutive frames in VH-IVUS pullback, with no fibrous tissue between the NC and the lumen. The latter practically means that all plaques with fibrous caps with the thickness ≤ 150 µm and which also in other respects fit the definition of VH-TCFA would be identified as VH-TCFA on VH-IVUS. However, all of them with fibrous caps with thicknesses between 65 and 150 µm would not fit the histological definition of TCFA and would therefore in histological terms not be considered as vulnerable plaques prone to spontaneous PR leading to thrombotic severe stenosis or occlusion of the IRA and to acute coronary syndrome [2, 3], and can be labeled as false positive TCFAs due to the resolution limitation of the method.

Yonetsu *et al.* reached a similar conclusion in their study, which showed that *in vivo* critical cap thicknesses were < 80 µm and < 188 for most representative fibrous cap thicknesses in non-ruptured lipid plaques [24]. This has to be confirmed by larger randomized trials; however, it suggests the need for changing the definition of a VP for *in vivo* assessment and that OCT has the potential to change primary PCI strategies in patients with acute coronary syndrome.

Several limitations in the present study should be appreciated. The main limitation was a relatively small number of patients enrolled. Second, the nature of enrollment was prospective. However, non-consecutive series

Table V. Plaque types in stent reference segments as identified by VH-IVUS compared to OCT and the OCT cap thickness in lipidic plaques in patients with acute myocardial infarction

No.	Distal reference segment			Proximal reference segment		
	VH-IVUS	OCT	Cap thickness [µm]	VH-IVUS	OCT	Cap thickness [µm]
1	CaTCFA	Lipidic	130	FCa	NA	/
2	AIT	Healthy vessel	/	PIT	Fibrous	/
3	AIT	Healthy vessel	/	TCFA	Lipidic	200
4	PIT	Fibrous	/	CaTCFA	Lipidic	180
5	AIT	NA	/	CaTCFA	Lipidic	170
6	TCFA	Lipidic	120	TCFA	Lipidic	100
7	NA	Fibrous	/	NA	Lipidic	180
8	FCa	Lipidic	120	TCFA	Lipidic	80
9	TCFA	Lipidic	140	PIT	Fibrous	/
10	PIT	Fibrous	/	CaTCFA	Lipidic	450
11	CaTCFA	Lipidic	160	CaTCFA	Lipidic	320
12	CaTCFA	Calcific	/	NA	NA	/
13	AIT	Fibrous	/	PIT	Fibrous	/
14	PIT	Fibrous	/	AIT	Healthy vessel	/
15	CaTCFA	Lipidic	90	TCFA	Lipidic	180
16	AIT	Fibrous	/	AIT	Fibrous	/
17	CaTCFA	Lipidic	170	NA	NA	/
18	FCa	Calcific	/	NA	Calcific	/
19	FA	Lipidic	100	TCFA	Lipidic	100
20	CaTCFA	Calcific	/	TCFA	Calcific	/
21	CaTCFA	Lipidic	180	CaTCFA	Lipidic (True TCFA)	60
22	PIT	NA	/	AIT	NA	/
23	AIT	NA	/	CaTCFA	Lipidic	85
24	AIT	Fibrous	/	NA	NA	/
25	NO PLAQUE	Fibrous	/	NA	Fibrous	/
26	PIT	Fibrous	/	PIT	Fibrous	/
27	NA	Fibrous	/	NA	Lipidic	220
28	FCa	Lipidic (True TCFA)	30	NA	Lipidic	150
29	PIT	Fibrous	/	PIT	Fibrous	/
30	TCFA	Lipidic	300	TCFA	Lipidic	140
31	CaTCFA	Lipidic	190	FCa	Calcific	/
32	NA	NA	/	NA	NA	/
33	PIT	Fibrous	/	NA	NA	/
34	FCa	Fibrous	/	CaTCFA	Lipidic	130
35	CaTCFA	Lipidic	90	CaTCFA	Lipidic	190
36	AIT	Fibrous	/	NA	NA	/
37	AIT	Healthy vessel	/	FA	Lipidic	200
38	PIT	Fibrous	/	PIT	Fibrous	/
39	AIT	Fibrous	/	TCFA	Lipidic	150
40	AIT	Fibrous	/	PIT	Fibrous	/

AIT – adaptive intimal thickening, CaTCFA – calcified thin-cap fibroatheroma, FA – fibroatheroma, FCa – fibrocalcific plaque, PIT – pathological intimal thickening, NA – not applicable, OCT – optical coherence tomography, TCFA – thin-cap fibroatheroma, VH-IVUS – intravascular ultrasound with virtual histology.

of subjects who met eligibility criteria and consented to the study were enrolled. The third limitation was the limitation of the VH-IVUS as a method – its relatively low resolution, which does not allow, as discussed above, proper identification of only the true TCFAs as per the histology-derived definition. Fourth, IVUS and VH-IVUS could not identify thrombus. Finally, thrombus aspiration, if performed, could affect qualitative characteristics of the CL, especially regarding thrombus assessment. Follow-up clinical data were also not assessed.

Conclusions

Due to its superior resolution, OCT identifies TCFA more precisely. OCT more often shows remaining problems related to stent implantation than IVUS after angiographically guided PCI.

Conflict of interest

The authors declare no conflict of interest.

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