intervention (PCI) for ST-segment elevation myocardial infarction (STEMI). Incomplete stent apposition (ISA) and the absence of strut endothelialization might be linked to stent thrombosis. STEMI might have a higher risk of thrombosis.

Methods: An early OCT evaluation of stents that were deployed in culprit lesions for STEMI was performed at 9- 18days. The primary end-point was the percentage of incomplete stent apposition and that of struts covered with a thrombus. Secondary end-points were the percentage of neointima covered struts.

Results: 20 lesions in 20 patients (4,614 struts) were analyzed. Median follow-up time was 12 (range 9 to 18) days. The frequency of incomplete stent apposition was 14%. The frequency of stent covered with thrombus was 11%. The percentage of incomplete stent apposition covered with thrombus and that of well apposition covered with thrombus were 73% and 27% (p < 0.05).

Conclusions: Stents implanted for STEMI had a high frequency of incompletely apposed struts at early phase using OCT. These struts of incomplete stent apposition were obviously coated a thrombus compared with apposition struts at the early stage.

TCT-393

Relation between the SYNTAX score and culprit vessel vulnerability in non-ST-segment elevation acute coronary syndrome-An optical coherence tomography study

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Background: High SYNTAX score (SS) has been associated with increased incidences of major adverse cardiac events, even after successful percutaneous coronary intervention (PCI) for culprit lesions. optical coherence tomography (OCT) imaging is a feasible technique for the identification of thin-cap fibroatheroma (TCFA) in vivo and may provide a better understanding of vulnerable plaques. The aim of this study was to examine the relation between SS and culprit and non-culprit lesion morphology as evaluated by OCT in patients with non-ST-segment elevation acute coronary syndrome (NSTEACS).

Methods: Culprit plaques in 144 patients with NSTEACS were interrogated by OCT before PCI. Non-culprit plaques were defined as independent plaques and had to be situated at least 5 mm from the stent edge in treated or non-treated vessels. A total of 81 non-culprit plaques in 81 patients were analyzed by OCT.

Results: Patients were classified into 3 groups according to the tertile of SS: 48 patients with an SS of <9; an intermediate SS group, 47 patients with an SS of ≥ 9 to < 16; and a high SS group, 49 patients with an SS of ≥ 16 . The high SS group had a significantly lesser minimum FCT in the culprit lesion as compared with the respective values in the intermediate SS group and low SS group (high vs. intermediate vs. low SS group; 60 vs. 70 vs. 77 µm, p< 0.01). Moreover, the minimum FCT in the intermediate group and low SS group (high vs. intermediate group and low SS group; 90 vs. 100 vs. 137 µm, p=0.03). The frequencies of lipid-rich plaque (90% vs. 85% vs. 68%, p=0.02), TCFA (73% vs. 40% vs. 40%, p< 0.01), plaque rupture (63% vs. 47% vs. 31%, p< 0.01), and multiple plaque ruptures in the culprit vessel (24% vs. 9% vs. 2%, p< 0.01) were significantly higher in the high SS group.

Conclusions: In patients with NSTEACS, patients with high SS may have heightened plaque vulnerability in culprit as well as non-culprit lesions. Our results are considered to highlight the importance of complete revascularization in patients with high SS.

TCT-394

Optical Coherence Tomography- versus Angiography-guided Percutaneous Coronary Intervention with Biolimus-eluting Stent Implantation in Patients with Myocardial Infarction: Dynamic Malapposition Patterns and Strut Coverage at 6 Months. The OCTACS Trial

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Background: Incomplete strut coverage after drug-eluting stent (DES) implantation has been identified as an important predictor of late stent thrombosis. Percutaneous coronary intervention (PCI) using DES in the setting of myocardial infarction causes a higher incidence of acute incomplete stent apposition (ISA), which may contribute to delayed healing. Guidance with optical coherence to-mography (OCT) may lower the incidence of acute ISA and thereby provide better strut coverage. We assess the proportion of uncovered struts 6 months after OCT- versus angio-guided implantation of the Biolimus-eluting Nobori stent (BES) (Terumo) with biodegradable polymer in patients with Non-ST segment Elevation Myocardial Infarction.

Methods: The OCTACS trial enrolled 100 patients. After obtainment of an optimal angiographic result, patients were randomized 1:1 to either (1) OCT- or (2) angio-guided PCI. OCT was performed in both groups, and the operator was blinded to the OCT imaging in group 2. OCT-criteria indicating further intervention in group 1 were: Stent under expansion, acute ISA, significant edge dissection and/or significant residual stenosis. If criteria were met, additional balloon dilatation(s) and/or stenting was performed followed by a final OCT. Primary endpoint is difference in proportion of uncovered struts at 6 months.

Results: Baseline characteristics were balanced between the OCT- vs. the angioguided group, including mean age (61.8 ± 9.4 years vs. 62.6 ± 11.0 years, p=0.68, respectively). Further intervention was done in 46% of the OCT-guided patients, and maximal balloon pressures were significantly higher (16.8 ± 3.9 atm. vs. 15.0 ± 2.6 atm., p<0.05), and procedure- and flouro times were substantially longer (46.0 (16.0-125.0) min. vs. 34.0 (17.0-99.0) min., p<0.05 and 11.8 ± 7.2 min. vs. 8.4 ± 5.9 min., p<0.05, respectively). Six months difference in proportion of uncovered struts and dynamic ISA patterns will be presented at TCT 2014.

Conclusions: OCT guided BES implantation might contribute to better strut coverage by reducing the incidence of acute ISA.

TCT-395

Serial Optical Coherence Tomography Evaluation 6-, 12- and 24-month Follow-up after Nobori Biolimus A9-eluting Stent Implantation

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Background: Nobori is a novel biolimus A9-eluting stent (BES) coated with a biodegradable polymer only on the abluminal side. Long-term vessel healing after BES deployment remains unclear.

Methods: Sixteen BES implanted in de novo coronary lesions of 13 patients were serially evaluated using optical coherence tomography (OCT) at 6, 12 and 24 months post-stenting. Average neointimal thickness, uncovered struts and neo-intimal unevenness score (NUS: each cross section as maximum neointimal thickness in one cross section divided by the average neointimal thickness of the same cross-section) were manually measured. In addition, the percentage of struts with peri-strut low intensity area (PLIA: a region around stent struts with a homogenous lower intensity appearance than surrounding tissue, which is suggestive of fibrin deposition or impaired neointimal addition, thrombi and atherogenic neointima (AN: a neointima containing a diffuse border and poorsignal region with invisible struts underneath due to marked signal attenuation)

Results: There was a significant increase in neointimal thickness $(78\pm22\mu m \text{ to } 86\pm25\mu m \text{ to } 110\pm32, p=0.001)$ from the 6- to the 24-month follow-up, without a significant decrease in mean lumen area (5.95 ± 1.76 mm2 to 5.66 ± 1.73 mm2 to 5.46 ± 1.88 , p=0.74). The incidences of uncovered struts and NUS significantly decreased from the 6- to the 24-month ($4.22\pm3.84\%$ to $1.43\pm1.64\%$ to 0.40 ± 0.81 , p=0.001 and $2.02\pm0.19\%$ to $1.89\pm0.22\%$ to 1.77 ± 0.21 , p=0.006, respectively). % strut with PLIA decreased significantly during the follow-up ($5.26\pm4.85\%$ to $3.49\pm4.12\%$ to 0.01 ± 0.01 , p=0.001) and thrombi also numerically decreased (7% to 0% to 0%, p=0.37). There observed no AN in the current study subjects.

Variable	6 months (n=16)	12 months (n=16)	24 months (n=16)	P for ANOVA
Mean neointimal thickness (µm)	78 ± 22	86 ± 25	110 \pm 32*	0.005
Frequency of uncovered struts (%)	4.22 ± 3.84	$\textbf{1.43} \pm \textbf{1.64}^{\star}$	$\textbf{0.40} \pm \textbf{0.81*}$	0.001
Mean lumen area (mm ²)	$\textbf{5.95} \pm \textbf{1.76}$	5.66± 1.73	$\textbf{5.46} \pm \textbf{1.88}$	0.74
Mean minimal lumen area (mm ²)	4.23 ± 1.89	4.15± 1.80	$\textbf{4.13} \pm \textbf{1.87}$	0.98
Mean stent area (mm ²)	6.16 ± 1.65	$\textbf{6.16} \pm \textbf{1.74}$	$\textbf{6.20} \pm \textbf{1.85}$	0.99
Neointimal unevenness score	$\textbf{2.02} \pm \textbf{0.19}$	$\textbf{1.89} \pm \textbf{0.22}$	$\textbf{1.77} \pm \textbf{0.21*}$	0.006
Frequency of strut with PLIA (%)	5.26 ± 4.85	3.49 ± 4.12	$\textbf{0.01} \pm \textbf{0.01}^{***}$	0.001
Frequency of stent with thrombi (n, %)	2 (7)	0 (0)	0 (0)	0.37
Frequency of stent with AN (n, %)	0 (0)	0 (0)	0 (0)	

PLIA, peri-strut low-intensity area; AN, atherogenic neointima.

Data are presented as mean \pm SD or percentages of patients.

* p < 0.05 vs. 6 months (Tukey), ** p < 0.05 vs. 12 months (Tukey).

Conclusions: Biodegradable polymer coated BES achieved acceptable vessel healing without atherogenic change at 24-month post implantation.

TCT-396

Head-to-Head Comparison of Automated versus Manual Detection for Lumen Contour and Stent Struts in Optical Coherence Tomography Analysis

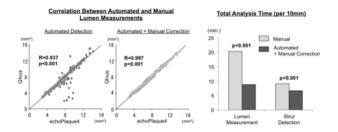
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Background: This study aimed to assess the accuracy and usefulness of automated lumen and stent strut detection algorithms (QIvus: Medis Medical Imaging Systems) in optical coherence tomography (OCT) analysis.

Methods: Automated detection algorithms were tested in 713 frames (5824 struts) obtained from 11 patients with Fourier-domain OCT imaging at post-stent implantation (length: 25.7 ± 11.0 mm, frame interval: 0.4 mm). The automated analysis results were compared with manual analysis performed by an expert physician using a conventional off-line analysis system (echoPlaque4, Indec Systems).

Results: In paired-frame analysis, automated lumen measurements showed good agreement with manual lumen area tracings (R=0.937, p< 0.001), except for several frames with significant underestimation resulted from residual blood or wire artifact within the lumen. Automated strut detection also showed high sensitivity (87.2±14.5%) with a relatively low false-detection rate (23.5±16.5%). In per-segment analysis, mean lumen area of the stent segment was comparable between automated and manual tracings (7.43±2.53 vs 7.45±2.46 mm, p=0.72); minimum lumen area by automated analysis was reasonably accurate (< 6% error) in 72.7% of the cases. Total analysis time (including automated analysis plus, if needed, manual correction) was significantly shorter for lumen measurements and strut detection (p< 0.001 for both) than manual analysis.

Conclusions: Despite the remaining need for some manual corrections, automated lumen and stent strut detection algorithms developed for OCT can facilitate rapid assessment of the stented coronary artery.



TCT-397

Estimation of Plaque Instability by Comparing Culprit versus Non-culprit Lesion Characteristics by OCT in Patients presenting with ACS

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Background: Identification of coronary plaque leading to ACS may be useful to improve clinical outcome of patients susceptive to atherosclerosis.

Methods: Consecutive ACS cases treated with OCT-guided PCI within 24 hours of arrival at our hospital from April 2013 to March 2014 were included.

Results: Detailed analysis was performed in STEMI (10), NSTEMI (9), and unstable angina (7) patients. The reasons for ACS determined by OCT were plaque rupture (46%), erosion (15%), calcified nodule (8%), and other (hemorrhage, fissure, severe stenosis, late stent thrombosis). Although slow flow phenomenon and distal emboli were seen in 4 (15%) and 2 (8%) patients during PCI, respectively, final TIMI 3 flow was obtained in 23 (88%) patients. OCT of culprit and non-culprit segments showed various features such as cholesterol cleft, macrophages, calcification, and micro-capillary, where the culprit lesions more frequently contained cholesterol cleft and were accompanied by intraplaque micro-capillary, with fewer sheet calcification as compared to non-culprit segments (Table), suggesting these features as potential parameters to evaluate plaque instability.

Conclusions: Recognition of OCT features associated with plaque instability may be useful to predict future event in patients with high risk of atherosclerosis.

(n=26)	Culprit	Non-culprit	P value
Cholesterol cleft	14 (54%)	6 (23%)	0.018
Macrophage	18 (69%)	16 (60%)	0.75
Sheet calcification	12 (46%)	19 (73%)	0.006
Ca nodule	2 (8%)	3 (12%)	0.66
Micro-capillary	18 (69%)	12 (46%)	0.056

