TCT-268

Incidence and predictors of tissue prolapse after percutaneous coronary intervention for saphenous vein graft disease: Intravascular ultrasound study

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Background: The aim of this study was to investigate the relationship between intravascular ultrasound (IVUS) findings and the no-reflow phenomenon and long-term outcome after percutaneous coronary intervention (PCI) of saphenous vein graft (SVG) lesions.
Methods: No-reflow was defined as post-PCI TIMI grade 0, 1, or 2 flow.
Results: Of 311 patients who underwent pre- and post-stenting IVUS, no-reflow was observed in 39 patients (13%). Degenerated SVG (62% vs. 36%, p=0.002) was observed more frequently in the no-reflow group. IVUS-detected intraluminal mass (82% vs. 43%, p<0.001), culprit lesion multiple plaque ruptures (23% vs. 6%, p<0.001), and tissue prolapse (51% vs. 35%, p=0.043) were significantly more common in patients with no-reflow. In the multivariate logistic regression analysis, an intraluminal mass (Odds ratio [OR]=4.84; 95% CI 1.98-10.49, p=0.001), culprit lesion multiple plaque ruptures (OR=3.46; 95% CI 1.46-8.41, p=0.014), and degenerated SVGs (OR=3.17; 95% CI 1.17-6.56, p=0.024) were the independent predictors of post-PCI no-reflow. At 5-year clinical follow-up, the rates of death [14 (36%) vs. 55 (20%), p=0.036] and myocardial infarction [13 (33%) vs. 52 (19%), p=0.039] were significantly higher in the no-reflow group. However, the rate of target vessel revascularization was not different significantly between two groups [15 (38%) vs. 90 (33%), p=0.3].
Conclusions: IVUS-detected intraluminal mass, multiple plaque ruptures, and degenerated SVGs are associated with post-PCI no-reflow in SVG lesions. No-reflow was associated with poor long-term clinical outcomes after PCI for SVG lesions.

TCT-269

Abstract Withdrawn

TCT-270

Abstract Withdrawn

TCT-271

Stent Thrombosis After Intravascular Ultrasound-guided Stent Implantation

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Background: Intravascular ultrasound (IVUS) is used frequently for percutaneous coronary intervention (PCI) in Japan. However, there is little information about the incidence of stent thrombosis after IVUS-guided stent implantation.
Methods: Between January 2005 and December 2011, 2,992 lesions in 2,685 patients underwent IVUS-guided bare metal stent (BMS) (n=981) and drug-eluting stent (DES) implantation (n=2,011). The Academic Research Consortium definition of stent thrombosis was used.
Results: Definite stent thrombosis was observed in 10 lesions (1.0%) with BMS (early 0.9%, late 0.1%, and very late 0%) and 9 lesions (0.4%) with DES (early 0.2%, late 0%, and very late 0.2%). Stent thrombosis occurred in 12 (1.6%) and 7 lesions (0.4%) that had undergone PCI for acute myocardial infarction and other indications, respectively. Stent underexpansion was observed in 6 (46%) of the 13 lesions with early stent thrombosis. On the other hand, none of the 6 lesions with late or very late stent thrombosis had stent underexpansion.
Conclusions: IVUS-guided stent implantation results in a low incidence of stent thrombosis. Stent underexpansion is a risk factor of early stent thrombosis, but it was not for late or very late stent thrombosis.

TCT-272

The Role of Macrophage Accumulations on Bare-Metal Stent Failure Presenting With Acute Coronary Syndrome: Observation by Optical Coherence Tomography

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Background: Little is known about the role of macrophage accumulations (Mφ) on bare-metal stent (BMS) failure.
Methods: We evaluated 43 consecutive BMS failure lesions in 43 patients (11 acute coronary syndrome [ACS] and 32 stable myocardial ischemia [SMI], median stent duration 9.6 [interquartile range 6.9-58.8] months) by optical coherence tomography. Neointima with Mφ was defined as thin bright layer with shadowing (peak intensity >180 and attenuation rate >2, when measurements were fitted to an approximate exponential function).
Results: The mean age was 67.4±10.0 years, 40 patients (93.0%) were male, and 16 (37.2%) were diabetic. Compared with SMI patients, ACS patients showed higher incidence of thin-cap fibroatheroma (TCFA)-containing neointima (63.6% versus 6.3%, p=0.0003), lesions with Mφ (72.7% versus 9.4%, p=0.0001), neointimal rupture (72.7% versus 15.6%, p=0.001), thrombus (81.8% versus 43.8%, p=0.039), and had higher admission LDL (116.4±26.6 versus 90.8±23.9 mg/dl, p=0.012) and lower HDL cholesterol level (39 [range 33-45] versus 47 [40-55] mg/dl, p=0.021). Stent failure with TCFA-containing neointima (n=9) expressed higher incidence of Mφ (88.9% versus 8.8%, p<0.0001), larger Mφ angle (143 [range 84-262] versus 0 [0-0] °, p=0.002), and longer Mφ longitudinal length (8 [range 5-13] versus 0 [0-0] mm, p=0.003) than non–TCFA-containing neointima (n=34). Fibrous cap thickness negatively correlated with Mφ longitudinal length (r=−0.00449, p=0.045). Compared with lesions without neointimal rupture (n=30), lesions with neointimal rupture (n=13) demonstrated higher incidence of Mφ (69.2% versus 6.7%, p<0.0001). Thrombotic stent failure lesions (n=23) showed more Mφ (39.1% versus 10.0%, p=0.039) than non-thrombotic lesions (n=20). Eleven stent failure patients with Mφ presented later than 32 patients without Mφ: 104.8 (range 58.8-142.4) versus 9.0 (6.6-13.3) months (p=0.0004). Using receiver-operating curve analysis, 16.7 months was the best predictor of the presence of Mφ with a sensitivity of 100% and a specificity of 84% (area under curve =0.960, p<0.0001).
Conclusions: Mφ might be associated with neoatherosclerosis and unstable features of BMS neointima.

TCT-273

Serial Evaluation Of Peri-strut Low Intensity Area On Optical Coherence Tomography After Drug-eluting Stents Implantation

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Background: Recent studies have demonstrated that peri-strut low intensity area (PLIA) seen on optical coherence tomography (OCT) represents neointimal fibrinogen and/or extracellular matrix and is associated with neointimal thickening (NIT) after 1st-generation drug-eluting stents (DES) implantation. However, there are no data regarding PLIA in new generation DES and its change in serial OCT evaluations.
Methods: A total of 83 patients underwent 9-month OCT after DES implantation (25 sirolimus-eluting stents [SES], 20 paclitaxel-eluting stents [PES], 30 zotarolimus-eluting stents [ZES], and 8 everolimus-eluting stents [ZES]). PLIA on OCT was defined as a region around stent struts with homogenous lower intensity than surrounding tissue without signal attenuation. Inter-stent analysis was performed between PLIA + and PLIA – stents, and then intra-stent analysis was performed between PLIA + and overall stent segments. The patients also underwent 2-year OCT evaluations and serial change of NIT was analyzed.
Results: The incidence of PLIA + stents on 9-month OCT was highest in PES and lowest in SES (90% in PES vs. 28% in SES vs. 60% in ZES vs. 63% in EES, p <0.001). In inter-stent analysis, PLIA + stents showed higher mean NIT than PLIA – stents (20.11 ± 9.69 μm vs. 10.86 ± 5.46 μm, p <0.001). Also in intra-stent analysis, PLIA + segments showed higher mean NIT than overall stent segments (25.79 ± 12.22 μm vs. 20.11 ± 9.69 μm, p = 0.013). On serial 2-year OCT, PLIA + stents showed smaller increase of NIT than PLIA – stents in inter-stent analysis (3.46 ± 10.50 μm vs. 7.41 ± 10.07 μm, p = 0.092). Also in intra-stent analysis, PLIA + segments showed smaller increase of NIT than overall stent segments (1.76 ± 11.63 μm vs. 3.46 ± 10.50 μm, p = 0.451). All of these findings were regardless of DES type.
Conclusions: Although PLIA was associated with increased NIT regardless of DES type, it may mean weak potential for further neointimal growth in long-term follow-up.

TCT-274

Relationship Between Arterial Remodeling, Fibrous Cap Thinning and Lipid Accumulation: A Serial Integrated Backscatter Intravascular Ultrasound and Optical Coherence Tomography Study

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Background: A serial intravascular ultrasound (IVUS) and optical coherence tomography (OCT) study has shown that positive arterial remodeling was related to thinning of fibrous cap. Serial changes in tissue components associated with fibrous cap and/or arterial remodeling is unknown Therefore, the purpose of this study was to evaluate the relationship between changes in fibrous cap thickness and changes in plaque tissue components by using optical coherence tomography (OCT) and integrated backscatter IVUS (IB-IVUS).
Methods: Serial (baseline and 6 months follow-up) IB-IVUS and OCT examinations were performed on 81 vessels from 56 patients with ischemic heart disease who underwent percutaneous coronary intervention. 81 fibroatheromas were selected from 48 culprit lesions and 33 non-culprit lesions. Serial changes and relationships between

IB-IVUS derived tissue components (calcification, dense fibrosis, fibrosis, lipid pool) and fibrous cap thickness were investigated.
Results: The percent changes in fibrous cap thickness correlated negatively and significantly with the percent changes in external elastic membrane cross sectional area (r=-0.5, P<0.0001). The %changes in fibrous cap thickness correlated negatively and significantly with %changes in lipid pool area (r=-0.61, P<0.0001). The %changes in fibrous cap thickness correlated positively with calcification, dense fibrosis or fibrosis, as well (r=0.44, P<0.0001, r=0.60, P<0.0001, and r=0.54, P<0.0001, respectively).
Conclusions: Arterial remodeling with changes in lipid pool component is related to changes in fibrous cap thickness. Positive arterial remodeling with lipid accumulation may represent an unstable or vulnerable process of the coronary arterial plaque.

TCT-275

The Evolution of Thin-Capped Fibroatheroma in Left Main Coronary Artery As Assessed by Serial Virtual Histology Intravascular Ultrasound Analysis

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Background: Statin theraphy have been shown the regression of coronary artery plaque. However a few data was reported about the change of left main coronary artery.
Methods: We used Virtual Histology Intravascular Ultrasound (VH-IVUS) to assess mild to moderate left main coronary artery (LMCA) disease in 500 consecutive pts and identified 50 pts with who had serial VH-IVUS examinations at baseline and at follow-up at 12 months. VH-TCFA (thin-capped fibroatheroma) was defined as necrotic core (NC) >10% of plaque area with a plaque burden of >40% and NC in contact with the lumen for ≥3 image slices. Positive remodeling was a remodeling index (lesion/reference EEM [external elastic membrane] area) >1.05.
Results: Pt age was 57±13 yrs and 20% were diabetics. The incidence of LM-TCFA was 11.2% (56/500). following 16 pts with VH-TCFA at baseline and serial VH-IVUS, distal reference lumen area was 14.6±4.2 mm^2. LMCA length was 6.4±4.0mm, and the rate of positive remodeling was 14% (8/56). 74% of VH-TCFA were located in mid-body of the LMCA and the rest at the distal bifurcation. After 12 months of statin therapy, minimal lumen area (12.8±3.8 to 12.4±4.5 mm^2, p=0.6) and maximum %NC (28.0±13.2 to 27.2±12.1, p=0.8) were similar; and only 37.5% (6/16) of VH-TCFA had healed to a non-VH-TCFA phenotype and 3 new VH-TCFA appeared (Table) even though low density lipoprotein (LDL) level was decreased in 87.5 % (14/16) pts with 12/16 having an LDL <100mg/dL.
Conclusions: Healing of VH-TCFA in the LMCA is not high during "routine" statin therapy. This suggests the need for more strict control of risk factors in these pts.

		Follow-up				
Baseline		PIT (n=14)	ThCFA (n=5)	TCFA (n=13)	Fibrotic (n=12)	Fibrocalcific (n=6)
PIT	(n=14)	14				
ThCFA	(n=1)		1			
TCFA	(n=16)		1	10	3	2
Fibrotic	(n=15)		3	3	9	
Fibrocalcific	(n=4)					4

TCT-276

Culprit lesion phenotype may depend on lesion location in patients with ST elevation acute myocardial infarction: TAMI-R study

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Background: Thin capped fibroatheroma(TCFA) is a principal pathophysiologic mechanism to occur acute coronary syndrome. However, the characteristics of each TCFA have not been evaluated.
Methods: We used virtual histology-intravascular ultrasound (VH-IVUS) to assess culprit lesions in 196 consecutive pts with ST elevation acute myocardial infarction (STEMI) and compared culprit lesions in the RCA (59 pts) with culprit lesions in the left coronary artery (LCA, 137 pts). VH-thin-capped fibroatheromas (VH-TCFAs) were defined as necrotic core (NC) >10% of plaque area, plaque burden >40%, and NC in contact with the lumen for ≥3 image slices.
Results: Plaque ruptures were seen in 85/196 of which 52 were located in the proximal 30mm of a coronary artery. Lesion length was similar; vessel size, plaque burden, minimal lumen area, and average %NC were larger; and VH-TCFA phenotype was more common (60% vs 31%, p=0.001) in RCA vs LCA-STEMI (Table). Importantly, culprit lesion plaque ruptures were more common in RCA (45.5%) vs LAD-STEMI (30.1%, p=0.034) as was the frequency of VH-TCFA phenotype in the setting of culprit plaque ruptures (72% in RCA vs 29% in LAD-STEMI, p=0.005).
Conclusions: Plaque ruptures are not always detected by IVUS in culprit lesions of pts presenting with STEMI, and VH-TCFA phenotype is not always seen in these culprit lesions whether or not plaque rupture is detected. Rather, plaque rupture and VH-TCFA phenotype in culprit lesions of STEMI pts appear to depend on location (RCA vs LCA) and vessel size.

	RCA	LCA	p-value
Lesion length (mm)	20.60+/−7.81	23.39+/−10.29	0.200
Distal reference lumen area (mm^2)	9.06+/−4.05	6.84+/−3.32	0.002
Minimal lumen area (mm^2)	4.52+/−2.58	3.49+/−1.85	0.007
Vessel area (mm^2) (ruptured site)	21.00+/−6.00	16.83+/−5.09	0.004
Lumen area (mm^2) (ruptured site)	7.59+/−2.81	6.05+/−2.33	0.020
Plaque area (mm^2) (ruptured site)	11.31+/−3.92	8.30+/−3.41	0.027
Ruptured cavity (mm^2)	1.18+/−1.05	0.83+/−0.69	0.145
Remodeling index (@ ruptured site)	1.04+/−0.19	1.13+/−0.29	0.193
average fibrotic plaque (%)	59.96+/−10.32	58.09+/−13.43	0.425
average fibrofatty plaque (%)	10.08+/−5.34	9.60+/−5.57	0.606
average necrotic core (%)	20.52+/−8.42	17.38+/−7.73	0.020
average dense calcium (%)	8.66+/−6.06	10.51+/−7.01	0.118
VH-IVUS phenotype			0.02
Pathologic intimal thickening	2	8	
Thick cap fibrotheroma	0	1	
VH-TCFA	22	21	
Fibrotic	2	13	
Fibrocalcific			

TCT-277

High Level of Copeptin in ST elevation Acute Myocardial Infarction patients is associated with In-Hospital Mortality and Plaque Rupture; TAMI-COP study

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Background: Copeptin has been known to predict heart failure and cardiovascular death in patients with acute coronary syndrome.
Methods: We collected coronary arterial blood samples from the infarct artery during primary percutaneous coronary intervention (PCI) in 80 STEMI pts and 28 controls. We assessed commonly used cardiac biomarkers (CK, CK-MB, troponin-I, CRP) and additionally measured recently introduced biomarkers (Copeptin [C-terminal Provasopressin] and N-terminal pro-B-type natriuretic peptide [NT-proBNP]).
Results: Pt age was 58±12 yrs and 93% were males. Intravascular ultrasound (IVUS) of the culprit lesion in 80 pts showed ruptured plaques in 36 pts. STEMI pts had a higher Copeptin level (265.89±183.10 pmol/L) vs193.67±60.17 pmol/L in normal controls, p=0.005. Especially, Copeptin levels were higher in ruptured plaque compared to non-ruptured plaques: 318.83±209.34 pmol/L vs 221.99±151.49 pmol/L, p=0.034. While troponin I, CK-MB, and CRP were not correlated with Copeptin, NT-proBNP was correlated with copeptin (r=0.579, p=0.0003). In hospital death occurred in 7 pts with a high Copeptin level (figure); all were due to cardiogenic shock after primary PCI.
Conclusions: Copeptin levels are associated with plaque rupture in STEMI pts and predict mortality after primary PCI.