Conclusions:
IVUS-detected intraluminal mass, multiple plaque ruptures, and degenerated SVGs are associated with post-PCI no-reflow in SVG lesions. No-reflow was between two groups [15 (38%) vs. 90 (33%), p = 0.346; 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Abstract Withdrawn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Abstract Withdrawn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%) that had undergone PCI for acute myocardial infarction and other indications, respectively. Stent underexpansion was observed in 6 (4.6%) 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 Preventing With Acute Coronary Syndrome: Observation by Optical Coherence Tomography
Yuuetsu Kikuta1, Hideo Takebayashi1, Shigeki Hiramatsu, Manabu Taniguchi2, Kenji Goto1, Masahito Taniguchi3, Katsumasa Sato4, Etsuko Ikeda1, Arata Hagikura1, Hiroki Yaman1e, Seiichiru Haruta4
1Fukuyama Cardiovascular Hospital, Fukuyama, Hiroshima

Background: Little is known about the role of macrophage accumulations (M₆) 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 6.9-8.8] months) by optical coherence tomography. Neointima with M₆ 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 M₆ (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 (114±4.26 versus 90.8±2.39 mg/dl, p = 0.012) and lower LDL cholesterol level (39 [range 33-45] vs. 47 [40-55] mg/dl, p = 0.021), Stent failure with TCFA-containing neointima (n = 9) expressed higher incidence of M₆ (89.8% versus 8.8%, p = 0.0001) and larger M₆ area (143 [range 84-362] versus 0 [0-0], p = 0.002, and longer M₆ 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 M₆ longitudinal length (r = -0.0049, p = 0.045). Compared with lesions without neointimal rupture (n = 30), lesions with neointimal rupture (n = 13) demonstrated higher incidence of M₆ (69.2% versus 6.7%, p = 0.0001). Thrombotic stent failure lesions (n = 23) showed more M₆ (39.1% versus 10.0%, p = 0.039) than non-thrombotic lesions (n = 20). Eleven stent failure patients with M₆ presented later than 32 patients without M₆: 104.8 (range 58.4-142.4) versus 90.6 (6.6-13.3) months (p = 0.0004). Using receiver-operating curve analysis, 16.7 months was the best predictor of the presence of M₆ with a sensitivity of 100% and a specificity of 84% (area under curve = 0.960, p < 0.0001).

Conclusions: M₆ might be associated with neoatherosclerosis and unstable features of BMS neointima.
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=-0.4, P=0.001). 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=0.001, and r=-0.54, P<0.001, respectively).

**Conclusions:** Arterial remodeling with changes in lipid pool component is related to 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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<th>Study</th>
<th>Method</th>
<th>Results</th>
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<td>Sang-Wook Kim1, Sang Youn Lee2, Gary Mintz2, Young Joon Hong3, Doo-Hyun Kim3, Du-Jeong Kim3, Dae-Joong Kim4</td>
<td><strong>Results:</strong> 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 at follow-up at 12 months. VH-TCFA (thin-capped fibroatheroma) was defined as necrotic core (NC) &gt;10% of plaque area with a plaque burden of &gt;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) &gt;1.05.</td>
<td><strong>Conclusions:</strong> Healing of VH-TCFA in the LMCA is not high during “routine” stent therapy. This suggests the need for more strict control of risk factors in these pts.</td>
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### TCT-276

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

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<tr>
<td>Sang-Wook Kim1, Sang Youn Lee1, Gary Mintz2, EunYoung Kim1, Jun Hwan Jo1, KwangJae Lee1, CheeJeong Kim1, TaeHo Kim1</td>
<td><strong>Results:</strong> Plaque ruptures were seen in 85/196 of which 52 were located in the proximal LMCA and 33 in a coronary artery. Lesion length = 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). Impolitrally, culprit lesion plaque rupture were more common in RCA (45.5%) vs LAD-STEMI (30.1%)</td>
<td><strong>Conclusions:</strong> 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 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.</td>
<td><strong>Conclusions:</strong> 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 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.</td>
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