	IVUS group (n=3343)	Non IVUS group (n=5233)	p value
Any ST within 1 year	0.52%	1.04%	0.01
- Acute (< 24 hours)	0.06%	0.04%	0.66
- Subacute (1-30 days)	0.27%	0.56%	0.05
- Late (31 days – 1 year)	0.25%	0.48%	0.1
All-cause death/MI within 1 year	3.96%	5.35%	0.004
Cardiac death within 1 year	0.84%	1.17%	0.1384
Peri-procedural MI	1.26%	1.53%	0.3
Non peri-procedural MI within 1 year	1.23%	2.17%	0.002

Conclusions: These data, drawn from the largest prospective registry of IVUS use to date, suggests that IVUS guidance during DES PCI may result in less ST, non peri-procedural MIs and cardiac mortality during 1 year follow-up.

TCT-22

Combination of High Wall Shear Stress, Plaque Burden, and Plaque Phenotype has Incremental Value for Prediction of Increased Plaque Vulnerability in Patients with Coronary Artery Disease

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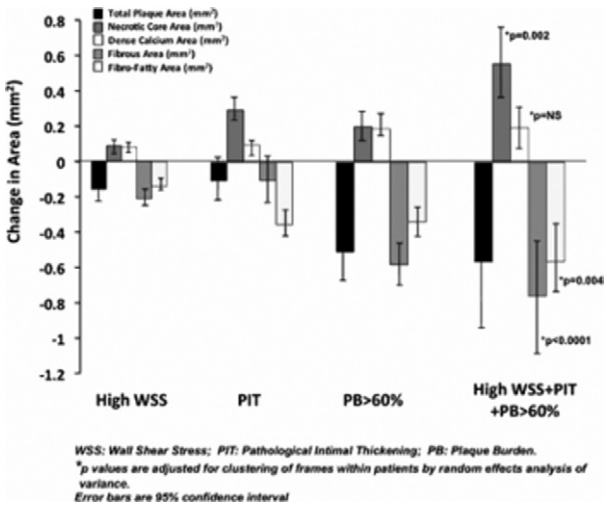
Background: High wall shear stress (WSS) and large plaque burden have each been associated with development of coronary plaque vulnerability. We hypothesized that high WSS, large plaque burden, and plaque phenotype have incremental value in prediction of increased plaque vulnerability.
Methods: Twenty patients with non-obstructive CAD, treated with 80 mg/day atorvastatin, underwent baseline and 6-month follow-up virtual histology-intravascular ultrasound (VH-IVUS), Doppler velocity, and computational fluid dynamics modeling for calculation of WSS. In each IVUS frame (n=2,249), plaque composition was classified as necrotic core (NC), dense calcium (DC), fibro-fatty (FF), and fibrous (FI) and plaque phenotype as pathological intimal thickening (PIT), fibrotic plaque, and fibroatheroma. Expansive remodeling was defined as change in (Δ)EEM area/Δplaque area >1 and high WSS as ≥25 dynes/cm2. Increased plaque vulnerability was considered as 1)increase in NC, 2)decrease in FI or FF, 3)expansive remodeling.
Results: Among 3 phenotypes, PIT had the greatest increase in NC area (p=0.06). At follow-up, compared to segments with baseline high WSS or plaque burden >60% or PIT alone, coronary segments with a combination of high WSS, plaque burden >60%, and PIT were associated with greater increase in NC area (p=0.002), greater decrease in FF (p=0.004) and FI areas (p<0.0001), and higher frequency of expansive remodeling (p=0.019).



Conclusions: In patients with CAD treated with statins, combination of high WSS, plaque burden >60%, and PIT has an incremental value in predicting increased plaque vulnerability at follow-up.

TCT-23

Detection by Near-infrared Spectroscopy of Large Lipid Core Plaques at Culprit Sites in Patients with Acute ST-Segment Elevation Myocardial Infarction

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Background: Autopsy studies suggest most ST-segment elevation myocardial infarctions (STEMI) are caused by a ruptured lipid core plaque (LCP), yet this has not been demonstrated in vivo. A novel combined near-infrared spectroscopy (NIRS) and intravascular ultrasound (IVUS) catheter can now accurately detect LCP in pts.
Methods: We performed NIRS-IVUS in the culprit vessel of 18 consecutive STEMI pts (age 59±12; 83% male). To minimize alteration of the culprit plaque, NIRS-IVUS was performed after establishment of TIMI 3 flow by a guidewire, small balloon or aspiration, but prior to stent placement. LCP was quantitated as the lipid-core burden index (LCBI) over a 10mm vessel segment, a measure of the fraction of NIRS measurements indicating lipid. NIRS in STEMI pts was compared to findings in 329 stable coronary pts without MI (age 64±10; 79% male) and to autopsy specimens from 57 pts (age 65±10; 64% male) without histologic LCP.
Results: NIRS identified large, often circumferential LCP at the culprit site in 17 of 18 STEMI pts (94.4%; Figure). The LCBI was 372±213 within the STEMI culprit vs 55±100 in 4325 segments in stable coronary pts (p<0.001) and vs 27±52 in 446 autopsy specimens without histologic LCPs (p<0.001; 13-fold lower than STEMI culprits).
Conclusions: A NIRS signature of a large, often circumferential LCP was significantly more frequent at STEMI culprits than in comparable regions in stable pts and in autopsy specimens without LCPs. A prospective study is indicated to determine if such plaques detected by NIRS prior to rupture place pts at risk for future acute coronary events and cardiac death.