irrespective of whether FFR or CFVR is ischemic. This underscores the requirement of both flow and pressure assessment for optimal risk stratification.

TCT-19
Fractional Flow Reserved Derived From Computed Tomographic Angiography (FFRCT) for Intermediate Severity Coronary Lesions: Results from the DeFACTO Trial (Determination of Fractional Flow Reserve by Anatomic Computed Tomographic Angiography)

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Background: Non-invasive fractional flow reserve (FFR) derived from typically acquired coronary computed tomographic (CT) angiography (FFRCT) is a novel method that employs computational fluid dynamics to diagnose coronary lesions that cause ischemia. To date, the diagnostic performance of FFRCT versus CT stenosis to effectively diagnose ischemia in vessels with stenosis of intermediate severity (30%-70% luminal diameter stenosis) has been inadequately studied.

Methods: Amongst 407 vessels from 170 centers in 4 countries who underwent CT, FFRCT, invasive coronary angiography and invasive FFR, we identified 150 vessels of intermediate stenosis severity by CT. CT stenosis, FFRCT, and invasive FFR were interpreted in blinded fashion by independent core laboratories. FFRCT and FFR <0.80 were considered ischemic, while CT stenosis >50% were considered obstructive. Diagnostic performance of CT stenosis alone versus FFRCT alone were evaluated for sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV); as well as employing a decision rule combining FFRCT and CT stenosis wherein maximal per-patient CT stenosis <50% was considered negative for ischemia and subclinical occlusions were considered positive for ischemia. Area under the receiver operating characteristics curve for FFRCT and CT stenosis were compared for discrimination.

Results: For lesions of intermediate stenosis severity, FFRCT demonstrated a sensitivity, specificity, PPV and NPV of 74%, 66%, 40%, and 99%; while CT stenosis demonstrated a sensitivity, specificity, PPV and NPV of 34%, 72%, 27%, 78%. When applying the decision rule, the combination of FFRCT and CT stenosis resulted in a sensitivity, specificity, PPV and NPV of 74%, 67%, 39% and 90%. The AUC of FFRCT for ischemia in stenoses of intermediate severity was higher than CT stenosis (0.80 vs. 0.55, p<0.001). All-cause death or MI also occurred significantly less frequently in the IVUS group (Table) due to fewer cardiac deaths and non-ACS deaths (Table).

Conclusions: In the present analysis from the largest prospective IVUS study to date, the presence of attenuated plaque, tissue protrusion, reference segment plaque burden and edge dissections were significant predictors of ST, whereas underexpansion and malapposition were not. This highlights the complex relationship between underlying lesion morphology, PCI results and outcomes in pts treated with DES.

TCT-20
Use of IVUS Reduces Stent Thrombosis: Results from the Prospective, multicenter ADAPT-DES Study

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Background: Previous IVUS studies examining correlates of drug-eluting stent (DES) thrombosis were retrospective and in small patient cohorts.

Methods: ADAPT-DES was a prospective, multicenter, real-world registry of 8,576 consecutive pts at 11 international centers treating percutaneous coronary intervention (PCI) with DES designed to determine the frequency, timing, and correlates (clinical, angiographic and platelet reactivity) of early and late stent thrombosis. During the index procedure, IVUS was used in 3,343 (39.0%) of cases and 1-year follow-up was completed.

Results: Average pt age was 64.0 years and 74.1% were male. Patients in the IVUS group were more likely to have ACS and high platelet reactivity to thienopyridine (PRU>200), and were treated with longer and larger stents, and more everolimus-eluting stents. Within 1 year, definite/probable stent thrombosis (ST) occurred in 17 (0.52%) pts in IVUS group vs in 53 (1.04%) pts in non-IVUS group (HR [95%CI] = 0.37 [0.20, 0.69], p=0.0016) (Table). In a propensity adjusted model to account for the predictors of IVUS use, IVUS guidance was independently associated with a reduced 1-year rate of ST (HR [95%CI] = 0.37 [0.20, 0.68], p=0.0014). All-cause death or MI also occurred significantly less frequently in the IVUS group (Table) due to fewer cardiac deaths and non-procedural MIs.

Conclusions: Whether IVUS guidance reduces stent thrombosis and improves clinical outcomes after DES is unknown, with only small observational studies and no randomized trials having been reported.

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Conclusions: These data, drawn from the largest prospective registry of IVUS use to date, suggest 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=3343), 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/Aplaque area >1 and high WSS as ≥25 dynes/cm². Increased plaque vulnerability was considered as 1)increase in NC, 2)expansion 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.

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

TCT-23
Detection of 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 ±55 in STEMI pts (p<0.001) and vs 27 ±42 in 446 autopsy specimens without histologic LCP (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.