frequently presented with ST-elevation myocardial infarction (STEMI; 45.7%, 40.0%, 22.9%, p=0.030) and with TIMI flow <3 (32.9%, 20.0%, 17.1%, p=0.042). According to the ratio of upstream and downstream RG, 69.5% of lesions were classified as upstream-dominant lesions and 30.5%, as downstream-dominant lesions. Among the 66 upstream-dominant lesions, 65 cases (98.5%) had upstream rupture and the RG ratio (RGupstream/RGdownstream) was an independent predictor for upstream rupture (OR 1.481, 95% CI 1.035-2.120, p=0.032). Upstream-dominant lesions more frequently presented with STEMI than downstream-dominant lesions (48.5% vs. 24.1%, p=0.026). In the idealized model and CFD analysis, axial plaque stress in the upstream segment was higher than in the downstream segment (10,968 dyne/cm² vs. 5,651 dyne/cm²) in upstream-dominant lesions. The inverse was also true for downstream-dominant lesions (7,667 dyne/cm² vs. 12,312 dyne/cm²).

CONCLUSIONS Both clinical presentation and degree of flow limitation were associated with the location of plaque rupture. Longitudinal lesion asymmetry assessed by RG, which can affect regional distribution of hemodynamic stress, was associated with the location of rupture as well as clinical presentation.

CATEGORIES IMAGING: Cath Lab of the Future

KEYWORDS Coronary artery disease, Plaque rupture, Plaque, vulnerable

TCT-315 Variation in Collagen in the Caps of Human Coronary Lipid-core Plaque Autopsy Specimens: A Possible Measure of Cap Weakness

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BACKGROUND The cap over a coronary lipid core plaque (LCP) protects the plaque from rupture and causation of a coronary event. Cap thickness has been considered the primary measure of likelihood of plaque rupture. However, caps of equal thickness may contain markedly different amounts of collagen. A thick cap may be low in collagen suggesting possible cap weakness. Poor correlation between cap thickness and collagen for caps above 200µm suggests that the clinical relevance of a dimensional measurement of cap thickness for detecting vulnerable plaques might be enhanced by assessing the amount of collagen in the plaque cap. Application of the aforementioned algorithm to patients in ongoing prospective clinical trials could increase the accuracy of vulnerable plaque detection.

CATEGORIES IMAGING: Vulnerable Plaque

KEYWORDS Collagen, Fibrous cap thickness, Histological analysis

TCT-316 Characteristics Of Culprit Lesions Vs. Non-Culprit Lesions In Patients With ST-Elevation Myocardial Infarction – An Optical Coherence Tomography Study. On Behalf Of The TOTAL-OCT Investigators

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BACKGROUND Autopsy and imaging studies have demonstrated that most ST-elevation myocardial infarctions (STEMI) are caused by plaque rupture of thin cap fibroatheroma. However, lesions with intact fibrous cap are also common as culprit lesions (CL) in myocardial infarction, suggesting that a thin fibrous cap may not be the only marker of vulnerable plaque. We compared OCT imaging findings between CL vs. non-culprit lesions (NCL) in the culprit vessel of patients undergoing primary percutaneous coronary intervention for STEMI.

METHODS We analyzed images from 65 patients recruited in the TOTAL-OCT sub-study (Thrombectomy versus PCI Alone) who had OCT imaging performed after thrombectomy and a plaque rupture or
intact fibrous cap CL. Lesion length (both CL and NCL) was defined by the number of consecutive frames with 2 or more diseased quadrants. We analyzed the plaque composition in quadrants on image frames at 1 mm intervals over the length of the lesion. The maximum lipid arc and minimum fibrous cap thickness (FCT) was measured. A comparison between STEMI CL vs. NCL in the same STEMI culprit artery was performed. Logistic regression was used to determine the contribution of quadrants of lipid, lipid arc and FCT to identify CL.

RESULTS Morphology findings of the 65 CL were 34 (52.3%) plaque ruptures and 31 (47.7%) intact fibrous cap; and morphology of the 58 NCL were 23 (39.7%) fibroatheroma and 35 (60.3%) non-fibroatheroma. CL had significantly more lipid quadrants than fibroatheroma NCL (33.89 ± 15.51 vs. 17.09 ± 8.14, p =<0.001), larger maximum lipid arc (342.36 ± 49.86 vs. 281.26 ± 80.74, p =<0.001) and lower FCT (80.57 ± 28.75 vs. 94.34 ± 17.44, p = 0.004) . Lipid quadrants were significantly more discriminative at identifying STEMI CL (AUC: 0.82, p =<0.001) or FCT (AUC: 0.70, p = 0.004). According to ROC curves, a threshold of 20 quadrants of lipid would have a sensitivity of 80 % and specificity of 70 % to identify a STEMI CL.

CONCLUSIONS Lipid content is most discriminatory marker identifying STEMI CL. Both FCT and lipid quadrants should be considered in prospective evaluation of vulnerable plaques.

CATEGORIES IMAGING: Vulnerable Plaque

KEYWORDS OCT, ST elevation myocardial infarction, Vulnerable plaque

TCT-317

Local Low Endothelial Shear Stress (ESS) Provides Incremental Prediction of Non-culprit MACE in Addition to Plaque Burden, Minimal Lumen Area, and Plaque Morphology: The PROSPECT Study

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BACKGROUND Low ESS, a pro-inflammatory stimulus, is an important predictor of coronary plaque development/progression. Whether low ESS adds incremental predictive value for future major adverse cardiac events (MACE) in untreated coronary lesions in high-risk patients with an acute coronary syndrome (ACS) is unknown.

METHODS In the PROSPECT study, 697 patients with ACS underwent 3-vessel intracoronary imaging. Independent predictors of non-culprit (nc) lesion MACE from untreated coronary lesions in 3 year follow-up were large plaque burden (PB), small minimum lumen area (MLA), and thin cap fibroatheroma (TCFA) morphology. In the present analysis, all nc-lesions leading to a new MACE in follow-up (nc-MACE lesions, n=50) and ~4-fold randomly selected control nc-lesions without follow-up MACE (nc-non-MACE lesions) were analyzed. Baseline ESS for each lesion was calculated using computational fluid dynamics. A propensity score for low ESS was determined accounting for PB, MLA, TCFA, artery and location in the artery. Local ESS (lowest ESS in 90° arc around the artery circumference) was then examined for incremental association with MACE.

RESULTS Imaging was sufficient for analysis in 32 nc-MACE lesions. Two nc-MACE lesions were excluded due to unreliable lesion morphology. Non-fibroatheromas were too few for analysis and they were excluded. The final dataset included 145 lesions: 13 nc-MACE TCFA, 10 nc-MACE thick cap fibroatheroma (ThCFA), and 122 non-nc-MACE lesions (63 TCFA, 59 ThCFA). Cumulative frequency distribution shows that the lesions responsible for future nc-MACE frequently exhibited low ESS at baseline (Figure). In a propensity-adjusted multivariable model, low ESS at baseline was strongly associated with nc-MACE in f/u (odds ratio 0.16 [95% CI 0.06-0.40], p <0.0001).

CONCLUSIONS After accounting for baseline large PB, small MLA, TCFA, and lesion location, low local ESS at baseline adds significant and substantial incremental predictive value to identify high-risk untreated lesions likely to cause MACE during 3 year followup.