necrotic core (NC) and dense calcium (DC). Quantitative assessment of these plaque components and the presence of VH-IUV-derived thin-cap fibroatheroma (VH-TCA) in the coronary arterial trees were compared to hs-CRP levels in individuals. hs-CRP levels were measured before coronary angiogram and IVUS study.

**Results:** Forty-nine patients (77.8%) were diagnosed with acute coronary syndrome in the population. The mean and median values of hs-CRP were 0.43 ± 1.52 mg/dl and 0.12 mg/dl, respectively. The total number of VH-TCA was 2.5 ± 1.9 per patient. The hs-CRP levels were positively correlated to mean plaque burden, total plaque volume index, volume index of FF and DC. But parameters of NC and the number of VH-TCA were not related with hs-CRP level. In multivariate analysis, the volume index of DC was more reliable factor to hs-CRP (β=5.490, 95% CI=5.072-5.908, p<0.001).

**Conclusions:** This three-vessel VH-IUV presented that hs-CRP were related to the lesions is feasible in both clinical scenarios, STEMI and SAP, whereas STEMI /C6 0,0045%, p<0.01) and greater macrophage index (STEMI <0.15, p<0.01). Mid parts also had increased lipid arc (proximal 12.9±2.0° vs middle 187±19° vs distal 136.6±18°; p<0.01) with a higher incidence of plaque disruption/thrombus (proximal 8% vs middle 29% vs distal 14%; p=0.071). In segments with plaque disruption/thrombus, ESS was higher (normalized ESS: 1.3:±0.2 vs 0.9:±1.0, p=0.17), lipid arc was greater (236.9±15 vs 150.0±3.5°; p<0.01), and fibrous cap was thinner (108±24 vs 206±1.31 mm, p<0.05).

**Conclusions:** In patients with ACS, the highest ESS values within a culprit lesion colocalized with plaque disruption/thrombus, which were more common in the mid part of the lesions. Further studies are warranted to elucidate whether ESS directly acts as a trigger for plaque rupture or whether other factors are involved in the rupture/thrombosis of a plaque which developed in a preceding low ESS environment.

**Results:** Six lesions (75%) had significant stenosis (>50% area stenosis by OCT). ESS was elevated in the mid part of the lesions (normalized ESS using the average within each lesion: proximal 0.7±0.2 vs mid 1.3±0.1 vs distal 0.4±0.2; p<0.01). Mid parts also had increased lipid arc (proximal 12.9±2.0° vs middle 187±19° vs distal 136.6±18°; p<0.01) with a higher incidence of plaque disruption/thrombus (proximal 8% vs middle 29% vs distal 14%; p=0.071). In segments with plaque disruption/thrombus, ESS was higher (normalized ESS: 1.3±0.2 vs 0.9±1.0, p=0.17), lipid arc was greater (236.9±15 vs 150.0±3.5°; p<0.01), and fibrous cap was thinner (108±24 vs 206±1.31 mm, p<0.05).

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**Background:** Plaque rupture/thrombosis is the most prominent mechanism leading to acute coronary syndromes (ACS). However, the factors responsible for the location of plaque rupture/thrombosis along a lesion are unclear. Endothelial shear stress (ESS) is a major determinant of vascular pathology. We investigated the local ESS patterns in association with the distribution of thrombosis/plaque features along the culprit lesions in patients with ACS prior to percutaneous coronary intervention using frequency domain optical coherence tomography (FD-OCT).

**Methods:** 3-dimensional coronary artery reconstruction using FD-OCT & coronary angiography was performed in the culprit vessel of 8 patients presenting with ACS. In each culprit lesion, we assessed local ESS (with computational fluid dynamics) and morphological features (by FD-OCT) in consecutive 1-mm segments distinguished in a proximal, mid and distal part. As thrombus interferes with accurate evaluation of underlying plaque characteristics, plaque disruption and local thrombus were combined in the analysis.

**Results:** Six lesions (75%) had significant stenosis (>50% area stenosis by OCT). ESS was elevated in the mid part of the lesions (normalized ESS using the average within each lesion: proximal 0.7±0.2 vs mid 1.3±0.1 vs distal 0.4±0.2; p<0.01). Mid parts also had increased lipid arc (proximal 12.9±2.0° vs middle 187±19° vs distal 136.6±18°; p<0.01) with a higher incidence of plaque disruption/thrombus (proximal 8% vs middle 29% vs distal 14%; p=0.071). In segments with plaque disruption/thrombus, ESS was higher (normalized ESS: 1.3±0.2 vs 0.9±1.0, p=0.17), lipid arc was greater (236.9±15 vs 150.0±3.5°; p<0.01), and fibrous cap was thinner (108±24 vs 206±1.31 mm, p<0.05).

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