Plaque progression is dependent on the local hemodynamics (time-averaged wall shear stress and TSVI) in combination with the plaque phenotype

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It is well established that, in the presence of risk factors, atherosclerotic plaques preferably grow at time-averaged low wall shear stress (TAWSS) locations. Although low TAWSS regions are at risk for plaque growth, not all low TAWSS regions are affected. We hypothesized that not only the magnitude of the WSS but also the temporal and spatial variations in WSS (contraction or expansion of stress at the endothelial cell, figure 1A) influence the endothelial cell response and thereby the risk on plaque growth. These variations can be captured by the recently introduced parameter TSVI. TSVI already proved to be more predictive for cardiovascular events than TAWSS [1].

Objective: To investigate the predictive power of TSVI for plaque growth in human coronary arteries in plaques with different phenotypes.

Methods: 38 non-stented, non-culprit coronary arteries (MPACT study) were imaged with near-infrared spectroscopy intravascular ultrasound (NIRS-IVUS) and optical coherence tomography (OCT) at the baseline and after 12 months [2]. Coronary reconstructions were based on fusion of the multimodality imaging and used as input for computational fluid dynamics. Vessel-specific flow was measured using Doppler wire and used as input to calculate TAWSS and TSVI [2]. The coronary arteries were subdivided into 1.5 mmx45° sectors and the TAWSS and TSVI were classified as low, mid and high based on the range per vessel. Per sector the plaque growth was quantified as plaque burden (Plaque area/total vessel area*100%) change over time (ΔPAV). Based on the NIRS and OCT data, the sectors were characterized as lipid rich plaque (wall thickness<0.5 mm & NIRS+, lipid rich(OCT)) or plaque without lipid (wall thickness >0.5 mm & NIRS-, fibrous (OCT)) or plaque free wall (wall thickness <0.5 mm &NIRS-, PFW (OCT)). The effect of TAWSS, TSVI and the combination with plaque phenotype on plaque progression was evaluated using Linear Mixed Model in SPSS.

Results: TSVI at baseline was significantly associated with PAV increase, with high PAV for high TSVI (p<0.001). A clear trend emerged for TAWSS, with high PAV for low TAWSS at baseline (Figure 1B). The highest plaque progression at 1-year follow-up was observed for lipid rich plaques (both based on NIRS and OCT) exposed to either low TAWSS or high TSVI (figure 2). Also in the PFW sectors clear plaque progression was observed dependent on either TAWSS or TSVI.

Conclusion: Lipid rich regions exposed to either low TAWSS or high TSVI are at the highest risk for plaque progression. These data suggest that although TAWSS and TSVI have a different mechanism of action on the endothelium, but both actions are involved in plaque progression.
Figure 2