Lipid-Rich Plaque Progression in Human Coronary Arteries can be Predicted Combining Multimodal Imaging and Computational Hemodynamics

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Introduction

Plaque composition and wall shear stress (WSS) magnitude act as well-established players in coronary plaque progression [1]. However, relying solely on WSS magnitude is insufficient to fully describe the biomechanical stimuli affecting atherosclerosis evolution, as endothelial cells experience also changes in the spatiotemporal configuration of WSS on the luminal surface. This study explores WSS profile and lipid content signatures of plaque progression to identify biomarkers of coronary atherosclerosis in a human follow-up study.

Methods

Thirty-eight non-culprit human coronary segments were imaged by coronary computed tomography angiography (CCTA), intravascular ultrasound (IVUS), near-infrared spectroscopy (NIRS), and optical coherence tomography (OCT) at time point T1 and at 1 year follow-up (T2). Baseline coronary artery geometries were reconstructed from IVUS and CCTA, and were combined with patient-specific flow information to perform CFD simulations assessing the time-average WSS magnitude (TAWSS) and the variability in the WSS contraction/expansion action on the endothelium along the cardiac cycle, quantified in terms of topological shear variation index (TSVI) [2,3]. Plaque progression was measured as IVUS-derived plaque atheroma volume change (ΔPAV) between T2-T1. Plaque composition was classified as lipid rich, fibrous or plaque free (PFW) based on NIRS and OCT images.

Results

An explanatory case of luminal distribution of TAWSS, TSVI and ΔPAV is presented in Figure 1A. Overall, the luminal exposure to high TSVI at T1 was associated with significantly higher ΔPAV in the T2-T1 time interval (4.00±0.69%) than sectors exposed to low or mid TSVI at T1 (Figure 1B). A clear trend emerged also for the exposure to low TAWSS at T1 and high ΔPAV (3.60±0.62%). Plaque phenotype acted synergistically with TAWSS or TSVI regarding plaque progression: at low TAWSS or high TSVI sectors in combination with lipid rich plaque, ΔPAV values were significantly higher than when considering the individual contribution of hemodynamics (≥5.90%, p<0.01; Figure 1C).

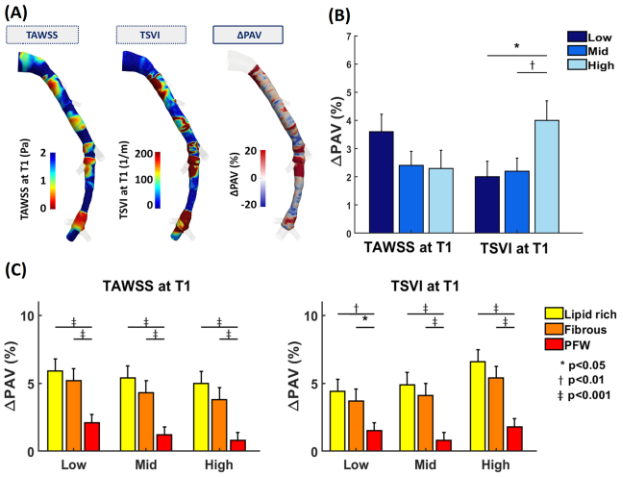

Figure 1: (A) TAWSS, TSVI and ΔPAV maps; (B) TAWSS and TSVI vs. estimated ΔPAV: (B) solely; (C) in combination with plaque phenotypes.

Conclusions

Luminal exposure to high TSVI, solely or combined with a lipid rich plaque phenotype, is associated with enhanced plaque progression at 1-year follow-up. Where plaque progression occurred, low TAWSS was also observed. These findings indicate TSVI, in addition to low TAWSS, as a potential biomechanical predictor for plaque progression, offering promise for clinical translation to enhance patient prognosis.

References

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