The variability of wall shear stress topological skeleton predicts plaque growth in human coronary arteries

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Introduction

There is an increasing interest on the wall shear stress (WSS) topological skeleton (TS), due to its capability to improve the description of the complex biomechanical stimulus affecting atherosclerosis evolution [1]. The WSS TS consists of fixed points, where the WSS vanishes, and unstable/stable manifolds connecting them, where WSS exerts a contraction/expansion action on the endothelium [2]. Here we test the ability of WSS TS to predict atherosclerotic plaque growth in a human follow-up study.

Methods

Forty-nine non-culprit human coronary segments were imaged at time point T1 and at 1 year follow-up (T2). Plaque burden (PB) growth along time interval T2-T1 was measured on IVUS images. Computational hemodynamics simulations were performed at T1 on the 3D reconstructed models using patient-specific boundary conditions. Possible links between PB growth and (1) the canonical cycle-average WSS magnitude (TAWSS, a hallmark of atherosclerotic progression) and (2) WSS TS features [2], using the recently introduced Topological Shear Variation Index (TSVI), were investigated. TSVI measures the variability of the contraction/expansion action exerted by the WSS on the endothelium along the cardiac cycle [1,3]. PB growth associated to low, mid and high tertiles of TAWSS and TSVI was analysed.

Results

Figure 1A presents the TSVI luminal distribution for an explanatory case. The average PB growth in luminal regions exposed to low, mid, or high TAWSS and TSVI values is also presented (Figure 1B). Overall, the exposure to high TSVI at T1 resulted in significantly higher PB growth than the exposure to low or mid TSVI. An association emerged also between the exposure to low TAWSS at T1 and PB growth.

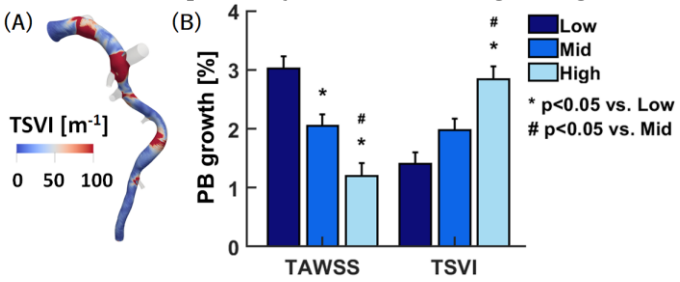

Figure 1. (A) TSVI map; (B) TAWSS and TSVI *vs.* PB growth

Conclusions

The luminal exposure to different hemodynamic stimuli, high TSVI and low TAWSS, is associated with significant PB growth in humans, confirming recent findings on early atherosclerosis on animal models [3]. TSVI confirms its effectiveness as a biomechanical marker of atherosclerotic disease at different stages [1].

References

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