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Wall shear stress topological skeleton variability predicts myocardial infarction

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Introduction

Coronary atherosclerotic plaques experience a variety of hemodynamic stimuli linked to plaque progression and destabilization that increases the risk of occlusion[1]. Peculiar wall shear stress (WSS) features obtained from patient-specific computational fluid dynamics (CFD) simulations have been associated with myocardial infarction (MI)[2]. Therefore, WSS-based quantities may prove to have predictive capability for MI, allowing the identification of lesions prone to rupture and improving the medical management of coronary artery disease (CAD) patients. Accordingly, the present study aims to explore the capability of WSS-based analysis derived from patient-specific CFD simulations in a clinical framework to predict MI.

Methods

Patients presenting with acute MI were retrospectively screened to identify those who had (1) a previous coronary angiography performed between 1 month and 5 years before the event, (2) a mildly stenosed lesion (i.e., ≤ 50% diameter stenosis) culprit for MI (future culprit, FC) at the baseline angiography, and (3) at least one additional lesion non-culprit for MI (non-future culprit, NFC) in the other two major epicardial vessels. This selection resulted in 80 patients and a total of 188 vessels, with 80 FC lesions and 108 NFC lesions.

The workflow of the study is presented in Figure 1. Three-dimensional vessel reconstructions, obtained using two angiographic projections, were discretized to perform transient CFD simulations using a finite element-based code (CAAS Workstation WSS software, Pie Medical Imaging). WSS-based analysis was focused on the widely adopted time average WSS (TAWSS) and the novel topological shear variation index (TSVI).[3,4] The TSVI quantifies the variability of the local contraction/expansion action exerted by the WSS on the endothelium along the cardiac cycle[5].

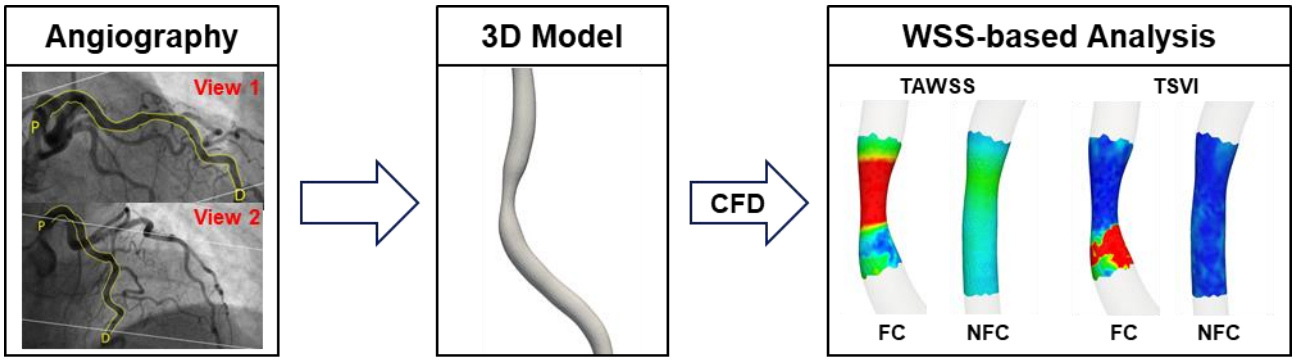


Figure 1. Workflow of the study

Results

As reported in Figure 2A, significant differences emerged between the FC and NFC distributions of lesion-average values of TAWSS (FC: 3.36 [IQR: 2.51-5.23] Pa; NFC: 2.94 [IQR: 2.36-3.80] Pa, p=0.011) and TSVI (FC: 71.08 [IQR: 44.42-116.20] m^{-1}; NFC: 33.21 [IQR: 24.77-60.11] m^{-1}, p<0.001). Notably, both TAWSS and TSVI were significantly higher in FC than NFC group, with a more marked statistical significance for TSVI. From ROC curves (Figure 2B), TAWSS resulted a moderate MI predictor (AUC=0.61; 95% CI: 0.53 to 0.69), while TSVI emerged as a good MI predictor (AUC=0.77; 95% CI: 0.70 to 0.84).

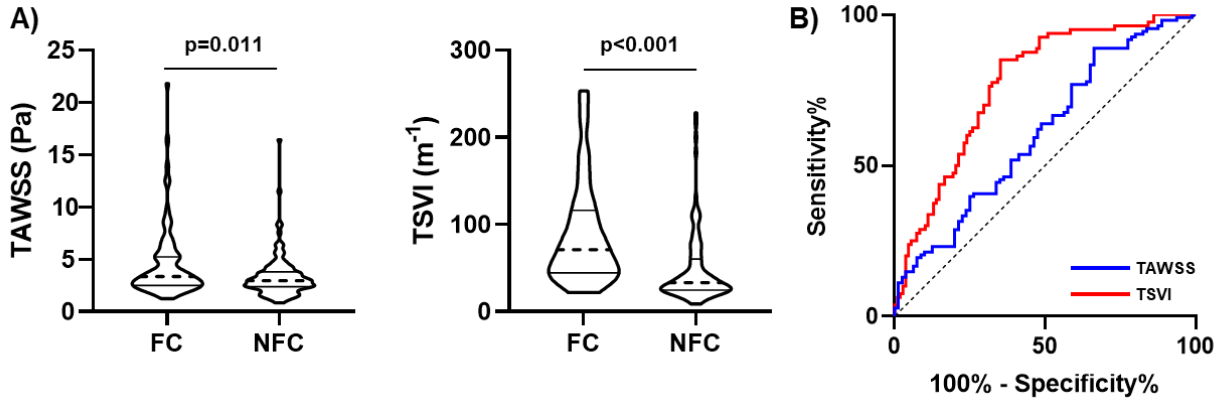


Figure 2. A) violin plots of TAWSS and TSVI averaged over the lesion. B) ROC curved based on TAWSS (blue) and TSVI (red) prediction of MI

Conclusion

The obtained results enforce the hypothesis that (1) WSS is a main actor in CAD, and (2) TSVI represents a relevant hemodynamic cue. The emergent predictive power of TSVI expands its association with long-term restenosis after carotid endarterectomy[3] and early atherosclerosis in coronary arteries[4]. A high temporal variability of the WSS contraction/expansion action may result in fibrous cap fragility, accelerated disease progression, plaque fatigue, ending in plaque rupture and subsequent MI. This hypothesis, which needs further investigation, is also supported by the ability of TSVI to identify blood flow stagnation, recirculation and separation regions, usually classified as flow disturbances. In conclusion, high TSVI resulted a strong predictor of MI in the analyzed population, encouraging further clinical trials to enforce the presented results and translate this concept into clinical practice.

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