VORTICITY TRANSPORT IN ABDOMINAL AORTIC ANEURYSMS WITH FOLLOW UP

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Introduction

Local blood flow disturbances play a key role in progression of the Abdominal Aortic Aneurysm (AAA), because they alter transport of biochemicals and fluid-wall interactions [1]. Therefore, a multitude of hemodynamic quantities has been proposed over the years to provide biomechanical markers of AAA evolution. However, these proposed quantities often fail in predicting aneurysm wall pathophysiology and remodeling, and in discriminating rupture risk based on hemodynamics. These results suggest that a different perspective to explore richness of fluid structures in AAAs is needed. Starting from the observation that the dynamics of large-scale vortices dominates the AAA hemodynamics, we propose a thorough characterization of the complex vortex structures produced and transported in the AAA, aiming at identifying vorticity-based quantities to be tested as potential markers of AAA progression, based on longitudinal data.

Methods

The lumen and the intraluminal thrombus (ILT) of three AAAs with their two years follow-up were reconstructed from CT-scan data. CFD was used to numerically solve the governing equations of fluid motion, using the finite element open-source code SimVascular [2]. The results from CFD simulations were used to quantify the vorticity transport equation:

$$\frac{\mathrm{D}\,\boldsymbol{\omega}}{\mathrm{D}\,t} = (\boldsymbol{\omega}\cdot\nabla)\boldsymbol{u} + \nu\Delta\boldsymbol{\omega} \qquad (1)$$

where $\boldsymbol{u}$ is the velocity vector, $\boldsymbol{\omega}$ is the vorticity vector and ν is the kinematic viscosity. The material derivative of the vorticity is given by the contribution of a stretching term (first term on the right side), quantifying the vortex lengthening due to velocity gradients, and of a term quantifying vorticity diffusion due to viscosity (second term on the right side). The vortex dynamics was here analyzed also in terms of the local swirling strength (λ_{ci}).

Results

Examples of instantaneous volumetric maps of the vorticity stretching term after systolic peak and at mid-diastole are presented in Figure 1 together with the time-histories of the volume-average values of the stretching term and of the swirling strength for one explanatory model and its two-years follow-up model. Both at baseline and at follow up, the AAA inflow jet rolls up into vortex rings transported inside the expansion region by the experienced pressure-gradient. Such vortex rings undergo stretching and tilting along the systolic deceleration phase and break-up in smaller vortical structures in diastole in the distal part of AAA sac in correspondence of the greatest ILT growth over time (gray arrows in Figure 1A). The vorticity in the AAA at baseline exhibits higher stretching and swirling strength than at follow-up, suggesting that those quantities may have contributed to AAA growth (Figure 1B).

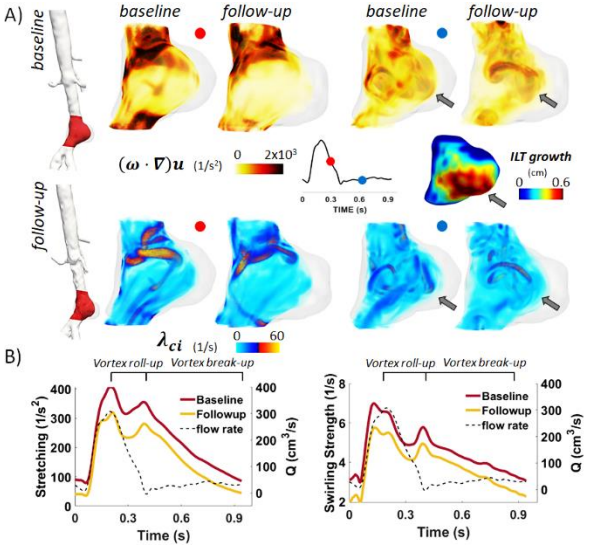

Figure 1: A) Volumetric maps and B) time-histories of the volume-average value of swirling strength and stretching in a AAA model and its two-years follow-up.

Discussion

In this study, a vorticity transport-based analysis is proposed on computational hemodynamics models of AAA, aiming at deciphering their intricate hemodynamics in a longitudinal investigation. A deeper understanding and interpretation of vorticity transport in AAAs could contribute to elucidate the role of flow disturbances in ILT formation, platelets dynamics, and inflammatory mechanisms [3], and in general of those hemodynamics-driven processes underlying AAA progression, potentially improving risk assessment and the clinical management of AAA patients.

References

1. Arzani, A et al., Phys Fluids, 24(8), 2012
2. Updegrove et al, Ann. Biomed Eng, 45(3):525-541, 2016
3. Biasetti, et al., J. R. Soc. Interface,8:1449–1461, 2011