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► To cite this version:

Kexin Yan, Wenfeng Ye, Michel Rochette, Pascal Haigron, Aline Bel-Brunon. Weak coupling between CFD and growth simulations to predict the evolution of an ascending aortic aneurysm. 2024. hal-04737965

HAL Id: hal-04737965

<https://hal.science/hal-04737965v1>

Preprint submitted on 15 Oct 2024

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Weak coupling between CFD and growth simulations to predict the evolution of an ascending aortic aneurysm

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Keywords: Finite elements; Growth and remodeling; Constrained mixture model; Ascending aorta aneurysm

1. Introduction

Predicting the evolution of an ascending aortic aneurysm (AscAA) is a challenge. Today the decision to perform surgery is based on the aneurysm diameter and more recently, on its evolution, measured on follow-up longitudinal images. However, due to the complexity of both the aortic geometry, the tissue behavior and the blood flow, these criteria may over- or under-estimate the risk of rupture or dissection in some cases. Numerical simulation of aneurysm growth could provide additional information on the tissue state to identify unstable cases requiring surgery.

In this work, we combine the hemodynamic study with a Growth and Remodeling (G&R) model based on the constrained mixture theory [1] to simulate a realistic ascending thoracic aorta. This study aims to assess the impact of flow biomarkers on the results of G&R and then compare the FSG (Flow-Structural G&R) results with CT longitudinal geometry to verify if our FSG model accurately describes the growth of AscAA.

2. Methods

The simulation framework consists in weakly coupled fluid and structural simulations using Computational Fluid Dynamics (CFD) and Finite Element (FE) methods respectively.

In the CFD simulation, we defined a flow jet perpendicular to the inlet surface with a pulsatile flow characterized by the mass flow rate shown in Figure 1. For the outlet boundary conditions, we used a Windkessel model. While turbulence models are generally employed in CFD simulations, as demonstrated in [5], the difference between turbulence and laminar models is minimal. Thus, to reduce computational cost, we opted for a laminar model. CFD simulation allows us to determine the distribution of time-averaged wall shear stress (TAWSS). As described by Mousavi et al. [2], Wall Shear Stress may cause endothelial damage and induce aneurysm growth. We use TAWSS as a fluid biomarker to define a zone of insult in our FE model, where elastin is degraded following Eq. (1):

$$\dot{q}^e(t) = -\frac{q^e(X,t)}{T^e} - \zeta_{TAWSS} \frac{D_{\max}}{t_{\text{dam}}} q^e(X,0) e^{-\frac{t}{t_{\text{dam}}}} \quad (1)$$

where $\dot{q}^e(t)$ is the rate of degradation of elastin, t_{dam} is the temporal damage spread parameter and D_{\max} is maximum damage, ζ_{TAWSS} is the normalized TAWSS, T^e is the turnover time of elastin.

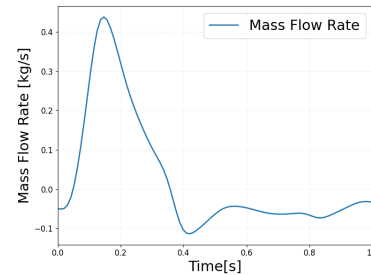


Figure 1: The mass flow rate follows the cardiac cycle and applied as the inlet boundary condition for the CFD simulation

For the FE simulation, we use a 3D structural mesh with hexahedral element to simulate G&R of an aneurysmal ascending aorta. The G&R model is implemented within FE software ANSYS with a coupled user material subroutine (USERMAT). In this model, we first apply a prescribed deposition stretch for the collagen and calibrate the deposition stretch of elastin so that the tissue reaches a homeostatic state [4]. It is therefore considered that all the tissue is in homeostatic state at the first step of simulation. The aortic wall is defined as a mixture of elastin, collagen and Smooth Muscle Cells (SMCs). We consider just one layer with different mass fractions of each

component, the strain energy density function is written as:

$$W = \varrho_t^e W^e(\bar{I}_1^e, J_{el}^e) + \sum_{j=1}^n \varrho_t^{c_j} W^{c_j}(I_4^{c_j}) + \varrho_t^m W^m(I_4^m) \quad (2)$$

where ϱ_t^i and W^i are the mass fraction and strain energy density of the i -th constituent, expressed in terms of first and fourth invariants. The strain energy of elastin W^e is described by a Neo-Hookean model and strain energy of collagen and SMCs W^c and W^m with a Fung-type function [1]. G&R consists in an evolution of ϱ_t^i ; elastin degradation is prescribed either homogeneous or by the above-mentioned local insult (Eq. 1); collagen can degrade and grow, its growth being defined by:

$$\dot{\varrho}^c = \varrho_t^c k_\sigma^c \frac{\sigma^c - \sigma_h^c}{\sigma_h^c} \quad (3)$$

where k_σ^c is a gain parameter and σ_h^c the homeostatic stress for collagen. In our case, we neglect the mass turnover of SMCs. During the simulation, the mass fraction of elastin varies according to the initial local insult defined by TAWSS (Eq. 1), and collagen deposition occurs following elastin degrades. (Eq. 3) As boundary conditions, we apply the heterogeneous systolic pressure field extracted from CFD, a mechanical support on the outer surface of aorta [3] and only enable radial movement on the inlet and outlet surfaces.

To illustrate the modelling method, a patient case is considered (Male, 62 YO, TAV), suffering from an ascending aorta aneurysm and for which a baseline and a 2-year follow-up CT-scans are available. This patient has a brachiocephalic artery and a left common carotid artery that share an origin. While we include the aortic arch and descending aorta to achieve more precise boundary conditions, our focus is primarily on the results for the ascending aorta. Therefore, we simplified the model of the aortic arch to include only two branches. The baseline geometry is obtained by segmentation; the simulation of growth is performed for 2 years with a time step of 1 month, to compute a simulated follow-up geometry to be compared to the ground-truth.

3. Results and discussion

Figure 2a displays the comparison between the baseline and follow-up geometries and shows that growth is localized on the upper outer curvature of the ascending aorta.

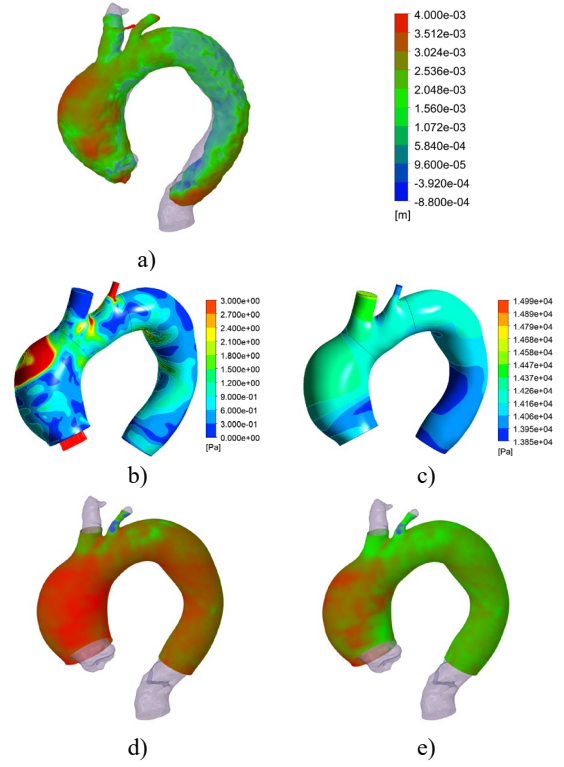


Figure 2: a) Distance between the baseline and 2-year follow-up geometries b) TAWSS field c) pressure field d) computed geometry after 2-years growth, for a homogeneous elastin degradation e) computed geometry after 2-years growth, for a TAWSS-driven elastin degradation

Figures 2b and 2c show the TAWSS and pressure fields respectively; TAWSS is particularly high in the area of observed growth due to the chosen normal inlet jet flow. The sensitivity of the inlet jet flow's effect should be studied in future research. While the pressure field, though higher on the outer curvature, is rather homogeneous.

Figures 2d and 2e correspond to 2-years growth computed by our simulation framework, either with a homogeneous elastin prescribed degradation (1d) or a TAWSS-driven elastin prescribed degradation (1e). It clearly shows that the pressure field heterogeneity cannot itself generate a localized growth, while the TAWSS-driven insult globally yields growth on the appropriate area.

4. Conclusions

Our study demonstrates that the combination of a CFD biomarker and a G&R model can aid in simulating AscAA growth more accurately and exhibits a correlation with a real patient longitudinal data. Limitation of our study are the following: it is not guaranteed that each tissue perfectly reaches the

homeostatic state at the initial time step; the inlet boundary condition does not account for the heart motion; the material parameters used in this study are not patient-specific. Future work will consist in identifying the G&R parameters based on several patients' longitudinal data.

Acknowledgements

This study was partially funded by ANRT (France) under CIFRE agreement n° 2021/1446.

Conflicts of Interest Statement

The authors declare that they have no conflict of interest.

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Received date:03/04/2024

Accepted date: 28/06/2024

Published date: XX/XX/2024

Volume: 1

Publication year: 2024