INFLUENCE OF FLUID-STRUCTURE INTERACTION IN A MODEL OF ATHEROMA PLAQUE GROWTH

Patricia Hernández-López (1), Myriam Cilla (1, 2), Estefanía Peña (1, 2), Mauro Malvè (2, 3), Miguel Ángel Martínez (1, 2)

1. *Aragon Institute of Engineering Research (I3A). University of Zaragoza, Spain*
2. *Biomedical Research Networking Center in Bioengineering, Biomaterials and Nanomedicine (CIBER-BBN), Spain*
3. *Public University of Navarra (UPNA), Pamplona, Spain*

Introduction

The consequences of atherosclerosis are one of the leading causes of mortality in developed countries today. Atherosclerosis can have serious outcomes, such as myocardial infarction, stroke or ischemia, depending on the affected artery. This disease causes a narrowing of the area available for blood circulation in blood vessels. This decrease in the lumen area is due to the formation of atheroma plaques in the arterial wall, caused by an increase in the endothelial permeability, which produces the flow of some substances from the bloodstream. The increase in the endothelial permeability may be due to several factors, including some mechanical stimuli caused by blood flow towards the arterial wall (such as Time Averaged Wall Shear Stress (TAWSS) or Oscillatory Shear Index (OSI), among others).

Methods

The formation of an atheroma plaque in an artery provokes changes in blood flow, and therefore in the mechanical stimuli that cause the aforementioned increase in the endothelial permeability. Therefore, we propose here a fluid-structure interaction analysis (FSI) based on a previously developed computational fluid dynamic model (CFD) of atheroma plaques development, to determine how these changes on the blood flow can affect the plaque growth.

There is a huge quantity of substances involved in atherosclerosis, but in the developed computational model we consider LDL, oxidised LDL, monocytes, macrophages, cytokines, foam cells, contractile and synthetic smooth muscle cells and collagen fibres. Due to the computational cost of a study in a three dimensional model, we analyse the effect of FSI on a two-dimension axisymmetric model.

We model blood flow in transient mode with Navier-Stokes equations, considering three cardiac cycles [1]. We also use Darcy's Law and Kedem-Katchalsky equations with the three-pore model [2] to calculate plasma and substance flows through the endothelium. Then, we use convection-diffusion-reaction equations to compute the inflammatory process in the arterial wall. The reactive terms of these equations are dependent on the considered substances. Finally, we model the arterial wall with a Yeoh hyperelastic constitutive law.

Due to big differences on the temporal scale of a cardiac cycle (ms) and the inflammatory process (several years), we have developed a semi-coupled model to address these different time scales.

Results

Our results compare atheroma plaques growth for the CFD and FSI models. Figure 1 shows the concentration of foam cells in the arterial wall, which are one of the substances that contribute most in the volume of the plaque. These results correspond to a calculation time of 10 years in both, the CFD and FSI models.

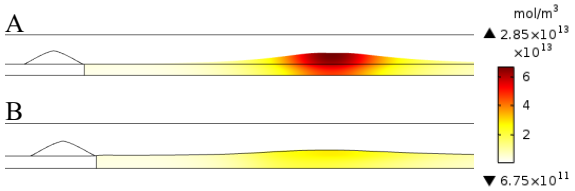

Figure 1: Concentration of foam cells for the CFD and FSI models (A and B, respectively).

As can be seen in Figure 1, the concentration of foam cells, as well as the growth of the plaque, are bigger for the case of CFD. In addition, it can be observed that, in the case of FSI, the arterial wall moves into the arterial lumen, whereas in the case of CFD this does not happen.

Discussion

A computational model of atheroma plaque formation has been developed, contrasting results for CFD and FSI. Results show that there is an important influence of the fluid-structure effect in the plaque formation.

References

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