THREE-DIMENSIONAL COMPUTER MODELING OF PLAQUE FORMATION AND THE LDL TRANSPORT WITHIN ARTERY AND THROUGH THE VESSEL WALL

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Introduction

Atherosclerosis is an inflammatory disease. Computational techniques and simulations have been developed to understand the blood flow dynamics and the mechanical behavior of the arterial wall.

We described mass transport of LDL through the wall and a simplified inflammatory process by coupling the Navier-Stokes equation, the Darcy equation for blood filtration and Kedem-Katchalsky equations for the solute and flux exchanges between the lumen and intima. A system of three additional reaction-diffusion equations is formed for the inflammatory process and lesion growth in the intima. Fluid-structure interaction is used for effective wall stress analysis.

Methods

The inflammatory process is modeled using three additional reaction-diffusion partial differential equations [Filipovic et al, 2012]:

$$\partial_t O = d_1 \Delta O - k_1 O \cdot M$$
$$\partial_t M + \mathrm{div}(v_w M) = d_2 \Delta M - k_1 O \cdot M + S/(1+S)$$
$$\partial_t S = d3 \Delta S - \lambda S + k_1 O \cdot M + \gamma(O - O^{thr})$$

where O is the oxidized LDL in the wall, M and S are concentrations in the intima of macrophages and cytokines, respectively; d_1, d_2, d_3 are the corresponding diffusion coefficients; λ and γ are degradation and LDL oxidized detection coefficients; and v_w is the inflammatory velocity of plaque growth, which satisfies Darcy's law and incompressibility continuity equation

In order to follow change of the vessel wall geometry during plaque growth, a 3D mesh moving algorithm ALE (Arbitrary Lagrangian Eulerian) is applied [Filipovic et al, 2006].

Results and Discussion

We compared changes in the cross-section areas for different patients with carotid artery progression. From 50 patients we choose two with significant evidence of MR plaque progression in order to estimate parameter for our model of plaque formation and development. From MR slices we segmented the inner and outer wall at nine cross-sections for baseline, three and twelve month times. It can be seen that almost all cross-section areas are increasing during follow-up time. Also using the generated results it can be concluded that in case patient #1 there is a significant correlation with large increasing of the cross-section areas and low wall shear stress shown in Figure 1 and large increasing in the cross-section area and low wall shear stress at the patient #2. Fluid-structure interaction method was implemented to analyze effective wall stress distribution. We fitted patient data for plaque volume progression with growth functions which depend on fluid shear stress and arterial wall effective stress. Matching computed plaque location, composition and volume progression over time with clinical observations demonstrates a potential benefit for future prediction of this vascular decease by using computer simulation.

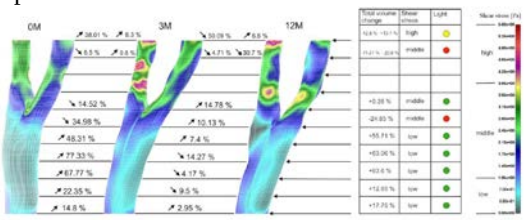

Figure 1: Correlation of cross-sections changes with wall shear stress for patient #1 red color denotes large decreasing in the cross-section area and middle wall shear stress, yellow color denotes small decreasing in the cross-section area and middle wall shear stress, while increasing in the cross-section area and low wall shear stress is the green color.

References

Filipovic et al, IEEE Trans Inf Technol Biomed, 16(2):272-278, 2012.

Filipovic et al, Comp. Meth. Appl. Mech. Eng. 195:6347-6361, 2006.