

Mass Transport of LDL in Stenotic Right Coronary Arteries

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Abstract: The blood flow and mass transport pattern of low-density lipoprotein (LDL) in a right coronary artery with two stenoses are studied. Computations were carried out under physiological conditions. Our results show a strong correlation between wall shear stress (WSS) and distribution patterns of LDL.

Keywords: Coronary artery; serial stenoses; LDL transport; wall shear stress

1 Introduction

Atherosclerosis is a cardiovascular disease of large- and medium-size arteries, in which plaque builds up inside arteries. Although real cause behind the formation of the plaque is still not fully established, low-density lipoprotein (LDL) is considered to be one of the main factors in causing atherosclerosis [1]. Therefore, revealing the transport of LDL macromolecules and blood flow in atherosclerotic artery is important in understanding the progression of the disease. Many studies have focused on computer simulations of LDL concentration in cardiovascular system [1-3]. Soulis *et. al.* examined the relationship between wall shear stress (WSS) and luminal surface concentration of LDL in the normal left coronary artery tree [2]. Chung and Vafai analyzed the LDL transport while incorporating the thickening of the arterial wall and cholesterol lipid accumulation [1]. The objectives of this research work are to study the mass transport pattern of LDL in a right coronary artery with two stenoses and the correlation between LDL distribution and wall shear stress (WSS).

2 Model

In the present study of blood flow and LDL transport in right coronary artery with two stenoses, the shape of the vessel is reconstructed based on the inlet and outlet diameters of the IVUS slices, the locations of the proximal and distal stenoses, and the centerline curve of the angiographic image of the coronary segment of a patient [4]. Both stenoses are moderate with a 75% of area reduction, as shown in Fig. 1. Blood flow was assumed to be incompressible, laminar, and non-Newtonian obeying the Carreau viscous model [5]. Time dependent 3D Navier-Stokes equations with no external force were used as the governing equations for blood flow. Blood density ρ was assumed to be constant at 1050 kg/m³. The unsteady state mass transport of LDL in flowing blood was described by a convection-diffusion equation. At the inlet boundary, a fully developed flow with a pulse waveform V(t) was imposed. This waveform was extracted from on-site blood flow data of the patient [4, 6] and was multiplied by a scalar to yield a typical average flow velocity under resting conditions of 0.17m/s. For the mass transport equation, a uniform concentration C_0 of LDL with 28.6*10⁻³ mol/m³ [1] was applied. At the outlet boundary, zero normal stress condition for blood flow and zero gradient of LDL concentration were imposed. A no-slip condition was applied to the flow on the wall. The boundary condition at the wall for the convection $c_w v_w - D \frac{\partial c}{\partial n} = K c_w$, where c_w is the concentration at the diffusion equation was described as

endothelial surface; $v_w = 0.6 \times 10^{-8}$ m/s is the transmural component of the fluid velocity at the wall; D = 15.0x10⁻¹² m²/s, is the diffusivity of LDL; *n* is the direction normal to the wall; K = 2x10⁻¹⁰ m/s is the

overall mass transfer coefficient of LDL at the artery wall [2, 3]. Computations were performed using COMSOL 5.3a, which applies a finite element method to solve the partial differential equations numerically over a tetrahedral mesh. Computations were repeated on refined meshes to endure a mesh independent solution and were performed over four consecutive cardiac cycles to ensure a truly periodic flow.

3 Results

Fig. 1(a) plots the contour of velocity along the central plan of the artery. Fig. 1(b)&(c) include the distributions of the WSS and the normalized LDL concentration (C/C_0) on lumen surface of the artery, respectively. Fig. 1(d) shows the path of the streamlines evenly spaced and started from the inlet boundary of the artery. All plots in Fig. 1 are corresponding to the data at the peak flow rate (t = 0.425s). From Fig. 1 we can see that LDL concentration elevates at the location where the WSS is low. The maximum LDL concentration occurs on the areas at the inner wall of the post-stenotic region of both proximal and distal stenoses, which are also the regions with disturbed flow.

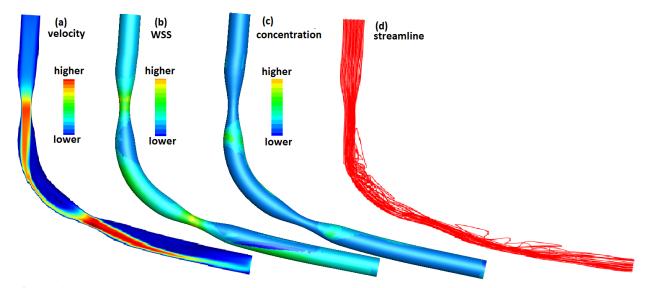


Figure 1. Streamline and contour plots of velocity, WSS and concentration at the peak flow (t = 0.425s)

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