

Chapter 13

Computer Predictive Model for Plaque Formation and Progression in the Artery

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ABSTRACT

In this chapter we described predictive model for plaque formation and progression in the coronary and carotid artery. A full three-dimensional model for plaque formation and progression, coupled with blood flow and LDL concentration is analysed. The Navier-Stokes equations together with the Darcy law for model blood filtration and Kedem-Katchalsky equations are implemented. Additionally, the system of three additional reaction-diffusion equations for simulation of the inflammatory process is coupled with full incremental iterative procedure. We developed hybrid genetic algorithm for fitting parameters of ODE model for oxidized LDL, macrophage, smooth muscle cell and foam cell concentration evolution in time. The animal carotid and coronary artery after 2 month of high fat diet are examined. We compared with CT our computer model of the plaque size for three groups of patients: De-novo, Old-lesions and Control patients. Detailed shear stress distributions for baseline and follow-up for these patients are given. There is a good matching for plaque size and location.

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INTRODUCTION

Atherosclerotic cardiovascular disease (CVD) is a chronic disorder developing throughout life and usually progressing to an advanced stage by the time symptoms occur. It remains the major cause of death in Europe, even though CVD mortality has fallen considerably over recent decades in many European countries.

CVD is strongly connected to lifestyle, especially the use of tobacco, unhealthy diet habits, physical inactivity, and psychosocial stress. It is well known that over three-quarters of all CVD mortality may be prevented with adequate changes in lifestyle.

The treatment of atherosclerosis is currently based on lipid lowering in combination with anti-inflammatory therapies that slow the progression of atherosclerosis. However, these therapies are not able to fully inhibit the formation or progression of atherosclerotic lesions. It has been proven that efficacy of these strategies in different clinical trials give only 30% to 40% (Shah 2007).

Lifestyle modifications including diet, exercise, and weight control are recommended for the treatment of dyslipidemia and associated coronary artery disease (CAD). The benefits of each of these modifications on health are dose-responsive (Haskell et al., 2007).

There is increasing evidence that exercise training can reduce endothelial dysfunction and the progression of atherosclerosis. Exercise training makes better the bioavailability of nitric oxide, diminishes the level of inflammatory markers, and can enhance the numbers of circulating endothelial progenitor cells (Ajijola et al., 2009). Exercise can reduce EPCs in the bone marrow (Rehman et al., 2004), with the potential of reducing atherosclerosis (Rauscher et al., 2003).

Atherosclerosis is a progressive disease characterized by the accumulation of lipids and fibrous elements in the large arteries. In the last decade, scientists come to appreciate a prominent role for inflammation in atherosclerosis. Formerly focused

on luminal narrowing due to the bulk of atheroma, the current concepts recognize the biological attributes of the atheroma as key determinants of its clinical significance (Libby 2002).

Inflammatory process starts with penetration of low density lipoprotein (LDL) in the intima. This penetration, if too high, is followed by leukocyte recruitment in the intima. One endothelial-leukocyte adhesion molecule has emerged as a particularly attractive candidate for the early adhesion of mononuclear leukocytes to arterial endothelium at sites of atheroma initiation: Vascular cell adhesion molecule-1 (VCAM-1). This process may participate in formation of the fatty streak, the initial lesion of atherosclerosis and then in formation of a plaque (Kaazempur-Mofrad et al., 2001).

Several mathematical models have recently been used for the transport of macromolecules, such as low-density lipoprotein, from the arterial lumen to the arterial wall and inside the wall (Tarbell 2003, Zunino 2002, Quarteroni et al 2002). These models are usually classified in three categories according to the level of description of the arterial wall. The simplest model is called the wall-free model, since in this model the arterial wall is simply described by means of an appropriate boundary condition. Kaazempur-Mofrad & Ethier 2001 simulated the mass transport in a realistic human right coronary artery and Wada et al., 2002 used a wall-free model to study the concentration polarization phenomenon. The wall-free model does not provide any information on the transmural flow and solute dynamics in the arterial wall. The fluid-wall models can be either single-layer or multilayer for the solute dynamics not only in the lumen, but also in the arterial wall. Stangeby & Ethier 2002 analysed the wall as single layer porous medium and solved the coupled luminal blood flow and transmural fluid flow using Brinkman's equations. Al & Vafai 2006 used multilayer models which represent intima and media separately. Olgac et al., 2008 used a three-pore model for LDL transport. Crosseto et

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