MODELLING OF ATHEROSCLEROTIC PLAQUE GROWTH USING FLUID–STRUCTURE INTERACTION

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Blood flow through a narrow arterial tube has been a classical mathematical problem dating back to the 1840s, after the pioneering experimental work conducted by Poiseuille [3] in 1836. However, numerical simulations of atherosclerosis only started to thrive in the 1990s, due to rapid advancement in computer technology. Many numerical models have been developed to study the behaviour of blood flow through a stenosed artery. There has been a steady progression in the complexity of the models, providing greater insight into different aspects of the biological process of atherosclerosis development. Such a process is, otherwise, extremely difficult to investigate experimentally. Some of the modelling complexities discussed in this thesis are:

(1) the pulsatile non-Newtonian blood flow condition with a Casson fluid model describing the blood rheology;

(2) the hyper-elastic artery wall governed by a neo-Hookean model for the wall material property;

(3) the two-way fluid–structural interaction (FSI) method providing more realistic simulations by considering not only the impact of the locally restricted fluid flow on the artery wall, but also the influence of the wall deformation on the fluid flow behaviour;

(4) a series of 3-D axis-asymmetric stenosis models with the severity level ranging from 45% to 79%;

(5) plaque morphologies varying from an initial bi-elliptical cross-sectional profile to an elliptical and a growth-updated arbitrary profile during the course of a plaque growth;

(6)   the plaque internal structure consisting of the diseased fibrous cap and a lipid pool of various sizes for adopting their own physiological material properties;

(7)   the use of a physiological blood flow profile.

The transportation of low-density lipoprotein (LDL) enables lipids like cholesterol and triglycerides to be carried within the water-based bloodstream. Numerous research papers have shown that LDL particles would adhere on the artery wall at the locations where the local wall shear stress (WSS) is too low to move them further, resulting in localised regions with high LDL concentrations. Atherosclerosis tends to develop at regions where LDL accumulation is high and consistent. It is well known in the medical community that a high level of LDL is closely related to myocardial infarction and/or stroke due to rupture of atherosclerotic plaques. Some rupture would release highly concentrated lipids and thrombogenic material into the bloodstream, leading to lethal blood clots that result in sudden cardiovascular casualties. In order to explore the dominant phenomenological mechanism, the thesis hypothesises that LDL accumulation has the sole influence on plaque growth. Therefore, the main objective of the thesis is to investigate how LDL accumulation affects the plaque morphology during its growth, aiming to provide a better understanding of atherosclerosis development.

Since LDL accumulation is directly linked to WSS disturbance of the blood flow, the WSS and fluid pressure are the primary concerns of the study. The first stage of the study is to validate the 3-D 45% axis-symmetric stenosis model with plaque morphology of a bi-elliptical cross-sectional profile for numerical accuracy [1]. In the second stage of the study, the model geometry is expanded to 3-D axis-asymmetric stenosis models with the same bi-elliptical morphology but two severity levels represented by the mild (45%) and the critical (78%) ones, respectively. These models are solved by applying the first four modelling complexities listed above. The numerical results show that the peak WSS is at the throat of the stenosis and the maximal WSS occurs in the systolic phase and the minimal WSS in the diastolic phase of the cardiac cycle. The fluid pressure behaves linearly, except at the stenosis contraction, where a sudden pressure drop is observed.

The third stage of the study models the growth of atherosclerotic plaques [2]. The mild 45% axis-asymmetric stenosis model with the bi-elliptical cross-sectional plaque morphology is used as the base model. By applying the hypothesis as the rule of growth, the plaque morphology is updated to a nonelliptical arbitrarily shaped profile across the centre of the plaque in the direction of the flow and elliptical profiles at various cross sections of the plaque that are perpendicular to the flow direction. The updated plaque morphology is determined according to the simulation results of WSS distribution in the vicinity of the previous plaque and the relationship between the WSS and LDL accumulation derived from the literature. The growth-updated model is then used as the new geometry for the next round of simulation. This process repeats until the stenosis severity is increased to 79%, which is 1% greater than the critical stenosis as reported in the literature. The numerical results of these growth-updated models

presented and discussed in the thesis are extensive, providing valuable insight into the plaque development.

The fourth stage of the study is to investigate the influence of the lipid pool size on the plaque growth and rupture. The size of the lipid pool and the stiffness of the fibrous cap are different in many cases of diseased arteries, affecting the flow behaviour. Considering these factors, the plaque geometry is further updated to include a lipid pool of either a small or a large size, along with different material properties for the artery wall and fibrous cap. Results show that lipid pool size plays a controlling role in stress levels within the stenosis. The plaque with a larger lipid pool is shown to have much higher stress within its fibrous cap, dramatically increasing the likelihood of plaque rupture. Altering the material properties of the artery wall results in excessive stress levels within the fibrous cap, which also leads to possible plaque rupture.

The final stage of the study compares plaque growth modelling between a sinusoidal pulsatile blood flow profile against a physiological blood flow profile. Results indicate that more horizontal growth is evident when a physiological blood flow profile is applied at the inlet boundary. The amounts of the vertical growth resulting from the sinusoidal and physiological flow profiles show no major differences. These results further contribute to the current understanding of plaque growth and rupture.

References

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