Effect of nonlinear blood viscosity on LDL transport and fluid-structure interaction biomechanics in a multi-stenosis left circumflex coronary artery

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Abstract. Low-density-lipoproteins (LDL) are widely associated with the initiation and progression of atherosclerosis. Biomechanical analysis has the potential to describe the highly nonlinear relationships between LDL transport, blood viscosity and wall shear/artery stress. To address this, we propose a fully-coupled fluid-structure interaction (FSI) model of an artery with two lipid-rich plaques. Variation in blood haematocrit is modelled using non-Newtonian power law models to assess the influence on LDL transport. Results show increased low-strain viscosity leads to decreased wall shear and LDL mass concentration; this result highlights the importance of patient specific fluid properties in biomechanical/LDL transport models.

Introduction

Atherosclerosis is a leading cause of myocardial infarction (heart attack) globally, with its pathophysiological representation often seen by an obstruction of flow or thrombus due to plaque rupture [1]. In particular, low-density lipoprotein (LDL) is known to be correlated to atherosclerotic plaque formation and wall shear stress results [2], making biomechanical analysis a useful tool for assessing potential disease sites. Literature, however, has not considered the effect of nonlinear blood viscosity [3] on LDL transport in fluid-structure interaction (FSI) models. Here we develop a dynamic, fully-coupled FSI model [4] based on angiography and include two lipid rich plaques in high risk areas (namely bends with low wall shear stress regions) and hyper/viscoelastic artery walls. We model blood as a non-Newtonian continuous phase, varying viscosity with haematocrit, and LDL as a discrete phase with concentration and properties adapted from the literature [5].

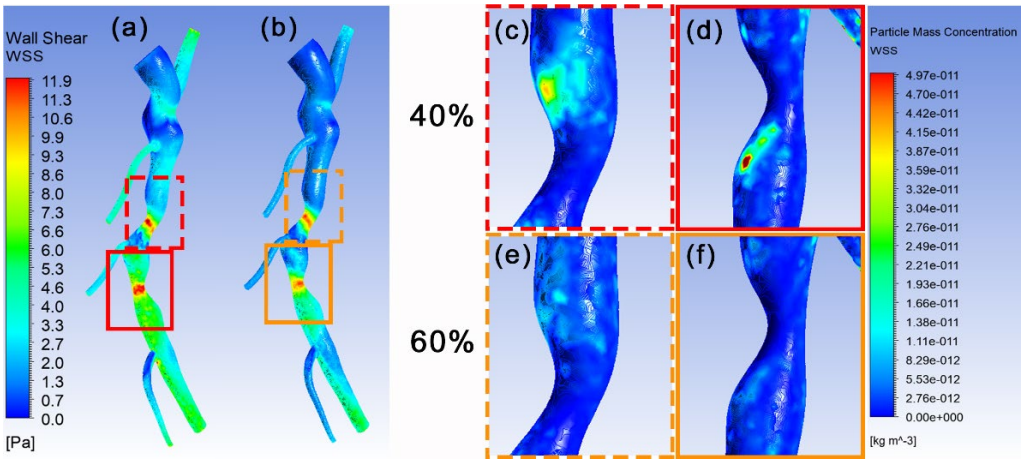

Figure 1: Wall shear stress at t=0.65 s for 40% (a) and 60% (b) haematocrit and LDL particle concentration comparison for the two haematocrit values at t=0.65 s for the first plaque (c, e) and the second plaque (d, f).

Results and discussion

Peak wall shear stress is lowered by up to 21% for a haematocrit of 60%, as described in Fig. 1(a-b). Plaque site one (c, e) and site two (d, f) both see a significant drop in LDL concentration for higher haematocrit values. Artery stress is not significantly affected. Results present interesting conclusions, with decreased wall shear stress and increased low strain rate viscosity leading to decreased LDL mass concentration, suggesting lower viscosity in the continuum phase could lead to an overestimation of LDL particle concentration at the artery wall, hence, an overestimated risk of plaque formation/growth.

References

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