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Non-Newtonian Effects on the Wall Shear Stress of the Blood Flow in Stenotic Right Coronary Arteries

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Abstract The effects of the non-Newtonian blood viscosity on the wall shear stress (WSS) of the blood flows in stenotic right coronary arteries have been investigated by computer simulations. The numerical calculations were performed using the Newtonian Model and the non-Newtonian models with the fluid obeying the Power Law and the Carreau models for the simulations of unsteady blood flows. The differences on the spatial and temporal WSS distribution patterns due to the different blood properties were compared. The computational results demonstrate that the blood viscosity properties do not affect the spatial WSS distribution pattern qualitatively. The region on the inner wall distal to the stenosis experiences a constantly low WSS during an entire cardiac cycle and the WSS in this region is lower than 1 N/m^2 anytime in the cardiac cycle. However, blood viscosity properties have considerable effect on the magnitude of the WSS, especially where disturbed flow occurs. The Newtonian model and the Carreau model show a good agreement quantitatively in the WSS along the artery wall. The Power Law model results in a much lower WSS where and when the WSS is relatively high, especially towards the end of the cardiac cycle.

Keywords: Wall shear stress, stenotic coronary artery, non-Newtonian.

1 Introduction

The wall shear stress (WSS) is widely believed to be one of the important local biomechanical factors responsible for the initiation and progression of atherosclerosis [1, 5]. Low WSS values are associated with low local velocity, which increases residence time and interaction between the blood lipoproteins and vessel endothelium. Long residence time with endothelium results in an increased lipoprotein intake and therefore the final outcome might be the thickening of arterial wall. A detailed hemodynamic evaluation of the spatial and temporal WSS distributions may give additional insight to understanding the progression of atherosclerosis and may have useful clinical value.

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During the last two decades, a lot of researches have been conducted to study the WSS distribution and to investigate the correlation between the WSS and the intima-media thickness [1, 5, 6]. Ku et al. [5] have estimated the shear stress with laser-Doppler anemometry and found that the intimal thickening bears an inverse relationship to both the maximum and the minimum shear stress. Gibson et al. [1] investigated the relationship between the vessel wall shear stress and the rate of atherosclerosis progression by means of quantitative angiography. Tang et al. [6] investigated the correlation between the human carotid atherosclerotic plaque progression with the plaque wall stress and the fluid shear stress based on in vivo patient tracking MRI data. Many numerical simulations have also been performed to examine the non-Newtonian effects on the pattern and distribution of the blood flow in human arteries [2, 3]. Some studies found non-Newtonian rheology important [2], while others suggested that under normal physiological conditions, the non-Newtonian effects may not be significant [3]. However, very few work has been focused on stenotic right coronary arteries. The objectives of the current work are to study the WSS distribution in a stenotic right coronary artery and to examine the effects of non-Newtonian blood properties on the spatial and temporal WSS distributions during a cardiac cycle.

2 Modeling

The blood is treated as an unsteady laminar, incompressible and non-Newtonian fluid obeying the non-Newtonian Carreau model with the viscosity-shear rate relation:

$$\eta = \eta_\infty + (\eta_0 - \eta_\infty)[1 + (\lambda\dot{\gamma})^2]^{\frac{n-1}{2}}, \qquad (1)$$

where η_0 = 0.056 *Pa·s* is the zero shear rate viscosity, η_∞ = 0.00345 *Pa·s* is the infinite shear rate viscosity, λ = 3.313*s* is a parameter, and n = 0.3568 is a dimensionless parameter [3]. Even though many non-Newtonian models of blood rheology have been used in blood researches, no one model is universally accepted. To investigate the influence of blood viscosity properties on the distribution of the WSS, computer simulations were also performed using other two models: 1) considering the blood as a Newtonian fluid with a constant viscosity 0.00345 *Pa·s*; 2) considering the blood as a non-Newtonian fluid obeying the Power Law with the viscosity-shear rate relation:

$$\eta = \eta_0(\dot{\gamma})^{n-1}, \qquad (2)$$

where η_0 = 0.035 *Pa·s*, and the Power Law index *n* equals to 0.6 [3]. The computational domain is a simplified geometry shown in Fig. 1(a), re-constructed based on the in vivo intravascular ultrasound (IVUS) image of a patient [7]. The radius of the cross-section at the inlet boundary is 0.00145*m*. The area stenosis severity is 50%. The inlet boundary is imposed with a fully

developed flow with a physiological human right coronary waveform [Fig. 1(b)], scaled to yield a time averaged flow velocity of 0.199 *m/s* at the centre-line. A no-slip condition is applied to the velocities at the wall boundary, treated to be inelastic and impermeable. The outlet boundary is treated as an open boundary, with the normal derivative of the flow velocity set to zero. The blood density ρ is assumed to be constant at 1050 kg/m^3.

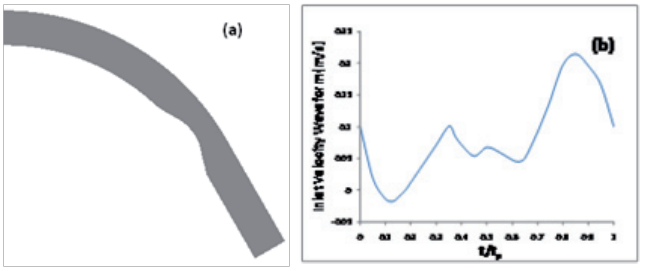

Figure 1: (a) Geometry of the computational domain with 50% stenosis; (b) Pulsatile coronary inlet velocity waveform

3 Observation and conclusion

Numerical computations were performed using Comsol Multiphysics. Navier-Stokes equations were solved using the finite element method with piecewise quadratic functions for velocity and piecewise linear functions for pressure over a tetrahedral mesh. Computations were repeated over different meshes to ensure the mesh independence of the numerical solutions. Four cycles were simulated to ensure that the flow was truly periodic.

Fig. 2 shows the contour plots of the WSS on the lumen surface for the baseline stenotic artery (with 50% stenosis by area) at the maximum flow rate (peak in diastole, t/t_p=0.85) based on (a) Carreau model, (b) Newtonian model, and (c) Power Law model, respectively. The location and magnitude of the maximum and the minimum WSS are clearly marked. We can see that all three models reveal basically the same spatial pattern of the WSS: The maximum WSS occurs at a point near the inner wall at the proximal side of the stenosis and the minimum WSS occurs at the inner wall distal to the stenosis region. But the magnitude of the WSS varies between models. The maximum and minimum WSS from the Carreau and Newtonian models are very close; However, the Power Law model shows a big difference compaerd to the other two models. The difference is significant where and when the WSS is relatively high (Plots for other t/t_p values are not included here). The Power Law model results in a much lower WSS at the peaks of the systole and diastole, especially towards the end of the cardiac cycle. The maximum WSS from the Power Law

model is only about a half compared to that from the other two modles when t/t_p=0.85.

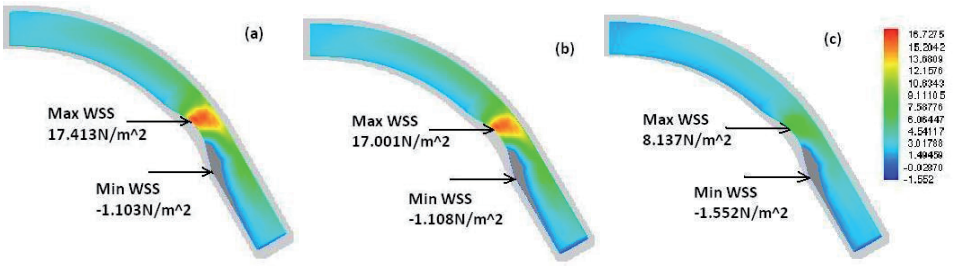

Figure 2: Contour plots of the WSS (N/m^2) along the wall at the maximum flow rate (t/t_p=0.85) (a) Carreau model, (b) Newtonian model, (c) Power Law model.

Evidence from in vitro and in vivo studies suggests that intimal thickening likely occurs when the average WSS is below 1 N/m^2 (=10 $dynes/cm^2$), which presents an inverse hyperlasia with respect to the shear stress [4]. Therefore, it is of special interest to know the size of the region on the surface of the artery wall where the WSS is lower than 1 N/m^2 and to examine the duration of the low WSS area in a cardiac cycle. To examine the size of the low WSS area on the inner wall distal to the stenosis, the WSS contour is plotted by replacing the area of the WSS < 1 N/m^2 with the dark grey color in the plots of Fig. 2. Comparing the grey areas in Fig. 2 (a), (b), and (c), we can see that the sizes of the low WSS areas are approximately the same for three different blood models. We observed that at each point along the artery wall, the WSS reaches the maximum at the peak flow of the diastole (t/t_p=0.85) during an entire cardiac cycle (Plots are not included). Based on this observation and Fig. 2, we can also conclude that the region on the inner wall distal to the stenosis experiences a constantly low WSS during the entire cardiac cycle and that the WSS in this region is lower than 1 N/m^2 during the entire cardiac cycle. The size of this low WSS area is the minimum at the peak flow of the diastole (showed in Fig. 2).

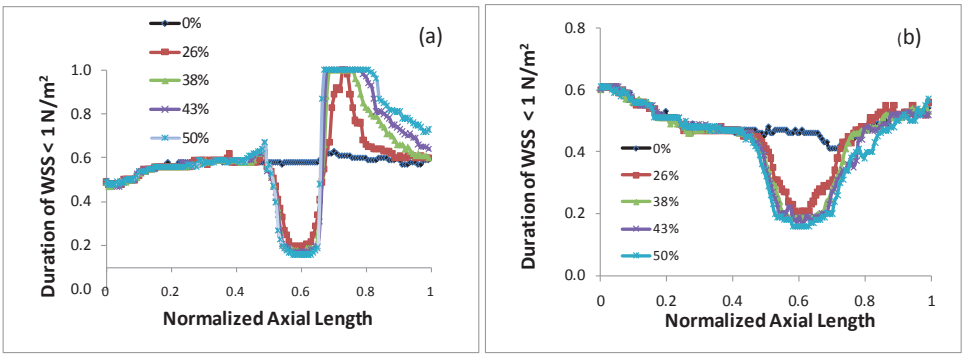

Figure 3: Duration of the WSS < 1 N/m^2 at each point on the inner wall (a) and on the outer wall (b) in a cardiac cycle.

At any point on the inner and outer walls, we calculated how long the location experiences a low WSS less than 1 N/m^2 (including the negative WSS resulted from the reverse flow or flow recirculation) in a cardiac cycle. Fig. 3 plots the duration of the WSS < 1 N/m^2 at each point on the inner wall (a) and on the outer wall (b) in a cardiac cycle for five arteries with stenosis sizes of 0%, 26%, 38%, 43%, and 50%, respectively. The horizontal axis is the normalized axial length of the artery. The stenosis stars at $x = 0.49$ and the neck of the stenosis is at $x = 0.62$. A point with a value of 1.0 on the graph indicates that the WSS at this point is lower than 1 N/m^2 anytime during the entire cardiac cycle. Fig. 3 shows the effect of stenosis size on the duration of the WSS < 1 N/m^2 at each point along the inner and outer walls. The size of stenosis does not affect the duration of the low WSS along the coronary artery segment proximal to the stenosis. Since the WSS elevates rapidly as the blood flows into the stenotic region proximal to the neck of the stenosis, the duration of the WSS < 1 N/m^2 in this region is much shorter along both inner and outer walls in a stenotic artery than that in a coronary artery with no stenosis (See Fig. 3). On the inner wall distal to the stenosis and downstream, the size of stenosis has a significant effect on the duration of the WSS < 1 N/m^2: The larger the size of stenosis, the longer of the duration of the low WSS that the region experiences, and the larger of the area of the low WSS region.

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