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Research note

Numerical simulation of LDL particles mass transport in human carotid artery under steady state conditions

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Abstract In this study, Lumen Surface Concentration (LSC) of Low Density Lipoprotein (LDL) particles in arteries with a permeable wall and up to 60% stenosis under steady state conditions, for Newtonian and non-Newtonian fluids, has been numerically investigated. The results show the Concentration Polarization (CP) phenomenon. Also, an increase in wall suction velocity (high blood pressure) and a reduction in Wall Shear Stress (WSS) are introduced as factors for an increase in LSC. Maximum LSC are observed for 40% stenosis.

1. Introduction

One of the most common arterial diseases is atherosclerosis. Atherosclerosis disease is the hardening of artery walls due to the growth of fatty plaques in medium and large arteries. Because fatty plaques are mainly composed of plasma lipoprotein particles, such as LDL, investigating the phenomenon of the mass transfer of LDL particles in the artery wall is an important subject in diagnosing this disease. Arterial diseases are the main cause of human fatalities in most parts of the world. Many studies show a direct relationship between flow patterns and atherosclerosis tissue.

In general, research activities in this field are classified into three categories: experimental, analytical and numerical methods. Wang et al. [1] studied experimentally the surface concentration of albumin in the carotid artery of a dog, and concluded that, due to CP, surface concentration is more than its bulk value in the flow, and, as wall suction velocity increases, LSC rises. Meng et al. [2] investigated CP and

showed that surface concentration is strongly and inversely dependent on WSS, and that disease develops in regions with low WSS. Shukla et al. [3], Chaturani and Samy [4] and Mistra and Chakravarty [5], considering blood analytically as a non-Newtonian fluid, found that the accumulation of cholesterol on an artery wall increases stenosis severity. Deng and Wang [6] numerically observed that under normal physiological conditions, LSC in a direct vessel is 5%–14% higher than the bulk concentration. LSC is also associated with fluid flow and changes linearly with filtration rate and, inversely, with WSS. These results are consistent with experimental results. Yang and Vafai [7] investigated the effect of blood pressure on LSC and concluded that an increase in blood pressure raises LSC, and is effective in disease development. Sun et al. [8], Olgac et al. [9], Soulis and Giannoglou [10] and Fazli et al. [11] showed that in regions with low WSS, the concentration of LDL particles is high.

Due to stenosis in the arteries and the existence of regions with low shear stress in the recirculation region, blood is treated as a non-Newtonian fluid, and it is necessary to consider the flow with non-Newtonian models. The aim of the present study is to investigate the factors affecting LSC. In this study, the artery wall is assumed rigid and permeable to plasma. To simulate the vessel wall, a wall-free model (lumen model) is used [12]. The advantages of this model are low computation cost and the achievement of qualitative information for mass transfer in blood lumen. This model is used by researchers to study the transfer of oxygen [13–15], LDL [16–18] and albumin [19].

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Nomenclature

C_0	Inlet concentration of LDL particles
C	Concentration of LDL particles
C_w	Wall concentration of LDL particles
D	Diffusion coefficient of LDL particles
$\boldsymbol{n}$	Direction perpendicular to the boundary
P	Pressure
r	Radial coordinate
R_0	Radius of vessel (3.5 mm)
Re	Mean inlet Reynolds number
Sc	Schmidt number ($\mathrm{Sc} = \nu/D$)
$\boldsymbol{U}$	Velocity vector
u	Velocity component in x-direction
U_0	Inlet average velocity
v	Velocity component in y-direction
V_w	Wall suction velocity (4×10^{-8} m/s)
w	Velocity component in z-direction

Greeks

ρ	Blood density (1050 kg/m^3)
μ	Blood viscosity (0.0035 kg/ms)
ν	Kinematic viscosity
τ_y	Blood yield stress.

2. Governing equations

The blood flow is assumed laminar, steady, incompressible and fully developed, and the blood fluid is assumed to be homogeneous, both Newtonian and non-Newtonian models are used.

To simulate the flow, continuity and Navier–Stokes equations are used.

$$\nabla \cdot \boldsymbol{u} = 0, \tag{1}$$

$$\rho(\boldsymbol{u} \cdot \nabla)\boldsymbol{u} = -\nabla P + \nabla \cdot (\mu \, \nabla \boldsymbol{u}). \tag{2}$$

In the case of the non-Newtonian model, the modified Casson model is used [20].

$$\mu = \left(\sqrt{\frac{\tau_y(1 - e^{-m\dot{\gamma}})}{\dot{\gamma}}} + \sqrt{\mu_c} \right)^2, \tag{3}$$

where, $\mu_c = 0.0035$ kg/ms, $m = 100$ s and $\tau_y = 0.01$ Pa for a hematocrit of 45% [21].

$\dot{\gamma}$ is shear strain rate and is defined as follows:

$$\dot{\gamma} = \left[2 \left\{ \left(\frac{\partial u}{\partial x} \right)^2 + \left(\frac{\partial v}{\partial y} \right)^2 + \left(\frac{\partial w}{\partial z} \right)^2 \right\} + \left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x} \right)^2 \right.$$
$$\left. + \left(\frac{\partial u}{\partial z} + \frac{\partial w}{\partial x} \right)^2 + \left(\frac{\partial v}{\partial z} + \frac{\partial w}{\partial y} \right)^2 \right]^{\frac{1}{2}}. \tag{4}$$

The mass transfer equation of LDL particles is described as follows:

$$\boldsymbol{u} \cdot \nabla C = D\nabla^2 C. \tag{5}$$

Because the size of LDL particles are variable, diffusion coefficient is in the range of 5×10^{-12} m^2/s to 2×10^{-11} m^2/s, and the Schmidt number, Sc, is in the range of 1.6×10^5 to 6.6×10^5 [16,22].

Table 1: Constant coefficient for stenosis severity.

Stenosis severity	30%	40%	50%	60%
λ	0.163	0.225	0.292	0.368

3. Geometry and boundary conditions

The artery is assumed an axisymmetric cylinder, with and without stenosis. The vessel wall is impermeable to LDL particles, and plasma with constant filtration velocity passes through it. Its dimensions are based on characteristics of the carotid artery, the diameter and length are 7 mm and 15.4 cm, respectively, and the distance of 2.8 cm from the beginning of artery is the stenosis region [16]. The geometry of the stenosis is described by the following bell-shaped Gaussian distribution profile [23].

$$R(x) = 1 - \lambda \, e^{-5x^2} \begin{cases} 0.163 \le \lambda \le 0.368 \\ |x| \le 4. \end{cases} \tag{6}$$

The value of λ depends on the stenosis severity (see Table 1).

Boundary conditions for Navier–Stokes equations are as follows:

At the inlet, a fully developed velocity profile is used. The average velocity (U_0), based on the average flow rate, 275 ml/min, is equal to 0.119 m/s, and the Reynolds number, Re, is 250.

$$u(0, r) = 2 \times U_0 \left(1 - \left(\frac{r}{R_0} \right)^2 \right). \tag{7}$$

At the outlet, the static gage pressure is set to zero. On the wall, the no slip condition and the radial filtration velocity are applied.

$$u(x, R_0) = 0, \tag{8}$$

$$v(x, R_0) = V_w. \tag{9}$$

Boundary conditions for concentration of LDL at the inlet, outlet and wall, sequentially, are as follows:

$$C(0, y) = C_0, \tag{10}$$

$$\left(\frac{\partial c}{\partial x} \right)_{(l,y)} = 0, \tag{11}$$

$$D \left(\frac{\partial c}{\partial \boldsymbol{n}} \right)_{(x,R_0)} = C_w V_w. \tag{12}$$

4. Numerical method

The structured grid is generated by Gambit software. To investigate the independency of the mesh, LDL concentration profiles along the wall and in the radial direction, at a section near the end of a simple artery (without stenosis), are considered for five different meshes with 18 900, 24 750, 31 500, 36 900 and 43 200 cells, and it is shown that 36 900 cells are adequate. For the artery with 60% stenosis, LDL concentration profiles in the stenosis region and in the radial direction for the number of meshes 52 800, 62 400, 74 880, 86 400, 96 000, and 105 600 are considered, and 96 000 cells have been shown to be adequate for this analysis. The governing equations are solved numerically by Ansys CFX software, using the finite volume and algebraic multigrid methods, based on iteration [24] and coupling pressure and velocity [25]. The convergence criterion for all equations is 10^{-6}, and they are solved using double precision.
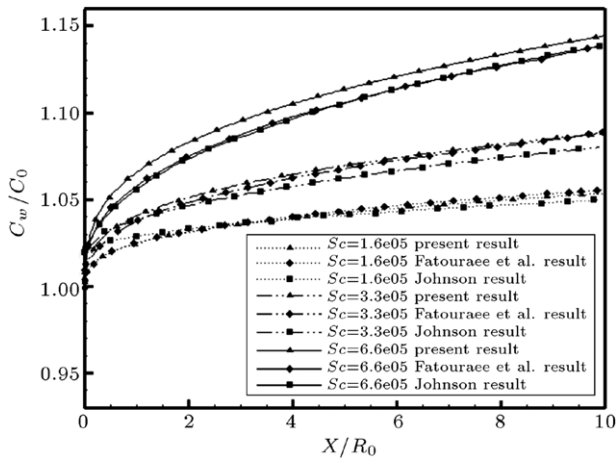
Figure 1: LSC for simple artery and Newtonian fluid.

Figure 2: LSC in terms of WSS for a simple artery and in the end of artery.

Figure 3: Effect of filtration velocity on LSC in a simple vessel.

5. Results and discussion

Numerical simulation of the mass transfer of LDL particles in arteries with and without stenosis, with a lumen model and with a filtration velocity of 10^{-8} m/s on the artery wall, under carotid artery steady flow, has been carried out. It should be noted that Sc is calculated based on the constant viscosity and the diffusion coefficient.

5.1. Concentration polarization (CP) phenomenon

Due to the existence of a filtration rate on the wall, and not passing through the endothelial, LDL particles accumulate on the artery wall, creating a very thin concentration boundary layer. This phenomenon is known as CP. In order to validate the numerical solution, LSC for the Newtonian fluid and the simple artery for various Sc, is given in Figure 1. These results have been compared with the analytical solution of Johnson et al. [26] and the numerical solution of Fatouraee et al. [16], and they are in good agreement. The results indicate higher LSC's at the end of the vessel compared to the concentration in the bulk flow for Sc of 1.6×10^5, 3.3×10^5 and 6.6×10^5 by 8.6%, 14.3% and 23.7%, respectively.

5.2. Effect of WSS on LSC

WSS is an important hemodynamic parameter and is an effective factor in the formation of atherosclerosis [27]. To investigate the correlation between WSS and LSC, Newtonian fluid flow in a simple vessel, with eight flow rates, is taken into consideration. Figure 2 shows that LSC decreased sharply at a low wall shear rate and, by increasing the inlet velocity and, consequently, increasing WSS, LSC decreases and then approaches a constant value asymptotically which is in agreement with results obtained by Ethier [28] and Deng and Wang [6]. As shown in regions where WSS is low, LSC is more sensitive to changes in the flow field.

5.3. Effect of filtration rate and blood pressure on LSC

By increasing the filtration velocity, the LSC of LDL particles, which cannot cross the vessel wall, is increased. From a medical point of view, high blood pressure is an important risk factor in the formation of atherosclerosis disease. The linear relationship
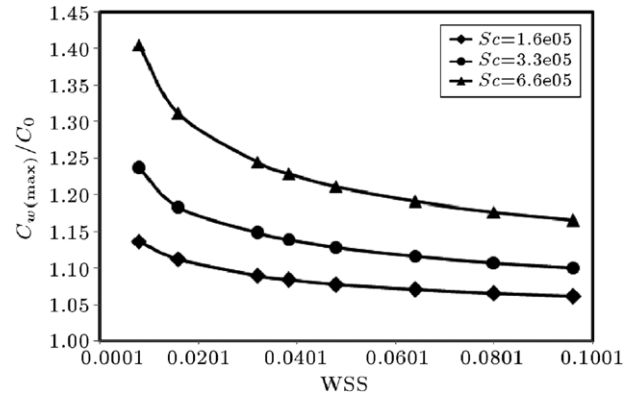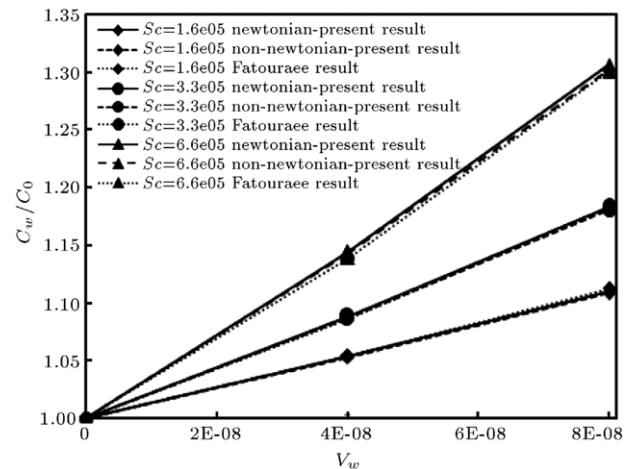
between filtration rate and blood pressure is expressed by Poiseuille's equation. To investigate the phenomenon of high blood pressure, a filtration velocity of 4×10^{-8} m/s, which corresponds to a blood pressure of 100 mmHg, and 8×10^{-8} m/s corresponding to 200 mmHg are used [1]. The filtration velocity 4×10^{-8} m/s belongs to normal blood pressure [29]. In Figure 3, the effect of filtration velocity on LSC for Newtonian fluid is shown. Also, these results have been compared with the numerical solution of Fatouraee et al. [16] and they are in very good agreement. The results suggest that by increasing the filtration rate, LSC increases linearly, and this is consistent with findings by Deng et al. [30], Deng and Wang [6] and Fazli et al. [11].

5.4. Effect of stenosis severity on LSC

Since accumulation of particles on the vessel wall is the primary cause of disease, in recent years, researchers have studied the transport of materials and the interaction of particles in the recirculation region [31]. In this region, LSC is increased and, therefore, is susceptible to disease progression. Figures 4–7 show WSS and LSC for Newtonian and non-Newtonian fluids for Sc $= 6.6 \times 10^5$. The results suggest that changes in LSC are well correlated with changes in WSS. It can be seen that for 30% stenosis, there is no separation (WSS > 0), and for non-Newtonian fluid, LSC is lower than that of Newtonian fluid, which is due to the difference in
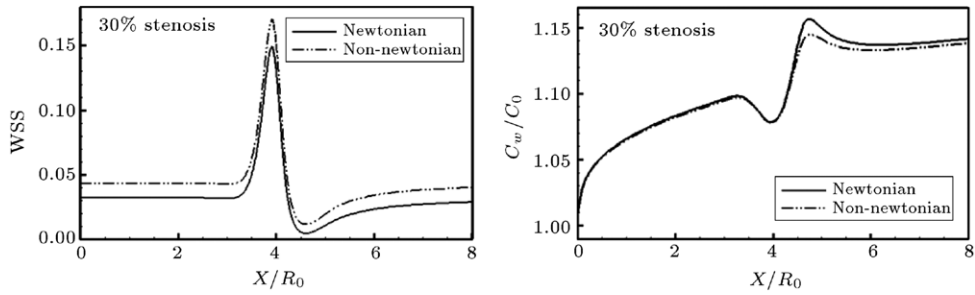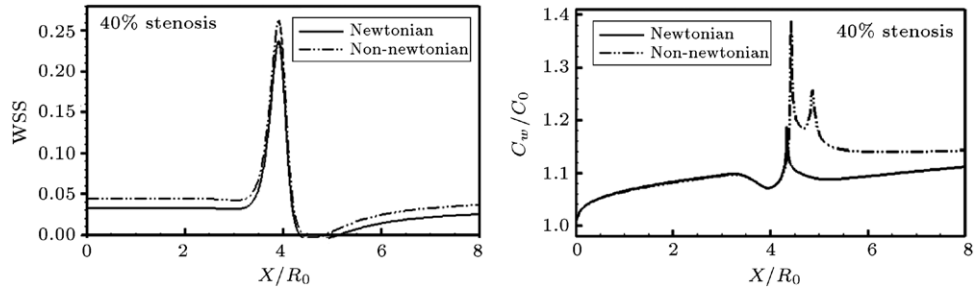
Figure 4: WSS and LSC for 30% stenosis.

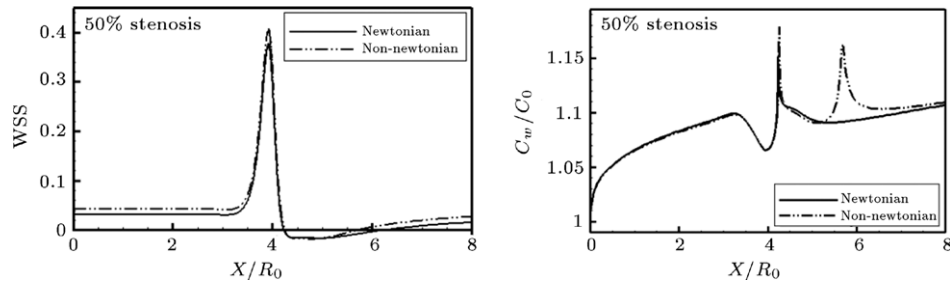Figure 5: WSS and LSC for 40% stenosis.
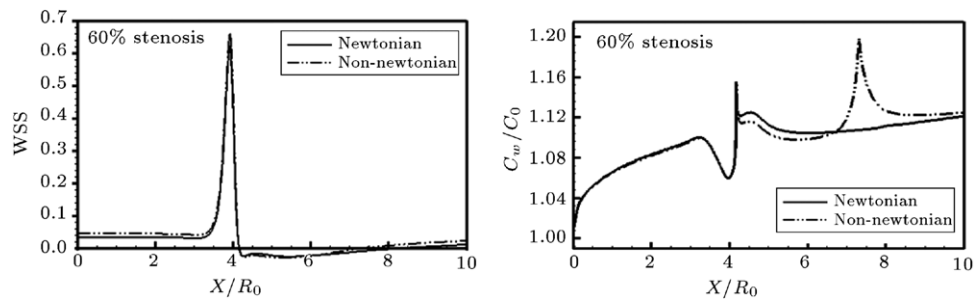

Figure 6: WSS and LSC for 50% stenosis.

Figure 7: WSS and LSC for 60% stenosis.

velocity profiles. For 40%–60% stenosis, flow separation occurs, which is evident from WSS curves. Due to the existence of the recirculation region, WSS is negative and its value for Newtonian fluid is more than that of non-Newtonian fluid, because the reverse velocity of non-Newtonian fluid is lower than that of Newtonian fluid. At the separation and reattachment points, where WSS is zero, LSC is higher for non-Newtonian fluid. The reason for the increase in LSC at the reattachment point is that in this point shear rate is zero,

and when the non-Newtonian model is used, viscosity and consequently the SC number strongly increases, as a reduction in the concentration boundary layer thickness will increase LSC. This finding is similar to the experimental observations of cholesterol uptake distribution along the stenosed arteries of dogs reported by Deng et al. [32] who showed that the surface concentration and, consequently, the uptake of the 3H-7-cholesterol in the arterial wall is elevated at the location of the reattachment point. Chen et al. [33] used an in-vitro reverse
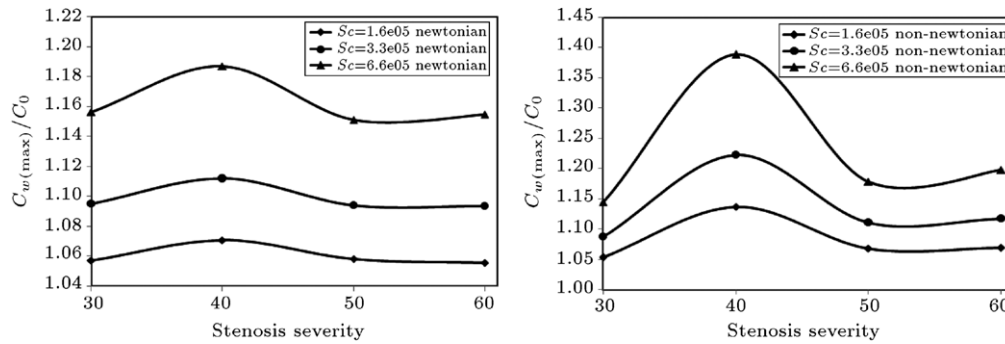
Figure 8: Maximum concentration in the separation region, in terms of stenosis severity for Newtonian and non-Newtonian models.

step model and showed that the particles in blood adhered and transmigrated more in the reattachment region than in the recirculation region. They stated that the reason for this event is the higher concentration of particles in this area.

Research findings suggest that, at separation and reattachment points, where WSS is low, LSC is high, which is an effective factor in the growth of plaque [22,30]. Also, angiography studies show that atherosclerosis plaques grow downstream of stenosis, where reduction in the velocity and instability is shown [34]. Unlike the non-Newtonian model, the Newtonian model (because it has constant viscosity) is unable to demonstrate the increase of LSC at the reattachment point. So, it can be concluded that the non-Newtonian model is more accurate than the Newtonian model. Also, it should be noted that for 40% stenosis, the concentration is highest compared to other stenosis, which is consistent with the findings of other researchers [11,35].

Figure 8 shows the maximum concentration changes in the separation region, with an increase in stenosis severity. The results show that the maximum LSC increases first, with an increase in the severity of stenosis, and then it tends to decrease. So, when stenosis starts, at first, it grows rapidly and then slowly goes forward, narrowing the section of the vessel.

6. Conclusion

The mass transfer of LDL particles in the carotid artery is numerically simulated, with symmetric 30%–60% stenosis, and without stenosis by the lumen model with a filtration velocity of the order 10^{-8} m/s under steady state flow. The results of the numerical solution show that, due to the occurrence of CP phenomena, in the Newtonian fluid, LSC is 8.6%–23.7% higher than that of the bulk flow. With increase in filtration rate (high blood pressure), LSC increase from 8% to 23%. By increasing stenosis severity, the recirculation region becomes larger, and the length of the recirculation region for non-Newtonian fluid is less than that of Newtonian fluid. As WSS increases, LSC decreases and then reaches an asymptotic value. Because of the drastic reduction of WSS at the separation and reattachment points, they are susceptible places for disease development. Also, unlike non-Newtonian fluid, Newtonian fluid is unable to demonstrate the increase of LSC at the reattachment point. So, it can be concluded that using a non-Newtonian model produces more accurate results compared with that of a Newtonian model.

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Conflict of interest

There are no financial and personal relationships with other people or organizations that could inappropriately influence (bias) their work.

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