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Effect of non-Newtonian behaviour on fluid structural interaction for flow through a model stenosed artery

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Abstract

The cause and development of many cardiovascular diseases are related to the nature of blood flow and the mechanical behaviour of the blood vessel. Moreover, the plaque (stenosis) rapture can be occurred as a result of interaction between the blood and plaque, leading to the clot formation and stroke. In the present study, the interaction of blood flow with plaque (stenosis) was numerically modelled. A pulsatile flow was used to mimic the real blood flow through the artery. The rheological properties of blood are considered as Newtonian as well as non-Newtonian. Fibrous cap thickness was varied from 0.1 mm to 2.0 mm. Many vortex rings are appeared at the pre- and post-stenotic region in the Newtonian model than in the non-Newtonian model. Deformation of stenosis, wall shear stress (WSS) and vomises stress all are found high in non-Newtonian model for the fibrous cap thicknesses studied here. Moreover, in Newtonian model, the vonmises stress was found to be 6500 pa for the case of 50% stenosis with 0.1mm fibrous cap thickness on the other hand it was around 10500 pa in case of non-Newtonian model.

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Keywords: stenosis; degree of stenosis, physiological flow; wall shear stress; wall shear stress gradient.

1. Introduction

Blood flow through the artery has the similar behavior with the fluid through a channel. This type of phenomenon can be described by the concept of fluid mechanics. This is known as Hemodynamics. Hemodynamics plays a great

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role to explain the fluid (Blood) flow behavior through the artery which is inherently unsteady. Structure of the blood vessel includes three layers: tunica intima (collagen and smooth muscle), tunica media (elastic fibers, collagen and smooth muscle), and tunica adventitia (connective tissues consisting of elastic and collagen fibers). There lies a small lumen at the center of artery through which blood flows at a very high pressure. However, due to bad food habit, lacking of physical exercise, oversleeping may cause serious plaque (stenosis) formation in the blood vessels.

The stenosis is defined as a partial constriction of the blood vessel due to the accumulation of cholesterols and fats and the abnormal growth of tissue. Plaque which actually constricts the blood vessel or cause of stenosis consists of Lipid core and fibrous cap. Fibrous cap which covers the lipid core is a thin domain and consists essentially of collagen and smooth muscle cells. The lipid core is formed by fatty composites. The disease caused by this is known as atherosclerosis[1]. Atherosclerotic plaques may rupture without warning under physiological conditions and cause fatal sub sequential diseases such as heart attack and stroke. The exact mechanism causing plaque rupture is not well understood. Stenosis severity has been widely used as a measure of seriousness of stenosis and basis for surgery decision. The stenosis causes the most frequent anomaly in blood circulation. Once the constrictions is formed ,the blood flow is significantly altered and fluid dynamical factors play important roles as the stenosis continues to enlarge leading to the development of cardiovascular diseases such as heart attack and stroke. The properties of the flow also have significant consequences on these cardiovascular diseases. Pressure losses in stenosed arteries become significant for severe stenosis leads to stroke or infraction, considered as one of the major causes of death and disability in humans. Many theoretical analyses have been assumed in the steady state. It is reported experimentally that there is a considerable difference between the blood flow and the water one through a stenosed tube and that the effect of the non-newtonian property of blood should not be neglected [2]. So,in the present study, Plaque interacts with the blood (both Newtonian and non-Newtonian) flow creating mechanical stresses which can lead to its rupture and which can produce recirculation downstream the plaque. Plaque rupture and recirculation can yield the formation of a thrombus and eventually lead to a heart attack. In this study, development of a FSI model in order to study the interactions between the blood flow and the plaque has been conducted and the effects of viscosity of the blood on this rupture process are studied.

In this research, a 2-D simplified model is considered to investigate the several fluid flow and structural behavior such as streamlines, vortex formation, and deformation of plaque, wall shear stress and vonmises stress for different cap thickness at different time for Newtonian and non- Newtonian model.

Nomenclature

H	model arterial diameter
S_0	degree of stenosis
T	time period
t	time instant
t_f	fibrous cap thickness
α	Womerseley parameter

2. Mathematical Modeling

A 2-D plannar (x, y) analysis has been considered in the present research. Blood is considered as a Newtonian, homogeneous and incompressible fluid as well as at the same time it is modeled as non-Newtonian Carreau model. Here modeled artery's width is H = 10mm as shown in Fig.1. The Governing equations for fluid domain are 2D Navier strokes equations and for solid domain structural properties are included for the Fluid Structural Interaction (FSI) study.

2.1 Fluid model

Governing equations are the Navier-Strokes equation as shown in vector notations:

$$\rho\frac{\partial\vec{V}}{\partial t}+\rho(\vec{V}.\nabla)\vec{V}=-\nabla P+\mu\nabla^2\vec{V} \tag{1}$$

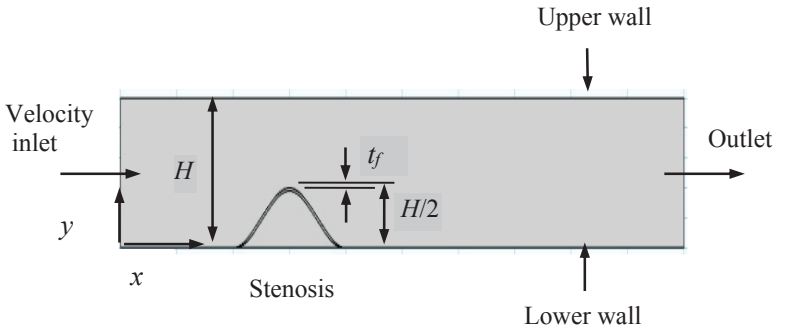

Fig.1. Schematic of an arterial stenosis

At solid-fluid interface boundary, the condition is governed by the following equations:

$$u_{fluid}=u_w \tag{2}$$

$$u_w=\frac{\partial u_{solid}}{\partial t} \tag{3}$$

At the inlet boundary the input velocity expression is given as follows and the waveform is shown in Fig. 2-

$$u_{mean}=u[1+a\sin(\omega\times{}^{t}/_{T})+b\cos(2\omega\times{}^{t}/_{T})] \tag{4}$$

where u = 0.1 m/s, $\omega = 6.28$ rad/s, a = 0.75, b = --0.75

The non-Newtonian model of the blood is governed by the Carreau model [1]:

$$\mu=\mu_{inf}+(\mu_0-\mu_{inf})\left[1+\left(\lambda\frac{\partial\gamma}{\partial t}\right)^2\right]^{\frac{n-1}{n}} \tag{5}$$

where, $\mu_0 = 0.4$ pa.s, $\mu_{inf} = 0.004$ Pa. s, $\lambda = 0.25$, $n = 1/3$

2.2. Structural model

A simple model of the artery with one-sided cosine shaped stenosis is considered in the present study which is identical to that of the experimental investigation of Ahmed and Giddens [3]. The shape of the stenosis is defined by following equation:

$$S(x)=\frac{S_0 H}{2}[1-cos\{2\pi(x-x_1)/(x_2-x_1)\}]/\ 2 \tag{6}$$

The lipid pool and the fibrous cap are modelled as isotropic nearly incompressible hyper elastic materials. For hyper-elastic materials, the stress-strain relationship is nonlinear and derives from a strain energy function W. For near incompressible material, the energy function W is splited into distortional and volumetric parts as shown by the following equation:

$$W=w(c)+U(j) \tag{7}$$

In the present study W is defined according to the Mooney-Rivlin model. Hence the energy function is stated by equation:

$$W_s=C_{10}(\bar{I}_1-3)+C_{01}(\bar{I}_2-3)+\frac{1}{2}\kappa(J_{el}-1)^2 \tag{8}$$

For fibrous cap the values of constants are C_{10} = 9200 Nm^{-2}, C_{01}= 0 Nm^{-2}, κ = 3000 MPa and for lipid deposit, the values of constants are taken as C_{10} = 500 Nm^{-2}, C_{01} = 0 Nm^{-2}, κ = 200 MPa [1]. The densities of fibrous cap and lipid deposits are considered as 1000 kg/m^3 [1].

The Womerseley number ($\alpha = H/2 \times \sqrt{2\pi\rho/T\mu}$), for the present investigation is found to be 8.26. The degree of stenosis, S_0 is kept constant at 50% as shown in Fig. 1. The computational domain with imposed boundary conditions are shown in Fig. 1. Pulsatile velocity (Fig. 2) inlet and constant pressure outlet conditions are imposed on inlet and outlet boundaries, respectively. All the arterial boundaries are given as no-slip boundary conditions. In the present study, the simulations are carried out using Finite element Method (FEM) where FSI is handled by means of ALE formulation [4]. The entire domain is subdivided into 7000~12424 number of triangular elements. Observation has been made at the point of maximum acceleration, maximum velocity and maximum deceleration as shown in Fig. 2.

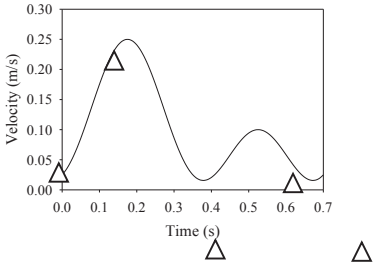

Fig.2. The inlet wave form

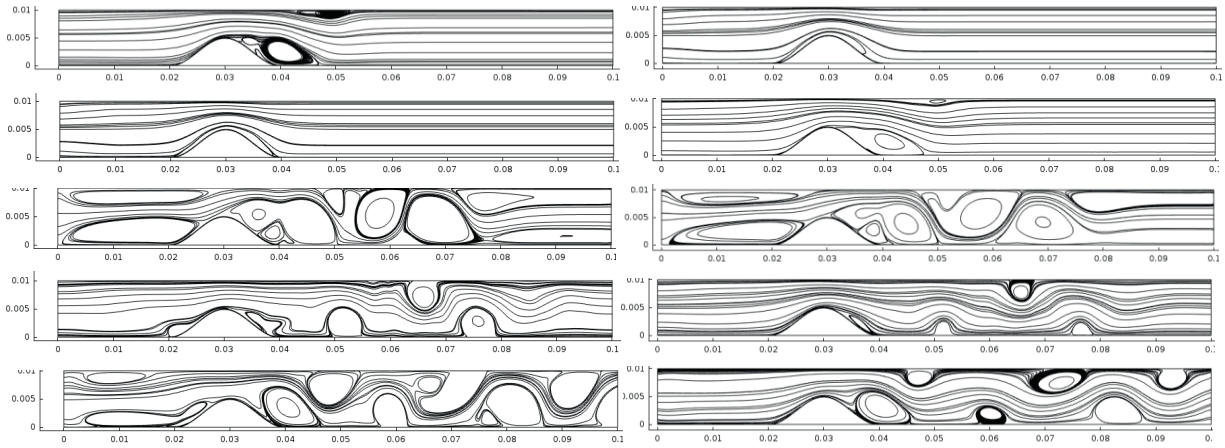

Fig. 3. Streamline for S_0= 50% and t_f = 0.1mm for (a) Newtonian; (b) Non-Newtonian

3. Results and Discussions

To describe the fluid flow characteristics, streamline is a very important parameter. In the present study, the streamline flow pattern is disturbed due to the presence of stenosis. Significant flow variations on the pre- and post stenotic region are observed due to stenosis, time dependency of flow and blood characteristics as Newtonian and non-Newtonian. Figures 3(a) and (b) show the results for stenosis severity, S_o = 50% and fibrous cap thickness, t_f = 0.1mm for Newtonian and non-Newtonian models, respectively. At t/T = 0.125, no vortex ring is observed but some vortex rings are initiated and with time advancement. The number of vortex rings are increased for both Newtonian and non-Newtonian model. When t/T = 0.25, with maximum velocity and zero acceleration, some small vortex rings are appeared only in post stenotic region along with upper and lower boundaries for both case. After that at time t/T=0.5, there are many vortex rings in post and pre stenotic region however, at point t/T = 0.75, the number of vortex rings is decreased due to less acceleration. On the other hand, at point t/T = 1, new cycle begins to start and

thus massive acceleration is introduced and thereby creates eight vortex rings with lengths of 0.02m, 0.01m, 0.011m, 0.009m, 0.008m, 0.012m, 0.01m and 0.007m, respectively in Newtonian model as shown in Fig. 3(a). On the other case, for same time instant, six vortex rings are formed with lengths of 0.01m, 0.004m, 0.0035m, 0.008m, 0.0085m and 0.005m, respectively in non-Newtonian model as shown in Fig. 3(b). As it is observed, the number of vortex ring formation is always higher in Newtonian model than in non-Newtonian model throughout the whole time period. The computations show that the Newtonian model significantly overestimates the recirculation hence overestimating the risk of clot formation in the dead zones of the blood flow.

The displacement field has been shown with color contour in Figs. 4(a) and (b) for two difference models for t_f = 0.1mm. In Fig. 4(a), the red portion shows a displacement value ranging from around 0.11 mm to 0.16 mm while in case of Fig. 4(b) this range is between 0.13 mm and 0.18 mm. Figure 5 shows the same deformation with higher

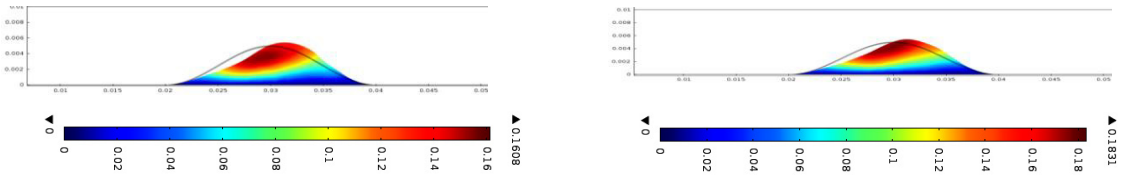

Fig. 4. Deformation of the stenosis in mm for t_f = 0.1mm a) Newtonian b) non-Newtonian

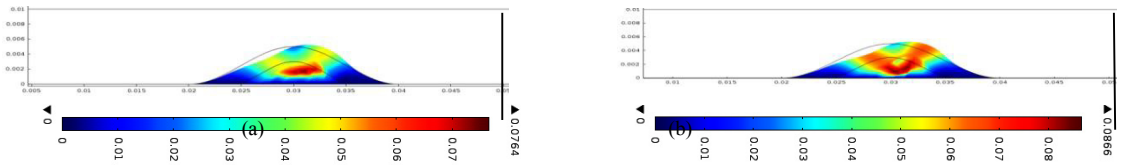

Fig. 5. Deformation of the stenosis in mm for t_f = 2.0 mm; a) Newtonian b) non-Newtonian

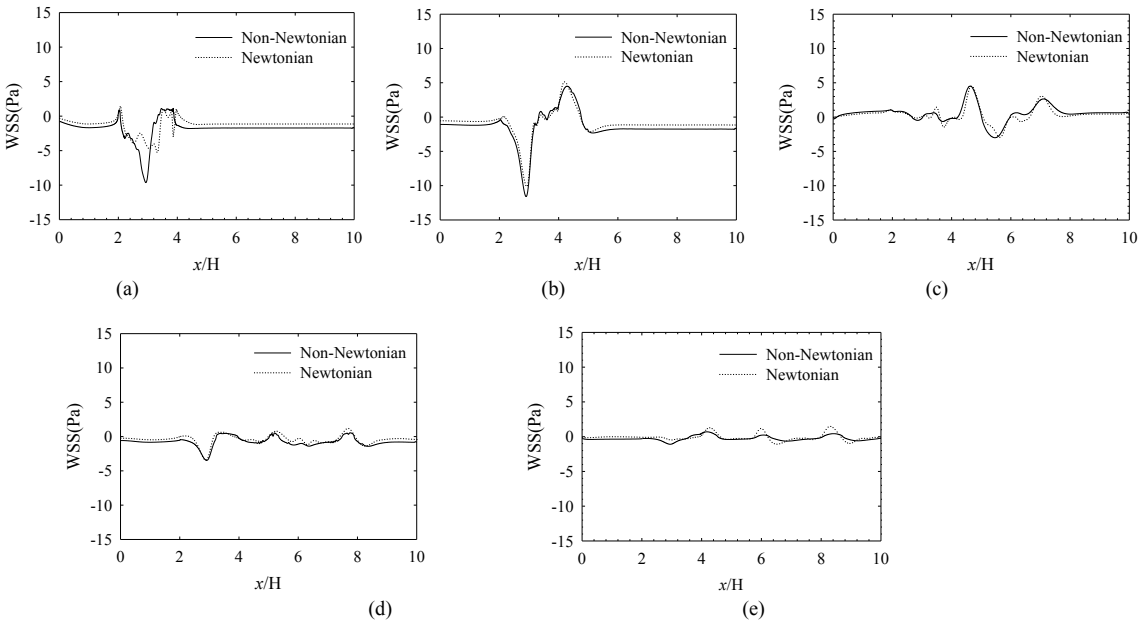

Fig. 6. Distribution of wall shear stress (WSS) along the lower boundary for t_f= 0.1mm; (a) t/T = 0.125, (b) t/T = 0.25, (c) t/T = 0.5, (d) t/T = 0.75, (e) t/T = 1.0

fibrous cap thickness of 2.0 mm. As is presented, it is clear that more deformation is computed in the non-Newtonian model compared to Newtonian model for the case of 0.1mm fibrous cap thickness.

Fig. 6 shows the distribution of wall shear stress for 0.1mm fibrous cap thickness along the lower boundary at different time instants. At t/T = 0.125, it is observed that, non-Newtonian flow shows a higher peak of value nearly -9pa than that of Newtonian one. The fluctuations are observed at x/H = 2.0 to 4.0. With the increase of time the value goes to -12pa at t/T=0.25. It is the highest value comparing to others as at this instant the velocity is maximum. At the middle of the cycle, the value starts from 4pa at x/H = 5, then decreases to nearly -2.5pa and at last increases to 2.0 pa. Finally, at the very end of the periodic pulsatile flow, the fluctuations are quite absent.

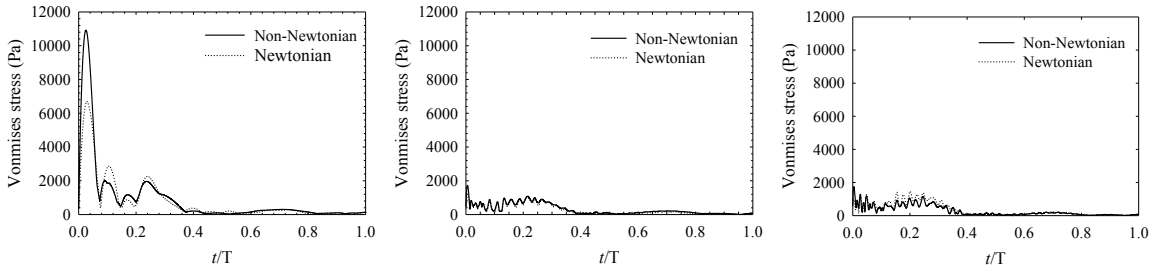

Fig. 7. Distribution of vonmises stress for (a) t_f= 0.1 mm, (b) t_f= 0.5 mm, (c) t_f= 2.0 mm

Variations of vonmises stress for 50% stenosis with various fibrous cap thicknesses are shown in Fig.7. It implies that with the increase of cap thickness, vonmises stress decreases and it shows a highest peak (10500pa) in case of 0.1mm fibrous cap thickness for Non-newtonian model The highest peak is attained at nearly t/T= 0.005, results highest distortion of the plaque that causes the peak vonmises stress. From Fig.7, it is observed that reducing cap thickness actually enhances the risk of rupture for a particular degree of stenosis (for present case it is 50%).

4. Conclusions

The aim of this paper is to study on the fluid-structure interactions between the blood flow and the plaque in a 2D geometry of a modelled arterial stenosis. The prime objective of the present research is to incorporate the non-Newtonian behaviour of the blood and to make the comparison with that of Newtonian model. It is observed that Carreau non-Newtonian model shows a higher peak than the Newtonian model for most of the critical parameters. Thus, it can be suggested that blood should be modelled as a non-Newtonian fluid in future research of biofluid dynamics.

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