International Journal of
Science and Engineering Investigations vol. 4, issue 38, March 2015
ISSN: 2251-8843

Effects of the Non-Newtonian Viscosity of Blood on Flow Field in a Constricted Artery with a Porous Plaque

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Abstract-Nowadays many people lose their lives due to cardiovascular diseases. Inappropriate food habits and lack of exercise expedite deposit process of fatty substances on inner surface of blood arteries. This abnormal lump disturbs uniform blood flow and reduces oxygen delivery to active organs. This work presents a numerical simulation of Non-Newtonian blood flow in a stenosis vessel. The vessel is considered as two dimensional channel and plaque area is modelled as a homogenous porous medium. To simulate blood flow reaction around stenosis region, we use C++ code and solve coupled Cauchy, Darcy, governing continuity and energy equations. The analyses results show that viscosity power (n) plays an important role in flow separation and the size of the eddy at the downstream edge of the plaque. It is also observed that with increasing (n) value, temperature discontinuity and likelihood of vessel rupture declined.

Keywords- *Stenotic Arteries, Blood Flow, Power Law Fluid, Porosity, Computational Fluid Dynamic.*

I. INTRODUCTION

Cardiovascular diseases are the main reason of death worldwide. The number of people who die due to heart attacks and cardiac strokes is quickly increasing and the average age of this disease is close to 38 years. The problem starts with sticking redundant cholesterol macromolecules to inner walls of artery. The situation becomes critical when the lump growths and increases the probability of vessel rupture or infract. Recent research in this are shows that in plaques region there is a microphage region which releases a large amount of heat. This phenomena makes higher the temperature along stenosis region and increase the probability of vessel rupture [1].

In literature there are several investigation of blood flow simulation in stenotic arteries [2-5]. In these studies, normal blood flow in a two dimensional constricted artery is analysed. In paper [6] a mathematical model for simulating heat and mass transfer of blood flow is presented. However, in all of these researches stenosis vessel walls and in fact atherosclerotic plaque region are considered as rigid boundary. And according to recent investigations modelling complex structure of atherosclerotic plaque has significant effect on the correct simulation of the flow field in stenotic arteries. Paper [7] assumed whole blood flow as a porous medium and examined the impact of pulsatile body acceleration. There is only a few papers which simulated an atherosclerotic plaque and free blood flow as two different domains. In 2014, H. Alimohamadi et al. considered both arterial walls and plaque region with two different porosity [8]. Their results were interesting and more close to realistic.

Calculating temperature distribution theoretically within the stenotic arterial walls is the focus of recent researches. O. Ley et al. in 2007 and 2008 used COMSOL software for simulation and studied temperature inhomogeneity in three different straight, bending and bifurcation constricted vessel [9, 10]. The effect of plaque porosity magnitude on the temperature fatigue occurrence and vessel rupture probability is brought up in [11]. Another paper demonstrated transient convective cooling mechanism of flowing blood [12]. Natural blood is a dilution of red blood cells in a plasma environment. Present of these particles gives blood flow non-Newtonian viscosity. To elucidate the significant effect of non-Newtonian modelling of the blood viscosity, H. Alimohamadi et al. [13] investigated the Power Law blood flow inside an abdominal aortic aneurysm vessel. They mainly examined the usage of external magnetic field in targeting drug delivery.

In this paper we want to extend the works of H. Alimohamadi et al. on non-Newtonian flow. Our main objective is to show the role of viscosity exponent (n) on the atherosclerotic plaque temperature distribution and likelihood of crack open in distal section. To obtain this target we solved governing equations in both free and porous region numerically. The rest of paper is organized as follow: In session 2 we explain the geometry of problem, governing equations and boundary condition, then in session 3 the numerical procedure is described. Finally, session 4 covered simulation results and discussion.

II. MATHEMATICAL MODELLING

In this section the arterial model is explained. The geometry is a straight two dimensional artery with a porous atherosclerotic plaque on the lower wall. The flow inside the vessel is assumed laminar, steady state, incompressible and viscous. The dimensions of vessel and plaque are completely match with human artery size and extracted from [14].

In this section we show the problem in three parts (i) governing equations, (ii) boundary conditions and (iii) dimensionalized and dimensionless parameters.

The Continuity equation is:

$$\int_S V.dS = 0$$

The momentum and energy equations are:

$$\int_S \rho u\vec{V}.d\vec{S} = -\int_S pi_x.d\vec{S} + \int_S (\tau_{xx}i_x + \tau_{yx}i_y).d\vec{S}$$

$$\int_S \rho v\vec{V}.d\vec{S} = -\int_S pi_y.dS + \int_S (\tau_{xy}i_x + \tau_{yy}i_y).d\vec{S}$$

$$\int_S \rho c_p T\vec{V}.d\vec{S} = \int_S (u\tau_{xy}i_x + v\tau_{yx}i_y).d\vec{S} + \int_S k(gradT).d\vec{S}$$

Where $\vec{V} = (u, v)$ is two dimensional velocity, $d\vec{S} = n.dS$ (n is unit vector normal to the surface), p is the pressure, T is the temperature, ρ and k are the blood density and conductivity respectively. In the case of non-Newtonian blood viscosity, Cho and Kensey [15] suggested Power Law model for shear-stress tensor, which is expressed as:

$$\bar{\tau} = m\left|\gamma\right|^{n-1}\bar{\gamma}$$

Where $\bar{\gamma}$ is the shear-rate tensor and m=0.0345 and n=0.6 are specific constant parameters in the case of blood.

For porous atherosclerotic plaque medium, there are lots of different models with different complexity. Here we used Darcy model to simulate the velocity in the permeable boundary.

$$grad(p) = -\frac{Da}{m\left|\bar{\gamma}\right|^{n-1}}\vec{V}$$

Da is the Darcy number which depends on the rigidity of the porous region.

For energy equation, it is assumed that blood with constant temperature Tin=37.5 ^{0}C [10] (human body temperature at normal condition) enters into the artery and both vessel walls are also at constant temperature Tw=Tin. Because of fully developed assumption at the end of domain the temperature, velocity and pressure gradient in x direction set zero

$$\frac{\partial T}{\partial x} = \frac{\partial u}{\partial x} = \frac{\partial v}{\partial x} = \frac{\partial p}{\partial x} = 0$$

The abdominal artery is far enough from heart pumping entrance, so at inlet section, fully developed velocity profile is assumed ($u = 4y - 4y^2, v = 0$). Moreover, no penetration and slip conditions are applied to the continuity and momentum equations (u=v=0 on the arterial walls).

On the plaque surface, the velocity field of porous medium must be equal to the velocity field of free blood flow ($\vec{V}_{Porous} = \vec{V}_{free-flow}$).

To have dimensionless equations we normalize the length with vessel diameter (d), velocity with maximum inlet velocity (U) and temperature with arterial temperature (Tw):

$$x^* = \frac{x}{d},\ y^* = \frac{y}{d},\ u^* = \frac{u}{U},\ p^* = \frac{p}{\rho U^2},\ T^* = \frac{T}{T_w}$$

Where the asterisk superscript shows non-dimensional variables and Reynolds number ($\mathrm{Re} = \frac{\rho U d}{m}$), Eckert number ($Ec = \frac{U^2}{c_p T_w}$) and Prandtl number ($\mathrm{Pr} = \frac{c_p m}{k}$) are dimensionless parameters.

III. NUMERICAL METHODOLOGY

The computational artery domain was meshed by stagger grid. A finite volume C++ code was used for blood flow simulation. In first step, the velocity- pressure coupling equations 1-4 were solved by pseudo-transient SIMPLE algorithm and then Alternating-Direction-Implicit (ADI) and Thomas methodology were applied for calculating temperature contour.

To obtain numerical results, the thermo-physical parameters must be set initially. For blood according O. Ley and T. Kim[9] kg/m^3, cp=4390 J/kg.^{0}C, k=0.549 W/m.^{0}C and for porous atherosclerotic plaque cp=4080 J/kg.^{0}C and k=0.484 W/m.^{0}C based on experimental results of them. There are numerous studies about the effect of Reynolds and Darcy numbers in stenosis arteries. In current work, based on [9, 16] simulation we assumed Re=300 and Da=1000 respectively.

IV. RESULTS

In this section, the effect of viscosity exponent (n) on the mechanism of vortex formation at the rear side of the construction and the heterogeneous temperature distribution along the atherosclerotic plaque surface.

The streamlines for four different n values are shown in figure 1. On the upstream side of all cases, there is no separation and the flow goes forward smoothly inside the artery. For normal condition (n=0.6), one large eddy is formed on the lee of stenosis. With increasing power value, the blood flow becomes more viscous, resists against separation occurrence and as it seen in this figure the size of eddy has reduced remarkably. As far as in the flow field of the Newtonian model (n=1), the downstream eddy has vanished and streamlines have only deviated. For case D (n > 1), the blood viscosity changes from shear-thinning to the shear-thickening fluid and one small vortex is created inside the porous plaque. The eddy formation on the downstream edge of the constriction exposes this section to the rupture and larger vortex in lower exponent increases this possibility.

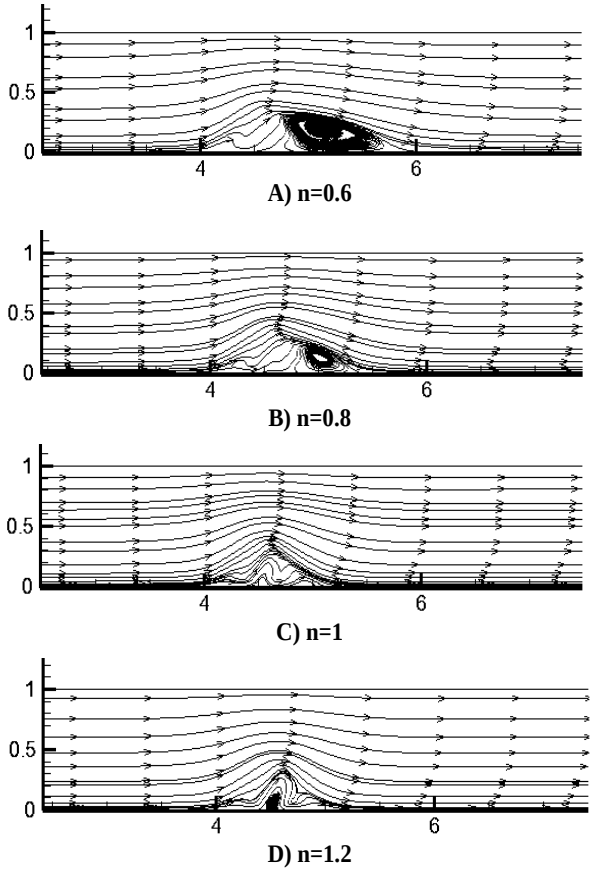

Figure 1. Velocity streamlines for different viscosity exponent

In Figure 2, the non-dimensional pressure contours for different exponent value are demonstrated. The maximum and minimum pressures are mentioned above each figure and the range between these values are divided into 30 levels. As seen, all cases have similar behavior: first, smooth pressure distribution and uniform decline in its value throughout upstream section. Second, in stenosis area and all over the rear section behind the atherosclerotic plaque, because of flow circulation, the pressure declines sharply, becomes negative and reaches its minimum value. Finally after the rear section, the pressure increases suddenly and drops gradually till it attains the fully developed condition at the outlet. It can be seen in this figure with doubling exponent from n=0.6 to n=1.2 the absolute value of maximum and minimum pressures in upstream and stenosis regions becomes 2.17 and 11.42 times higher respectively.

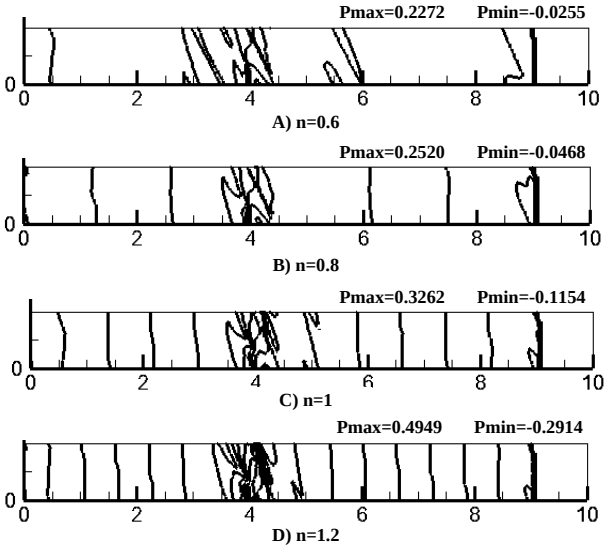

Figure 2. Pressure distribution for different viscosity exponent

The temperature difference between the plaque interface and arterial walls is indicated in Figure3. It is observed that exponent number played an important role in the maximum temperature increment and the temperature distribution profile. In cases n<1 (n=0.6 and n=0.8) the maximum temperature is registered at the back of the plaque; whereas, for the Newtonian (n=1) and shear-thickening (n=1.2) cases inside the plaque experienced the highest temperature. The difference in the location of maximum temperature can be explained by the flow separation phenomena. For low exponent, n=0.6 and n=0.8, eddy formation at the back of the plaque increases the maximum blood flow temperature 0.72% and 0.61% respect to the arterial walls' temperature. For Newtonian case, there is no eddy and temperature goes up slightly (less than 0.1%) inside the plaque. As can be seen in this figure, in n>1 assumption, vortex reformation inside the plaque increases the maximum temperature but the eddy is small and has trivial impact.

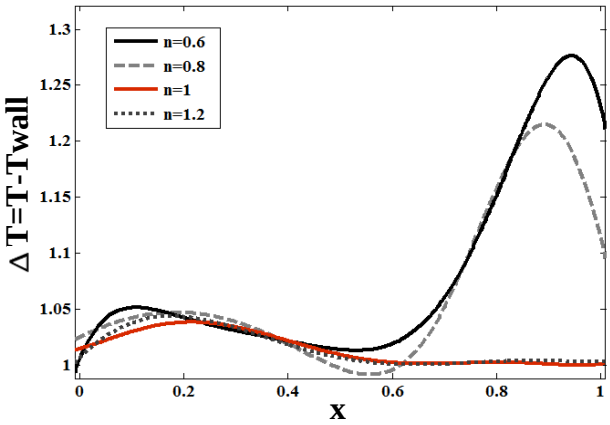

Figure 3. Temperature difference distribution with plaque surface for different viscosity exponent

The maximum lateral velocity, temperature and viscosity for different power law exponent are shown in Figure 4 and table 1. With increasing the exponent number (n) and consequently the viscosity of blood flow, the fluid elements near the arterial walls move slowly and stick to the surface. Hence, due to the law of conservation of mass, the flow at the center of artery accelerates and pushes forward with higher speed. According to table 1 with increasing exponent number from n=0.6 to n=0.8 and then n=1 the maximum viscosity becomes 1.96X and 2.67X lager. However, in higher n value (n>1) the maximum viscosity jumps up and increases in order of magnitude.

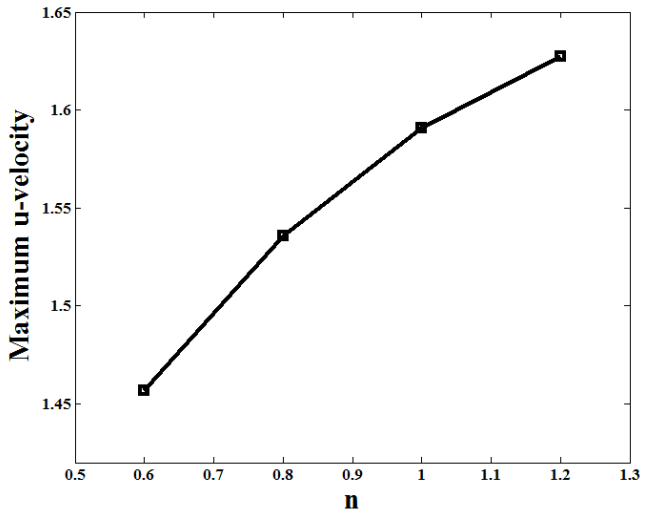

Figure 4. Maximum velocity for different viscosity exponent

TABLE 1. MAXIMUM TEMPERATURE, MAXIMUM AND MINIMUM VISCOSITY FOR DIFFERENT "N" VALUE

Viscosity power	Tmax	η_{max}	η_{min}
n=0.6	38.777	0.0127	0.007
n=0.8	38.715	0.025	0.008
n=1	38.542	0.034	0.034
n=1.2	38.601	0.42	0.05

V. CONCLUSION

In this work, blood flow in a two dimensional constricted channel was studied. Power law model was used for simulating non-Newtonian viscosity of the flow. Four different value of exponent number "n" in viscosity equation were considered. The results show flow separation, the size of the eddy at the downstream edge of the plaque and thermal stress are all depend on the viscosity exponent. It is found that Newtonian model is the safest case with lowest possibility of rupture.

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