

Numerical Simulation Of Porosity Effect On Blood Flow Pattern And Atherosclerotic Plaques Temperature

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ABSTRACT: The present work is a numerical simulation of the blood flow around the atherosclerotic plaques in a two dimensional straight stenosis vessel. With assuming the plaque as a homogenous porous medium, the governing continuity, Navier-Stokes, Brinkman- Forchheimer and energy equations are solved simultaneously. This analyze characterizes the effect of plaque porosity on the temperature heterogeneity and likelihood of vessel rupture. It was shown by diseases development and decreasing porosity from 0.8 to 0.4, the maximum plaque surface temperature increases 90% and also 2.35 times higher shear force is exerted to the distal region.

Keywords : Blood Flow, Atherosclerotic Plaques, Porous Medium, Stenosis Artery, Metabolic Heat

1 INTRODUCTION

Nowadays, heart attack takes the lives of many people all around the world. This disease mostly occurs by accumulation of fatty deposits and macromolecules, which are called atherosclerotic plaques, on inner surface of arterial walls. Disturbing normal blood delivery mechanism is the first negative effect of this common cardiovascular illness. However, it is shown recently, by macrophages cells penetration into the plaque structure, metabolic activation of this region goes up and noticeable amount of heat is released by the inflammatory layer [1]. The appearance of these hot spots along the arterial walls makes the stenosis section vulnerable and likelihood for rupture [2]. Numerical study of blood flow patterns and temperature distribution over the arteries is the focus of relatively new researches [3-8]. Moreover, a little attention have been adverted to macrophage layer heat generation and thermal fatigue phenomena in atherosclerotic plaques [9-11] although, in these papers the porosity of plaque is ignored. In the present work, our main objective is to investigate the effects of plaque porosity factor on the blood flow pattern and heterogeneous temperature distribution along the arterial walls. To that end, the organization of this work is as follows: in section 2, the detail of vessel geometry and macrophage layer are

presented. The governing equations and boundary conditions are described in next. Numerical results are brought up in section 4 with addressing the effects of plaque porosity on arterial wall thermal stress and vessel rupture probability. The work is finalized by highlighting the major results.

2 PROBLEM DESCRIPTION

In this paper, a laminar, steady state, incompressible and Newtonian blood flows inside a two dimensional atherosclerosis straight artery. The geometry of problem is shown in Figure 45 as we assumed the macrophage layer is focused at the center of plaque. The vessel and plaque dimension are based on abdominal human aorta artery as the $L_2=6.2$ mm is the plaque length, $L_1=3.35$ mm is the macrophage length, $H=2.48$ mm is the plaque height, $h_1=1.24$ mm is the macrophage thickness and vessel diameter (d) and length (L) are 6.2 and 62 mm respectively [12].

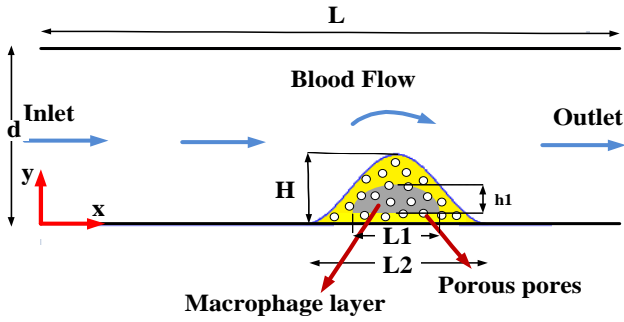


Figure 1. Geometry of problem

3 GOVERNING EQUATIONS

3-1 Blood Flow and Energy Equations

The governing equations of continuity, Navier-stokes and energy equations are as follows:

$$\text{div}(\vec{V}) = 0 \quad (1)$$

$$\rho(\vec{V} \cdot \text{grad})\vec{V} = -\text{grad}(P) + \mu(\text{div}(\text{grad}(\vec{V}))) \quad (2)$$

$$\rho C_p ((\vec{V} \cdot \text{grad})T) = \text{div}(k \cdot \text{grad}(T)) + \Phi \quad (3)$$

Where \vec{V} is the two dimensional velocity vector, ρ is the density, P is isotropic pressure, μ is the blood viscosity, T is temperature, k is the thermal conductivity, C_p the specific heat transfer of the blood and Φ is viscous heat dissipation which is given by:

$$\Phi = \left(\frac{\partial u}{\partial x}\right)^2 + \left(\frac{\partial v}{\partial y}\right)^2 + 2\left(\frac{\partial u}{\partial y} + \frac{\partial v}{\partial x}\right)^2 \quad (4)$$

3-2 Governing Equations in Atherosclerotic Porous Medium

There are numerous mathematical models for simulating blood flow through the porous plaque medium. In this work, we used from Brinkman-Forchheimer model where the inertial forces are neglected and can be written as:

$$\text{grad}(P) = \frac{\mu}{\phi} \text{div}(\text{grad}(\vec{V})) - \frac{\mu}{\kappa} \vec{V} + C_F \kappa^{-\frac{1}{2}} \rho |\vec{V}| \vec{V} \quad (5)$$

Where ϕ and κ are porosity and permeability of plaque respectively. C_F is Forchheimer constant parameter which is calculated by [13]:

$$C_F = \frac{1.75}{\sqrt{150}} \phi^{-\frac{3}{2}} \quad (6)$$

As was mentioned before, solid part of macrophage layer inside the atherosclerotic plaque produces noticeable amount of metabolic heat. This heat is constrained inside the plaque and varies between $0.05-0.2 \text{ W/mm}^3$. Assuming solid and fluid parts of porous plaque in same temperature, the governing energy equation of this region is defined as:

$$\rho C_p (V \cdot \nabla T) = \nabla(k_m \nabla T) + (1-\phi)\dot{q}_s \quad (7)$$

In which k_m is combination of solid and fluid thermal conductivity:

$$k_m = \phi k_f + (1-\phi)k_s \quad (8)$$

Finally, the applied boundary conditions for this problems include:

- No penetration and slip velocity on the upper and lower arterial walls $u = v = 0$

- Fully developed inlet velocity $u = 4y - 4y^2, v = 0$

- Constant arterial walls and blood entrance temperature

$$T = T_w$$

- Fully developed condition for all parameters at the outlet

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

- Equal velocity and temperature on plaque-blood interface

$$(\vec{V}_p = \vec{V}_b, T_p = T_b)$$

3-3 Transformation of Equations

In order to speed up the numerical solution and work with dimensionless parameters, we substitute:

$$x^* = \frac{x}{d}, y^* = \frac{y}{d}, u^* = \frac{u}{U}, p^* = \frac{P}{\rho U^2}, T^* = \frac{T}{T_w} \quad (9)$$

Where d is vessel diameter, U is maximum inlet velocity and T_w is arterial temperature that in this paper is chosen 37.5°C [11]. With Eq.(9), the dimensionless form of governing Eq. (1-3), (5) and (8) are obtained as: Continuity

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0 \quad (10)$$

a) For free blood region

X momentum

$$u \frac{\partial v}{\partial x} + v \frac{\partial u}{\partial y} = -\frac{\partial P}{\partial x} + \frac{1}{\text{Re}} \times \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right) \quad (11)$$

Y momentum

$$u \frac{\partial v}{\partial x} + v \frac{\partial v}{\partial y} = -\frac{\partial P}{\partial y} + \frac{1}{\text{Re}} \times \left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2} \right) \quad (12)$$

Energy equation

$$u \frac{\partial T}{\partial x} + v \frac{\partial T}{\partial y} = \frac{1}{\text{Pr} \times \text{Re}} \left(\frac{\partial^2 T}{\partial x^2} + \frac{\partial^2 T}{\partial y^2} \right) + \frac{2Ec}{\text{Re}} \times \Phi \quad (13)$$

b) For porous plaque medium

X momentum

$$\frac{\partial p}{\partial x} = \frac{1}{\text{Re}} \times \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right) - \frac{Da}{\text{Re} \times \phi} u - F_0 \sqrt{(u^2 + v^2)} u \quad (14)$$

Y momentum

$$\frac{\partial p}{\partial y} = \frac{1}{\text{Re}} \times \left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2} \right) - \frac{Da}{\text{Re} \times \phi} v - F_0 \sqrt{(u^2 + v^2)} v \quad (15)$$

Energy equation

$$u \frac{\partial T}{\partial x} + v \frac{\partial T}{\partial y} = \frac{1}{\text{Pr} \times \text{Re}} \left(\frac{\partial^2 T}{\partial x^2} + \frac{\partial^2 T}{\partial y^2} \right) + \frac{2Ec}{\text{Re}} \times \Phi + (1 - \phi) \dot{q}_s \quad (16)$$

It is worth mentioning, the asterisk (*) symbol above dimensionless parameters have dropped for convenience. In above equations, the non-dimensional parameters including Reynolds (Re), Eckert (Ec), Prandtl (Pr), Darcy (Da) and Forchheimer (Fo) numbers are defined respectively by:

$$\text{Re} = \frac{\rho U d}{\mu} \quad (17)$$

$$\text{Ec} = \frac{U^2}{C_p T_w} \quad (18)$$

$$\text{Pr} = \frac{C_p \mu}{k} \quad (19)$$

$$\text{Da} = \frac{d^2}{\kappa} \quad (20)$$

$$F_0 = \frac{C_F}{\sqrt{\kappa}} d \quad (21)$$

4 RESULTS AND CONCLUSION

For solving partial differential Eq. (10-16), a computer C++ code has been developed. Finite volume method is used for domain discretization and diffusive terms are calculated by second order upwind scheme. The pressure and velocity magnitudes are stored in stagger grid cell centers and pseudo-transient SIMPLE algorithm is applied for solving velocity-pressure coupling equations (Eq. (10-12), (14) and (15)). After obtaining velocity field, in the second stage, the temperature distribution is calculated by Alternating-Direction-Implicit (ADI) methodology. The appeared tridiagonal matrix in this method is solved by Thomas procedure. For numerical solution, $\dot{q} = 0.1 \text{ W/mm}^3$ value is used for macrophage layer heat generation [10] and thermophysical properties of both blood and plaques are considered respectively as $\rho = 1050$

kg/m^3 , $C_p = 4390 \text{ J/kg}^\circ\text{C}$, $k = 0.049 \text{ W/m}^\circ\text{C}$, $\mu = 3.2 \times 10^{-3}$

Kg/m.s , $C_p = 4080 \text{ J/kg}^\circ\text{C}$, $k = 0.484 \text{ W/m}^\circ\text{C}$ [11].

Moreover, in the present work, two Reynolds and Darcy numbers are set 300 and 100. Plaque porosity is an important factor that changes by passage of time and stenosis disease progress. The effect of this parameter on plaque surface temperature distribution is depicted in Figure 2. By decreasing porosity, plaque becomes more rigid and stands out hardly against the passage of flow. So, the strength of vortex at the posterior regions of the plaque increases, cooling mechanism of flowing blood along the plaque/lumen interface is collapsed completely and finally as shown in figure 4 plaque surface temperature goes up sharply. Such that with declining plaque porosity from 0.8 to 0.4 the maximum plaque temperature rises about 90%. This remarkable temperature heterogeneity along the arterial wall provides a suitable condition for thermal fatigue phenomena and crack open in stenosis vessels. Wall shear stress variation along the lower arterial wall with respect to porosity factor is shown

in Figure 3. The shear stress has low value and smooth trend before the interior edge of the plaque. From that point due to blood flow acceleration and increasing velocity gradient, the shear stress value increases steeply and reaches the extremum point. After that, the shear stress magnitude falls down and in the point of zero shear stress detachment of flow occurs. As shown in the figure, flow circulation and reversal flow at the downstream edge of the plaque applies negative shear force to the arterial inner surface and increases the probability of vessel rupture.

metabolic heat generation, porous lump region has noticeable higher temperature than arterial walls (about 0.9°C).

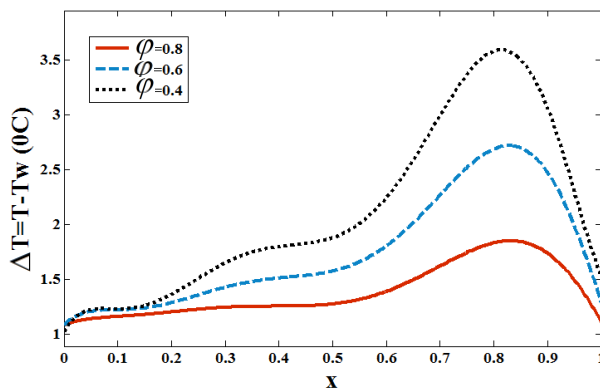


Figure 2. Temperature distribution on the plaque surface for three different porosity factor

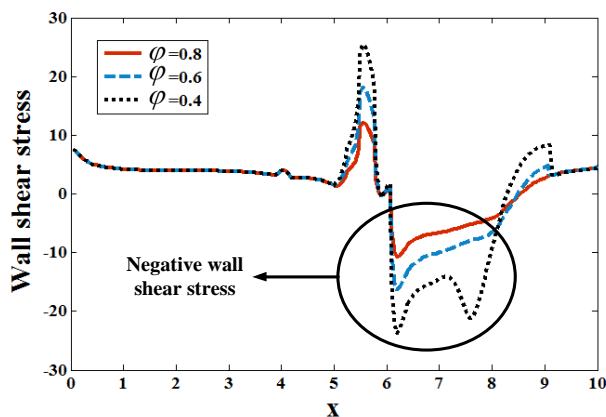


Figure 3. Wall shear stress distribution on the arterial wall for different porosity factor.

it, in the rear section of plaque ($X=7$ and $X=8$) the negative vertical velocity exerts downward force to the arterial wall and put it in likelihood condition for rupture. For same porosity factor ($\phi=0.8$), the temperature contour as well as the variation of blood temperature at different cross sectional positions is depicted in Figure 5. Due to macrophage

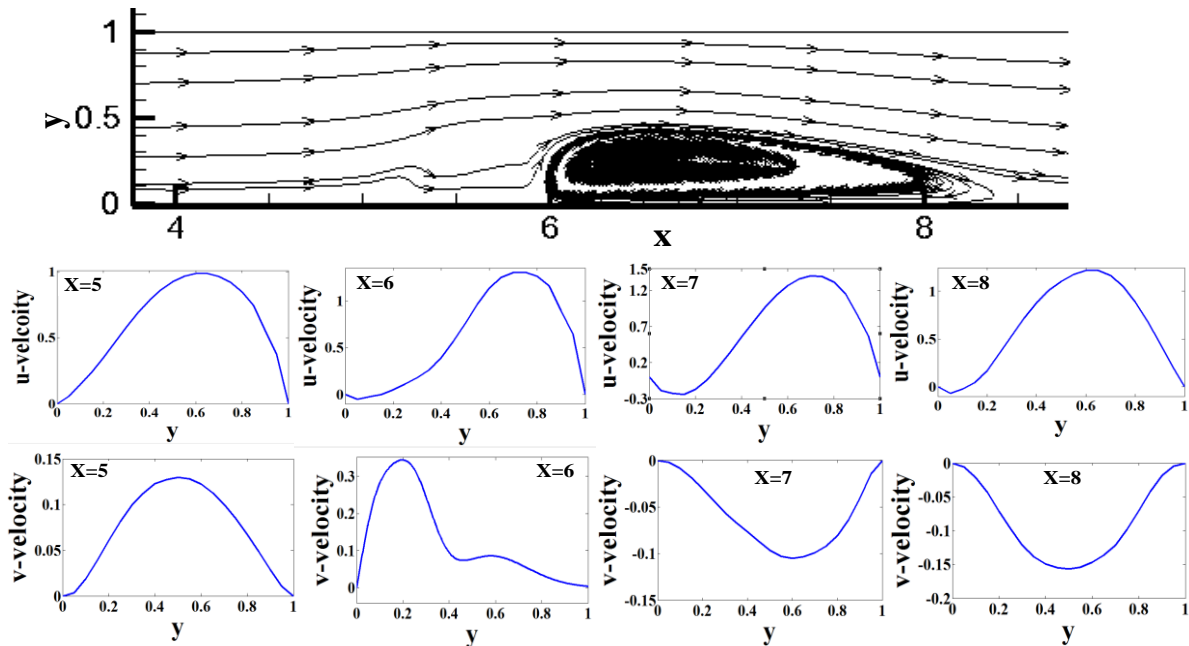


Figure 4. Streamline pattern and velocity components profiles in different positions

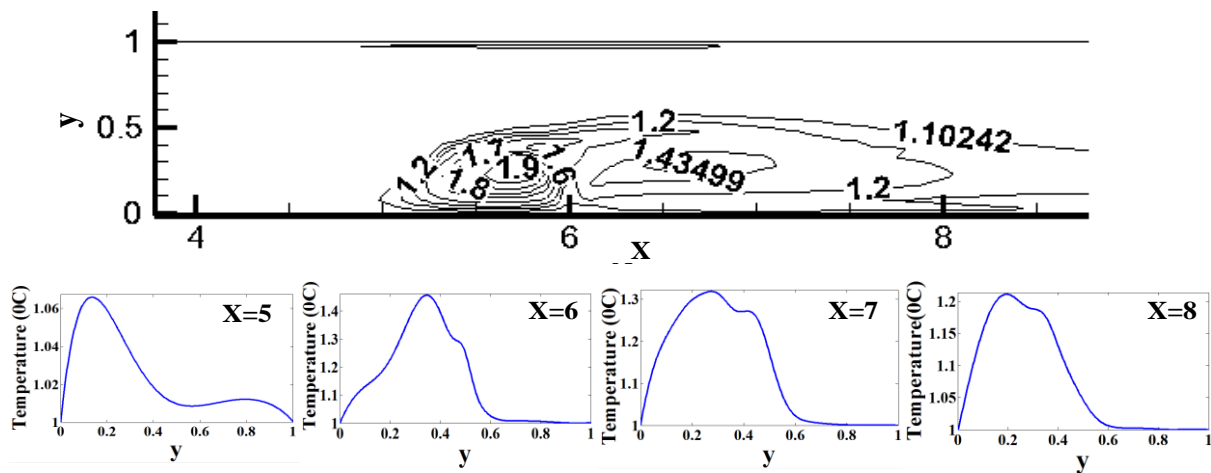


Figure 5. Temperature contours and profiles in different positions

In addition, by advent of strong vortex in the rear section, heat transfer mechanism between cooling blood flow and hot atherosclerotic plaque is blocked and consequently inhomogeneity temperature along the arterial wall is exacerbated. In Figure 6 negative shear force comparison for three different plaque porosity is demonstrated. As was expected, by decreasing plaque porosity, the vessel environment with 2.35 times higher shear force.

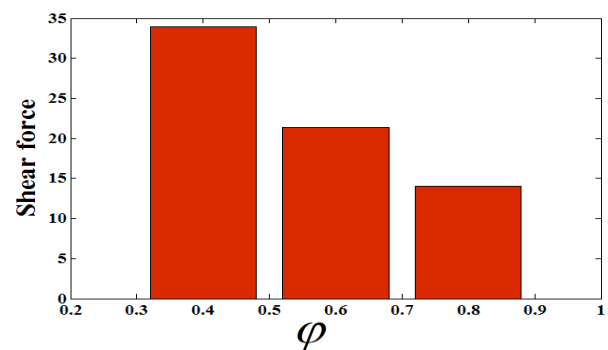


Figure 6. Shear force comparison for different porosity

5 CONCLUSION

In this paper, the role of porosity factor in atherosclerotic plaque temperature distribution and likelihood vessel rupture has been studied. Because of metabolic heat generation of macrophage layer and flow circulation at the downstream edge of the plaque, the stenosis arterial wall withstands remarkable temperature inhomogeneity and negative shear force. The results show by increasing the rigidity of plaque, the temperature difference between stenosis section and arterial walls rises and higher shear force is applied to the distal region.

REFERENCES

- [1] A. M. Malek, S. L. Alper, and S. Izumo, "Hemodynamic shear stress and its role in atherosclerosis," *Jama*, vol. 282, pp. 2035-2042, 1999.
- [2] M. Madjid, M. Naghavi, B. A. Malik, S. Litovsky, J. T. Willerson, and W. Casscells, "Thermal detection of vulnerable plaque," *The American journal of cardiology*, vol. 90, pp. L36-L39, 2002.
- [3] H. Alimohamadi and M. Imani, "Finite Element Simulation of Two-Dimensional Pulsatile Blood Flow thorough a Stenosed Artery in the Presence of External Magnetic Field," *International Journal for Computational Methods in Engineering Science and Mechanics*, pp. 00-00, 2014.
- [4] R. Ouared and B. Chopard, "Lattice Boltzmann simulations of blood flow: non-Newtonian rheology and clotting processes," *Journal of statistical physics*, vol. 121, pp. 209-221, 2005.
- [5] L. Diamantopoulos, X. Liu, I. De Scheerder, R. Krams, S. Li, J. Van Cleemput, *et al.*, "The effect of reduced blood-flow on the coronary wall temperature Are significant lesions suitable for intravascular thermography?," *European heart journal*, vol. 24, pp. 1788-1795, 2003.
- [6] H. Alimohamadi, M. Imani, and M. Shojae-Zadeh, "Computational Analysis of Pulsatile Biofluid in Locally Expanded Vessel under the Action of Magnetic Field," *Advances in Applied Science Research Journal*, vol. 5, pp. 1-8, 2013.
- [7] H. Alimohamadi, M. Imani, and M. Shojaezadeh, "Non-Newtonian Blood Flow In A Stenosed Artery With Porous Walls In The Present Of Magnetic Field Effect," 2014.
- [8] H. Alimohamadi and M. Imani, "Transient Non-Newtonian Blood Flow under Magnetic Targeting Drug Delivery in an Aneurysm Blood Vessel with Porous Walls," *International Journal for Computational Methods in Engineering Science and Mechanics*, vol. 15, 2014.
- [9] A. Ten Have, F. Gijzen, J. Wentzel, C. Slager, P. Serruys, and A. van der Steen, "A numerical study on the influence of vulnerable plaque composition on intravascular thermography measurements," *Physics in medicine and biology*, vol. 51, p. 5875, 2006.
- [10] O. Ley and T. Kim, "Determination of atherosclerotic plaque temperature in large arteries," *International Journal of Thermal Sciences*, vol. 47, pp. 147-156, 2008.
- [11] O. Ley and T. Kim, "Calculation of arterial wall temperature in atherosclerotic arteries: effect of pulsatile flow, arterial geometry, and plaque structure," *Biomedical engineering online*, vol. 6, p. 8, 2007.
- [12] D. K. Stangeby and C. R. Ethier, "Computational analysis of coupled blood-wall arterial LDL transport," *Journal of biomechanical engineering*, vol. 124, pp. 1-8, 2002.
- [13] S. Ergun and A. A. Orning, "Fluid flow through randomly packed columns and fluidized beds," *Industrial & Engineering Chemistry*, vol. 41, pp. 1179-1184, 1949.