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Computational Analysis of Blood Flow Through the Human Artery with Axi-Symmetric Blockage

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Abstract: An axi-symmetric model was developed and validated for hemodynamic pulsatile blood flow through a symmetrically stenosed artery. The blood flow was considered to be a Newtonian fluid, designed for a 2D idealized elastic arteries and is characterized as a steady, laminar, incompressible and unidirectional flow velocity at the inflow and various values of blood-pressure at the outflow, while the arterial walls as well as the surrounding muscles were modeled as a hyperelastic neo-Hookean material. The results were obtained for axial velocities, total flow rate, pressure gradient and wall shear stresses (WSS). The result showed significant strengthened WSS at the stenotis throat and weakened WSS at the distal side of stenosis neck. It is found that the increase of stenosis size (height) increased the pressure drop and WSS, whereas velocity and flow rate decreased. The wall deformation and WSS play an important role in the flow mechanics of the blood in the stenosed artery. This work may enhance to regulate the blood flow in hypertensive patients and those who have blockage in their arteries. Such kind of computational work may be helpful for the physiologists to treat their patients more accurately and more effectively.

Key words: Hemodynamics · Blood · Human Artery · Wall Shear Stress

INTRODUCTION

A very common fact, now-a-days, is the abnormal growths in the lumen of the arterial wall developed at various locations of the cardiovascular system. Stenosis is one of the most widespread arterial diseases [1]. A schematic of human normal and stenosed artery is shown in Fig. 1. The fluid dynamical factors play an important role in the development of such arterial diseases. The interest in hemodynamic studies, in recent years, has grown appreciably due to the fact that many cardiovascular diseases are closely related to the flow conditions in the blood vessels [2]. A steady flow through an axisymmetric stenosis has been investigated extensively by Smith [3] using an analytical approach indicating that the flow patterns strongly depend on the geometry of the stenosis and upstream Reynolds number. Realizing the fact that the pulsatile nature of the flow cannot be neglected, many theoretical analysis and experimental studies of the flow through stenosis have been performed [4-5]. In most of these studies, the flowing blood is assumed to be Newtonian. The assumption of the Newtonian behavior of blood is acceptable for a high shear rate flow in the case of a flow through larger arteries. It has now been well accepted that blood, being a suspension of cells, behaves like a non-Newtonian fluid at a low shear rate in smaller arteries under certain flow conditions [6-7]. Some researchers [8-9] propounded that for blood flowing through small vessels there is an erythrocyte-free plasma (Newtonian) layer adjacent to the vessel wall and a core layer of a suspension of all erythrocytes (non-Newtonian). Accepting this idea,

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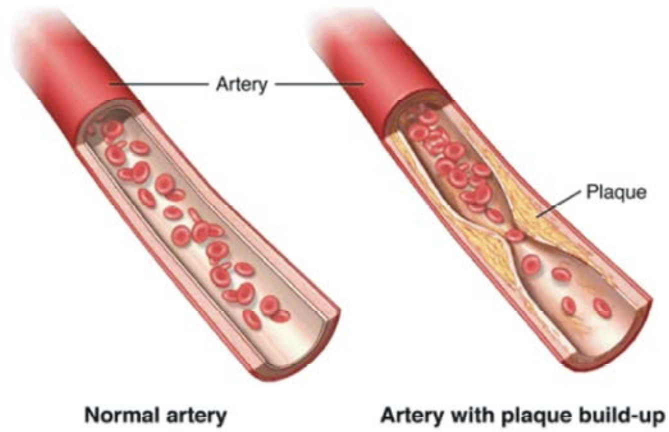

Fig. 1: Schematic of human regular and restricted artery

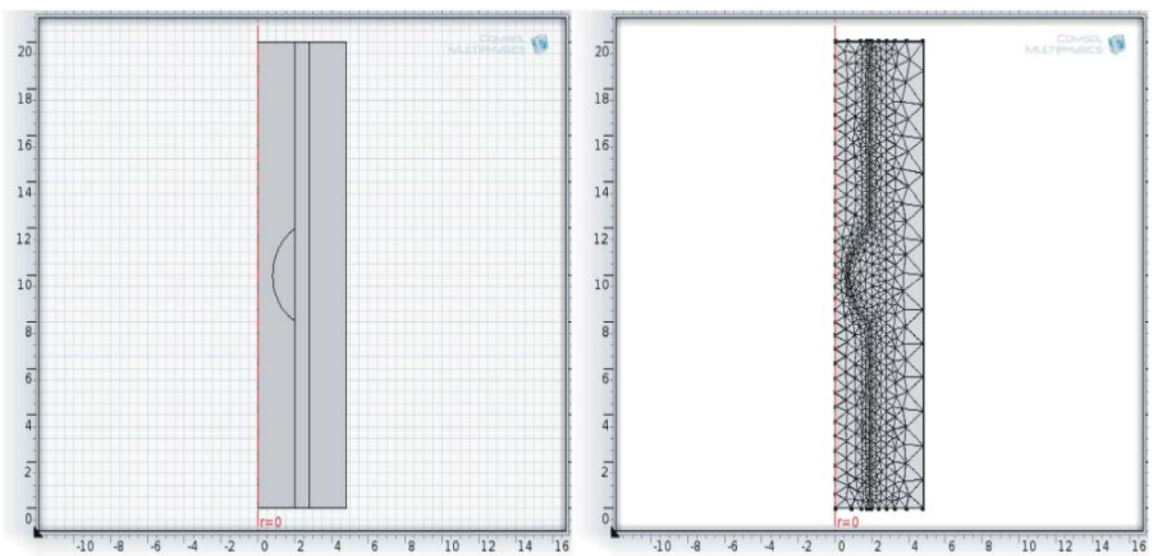

Fig. 2: Flow geometry of the stenosed artery (left) and meshing of domain (right)

Charm and Kurland [10] pointed out in their experimental findings that the Casson fluid model would be the best representative of blood and that it would be applied to the blood of humans.

In this research, an attempt has been made to provide a model to simulate the blood flow dynamics through a section of human artery with symmetric-stenosis

Formulation of the Problem: The axi-symmetric geometry of stenosed artery and its finite element meshing are shown in Fig. 2.

$$R(z) = \left\{ \begin{array}{c|c} R_0 - \frac{2\delta_s}{L_0}(z-d) & d \le z \le d + \frac{L_0}{2} \\ R_0 - \frac{\delta_s}{2}\{1 + cos\frac{2\pi}{L_0}(z - d - \frac{L_0}{2})\} & d + \frac{L_0}{2} \le z \le d + L_0 \\ R_0 & otherwise \end{array} \right\} \tag{1}$$

where $R(z)$ is the radius of the stenosed tube and R_0 is the radius of unobstructed blood vessel. L_0 is the length of stenosis, d is the position of stenosis, δ_s is the height of stenosis.

Modeling Blood Flow: The Navier-Stokes equations are the principle equations to model fluid flow. Under the conditions of an incompressible material, the gravitational external force $\rho_f g$ is assumed to be zero and the reduced Navier-Stokes equations (conservation of mass and conservation of momentum) are;

$$\frac{\partial u_i}{\partial x_i} = 0 \tag{2}$$

$$\rho u_j \frac{\partial u_j}{\partial x_j} = -\frac{\partial p}{\partial x_i} + \mu \frac{\partial^2 u_i}{\partial x_j^2} + F_j \tag{3}$$

where $u_i = (u, v, w)$ is the local velocity, $x_i = (x, y, z)$ is the length coordinate, p is the fluid pressure, μ is the dynamic viscosity and F_i is the force due to the induced magnetic field (volume force) on the blood vessel.

The blood-mimicking fluid was modeled in isothermal, incompressible and Newtonian (constant viscosity) conditions and the stress tensor is generated in the blood defined by the constitutive equations;

$$\sigma_{ij} = -p\delta_{ij} + 2\mu e_{ij} \tag{4}$$

wshere σ_{ij} is the stress tensor, $\mu = 3.5 \times 10^{-3} m^2$/sec is the fluid viscosity and δ_{ij} is the kronecker delta.

Modeling Vessel Wall and Elastic Muscle: The governing equations for the motion of an elastic solid are mathematically described by the following equation:

$$\rho_w \frac{\delta^2 \varepsilon_{ij}}{\delta t^2} = \frac{\delta \sigma_{ij}}{\delta x_j} + \rho_w F_i \quad \text{for } i = 1,2,3 \tag{5}$$

where ε_{ij} and σ_{ij} are the components of the displacements and stress tensor in a solid respectively, ρ_w is the solid density, F_i are the components of body force acting on solid and σ_{ij} can be obtained from constitutive equation of the material.

Using the relation for the Cauchy stress for an incompressible neo-Hookean material we get.

$$\sigma_{ij} = \lambda e_{\alpha\alpha}\delta_{ij} + 2G\varepsilon_{ij} \tag{6}$$

Boundary Conditions and Parameter Values: Two types of domains exist in this analysis, the solid domain, which represents the vessel wall, muscle surrounding the vessel and the stenosis part, which was considered as an elastic rigid body where this limitation was only applied to validate the computational model. The wall was assumed as an incompressible, isotropic and linearly elastic material, with high Young's Modulus of 80 MPa, a Poisson ratio of 0.45 and a density of 1000 Kg/m^3 with thick wall and no-slip wall condition. While, in the fluid domain, blood-mimicking fluid was modeled in isothermal conditions and as incompressible and Newtonian, in which the fluid density was $\rho = 1050$ Kg/m^3 and viscosity was $\mu = 3.5 \times 10^{-3}$ m^2/s. A steady flow with laminar profile with mean velocity of 42 mm/s was selected as boundary condition at the inflow boundaries. At the outlet it was considered as free pressure boundary.

Numerical Implementation: First blood flow velocity was computed from equation (3) associated with equation (2) at every time step using the appropriate boundary conditions illustrated above. The deformation and the shear stress resulted in the blood vessel and the surrounding muscles are obtained using equation (5) along with (6). Commercial finite element software package COMSOL Multiphysics 5.2 is used to simulate the model. The interaction of the flow of fluid (blood) on the solid (clotted stenosis, blood vessel and surrounding muscles) is examined using the FSI module of the above mentioned simulation software.

RESULT AND DISCUSSION

In the present model, an attempt has been made to evaluate some of the important characteristics of blood flow past an arterial stenosis with the effect of body acceleration and pulsatile pressure gradient. A computational analysis and simulation of blood flow through symmetric stenosis with various flow rates have been studied. A pulsatile flow of blood, assumed to be steady and Newtonian, is produced by using a specific pressure gradient at inlet (11208 Pa) and outlet (11148 Pa). The arterial walls and the surrounding muscles are modeled as a hyperelastic Neo-Hookean material and the no-slip conditions were used for velocity at blood vessel.

Velocity Field and Streamlines: The fluid velocity was much varied in the stenosis region and become highest at the stenosis throat (high velocity gradient). The flow velocity field and the resulting streamlines are shown in Fig. 3. The area of interest is the region after the stenosis where the flow separation occurs. There is high particle concentration in the recirculation zone.

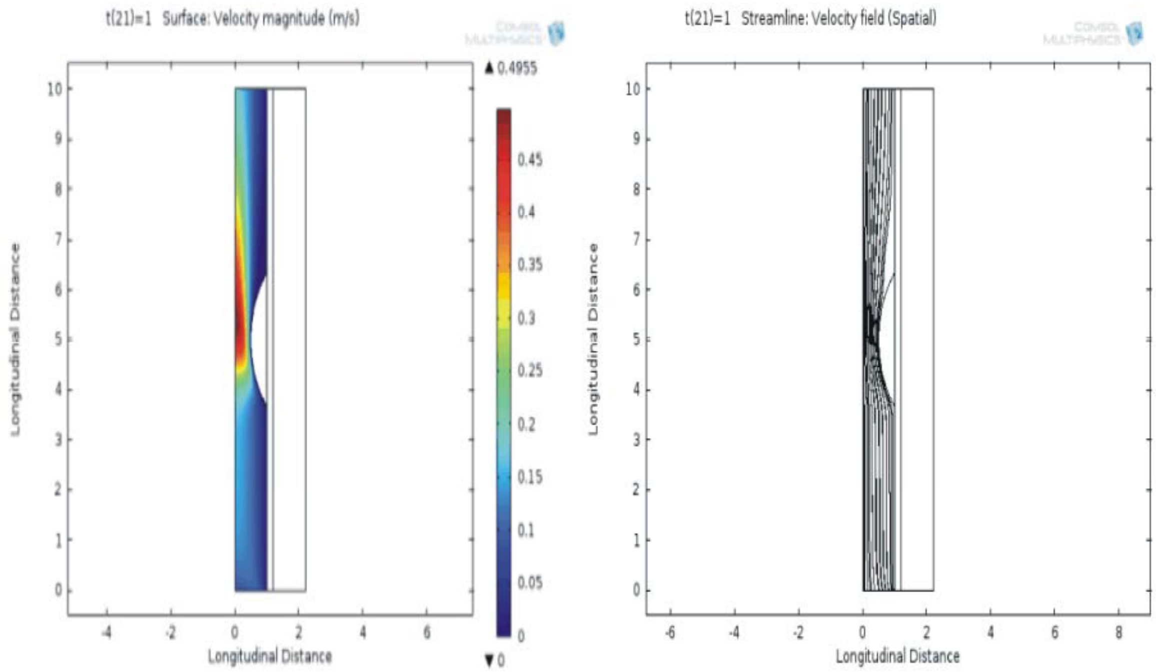

Fig. 3: Blood velocity field (in ms^{-1}) at Time = 1s (left) and the streamlines at Time = 1s (right)

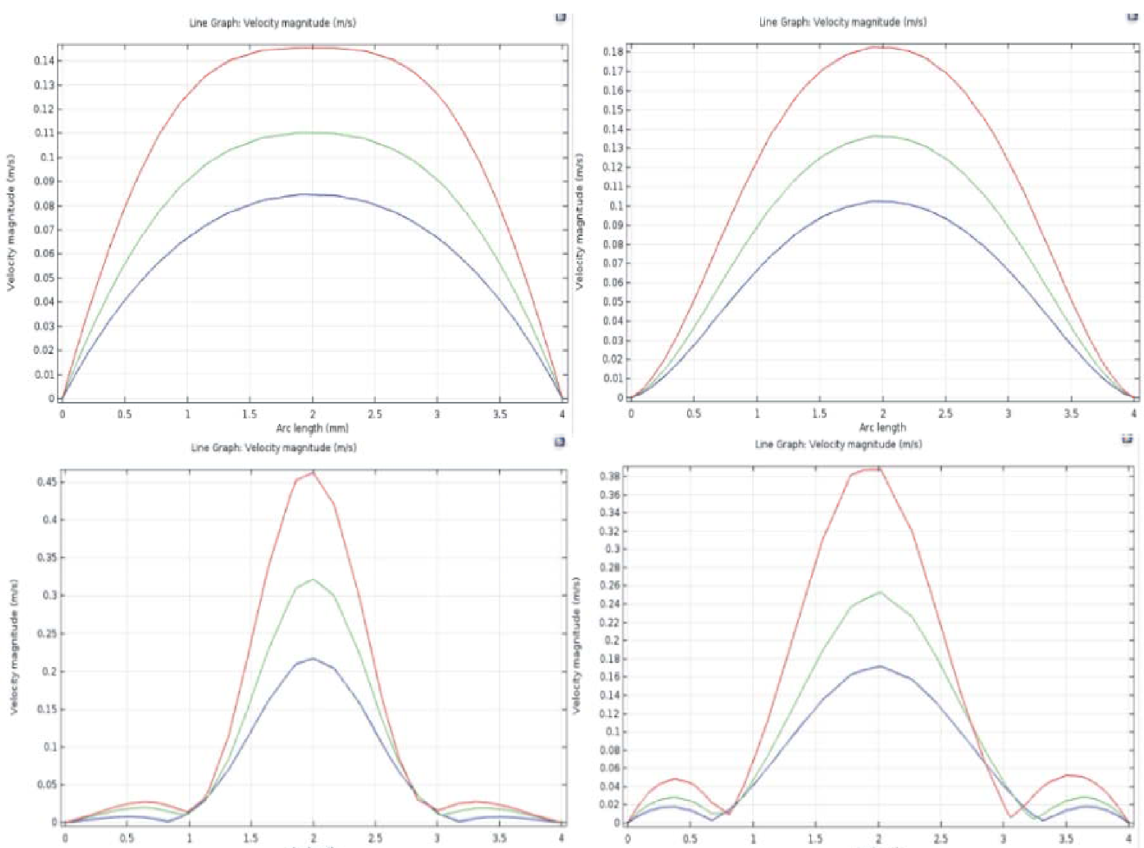

Fig. 4: Velocity plot group at artery length L = 5 *mm*, 7.5 *mm*, 12.5 *mm*, 15 *mm*

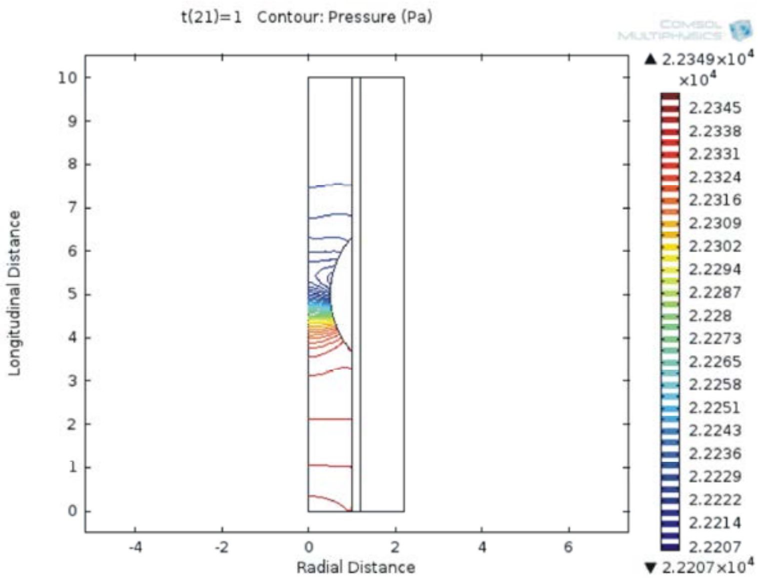

Fig. 5: Pressure contours

Velocity Plot Group: The followings are some plot group created for the velocity profile at four positions, e.g. $L = 5$ *mm*, 7.5 *mm*, 12.5 *mm* and 15 *mm*. These figure shows that there is a fluctuation of velocities at stenosis throat and the latter positions. As a result the profiles show some variations in comparison with the profile of Hagen-Poiseulle flow.

Pressure Contours: Results show that blood pressure is increased from the normal artery gradually when passes the stenosed region and pressure profile is of the parabolic form.

Pressure is much reduced in the next portion of the artery and pressure profile is also changed. The results also show the intensified WSS at the stenosis throat and significantly weakened WSS at the distal side of stenosis. Due to the largest stress values, large deformation takes place at the stenosis part than at the regular artery.

CONCLUSIONS

It can be concluded that, the peak velocity was maximum at the stenosis throat. Besides that, the increment in the severity of stenosis also demonstrate an increment of the pressure, WSS, velocity profile due to the more decreased region of occlusion in the stenotic region. Our analysis has revealed that hemodynamic stresses are pressure-dependent and vary greatly over the pressure range values varying considerably all through the stenosis. For future work, additional computational work may involve dividing the stenotic wall geometry into different regions and using a separate constitutive equation for each region. Such approach will be useful in defining realistic geometries. New more elaborated patient-specific models could lead to a complete hemodynamic analysis that hopefully will incite new research that would result in enhanced treatment of multiple pathological conditions, such as atherosclerosis (stenosis) or ICAs (intracranial aneurysms).

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