NUMERICAL ANALYSIS OF BLOOD FLOW THROUGH BLOOD VESSELS WITH ATHEROSCLEROSIS USING THE NEWTONIAN FLOW MODEL

Abstract

This study quantitatively investigates a Newtonian model for blood flow in a human blood vessel with an atherosclerotic artery. For numerical investigation, the Newtonian flow model of blood flow is used. COMSOL Multiphysics is used for the simulation of the model. The governing equation system, that is depends on incompressible Navier-Stokes equations, considers blood characteristics. Examining the blood flow pattern through an atherosclerotic artery is the aim of this investigation. To solve the governing system of equations with boundary conditions, the finite element model by COMSOL Multiphysics is used. The results have been shown concerning velocity, pressure, and streamlines. Graphical cross-sectional maps of velocity magnitude, pressure, and streamline over the atherosclerotic contraction are also shown. The blood flow simulation findings indicate that the blood flow velocity rises near the plaques. This paper will analyze how the quantity of atherosclerotic plaque controls blood flow through an atherosclerotic artery, assuming the flow is steady and the blood is treated as Newtonian Fluid Model.

***Keywords*:** Newtonian model, COMSOL Multiphysics, Velocity, Pressure, and Streamlines.

1. Introduction

The primary cause of mortality is cardiovascular diseases (CVD), which include stroke and coronary artery disease. Any illness affecting the heart or blood vessels is referred to as cardiovascular disease (CVD). There is an involvement of atherosclerosis in peripheral arterial disease, coronary artery disease, and stroke. Among other things, high blood pressure, smoking, diabetes mellitus, inactivity, obesity, high blood cholesterol, poor nutrition, excessivealcohol intake, and restless nights might be the cause of this. An estimated 13% of fatalities from CVD are thought to be related to high blood pressure, compared to 9% from smoking, 6% from diabetes, 6% from inactivity, and 5% from obesity. In all, cardiovascular disease (CVD)caused 17.9 million deaths (32.1%) in 2015, up to 12.3 million (25.8%) in 1990. [1]

Atherosclerosis is a kind of arteriosclerosis, which is defined by the formation of abnormalities in the artery walls known as lesions. The accumulation of athermanous plaques resulting from these diseases may cause the artery walls to narrow.

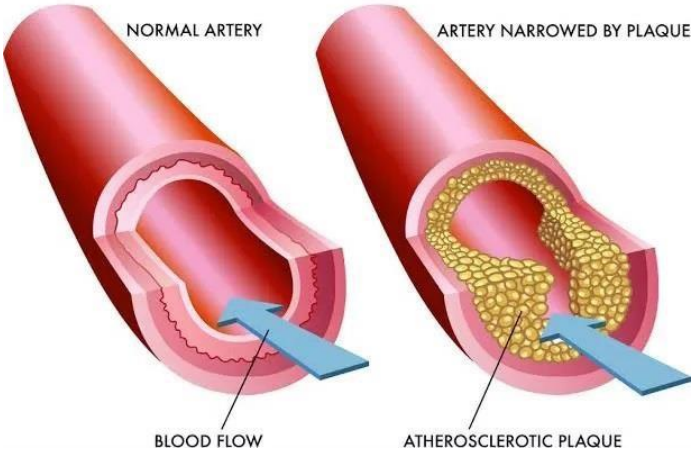

Figure 1: Blood flow in Normal and Narrowed Vessel

More specifically, atherosclerosis is a degenerative condition characterized by inflammation of the inner artery wall (intima) caused by an excess of LDL in the circulation. The tighteningof arteries restricts the flow of oxygen-rich blood to various regions of the body. The specific etiology of atherosclerosis is uncertain; however, it is thought to be complex. Therefore, a more comprehensive understanding of the processes of atherosclerosis is crucial for creating novel treatment strategies, and advancements in numerical simulations and mathematical modeling of the related phenomena play a key part in this research context.

To simplify the situation, most researchers in the first investigations on atherosclerotic arteries believed that human blood was a Newtonian fluid and that atherosclerosis was a symmetric constriction. W. Wang, D. Yang, and Y. Lu [2] investigated the steady flow via an axisymmetric sclerotic artery theoretically. Four non-Newtonian blood models Power Law, Casson, Carreau-Yasuda, and Generalized Power Law as well as the Newtonian modelhave been utilized in [3] to evaluate the relevant blood flow behavior and measure the differences between them to analyze their significance.

The effects of blood flow and stress on the vascular wall of semicircular obstructed long cylindrical arteries were investigated by Apoorva Garje and Y.G. Adhav [7]. Wang, Weiduo, Dan Yang, and Yi Lu. [8] found that the body is not significantly affected by a tiny obstruction. Thinning blood vessel walls put a great deal of strain on the walls, which raises the risk of ischemic cerebrovascular disorders, which can be deadly. Thus, obstructions ought to be identified promptly. Rothwell, Peter M., R. Gibson, and C. P. Warlow Author [10] concluded that a key mechanism of ischemic strokelikely occurs distal to a newly clinical arterial stenosis, and is related to the pathophysiology ofacute coronary syndromes, namely local thrombus generation caused by an unsustainable arterial plaque with atherosclerosis. Studies of the hydrodynamic characteristics in stenosis zones are crucial because it is thought that the development of plaque is associated with wall shear stress at the vessel walls [11], which is directly related to the dynamics of blood flow. Research on the flow and pressure patterns in the arteries may help us avoid plaque buildup byrevealing the locations of stenoses. According to a recent study [12], regular exercise may raisethe hydrodynamic pressure on the artery walls, which may have a

mechanically beneficial impact on soft plaque that is comparable to percutaneous coronary intervention therapy. The pulsatile blood flow inside elastic and stiff arteries was examined by the author of [13] using the non-Newtonian Power Law model. Using the images from the CT scan, they created a virtual healthy and stenosed abdominal aorta using the ANSYS-CFX program. According to their findings, the maximal wall shear stress in an unhealthy abdominal aorta is more than it would be in a healthy one. Using the lattice Boltzmann method, De Rosis [14] quantitatively investigated the non-Newtonian blood circulation inside hyperelastic arteries. He used the Casson model to simulate blood flow, and the effects of anisotropic, hardening, and hyperelastic artery walls were explored. A fluid-structure interaction model has been presented to simulate the relationship between blood circulation and plaque caused by atherosclerosis. It was made up of a peripheral layer of plasma that was thought to be a Newtonian fluid and a central area of suspension madeup of all the erythrocytes. The fluid is taken to be laminar, Newtonian, and incompressible to evaluate the impact of fibrous cap thickness and degree of stenosis on susceptible plaque [16]. Corresponding to [17], which deals with coupling an elastic structure characterizing the exterior of a vessel with a generalized Newtonian fluid that accounts for theshear thinning behavior of blood to capture the pulse wave resulting from the connection between blood and the vessel wall in a three-dimensional case study of a healthyartery. To solve some concerns, this interaction might be crudely approximated or ignored. A comparison of Newtonian and non-Newtonian blood flow rates via a stenosed artery known as the carotid was carried out by Rahman [18].

2. Governing Equations

The two-dimensional numerical simulation was run in steady-state conditions. Newtonian blood flow's governing partial differential equations are as follows:

Continuity Equation:

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0$$

Navier Stokes Equation:

$$\rho(u \,.\nabla)\, u = \nabla.(-pI + K) + F$$

$$\rho(\nabla.u) = 0$$

$$K = \mu(\nabla u + (\nabla u)^T)$$

Density of blood	1060 kgm^{-3}
Viscosity of blood	0.004 $Pa.s$
On the inlet	u = 0.1 ms^{-1} and v = 0 ms^{-1}

On the outlet	No viscous stress and Pressure = 0 Pa
Other Boundaries	No slip condition u = v = 0 ms^{-1}
Density of fat	$911 kgm^{-3}$

3. Result& Discussion

The current numerical study is concerned with blood flow via an atherosclerotic human artery. The results were presented in the form of velocity, streamlines, pressure distribution, and viscosity contours.

3.1 Velocity Analysis

Figures 2-5 show the velocity contour for an atherosclerotic artery with inlet velocity 0.1 ms^{-1}. It is observed that, in Figure 2 higher velocity is seen at the central area of the artery whereas the lower velocity is at the wall because of the no-slip condition. The velocity at the entrance is thesame as the initial velocity, but after the atherosclerotic plaque comes, the velocity increases. Furthermore, after the plaque at the outflow, velocity equals that at the entrance. However, in Figure 3, the blood velocity is only that high in the middle portion of the artery. Blood is seen to get blocked in the middle. That is, when the plaque builds up, blood becomes increasingly clogged at the core. Figure 4 shows the maximum velocity near the inlet wall when the plaque is close to it, and Figure 5 shows the highest velocity near the outlet when the plaque is closeto it. This indicates that the region closest to the plaque has the maximum velocity.

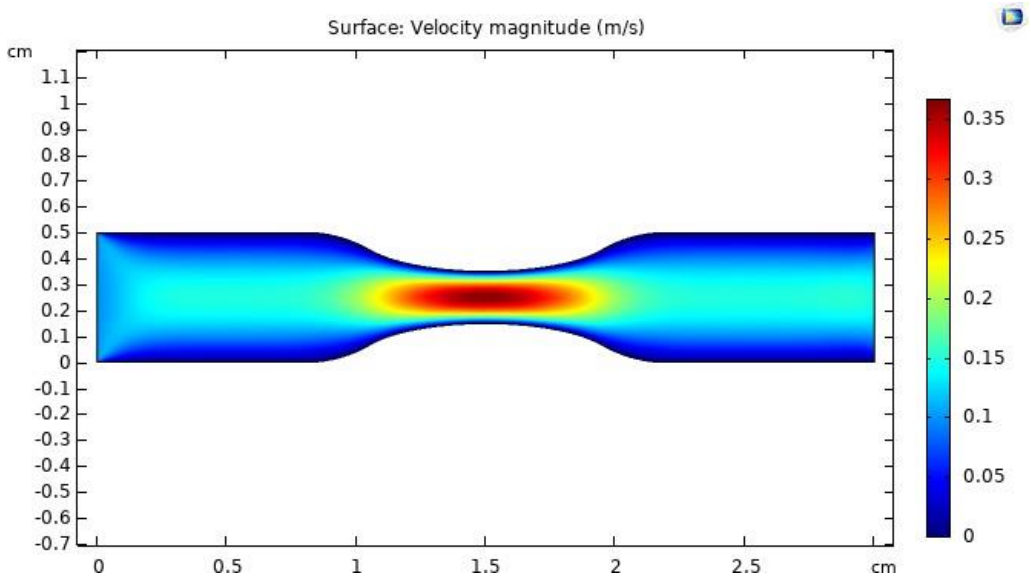

Figure 2: Velocity distribution in atherosclerotic artery

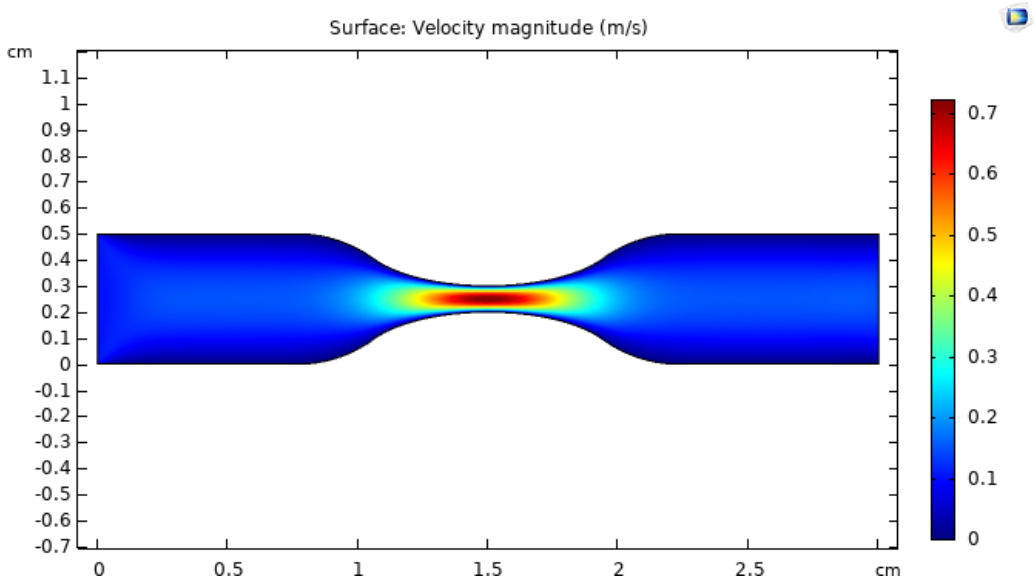

Figure 3: Velocity distribution in atherosclerotic artery

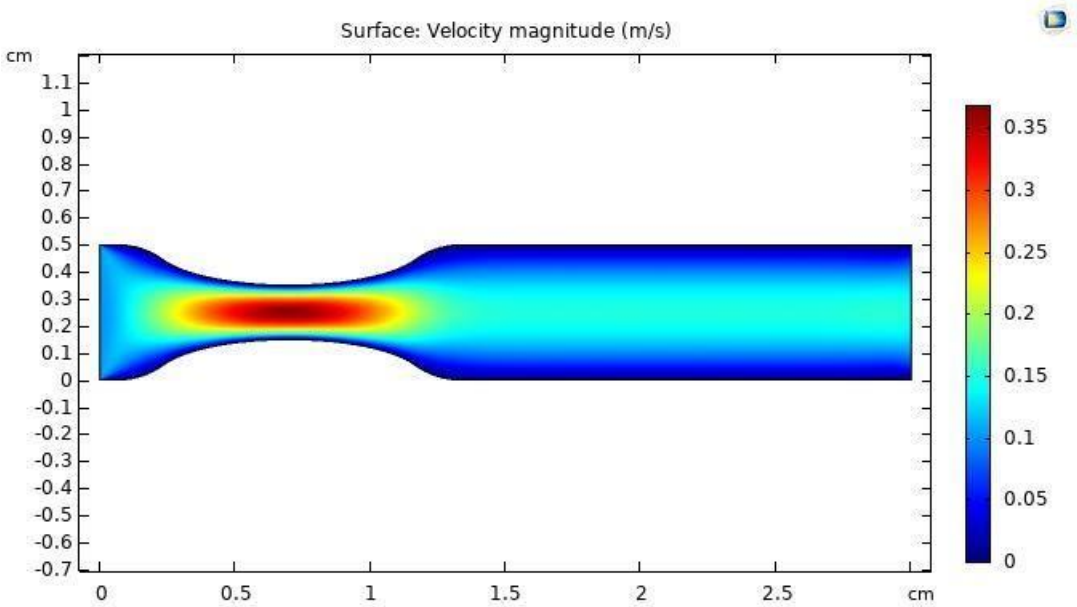

Figure 4: Velocity distribution in atherosclerotic artery

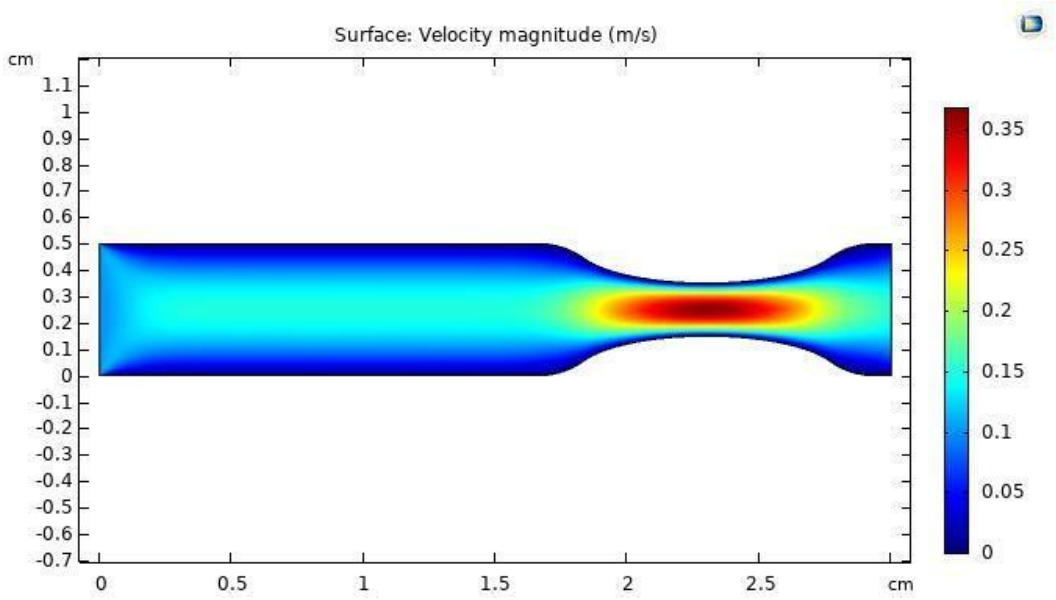

Figure 5: Velocity distribution in atherosclerotic artery

3.2 Pressure Distribution

Figures 6-9 depict the pressure contour for an atherosclerotic artery. Figure 6 shows that pressure is high at the entrance but decreases over time near the atherosclerotic plaque and becomes lower at the outflow. However, in Figure 7, the pressure is higher than in Figure 6 at the entrance and remains constant until the plaque arrives. Following that, the pressure decreases and lowers toward the outflow. Also, when the plaque is near the inlet and outlet in Figures 8 & 9 respectively, pressure decreases as the plaque arrives.

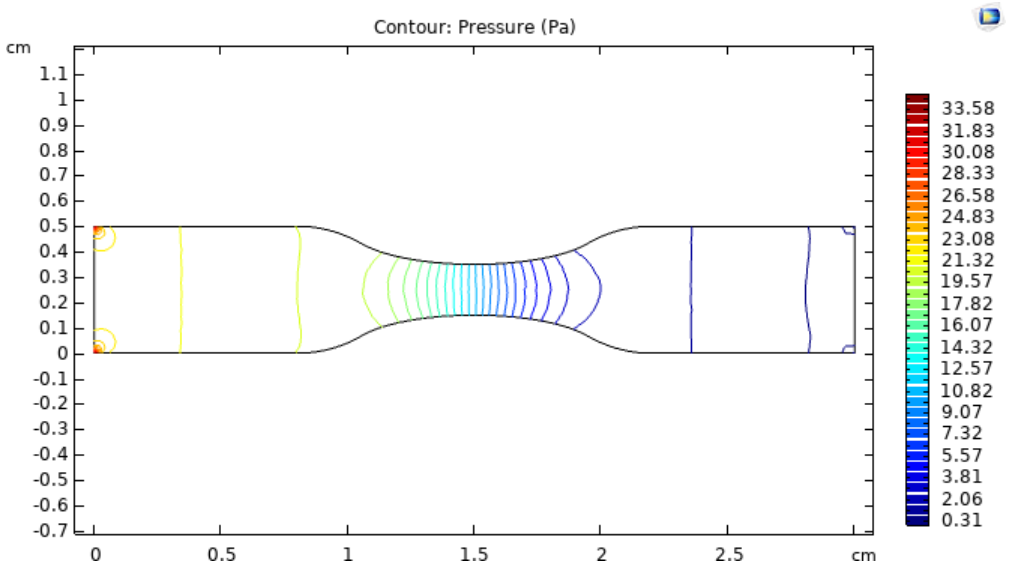

Figure 6: Pressure contour in atherosclerotic artery

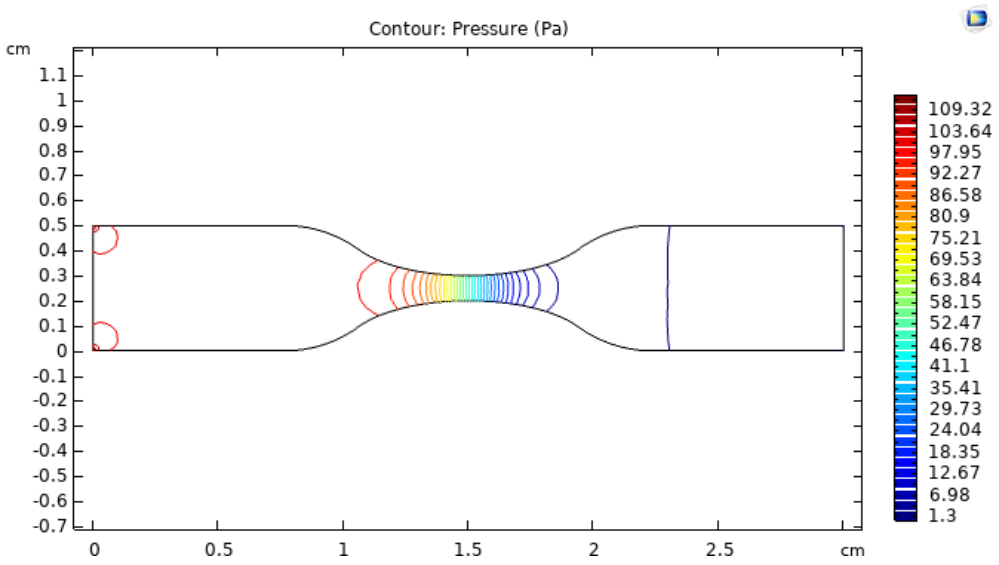

Figure 7: Pressure contour in atherosclerotic artery

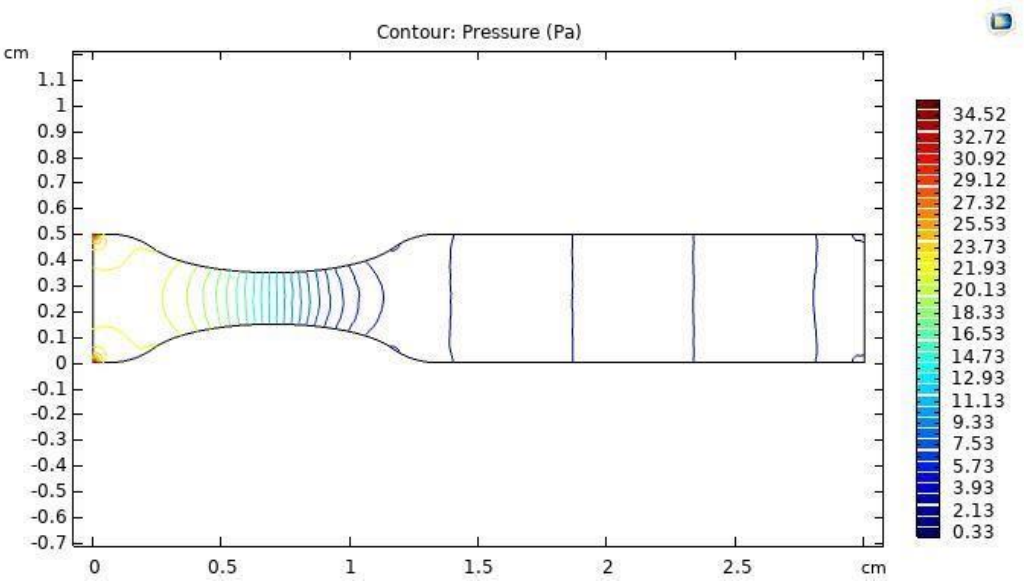

Figure 8: Pressure contour in atherosclerotic artery

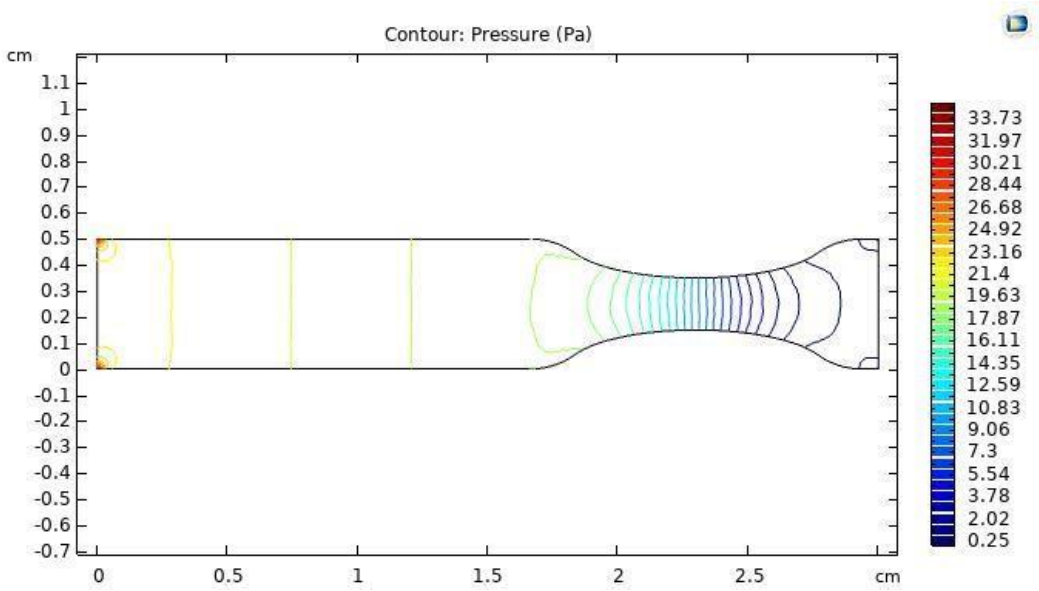

Figure 9: Pressure contour in atherosclerotic artery

3.3 Flow Patterns

Figures 10-13 also illustrate streamlines in the velocity field of the blood in the atherosclerotic artery. The streamlines are nearly identical in Figures 10 & 11, although, toward the middle, the streamlines in Figure 12 are denser than in Figure 13. Since the plaque is close to the inlet in Figure 12, streamlines are deep at the inlet. Furthermore, when the plaque is close to the outlet in Figure 13, streamlines are deep at the outlet.

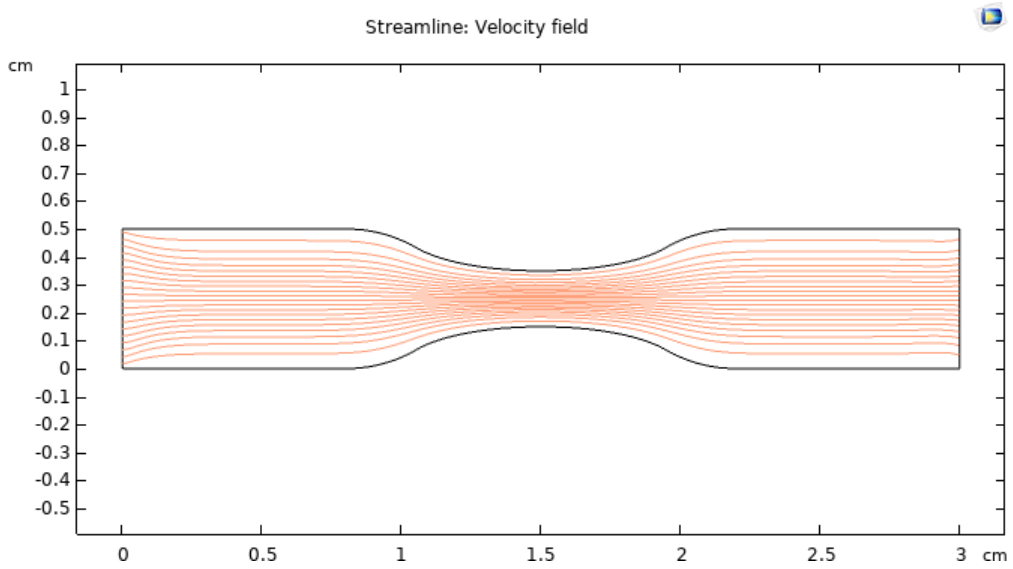

Figure 10: streamlines in the atherosclerotic artery

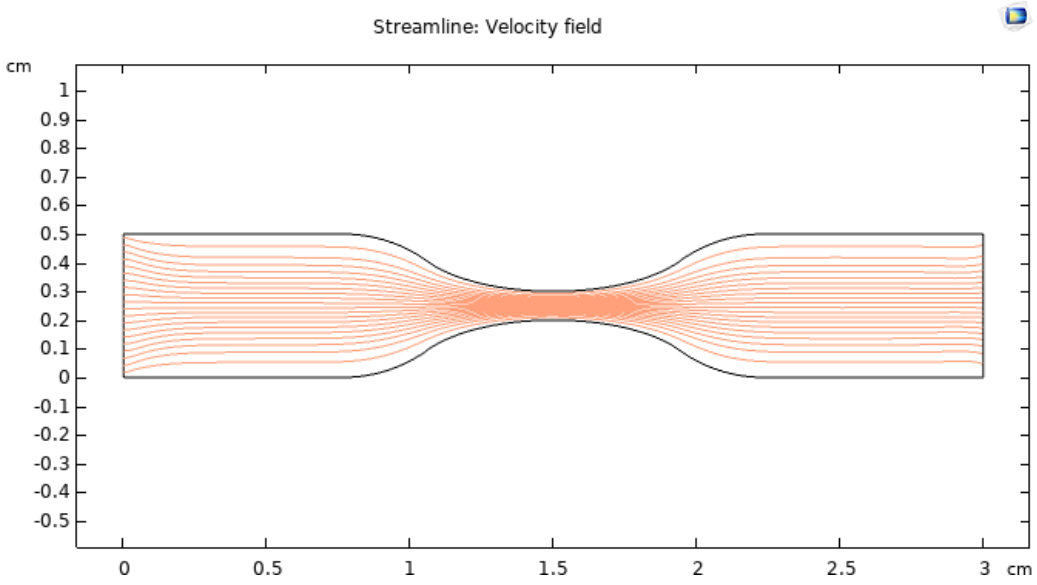

Figure 11: streamlines in the atherosclerotic artery

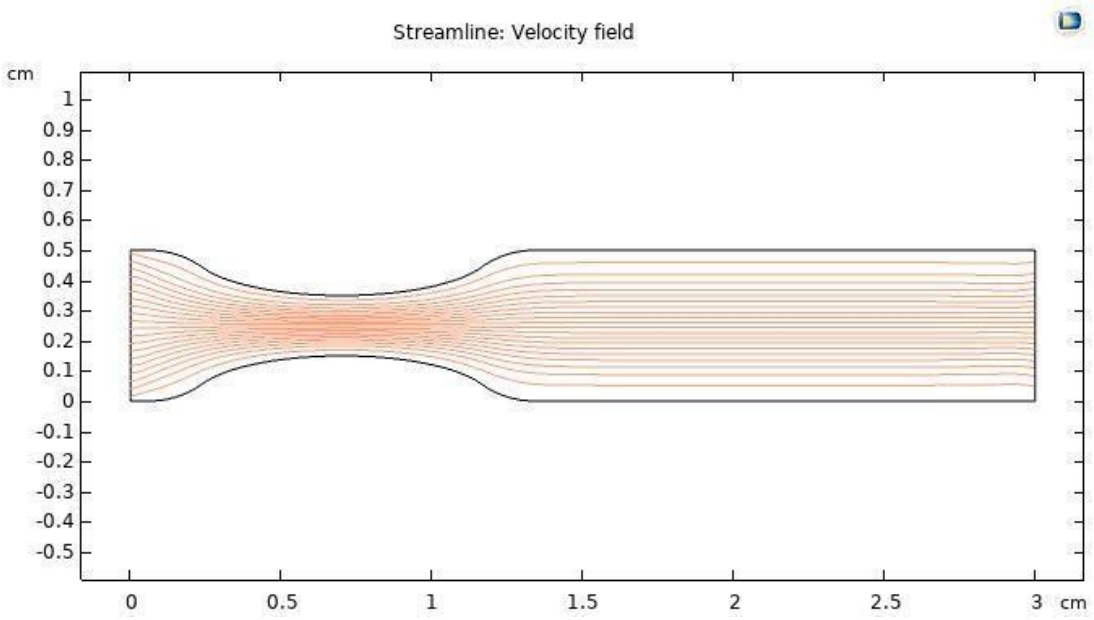

Figure 12: streamlines in the atherosclerotic artery

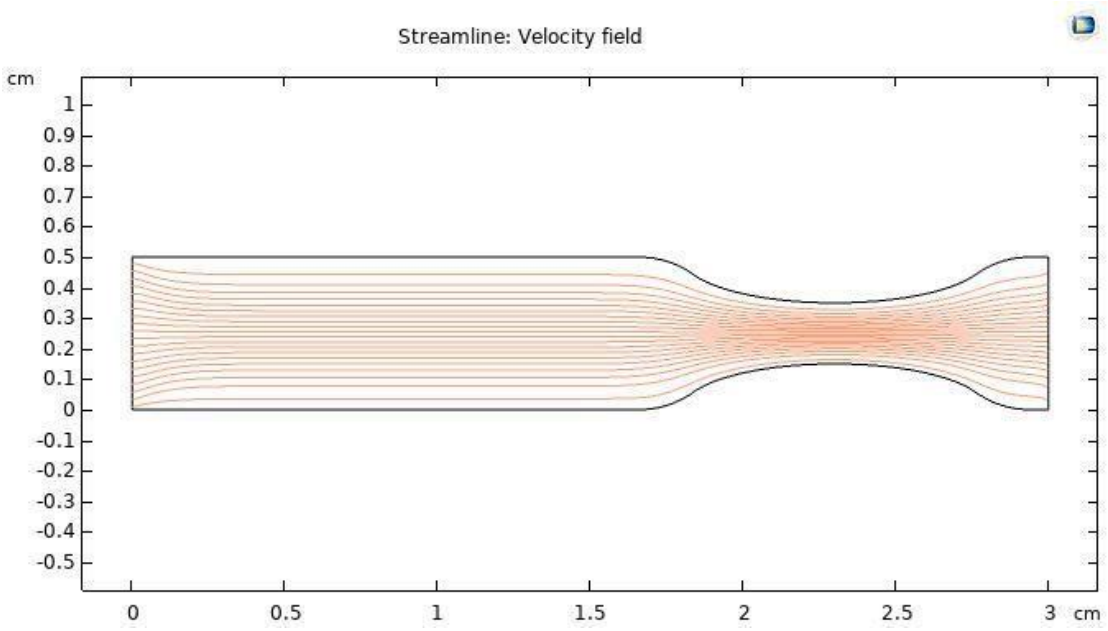

Figure 13: streamlines in the atherosclerotic artery

Conclusion

A numerical investigation of regulating blood flow via an atherosclerotic artery using a Newtonian flow model is carried out in this paper. The governing equations are solved using the finite element approach. The effect of atherosclerotic plaque on velocity surface, pressure, and streamlines has been studied in detail in atherosclerotic arteries. The following findings may be taken from the current investigation:

Different quantity of atherosclerotic plaque has different effects on velocity, and pressure of blood flow. High velocity is observed near the atherosclerotic plaque. Pressure is distributed from high to low on the inlet to the outlet side.

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