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Comprehensive Mathematical Modeling of Atherosclerotic Blood Flow: Impact of Porous Media on Hemodynamics

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Abstract: ***Atherosclerosis, characterized by the narrowing of arteries due to plaque buildup, disrupts normal blood flow and is a major contributor to cardiovascular diseases. This study develops a detailed mathematical model to investigate the hemodynamics of blood flow in arteries affected by atherosclerosis, specifically considering the porous nature of the arterial wall. By treating blood as a non - Newtonian fluid, modeled using Casson's framework, and applying Navier - Stokes and continuity equations, study examines how these factors affect flow velocity, pressure distribution, and wall shear stress. The model addresses laminar, incompressible, and fully developed flow in an artery featuring axially non - symmetric but radially symmetric stenosis. Numerical solutions for volumetric flow rate, pressure drop, and wall shear stress are derived under relevant boundary conditions and presented graphically. The investigation shows that wall shear stress rises with the porous parameter, stenosis size, and stenosis length, but decreases as stenosis shape parameter increases. These findings are consistent with existing research, validating the model's relevance. The insights provided by this study are valuable for both biomedical researchers and medical practitioners, offering a deeper understanding of the impact of atherosclerosis on blood flow and potentially guiding better diagnostic and therapeutic approaches.***

Keywords: Atherosclerosis, Cardiovascular diseases, Porous media, Non - Newtonian fluid, Casson's fluid model, Stenosis, Blood flow dynamics

1. Introduction

Atherosclerosis is a leading cause of cardiovascular diseases, resulting from the accumulation of plaques within the arterial walls, which can impede blood flow and increase the risk of heart attacks and strokes [3, 6, 68, 76, 83]. Understanding the hemodynamic changes due to atherosclerosis is crucial for effective diagnosis and treatment. Traditional models often treat arteries as rigid or elastic tubes without considering the porous nature of arterial walls. This study integrates the porous media effects into the mathematical modeling of blood flow through atherosclerotic arteries, providing a more comprehensive view of the fluid dynamics involved. Several studies have explored blood flow in stenotic arteries using various mathematical models [9, 45, 54, 75, 88]. The porous media theory, initially developed for fluid flow through geological formations, has been adapted to model the transport phenomena in biological tissues. Atherosclerosis, characterized by stenosis a partial blockage of blood vessels caused by the buildup of cholesterol, fats, and abnormal tissue growth significantly impacts blood circulation. Atherosclerotic stenosis is one of the most common abnormalities affecting blood flow [27, 39, 60, 77]. When such a constriction forms, it significantly alters the flow of blood, and fluid dynamics become critical in understanding how stenosis enlarges over time, potentially leading to severe cardiovascular conditions like heart attacks and strokes. The impact of stenosis in a circular tube was first rigorously analyzed by [57, 63, 55, 71, 91]. Stenosis, or the localized narrowing of a blood vessel, often results from abnormal intravascular growths. This condition is particularly prevalent in mammalian arteries, and when severe, it can lead to significant morbidity and mortality [32, 40, 62, 81]. Although the exact mechanisms behind the development of stenosis remain unclear, researchers have suggested that factors such as formation of intravascular plaques and pressure exerted by ligaments and spurs on vessel walls are critical in initiating and progressing this vascular disease. Further investigations by [5, 34, 56, 64, 80] explored various non - Newtonian models to simulate blood flow in large arteries. Their research demonstrated that yield stress of blood essentially force required to start blood flow has little to no impact on either velocity profiles or wall shear stress within these arteries. Some researchers [8, 25, 66] highlighted the limitations of existing studies in accurately modeling plaque growth, pointing to the need for more advanced research in this area. These insights pave the way for future exploration into more effective models of plaque development. Following this, [11, 15, 23, 36] identified critical hemodynamic factors resistance in coronary arteries equipped with catheters, both under normal conditions and in the presence of stenosis. These studies are essential for understanding how these parameters change in pathological states. Similarly, research by [7, 12, 53, 67, 84] examined how the morphology of stenosis affects flow dynamics and parameters like wall shear stress in vessels with very mild stenosis, providing valuable insights into the early stages of arterial disease. Mathematical modeling approaches have also been employed to analyze blood flow through arteries. Some researchers [2, 21, 72, 85] applied model to study interstitial flow through tunica of artery walls contributing to our understanding of flow through porous arterial walls. In a related domain, [19, 58, 69, 87, 90] investigated flow and heat transport in porous media, using mass diffusion and various convective flow models like the Darcy and Brinkman models. They extended work to analyzing energy transport within tissues, providing a comprehensive view of how heat and mass transfer occur in biological systems [29, 43, 49, 82]. These studies collectively enhance our understanding of the complex interactions within stenosed arteries and highlight the importance of integrating diverse modeling techniques to tackle cardiovascular issues

effectively. Some researchers have [4, 13, 48, 51, 65] investigated the flow of a Casson fluid through a tube filled with a porous medium under periodic body acceleration, with applications in artificial organs. Their research highlighted the dynamic effects of body acceleration on fluid flow characteristics. Some have [1, 10, 28, 59, 88] explored the impact of body acceleration on the pulsatile flow of non - Newtonian fluids through stenosed arteries, revealing significant alterations in instantaneous flow characteristics due to body acceleration and also focused on pulsatile blood flow in a constricted artery under body acceleration conditions, observing an increase in velocity and flow rate, alongside a decrease in effective viscosity due to slip at the wall [16, 44, 73, 81]. Present investigation aims to study influence of the porous parameter on blood flow, modeled as Casson's fluid. The numerical analysis provides insights into variations in velocity profiles, volumetric flow rates, and wall shear stress under these conditions. This study contributes to understanding how porous media and fluid characteristics interact in the context of blood flow dynamics, offering implications for biomedical applications and physiological research.

Formulation of the problem: Let's consider axisymmetric flow of blood within a uniform circular artery that exhibits an axially non - symmetric but radially symmetric mild stenosis. Geometry of this stenosis is characterized as [20, 52, 74]:

$$\frac{R(z)}{R_0} = 1 - A[L_0^{(m-1)}(z-d) - (z-d)^m], \qquad d \le z \le d + L_0$$
$$= 1, \qquad \text{otherwise,} \tag{1}$$

$$A = \frac{\delta}{R_0 L_0^m} \frac{m^{m/(m-1)}}{(m-1)},$$

where, R_0 represents radius of normal artery, R (z) is radius in stenotic region, L denotes total length of artery, L_0 is length of stenosed segment, and d is distance between equidistant points. The variable δ indicates maximum height of stenosis, with δ being much smaller than ($\delta << R_0$). The parameter mmm determines shape of stenosis, with m being equal to or greater than 2 ($m \ge 2$) [14, 37, 42, 46].

Casson's fluid model: For Casson's fluid, the relationship between shear stress and shear rate is expressed by the following equation:

$$\tau^{1/2} = \tau_0^{1/2} + (\mu e)^{1/2}, \qquad \tau \ge \tau_0 \tag{2}$$
$$e = \left(-\frac{du}{dr}\right)$$

- τ_0 represents the yield stress of the fluid.
- τ denotes the shear stress tensor.
- e corresponds to the shear strain rate
- u signifies the velocity of the fluid.
- μ stands for the viscosity of the blood, also known as Casson's viscosity coefficient.
- r is the radius of the artery.

Darcy flow model:
The Darcy model, commonly used to describe flow through porous media, is represented by [26, 31, 50]:

$$U = \left(-\frac{kp}{\mu}\right) \tag{3}$$

where, k is porous parameter, μ is viscosity of blood, p is pressure gradient.

Conservation equations: In this study, we investigate the flow dynamics using the Navier - Stokes equations tailored for incompressible non - Newtonian fluids, specifically incorporating the effects of porosity. The fundamental equations of motion in cylindrical polar coordinates are given by [22, 90]:

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial z} + v\frac{\partial v}{\partial t} = -\frac{1}{\rho}\frac{\partial p}{\partial z} + \frac{\mu}{\rho}\left(\frac{\partial^2 u}{\partial^2 r} + \frac{1}{r}\frac{\partial u}{\partial r} + \frac{\partial^2 u}{\partial r^2}\right) \tag{4}$$

$$\frac{\partial u}{\partial t} + u\frac{\partial u}{\partial z} + v\frac{\partial v}{\partial r} = -\frac{1}{\rho}\frac{\partial p}{\partial z} + \frac{\mu}{\rho}\left(\frac{\partial^2 v}{\partial^2 r} + \frac{1}{r}\frac{\partial v}{\partial r} + \frac{\partial^2 v}{\partial r^2} - \frac{v}{r^2}\right) \tag{5}$$

$$\frac{\partial u}{\partial z} + \frac{1}{r}\frac{\partial (vr)}{\partial r} = 0 \tag{6}$$

Boundary conditions: The boundary conditions imposed by the no - slip condition on the stenosis surface are expressed as follows: [30, 61, 78]:

$$u = 0 \qquad \text{at} \qquad r = R_0$$
$$u = 0 \qquad \text{at} \qquad r = R(z) \tag{7}$$

Solution of the problem: By solving this equation, we can determine the velocity of the blood flow, the volumetric flow rate, and the pressure distribution as described in references [33, 86]:

$$v = -\frac{p}{4\mu}\left(R^2 - r^2 - \frac{8}{3}R^{1/2}\left(R^{3/2} - r^{3/2}\right) + 2R\,(R - r)\right) \tag{8}$$

Now total flow flux

$$Q = \int_0^R 2\pi u r\, dr = \pi \int_0^R r\left(-\frac{du}{dr}\right) dr, \tag{9}$$

$$Q = -\frac{\pi p R^4}{8\mu}\left(1 - \frac{16}{7}\left(\frac{r}{R}\right)^{1/2} + \frac{4}{3}\left(\frac{r}{R}\right) - \frac{1}{21}\left(\frac{r}{R}\right)^4\right) \tag{10}$$

$$\frac{dp}{dz} = \left(-\frac{8\mu Q}{\pi R_0^4}\varphi(z)\right) \tag{11}$$

$$\varphi(z) = 1/\left[1 - \frac{16}{7}\left(\frac{r}{R}\right)^{1/2} + \frac{4}{3}\left(\frac{r}{R}\right) - \frac{1}{21}\left(\frac{r}{R}\right)^4\right]$$

$$\Delta p=\int_0^L\left(\frac{dp}{dz}\right)dz=\left(\frac{8\mu Q}{\pi R_0^4}\psi\right) \tag{12}$$

where

$$\psi=\int_0^L\varphi(z)dz=\int_0^d\left[\varphi(z)\right]_{R/R_0=1}dz+\int_d^{d+L_0}\varphi(z)dz+\int_{d+L_0}^L\left[\varphi(z)\right]_{R/R_0=1}dz$$

In the expression derived above, the first and third integrals are relatively straightforward to evaluate. However, the second integral poses a significant challenge and, as such, will be computed numerically [38, 41, 70, 89].

$$p=\frac{8\mu Q}{\pi R_0^4}\int_0^L\frac{dz}{\left(\frac{R}{R_0}\right)^4\left(1-\frac{16}{7}\left(\frac{r}{R}\right)^{1/2}+\frac{4}{3}\left(\frac{r}{R}\right)-\frac{1}{21}\left(\frac{r}{R}\right)^4\right)} \tag{13}$$

Using equation (1) and equation (3) we have,

$$\tau=\left(\frac{U\mu R_0}{2k}\right)^{1/2}\left(\begin{array}{l}\left(1-\frac{16}{7}\frac{\delta}{R_0L_0^m}\frac{m^{m/(m-1)}}{(m-1)}[L_0^{(m-1)}(z-d)-(z-d)^m]\right)^{1/2}\\ \int_d^{(d+L_0)}+\frac{4}{3}\left(\frac{\delta}{R_0L_0^m}\frac{m^{m/(m-1)}}{(m-1)}[L_0^{(m-1)}(z-d)-(z-d)^m]\right)\\ -\frac{1}{21}\left(\frac{\delta}{R_0L_0^m}\frac{m^{m/(m-1)}}{(m-1)}[L_0^{(m-1)}(z-d)-(z-d)^m]\right)^4\end{array}\right) \tag{14}$$

2. Results and Discussions

Simulations reveal how stenosis and arterial wall porosity affect flow velocity and pressure distribution within the artery. As stenosis severity increases, there is a significant drop in flow velocity downstream of the stenosis, accompanied by a steep pressure gradient. The porous media effect moderates the pressure drop, indicating fluid exchange between the blood and the arterial wall. Wall shear stress, a critical factor in endothelial function and plaque stability, is significantly elevated near the stenotic regions. The non - Newtonian model captures the shear - thinning behavior of blood, showing a decrease in apparent viscosity at higher shear rates, which is crucial for understanding blood flow under pathological conditions. Incorporating porous media into the model highlights its role in reducing flow resistance and pressure build - up. The permeability of the arterial wall influences the extent to which fluid exchange occurs, affecting overall hemodynamics. Higher permeability leads to increased fluid absorption by the arterial wall, reducing the shear stress and stabilizing the flow. The findings suggest that therapies targeting the reduction of blood viscosity and enhancing arterial wall permeability could mitigate the adverse effects of atherosclerosis. Medications like Hydroxychloroquine and Doxycycline, which reduce blood viscosity and treat inflammation, could be beneficial for patients with high resistance to blood flow. The development of this mathematical model aims to investigate various aspects of blood flow through a stenosed artery under influence of porous parameters, featuring an axially non - symmetric but radially symmetric mild stenosis. Theoretical results such as velocity profiles, volumetric flow rates, wall shear stress, and pressure gradients have been analyzed. Specifically, numerical solutions for wall shear stress are presented in this study. Wall shear stress plays a critical role in understanding blood flow dynamics, especially in stenosed arteries with porous effects. Computer codes were developed to quantify the effects of porous parameters on wall shear stress. Numerical simulations were conducted to evaluate wall shear stress in diseased systems affected by stenosis, caused by local lipid deposition. To comprehensively analyze blood flow characteristics in presence of porous parameters, numerical results were computed using experimental data from stenosed arteries. The findings are depicted in Figures 1 - 2, illustrating the impact of these parameters on wall shear stress distribution. Figure 1 illustrates the variation of wall shear stress with the porous parameter along the stenosed segment of the artery. It is evident that as the porous parameter increases, the wall shear stress in the stenotic region of the artery also increases, aligning with findings from [24, 47]. Figure 2 shows the relationship between wall shear stress and stenosis size. As the size of the stenosis increases, there is a corresponding increase in wall shear stress. This trend is consistent with observations by [18, 35], where larger stenoses lead to higher wall shear stress in the affected region.

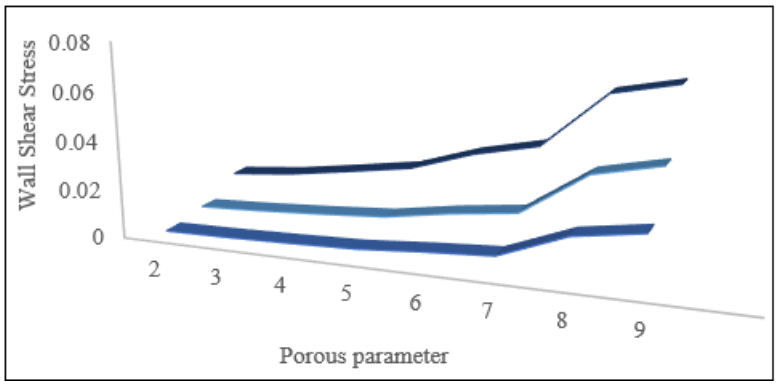

Figure 1: Variation of wall shear stress with porous parameter

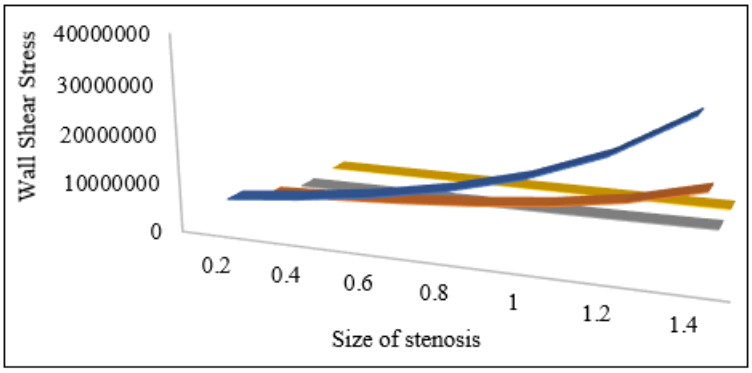

Figure 2: Variation of wall shear stress with stenosis size

3. Conclusion

This study offers a comprehensive analysis of blood flow dynamics in atherosclerotic arteries by incorporating the effects of porous media. The mathematical model developed provides significant insights into how stenosis and arterial wall porosity influence hemodynamics. By treating blood as a non - Newtonian fluid using Casson's model and analyzing flow through arteries with axially non - symmetric but radially symmetric stenosis, we explored key hemodynamic parameters such as wall shear stress, pressure distribution, and volumetric flow rate. Our findings reveal that wall shear stress increases with porous parameter, stenosis size, and stenosis length, while it decreases as the stenosis shape parameter increases. This indicates that the structural and porous characteristics of stenosed arteries significantly affect blood flow dynamics. The study highlights how variations in these parameters can profoundly impact the physiological behavior of blood flow in diseased arteries. The numerical results and graphical representations presented in this research underscore the complex interplay between blood flow and arterial structure in the presence of atherosclerosis. These insights are crucial for enhancing our understanding of the disease and can inform the development of more effective diagnostic and therapeutic strategies for cardiovascular conditions. This model, by accurately predicting the main characteristics of physiological flows, holds valuable implications for biomedical researchers and medical practitioners focused on treating cardiovascular diseases.

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