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Mathematical Modeling for Casson Blood Flow in Stenosed Arteries: A Comparative Study

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Abstract:

Cardiovascular diseases are the leading cause of mortality worldwide. A significant contributor to these conditions is arterial stenosis, which disrupts normal blood flow and increases the risk of critical events like myocardial infarction and stroke.

This study explores the dynamics of blood flow in stenosed arteries using the Casson fluid model to capture its non-Newtonian properties. Focusing on arteries narrowed by plaque accumulation, the study examines the effects of critical parameters such as different stenosis geometries on hemodynamic behaviour. Employing the numerical approaches, the findings reveal significant deviations in velocity profiles in different degree of stenosis. The study provides valuable insights for improving diagnostic and therapeutic strategies in cardiovascular care.

Keywords: Casson fluid, Stenosed arteries, Hemodynamics, Velocity profile, Finite Difference methods

1. Introduction:

Cardiovascular diseases remain the leading cause of mortality worldwide, accounting for millions of deaths annually and placing an immense burden on global healthcare systems. Among these, arterial stenosis, the pathological narrowing of arteries due to plaque buildup, stands out as a critical condition that disrupts normal blood flow and significantly elevates the risk of life-threatening events such as heart attacks and strokes. The interplay between hemodynamic factors and vascular biology in stenosed arteries is both intricate and clinically significant. As blood flows through a narrowed vessel, it encounters increased resistance and turbulence, leading to elevated pressure gradients and shear stresses. These abnormal conditions not only impair oxygen and nutrient delivery to tissues but also contribute to the progression of vascular diseases by promoting endothelial dysfunction, inflammation, and thrombogenesis.

Understanding the hemodynamics of stenosed arteries is therefore fundamental for developing effective diagnostic, therapeutic, and preventive strategies. Mathematical modeling serves as a powerful tool in this endeavor, enabling researchers to simulate and analyze the complex interactions within the circulatory system under both healthy and pathological states. Historically, early models treated blood as a Newtonian fluid, a simplification that overlooked its unique

rheological properties such as shear-thinning behavior and yield stress. The advent of non-Newtonian models, particularly the Casson fluid model, marked a significant advancement, providing a more realistic representation of blood flow dynamics. This study builds on this foundation, focusing on the application of the Casson model to elucidate the effects of critical parameters such as yield stress, hematocrit levels, and stenosis severity—on flow patterns and wall shear stress distributions.

The circulatory system is a complex network responsible for delivering oxygen and nutrients while removing waste products. Blood, a complex suspension of cells in plasma, exhibits non-Newtonian behavior, making its flow distinctly different from simple fluids like water. Hemodynamic changes in stenosed arteries influence systemic health by affecting oxygen delivery and imposing abnormal mechanical stresses on the vascular walls. Moreover, localized flow disruptions contribute to conditions such as myocardial infarction and stroke, underscoring the clinical significance of accurate blood flow modeling.

The rheological properties of blood are critical in determining its flow characteristics. Blood's viscosity varies with shear rate due to its composition of red blood cells, white blood cells, platelets, and plasma proteins. Additionally, a yield stress—the threshold stress required for flow initiation is a

hallmark of blood's behavior. The Casson fluid model, specifically designed to capture these properties, provides a robust framework for analyzing non-Newtonian blood flow in arteries.

Mathematical models have been instrumental in advancing our understanding of hemodynamic processes. They allow for the simulation of physiological conditions that are difficult to replicate experimentally. Such models are critical in predicting clinical outcomes and optimizing treatments. The Casson fluid model, originally developed for pigment-oil suspensions, has been adapted to study blood flow due to its ability to describe shear-thinning behavior and yield stress [1,4,16].

Mathematical modeling is a cornerstone of hemodynamic research, enabling the exploration of blood flow dynamics in healthy and pathological conditions. These models integrate principles from fluid mechanics, computational physics, and biological systems to simulate real-world scenarios and provide insights into vascular behavior under varying physiological conditions. This section provides an in-depth exploration of mathematical modeling, its historical evolution, applications, and challenges in the context of blood flow and arterial stenosis [5,20].

The mathematical modeling of hemodynamics has its roots in the application of classical fluid dynamics to biological systems. Early models treated blood as a Newtonian fluid, simplifying the analysis but neglecting key rheological properties. Over the decades, advancements in computational methods and the availability of experimental data led to the development of non-Newtonian models, including the Casson model. These models addressed limitations by incorporating factors like shear thinning, yield stress, and viscoelasticity [2,3,8,17].

By employing a combination of numerical techniques and computational simulations, the research aims to bridge gaps in current knowledge and offer insights that are directly relevant to clinical practice. The ultimate goal is to enhance our understanding of how stenosis alters hemodynamics and to inform the design of better diagnostic tools and medical devices, such as stents and grafts, tailored to individual patient needs [7,9,15].

Modeling blood flow in stenosed arteries presents unique challenges due to the intricate interplay of fluid dynamics and vascular geometry. Arterial stenosis disrupts laminar flow, creating regions of high shear stress and complex vortices. Accurately capturing these phenomena requires sophisticated mathematical formulations and computational methods. Traditional Newtonian models fall short in representing the

complexities of blood flow under such conditions, highlighting the need for advanced non-Newtonian models like the Casson fluid framework.

Yang er. al. [21] developed and numerically solved a model evolution of plaques in arteries. Siddiqui et. al. [14] studied the effect of pulsatility and effect of non-Newtonian nature of blood flow through an artery having stenosis by considering blood as a casson fluid. Priyadharshini and Ponalagusamy [6] used numerical finite difference method to solve mathematical model of flow of blood carrying magnetic nanoparticles and investigated the magnetohydrodynamics effects. In the study of modelling blood flow Shah et. al. [11] found that if the hight of stenosis increases, the resistance of flow also increases. Srivastava and Saxena [15] analysed the blood flow physiological characteristic in stenotic artery by considering blood as two fluid model. Sarifuddin et. al. [10] studied the flow of blood in asymmetric constriction of Artery. Some authors analysed the flow properties of blood in stenotic artery (Varshney et al. [18,19,20], Mishra and Pandey [14]). In this paper, a mathematical model is developed to study the effect of stenosis geometry on blood flow in artery by considering blood as Casson fluid.

2. Mathematical Model

The artery with stenosis is modeled as a cylindrical elastic tube with a circular cross-section containing an incompressible, non-Newtonian fluid. The blood flow is assumed to be laminar, unsteady, two-dimensional, axisymmetric, and fully developed. The Casson model characterizes the blood's rheological properties. The flow occurs under the influence of an externally imposed periodic body acceleration in the axial direction. Based on these assumptions, the governing equations are formulated in the cylindrical coordinate system (r, z, θ) as follows.

$$\begin{split} \frac{\partial u_r}{\partial r} + \frac{u_r}{r} + \frac{\partial u_z}{\partial z} &= 0 \\ \frac{\partial u_z}{\partial t} + u_r \frac{\partial u_z}{\partial r} + u_z \frac{\partial u_z}{\partial z} &= \frac{1}{\rho} \frac{\partial p}{\partial z} - \frac{1}{\rho} \left[\frac{1}{r} \frac{\partial}{\partial r} \left(\sigma_{rz} + \frac{\partial}{\partial z} \left(\sigma_{zz} \right) \right] + \alpha(t) \end{split}$$

$$(2)$$

$$\frac{\partial u_r}{\partial t} + u_r \frac{\partial u_r}{\partial r} + u_z \frac{u_r}{\partial z} &= -\frac{1}{\rho} \frac{\partial p}{\partial r} - \frac{1}{\rho} \left[\frac{1}{r} \frac{\partial}{\partial r} \left(\sigma_{rr} \right) + \frac{\partial}{\partial z} \left(\sigma_{rz} \right) \right] \end{split}$$

$$(3)$$

Here $u_z(r,z,t)$ and $u_r(r,z,t)$ denotes the axial and the radial velocity components respectively. p is the pressure and ρ is the density of flowing blood. The pressure gradient $\partial p/\partial z$ appearing in equation (2), is given by

$$-\frac{\partial p}{\partial z} = P_0 + P_1 Cos\mu t, t > 0.$$
(4)

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Where $P_0 \rightarrow$ constant amplitude of the pressure gradient

 $P_1 \rightarrow$ amplitude of the pulsatile component giving rise to systolic and diastolic pressure

and $\mu = 2\pi c_p$, where c_p is the pulse frequency.

The externally imposed body acceleration $\alpha(t)$ is given by

$$\alpha(t) = a_0 \cos(\omega_b t + \phi) \text{ for } t \ge 0.$$
(5)

Where a_0 is its amplitude, $\omega_b = 2\pi f_b$, f_b is its frequency and φ is its phase difference.

3. Boundary Conditions

(1)
$$u_r(r, z, t) = 0, \frac{\partial u_z(r, z, t)}{\partial r} = 0 \text{ at } r = 0$$

(2)
$$u_r(r, z, t) = \frac{\partial R}{\partial t}$$
 and $u_z(r, z, t) = 0$ at $r = R(z, t)$
(7)

(3)
$$u_r(r, z, t) = 0$$
 and $u(r, z, t) = 0$ at $t = 0$
(8)

4. Non-Newtonian Model

In Blood is considered a Casson fluid because it exhibits non-Newtonian behavior, meaning its viscosity changes with shear rate rather than remaining constant. Unlike Newtonian fluids, blood requires a minimum shear stress before it starts flowing due to the presence of red blood cell aggregates at low shear rates, which resist movement. This property is well captured by the Casson model, making it a suitable choice for studying blood flow in various physiological and pathological conditions. Additionally, blood behaves as a shear-thinning fluid, where its viscosity decreases as the shear rate increases, caused by the breakdown and alignment of RBCs in the flow direction. The Casson model provides a better fit to experimental data, particularly at low shear rates, compared to Newtonian models, making it useful for hemodynamic studies. This is especially important in analyzing blood flow in stenosed arteries, where both low and high shear regions exist, significantly affecting velocity profiles, wall shear stress, and pressure drop. By incorporating the yield stress and shear-thinning properties, the Casson model enhances the accuracy of predictions in mathematical and computational studies, making it a valuable tool for understanding realistic blood flow dynamics in the circulatory system.

$$\begin{split} \tau^{1/2} &= \mu^{1/2} e^{1/2} + \ \tau_0^{1/2} \qquad (\tau \geq \tau_0) \ , \\ e &= 0 \qquad (\tau \leq \tau_0). \end{split}$$

5. Geometry of stenosed artery:

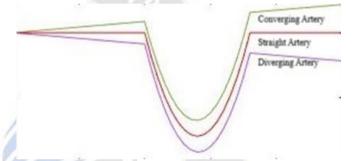
The geometry of the time-variant mild stenosis arterial segment for different taper angle is mathematically given by

$$R(z,t) = \begin{cases} \left[dz + \delta - \frac{4\tau_m Sec\varphi(z-b)}{l^2} (l - (z-b)) \right] \delta_1(t), & b \le z \le b+l \\ (mz + \delta)\delta_1(t), & ; otherwise \end{cases}$$
(9)

Where the slop for tapering in artery is represented as $\tan \phi$.

The time variant parameter $\delta_1(t) = 1 - b \cos(\omega t - 1)$ $e^{-b\omega t}$; $\omega = 2\pi f_p$.

Here $\phi < 0$ is represented as the converging artery, $\phi = 0$ is straight artery and $\phi > 0$ is the diverging artery.



Conclusion:

This study presented a mathematical model for Casson fluid flow through stenosed arteries, aiming to enhance the understanding of non-Newtonian blood flow characteristics. The Casson model effectively captures the yield stress behavior of blood, providing a more realistic representation compared to Newtonian models.

The developed mathematical model illustrated that stenosis significantly affects velocity profiles, shear stress distribution, and pressure drop. The yield stress inherent in Casson fluid alters the flow dynamics, reducing flow resistance in lower shear regions while increasing it in high shear regions near the arterial walls. The model also demonstrated that an increase in stenosis severity leads to a considerable rise in pressure drop, which has clinical implications for cardiovascular diseases. Overall, the study contributes to the study of hemodynamics by offering insights into the role of non-Newtonian properties in blood flow through stenosed arteries.

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