

Characterizing Blood Flow in Constricted Radially Non-Symmetric Multiple Stenosed Arteries: A Herschek-Bulkley Fluid Model Approach

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Abstract: *This work delves into the study of blood flow in a complex scenario: a two-dimensional model of a constricted, radially non-symmetric, multiple stenosed artery. Blood, in this case, is modeled as a Herschek-Bulkley fluid, with its behavior governed by the generalized form of the Navier-Stokes equation. The set of expressions have derived for various blood flow characteristics, including resistance to flow and wall shear stress. Specifically, these characteristics have investigated at points of maximum depression within a single loop stenosis. The findings revealed that as the height of stenosis increases, the resistance to flow also increases. Additionally, it was observed that wall shear stress increases with axial velocity for higher values of the stenosis shape parameter. This research sheds light on the intricate dynamics of blood flow in complex arterial geometries, providing insights that could be valuable for understanding and potentially treating conditions related to arterial stenosis.*

Keywords: Herschek-Bulkley fluid, Arterial stenosis, Navier-Stokes equation, Radially non-symmetric, Multiple stenoses, Resistance to flow, Wall shear stress, Stenosis shape parameter

1. Introduction

Atherosclerosis stands as a primary culprit behind heart attacks and strokes, characterized by the ongoing narrowing of arterial lumens and the hardening of artery walls due to lipid accumulation in the intima layer. This progressive buildup of deposits within arterial walls can lead to the formation of plaques, which protrude into the lumen, obstructing blood flow. If the carotid artery is affected, it can precipitate strokes, while coronary artery involvement can lead to heart attacks [7,14,27,46]. Moreover, the forces exerted by flowing blood on the plaque surface undergo corresponding changes. Stenosis, the abnormal narrowing of blood vessels, contributes significantly to this condition [9,19,58]. Researchers have underscored the formation of intravascular plaques and the impact of ligaments and spurs on blood vessel walls as major factors driving the initiation and progression of atherosclerosis [4,33,54,73]. In the realm of fluid dynamics, numerous investigations have delved into the complexities surrounding non-Newtonian fluids, particularly in relation to nanoparticle utilization across various conditions [2,16,38,44,48,64]. Some researchers have employed a Casson-like fluid model to delve into the longitudinal transport of nanoparticles within blood, highlighting the importance of understanding blood rheology and vascular permeability in this context. Others have expanded this inquiry by studying the flow of nanofluids through arteries affected by composite stenosis and porous walls. Numerous scholars have contributed to comprehending the dynamics of blood flow within stenosed arteries, analyzing the intricate characteristics of blood flow in these conditions [22,43,56,68]. A consensus across several publications emphasizes the crucial role of understanding blood flow and its properties in elucidating its impact on the cardiovascular system. At low shear rates, blood demonstrates non-Newtonian behavior due to its composition as a suspension of cells. Some researchers have developed mathematical models for blood flow, considering factors such

as the velocity slip condition at the artery wall [1,36,45,62]. Additionally, some have explored the influence of slip velocity on non-Newtonian Power-law fluids over a surface generating heat. This research aims to clarify the effects of the peripheral layer and slip condition on nanoparticle transport within capillaries, while also investigating the impact of the power law index on nanoparticle velocity and concentration within a base fluid [17,34,59]. The exploration of blood flow within arterial stenosis holds significant implications for both medical and engineering fields. Arterial stenosis, characterized by the narrowing or development of plaques within the arterial system, disrupts the normal flow of blood, hindering the adequate delivery of oxygen-rich blood to essential organs [30,53,75]. This disruption can result in elevated blood pressure and various injuries, posing significant health risks over time, potentially leading to fatalities. Researchers have explored blood flow through stenosed arteries using various methodologies. Some have utilized perturbation methods to investigate blood flow through permeable-wall stenosis, while others have employed mathematical analysis and finite difference methods to study blood flow behavior in constricted arteries [10,35,55]. Additionally, some researchers have examined flow dynamics in tapered arteries, treating blood as a non-Newtonian fluid and conducting numerical investigations. Comparative studies have been conducted, treating narrowed artery surfaces as two-fluid models, revealing that resistance to flow increases with stenosis width and height. Furthermore, some researchers have delved into the realm of nanoscience, focusing on nanoparticle concentration in blood flow and analyzing parameters such as source, sink, clot size, and stenosis height [29,41,47,77]. Others have examined abnormal blood flow through diseased arteries using Navier-Stokes equations, evaluating parameters like pressure drop and the ratio of minimum to maximum shear stress. Several investigations have aimed to understand the consequences of arterial stenosis on blood flow, including examinations of non-Newtonian fluid behavior in microchannels, along with

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research into additional arterial diseases. The significant findings of studies [3,6,31] highlight the physiological importance of variations in resistance to flow and wall shear stress with axial distance. Theoretical results presented by [8,26,52] elucidate velocity profiles, pressure drop, wall shearing stress, and separation phenomena for specific geometries within the Newtonian model of blood flow. The effects on the cardiovascular system, as explored in the series of papers by [7,40,69,79], are discernible through the study of blood flow in its immediate vicinity. However, it's worth noting that blood doesn't always adhere to Newtonian behavior. Given its composition as a suspension of cells, blood behaves as a non-Newtonian fluid, particularly at low shear rates, as affirmed by [9]. Additionally, [20,42,49,71] indicates that blood flow in tubes with small diameters (less than 0.2 mm) and at shear rates below 20 sec⁻¹ can be represented by a power-law fluid model. Notably, the discussed models haven't addressed radially non-symmetrical stenosis. In this current analysis, a mathematical model for blood flow through radially non-symmetrical stenosis has been developed, considering an improved generalized geometry of multiple stenoses located at equispaced points

[5,21,51,65,74]. For simplicity, graphical analysis is conducted for a single loop of stenosis, featuring maximum depression at various points.

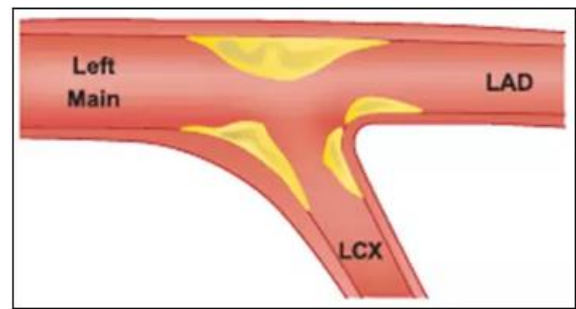


Figure 1: Artery with multiple stenosis

Formulation of the problem: In the current analysis, we assume that the stenosis forms within the arterial wall and is symmetrical about the axis but asymmetrical concerning radial coordinates. In such instances, the radius of the artery, denoted as R(z), can be expressed as:

$$\frac{R(z)}{R_0} = \begin{cases} 1 - A[L_0^{(m-1)}(\alpha z - kd - (k-1)L_0) - (\alpha z - kd - (k-1)L_0)^m] & k(d+L_0) - L_0 \leq \alpha z \leq k(d+L_0) \\ 1 & \text{otherwise} \end{cases} \quad (1)$$

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

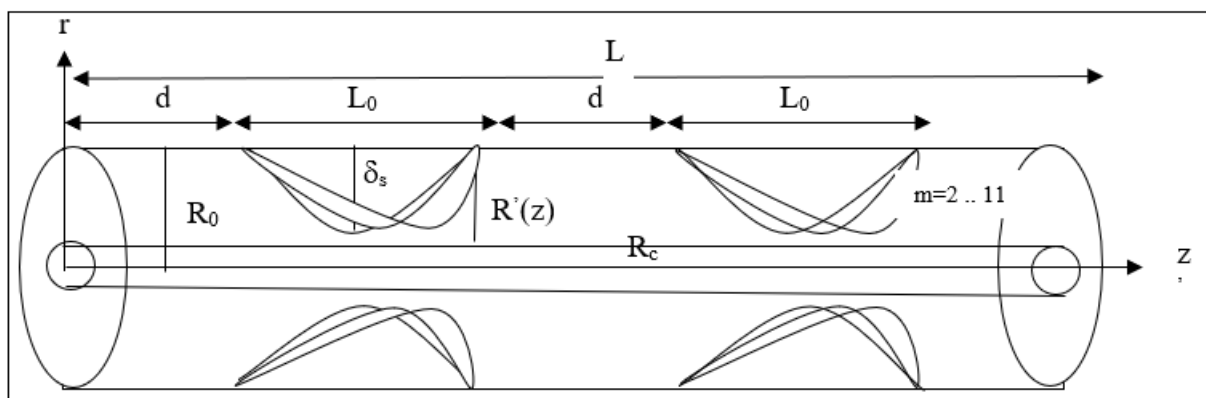


Figure 2: Stenosed artery

$$z = \left[\frac{kd + (k-1)L_0 + L_0 / m^{1/(m-1)}}{\alpha} \right] \quad (2)$$

Following boundary conditions are introduced [24,37];

$$\partial u / \partial r = 0 \text{ at } r = 0 \quad u = 0 \text{ at } r = R(z).$$

$$\tau \text{ is finite at } r = 0 \quad (4)$$

$$P = P_0 \text{ at } z = 0 \quad P = P_L \text{ at } z = L$$

Conservation Equation and boundary conditions: It has studied that the smooth and unchanging flow of blood in a straight artery, assuming it's fully developed and doesn't compress, and considering that its thickness changes as you move outwards from the center [71,82], we end up with a specific equation that describes how the blood moves [5,12];

$$\left. \begin{aligned} 0 &= -\frac{\partial P}{\partial r} + \frac{1}{r} \frac{\partial(r\tau)}{\partial z} \\ 0 &= -\frac{\partial P}{\partial r} \end{aligned} \right\} \quad (3)$$

Analysis of the problem:

Herschel-Bulkley fluid model- The stress-strain relation of Herschel-Bulkley fluid is given as [11,13,32]:

$$f(\tau) = \left(-\frac{du}{dr}\right) = \frac{1}{\mu}(\tau - \tau_0)^n, \quad \tau \geq \tau_0$$

$$f(\tau) = \left(-\frac{du}{dr}\right) = 0, \quad \tau \leq \tau_0 \quad (5)$$

$$\text{where } \tau = \left(-\frac{dp}{dz} \frac{r}{2}\right), \quad \tau_0 = \left(-\frac{dp}{dz} \frac{R_c}{2}\right),$$

By equation (2) and (3) we get,

$$\left(\frac{du}{dr}\right) = -\left(\frac{p}{2\mu}\right)^{1/n} \left[(r - R_c)^{1/n}\right], \quad (6)$$

Flow of flux is as below;

$$Q = \int_0^R 2 p u r dr = p \int_0^R r^2 \left(-\frac{du}{dr}\right) dr, \quad (7)$$

$$Q = \frac{\pi}{2} \left(\frac{P}{2\mu}\right)^{1/n} \frac{R^{(3+1/n)}}{(1+1/n)} f(y), \quad (8)$$

$$\text{where } f(y) = \left[2\left(1 - \frac{R_c}{R}\right)^{((1/n)+1)} - \frac{4}{((1/n)+2)} \left(1 - \frac{R_c}{R}\right)^{((1/n)+2)} + \frac{4}{((1/n)+2)((1/n)+3)} \right.$$

$$\left. \left(\left(1 - \frac{R_c}{R}\right)^{((1/n)+3)} - \left((-1)^{((1/n)+3)} \left(\frac{R_c}{R}\right)\right) \right)\right],$$

$$P = \left(-\frac{dp}{dz}\right) = \frac{2\mu}{R^{(1+3n)}} \left(\frac{2Q}{\pi f(\bar{y})} \left(1 + \frac{1}{n}\right)\right)^n \quad (9)$$

$$\Delta P = P_L - P_0 = \frac{2\mu}{\pi R_0^{1+3n}} \left(2Q \left(\frac{1}{n} + 1\right)\right)^n \int_0^L \frac{dz}{\left(\frac{R(z)}{R_0}\right)^{(1+3n)} (f(\bar{y}))^n} \quad (10)$$

The resistance to flow is as below [25,50];

$$\lambda = \frac{P_L - P_0}{Q} \quad (11)$$

$$\lambda_0 = \frac{2\mu}{R_0^{1+3n}} \left(\frac{2Q(1+1/n)}{\pi}\right)^n (M) \quad (12)$$

$$M = \left(\int_0^d \frac{dz}{(f_0)^n} + \int_d^{d+L_0} \frac{dz}{\left(\frac{R(z)}{R_0}\right)^{1+3n} (f(\bar{y}))^n} + \int_{d+L_0}^L \frac{dz}{(f_0)^n} \right)$$

$$f_0 = \left[2\left(1 - \bar{y}_1\right)^{(1+1/n)} - \frac{4}{(1/n+2)} \left(1 - \bar{y}_1\right)^{(2+1/n)} + \frac{4}{(2+1/n)(3+1/n)} \left(\left(1 - \bar{y}_1\right)^{(3+1/n)} - \left((-1)^{(3+1/n)} \bar{y}_1\right) \right) \right],$$

$$\text{where } \bar{y}_1 = \frac{R_c}{R_0}$$

$$\lambda_N = \frac{2\mu}{R_0^{1+3n}} \left(\frac{2Q(1+1/n)}{\pi}\right)^n \frac{L}{(f_0)^n} \quad (13)$$

$$\lambda = \frac{\lambda_0}{\lambda_N} = 1 - \frac{L_0}{L} + \frac{(f_0)^n}{L} \int_d^{d+L_0} \frac{dz}{\left(\frac{R(z)}{R_0}\right)^{1+3n} (f(\bar{y}))^n} \quad (14)$$

2. Result and Discussion

To assess the quantitative impact of the stenosis shape parameter ($m= 2...11$) and stenosis size on resistance to flow and wall shear stress, computer codes were developed. These codes were used to evaluate analytical results for resistance to blood flow and wall shear stress in diseased systems afflicted with stenosis due to localized lipid deposition [15,39,40].

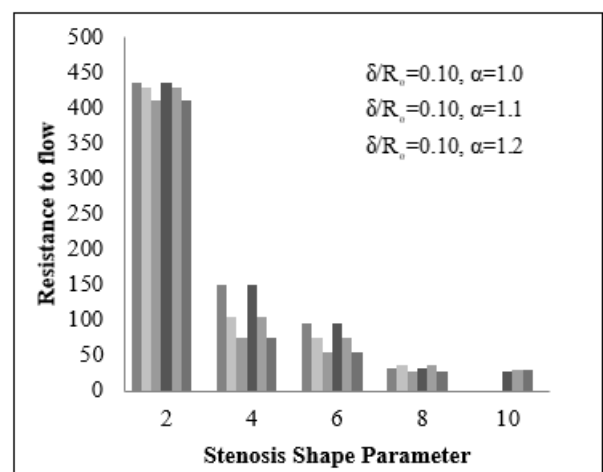


Figure 2: Variation of Resistance to flow with Stenosis shape parameter

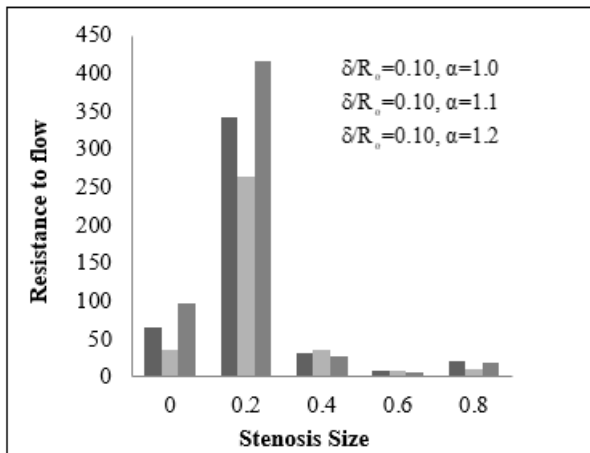


Figure 3: Variation of Resistance to flow with stenosis size

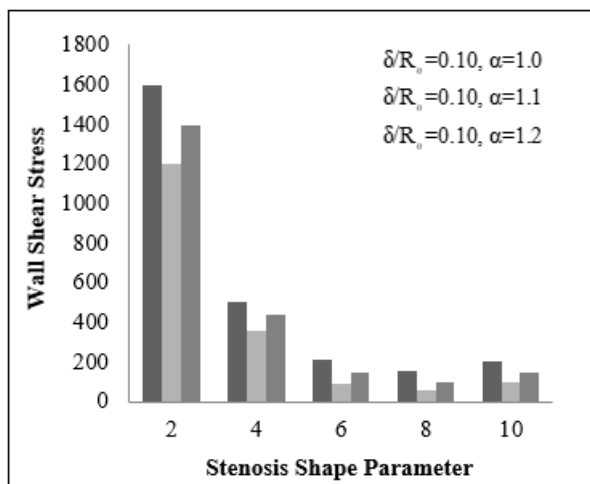


Figure 4: Variation of Wall Shear Stress with Stenosis shape parameter

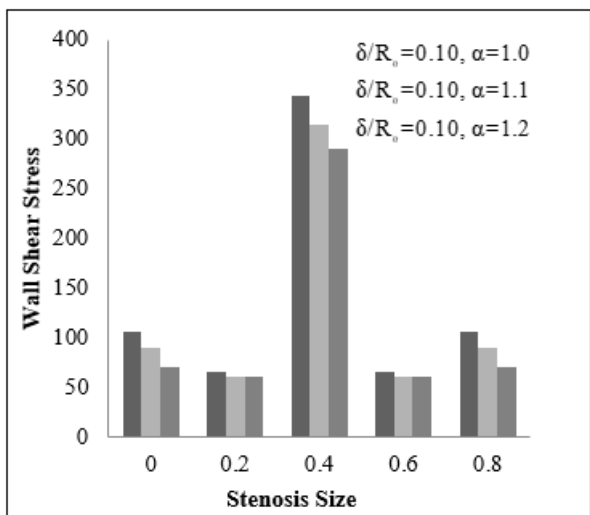


Figure 5: Variation of wall shear stress with stenosis size

The obtained results, based on experimental data from stenosed arteries, are illustrated in Figures 3-6. Figure 3 depicts the relationship between resistance to flow (λ) and the stenosis shape parameter (m). It is noted that resistance to flow decreases as the stenosis shape parameter (m) increases, with maximum resistance occurring at $m = 2$, indicating symmetric stenosis [57,60,78]. This aligns with previous findings. Figure 4 shows the relationship between resistance to flow (λ) and stenosis size (δ/R_0), indicating an increase in

resistance as stenosis size increases. This phenomenon is consistent with the Fahraeus-Lindquist effect observed in very thin tubes [72,81,84]. Figure 5 illustrates the relationship between wall shear stress and the stenosis shape parameter (m), demonstrating a decrease in wall shear stress as the stenosis shape parameter (m) increases. These findings correspond to previous research [60,67,80]. Figure 6 displays the relationship between wall shear stress (τ) and stenosis size, indicating an increase in wall shear stress as stenosis size increases, in line with previous observations [61,66,76,83].

3. Conclusion

This study described a theoretical and analytical investigation into blood flow through arteries affected by multiple stenoses of various shapes. The study employs numerical experiments to understand the impact of blood flow in arteries with radially non-symmetric multiple stenoses. Unlike single, regular-shaped stenoses, radially non-symmetric multiple stenoses have a more significant effect on blood flow dynamics. The findings from this study can be valuable for biologists and medical practitioners, providing insights into how blood flow is altered in the presence of complex arterial geometries, which could aid in the diagnosis and treatment of cardiovascular diseases.

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