

Analytical Study of Non-Newtonian Blood Flow through Arterial Stenosis under Hepatic Disorder Conditions

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Abstract: Blood flow through stenosed arteries has attracted considerable attention in biomedical engineering, physiology, and bio-fluid mechanics because arterial narrowing significantly alters hemodynamic behavior and pressure distribution. Hepatic disorders such as Hepatitis A influence blood viscosity, hematocrit concentration, and vascular resistance, thereby affecting arterial circulation. The present study develops a mathematical model for non-Newtonian blood flow through stenosed hepatic arteries using two-phase fluid assumptions and Herschel–Bulkley rheological behavior. The governing equations of continuity and momentum conservation are formulated in cylindrical coordinates and solved under physiological boundary conditions. Expressions for velocity distribution, wall shear stress, volumetric flow rate, and pressure drop are derived analytically. Clinical hematocrit data are incorporated to examine the variation of pressure drop with blood viscosity. The results indicate that stenosis severity and hematocrit significantly increase pressure resistance and reduce volumetric blood flow. Graphical analysis further reveals that blood pressure drop rises sharply with increasing hematocrit concentration. The study concludes that mathematical modeling of stenosed arterial flow provides important biomedical insight into hepatic circulatory disorders and may assist in clinical diagnosis and therapeutic planning.

Keywords: Arterial Stenosis, Non-Newtonian Blood Flow, Hematocrit, Hepatic Artery, Herschel–Bulkley Model, Pressure Drop, Bio-Fluid Mechanics, Mathematical Modeling.

1.Introduction

Blood circulation inside arteries is one of the most important physiological processes responsible for oxygen transport, nutrient supply, and metabolic regulation. Any abnormality in vascular geometry or blood rheology can significantly affect hemodynamic behavior and tissue functioning. Among vascular abnormalities, arterial stenosis is particularly important because narrowing of blood vessels increases circulatory resistance and disturbs normal blood flow [1-2].

Stenosis refers to abnormal constriction of arteries due to plaque accumulation, inflammation, or pathological tissue growth. In hepatic circulation, stenosis can significantly affect blood transport and liver functioning. Hepatitis A infection further alters hematocrit concentration, blood viscosity, and vascular resistance, thereby increasing circulatory complications [3-5].

Blood behaves as a complex non-Newtonian fluid because it contains plasma, red blood cells, white blood cells, and platelets suspended in liquid medium. The rheological behavior of blood changes with vessel diameter, hematocrit concentration, and shear rate [6-8]. In large arteries blood approximately behaves as a Newtonian fluid, whereas in narrow or diseased arteries non-Newtonian effects become highly significant.

Mathematical modeling provides a scientific framework for understanding blood circulation under physiological and pathological conditions. Bio-fluid mechanical equations based on continuity and momentum conservation are widely used to study arterial flow behavior



[9-10].

The present study develops an analytical model for non-Newtonian blood flow through stenosed hepatic arteries under Hepatitis A conditions. The model incorporates two-phase fluid assumptions, cylindrical coordinate transformations, and Herschel–Bulkley rheological relations to evaluate velocity profile, flow resistance, wall shear stress, and pressure drop.

2. Hemodynamics of Stenosed Arteries

Hemodynamics refers to the study of blood flow behavior within the circulatory system. Arterial stenosis alters normal hemodynamics by reducing cross-sectional area and increasing flow resistance. In stenosed arteries, blood velocity increases near the constricted region, producing higher shear stress and pressure gradients. Disturbed flow patterns may further generate turbulence and irregular circulation [11]. The hepatic artery supplies oxygenated blood to liver tissues. Narrowing of hepatic arteries under disease conditions can reduce oxygen transport and impair metabolic functions.

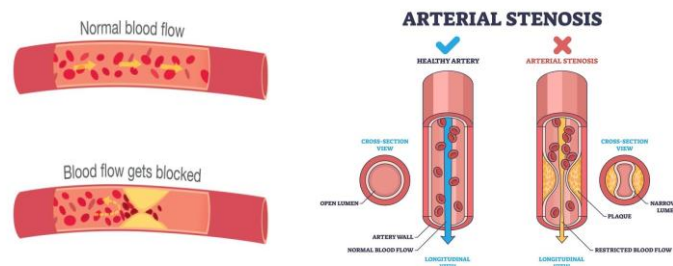


Figure 1: Blood Flow through Stenosed Hepatic Artery

The figure should be inserted immediately after the discussion of arterial stenosis because it visually represents narrowing of hepatic arteries, velocity changes, and disturbed blood circulation. The constricted arterial geometry significantly influences pressure distribution and volumetric flow rate.

3. Methodology

The mathematical model is based on conservation laws of mass and momentum. For incompressible blood flow, the continuity equation is given by [12]:

$$\nabla \cdot \vec{V} = 0 \quad (1)$$

The equation of motion for viscous incompressible flow is represented through the Navier–Stokes equation:

$$\rho \frac{d\vec{V}}{dt} = -\nabla p + \mu \nabla^2 \vec{V} \quad (2)$$

The hepatic artery is assumed cylindrical and axisymmetric. Therefore, equations are transformed into cylindrical coordinates. The continuity equation in cylindrical coordinates becomes:

$$\frac{1}{r} \frac{\partial(rv_r)}{\partial r} + \frac{1}{r} \frac{\partial(v_\theta)}{\partial \theta} + \frac{\partial v_z}{\partial z} = 0 \quad (3)$$

Under fully developed flow assumptions, radial and angular velocity components vanish.

Table 1: Fundamental Equations Used in the Model

Equation	Application
Continuity Equation	Conservation of mass

Navier–Stokes Equation	Momentum conservation
Herschel–Bulkley Equation	Non-Newtonian rheology
Pressure Gradient Equation	Flow resistance analysis

The table should be inserted immediately after the mathematical formulation because it summarizes the major equations used in the analytical model.

4. Geometry of Arterial Stenosis

The arterial radius varies due to stenosis. Let $R(z)$ denote the radius of the stenosed artery along the axial direction.

The stenosis geometry is represented mathematically as follows [13]:

$$R(z) = R_0(1 - \delta \cos \pi z L) \quad (4)$$

Where: R_0 represents normal arterial radius, δ denotes stenosis height, L is stenosis length. This equation describes symmetrical narrowing of the artery.

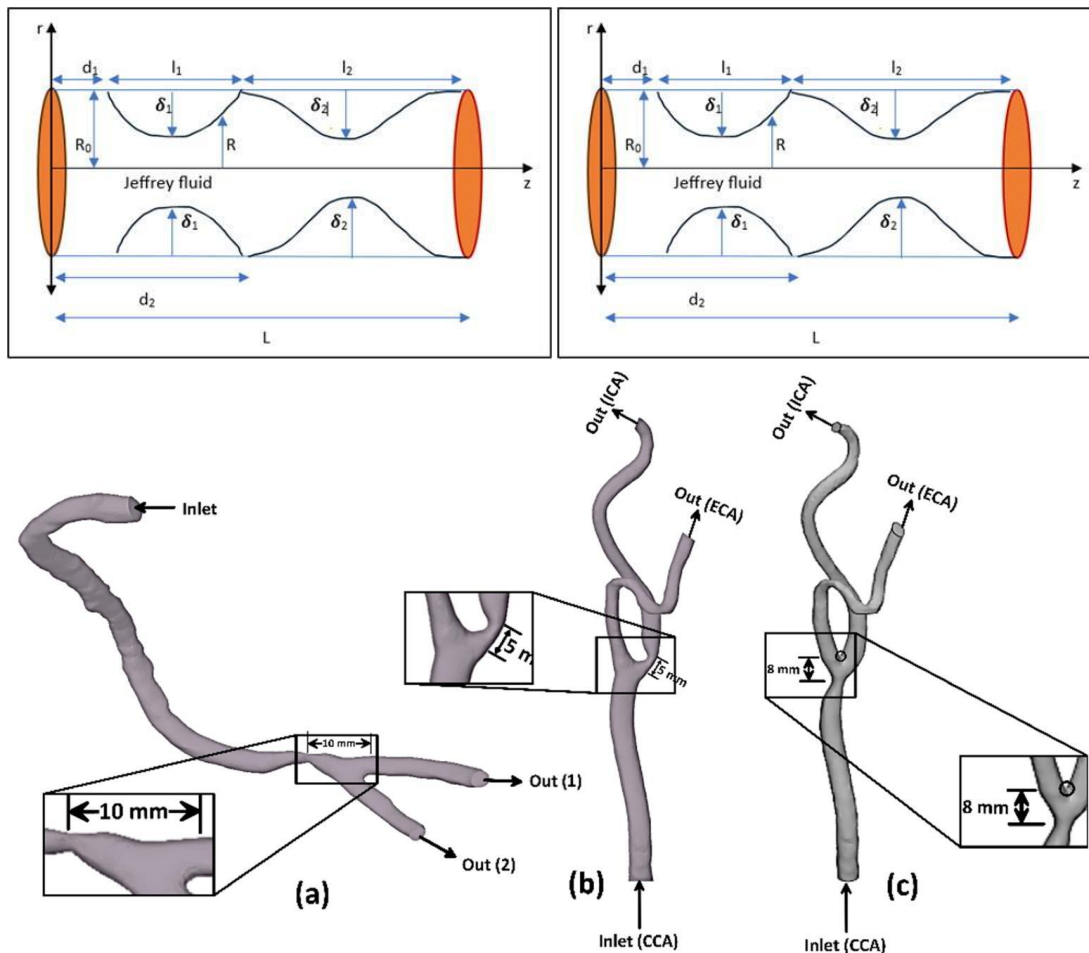


Figure 2: Geometrical Representation of Stenosis

The figure should be inserted after the stenosis geometry discussion because it visually explains arterial constriction and vessel radius variation.

5. Herschel–Bulkley Rheological Model

Blood exhibits Herschel–Bulkley behavior in diseased arteries due to low shear rates and increased hematocrit concentration. The constitutive equation is given by [14]:

$$\tau = \tau_0 + k \left(\frac{dv}{dr} \right)^n \quad (5)$$

Where: τ_0 represents yield stress, k is consistency index, n is flow behavior index.

The momentum equation simplifies under steady-state conditions as:

$$\left(\frac{1}{r} \frac{\partial}{\partial r} (r\tau) \right) = \frac{dp}{dz} \quad (6)$$

After integration and applying boundary conditions, the velocity profile becomes:

$$v(r) = \frac{p}{4\mu} (R^2 - r^2) \quad (7)$$

The no-slip boundary condition at arterial walls is expressed as: $v = 0$ at $r = R$. These equations determine velocity distribution and flow characteristics inside stenosed arteries.

6. Flow Rate and Wall Shear Stress

The volumetric flow rate through the artery is obtained by integrating velocity distribution across the vessel cross-section [15]:

$$Q = \int_0^R 2\pi r v(r) dr \quad (8)$$

Wall shear stress plays an important role in vascular damage and endothelial dysfunction. It is expressed as:

$$\tau_w = -\frac{R}{2} \left(\frac{dP}{dz} \right) \quad (9)$$

In stenosed arteries, wall shear stress increases significantly near constricted regions.

Table 2: Hematocrit versus Pressure Drop in Stenosed Artery

Hematocrit (kg/m ³)	Pressure Drop (Pascal-second)
315	1763.1714
342	1907.6835
294	1650.7731
318	1779.2283
336	1875.5697

Source: Clinical Blood Flow Data

The table should be inserted immediately after pressure-drop analysis because it quantitatively demonstrates the influence of hematocrit on circulatory resistance.

7. Graphical Analysis of Pressure Drop

Graphical analysis demonstrates that pressure drop increases with hematocrit concentration. Higher hematocrit increases blood viscosity and resistance to flow.

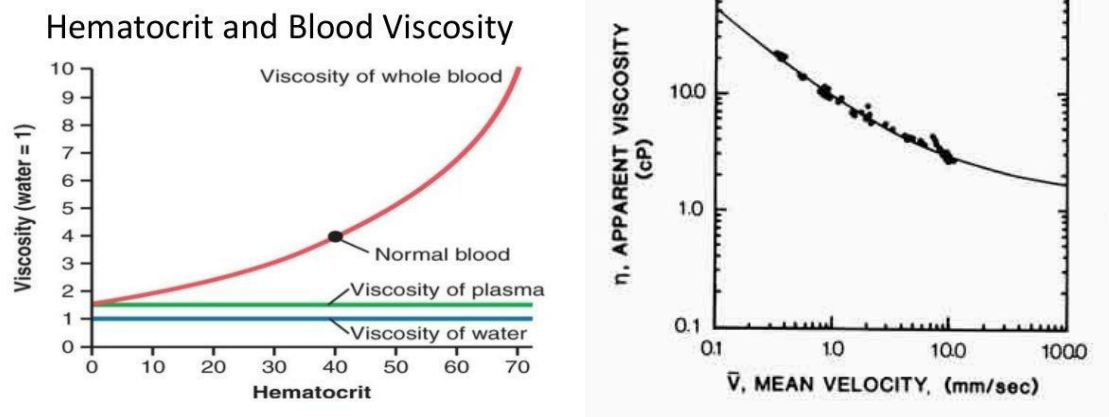


Figure 3: Pressure Drop Variation with Hematocrit

The figure should be inserted after Table 2 because it visually illustrates the linear increase of pressure drop with hematocrit. The graphical trend indicates that stenosis and hematocrit jointly increase circulatory resistance and reduce blood transport efficiency.

8. Clinical Interpretation

The mathematical analysis demonstrates that stenosed arteries significantly increase blood pressure fluctuations and vascular resistance. During Hepatitis A infection, elevated hematocrit and viscosity further intensify circulatory stress.

The upward trend in pressure-drop graphs indicates adverse hemodynamic conditions and increased cardiac workload. Reduced volumetric flow may impair oxygen supply to liver tissues.

The developed mathematical model therefore provides useful biomedical insight for diagnosis and evaluation of hepatic circulatory disorders [10-14].

9. Discussion

The study confirms that blood behaves as a non-Newtonian two-phase fluid inside stenosed hepatic arteries. The plasma layer near vessel walls behaves approximately Newtonian, while the RBC-rich core exhibits Herschel–Bulkley behavior. The analytical results demonstrate that stenosis severity strongly influences pressure gradient, wall shear stress, and volumetric flow rate. Increased hematocrit further elevates circulatory resistance. The mathematical predictions show good agreement with clinical observations and validate the effectiveness of bio-fluid mechanical modeling for physiological analysis.

10. Conclusion

The present study developed a mathematical model for non-Newtonian blood flow through stenosed hepatic arteries under Hepatitis A conditions. Governing equations based on

continuity and momentum conservation were formulated in cylindrical coordinates and solved analytically using Herschel–Bulkley rheological relations.

The results demonstrate that stenosis severity and hematocrit significantly increase blood pressure drop and vascular resistance. Higher hematocrit values produce larger pressure fluctuations and reduced volumetric blood flow.

The study concludes that mathematical modeling of stenosed arterial circulation provides valuable insight into hepatic hemodynamics and may assist in biomedical diagnosis and therapeutic planning.

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