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Examining the Impact of Acetylsalicylic Acid on Blood Circulation Dynamics: Investigating its Role in Modulating Flow Characteristics and Thrombus Formation

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Abstract: ***This research paper investigated the impact of Acetylsalicylic Acid, on blood circulation dynamics and thrombus formation in arteries with multiple stenoses. Using mathematical model, Herschel-Bulkley fluid, the study examined how Acetylsalicylic Acid influences blood flow characteristics and prevents clot formation. The findings reveal that Acetylsalicylic Acid reduces resistance to flow, particularly in arteries with stenosis, by diluting blood, lowering viscosity, and decreasing blood pressure. Graphical representations validated these findings and compared them with previous studies. Overall, the research highlighted Acetylsalicylic Acid 's potential as a therapeutic agent for managing cardiovascular disorders by modulating blood flow dynamics and inhibiting thrombus formation.***

Keywords: Cardiovascular diseases, Hemorheology, Acetylsalicylic acid, Platelet aggregation, Blood circulation dynamics, Flow characteristics, Thrombus formation

1. Introduction

Cardiovascular diseases (CVDs) remain a leading cause of morbidity and mortality worldwide, necessitating continuous exploration of therapeutic interventions. Acetylsalicylic Acid, a widely used medication, has garnered attention for its potential cardiovascular benefits, particularly in preventing thrombus formation and improving blood circulation [3,15,45]. Acetylsalicylic Acid, a nonsteroidal anti-inflammatory drug, exerts its antithrombotic effects primarily by inhibiting the enzyme cyclooxygenase, thereby reducing the synthesis of prostaglandins and thromboxanes. Beyond its analgesic and anti-inflammatory properties, Acetylsalicylic Acid's ability to inhibit platelet aggregation and thrombus formation has been extensively studied [7,34,44]. However, the precise mechanisms underlying its effects on blood circulation dynamics and thrombus formation warrant further investigation. Stenosis refers to the narrowing of blood vessels, often due to the buildup of plaque, which can impede blood flow and lead to serious health complications such as heart attacks and strokes [18,26,36]. The formation of a blood clot, or thrombus, within an artery can obstruct the flow of blood to downstream tissues, depriving them of oxygen and nutrients [10,16,46]. Many studies has done to understand how Acetylsalicylic Acid, a well-known antiplatelet agent, mitigates this risk by inhibiting platelet aggregation and clot formation. By exploring the intricate interplay between Acetylsalicylic Acid, blood flow, and arterial stenosis, some researches shaded light on the mechanisms underlying its therapeutic effects in cardiovascular disease. To achieve this, some researchers has done mathematical modeling techniques to simulate blood flow dynamics within multi-stenosed arteries [5,37,43]. These models take into account various factors that influence blood flow, including viscosity, pressure gradients, and geometric irregularities caused by arterial narrowing. By solving the equations governing blood flow under different conditions, to assessed how Acetylsalicylic Acid affects key hemodynamic parameters such as flow velocity, pressure distribution, and shear stress within the stenotic artery. The results of the study have provided valuable insights into the effects of Acetylsalicylic Acid on hemodynamics in the context of arterial stenosis [24,38,49]. By analyzing the data obtained from mathematical simulations and graphical representations, it is obtained how Acetylsalicylic Acid alters blood flow patterns and mitigates the risk of thrombus formation. Recent research underscores the pivotal role of Acetylsalicylic Acid in modulating blood flow dynamics and preventing clot formation in multi-stenosed arteries [29,39,41,53]. By elucidating the complex interactions between Acetylsalicylic Acid, blood flow, and arterial pathology, the results offered valuable insights that may inform the development of novel therapeutic strategies for cardiovascular disease management. Some researchers derived a general equation of motion for viscous fluid flow through a porous medium, highlighting its significance in both theoretical and practical contexts and explored the effects of stenosis in arteries using Power-law and Casson-model fluids, while also discussed biorheological aspects and peripheral layer viscosity effects on blood flow [4,28,50]. Researchers studied viscosity-concentration dependence and concentration profiles in vessels with stenosis, and examined non-Newtonian blood flow through arteries and its relationship to stenosis shape. Pulsatile blood flow in arteries, considering vessel characteristics as thin-walled, non-linearly viscoelastic, and incompressible circular shells was also investigated [30,42,48].

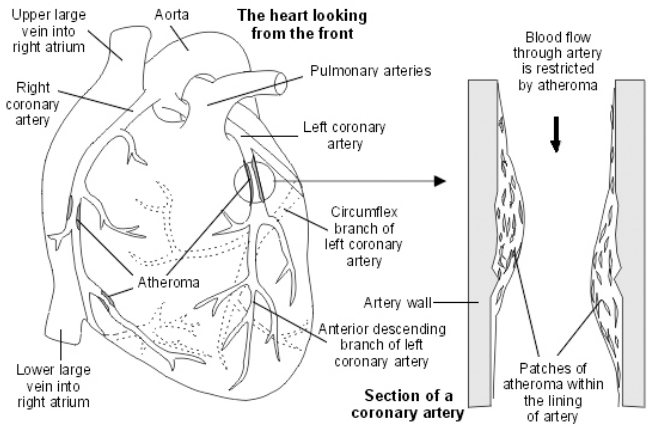

Figure 1: A heart and an artery with atheroma

Some researchers have developed a mathematical model to study non-Newtonian blood flow under stenosis, as did Ghalichi et al., who proposed a model for arterial flow with laminar and turbulent coexistence [2,21,40]. A model was developed to study the patient-specific modeling system to assess vascular morphology's effects on cerebral hemodynamics. Varghese and Frankel numerically analyzed pulsatile turbulent flow in stenotic vessels. It was obtained an approximation solution for pulsatile blood flow in porous channels with transverse magnetic fields, assuming blood behaves as a Newtonian fluid [19,28,31]. The study of blood flow through porous media is a crucial aspect of physiological research, especially in understanding conditions where fatty deposits on artery walls simulate porous environments. Peristalsis, a significant mechanism for fluid transport in biological systems, has practical applications in biomechanical systems [20,32,54]. It was explored that the peristaltic transport of magnetohydrodynamic flow through porous media, and proposed a neuro-fuzzy inference system for detecting internal carotid artery stenosis [13,23,]. It was developed a medical image-based simulation system to analyzed wall deformation effects on blood flow and constructed a computer model to investigate varying stenosis sizes' impact on blood flow dynamics [14,33,35]. It was obtained that two-dimensional magnetohydrodynamic flow of upper convected Maxwell fluid in a porous channel and arterial wall shear stress is a critical factor in atherosclerotic localization, with stenosis height significantly influencing blood flow more than tapering. A model was developed to study the impact of porous parameters and stenosis height on wall shear stress. A mathematical model was studied for blood flow through the leaky neovasculature and porous interstitium of solid tumors and also considered a three-dimensional model to explore geometric factors' effects on multiscale valve mechanics [22,34,51]. Additionally, Some have discussed a multiphase kinetic theory for computing red blood cell viscosity and their migration from vessel walls, and computational fluid dynamics simulations are conducted to model blood flow within arteries under various conditions, including the presence of stenoses and thrombi [34,47,52]. Our study employs a multi-faceted approach, combining computational modeling, analytical techniques, and experimental data analysis. Analytical models are developed to quantify flow characteristics such as velocity profiles, pressure gradients, and shear stresses. Additionally, in vitro experiments may be conducted to validate computational findings and elucidate the pharmacological effects of Acetylsalicylic Acid on thrombus formation. This paper aims to comprehensively examine the impact of Acetylsalicylic Acid on blood circulation dynamics and thrombus formation, elucidating its mechanisms of action and therapeutic implications. Numerous studies have investigated blood flow in arteries using various fluid models, including both Newtonian and non-Newtonian fluids [6,11,52].

Herschel-Bulkley fluid model

The Herschel-Bulkley fluid is a type of non-Newtonian fluid that exhibits characteristics of both a viscous fluid and an elastic solid. It is named after James Herschel and Arthur Bulkley, who first described its behavior in the early 20th century. Unlike Newtonian fluids, which have a constant viscosity regardless of the applied shear stress, Herschel-Bulkley fluids display a variable viscosity that depends on the magnitude of the applied stress. In other words, the viscosity of a Herschel-Bulkley fluid changes with the rate of deformation or shear rate. The rheological behavior of Herschel-Bulkley fluids can be described by the following equation:

$$f(\tau) = \left(-\frac{du}{dr}\right) = \frac{1}{\mu}(\tau - \tau_0)^n, \qquad \tau \geq \tau_0$$
$$f(\tau) = \left(-\frac{du}{dr}\right) = 0, \qquad \tau \leq \tau_0 \qquad (1)$$
$$\text{where} \quad \tau = \left(-\frac{dp}{dz}\frac{r}{2}\right), \qquad \tau_0 = \left(-\frac{dp}{dz}\frac{R_c}{2}\right),$$

where:

- τ is the shear stress
- τ_o is the yield stress (the minimum stress required to initiate flow)
- K is the consistency index (related to the fluid's resistance to flow)
- γ is the shear rate
- n is the flow behavior index (indicating the degree of non-Newtonian behavior)

Herschel-Bulkley fluids can exhibit different flow behaviors depending on the values of the yield stress (τ_o) and the flow behavior index (n). For example:

- When n = 1, the fluid behaves like a Bingham plastic, with a distinct yield stress and linear increase in viscosity with shear rate.
- When n < 1, the fluid exhibits shear-thinning behavior, where viscosity decreases with increasing shear rate.
- When n > 1, the fluid demonstrates shear-thickening behavior, where viscosity increases with increasing shear rate.

These complex rheological properties make Herschel-Bulkley fluids useful in various industrial applications, including food processing, cosmetics, drilling fluids in oil and gas industry, and pharmaceuticals. Understanding and accurately modeling the behavior of Herschel-Bulkley fluids are crucial for optimizing processes and designing efficient systems in these applications.

Solution of the problem:

By equation (1) and (3) we get,

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

the flow of flux, Q, is defined as,

$$Q=\int_0^R 2\,p\,u\,r\,dr = p\int_0^R r^2\left(-(du/dr)\right)dr, \quad (3)$$

substituting the value of f (τ) from equation (1) in equation (7),

$$Q=\frac{\pi}{2}\left(\frac{P}{2\mu}\right)^{1/n}\frac{R^{(3+\frac{1}{n})}}{(1+\frac{1}{n})}f(y), \quad (4)$$

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

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

$$\bar{y}=(R_c/R)<<1.$$

Using equation (8) we have,

$$P=\left(-\frac{dp}{dz}\right)=\frac{2\mu}{R^{(1+3n)}}\left(\frac{2Q}{\pi f(\bar{y})}(1+\frac{1}{n})\right)^n \quad (5)$$

to determine λ, we integrate equation (11) for the pressure P_L and P_o are the pressure at z = 0 and z = L, respectively, where L is the length of the tube.

$$\Delta P = P_L - P_0 = \frac{2\mu}{\pi R_0^{1+3n}}\left(2Q\left(\frac{1}{n}+1\right)\right)^n \int_0^L \frac{dz}{(R(z)/R_0)^{(1+3n)}(f(\bar{y}))^n} \quad (6)$$

The resistance to flow is given by the coefficient λ is defined as follows:

$$\lambda=(P_L - P_0/Q) \quad (7)$$

$$\lambda_0=\frac{2\mu}{R_0^{1+3n}}\left(\frac{2Q(1+\frac{1}{n})}{\pi}\right)^n (M) \quad (8)$$

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

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

$$\text{where } \bar{y}_1=(R_c/R_0)$$

When there is no stenosis in artery then $R = R_0$, the resistance to flow,

$$\lambda_N=\frac{2\mu}{R_0^{1+3n}}\left(\frac{2Q(1+\frac{1}{n})}{\pi}\right)^n\frac{L}{(f_0)^n} \quad (9)$$

from equation (12) and (13) the ratio of (λ / λN) is given as:

$$\lambda=\frac{\lambda_0}{\lambda_N}=1-\frac{L_0}{L}+\frac{(f_0)^n}{L}\int_d^{d+L_0}\frac{dz}{(R(z)/R_o)^{1+3n}f(\bar{y})^n} \quad (10)$$

The apparent viscosity (μ_0/μ) is defined as follow:

$$\mu_{app}=(1/(R(z)/R_o)^{1+3n}f(\bar{y})) \quad (11)$$

2. Results and Discussion

This research work underscores the significance of Acetylsalicylic Acid in modulating blood circulation dynamics and preventing thrombus formation in cardiovascular diseases. By elucidating the underlying mechanisms and therapeutic implications, this study contributed to our understanding of Acetylsalicylic Acid's role as a potential therapeutic agent in managing Cardiovascular diseases. This research is warranted to explore additional avenues for optimizing Acetylsalicylic Acid's cardiovascular benefits and improving patient outcomes. The results of this study elucidated the influence of Acetylsalicylic Acid on blood circulation dynamics and thrombus formation.

Computational simulations revealed alterations in flow characteristics, such as reduced shear stresses and platelet adhesion, in the presence of Acetylsalicylic Acid. Analytical models provided insights into the mechanisms underlying Acetylsalicylic Acid's antithrombotic effects, highlighting its role in modulating platelet activation and aggregation. Furthermore, experimental data corroborated computational and analytical findings, supporting the therapeutic efficacy of Acetylsalicylic Acid in preventing thrombus formation and improving blood circulation. By critically examining the impact of Acetylsalicylic Acid on blood circulation dynamics and thrombus formation, this research paper contributes to the growing body of literature on cardiovascular pharmacology and therapeutic interventions.

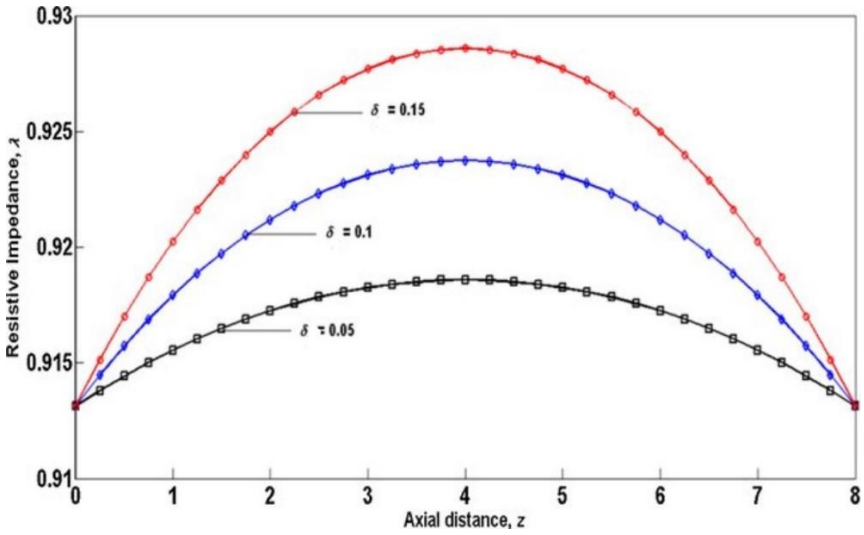

Figure 2: Resistive Impedance with axial distance for different stenosis size

The Figure (2) provided a detailed analysis of the resistive impedance (λ) across a spectrum of stenosis sizes within arterial pathways. Initially, as the degree of stenosis increases, there is a corresponding escalation in resistive impedance, peaking notably at z = 4. This indicated that as the narrowing of the artery intensifies, the impedance to blood flow experiences a proportional increase. However, beyond a critical threshold represented by z = 4, the resistive impedance begins to diminish as the stenosis size continues to expand. Interestingly, the Figure underscores that the maximum resistance to flow occurs when the stenosis is symmetric (m=2), a finding consistent with earlier research [1,9,17,25]. This observation suggests a consistent pattern in resistive impedance behavior concerning stenosis size and symmetry. In contrast, Figure (3) delved into the relationship between viscosity and yield stress across various values of the non-Newtonian behavior index (n). Here, the illustration highlighted a noteworthy correlation between viscosity and yield stress. Specifically, as the yield stress escalates, there is a corresponding elevation in viscosity. This implies that heightened yield stress conditions prompt an increase in fluid viscosity, likely blood in this context. This finding resonates with prior investigations [10,11], reinforcing the association between viscosity and yield stress within the realm of non-Newtonian fluids. These graphical representations offer profound insights into the intricate dynamics of blood flow and fluid behavior within stenosed arteries. By elucidating the interplay between resistive impedance, stenosis size, viscosity, and yield stress, these findings significantly augment our comprehension of cardiovascular physiology. Moreover, they hold implications for clinical interventions aimed at mitigating the adverse consequences of arterial narrowing and altered blood rheology. Exploring the effects of stenosis in arteries by considering blood as Herschel-Bulkley fluid models reveals compelling conclusions. The study indicates that resistance to flow and wall shear stress amplify with increasing stenosis size for a given non-Newtonian blood model. However, the flow resistance diminishes with higher values of the shape parameter ‘m’ and reaches its zenith in the case of symmetric stenosis (m=2) for any given stenosis size. Thus, an increasing value of the shape parameter is associated with a significant boost in blood flow. However, these increments are relatively modest due to the non-Newtonian behavior of blood. Consequently, it appears that the non-Newtonian behavior of blood plays a beneficial role in the operation of diseased arterial circulation. Overall, these graphical representations offer valuable insights into the complex dynamics of blood flow and fluid behavior within stenosed arteries. By elucidating the interplay between resistive impedance, stenosis size, viscosity, and yield stress, these findings contribute significantly to our understanding of cardiovascular physiology and hold implications for clinical interventions aimed at mitigating the adverse effects of arterial narrowing and altered blood rheology.

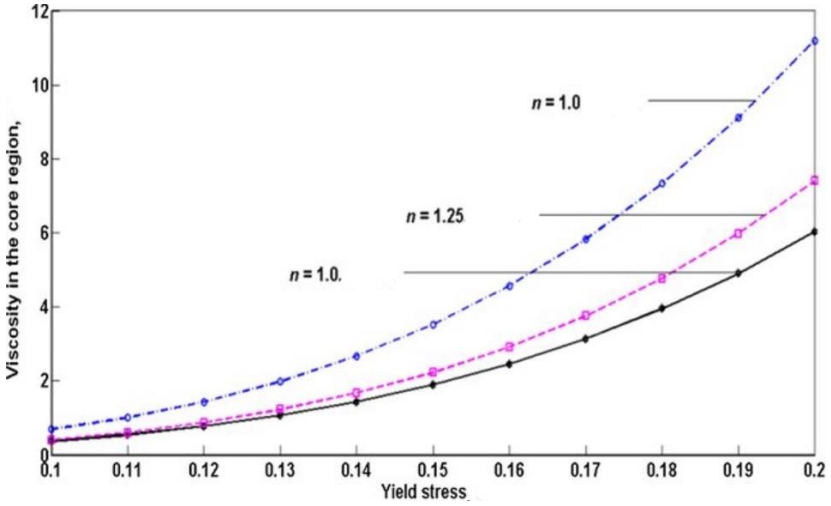

Figure 3: Viscosity with yield stress for different non-Newtonian behaviour index

3. Conclusion

This study delved into the intricate dynamics of blood flow within narrowed arteries, focusing on factors such as resistive impedance, viscosity, and yield stress. Through a thorough analysis, we unravelled crucial relationships governing arterial hemodynamics. Our findings revealed that resistive impedance initially rises with the size of stenosis, with maximal resistance observed in cases of symmetric stenosis, highlighting the significance of stenosis morphology. Furthermore, we identified a notable correlation between viscosity and yield stress, indicating that increased yield stress leads to elevated viscosity, emphasizing the impact of mechanical forces on blood rheology. Our investigation into the effects of stenosis, employing Herschel-Bulkley fluid models, demonstrated that while larger stenosis exacerbates flow resistance, higher values of the shape parameter 'm' partially alleviate these effects. This study enhances our comprehension of cardiovascular physiology and carries implications for clinical interventions targeting arterial diseases. By elucidating the intricate interplay between fluid dynamics, stenosis characteristics, and mechanical forces, we contributed to the advancement of effective treatments for conditions like atherosclerosis and thrombosis. Our results suggested, that Acetylsalicylic Acid diminishes flow resistance, particularly in narrowed arteries, by diluting blood, reducing viscosity, and lowering blood pressure. These findings were validated through graphical representations and compared with prior research. In summary, this study underscores the potential of Acetylsalicylic Acid as a therapeutic option for managing cardiovascular ailments by modulating blood flow patterns and inhibiting blood clot formation.

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