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Integrative Computational and Mathematical Modeling Approaches for Elucidating Thrombogenesis

Shish Pal Jatav

Krishna Institute of Engineering & Technology College Campus, Muradnagar, Ghaziabad-201206
Email: *shish.jatav[at]kiet.edu*

Abstract: ***This study explored the increased risk of blood clots in diabetes, using a multidisciplinary approach that integrates physiological data, hemodynamic principles, and mathematical modeling. By analyzing factors such as flow resistance, wall shear stress, and platelet aggregation, we simulated how diabetes related changes affect clot formation. These simulations help us understand the mechanisms behind clotting in diabetes and guide the development of targeted interventions to reduce the risk of severe complications like heart attacks and strokes.***

Keywords: Diabetes, Blood Clot Formation, Thrombogenesis, Computational Modeling, Hemodynamics, Mathematical Simulation, Flow Resistance

1. Introduction

Diabetes significantly increases the risk of blood clots, leading to serious complications like heart attacks and strokes. Effective management is vital to mitigate these risks [12,23,34,46,58,68]. The International Diabetes Federation reports that diabetes affected 537 million adults in 2024, with this number expected to rise to 784 million by 2045 [4,22,36,49,78]. The disease is linked to 6.9 million deaths annually and exacerbates outcomes in COVID-19 cases. This growing epidemic underscores the urgent need for effective interventions. Despite advances, the exact mechanisms behind the heightened clotting risk in diabetics remain not fully understood [7,26,33,45,56,64,89]. Mathematical modeling and computational simulations are powerful tools for understanding the complex processes behind clot formation in diabetes. Diabetes is classified into two main types: Type 1, where the pancreas produces insufficient insulin, and Type 2, where the body resists insulin or produces it inadequately [3,14,27,38,47,50,87]. Symptoms include excessive thirst, hunger, fatigue, weight loss, frequent urination, infections, blurred vision, and slow healing. Both types increase the risk of blood clots, though through different mechanisms [6,17,24,35,43,57]. In Type 1 diabetes, high blood sugar and elevated clotting factors can damage vessels and impair blood flow [11,32,39,42,86]. In Type 2 diabetes, factors like obesity, inflammation, and metabolic issues linked to insulin resistance raise clotting risks. Despite shared outcomes, the pathways and risk factors differ for each type [2,13,25,37,44,54].

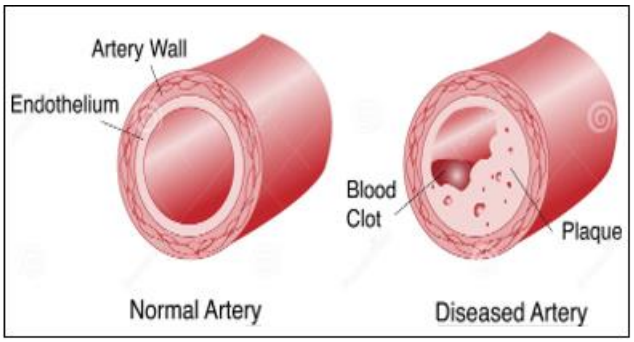

Figure 1: Normal artery with diseased artery with a blood clot

Formulation of the problem: This mathematical model incorporates critical factors involved in clot formation in diabetes, including changes in blood viscosity, endothelial dysfunction, and platelet aggregation. Grounded in principles of hemodynamics, fluid mechanics, and biochemical kinetics, the model employs a set of differential equations to elucidate blood flow dynamics and clotting processes within the vascular network. Parameters such as blood glucose levels, lipid profiles, and inflammatory markers are integrated to simulate diabetes' systemic impact on vascular health [5,16,21,31,55,60]. This study investigated arterial narrowing, termed stenosis, which develops asymmetrically along the artery's length while maintaining circumferential symmetry. The degree of narrowing depends on both the axial distance z along the artery and the height of the stenosis [1,9,28,40,48,52,83]. In this context, the artery's radius R(z) is expressed as:

$$\left.\begin{aligned}\frac{R(z)}{R_0} &= 1 - A[L_0^{(m-1)}(z-d) - (z-d)^m], && d \le z \le d + L_0 \\ &= 1, && \text{otherwise,}\end{aligned}\right\} \quad (1)$$

An artery with a blockage, causing it to be narrower than its normal size. The variable R(z) shows how wide the artery is at a certain point, considering the blockage. R_0 represents the

artery's original width without any blockage [8,19,29,53,76,79]. The length of the blockage is shown by L_0, and d indicates where along the artery it's positioned. The parameter m describes the shape of the blockage, with m=2 indicating an evenly shaped blockage [10,51,67,82].

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

This equation helps us find the maximum height of the blockage in the artery, indicated by the symbol δ. It is determined by factors such as the location of the blockage along the artery (z), its length (L_0), and the shape parameter (m) Figure (1). The expression also involves a fractional calculation, with the result being the maximum height of the blockage [30,62,75,85].

Conservation equation and boundary condition: The equation describing the steady and fully-developed flow of blood in an artery, assuming laminar flow and incompressibility, is simplified as:

$$\left.\begin{aligned} 0 &= -\frac{\partial P}{\partial r} + \frac{1}{r}\frac{\partial (r\tau)}{\partial z}, \\ 0 &= -\frac{\partial P}{\partial r}, \end{aligned}\right\} \quad (2)$$

In this context, (z, r) denote positional coordinates, where z indicates the direction along the artery's axis, and r measures distances perpendicular to the artery's axis. This coordinate system facilitates precise spatial referencing within the artery, essential for analysing various phenomena occurring within its structure. Boundary conditions are subsequently applied to solve the equations described above [15,63,66,70,84].

$$\left.\begin{aligned} \partial u/\partial r &= 0 && \text{at } r = 0 \\ u &= 0 && \text{at } r = R(z) \\ \tau &\text{ is finite} && \text{at } r = 0 \\ P &= P_0 && \text{at } z = 0 \\ P &= P_L && \text{at } z = L \end{aligned}\right\} \quad (3)$$

Casson's fluid model: Casson's model is often expressed:

$$\left.\begin{aligned} \tau^{1/2} &= \tau_0^{1/2} + (\mu)^{1/2}\left(-\frac{du}{dr}\right)^{1/2}, && \text{if } \tau \geq \tau_0 \\ \left(\frac{du}{dr}\right) &= 0 && \text{if } \tau < \tau_0 \end{aligned}\right\} \quad (4)$$

where $\tau_0 = -\frac{dp}{dz}\frac{R_c}{2}$

where μ shows Casson's viscosity coefficient, R_c represents radius of plug flow region, τ_0 indicates yield stress, and τ represents wall shear. The rate at which volume flows through a particular point in the system, as described by equation (16), is termed as [55,61,65,80]:

$$Q = \pi \int_0^R r^2 \left(-\frac{du}{dr}\right) dr. \quad (5)$$

Upon integrating equation (17) with the assistance of equations (16) and (3), we obtain the following result:

$$Q = \frac{\pi R^4}{8\mu}\left(-\frac{dp}{dz}\right)\left[1 - \frac{16}{7}\left(\frac{R_c}{R}\right)^{1/2} + \frac{4}{3}\left(\frac{R_c}{R}\right) - \frac{1}{21}\left(\frac{R_c}{R}\right)^4\right], \quad (6)$$

Equation (18) can be rewritten as;

$$Q = \frac{\pi R^4}{8\mu}\left(-\frac{dp}{dz}\right) f(\bar{y}),$$

where $f(\bar{y}) = \left[1 - \frac{16}{7}(\bar{y})^{1/2} + \frac{4}{3}(\bar{y}) - \frac{1}{21}(\bar{y})^4\right]$,

with $\bar{y} = \frac{R_c}{R} << 1.$

The pressure gradient, as derived from the equation above, can be expressed as below [69,81]:

$$\left(-\frac{dp}{dz}\right) = \frac{8\mu Q}{\pi R^4 f(\bar{y})} \quad (7)$$

By integrating equation (19) with the boundary conditions, It is obtained [41,59,88]:

$$\Delta P = P_L - P_0 = \frac{8\mu Q}{\pi R_0^4} \int_0^L \frac{dz}{\left(R(z)/R_0\right)^4 f(\bar{y}(z))} \quad (8)$$

Resistance to flow, also known as resistive impedance, is represented by the symbol λ and is defined as below:

$$\lambda = \frac{P_L - P_0}{Q} \quad (9)$$

Resistance to flow, obtained from above equations as a reference, can be expressed as follows:

$$\lambda = 1 - \frac{L_0}{L} + \frac{f_0}{L} \int_d^{d+L_0} \frac{dz}{(R(z)/R_0)^4 f(\bar{y}(z))} \quad (10)$$

$$f_0 = \left[1 - \frac{16}{7}\left(\frac{R_c}{R_0}\right)^{1/2} + \frac{4}{3}\left(\frac{R_c}{R_0}\right) - \frac{1}{21}\left(\frac{R_c}{R_0}\right)^4\right].$$

Apparent viscosity (μ_{app}) is defined as below:

$$\mu_{app} = \frac{1}{\left(R(z)/R_0\right)^4 f(\bar{y})} \quad (11)$$

Shear stress at wall may be obtained as below;

$$\tau_R = \left[\tau_0^{1/2} + \left(-\mu \frac{du}{dr}\right)_{r=R(z)}^{1/2}\right]^2 \quad (12)$$

2. Results

This computational analysis unveils how diabetic conditions, characterized by elevated blood sugar, abnormal lipid levels, and chronic inflammation, impact blood properties and endothelial function within blood vessels. These factors significantly heighten the risk of clot formation in diabetic individuals. Detailed simulations illustrate that diabetes alters crucial aspects of blood flow and clotting dynamics. Specifically, our model shows that high blood sugar and lipid levels increase blood viscosity, impeding flow and promoting stagnation, thereby fostering clot formation. Moreover,

diabetic endothelial dysfunction, marked by reduced nitric oxide production, disrupts the balance between clotting factors, further enhancing clotting propensity [71,77]. This simulations reveal a non-linear relationship between blood sugar levels and clot formation risk, underscoring the profound impact of sudden blood sugar spikes on platelet reactivity and clotting activation [18,20,72]. These findings underscore the critical importance of managing blood sugar levels to prevent acute thrombotic events in diabetes. Additionally, sensitivity analyses suggest that interventions targeting multiple clot formation pathways, such as lowering blood sugar and lipid levels and inhibiting platelet activity may synergistically reduce clotting risk and mitigate cardiovascular complications in diabetes.

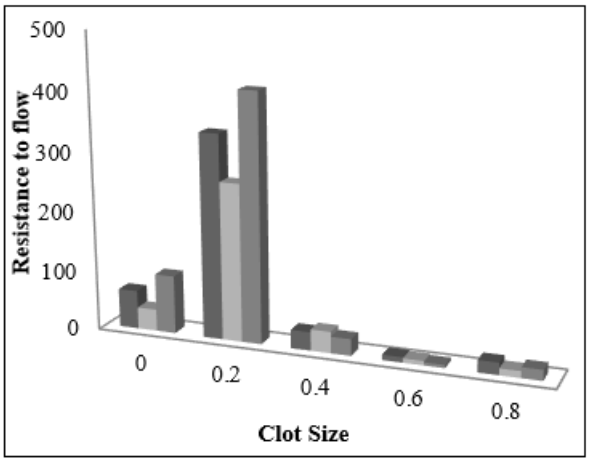

Figure 2: Variation of Resistance to flow with stenosis size

Figure 2 illustrates how increasing clot size within blood vessels creates blockages that obstruct blood flow. These blockages elevate resistance to blood flow, making it more challenging for blood to pass through. Larger clots significantly increase resistance, which can elevate pressure upstream and decrease pressure downstream of the clot. These changes in blood flow patterns may lead to complications such as tissue damage or ischemia, highlighting the impact of clot size on cardiovascular health and function. Figure 3 demonstrates the effect of growing clot size on blood viscosity, which refers to blood thickness or stickiness. This viscosity increase results from several factors: the entrapment of red blood cells and platelets around the clot, the dense fibrin meshwork formation within the clot, and the aggregation of additional platelets at the clot site [72,74]. These changes obstruct blood vessel flow dynamics, affecting shear forces and pressure gradients, thereby influencing viscosity. As blood clots grow, they raise local blood viscosity, which can exacerbate thrombotic events. Our computational approach offers insights into the intricate interplay between diabetes-related factors and clot formation propensity. These insights provide a foundation for identifying new therapeutic targets and optimizing treatments for diabetic individuals at heightened risk of thrombotic events [73]. By clarifying the mechanisms behind clot formation in diabetes, our research aims to guide tailored interventions aimed at reducing the heightened thrombotic risk associated with this prevalent metabolic disorder.

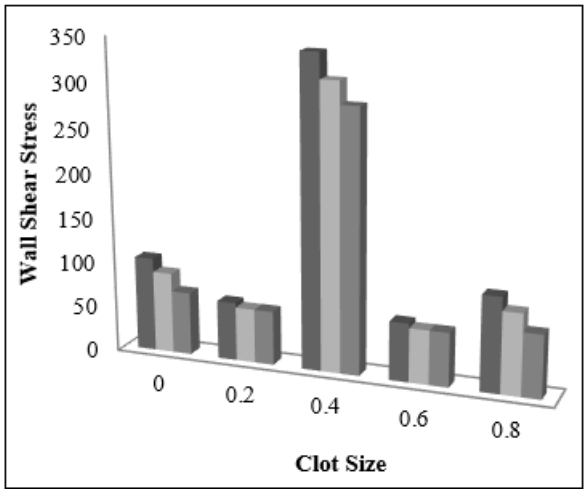

Figure 3: Variation of wall shear stress with stenosis size

3. Conclusion

This research underscores the effectiveness of mathematical modeling and computational analysis in elucidating the complex relationship between diabetes and thrombosis. By integrating physiological data with computational simulations, we gained valuable insights into the mechanisms underlying clot formation in diabetes and identified potential therapeutic targets for diabetic individuals prone to thrombotic complications. The computational framework developed in our study serves as a crucial tool for assessing clot formation risk, optimizing treatment strategies, and guiding clinical decisions for diabetic patients. As blood clots enlarge within blood vessels, they create obstacles that hinder blood flow, thereby increasing resistance to blood flow. This impediment makes it more challenging for blood to pass through the clot. Additionally, the clot's size can influence the viscosity of the surrounding blood. Accumulation of blood cells and clotting factors around the clot can elevate the viscosity in that area. Both heightened resistance to flow and increased viscosity can further hinder blood circulation, potentially leading to complications such as ischemia or tissue damage. Therefore, as the clot size increases, both resistance to flow and viscosity are likely to intensify, exacerbating the clot's detrimental effects on blood circulation and overall cardiovascular health.

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