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FLUID-STRUCTURE ANALYSIS OF BLOOD FLOW IN AN OCCLUDED DEFORMABLE ARTERY

Deore Mansi[1,2]*, Raja Jayendiran[2], Venkat Perumal[2], V M Phalle[1]

[1]Veermata Jijabai Technological Institute (VJTI), Mumbai, India, 400019

[2]Stryker Global Technology Center, Gurgaon, India, 122102

* Corresponding Author (e-mail : mddeore_m22@me.vjti.ac.in)

ABSTRACT

Blocked arteries, due to the build-up of fibrous and fatty material inside the arteries is the underlying condition that causes coronary heart disease and other circulatory diseases. This can ultimately bring on symptoms such as chest pain (angina) or lead to life-threatening conditions such as a heart attack or stroke. It is important to have a detailed understanding of the complex interactions that occur between blood flow and vessel deformations under this medical condition. The objective of this study is to provide a more realistic description of blocked artery physiology by presenting a unique approach that includes fluid-structure interaction (FSI) analysis. Unlike conventional techniques, which generally regard either the clot or the vessel as rigid entities or focus primarily on fluid flow or structural mechanics, our approach overcomes these gaps by considering both blood flow and clogged-vascular deformability. To represent the intricacies of hemodynamic interactions and soft tissue mechanics, we use an idealized cylindrical vascular model. One of the main contributions is a thorough modeling framework that combines structural mechanics and fluid dynamics to allow for a dynamic evaluation of blocked-vessel interactions with the blood. Examining the patterns of stress, strain, and deformation in vessel walls offers valuable information about how occluded vessels respond mechanically to changes in blood flow. This research holds significant implications for disease modeling, clinical diagnostics, and therapeutic interventions. Our goal is to contribute to the development of more effective treatment options and patient-specific treatments by understanding the bio tribological mechanisms that underlie the physiology.

KEYWORDS: Friction, Fluid-structure interaction, Blood clot, Computational Fluid Dynamics, Artery

1. INTRODUCTION

Arteries play a pivotal role in the circulatory system by transporting oxygen-rich blood from the heart to tissues and organs. Characterized by distinct layers including the tunica intima, tunica media, and tunica externa, arteries exhibit unique structural features that contribute to their function. The tunica intima, composed of endothelial cells and connective tissue, provides a smooth surface for blood flow. In contrast, the tunica media, consisting of smooth muscle cells and elastic fibers, regulates blood pressure and flow. Arteries, with their thicker walls compared to veins, are designed to withstand high pressure, ensuring efficient blood distribution. Understanding arterial anatomy and physiology is essential for comprehending blood flow dynamics and vascular function [3][7].

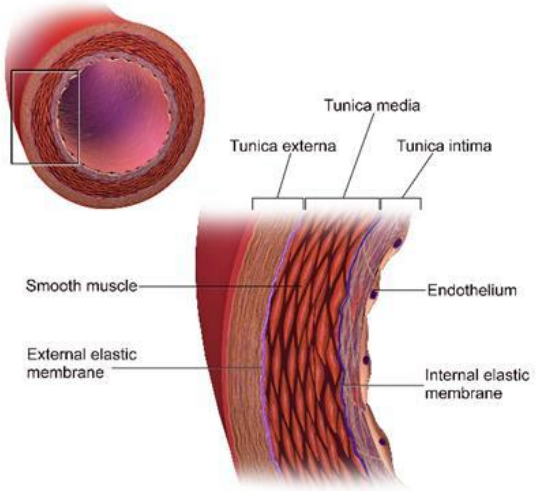


Fig.1. Structure of an artery wall [7]

In the context of this paper, the study of arterial diameters, velocities, and vascular resistance becomes crucial. These parameters influence the hemodynamic behavior within the arterial system, impacting tissue perfusion and metabolic balance. By investigating the intricate details of arterial structure and function, this research aims to elucidate the interplay between fluid dynamics and arterial mechanics in occluded arteries, providing insights into the pathophysiology of vascular diseases and potential strategies for tissue engineering and therapeutic interventions.

Pathological conditions affecting arteries include aneurysms (bulges in artery walls), atherosclerosis (accumulation of plaque inside vessels leading to hardening, narrowing, and blockages), clots (accumulation of blood cells or other materials causing vessel blockages), stenosis (artery narrowing impeding blood flow), vasculitis (vessel inflammation), and genetic or congenital disorders (vessel formation or function issues stemming from genetics or present at birth) [4]. The formation of blood clots can have serious consequences, potentially manifesting as symptoms like chest pain (angina) or progressing to life-threatening conditions such as heart attacks or strokes. Strokes, in particular, can be classified into different types, including ischemic strokes, hemorrhagic strokes, and transient ischemic attacks (TIAs). Ischemic strokes occur when a blood clot obstructs a blood vessel supplying the brain, leading to reduced blood flow and oxygen delivery. Hemorrhagic strokes, on the other hand, result from a ruptured blood vessel in the brain, causing bleeding and pressure on surrounding brain tissue. TIAs, often referred to as "mini-strokes," are temporary blockages that resolve quickly but serve as warning signs for potential major strokes. Each type of stroke requires specific management and treatment approaches to minimize damage and prevent recurrence.

Blood clots play a crucial role in the pathogenesis of ischemic stroke, with changes in clot structure and composition observed in affected patients. In vitro clot structures from plasma samples consistently show dense fibrin networks resistant to fibrinolysis, while ex vivo clot compositions obtained through thrombectomy exhibit variability, with some clots being rich in red blood cells (RBCs) and others in platelets [5][6] (Fig.2.). Challenges in numerical modeling of clots exist due to a lack of comprehensive experimental data, highlighting the need for further in vivo research to understand the impact of clot contraction on stroke pathogenesis. Advanced technologies for imaging and clot retrieval hold promise for generating crucial data for diagnosing, treating, and managing ischemic stroke patients.

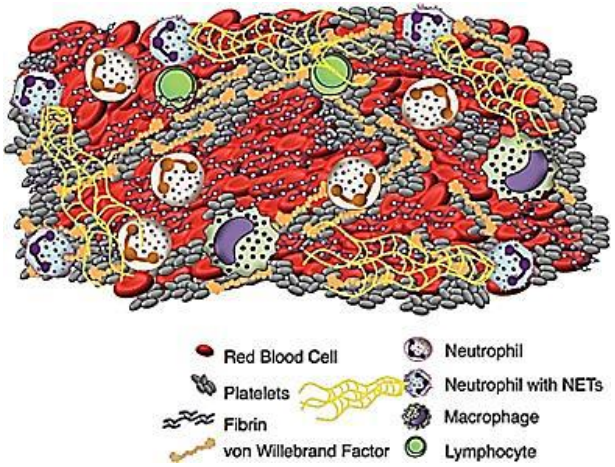


Fig.2. Schematic diagram of the blood clot's constituent parts.
(Note that the ratios of the different parts are not indicative of a particular subtype of blood clot; rather, they are arbitrary.) [6]

Arteries have a wide range of diameters, thicknesses, and lengths, reflecting their various functions and locations. Large arteries, such as the aorta and carotid arteries, have diameters of 2 to 3 centimeters or greater. These arteries typically have thicker walls, ranging from 1 to 2 millimeters, to withstand the high pressures generated by the heart's contractions. Large arteries vary in length from 20 to 30 centimeters or longer, based on where in the body they are located. The brachial and femoral arteries are examples of medium-sized arteries, with diameters between 0.5 and 1 centimeter. Medium-sized arteries have walls that are between 0.5 and 1 millimeters thick, which gives them flexibility and structural support. These arteries can range in length from 30 to 40 centimeters, which helps the body distribute oxygen-rich blood to different parts of the body. Smaller arteries, such as the radial and tibial arteries, have diameters of 2 to 3 millimeters. Smaller arteries typically have a wall thickness of 0.1 to 0.3 millimeters, which allows for flexibility and efficient nutrient exchange. These arteries range in length from a few centimeters to approximately 20 centimeters, depending on their location and function in the circulatory system.

From a mechanical standpoint, vascular tissue exhibits considerable deformability and non-linearity. The mechanical behavior of rubber tubes and rat carotid arteries under physiological conditions was shown to differ significantly. Rubber tubes showed linear elasticity and isotropy, whereas arteries showed nonlinear, anisotropic properties. Remarkably, arteries behaved like rubber tubes at physiological pressure, exhibiting stepwise isotropy in the physiological range as opposed to the global isotropy seen in rubber tubes. Isotropic numerical modeling is favored over anisotropic modeling due to its simplicity, computational efficiency, and generalizability, making it practical when detailed anisotropic data is lacking. Anisotropic modeling, while more accurate, is complex, computationally demanding, and prone to errors due to challenges in data availability and model validation. The trade-off between accuracy and computational feasibility guides the choice between isotropic and anisotropic models, with isotropic models offering a more straightforward approach for certain applications despite potential limitations in capturing directional variations in tissue mechanics.[8]

Fluid Structure Interaction (FSI) is a multidisciplinary field that investigates the dynamic interaction of fluids with structures. This problem exemplifies a Multiphysics approach in which two distinct physics phenomena are analyzed independently and their interaction is being considered. The deformation and response of solid structure is directly influenced by the fluid flow behavior in FSI problems, and vice versa. Numerous scientific and engineering fields, such as aerospace, civil engineering, biomechanics, and environmental studies, frequently use FSI. Based on how the fluid and structure domains are coupled, FSI can be divided into two primary categories: monolithic approaches, where the fluid and structure equations are solved simultaneously in a single system, and partitioned approaches, where the fluid and structure equations are solved separately and coupled at the interface. Modeling FSI involves representing the fluid and solid domains, defining the interface between them, and solving the governing equations for fluid dynamics and structural mechanics. ANSYS supports two types of Fluid-structure Interaction: one-way transfer and two-way transfer. In one-way FSI, CFD findings are used as loads in the Mechanical analysis, but the results of the Mechanical analysis are not fed back into the fluid analysis. The outcomes of the mechanical analysis are fed back as loads into the fluids model in a two-way FSI. One-way transfer is appropriate when the calculated displacements and temperature differentials in the Mechanical application are insignificant enough to influence the fluid analysis, whereas two-way transfer is appropriate when these factors are significant enough to affect the fluid

analysis.

This research is highly relevant to the field of bio-tribology as it explores the friction, wear, and lubrication phenomena within biological systems, particularly focusing on the interactions between blood flow and the vascular walls. The fluid-structure interaction (FSI) framework developed in this study allows for a deeper understanding of how mechanical forces, generated by blood flow, influence the deformation and stress distribution within the arterial walls and clots. This insight is critical for advancing our knowledge of vascular diseases, where the tribological aspects play an important role in the onset and progression of conditions like atherosclerosis and thrombosis.

2. FLUID MODEL

The FSI simulations in ANSYS are based on the insertion of a fluid domain into a solid hollow domain with the same dimensions. The idealized fluid domain for blood is a curved, solid cylindrical domain with a length of 80 mm and a radius of 1.5 mm, which is equivalent to the inner radius of the hollow, cylindrical domain utilized for the artery. (Fig.3.). The blood flow is described as unsteady, laminar, incompressible (volume or density does not vary with pressure), and Newtonian (constant viscosity that does not change, no matter the pressure being applied to the fluid). The inlet and outflow surfaces define the domain's two boundaries.

The modeling of blood flow in a vessel is a challenging fluid-structure interaction (FSI) mathematical problem. Numerous unanswered questions remain in the areas of analytical techniques and numerical scheme development issues. The present study used ANSYS's two-way fluid-structure interaction (FSI) capability to investigate the impact of blood clots on blood flow and arterial structure. The flow is simulated using two alternative approaches: Artery rigid wall and flexible wall assumption. FSI simulations are based on a computational fluid dynamic analysis for the fluid (CFD) coupled to a finite element model (FEM) for the solid provided conditions at the interface of the fluid-solid domain are being enforced.[9]

Pressure outflow of 10 kPa and velocity of 0.57 m/s at inlet is assumed. System coupling dynamic mesh is introduced at fluid wall and clot wall interface. In this case, the blood's viscosity is set to 0.0035 Pa.s. and its density is 1060 kg/m3. Navier- Stokes equations for an incompressible fluid with constant viscosity, which are given in standard textbooks, are used to compute fluid flow in computational fluid dynamics (CFD) [10]. The governing equation for fluid domain is given by continuity equation, Eq. (1) and Navier-stokes or momentum equation, Eq. (2),

$$\nabla \cdot u = 0 \tag{1}$$

$$\rho \frac{Du_i}{Dt} + \nabla p = \rho g + \mu \nabla^2 u, \tag{2}$$

where u is the velocity, p is the pressure, μ is the constant viscosity and ρ is the constant density. The Navier-stokes equation provides information of velocity variation and pressure gradients in numerous flow situations.

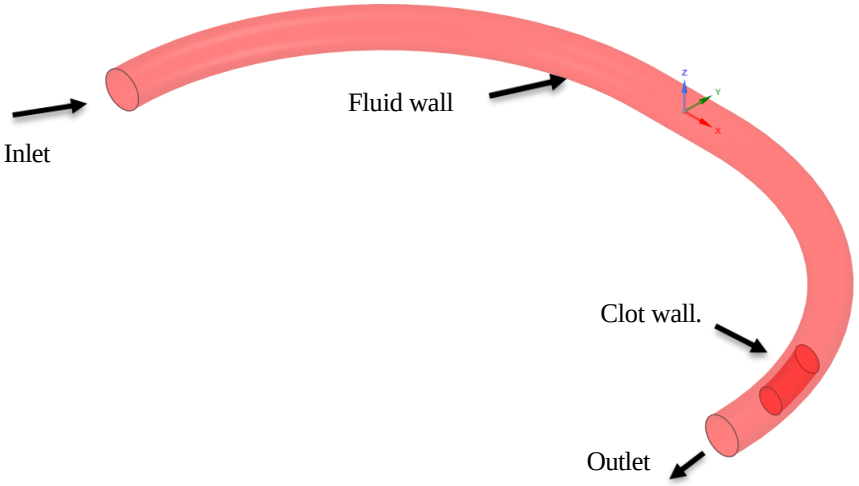


***Fig.3**. Fluid Domain*

3. FINITE ELEMENT MODEL FOR ARTERY AND CLOT

The artery's hollow, cylindrical shape measures 80 mm in length, inner radius of 1.5 mm and 0.1 mm in thickness. The clot is positioned 5 mm from the outlet inside the artery. It has a diameter of 2 mm and a length of 5 mm. In this work the artery wall and clot are modelled by an isotropic linearly elastic material. Selection of the artery and clot material properties is based on literature sources that correspond with the typical properties of blood flow in small arteries (Table 1). Clot is assumed to be slightly stiffer as compared to artery. Several challenges lie in defining mechanical properties for clot.

Table 1. Material properties of human artery and blood clot

Material	Density [kg/m³]	Young's Modulus [MPa]	Poisson's ratio
Artery	1000	1	0.45
Blood Clot	1080	1.7	0.45

Fluid solid interface is assigned at artery inner wall and clot outer surface.

The governing equation for transient dynamic analysis is given by the equation of motion, Eq. (3).

$$[M]\{\ddot{u}\} + [C]\{\dot{u}\} + [K]\{u\} = \{F(t)\}, \quad (3)$$

With damping and inertia effects considered, the equation mentioned above can be used to find the unknown displacements that satisfy the equilibrium at each time step.

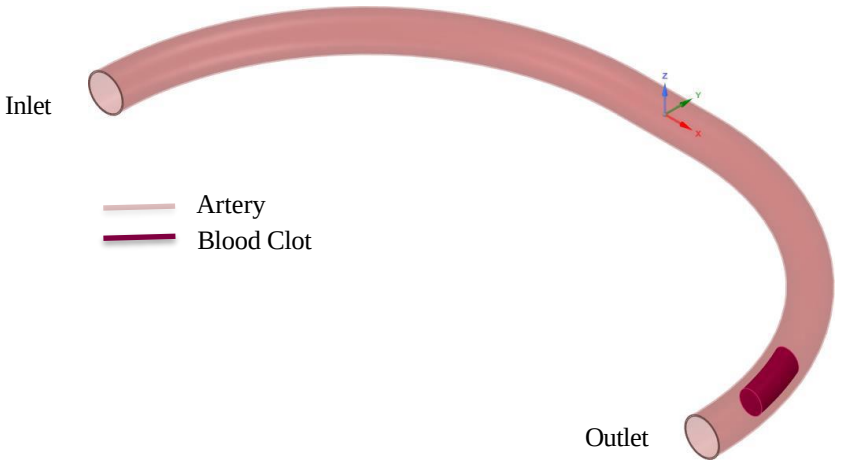
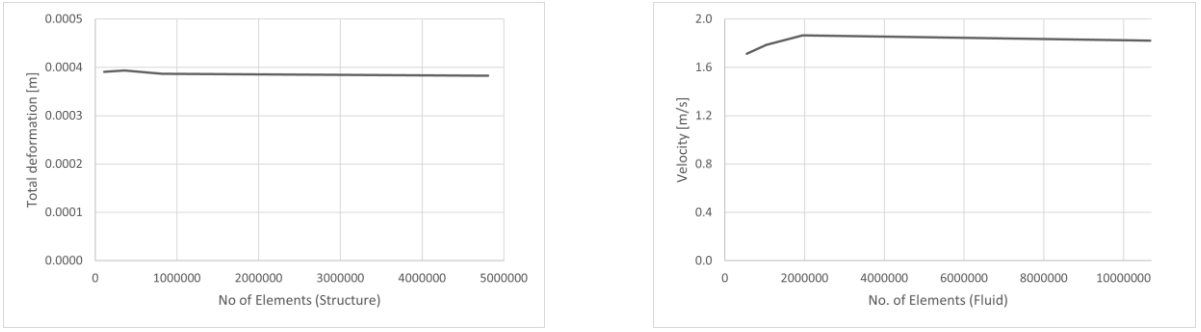


Fig.4. Solid Domain

4. FLUID-STRUCTURE INTERACTION ANALYSIS

The mathematical model is discretized in space and time, with time integration for both the structure and the fluid domain, resulting in an algebraic equation system. In FSI analysis, loads are transferred from fluid to structure at the interface, and vice versa. In this case, the boundary conditions will change, and the mesh must be updated at each step of the analysis. The conservation of mass equation applies to fluids as well, but the general momentum equation cannot be used in transient analysis because the solution domain changes all the time, requiring the grid to be updated to reflect the changed flow boundary.

The solid domain is meshed into 4807738 elements with 22714829 nodes and the fluid domain is discretized into 10665789 elements with 1982521 nodes. A convergence study of artery with clot model revealed that no additional gain was attained with a larger number of elements for the solid or the fluid (Fig.5.).



(a) (b)

Fig. 5. Convergence study Solid domain (a); Fluid domain (b)

The fluid flow has the same characteristics as previously stated in Section 2. At the inlet there is a velocity condition and at the outlet pressure conditions in assumed.

System Coupling is a method used to link independent analyses, facilitating FSI. The interface represents the junction of the outside surface of the fluid domain and inside surface of the artery walls. At the interface, continuity of stress and displacement (i.e., $U_s = U_f$ and $\sigma_s \cdot n = \sigma_f \cdot n$ where 's' and 'f' denote solid and fluid domain respectively) are enforced.

The two-way FSI problems are solved with the Fluid Flow (Fluent) and ANSYS Mechanical solvers. Characteristic durations of the simulations on a 32-core machines are:

- Two-way FSI for artery rigid wall condition: 20 hours 10 mins 42 seconds.
- Two-way FSI for artery flexible wall condition: 7 days 11 hours 14 mins 53 seconds

5. SIMULATION SCHEME

ANSYS employs system coupling scheme to solve strongly coupled fluid-structure-interaction problems (ANSYS 2023R2). In System Coupling analyses, Ansys supports various capabilities, including the transfer of force and displacement data along wall boundaries, utilization of triangular and quadrilateral-faced interface cell types, and full accommodation of local and distributed parallel solver execution. It further enhances the flexibility and effectiveness of System Coupling analyses within Ansys by enabling the use of custom solver input and restart files and sharing convergence data for all equations, including continuity, momentum, and energy, with the System Coupling service during runtime [11].

To create a coupled analysis in Workbench, a system coupling system is added to the Project Schematic along with Transient structural system for Mechanical and Fluid Flow (Fluent) system for CFD. Here system coupling will be used to connect the participants once fluid and solid physics setup is completed.

6. RESULTS

6.1 Structural Integrity of Vessel and Clot

The displacement and stress magnitudes have been analyzed and compared for flexible and rigid artery walls. The maximum deformation for a blood clot is 0.031 mm, while the maximum deformation for a flexible artery wall is 0.38 mm. On the other hand, the maximum deformation for the blood clot is marginally smaller at 0.027 mm when considering the rigid artery wall assumption. (Fig.6 & 7.).

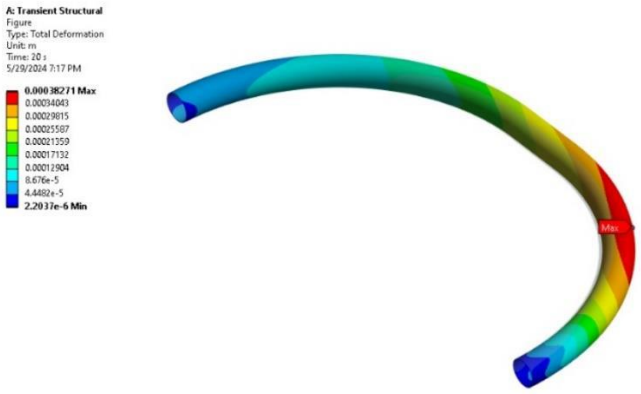
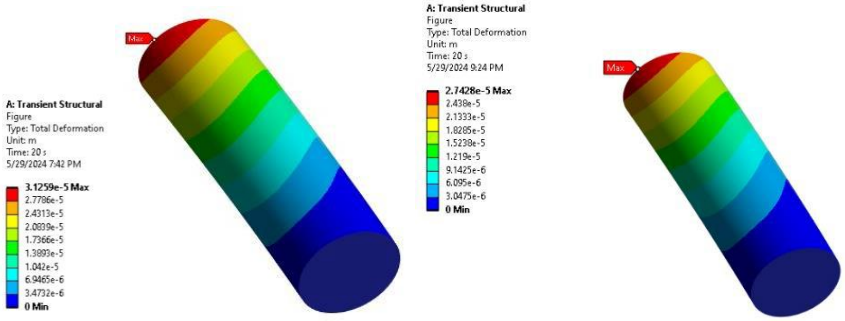


Fig.6. Displacement contour for flexible wall



(a) (b)

Fig.7. Displacement contour for clot when vessel wall is (a) Flexible (b) Rigid

(Fig. 8 & 9) show the stress contour for the flexible and rigid artery wall. The flexible artery wall's von Mises stress has a maximum value of 69.47 kPa. The von Mises stress for the blood clot is 9.31 kPa when the artery wall is flexible, as opposed to 13.18 kPa when the artery wall is rigid. These results show that the clot's stress distribution is significantly influenced by the stiffness of the artery wall, with higher stresses observed in the case of the rigid wall.

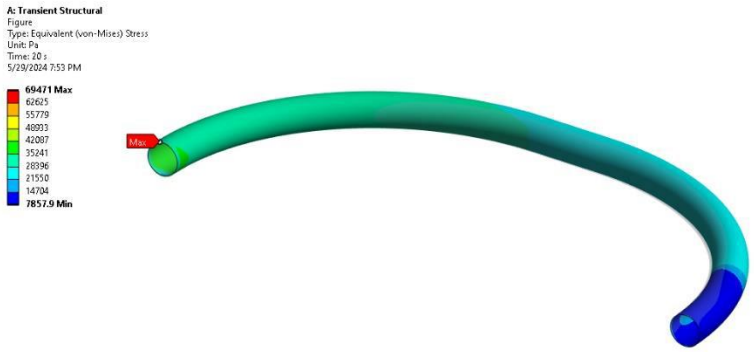
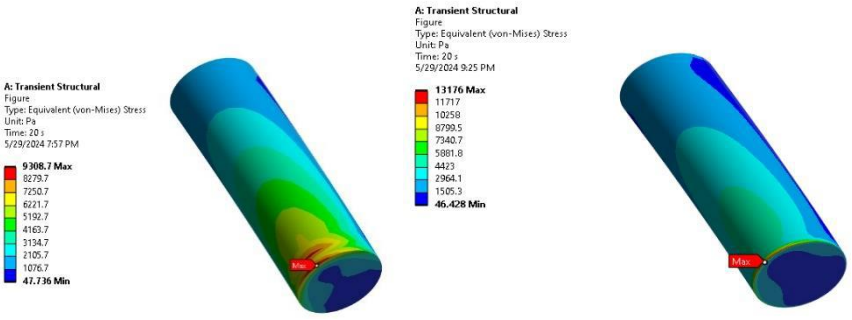


Fig.8. Von mises stress contour for flexible wall



(a) *(b)*

Fig.9. Von mises stress contour for clot (a) flexible wall (b) rigid wall

6.2 Flow Parameters

In this section, we analyze the flow parameters such as pressure distribution, velocity, and wall shear stress (WSS) for both rigid and flexible artery wall conditions. The comparison of these parameters provides insight into how arterial wall flexibility influences the hemodynamics of blood flow, particularly in the presence of a blood clot.

For the flexible artery wall, the pressure distribution value ranges from 9.79 kPa to 12.95 kPa, while for the rigid artery wall, it ranges from 9.71 kPa to 12.67 kPa. In both cases, there is a noticeable decrease in pressure in the vicinity of the clot, suggesting that the clot has a major effect on the local hemodynamics (Fig.10.).

The velocity distribution also shows distinct differences between the two wall conditions. The maximum velocity recorded is 1.86 m/s for the flexible artery wall and 1.58 m/s for the rigid wall. The highest velocities are observed near the clot, suggesting that the presence of the clot accelerates blood flow in its vicinity due to the restricted lumen (Fig.11.).

The wall shear stress (WSS) analysis yields maximum values of 0.189 kPa for the flexible wall and 0.120 kPa for the rigid wall. The higher WSS values near the clot region indicate increased frictional forces acting on the vessel wall in the presence of a clot, which are more pronounced when the wall is flexible. The WSS values remain relatively low throughout the artery, highlighting the clot's localized effect on shear stress distribution (Fig.12.).

6.3 Discussion

The presence of a blood clot in an artery has a significant impact on the endothelial layer and vessel wall integrity because of increased wall shear stress (WSS) and blood velocity. Elevated WSS near the clot increases frictional forces on the endothelial cells, leading to degradation. High blood velocity further amplifies these frictional interactions, causing wear, elongation, and micro-tears in the arterial wall. Over time, these frictional stresses reduce the wall's elasticity, raising the risk of aneurysms and ruptures. High von Mises stress within a blood clot can lead to its fragmentation due to the internal frictional forces exceeding the clot's structural cohesion. These forces act on the clot's surface and interior, causing it to break apart. The fragmented pieces then interact with the blood flow, increasing the overall friction within the vessel. These clot fragments can lodge in smaller vessels and obstruct blood flow.

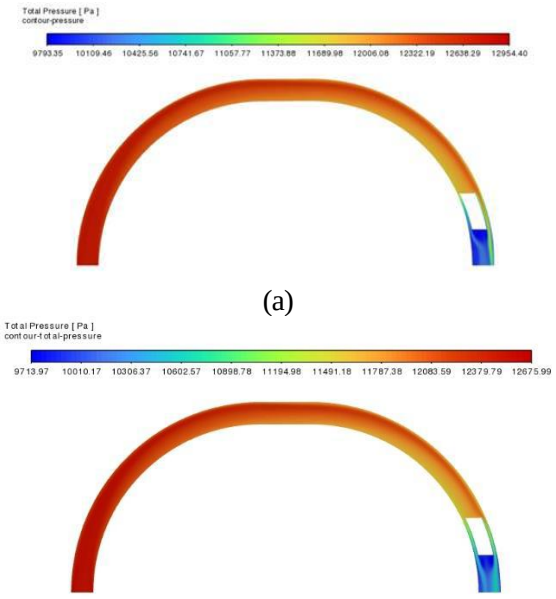


Fig.10. Pressure contour obtained for (a) flexible wall (b) rigid wall.

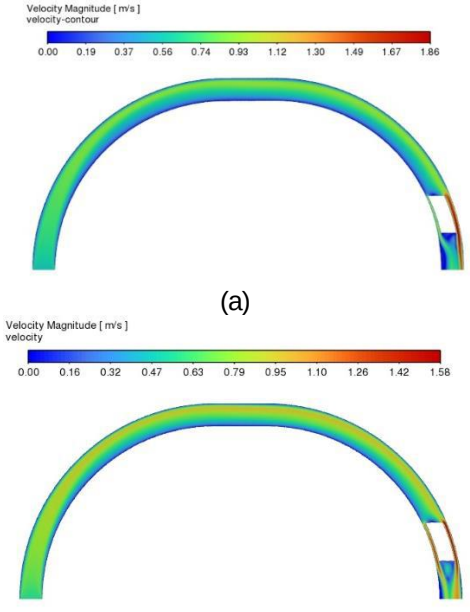


Fig. 11 Velocity contour (cross-sectional view) obtained for (a) flexible wall (b) rigid wall

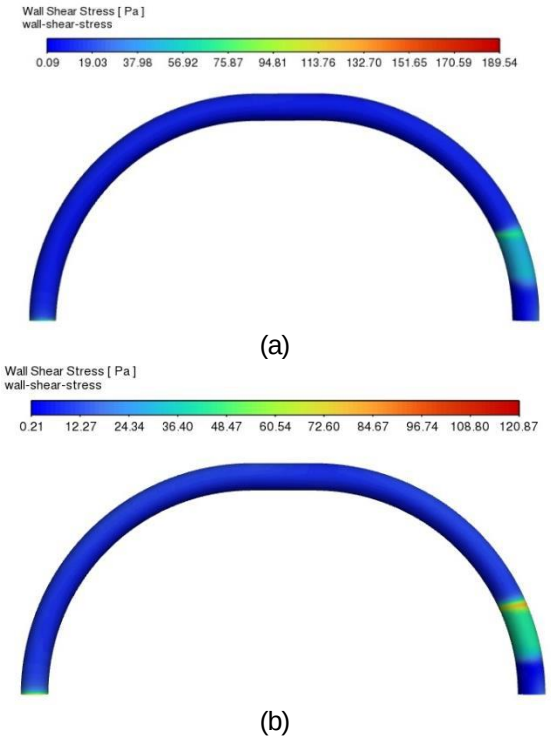


Fig.12 Wall shear stress contour obtained for (a) flexible wall (b) rigid wall

7. CONCLUSION

We have developed a fluid-structure interaction (FSI) framework to better understand blood vessel behavior in the presence of blood clot. Using FSI we are able to evaluate the structural integrity of both the clot and the arterial wall. Our findings show that considering a rigid wall will not capture the stress distribution and deformation experienced by the clot which can help us to understand the artery and clot behavior under blood flow. By using FSI, we provide a more realistic analysis of these interactions, aiding researchers in understanding how blood vessels and clots behave under blood flow. The relevance of this work to bio-tribology lies in its ability to provide analysis of the frictional and mechanical interactions between blood flow and arterial walls. The study not only enhances the understanding of the mechanical behavior of arteries under various physiological conditions but also highlights the importance of considering tribological factors in the development of therapeutic strategies for vascular diseases. This work contributes to improved insights into vascular health and the development of effective medical interventions especially involving blockage of arteries due to blood clot.

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