

Computational Study of Blood Flow Analysis for Coronary Artery Disease

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Abstract—The aim of this study is to estimate the effect of blood flow through the coronary artery in human heart so as to assess the coronary artery disease. Velocity, wall shear stress (WSS), strain rate and wall pressure distribution are some of the important hemodynamic parameters that are non-invasively assessed with computational fluid dynamics (CFD). These parameters are used to identify the mechanical factors responsible for the plaque progression and/or rupture in left coronary arteries (LCA) in coronary arteries. The initial step for CFD simulations was the construction of a geometrical model of the LCA. Patient specific artery model is constructed using computed tomography (CT) scan data with the help of MIMICS Research 19.0. For CFD analysis ANSYS FLUENT-14.5 is used. Hemodynamic parameters were quantified and flow patterns were visualized both in the absence and presence of coronary plaques. The wall pressure continuously decreased towards distal segments and showed pressure drops in stenotic segments. Areas of high WSS and high flow velocities were found adjacent to plaques deposition.

Keywords—Computational fluid dynamics, hemodynamics, velocity, strain rate, wall pressure, wall shear stress.

I. INTRODUCTION

BLOOD is the major component of human circulatory system which is used as working fluid in this present analysis of blood flow. It comprises of concentrated suspension of several cellular elements, RBCs (red blood cell or erythrocytes), WBCs (white blood cells or leukocytes) and platelets (thrombocytes) in aqueous solution. Plasma which is the liquid component of blood constitutes of mainly 95% water by volume and 5% other cellular matrix, namely electrolytes, proteins and waste products. Transportation of these substances in the circulatory system is being carried out using the plasma [7]. In order to study the mechanical properties of blood, suspended particles of fluid are taken into consideration. A fluid is said to be Newtonian if it satisfies the Newton's law of viscosity. Plasma is considered as Newtonian fluid as it mostly consists of water. Blood has complex mechanical properties and it becomes significant when the size of the particle is large as compared to lumen size. Since LCA is very small compared to other arteries, modelling of blood should not be considered as homogeneous fluid. RBCs suspension in plasma is also considered. Interaction between the blood cellular elements affects the rheological properties of blood. Based on the size of the blood vessels and blood flow behavior, fluid can be considered as non-Newtonian [8]. Blood has higher viscosity as compared to plasma. Rise in haematocrit at very low shear rates

leads to increase in viscosity of suspended particles and hence non-Newtonian behaviour of blood is relevant [9]. To represent the viscous property of blood, there is no universally accepted model as rheology of blood flow is complex. Some popular non-Newtonian models like Power law, Casson and Carreau models are used to simulate the blood behaviour to different degrees of accuracy.

Over the past few decades, stroke has become one of the most common cause deaths. Calcified plaque formation occurs gradually with due course of time which narrow down the lumen diameter of vessel thereby hardening the artery. These result in deficit blood supply to vital organs especially heart leading to stroke [1]. Due to the presence of plaque, lumen diameter reduces and variation of haemodynamics is observed [2]. Plaque occurs mostly on the region of high curvature, complex geometry and bifurcation in arteries where shear stress of fluid and other fluid properties deviates from normal healthy artery [3]. Plaques were found to be closely related to WSS on the side branches. The effect of blood flow on the arterial wall is due to the shear stress which influences the endothelial cells. Thus, deformation at plaque region and phenotype of these endothelial cells are affected. Inflammation occurs on that region and initiation of plaque progression takes place. With these findings, understanding of the atherosclerosis development by exploring and quantifying the haemodynamic effect of coronary plaques using CFD technique has improved [4].

The haemodynamic parameters of blood flow cannot be measured directly on blood vessels. Therefore, CFD techniques are employed to provide alternative ways for diagnosis of coronary artery disease [5]. CFD analysis of main coronary artery, side branches impact on WSS should not be neglected. Wellnhofer et al. [6] studied the impact of side branches on WSS calculation in 17 patients and concluded that side branches showed significant impact on coronary flow and WSS profile in the LCA.

In the present study, cardiovascular haemodynamics are analyzed along with the simulation tools to predict the behavior of circulatory blood flow in the human artery and comparisons are made for the obtained haemodynamic parameters in order to locate the position and initiation of plaque in artery.

II. GEOMETRY CREATION

For complex anatomical model, CT and MRI scan are

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preferable methods for geometry creation. CT angiography of patient suspected of coronary artery disease is taken for the study purpose. DICOM (digital imaging and communication in medicine) data were transferred to a separate workstation equipped with MIMICS version 19.0 (Materialise Interactive Medical Image Control System) for image post-processing and segmentation. Geometrical shape of LCA is acquired from grayscale images from the CT scan since a grey value signifies the material density. Three-dimensional (3D) volume data were post-processed and segmented using a semi-automatic method with a CT number thresholding technique and manual editing was performed in some slices to remove tissues that are not required. The specification of the DICOM image is given in Table I.

TABLE I MATERIAL (CT SCAN SLICE) INFORMATION	
Geometrical parameters	Values Measured
Pixel size	0.855469 mm
Image resolution	512*512 pixels
No. of DICOM slices	355
Image width	85.5469
Image height	65.0024
Slice thickness	1 mm

DICOM files are loaded onto MIMICS software where anatomy of heart can be seen by adjusting thresholding the mask properties. On manipulating the values based on HU (Hounsfield) scale (a quantitative scale for describing radio density), luminal region is segmented automatically. In this model lower threshold value is 226 HU and higher value is set at 3071 HU. Fig. 1 describes the process of geometry modeling from CT scan data using MIMICS and finally 3D model.

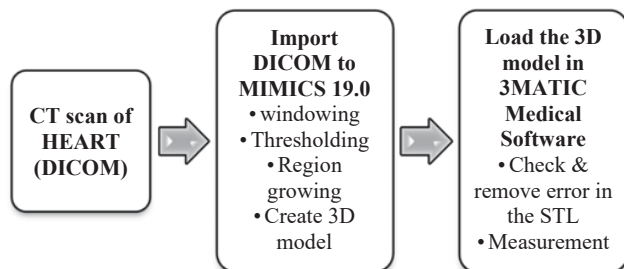


Fig. 1 Flow chart of geometry modeling

Fig. 2 shows the visualization of human heart. 3D model obtained from CT scan clearly shows the orientation and shape of LCA and RCA in heart.

LCA was extracted from the segmented model along with special focus given on branches. It is then saved in “STL format” for further computation purposes. Fig. 3 shows the anatomical details of the LCA.

The created 3D model of LCA using MIMICS software is imported to 3-Matic software (Materialise) and the geometrical measurement are made at different regions of LCA model with good accuracy as shown in Fig. 4.

From Fig. 4, geometrical dimensions of LCx and LAD artery

and their bifurcation angle were measured using 3-Matic software. The measured geometry values are listed in Table II.

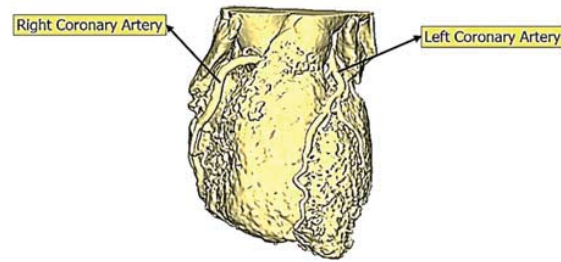


Fig. 2 3D CT visualization of a human heart

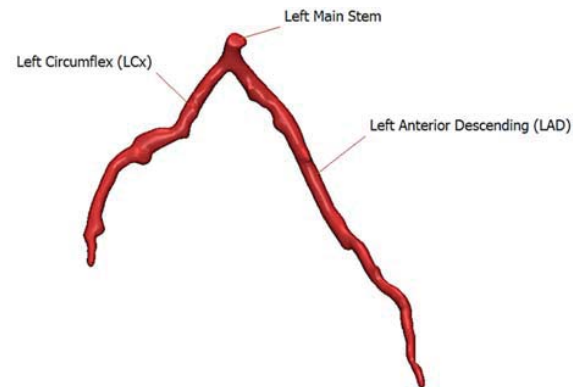


Fig. 3 3D CT visualization of a LCA

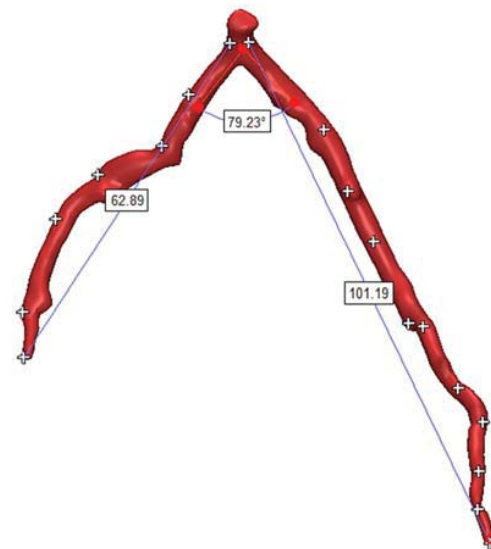


Fig. 4 Actual measurements of LCA

On measuring the patient specific artery, bifurcation angle of 80° (approx.) was found between LAD and LCx. The previous study conducted on LCA, 80° bifurcation angle was taken as a cut off value in order to determine the presence of coronary artery disease based on the study conducted for natural distribution of coronary artery bifurcation angles [12], [13].

TABLE II
CHARACTERISTIC LENGTHS (MM) OF GEOMETRICAL MODEL

Vessel	Inlet Diameter(Mean)	Length
LM	4	1
LCX	3	64
LAD	3.6	100

A. Generation of Computational Models

Once the artery is extracted from CT scan and 3D model is obtained from MIMICS software, it is exported to 3-Matic software where rough data can be cleaned up and saved in “STL format”. The tetrahedral mesh configuration for the LCA model was 279515 elements and 57558 nodes in fine meshing. The meshes were generated using ANSYS ICEM CFD version 14.5 (ANSYS, Inc., Canonsburg, PA, USA). Finally, the mesh models were saved in “IGS format” to be imported in ANSYS Fluent for simulation.

III. MATHEMATICAL MODELING

It is assumed that the flow of blood in the LCA is governed by the Navier–Stokes equations.

$$\rho \left(\frac{\partial v}{\partial t} + V \cdot \nabla V \right) = -\nabla \tau - \nabla P \quad (1)$$

The continuity equation for an incompressible fluid:

$$\nabla \cdot V = 0 \quad (2)$$

where V is the two-dimensional velocity vector, t the time, P the pressure, ρ the density and τ the stress tensor. Writing the Navier–Stokes equations in this form allows the flexibility to use an arbitrary non-Newtonian blood model. Johnston et al. [11] assumed that the fluid is incompressible and non-Newtonian. The steady flow through the stenosed artery model is therefore governed by the stationary conservation equations for mass and momentum, i.e. the Navier Stokes equations. The equations were solved using the commercially available software package ANSYS Fluent 14.5.

IV. SETUP AND FLOW SPECIFICATIONS

TABLE III
MATERIAL (CT SCAN SLICE) INFORMATION

Zero shear rate limit (μ_0)	0.056 Pa-s
Infinite shear rate limit (μ_∞)	0.0035 Pa-s
Relaxation time constant (λ)	3.313 S
Power law index in Carreau model (n)	0.3568
Blood specific heat capacity	3594 JK ⁻¹ K ⁻¹
Density	1060Kg/m ³
Dynamic viscosity (μ)	0.0035 (Ns/m ²)
Blood flow type	Laminar

Left coronary artery model obtained comprises of tetrahedral meshing. Physical and flow properties are specified in FLUENT setup with inlet velocity of 0.25 m/s is maintained. Blood viscosity properties is as per Carreau Model [11] maintained in Table III.

A. Application of Physiological Parameters

In order to ensure that analysis reflects perfect simulation of *in vivo* conditions, realistic physiological boundary conditions were applied for 3D numerical analysis. To solve governing equations, some assumptions such as flow was taken as steady and laminar with no external forces applied on it. Blood was taken as working fluid and it is considered incompressible and non-Newtonian. At outlet, stress free conditions are applied, and gauge pressure is set zero to be steady state outlet flow for LCX and LAD artery. No slip condition is assumed along the walls and considered as non-flexible and impermeable material. Plaque was assumed to be a rigid body [14]. Blood was assumed to be a Newtonian and incompressible fluid [14], [15]. The blood is treated as a non-Newtonian fluid obeying the non-Newtonian Carreau model with the viscosity-shear rate relation as:

$$\mu(x, t) = \mu_\infty + (\mu_0 - \mu_\infty) [1 + (\lambda \dot{\gamma}(x, t))^a]^{(n-1)/a} \quad (3)$$

Where μ_0 is the zero-shear rate viscosity, μ_∞ is the infinite shear rate viscosity, λ is a parameter, and n is a dimensionless parameter [10], [11].

V. RESULTS AND DISCUSSION

Haemodynamic parameters such as velocity magnitude, wall pressure and strain rate are obtained on CFD analysis for left coronary artery. As per previous study, plaque mostly occurs near the bifurcation region or any complex curvature. Fig. 5 shows the meshed model of LCA.

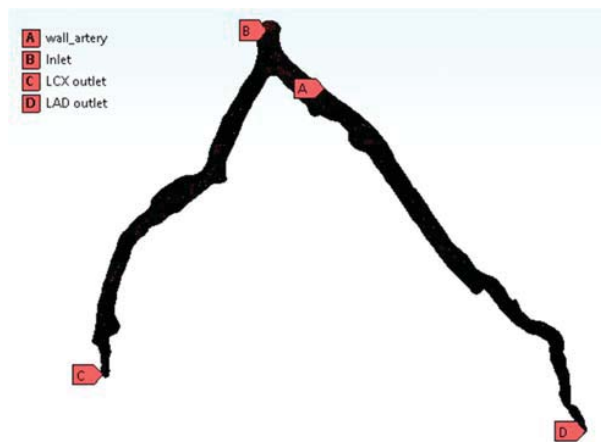


Fig. 5 Inlet and Outlet boundary conditions set up for meshed model of LCA

In the present work, an emphasis was placed on modeling the patient specific vascular domain of LCA. However fluid dynamic problem is not defined without the specification of appropriate boundary conditions. Therefore, proper setting of named selection is required so that results obtained do not miss any minor region.

Various planes were selected at different location to visualize the velocity effect. Inlet value for this simulation was 0.25 m/s.

From Fig. 6, maximum velocity of 0.3 m/s is obtained. Three different sections of left coronary artery were taken where plaque formation mostly occurs as per previous researchers.

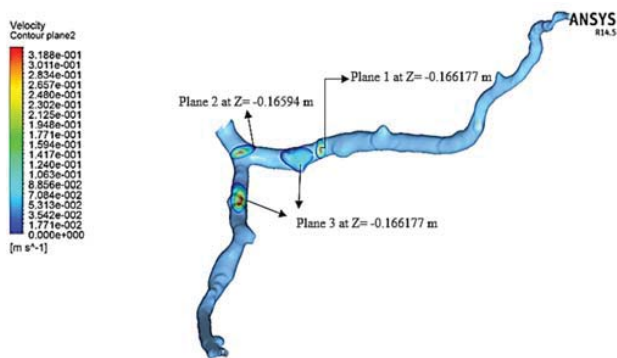


Fig. 6 Velocity distribution across planar cross section 1, 2 and 3 i.e. LM, LAD & LCx at Re = 303

Plaque can cause decrease in the luminal section of artery, hence velocity increases post plaque region. Velocity distribution across plane 2 is shown in Fig. 8. This plane is located at the LAD artery which has shown more prone region for plaque development due to complex curvature.

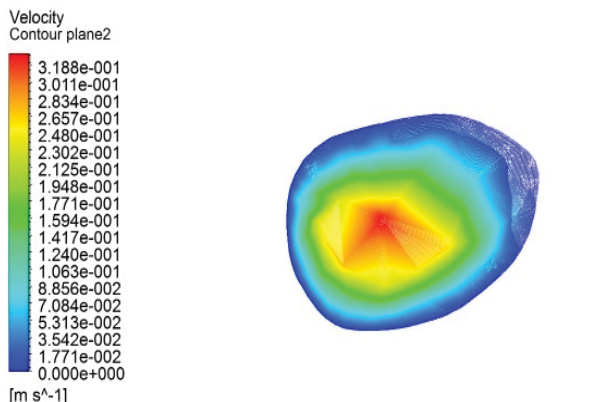


Fig. 7 Velocity contour at inlet (LM) in plane 2 at Z = -0.16594 m

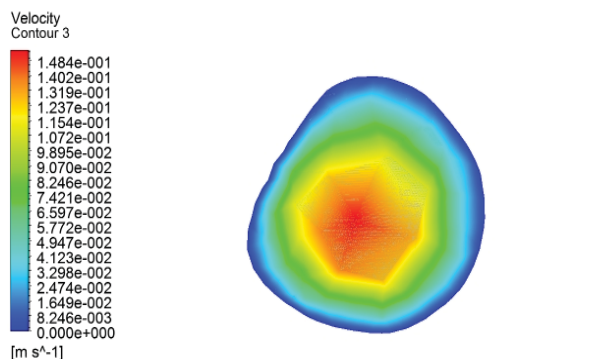


Fig. 8 Velocity contour for LAD in plane 1 at Z = -0.166177m

Presence of plaque can cause local deformation due to the blood flow. Deformation with respect to time is known as strain

rate and strain rate contour for LCA is shown in Fig. 9. As the value of strain rate is more, it can give the information about the size and position of the plaque in the artery. Due to early atherosclerosis, there is a variation in the arterial stiffness which changes the strain rate values.

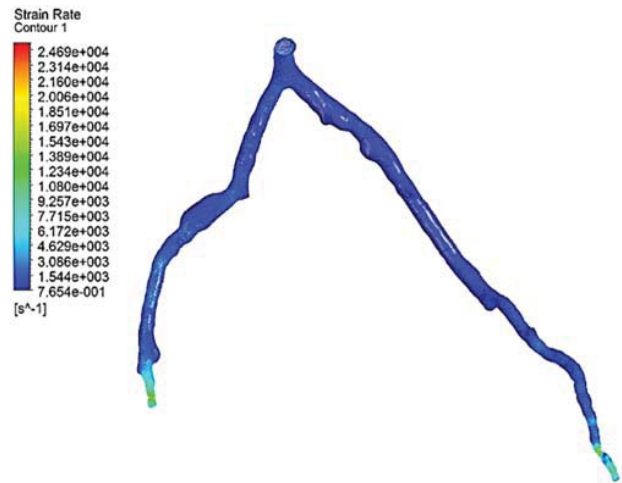


Fig. 9 Strain Rate distribution within LCA

An elaborated view of Wall Pressure distribution within LCA artery is shown in Fig.10. Pressure drop intensifies through the branch i.e. LAD and LCx depending on the local morphology, size and branching angle. The wall pressure continuously decreased towards distal segments and showed pressure drops in stenotic segments.

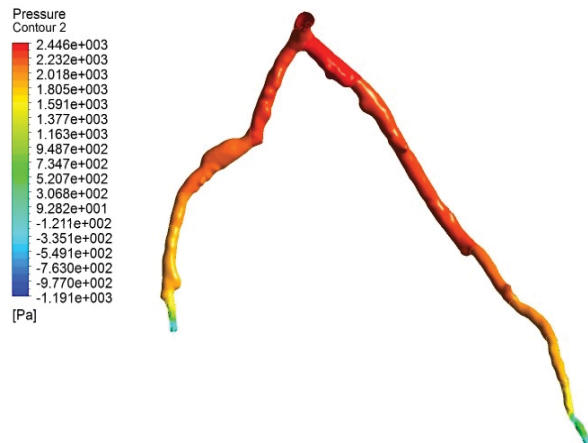


Fig. 10 Wall Pressure distribution within LCA

In this study, CFD was used to analyze the hemodynamics parameters of normal and atherosclerotic artery. Non-Newtonian model was simulated using the Carreau model since it is capable of predicting the hemodynamics parameters more accurately to physiologists as confirmed by previous study. In the stenosis region of artery, velocity magnitude, strain rate, wall pressure and WSS are found to be higher because blood flow area is reduced. This indicates the presence of plaque in

the artery. It has been revealed that atherogenesis preferentially involves the outer walls of vessel bifurcations, side branches and regions of high curvature in the arterial tree. Hence areas of high WSS and high flow velocities were found adjacent to plaques deposition.

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