Numerical Analysis of Blood Flow through Multiple Stenosis Right Coronary Artery

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Abstract
The effect of stenosis on pulsatile blood flow through multiple stenosis right coronary artery (RCA) was found by using computational fluid dynamics (CFD). The data of this study were taken from Catheterization Laboratory in Basra Cardiac Center. The case used is for woman has three stenosis in her RCA with different stenosis ratios which are; 75.15%, 41.21%, and 32.09% respectively. A non-Newtonian blood model characterized by Carreau equation, as well as Newtonian model of blood viscosity was used in the flow simulation. The simulations were performed by using ANSYS FLUENT software based on the finite volume method. The study shows that, non-Newtonian viscosity of blood flow is inversely proportional with shear rate distributions and its effects occurring close to the centerline of flow. The hemodynamics characteristics (velocity, pressure drop, and wall shear stress) increases with increase stenosis ratio for multiple stenosis RCA. In addition, comparison between two viscosity models shows very similar behavior for axial velocity profile, also the non-Newtonian property strengthen the peak values of WSS and increasing pressure drop.

Keywords: Pulsatile flow; Non-Newtonian; Hemodynamic characteristics; Right coronary artery; Multiple stenosis.

1. INTRODUCTION
Recent developments in the field of computational fluid dynamics CFD have capability to simulate the blood flow in the cardiovascular system. Computer can show you the flow pattern of blood for a various disease artery. Therefore, CFD is nowadays become a clinical diagnostic tool for the medical practicing throughout the region of vascular diseases.

Blood does the main functions of delivering nutrients and oxygen to all the human tissues, protecting the body against infection by removing waste products through the work of antibodies. The heart is hollow muscular organ responsible for providing the driving pump to move blood in body, the heart is composed of two separate pumps: a left heart that pumps blood through systemic circulation that provides blood to all organs and tissues of the body except lungs, and a right heart that pumps blood in pulmonary circulation that provides blood to lungs. Each of these hearts is a pulsatile two separate pump composed of an atrium and a ventricle [1]. Arterial stenosis is a partial occlusion of the artery caused by abnormal growth of tissues or plaque build-up on the arterial wall which cause atherosclerosis, this plaque cause a narrowing of the arterial passage [2].

There were strong proof that hemodynamic factors like; flow recirculation, flow separation, low and oscillatory wall shear stress (WSS), and changes in the rheological properties of blood and its components, play a main role in the development and progression of atherosclerotic and other arterial lesions [3].

X. Xu and M. Collins (1994) [4], and T. Ishikawa et al. (1998) [5] they were investigated the effects of the non-Newtonian viscosity of blood flow in general three-dimensional arterial bifurcation, they found that the duration of flow separation is shorter and the reverse flow is weaker with the non-Newtonian fluid compared with Newtonian fluid except this it does not have significant effects on the basic features of the flow field. B. Johnston et al. (2006) [6] seen transient study of pulsatile blood flow through four right coronary arteries using both the Newtonian and non-Newtonian models used was the Generalised Power Law of blood viscosity, the results shows a little practical difference between Newtonian and non-Newtonian models of blood viscosity about 30% of the cardiac cycle. Rabby et al., (2013) [7] find out how pulsatile blood flow behaves in arterial stenosis, they found how pressure drops at stenosis and its shear stress increases suddenly at the center of the stenosis. Jahangiri et al, (2018) [8] presented the behavior of hemodynamic parameters of blood by using six non-Newtonian models and compared the results with Newtonian model, they found that Walburn-Schneck and Power-law models have the lowest axial velocity, pressure difference, and shear stress as compared with other viscosity models.

A number of researchers have studied the flow of blood through single stenosis arteries as illustrated in the upper part, but a very few researchers have studied blood flow through multiple stenosis arteries. One of the important factors that effect on blood flow through arteries is the geometry of the artery. In the present study, we show hemodynamic parameters behavior for blood characterized by Carreau model that flow through real multiple stenosis arterial geometric. Moreover, compare the effects of two viscosity models (Carreau and Newtonian) on hemodynamic factors.
2. METHOD

2.1. Right Coronary Artery

Coronary arteries are the blood vessels which deliver the oxygenated blood to the cardiac muscles. The left coronary artery supplies the left lateral and anterior portions of the left ventricle, while the right coronary artery supplies the right ventricle and the posterior part of the left ventricle [1]. Coronary arteries in the human body will generally curve and bend. As seen from x-ray pictures in Fig. (1). The data was taken from Catheterization Laboratory in Basra Cardiac Center. The picture shows that this patient has a three stenosis in RCA with stenosis ratios 75.15%, 41.21%, and 32.09% respectively. In order to study the hemodynamics of flow through RCA, we need to simplify the geometry while keeping the essential features intact. Two-dimensional blood flow through rigid RCA walls was numerically simulated. AutoCAD 2013 software is used to modeling RCA, with a scale appropriate with RCA dimensions.

Figure 1: Multiple Stenosis RCA.

2.2 Governing Equations

The governing equations represent mathematical statement of conservation laws of physics, which were used to formulate a solution based on the flow properties and the boundary conditions. The continuity and momentum equations for two-dimensional, unsteady, incompressible, and laminar flow are written respectively as follows [9]:

The Continuity Equation:

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0$$  \hspace{1cm} (1)

The Momentum Equations:

X-Direction:

$$\rho \left( \frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} + v \frac{\partial u}{\partial y} \right) = - \frac{\partial p}{\partial x} + \mu \left( \frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right)$$  \hspace{1cm} (2)

Y-Direction:

$$\rho \left( \frac{\partial v}{\partial t} + u \frac{\partial v}{\partial x} + v \frac{\partial v}{\partial y} \right) = - \frac{\partial p}{\partial y} + \mu \left( \frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2} \right)$$  \hspace{1cm} (3)

Where $u$ and $v$ velocity components in x and y directions respectively, $p$ is pressure, $\rho$ is the density of blood, and $\mu$ is blood viscosity.

2.3 Flow Conditions

The governing equations normally contain arbitrary functions, and to solve governing equations a set of boundary conditions are required. It was assumed that initially no flow takes place when the system is at rest. The boundary conditions ($t>0$) can be summarized as follows:

Inlet RCA: using mean pressure readings were appearing in monitor’s screen when catheter reaches RCA in Catheterization Laboratory. Pressure reading at artery inlet is 16998.3 Pa (127.5 mmHg).

Outlet RCA: unsteady parabolic velocity profile was imposed. The outlet velocity was provided in a sine waveform Equation for the pulsatile flow simulations, and it is represented by the following equation [10]:

$$U(t) = 0.25 \left( 1 + \sin \left( 2\pi \frac{t}{T} \right) \right)$$  \hspace{1cm} (5)

Where $t$ is a local time and $T$ is the period of oscillation time. In the present study $T=0.8s$.

2.4 Non-Newtonian Model

Non-Newtonian blood flow was modeled as Carreau viscosity model. Which is the most popular models in non-Newtonian blood modeling. The complex rheological behavior of blood is approximated using a shear-thinning model by the Carreau model [11]:

$$\mu = \mu_\infty + (\mu_0 - \mu_\infty)(1 + (\lambda \dot{\gamma})^2)^{\frac{n-1}{2}}$$  \hspace{1cm} (6)

Where: $\mu_0 = 0.056 Pa.s$ is zero shear viscosity, $\mu_\infty = 0.0035 Pa.s$ infinite shear viscosity, $\lambda = 3.313 s$ is time constant, $n= 0.6$ flow behavior index, and $\dot{\gamma}$ is the shear rate tensor was given as [11]:

$$\dot{\gamma} = \sqrt{\frac{1}{2} \sum_j \sum_i d_{ij} d_{ji}}$$  \hspace{1cm} (7)

Where $d_{ij}$ is the rate of deformation tensor. Final shape of the shear rate tensor for 2D (x-y) is:

$$\dot{\gamma} = \left[ 2 \left( \frac{\partial u}{\partial x} \right)^2 + 2 \left( \frac{\partial v}{\partial y} \right)^2 + \left( \frac{\partial v}{\partial x} + \frac{\partial u}{\partial y} \right)^2 \right]^\frac{1}{2}$$  \hspace{1cm} (8)

2.5 Computational Details

The 2D unsteady Navier-Stokes equations were solved by means of a commercial CFD package FLUENT 15.0 based on the finite volume method. The upwind differencing scheme was employed for all equations and the velocity-pressure coupling algorithm was SIMPLE (Semi-Implicit Method for Pressure
Linked Equations) in the CFD calculations. For this study, 80 time steps per cycle were chosen, with 0.01 time step size.

3. RESULTS AND DISCUSSION

Transient simulations were performed for each of the three arteries described above. Each simulation was from \( t=0 \) to 0.8s, yielding a heart rate of 75 beats per minute. The simulations were performed using Carreau blood viscosity model with additional simulation performed using a Newtonian model. This simulation is used to study the difference between the two viscosity models. The waveform in Fig. (2) represent velocity equation at artery outlet and can be divided into four different phases; early systole, peak systole, early diastole and peak diastole corresponded to \( \frac{t}{T} = 0, 0.25, 0.5, \) and 0.75 respectively. Velocity, pressure drops, and wall shear stress (WSS) distributions results are presented in this section.

3.1 Non-Newtonian Viscosity

The dynamic viscosity distributions at four phases (early diastolic, peak diastolic, early systolic, and peak systolic) with in one cardiac cycle of blood flow through multiple stenosis RCA are show in fig. (3). They would seem that more of the flow region has a viscosity near to the Newtonian viscosity value (0.00345 Pa.s), as observed by the majority of the contours plot falling within the legend containing the non-Newtonian viscosity values. This deduces that the majority of the flow has a high shear rate, in which blood losses its non-Newtonian properties.

The regions in which the non-Newtonian viscosity is more dominate, is the regions close to the centerline of flow. There is a noticeable peak in viscosity values at this place, which corresponds to the place of highest viscosity. Where blood flow there is shear forces from the boundary layer resist flow therefore, a high shear occurs near the wall but low shear rates exists near the centerline of flow. These regions of high viscosity dose not vary with time of cardiac cycle, particularly they clearly seen in peak systolic phase of cardiac cycle, which is the end-deceleration phase where velocity are very low. Mean viscosity values at peak diastolic phase at throats of multiple stenosis RCA are 0.00366, 0.00393, and 0.00396 Pa.s for 75.15%, 41.21% and 32.09% respectively. From these result we can conclude that blood flow viscosity decreases with increases stenosis ratio of RCA because of increasing shear rate as stenosis ratio increase in stenosis regions.

![Velocity waveform at the artery outlet.](image)

**Figure 2:** Velocity waveform at the artery outlet.

3.2 Velocity Distribution

The velocity distributions results are shown at four phases (early diastolic, peak diastolic, early systolic, and peak systolic) with in one cardiac cycle. Fig.(4) presents the mean velocity along the length of multiple stenosis RCA, it seen that the magnitudes of mean velocities are larger during peak diastolic phase, and velocity value increase as blockage artery increase. Peak velocity value at 75.15% stenosis is 53% larger than peak velocity value at 41.21% stenosis and 56% larger than peak velocity value at 32.09% stenosis. Opposite of peak diastolic phase is peak systolic phase, in this phase velocity value decreases as blockage artery increase reach minimum value 0.0135 m/s at the throat of 75.15% stenosis due to reverse flow during peak systolic phase.

![Mean velocity along length of multiple stenosis RCA during one cardiac cycle.](image)

**Figure 3:** Viscosity counters of multiple stenosis RCA for one cardiac cycle.

![Mean velocity along length of multiple stenosis RCA during one cardiac cycle.](image)

**Figure 4:** Mean velocity along length of multiple stenosis RCA during one cardiac cycle.

To show the effects of stenosis ratio on axial velocity profiles in multiple stenosis RCA, Fig.(5) (a)-(c) present axial velocity profiles during four phases of one cardiac cycle and at three locations; at throats of 75.15% , 41.21%, and 32.09 % stenosis
respectively. As stenosis ratio increase the effect of jet flow increase and axial velocity profiles becomes uniform, furthermore RCA is sinuate so the effects of curvatures cause inclined velocity profiles.

In Fig. (6), here we can observe the velocity profile of the Newtonian and non-Newtonian models through multiple stenosis RCA for three different axial locations and at peak diastolic phase. Very little variation occurred between the two models at the throat of stenosis and largest difference occurred in the before stenosis lines during the low velocity portion. It can be observed that the axial velocity profiles in non-Newtonian model will be bending inwards in comparison with Newtonian model. Because of non-Newtonian fluid provides higher resistance to flow due to increase viscosity values toward centerline of blood flow compared with constant viscosity value to Newtonian model.
3.3 Wall Shear Stresses

Fig.(7) shows WSS distribution with the two profiles represents both sides of multiple stenosis RCA at a particular phases of the complete cardiac cycle. The maximum values of WSS at any point for all phases are achieved with outer wall profile. At outer wall for throat of 75.15% stenosis, shearing stress increases sharply compared with its value at inner wall, because of the outer wall has a protrusion. Furthermore, there is slightly different in WSS magnitude at throat of 32.09% stenosis on both sides due to approximately symmetric flow in its region. The maximum values of WSS for outer side and at the throat of stenosis for stenosis ratios 75.15%, 41.21% and 32.09% are 340, 112 and 100 Pa respectively. Therefore, we can conclude that as stenosis ratio increase WSS values increase at stenosis region.

WSS distributions along inner and outer walls during peak diastolic phase are shown in Fig. (8). Where the solid lines correspond to Non-Newtonian and the dashed lines correspond to Newtonian cases. Furthermore, maximum WSS values are at the throat of stenosis for both the Newtonian and Non-Newtonian cases. However, from the graphs we can notice that non-Newtonian WSS is higher than Newtonian case at the stenosis place due to larger viscosity values for non-Newtonian compared with constant value for Newtonian case.

Figure 6: Axial velocity profiles of multiple stenosis RCA for two viscosity models and at three lines.
3.4 Pressure drops

Fig. (9) present pressure drops curves in multiple stenosis RCA. Along the first stenosis (75.15%) the pressure decreased owing to spatial acceleration of flow and viscous effect, pressure drops value is 11.54 mmHg along first stenosis at peak diastolic phase. Also at same phase, pressure drops increases again as flow accelerated in the region of decreasing area at 41.21% stenosis along this region pressure drops value is 1.6 mmHg. In the flow separation region between the stenosis, the pressure remained relatively constant. At 32.09% stenosis pressure drops along its region equal to 1.3 mmHg. Therefore, pressure drops along stenosis region for multiply stenosis RCA increase as stenosis ratio increase.

Figure 7: WSS along inner and outer multiple stenosis RCA walls for one cardiac cycle.

Figure 8: WSS along inner and outer multiple stenosis RCA walls at peak diastolic phase for two viscosity models.
4. CONCLUSION

The hemodynamics characteristics of laminar flow in two-dimensional under pulsatile conditions for blood flow through multiple stenosis RCA are obtain. Geometrical date of RCA is taken from catheterization laboratory in Basra Cardiac Center, and commercial computational fluid dynamics code FLUENT is employ to solve the governing equations. The main conclusions from this study can be summary as follows:

- The viscosity distributions for the Carreau model show significant non-Newtonian effects occurring close to the centerline of flow.
- In stenosis region, viscosity values decreases due to increasing shear rate this leads to increasing blood flow velocity. Furthermore, increasing stenosis ratio leads to increasing velocity and decreasing viscosity for blood flowing through stenosis.
- At stenosis region, WSS increases rapidly especially at peak flow phase, WSS values increase as stenosis ratio increase. In addition, WSS for the non-Newtonian model tended to have a slightly higher magnitude than the Newtonian model.
- Pressure drops along stenosis region increase as stenosis ratio increase. The non-Newtonian model shows higher pressure drops from the inlet to the outlet throughout the flow phase than the Newtonian model.

REFERENCES


