A Comparative CFD Study on the Hemodynamics of Flow through an Idealized Symmetric and Asymmetric Stenosed Arteries

B. Prashantha, S. Anish

Abstract—The aim of the present study is to computationally evaluate the hemodynamic factors which affect the formation of atherosclerosis and plaque rupture in the human artery. An increase of atherosclerosis disease in the artery causes geometry changes, which results in hemodynamic changes such as flow separation, reattachment, and adhesion of new cells (chemotactic) in the artery. Hence, geometry plays an important role in the determining the nature of hemodynamic patterns. Influence of stenosis in the non-bifurcating artery, under pulsatile flow condition, has been studied on an idealized geometry. Analysis of flow through symmetric and asymmetric stenosis in the artery revealed the significance of oscillating shear index (OSI), flow separation, low WSS zones and secondary flow patterns on plaque formation. The observed characteristic of flow in the post-stenotic region highlight the importance of plaque eccentricity on the formation of secondary stenosis on the arterial wall.

Keywords—Atherosclerotic plaque, Oscillatory Shear Index, Stenosis nature, Wall Shear Stress.

I. INTRODUCTION

ATHEROSCLEROSIS is a common dangerous disease and it causes death in many countries because of pulsatile blood behavior in the human artery [1]. Blood is an inhomogeneous fluid consisting of red cells, platelets and a number of white cells. Several autopsy study have been demonstrated 70-80% of cardiovascular diseases (atherosclerosis plaque) occurs at complex geometries [2]. This continuous development causes narrowing of the vessel lumen (known as stenosis) creating significant flow disturbances [3]. These disturbances and turbulences in the flow influence the variations in the mechanical parameters such as stress acting on wall and wall elongation. Large plaque formation in the artery changes the wall shear stress distribution on the arterial walls [4]. Flow behavior in the stenotic arteries influences the stress on the plaque and thereby stimulates the plaque rupture also.

Quite often, the adhesion of new cells on the wall occurs at regions where the wall shear stress is lower [5]. In a normal healthy person, blood flow through the artery is laminar. However, the presence of new cells in the blood flow and its adhesion on the arterial wall make the flow transitional or turbulent [6] and change of pulse frequency causes laminar to turbulence flow in the artery. Certain combination of pulsatile flows and geometries will generate vortices and secondary flows in the flow and it promotes accumulation of new cells on to the arterial wall [7]. For the accurate prediction of plaque formation and development a thorough understanding of the flow behavior at the post stenotic region is essential.

The objective of the present study is to understand the effect of pulsatile blood flow behavior through an idealized symmetric and asymmetric stenosed channel. The analysis is carried out for severe stenosis case (75% of area reduction). During pulsatile flow, distributed streamlines, velocity vector fields and Oscillatory shear stresses in the post stenotic region clearly defines the influence of stenosis location (eccentricity) in the arterial wall. Symmetric case results were validated with experimental results of [8].

II. METHODOLOGY

Real artery geometry is very difficult to reconstruct due to its complex internal structure and the difficulty in obtaining real patient data. Hence, present computational study is an ideal geometry model has been considered [8]. The computational fluid model is set up for 75% reduction in area and was modelled using CATIA. The geometry of an idealized artery model has been scaled up keeping the dynamic similarity. Inlet and outlet diameter has been considered as 2 inch and upstream length will be considered as 100 times of diameter, downstream length will be considered as 14 times of the diameter from the throat section as shown Fig. 1 (a).

Asymmetric stenosis model will be constructed by varying eccentricity of the minimum constricted flow diameter in the passage (stenosis) as shown in the Fig. 1 (b). Three-dimensional smooth geometries are developed by using CATIA and meshing has been done through ICEM CFD. Grid independence study has been carried out to arrive at an optimum number of grid elements for the computational study. This optimizes the computational time without compromising with accuracy of the solution. Computational simulations have been carried out using Finite volume method (FVM) by commercial software ANSYS FLUENT. Two equation turbulence modelling (K-omega Standard) Naiver strokes equations has been solved by using second order upwind discretization method and Pressure Implicit Splitting Operator algorithm (PISO).

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World Academy of Science, Engineering and Technology
International Journal of Mechanical and Mechatronics Engineering
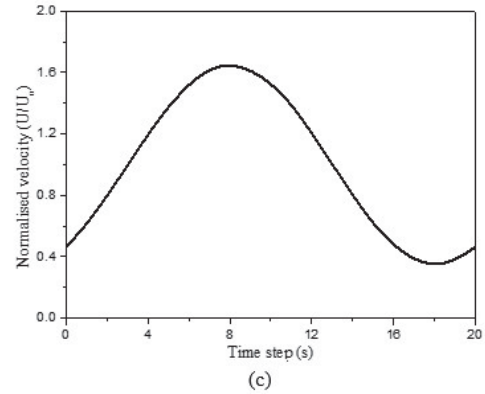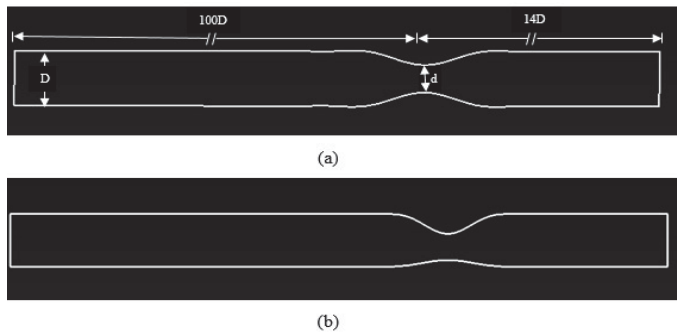Vol:10, No:5, 2016

Fig. 1 Geometric representation of (a) symmetric and (b) asymmetric stenosis (c) Inlet velocity profile Boundary condition

Blood is assumed as Newtonian fluid with density of 1060 kg/m^3 and viscosity of 0.0036104 Pa s [8]. Velocity wave form Fig. 1 (c) taken out from the Ahmed and Giddens experimental result and created as a User Defined Function (UDF) using C language, was applied as an input velocity boundary condition and outlet can be defined as outflow boundary condition within the FLUENT. After the completion of three inlet velocity wave form, the simulation results were extracted from third cycle [6].

III. Results and Discussion

This study is an extension of symmetric stenosis case study carried out by simulation work [6] to asymmetric case of deposited plaque on the arterial wall, to examine the detailed hemodynamic flow behavior during the presence of symmetric and asymmetric stenosis in the rigid arterial wall. Previous Ahmed and Giddens literature data describes hemodynamic behavior in the different degrees of symmetric stenosis in an artery. Present study focuses on the hemodynamic behavior in the presence of protruded asymmetric plaque over the lumen area. In the post stenotic region flow becomes turbulent when the stenosis exceeds 50% of area [8], hence, we extended our study for the severe case (75%). In the present study, analysis has been done at three axial locations in the post stenotic region.

A. Validation

The present computational work has been validated with experimental result of [8] as shown in Fig. 2. These plots show that computed values are in good agreement with the experimental results. Available experimental velocity profile at -1.5D axial location, Fig. 1 (c), has been considered for the simulation inlet boundary condition for the analysis, (1) shows the similar velocity profile as taken out from the experimental velocity profile at -1.5D location. Where U/U_m the normalized inlet velocity along centerline axis and Um is the mean velocity of 0.04254 ms^{-1}

$$V (t) = [0.042517+0.027969*cos (2*3.1415*t/20-2.53)] \quad (1)$$
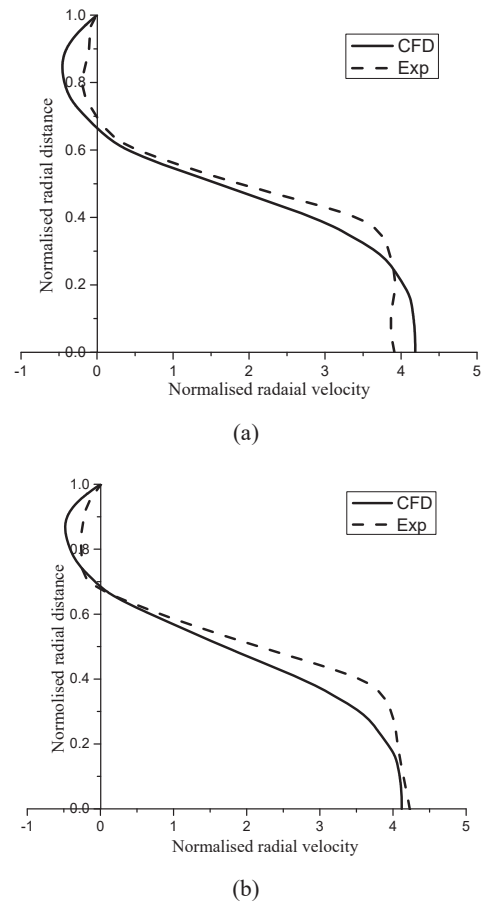
where 't' is the time step.

(a)

(b)

Fig. 2 Comparison of radial velocity profile at peak time step in the post stenosis region (a) Plane at 1D location (b) Plane at 1.5D location

B. Effect of Stenosis Nature on Hemodynamic Flow Field

Flow streamlines at two different instance of the pulsating flow is shown in Fig. 3. One is in the accelerating and other is in the deceleration phase of the systole and diastole. It is observed that the recirculation zone is quite large in the deceleration phase (t=14s). The reattachment occurs at 5D distance for the symmetric model whereas for the asymmetric model it occurs at 9D during the deceleration phase. During deceleration diastolic time phase, asymmetric stenosis distributes strong recirculated secondary vortices up to 9D

World Academy of Science, Engineering and Technology
International Journal of Mechanical and Mechatronics Engineering
Vol:10, No:5, 2016

locations, however in the case of symmetric stenosis only mild fluctuated turbulent flow pattern can be observed along the
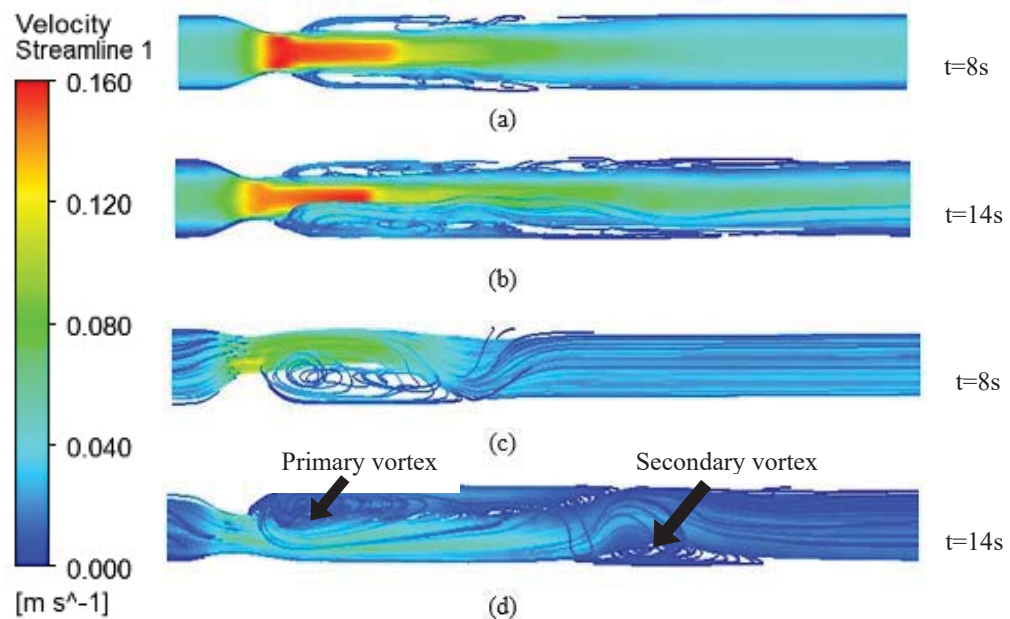
stream wise distance.

Fig. 3 Effect of stenosis eccentricity allots recirculation regions in the post stenotic region towards outer wall (a) Symmetric case at peak time step (b) Symmetric case at deceleration time step (c) Asymmetric case at peak time step (d) Asymmetric case at deceleration time step

In the case of peak systolic time phase (t=8s), flow reattachment in both the cases have nearly same length (3D) but vortices are unevenly distributed. Whereas symmetric stenosed atrial wall generates a set of symmetric vortices immediate next to the stenosis, those generated vortices will not continue their growth. Hence, after small region of stream distance fully developed (reattachment) flow can be observed. But in the asymmetric case strong vortices will grow with the time step and more recirculation region length occurs along the stream wise distance (9D).

Recirculation region length of velocity stream lines clearly signifies developed strong recirculating vortex patterns and with time steps. In asymmetric cases primary vortex are detached as soon and reattach next opposite to the wall at a certain region of length as shown in Figs. 4 (c), (d). Hence, clear picture of these results defines there will be more chances of deposition of new cells (plaque) irregularly in the post stenotic region as compare with the symmetric case. This signifies importance of the lumen geometry in the distribution of wall shear stress.

C. Effect of Wall Shear Stress

Flow streamlines at two different instance of the pulsating flow is shown in Fig. 3. One is in the accelerating and other is in the deceleration phase of the systole and diastole. It is observed that the recirculation zone is quite large in the deceleration phase (t=14s). The reattachment occurs at 5D distance for the symmetric model whereas for the asymmetric model it occurs at 9DD during the deceleration phase. In the case of peak systolic time phase (t=8s), flow reattachment in both the cases have nearly same length (3D) but vortices are

unevenly distributed. Whereas symmetric stenosed arterial wall generates a set of symmetric vortices immediate next to the stenosis. These generated vortices will not continue their growth but after small distance downstream reattachment can be observed. In the asymmetric case strong vortices will grow with the time step and more recirculation region length occurs along the stream wise distance (9D).

The length of the recirculation region differs in both cases and so the wall shear stress distribution. Asymmetric channel develops strong recirculating vortex patterns which evolves with time step. In asymmetric cases the detached primary vortex soon reattaches opposite to the wall at a certain length which may depends on the degree of stenosis. Hence with asymmetric stenosis there will be more chances of deposition of new cells (plaque) irregularly in the post stenotic region as compare with the symmetric case, signifying the importance of lumen geometry in the atherogenesis progression.

D. Effect of Oscillatory Shear Stress

Oscillatory shear index is an important parameter in the pulsatile blood flow through the arteries, which is used to quantify the multiple sign changes of WSS (unsteady) in the cardiac cycle. OSI can be calculated as,

$$OSI = \frac{1}{2}\left(1 - \frac{|\int_0^n \tau|}{\int_0^n |\tau|}\right) \qquad (2)$$

where 'τ' is the wall shear stress derived from velocity fields.

World Academy of Science, Engineering and Technology
International Journal of Mechanical and Mechatronics Engineering
Vol:10, No:5, 2016

It ranges from 0 to 0.5, small values of OSI indicates flow separation and reattachment, whereas large values of index highlights fully reversed steady flow [7].
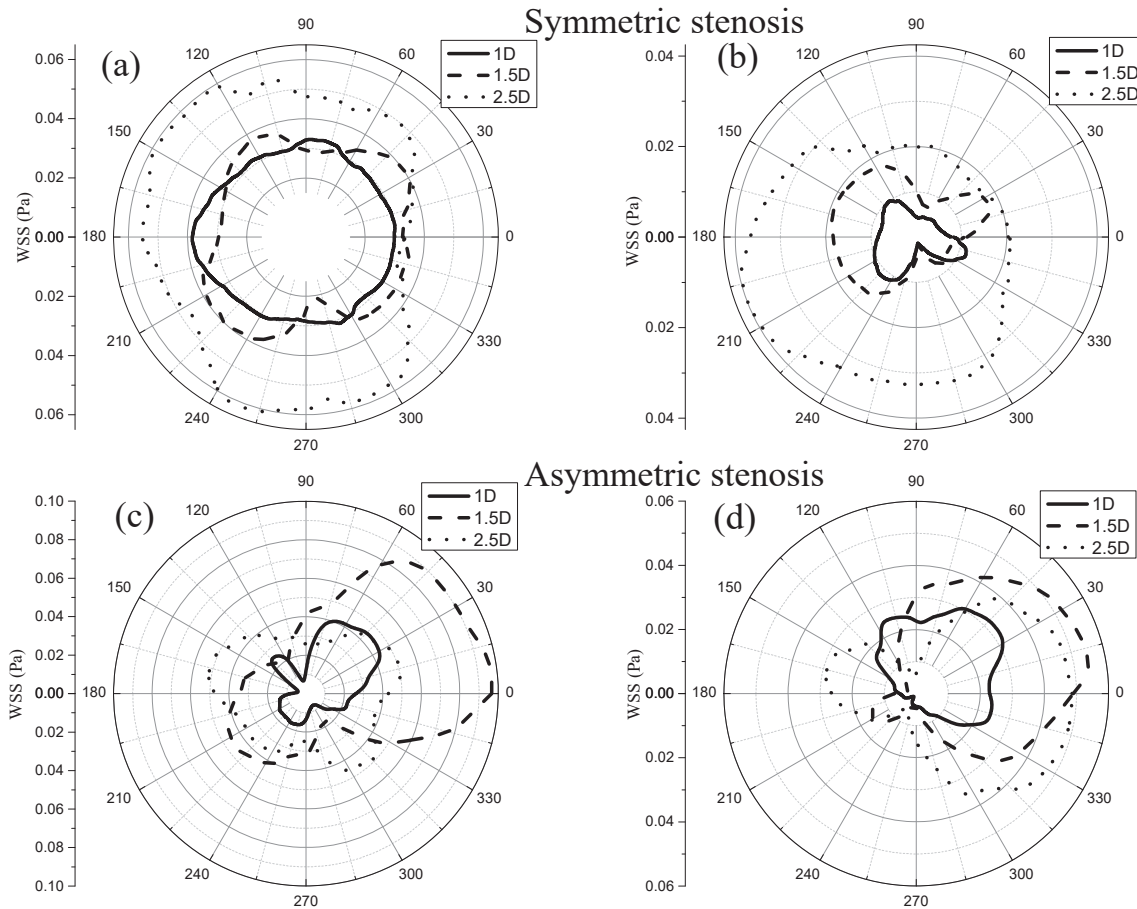

Fig. 4 Angular variation of WSS at different axial locations (a) Symmetric case at peak time step (b) Symmetric case at deceleration time step (c) Asymmetric case at peak time step (d) Asymmetric case at deceleration time step.

The OSI values are localized to a comparatively small angular space for the symmetric stenosis case. Especially for the decelerating phase the OSI values are very small at 1.5D and almost nil at 2.5D. On the contrary, for the asymmetric case the oscillatory shear index exhibits a significantly strong presence over a wide range of angular location. For each axial location, the peak OSI value occurs at almost similar angular location for the symmetric stenosis. Whereas for the asymmetric stenosis the angular location of the peak OSI keeps on shifting along the axial distance. This is possibly due to the formation of the secondary vortex along the downstream.

complex geometries are responsible for the asymmetric plaque formations in the artery. Even though the degrees of stenosis are same for both symmetric and asymmetric case the pulsatile flow and the eccentricity creates a complex secondary vortex generation in the asymmetric channel. As the adhesion of new cells and subsequent plaque formation happens at low wall shear stress region, an increase in the irregularity of the artery lumen may be conducive for secondary plaque formation and atherogenesis. The angular variation of wall shear stress and OSI values show that asymmetric stenosed channel is more prone to atherogenesis than the symmetric channel.

IV. CONCLUSION

In this CFD analysis, a comparison of hemodynamics features through two idealized stenosed artery models are carried out. These two models differ in such a way that one has symmetric stenosis and the other has asymmetric stenosis.

The symmetric model has been validated well with the established experimental results. From the analysis of flow streamlines, it is understood that pulsatile flow nature and

World Academy of Science, Engineering and Technology
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Vol:10, No:5, 2016

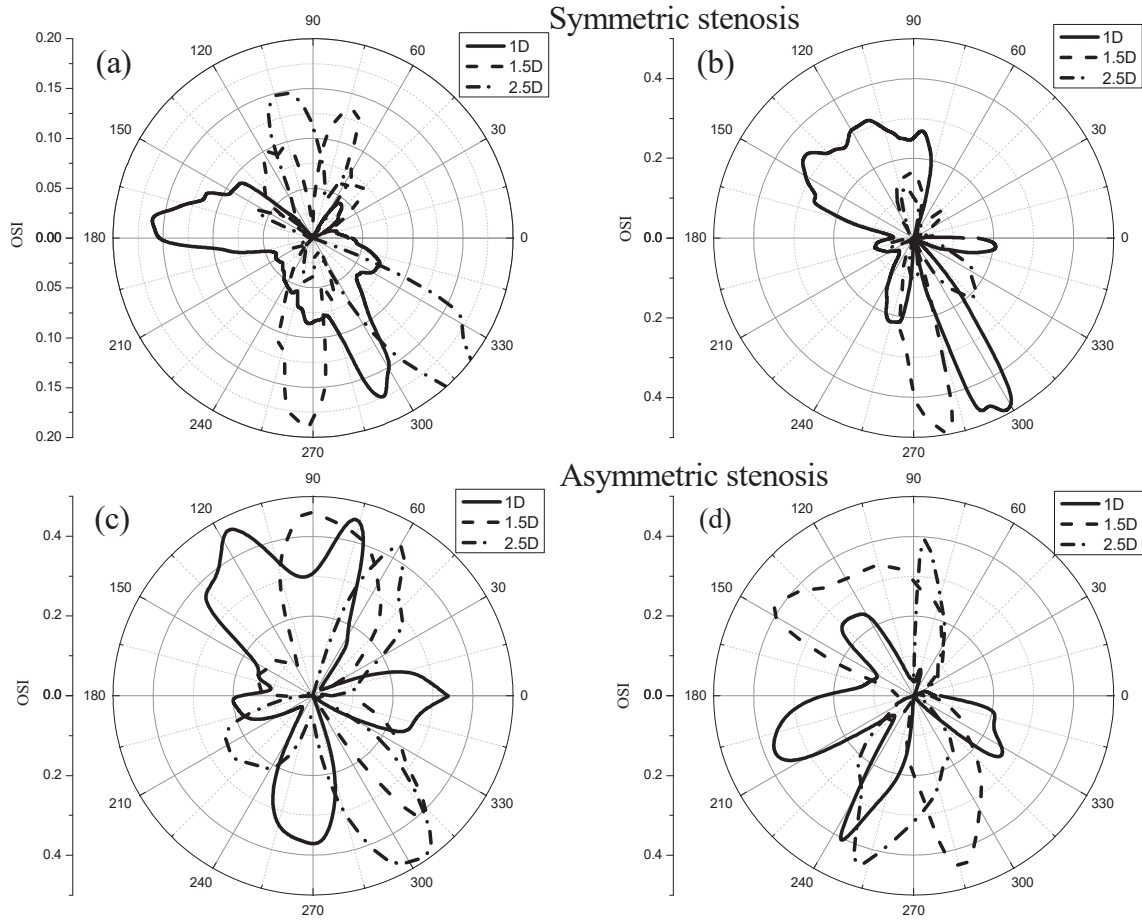## Symmetric stenosis

Asymmetric stenosis

Fig. 5 Variation of OSI in the post stenotic region (a) Symmetric case at peak time step (b) Symmetric case at deceleration time step (c) Asymmetric case at peak time step (d) Asymmetric case at deceleration time step

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