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Research Paper

Study of Effect of Wall Shear Stress during a Cardiac Phase on Plaque Progression.

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Abstract: In this paper various hemodynamic parameters has been introduced, how it is related to plaque progression is discussed. Computational fluid dynamic is used for analysis of flow velocity, wall shear stress *(WSS)* and Wall pressure gradient. How WSS is related to plaque progression is analyzed. Heart diseases causes millions of death every year and still increasing. Studying hemodynamic parameter can be helpful to take preventive measures. Analyzing various parameters proved that studying effect of *WSS* during entire cardiac phase can be useful. Future study requires to analyze the realistic coronary models from patient's data with different degree of coronary stenosis.

Keywords: Coronary artery disease *(CAD), WSS*, Blood flow, Flow velocity, Plaque, Simulation.

1. Introduction

Coronary artery disease *(CAD)* is the major cause of death in countries. The most common cause of *CAD* is atherosclerosis which is caused by the presence of plaques on the artery wall, resulting in the lumen stenosis. Plaques have been particularly involved in blood clot and blocks blood stream to myocardium. This occurs when the coronary plaques suddenly rupture; if a clot cannot be treated in time, then the heart muscle will be impaired due to ischemic changes, leading to ischemia or myocardial infarction or necrosis **[1].** Therefore, an early detection and diagnosis of *CAD* is particularly important for reduction of the mortality and subsequent complications **[1].** The natural history of coronary plaque is dependent not only on the formation and progression of atherosclerosis, but also on the Hemodynamic response. If the local wall shear stress is low, a proliferative plaque will form. Local inflammatory response will stimulate the formation of so called "vulnerable plaque" which is prone to rupture with superimposed thrombus formation. The vast majority of these inflamed high-risk vulnerable plaques cannot be detected by anatomic imaging and myocardial perfusion imaging. Since the progression and development of vulnerable plaque is associated with low wall shear stress, measurement of these characteristics in vivo will enables risk stratification for the entire coronary circulation **[2].** The wall shear stress *(WSS),* wall pressure and blood flow changes in human body cannot be measured directly on blood vessels, Computational fluid dynamics *(CFD)* can provide alternative ways to diagnose *CAD* **[3].** Wall shear stress is Tangential drag force produced by blood moving across the endothelial surface. It is a function of the velocity gradient of blood near the endothelial surface. **[9]** The *WSS* factor in coronary artery plays significant role in the formation of *CAD* **[4].** In addition, the prediction of coronary disease with the normal coronary artery and *WSS* as the local vessels wall demonstrates anatomical section predisposed for atherosclerosis development **[5].** *CFD* allows for efficient and accurate computations of hemodynamic features of both normal and abnormal situations in the cardiovascular system **[3].** *CFD* is different from medical imaging visualization as medical imaging techniques such as coronary angiography or computed tomography angiography provide anatomic alterations of the coronary artery wall due to presence of plaques, thus assessing the degree of lumen stenosis. In contrast, *CFD* analysis enables hemodynamic changes of the coronary artery, even before the plaques are actually formed in the artery wall or occlude the vessels. Therefore, to some extent, *CFD* allows an early detection of coronary artery disease and improves understanding the progression of plaques which are considered paramount importance to clinical treatment. This study aims to describe the variation of *WSS* during entire cardiac phase. The assumption of this is differences in the blood velocity during entire cardiac cycle could affect the blood wall and will Show variable effects on WSS, thus the proposed research will serve the purpose of identifying patients with potential risk of developing coronary artery disease.

2. Materials and Method

2.1. Anatomical details of left coronary artery

The Existing Computed tomography *(CT)* data of left coronary artery anatomy was used in this study to generate the anatomical model.

The left coronary artery *(LCA)* branches consist of the left main stem (LMS), left anterior descending *(LAD)* and left circumflex *(LCX),* as shown in Figure 1. Simulation of different flow velocity during cardiac phase has done in this study.

Figure – 1: LCA branches on 3D CT visualization image ***[6].***

2.2: Measurement of CT volume data

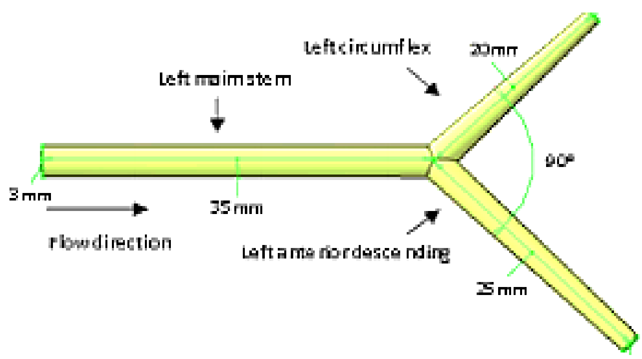

Figure - 2: The 90° model based on the anatomic details ***[6].***

2.3: CFD simulation in left coronary artery

2.3.1: Generation of Mesh Models

The Two-Dimensional models of left coronary were used for generation of mesh models for performance of *CFD* simulation. The mesh models were generated with the ANSYS. Mesh models were saved as *GTM* type to be used for *CFD* computation.

2.3.2: Application of physiological parameters

In order to ensure that analysis reflected the realistic simulation of in vivo conditions, application of the physiological parameters were considered for the Two-dimensional numerical analysis. The transient simulation was performed, and accurate hemodynamic rheological and material properties were used in this study, as referred to studies **[7].** According to Bertolotti et al **[8],** flow rate graph at *LCA* is used. Accurate physiological parameters were applied with a blood density of *1060* kg/m^3, blood viscosity of *0.0035* Pas. The blood flow in wall assumed to be Laminar flow and Blood is assumed to be a Newtonian and incompressible fluid.

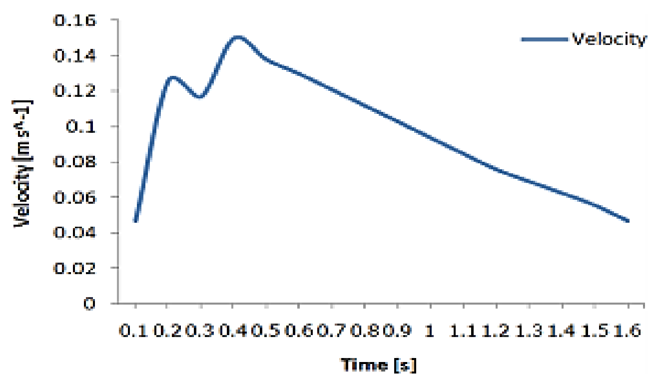

Figure - 3: Pulsatile velocity at left coronary artery ***[6]***

2.3.3: Performance of CFD computation

To ensure that results are valid, the simulation can be performed using *ANSYS.*

2.3.4: Brief about parameters

Wall shear stress *(WSS):* Low value of WSS high chances of plaque progression. **[14]**

Change in pressure *(WPSG):* Reduction in *WPSG* decreased the likelihood of plaque formation **[15].**

Angle: Wide angle high disturbance more chances of plaque progression **[14].**

Viscosity: It represents thickness of blood (ability of blood to flow through the blood vessels). Rate of deformation that fluid experiences when shear stress is applied to it. **[16]**

3. Possible Approach

AS our Aim is To Analyze Wall shear stress value variation, during one cardiac phase (effect during cardiac cycle).Equation can be used is

$$\mathrm{WSS} = \frac{1}{t}\int_0^t \left|\mu \frac{dv}{dn}\right| dt \qquad (1)$$

Where, μ is Blood viscosity, v is Velocity vector near wall perpendicular to surface, ***d*** is Time derivative of local shear stress, n is Distance to the wall surface, t is Pulsatile period.

Local Shear Stress

$$\tau = 32\mu\frac{Q}{\pi d^3} \qquad (2)$$

Where, μ is Blood viscosity, v is Velocity vector near wall perpendicular to surface, ***dt*** is Time derivative of local shear stress, n is Distance to the wall surface, t is Pulsatile period.

To study, lets occlusion percentage of plaque considered is *25%*. (As *50%* and greater lumen stenosis have significant *CAD* **[11]** at the bifurcation of *LCA* (which have high chances of plaque formation).

$$\text{Occlusion percentage [12] } (o) = \frac{R}{D_{lad}} \times 100 \qquad (3)$$

R is radius of minor axis.Here ***R= 0.8*** mm, ***d= 2*** mm, ***o = 25%.***

Figure -4: Frontal view of obstruction **[12]**

The following geometric assumptions should be adopted:

- The bifurcation is located in one Plane bifurcation plane
- The *LM* is a straight tube with a circular cross section,
- The *LCX* and *LAD* are curved tubes with circular cross sections and constant radii of curvatures,
- The curvature planes for the *LAD* and the *LCX* were perpendicular to the Bifurcation Plane.
- As it is difficult to obtain the outer wall boundary of the artery from *CT* images, the vessel wall was artificially constructed with a constant thickness *0.4mm* Although arterial wall is known to be a composite tissue including collagen fibers, its heterogeneous and anisotropic structure properties were simplified by adopting a nine parameter Mooney–Rivlin hyper elastic model due to lack of in vivo data.
- The blood flow distribution in the bifurcation adopts the method of Boutsianis et al, where *71%* is directed through *LAD* and *29%* through *LCx*, and this is maintained unchanged through the entire cardiac cycle. Pulsatile aortic pressure was applied as an inlet boundary condition at the entrance of *LM*, and pulsatile velocity conditions were imposed on both the *LAD* and the *LCx* outlet boundaries. As this study focuses on the *WSS* changes under different branch, global coronary wall motion due to its attachment to the moving myocardium is neglected to isolate the effects of wall compliance. Fine mesh size (*0.15mm*) was chosen to conduct the simulations from accuracy and efficiency points of view. **[13]**

4. Future Work

From the survey we made on various research paper on effect of WSS on plaque progression we understood that more research on effect of WSS on plaque progression can provide us important data which we can use in practical life and can better understand the patient specific need.

5. Conclusion

The main aim of this paper is to provide insight of how plaque progression chances can be analyzed and can be useful for taking precaution and needed treatment. From study we can get accurate values but three dimensional model study could help better and more patient specific plus various properties of wall material can be considered.

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