A 2-D NUMERICAL SIMULATION ON FLOW-INDUCED WALL SHEAR STRESS FOR AN ABDOMINAL AORTIC ANEURYSM MODEL

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Abstract: The aim of this study is to numerically predict the flow-induced WSS for a rigid abdominal aortic aneurysm (AAA) model. Laminar numerical simulations of steady flow of the aneurysm model have been carried out with the variation of dilation ratio (DR) and Re, ranging from 100 to 400. Using CFD software GAMBIT 2.3.16 and FLUENT 6.3.26, variation of WSS has been investigated. It has been observed that the WSS decreases rapidly to zero at the separation point, and maintained a negative value along the aneurysm. Then, the WSS rises sharply to a peak value at the reattachment point. It is also noted that as Re and DR increases, the value of negative WSS decreases and the value of peak WSS increases.

Keywords: Abdominal aortic aneurysm, Steady flow, Computational fluid dynamics, Hemodynamics, Wall shear stress.

Introduction: An abdominal aortic aneurysm (AAA) is a localized, permanent enlargement (dilation) of the infrarenal abdominal aorta more than 1.5 times of the normal size. The main factors which can cause aneurysm are atherosclerosis, genetic disorders, infection, injury etc. Once the weak area forms in the arterial wall, the hemodynamic force try to affect the weaker part of the arterial wall and moves the wall in outward direction. The final effect is the bulging of the artery. In this case, the wall thickness decreases with the increase in the level of disease aneurysm. Moreover, as the age of human being increases, the elasticity of the vessel wall deteriorates. If an aneurysm grows without treatment, the bulge becomes bigger and bigger. Finally, the rupture of the artery would occur. A ruptured aneurysm can lead to uncontrolled bleeding, causing the possible death of the victim. From the review of literatures, it has been observed that a number of researchers have worked on different AAA models. Among them, Zhang *et al.* [1] have carried out a research on numerical and experimental investigation for a pulsatile flow fields in rigid AAA models. They have found that there are one or more vortexes are formed in the AAA bulge and a fairly high WSS exists at the distal end. Both experimental and numerical methods have been used by Bluestein *et al.* [2] to study steady in-vitro flow patterns through a model aneurysm under laminar and turbulent flow conditions. They have revealed that the fluid dynamics characterizing the recirculation zone formed inside the aneurysm cavity create conditions in promoting thrombus formation and the viability of rupture. They have also noted a pronounced WSS peak at the distal end of the aneurysm. In a numerical work, Finol and Amon [3] have considered a two-aneurysm, axisymmetric, rigid wall AAA model to study steady flow field. They have noted that the spatial distribution of Wall Shear Stress Gradient may cause damage to the arterial wall at an intermediate stage of AAA growth. Sheard [4] has performed a numerical simulation in order to determine the flow dynamics and WSS for a pulsatile flow through a human AAA model. He has found that the peak instantaneous WSS for aneurysm dimensions (LR = 2.9, DR = 1.9), is 2.4 times greater than the peak WSS in a healthy vessel. Shupti *et al.* [5] have performed numerical simulation to study the flow behaviour of a pulsatile Newtonian fluid confined within a 2D asymmetric shaped aneurysm. They have found a lower centreline velocity but comparatively higher wall pressure and WSS at the distal neck of the aneurysm. In this present work an effort has been made to numerically predict the flow-induced WSS for a two dimensional, rigid AAA model.

Mathematical Formulation AAA geometry: A rigid AAA model is used in this study, as computational domain. The schematic diagram of the computational domain is illustrated in fig.1. The dilation ratio (DR=D/d) of the model has been varied as 1.5, 2, and 2.5, keeping the aspect ratio (AR=L/d) constant as 2.5. We have also considered the total length of artery with aneurysm as 350 mm and diameter of artery, d=20 mm all the cases.

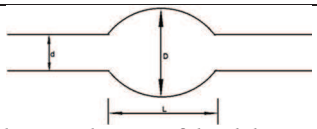

Fig.1: Schematic diagram of the abdominal aortic aneurysm (AAA) model.

Governing Equations:

Assumptions: The flow under consideration has been assumed to be steady, two-dimensional and laminar. Here, the fluid (blood) has been considered to be incompressible. Although blood has actually non-Newtonian behaviour, in the simulation it is considered as Newtonian fluid (Finol and Amon [2]).

Under this assumption the continuity and Navier-Stokes equations can be written as follows:

Continuity equation:

$$\nabla \vec{V} = 0 \qquad (1)$$

Momentum equation:

$$\rho(\vec{V}.\nabla \vec{V}) = -\nabla p + \mu \nabla^2 \vec{V} \qquad (2)$$

Where, $\vec{V}$ is velocity field, p is pressure, ρ is density, μ is the coefficient of dynamic viscosity.

Boundary Conditions: Three different types of boundary conditions have been applied to the present problem. They are as follows, (i) At the walls: No slip condition, i.e. velocity component at the wall is zero. (ii) At the inlet: Axial velocity has been specified and the transverse velocity has been set to zero and flow considered to be a fully developed. (iii) At the exit: pressure outlet has been specified.

Numerical Procedure: The geometry and mesh of the computational domain have been made in commercial software GAMBIT 2.3.16 which is the pre-processor for FLUENT. The Nevier-Stokes equation and the continuity equation are solved using the commercial CFD software FLUENT 6.3.26 that employs the control volume technique on a uniform staggered grid following SIMPLE algorithm. The convergence of the iterative scheme is achieved when the normalized residuals of mass and momentum equations summed over the entire calculation domain fall below 10^{-5}.

Results and Discussion: In this work, the effect of DR and Re on the flow-induced WSS has been investigated. The Re has been varied from 100 to 400 and the inlet velocity distribution has been considered to be fully developed.

Variation of Wall Shear Stress: Fig. 2(a), fig. 2(b) and fig. 2(c) show the effect of flow Re on WSS for DR 1.5, 2 and 2.5 respectively. For all the cases, the WSS decreases rapidly to zero at the separation point, and maintained a negative value, because of the flow reversal in the recirculation zone along the aneurysm. Then, the WSS rises sharply to a peak value at the reattachment point. It is also noted that as Re increases the value of negative WSS decreases and the value of peak WSS increases. This agrees with results by Shupti *et al.* [5].

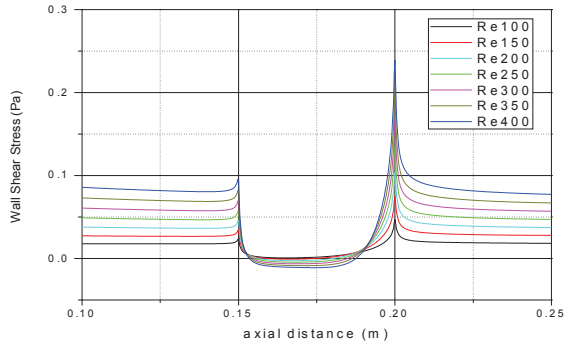

Fig. 2(a): Variation of WSS with the variation of Re for DR=1.5.

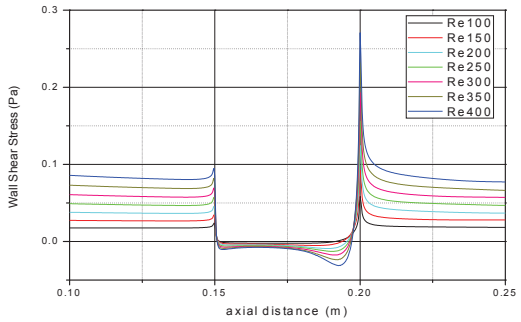

Fig. 2(b): Variation of WSS with the variation of Re for DR=2

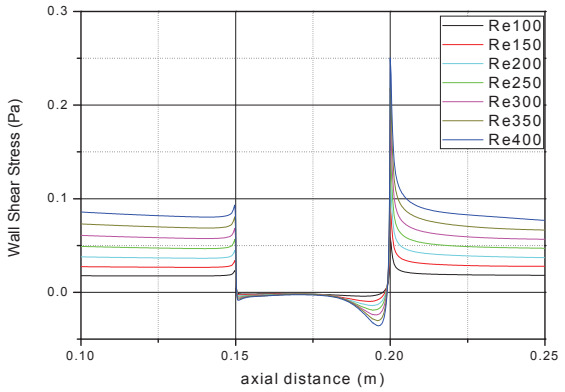

Fig. 2(c): Variation of WSS with the variation of Re for DR=2.5

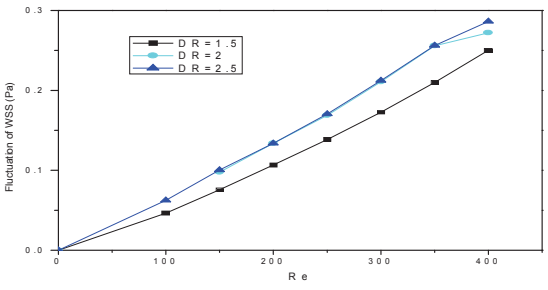

Fig. 2(d): Fluctuation of WSS with the variation of DR for different Re.

Fig. 2(d) shows the fluctuation of WSS with the variation of DR at distal end for different Re. It has been observed that the fluctuation of WSS increases with the variation of DR for different Re. This mechanism can be explained by the fact that due to convective deceleration of the flow, the velocity decreases in the proximal half of the aneurysm, which results in a decrease in the WSS to a zero value at the point of separation. Similarly, the WSS value at the distal end is also zero because of the site of boundary

layer reattachment. The WSS value increases sharply because the velocity increases sharply after the point of reattachment, due to the convective acceleration. So, the vicinity of the reattachment point is a site of localized low negative and high positive WSS for all the Re. The elevated level of WSS may lead to damage of endothelium layer of the distal aneurysm wall (Sheared [4]). Due to low WSS, the platelets lead to a prolonged contact with the wall, promoting adhesion to the wall (Bluestin *et al*.[2]).

Conclusion: In the present work, a numerical study on flow-induced WSS for a rigid AAA model has been carried out. The effect of important parameter like flow Re and DR have been investigated. The Re is in the range of 100 to 400 and dilation ratio of 1.5, 2 and 2.5 are considered. It leads to some important observation. For all the cases, the WSS value is negative along the aneurysm. Then, the WSS rises sharply to a peak value at the reattachment point. It is also noted that as Re increases, the value of negative WSS decreases and the value of peak WSS increases. The elevated level of WSS may lead to damage of endothelium layer of the distal aneurysm wall. Due to low WSS, the platelets lead to a prolonged contact with the wall, promoting adhesion to the wall.

Nomenclature

Symbol	Meaning	Units
DR	Dilation Ratio	_
AR	Aspect Ratio	_
Re	Reynolds number	–
$\vec{V}$	Velocity field	ms^{-1}
P	Pressure	Pa
ρ	Density	$kg\ m^{-3}$
μ	Coefficient of dynamic viscosity	$kg\ m^{-1}s^{-1}$
WSS	Wall shear stress	Pa
CFD	Computational fluid dynamics	_

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