NON-NEWTONIAN FLUID FLOW SIMULATION IN A PROGRESSIVELY ENLARGED ANEURYSM MODEL

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Summary The current study is focused on the numerical investigation of the flow field induced by the unsteady flow in the vicinity of an abdominal aortic aneurysm model using a recently-developed Computational Fluid Dynamics code. For modelling the rheological behaviour of blood, the Quemada non-Newtonian model is employed, which is suitable for simulating the two-phase character of blood. Furthermore, the investigation is focused on the distribution of the flow-induced forces on the interior wall by assuming three different degrees of aneurysm-growth. Finally and for examining the effect of the distribution on the aneurysm growth, a comparison is made between the pressure and wall shear-stress distributions at the wall for each growth-degree.

INTRODUCTION

The disturbances in the flow field within the circulatory system caused by the irregular geometry of an abnormality can affect the severity of the abnormality itself. Particularly in the case of an Abdominal Aortic Aneurysm (AAA) the abrupt expansion in the geometry induces the recirculation of the flow within the aneurysm. This, in conjunction with the periodicity of the flow rate, causes unusual fluctuations of the pressure applied on the vascular wall which is already tensed and weakened due to the enlargement. In the current paper, the flow field within 3D models of axisymmetric aneurysm with different growth degrees is investigated by employing the Quemada constitutive equation [1] due to the fact that – to the best of the authors' knowledge – a blood constitutive equation was never used for this type of flow although there are generally many studies on the field of biomechanics of AAAs. This is considered as necessary given the fact that the blood flow field when using Newtonian modelling for blood differs markedly from when using non-Newtonian modelling. Therefore, the aim of the current study is twofold: (i) accurate representation of the flow field and wall stress distribution within an AAA during the cardiac cycle; (ii) accurate assessment of the most stressed region of the vascular wall.

METHOD

Due to the laminar, incompressible and unsteady nature of the flow under investigation, a code developed for solving the Navier-Stokes equations for incompressible flow in integral form is used. The code incorporates the SIMPLE scheme in conjunction with collocated arrangement of variables. The approximation of the convection terms is carried out using the QUICK differencing scheme, whereas the code enables also multi-block computations, which are useful in order to cope with the two-block grid structure of the current computational domain. The relation between the shear stress and shear rate in tensorial form in the Navier-Stokes equations is given by $\bar{\tau} = \mu(|\bar{\gamma}|)\bar{\gamma}$ where $\bar{\tau}$ is the shear-stress tensor, $\bar{\gamma}$ is the shear-rate tensor and μ is the viscosity. For the Quemada model, which is a model developed for simulating the rheological behaviour of concentrated disperse systems, μ is given by $\mu = \mu_F \left(1 - \frac{1}{2}\frac{k_o + k_\infty \sqrt{|\bar{\gamma}|/\gamma_c}}{1+\sqrt{|\bar{\gamma}|/\gamma_c}}\varphi\right)^{-2}$ where μ_F is the viscosity of plasma (suspending medium), φ is hematocrit and γ_c, k_∞ , k_o are parameters determining the rheological behaviour of the suspension. The geometry consists of a tube divided in three segments namely the inlet segment, the aneurysm segment and the outlet segment. The radius within the aneurysm segment is given by a circle-arc function. The grid used in the computations is based on multiblock structure and is denser with respect to the longitudinal direction at the aneurysm area in order to capture the flow variations in greater detail. The inner block is of a rectangular cross section and is encircled by the outer block as shown in Figure 1(i). As inlet boundary condition, the flow rate waveform in the abdominal artery is used representing a typical flow condition derived from i*n vivo* measurements [2] and is shown in Figure 1(ii).

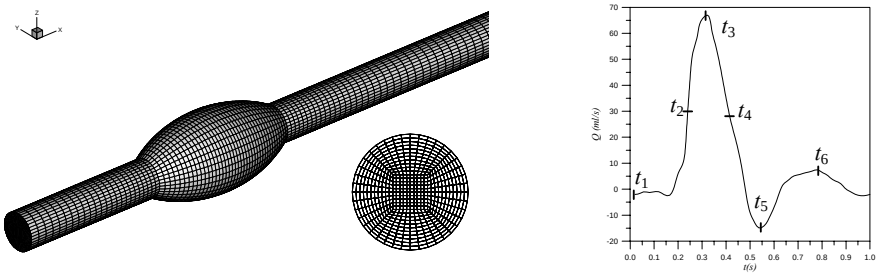

Figure 1: (i) 3D grid with cross section; (ii) Inlet flow rate $Q(ml/s)$ over a period.

RESULTS

The flow field in the vicinity of the aneurysm for all three growth-degrees considered is shown in Figure 2(i) for various time instants that correspond to different phases of the cardiac cycle, namely t_1 – late diastole, t_2 – systolic acceleration, t_3 – peak systolic flow, t_4 – systolic deceleration, t_5 – early diastole/peak retrograde flow, t_6 – peak diastolic flow (Fig. 1(ii)). At $t=t_2$ acceleration causes vortices formed from retrograde flow during late diastole to vanish. At $t=t_3$, the flow rate is the maximum throughout the cardiac cycle and acceleration of the flow ceases. At this stage a vortex starts to form at the beginning of the aneurysm section, which is progressively bigger for larger aneurysm degrees. At $t=t_4$, the flow rate gradually drops whereas the deceleration causes increase of the vortex size. The vortex grows even further at $t=t_5$ and covers the whole aneurysm area for all growth-degree cases. This vortex later produces a counter rotating vortex which splits the main vortex into two corotating vortices with the counter rotating vortex lying between the other two ($t=t_6$). As the cycle reaches late diastole ($t=t_1$) the flow again decelerates thus inducing vortex formation. Due to the occurrence of several prominent peaks at the distal side of the aneurysm, the pressure levels at this point are monitored throughout the cycle as shown in Figure 2(ii). The variation does not seem to differ between the aneurysm-degree cases; however the peaks occurring are slightly more prominent than in a normal subject i.e. without aneurysm. Due to the seemingly high strain at the aforementioned location, the variation of the wall shear-stress (WSS) at the same point is also monitored and is shown in Figure 2(iii). It can be seen that the peak at the systolic phase is much higher in a normal vessel than in vessel with an aneurysm. However, the minimum values occurring during the diastolic phase are much more prominent for aneurysm cases than for normal conditions.

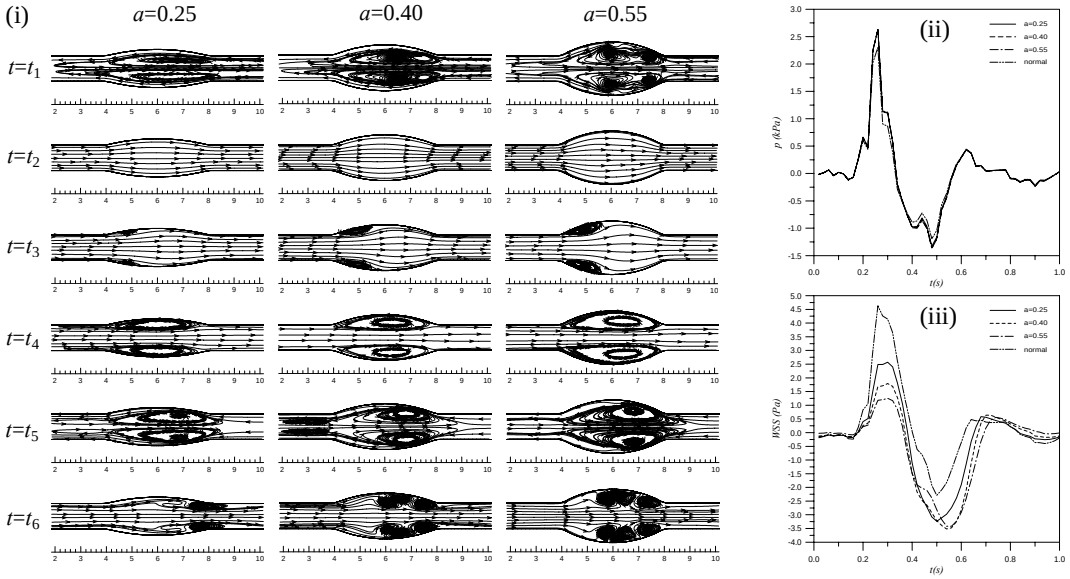

Figure 2: (i) Instantaneous streamlines for various time instants and for the three different growth degrees; Variation at the distal end of the aneurysm wall throughout the period cycle of (ii) the pressure and (iii) the shear-stress.

DISCUSION

The most stressed region in terms of pressure levels seems to be the distal wall of the aneurysm on which intense flow impingement occurs due to the sudden enlargement of the cross-section and the consequent expansion of the flow. These findings are in agreement with Hoi *et al.* [3] that argue that flow impingement induces further degradation and stretch of the aneurysm. The explanation for this begins with the fact that endothelial cells respond to elevated WSS by releasing vasodilation factors to enlarge the tube diameter so as to return the WSS to baseline levels. These factors degrade the extracellular matrix and dilate the arterial wall by relaxing smooth-muscle cells in the flow impingement zone [3]. The absence or disorganisation of key extracellular matrix components decrease the mechanical strength of the aneurysm wall and in conjunction with the elevated pulsatile pressure, renders the aneurysm wall more susceptible to dilation. Hoi *et al.* [3] also argue that as the wall continues to degrade and be stretched from the distal end of the aneurysm, healthier portions of the parent artery become incorporated into the aneurysm. Therefore, the increased pressure and high WSS levels at this may lead to subsequent enlargement, elongation and possible rupture of the aneurysm.

References

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