ENDOVASCULAR WALL PRESSURE-FORCES 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. As inlet boundary condition, the realistic flow rate waveform in the abdominal aorta is used. For modelling the rheological behaviour of blood, the Quemada non-Newtonian model is employed. In view of the haemodynamical mechanisms related to the enlargement and subsequent rupture of an aneurysm, the investigation is focused on the distribution of the flow-induced pressure on the interior wall of the aneurysm. In order to study the development of the distribution with the gradual enlargement of the aneurysm, three different degrees of aneurysm-growth have been assumed. Finally and for examining the effect of the distribution to the aneurysm growth, a comparison is made between the pressure-distributions for each growth-degree and also the areas of maximum strain on the arterial wall are identified namely the areas of the highest pressure and highest pressure-variation.

1 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 endovascular wall which is already tensed and weakened due to the enlargement. The investigation of the correlation of various flow effects in the vicinity of an AAA has be the aim of several research studies.

The study by Finol and Amon [1] focused on the numerical predictions of blood flow patterns and hemodynamic stresses in AAAs employing a realistic aortic blood flow simulated under pulsatile conditions. Their results mainly concern the wall shear stress distribution on a 2D model of multiple aneurysms and illustrate the importance of considering time-dependent flow for the evaluation of hemodynamic indicators. In a similar study concerning multiple aneurysms, Kumar [2] investigated the flow field within an arterial vessel model with two asymmetric aneurysm models under pulsatile flow conditions. Results show the influence of the Reynolds number (Re) and Strouhal number (Str) on the wall shear stress (WSS) and wall pressure and conclude that while the WSS increase with increasing either Str or Re, the wall pressure seems to get lowered. In another study Finol *et al.* [3] deal with the effect of the asymmetry in the geometry of an AAA by numerically investigating the pulsatile flow in a 3D model. In their results they argue that the effect of the asymmetry is to increase the maximum wall shear stress at peak flow and to induce the appearance of secondary flows in late diastole. Numerical investigation is also carried out by Viswanath *et al.* [4] for studying the flow field in axisymmetric AAAs using the actual pressure-velocity pulse as the abdominal aorta as the inlet boundary condition. The analysis given by this study confirms that the mechanical forces on the arterial wall, developed by the blood flow, may play an important role in both the development and growth of aneurysms.

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 [5] for more accurately simulating the rheological properties of blood. Whereas in a previous study [6] only steady flow is concerned, the current study employs a realistic flow rate waveform as the inlet flow condition so as to investigate the subsequent pressure variations over the interior wall of the aneurysm.

2 METHODOLOGY

Due to the laminar, incompressible and unsteady nature of the flow under investigation, the Navier-Stokes equations for unsteady, incompressible flow in integral form are used as given in [6]. For Newtonian flows the viscosity μ is constant whereas for non-Newtonian flows, as in the current study, it is expressed as a function of the second invariant of $\bar{\gamma}$. For the Quemada model, which is a model developed for simulating the rheological behaviour of concentrated disperse systems, μ is given by

$$\mu = \mu_F \left(1 - \frac{1}{2} \frac{k_o + k_\infty \sqrt{|\bar{\gamma}|/\gamma_c}}{1 + \sqrt{|\bar{\gamma}|/\gamma_c}} \varphi \right)^{-2} \tag{1}$$

where μ_F the viscosity of plasma (suspending medium), φ is haematocrit and γ_c, k_∞ , k_o are parameters determining the rheological behaviour of the suspension [5].

The geometry consists of a tube of diameter D and can be divided in three segments namely the inlet segment with length $4D$, the aneurysm segment with length b=$4D$ and the outlet segment with length $18D$. The radius R_0 of the inlet and outlet segments is undeformed and equal to $D/2$, whereas the radius of the deformed segment is given by

$$R = R_0 + \left(a - R_c + \sqrt{R_c^2 - [b/2 - x]^2} \right), \; 0 \le x \le b \tag{2}$$

where $R_c = \dfrac{a^2 + (b/2)^2}{2a}$ and a is the maximum width of the dilated segment. Both edges of the aneurysm are smoothed following a function of cycle-arc for which w=0.12 as shown in Figure 1. For the current study, three different values of a were used namely 0.25, 0.4 and 0.55.

As inlet boundary condition, the flow rate waveform in the abdominal artery is used [7] and is shown in Figure 2. At the outlet the pressure is constant and equal to 0 whereas at the walls the non-slip condition is applied. Based on measurements [7] showing that the radius of the abdominal artery is R_0=0.63cm the mean velocity over the period and cross section would be u_m=8.2cm/s.

The parameters of the flow are the Reynolds (Re) and Strouhal (Str) numbers defined as

$$\mathrm{Re} = \frac{\rho u_m D_0}{\mu}, \; \mathrm{Str} = \frac{D_0}{u_m T} \tag{3}$$

where D_0=$2R_0$,T=1s and μ is taken as the viscosity for infinite shear rate ($\bar{\gamma} \to \infty$) from (1). According to these, Re=352 and Str =0.154.

3 RESULTS AND DISCUSSION

The flow field in the vicinity of the aneurysm for all three growth-degrees considered is shown in Figure 3 for various time instants. These instants correspond to different phases of the cardiac cycle, namely t_1=0 (late diastole), t_2=0.24 (systolic acceleration), t_3=0.32 (peak

systolic flow), t_4=0.42 (systolic deceleration), t_5=0.54 (early diastole – peak retrograde flow), t_3=0.78 (peak diastolic flow).

At the systolic acceleration phase ($t=t_2$) ventricle contraction occurs thus forcing the flow to accelerate. Acceleration causes vortices formed from retrograde flow during late diastole to vanish. The aneurysm degree does not seem to affect the flow field at this stage as can be seen from the flow patterns that are the same for all three aneurysm degrees. At peak systolic flow ($t=t_3$) 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. In the systolic deceleration phase ($t=t_4$) the flow rate gradually drops whereas the deceleration causes increasing of the vortex size as also observed in the past [9]. The vortex grows even further at early diastole where peak retrograde flow occurs ($t=t_5$) and covers the whole aneurysm area for all growth-degree cases. It can be seen that for higher growth degrees the vortex-centre is located further to the distal end of the aneurysm. This vortex later breaks into two corotating vortices (eddy breaking) one of which produces a counter rotating vortex (eddy doubling) lying between the other two ($t=t_6$). This phenomenon is more prominent for larger growth-degrees whereas the core flow formed during acceleration at early diastole and reaching a maximum at $t=t_6$ causes the vortices to move downstream. As the cycle reaches late diastole ($t=t_1$) the flow again decelerates thus inducing vortex formation. The aneurysm growth seems to play a more important role in the fluid structure at this stage due to the fact that larger growth degrees seem to play a catalytic role in eddy-breaking and consequent existing of two vortices instead of only one within the aneurysm. Subsequent flow acceleration at early systole causes ejecting of the vortices from the aneurysm towards the downstream end of the model and the flow is thus reattached to the walls.

A direct comparison at each time instant between aneurysm-growth cases is shown in Figure 4 for the selected time instants as shown in Figure 2. In agreement with the flow field observations, the systolic deceleration phase is dominated by a steep pressure gradient which forces the flow to accelerate ($t=t_2$). At the next stage ($t=t_3$) where maximum flow rate occurs, the pressure gradient is lower whereas the pressure on the aneurysm wall is increased. This increase is more prominent for larger growth degrees (Fig. 4iii). The flow then decelerates during late systole ($t=t_4$) owing to the negative pressure gradient (in the sense that it causes retrograde flow) and the pressure distribution barely differs between the growth-degree cases (Fig. 4iv). The pressure gradient remains negative at early diastole ($t=t_5$) but it is now lower and there is a marked negative peak occurring at the aneurysm wall at around x=7 (Fig. 4v). This seems to be caused by the acute impingement on the wall subsequent to the recirculation of the flow at this particular point where the vortex appears to be closest to the wall. At the phase of peak diastolic flow ($t=t_6$) the pressure gradient again changes sign and the distribution exhibits a rapid fluctuation in the vicinity of the aneurysm (Fig. 4vi) that is more marked for larger growth degrees and is caused by the presence of multiple vortices in the aneurysm. Finally, at the late diastolic phase the pressure gradient remains negative and is the lowest in absolute compared to the other instants studied. The fluctuations in the aneurysm region are still present owing to the complex structure of the flow field that is caused by accumulation of transient effects from the middle of the cycle.

4 CONCLUSIONS

The flow field and pressure distributions in a vascular model with various aneurysm-growth degrees are studied by means of computational fluid dynamics. The results show that vortex forming is dominant in the aneurysm throughout the cardiac cycle except during the early systolic phase in which rapid acceleration of the flow occurs. Furthermore, it seems that larger growth degrees favour the process of eddy-breaking and eddy-doubling thus causing a

more complex flow field. This reflects on the pressure distribution in the form of fluctuations that occur in the wall pressure distribution. These fluctuations are more prominent at specific cycle phases where either positive or negative peaks take place inside the aneurysm wall. 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. The increased pressure variations at this point in conjunction with the already tensed and weakened endovascular wall may lead to subsequent enlargement and possible rapture of the aneurysm.

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FIGURE CAPTIONS

Figure 1. Geometrical model of the vessel with the aneurysm.
Figure 2. Inlet flow rate over a period
Figure 3. Instantaneous streamlines for various time instants and for the three different growth degrees.
Figure 4. Pressure distributions for the three growth degrees and for (i) t=0 (t_1); (ii) t=0.24 (t_2); (iii) t=0.32 (t_3); (iv) t=0.42; (t_4); (v) t=0.54 (t_5); (vi) t=0.78 (t_6).