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FLUID-STRUCTURE INTERACTION IN A STENTED ABDOMINAL AORTIC ANEURYSM: EFFECTS OF ENDOLEAKS

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ABSTRACT

The treatment of aortic aneurysms with endovascular graft (EVAR) has become the preferred alternative to treat this condition in patients with a suitable geometry [1]. Although EVAR is attractive as a less invasive procedure, a complication termed endoleak may occur: this is the persistence of pulsatile flow in the sac of the excluded aneurysm. Endoleaks result from either poor mechanical seal between the endograft and the wall of the aorta (type 1) or retrograde flow into the sac from a branch taking origin from it (type 2). Type-1a endoleaks occur at the proximal attachment, type-1b at the distal one. It has been shown that endoleaks result in continuous expansion and eventual rupture of aneurysms [2-3]. Parodi et al. [2] developed an experimental in vitro circulatory model to analyze hemodynamic changes within the aneurysm sac in the presence of endoleaks of different sizes. The walls of the aneurysm were assumed to be rigid. Their results showed that the intrasac and mean diastolic pressures were significantly higher than systemic diastolic and mean pressures. This explains the continuous expansion of an incompletely excluded aneurysm. The most threatening endoleak is the one designated as type 1 where there is a gap in the attachment of the endograft to the native aorta. The objective of this study is to investigate in a computational fluid-structure interaction (FSI) model the effects of wall flexibility in a type-1 endoleak occurring in an aneurysm treated by EVAR. We also compared the flow patterns, wall stress and deformation values between FSI and rigid wall aneurysm models.

MATHEMATICAL FORMULATION

The blood velocity and wall shear stress distribution in the aortic aneurysm (AA) are computed by solving the Navier-Stokes equations on the deforming fluid motion of the AA model based on the arbitrary Lagrangian-Eulerian (ALE) method. The endovascular graft is assumed to be a thin uniform shell and its migration was not considered. To determine the leakage transients and evaluate their consequences, we created a 1.5 mm diameter “leak”. Physiologic inflow velocity and outlet pressure waveforms were used. Shear thinning behavior is incorporated by making the viscosity dependent on the shear rate according to the Carreau model.

RESULTS AND CONCLUSIONS

A finite element formulation based on the Galerkin method was utilized to solve the governing equations. A sufficient number of cycles were performed numerically to overcome the transient period until convergence was achieved when the flow became time periodic. The effect of a type-1 endoleak on the velocity field within the aneurysm was analyzed for rigid and flexible walls. There were significant flow field differences between both models.

For the coexistence of type-1a and type-1b, Figure 1 shows that the temporal variation of the leakage rates (m/s) at the proximal and distal ends are identical in the rigid wall model. However, when we introduced a flexible wall in the model as shown in Figure 2, flow occurred into the sac during peak systole from both the type-1a and type-1b leaks (Figures 1&3) which can cause high wall stress. In the presence of type-1 endoleaks, the AA wall stress ($\sigma_{Von\ Mises} = 32\,N/cm^2$) is similar to that of a non-stented AA ($\sigma_{Von\ Mises} = 33\,N/cm^2$) and poses the same risk of rupture. In other words, an aneurysm that has been imperfectly excluded because of a type-1 endoleak encounters the same stresses and outcomes as an untreated aneurysm

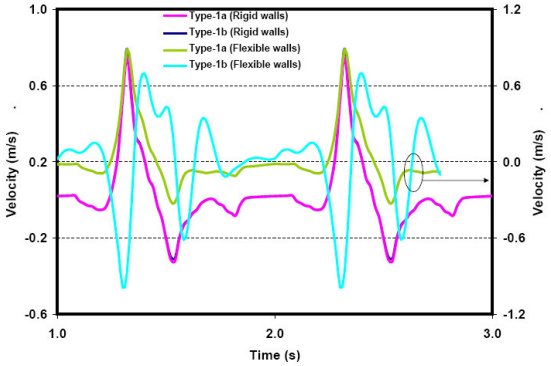

Figure 1. Temporal variation of the leakage rate (m/s) at the proximal end (type-1a) and distal end (type-1b) in rigid wall and flexible wall models

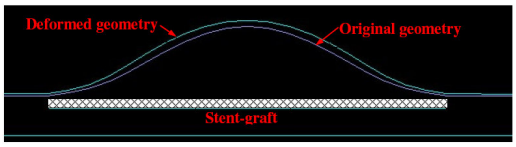

Figure 2. Wall displacement at peak systole

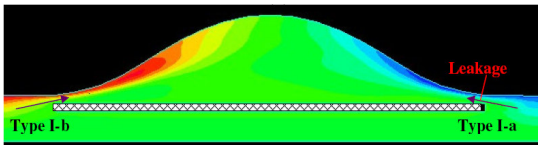

Figure 3. Velocity distribution in axisymmetric aortic aneurysm with flexible walls at peak systole

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