Intraluminal thrombus and risk of rupture in patient specific abdominal aortic aneurysm – FSI modelling

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Recent numerical studies of abdominal aortic aneurysm (AAA) suggest that intraluminal thrombus (ILT) may reduce the stress loading on the aneurysmal wall. Detailed fluid structure interaction (FSI) in the presence and absence of ILT may help predict AAA rupture risk better. Two patients, with varied AAA geometries and ILT structures, were studied and compared in detail. The patient specific 3D geometries were reconstructed from CT scans, and uncoupled FSI approach was applied. Complex flow trajectories within the AAA lumen indicated a viable mechanism for the formation and growth of the ILT. The resulting magnitude and location of the peak wall stresses was dependent on the shape of the AAA, and the ILT appeared to reduce wall stresses for both patients. Accordingly, the inclusion of ILT in stress analysis of AAA is of importance and would likely increase the accuracy of predicting AAA risk of rupture.

Keywords: intraluminal thrombus; abdominal aortic aneurysm; FSI modelling; risk of rupture

1. Background

Abdominal aortic aneurysm (AAA) represents a common vascular pathology with possibly fatal implications. AAA is an irreversible dilation, i.e. ballooning, of an artery due to gradual wall weakening. As the aneurysm expands, it may eventually rupture. In the current clinical management of AAA patients, the maximum transverse dimension of the aneurysm is often used as the primary indicator of potential for rupture. Decisions to repair asymptomatic AAAs are currently based on diameter (5.5 cm) as a sole predictor of rupture, along with consideration of the risks associated with undergoing repair. Accordingly, repair is warranted when the risk of rupture exceeds that of the repair as to justify an elective surgical resection (Raghavan and Vorp 2000; Fillinger et al. 2002). As there exist aneurysms smaller than 5.5 cm that do rupture and larger ones that remain intact, there is a need to more accurately predict risk of rupture based on patient specific parameters other than diameter alone. Because frequent observation and a low threshold for intervention will not prevent all ruptures, further refinement of the ability to predict AAA rupture risk is essential.

Current advances in imaging and software facilitate extraction of patient specific 3D data of human anatomy from clinical visualisation modalities that are commonplace in the modern clinical practice. These imaging techniques can provide the basis for applying computational fluid dynamic (CFD) and finite elements analyses to patient specific geometries. Recently, such patient based CFDs models were reconstructed from medical images (Vorp et al. 1996a; Di Martino and Vorp 2003; Fillinger et al. 2003; Steinman et al. 2003), currently incorporating fluid structure interaction (FSI) modelling (Wolters et al. 2005; Leung et al. 2006; Papaharilaou et al. 2006), paving the way to use these techniques for diagnosis, surgical planning and follow up.

AAA may be filled with intraluminal thrombus (ILT; Mower et al. 1997; Vorp et al. 1998; Schurink et al. 2000; Wang et al. 2002; Di Martino and Vorp 2003; Thubrikar et al. 2003). The effect ILT may have on the risk of rupture is debated. There has been an investigation on the pressure within the aneurysmal thrombus compared to the systemic pressure (Schurink et al. 2000). Results indicated that thrombus within the aneurysm did not reduce the mean and the pulse pressure near the aneurysmal wall, and as such the authors concluded that the thrombus will not reduce the risk of rupture of the aneurysm. Vorp et al. (1996a, 1996b, 2001) indicated that the presence of ILT alters the normal pattern of oxygen supply to the AAA wall. This hypoxic condition may lead to cell dysfunction through expressing extracellular matrix degrading factors, which may further lead to wall weakening and increased potential for rupture (Vorp et al. 1998, 2001; Kazi et al. 2003). However, parametric studies of well-organised thrombus of clearly defined shape (Di Martino and Vorp...
2003) and additional studies (Mower et al. 1997; Di Martino and Vorp 2003; Thubrikar et al. 2003) indicated that such thrombus reduces the effect of the pressure load on the aneurysmal aortic wall. The presence of the thrombus, the mechanical properties of the thrombus and the eccentricity of the patient lumen influence the aortic wall stress distribution (Mower et al. 1997; Wang et al. 2002; Di Martino and Vorp 2003; Thubrikar et al. 2003). The relatively constant area of ILT over cardiac cycles indicated the incompressibility and isotropic nature of this tissue, suggesting a possible ‘mechanical cushioning effect’ of the ILT (Vorp and Vande Geest 2005). Thubrikar et al. (2003) found that even though thrombus allows luminal pressure to transfer to the wall, it prevents aneurysm rupture by reducing the strain on the wall. Other experimental studies (Wang et al. 2002), including one from Vorp’s group (2003), have shown that ILT reduces peak wall stress, indicating that while the long-term presence of ILT on rupture is harmful, the immediate effects are beneficial (Leung et al. 2006).

Recent numerical studies of the AAA wall stress calculation were based on patient specific image reconstruction, but assumed uniform pressure inside the AAA (Fillinger et al. 2002, 2003) and did not account for flow induced stresses within the AAA, and for the different flow patterns in the presence of ILT. Recent numerical studies accounted for the interaction between the blood and the vessel wall of the AAA (Li and Kleinstreuer 2005a, 2005b, 2005c; Wolters et al. 2005; Leung et al. 2006; Papaharilaou et al. 2006). In addition, these studies have indicated that a subset of small aneurysms have an unexpectedly high wall stresses and are at a higher risk of rupture than would have been predicted by traditional means.

In this paper, we present a patient specific analysis of an uncoupled FSI between the fluid flow and the AAA wall using a fluid solver (Fluent Inc., Lebanon, NH, USA) and a solid solver (ABAQUS Inc.) for the ILT and wall domains. For the flow domain, the inlet flow at the proximal aorta was applied as uniform steady flow of 0.4 m/s, representing typical physiologic peak flow conditions during the cardiac cycle. The fluid was assumed laminar and Newtonian with a density of 1050 kg/m^3 and viscosity of 0.004 kg/m per second, representing human blood properties at 37°C.

2.2. Numerical methods
An uncoupled interaction between the blood flow and the AAA wall was applied to study, the details of the blood flow domain and its interaction with the wall mechanics, using a fluid solver (Fluent Inc.) for the flow domain and a solid solver (ABAQUS Inc.) for the ILT and wall domains. For the flow domain, the inlet flow at the proximal aorta was applied as uniform steady flow of 0.4 m/s, representing typical physiologic peak flow conditions during the cardiac cycle. The fluid was assumed laminar and Newtonian with a density of 1050 kg/m^3 and viscosity of 0.004 kg/m per second, representing human blood properties at 37°C.
The pressure distribution resulting from the flow calculation was superimposed on a uniform systolic pressure of 120 mmHg (\(16\) kPa) and was applied as boundary conditions for the vessel wall calculation using ABAQUS. The material for characterising the AAA wall was assumed to be hyperelastic, isotropic, incompressible and homogenous, using the constitutive model proposed by Raghavan and Vorp (2000). The strain–energy function is given as:

\[
W = a(I_B - 3) + \beta(I_B - 3)^2,
\]

where \(W\) is the strain–energy density, \(\beta\) is the left Cauchy–Green tensor and \(I_B\) is the first invariant of the left Cauchy–Green tensor \(B\); \(a\) and \(\beta\) are the model parameters indicative of the mechanical properties of the AAA wall. The model parameters were set to \(a = 17.4\) N/cm\(^2\) and \(\beta = 188.1\) N/cm\(^2\), corresponding to population mean values obtained from uniaxial loading tests on excised AAA wall specimens (Raghavan and Vorp 2000). The ILT was modelled as an incompressible, isotropic, homogenous, linear elastic material with Young modulus \(E = 0.11\) MPa and Poisson ratio \(\nu = 0.45\), representing population mean values obtained from uniaxial loading tests performed on ILT specimens harvested during AAA surgery (Di Martino and Vorp 2003; Vorp and Vande Geest 2005). In order to estimate the effect of ILT inclusion on stress distribution within the aneurysmal wall, two analyses were carried out for each patient. One simulation was performed with the 3D reconstructed ILT included in the geometry, while in the other simulation the ILT was excluded.

2.2.1. Mesh convergence study

The computational grid, generated from first-order tetrahedral elements (C3D4) which are compatible with Fluent’s package finite volume approach, was created with Gambit. Fluent was then used to create an ABAQUS input file with first-order elements (C3D4). By means of an in-house developed mesh converter the mesh was subsequently converted to one that used second-order ‘modified’ elements (C3D10M). These second-order elements allow for more accurate stress/displacement calculations with ABAQUS. Specifically, the elements used were hybrid type elements, as required for achieving convergence with the hyperelastic material model used for the AAA wall properties (ABAQUS 2004).

A mesh convergence study was performed on the AAA without ILT (first patient). Stress/displacement analyses with coarser and finer meshes showed negligible error, when comparing the von Mises stress and total displacement patterns. The resulting mesh that guaranteed mesh density independence contained 29,558 elements for the arterial wall.

3. Results

The reconstructed AAA wall and ILT surface that were used for the numerical simulations are depicted in Figure 2 for each of the two subjects. These figures show the reconstructed CT images of Figure 1 using the methods described above. The AAA cases in Figure 2 are shown using cross-sectioning to reveal the ILT shape (The ILT is shown in red in Figures 1 and 2, and the lumen in yellow). The ILT in the first AAA occupied a significant part of the aneurysm volume. The second AAA ILT mostly occupied the upper half of the aneurysm.

Pathlines and wall shear stresses (WSS) in both AAA studied are depicted in Figures 3 and 4. Results are shown in right and/or left lateral views. We have chosen to show only the views, which depicted relevant differences in the results within the specific patients for the AAA with and without ILT. The highest shear stress values were found on the anterior side of the AAA’s (as indicated in the Figures 3 and 4). Significant differences are noted when comparing for each patient the case with or without the ILT. Those are evident in the differing flow patterns (as indicated by the pathlines) and the wall shear stress distributions with their apparent different zones of maximal values (as indicated by arrows in Figures 3 and 4). Significant differences were also found in the maximal shear stress levels between the two AAA cases. The shear stress levels on the anterior side of the AAA was in the range of 10–20 N/m\(^2\) for patient 1, while 70–80 N/m\(^2\) for patient 2. In both AAAs, with and without ILT, a large amount of particles were recirculating along complex flow trajectories with a long residence time within the AAA (Figures 3(a) and 4(a)). The deposition
patterns resulting from such flow recirculation regions offer a viable mechanism for the formation and growth of the observed ILT in both AAA’s (Bluestein et al. 1996), although, it must be cautioned that as the ILT progresses the recirculating flow patterns are bound to change. The overall shear stress distribution patterns are similar, with low wall shear stress values within the AAA and higher values on the wall opposite to the AAA itself.

Von Mises stress distributions are shown in Figures 5 and 6 for each AAA with and without ILT. In order to visualise the von Mises stress pattern on the inner side of the AAA wall, the AAA’s were cross-sectioned by lateral (sagittal) planes, with both sides (left and right lateral view) of the AAA’s shown. To determine the effect of the ILT on the von Mises stress distribution, the numerical results of the patients with ILT are depicted with and without the ILT. For the first AAA, the peak stress was about 70 N/cm² without ILT and 57 N/cm² with ILT. For the second AAA, peak wall stress reduced from about 40 N/cm² without ILT, to 30 N/cm² with ILT. The magnitude of von Mises stress within the ILT itself was much lower than the stress within the wall, with peak stresses values reaching 5–5.5 N/cm², for the first and second patient, respectively.

4. Discussion
The numerical study presented here provides detailed results from patient specific reconstructed AAA geometries,
including the effects of ILT, based on hyperelastic models for characterising the wall properties. As mentioned in the introduction, the role of ILT in reducing stresses in the aneurysm wall is still debated, in lieu of the disruption of oxygen supply to the AAA wall (Vorp et al. 1996a, 1996b, 2001), while other experimental studies (including some from the same groups) indicate that ILT reduces peak wall stress (Wang et al. 2002; Di Martino and Vorp 2003; Thubrikar et al. 2003; Leung et al. 2006). This indication is reinforced by several numerical studies that have demonstrated that ILT reduces wall stresses, by reducing the pressure load on the aneurysmal aortic wall (Mower et al. 1997; Di Martino and Vorp 2003; Thubrikar et al. 2003). However, a more accurate analysis has to include the combined dynamic effects of flow induced WSS and the stresses developing in the aneurysm wall in order to better predict the effects of ILT and its possible effect on the risk of rupture. It should be examined whether an ILT offers some protection by altering flow patterns and pressure distribution within the AAA lumen, as well as wall shear stress distribution at the AAA wall. These changes may have a significant effect on the stresses developing in the AAA wall. It is clear that the presence of a large ILT significantly changes the flow patterns within the AAA lumen by reducing the flow channel, leading to widely varying values of WSS acting from the fluid side on the AAA wall and the resulting von Mises stresses developing within the wall.

Figure 4. Pathlines depicting the flow field and the resulting wall shear stress distribution in the lumen of the second patient AAA. (a) Pathlines showing velocity magnitude in colour. (b) Shear stress contour plots. Figure available in colour online.
It should be noted that it is impossible to have a patient based AAA in which ILT would not significantly alter the lumen flow dynamics and the resultant wall stresses. The rationale of the current study was to compare an hypothetical clinical scenario (inclusion or exclusion of the ILT) by demonstrating the role of the altered flow dynamics due to the presence of ILT and its effect on the wall stresses distribution and magnitude. The specific

Figure 5. von Mises stress distribution (N/m²) in the wall of the AAA of the first patient AAA. Figure available in colour online.

Figure 6. von Mises stress distribution (N/m²) in the wall of the AAA of the second patient AAA. Figure available in colour online.
question addressed by our study is whether the apparent change in the lumen geometry with its resultant change in the flow dynamics, and the potential cushioning effect of the ILT interface, would serve to increase or reduce the AAA wall stresses.

Based on the two aneurysms studied, it appears that the presence of ILT significantly changes the stress distribution patterns and also reduces the peak wall stress (Figures 5 and 6, AAA without ILT versus AAA with ILT (ILT not shown)). The numerical simulations of an AAA with ILT included clearly indicate reduced stresses at the location of the ILT as compared to the simulation of the same patient aneurysm without the ILT.

The shape of the first AAA resembles an elongated sac with a size between 5 and 6.5 cm. The second AAA is spherical in shape and is much smaller in size, with a diameter of about 4.3 cm. Comparing the size with the stress results, it is clear that the size of the AAA remains an important parameter for the decision making of the surgeon. The largest AAA also has the largest stresses in the wall. It should be emphasised, however, that the stress levels are highly dependent on the geometry of the AAA and thus patient specific. In this view, numerical simulations of the wall stress distribution in AAA’s by our decoupled fluid–structure approach could provide the surgeon additional and useful information for predicting AAA rupture risk.

The authors are well aware of the fact that the computed stress distribution is affected by assuming uniform wall thickness, leading to an increase of the uncertainty of the results as compared to the correct in vivo conditions. This assumption is due to the inherent limitations in the imaging technique and reconstruction. However, as this approach was applied for both patients, the value of the comparative results is still valid (Papaharilaou et al. 2006). Although the analysis was of patient specific AAA, the inlet velocity for each patient was not available. Instead, a typical value of peak flow conditions in AAA was applied for both patients. While patient specific velocity would have a certain effect on the results, for a parametric and comparative study such as presented here keeping the same boundary conditions for both patients appears to be a valid assumption. The boundary conditions representing peak systolic flow and pressure were of steady flow conditions, and a fully coupled FSI approach with dynamic waveforms may yield different results, although, those may vary only slightly during peak systolic conditions when quasi-steady flow conditions may possibly dominate.

Although the flow patterns and shear stress values in both AAA studied showed similar flow patterns and shear stress contours, we found a significant difference in the wall shear stress values on the anterior side of the AAAs. We found higher WSS on the anterior side of the AAA in the patient with the smallest AAA. This could eventually influence the progression of the disease process and give an indication of progression of the disease and further weakening of the AAA wall. The influence of the WSS on the AAA should be studied in the future, by following up a number of patients during the progression of the aneurismal disease and performing the numerical simulations according to this methodology. If the model shows a correlation between wall shear stress values and changing size of the AAA, it could help the diagnosis and follow up of such AAA patients.

The significant difference in von Mises stresses within the walls of both AAA’s between the two patients (stresses in the first AAA were almost double the value of the stresses in the second AAA; Figures 5 and 6), is a clear indication that the risk of rupture in the first patient was much higher than that in the second patient. The presence of the ILT appears to be a stabilising factor for the AAA. A well orientated ILT, which generates a channel like geometry with streamlined flow patterns, appears to reduce the stress in the AAA wall significantly. It should be cautioned that our analysis did not include species transport (e.g. oxygen) to the wall, so potential detrimental effects of the presence of ILT mentioned in the literature could not be verified.

Some recent simulations include fully coupled fluid–structure interaction of AAA. However, those are very time consuming and currently are not able to provide results on a short-term basis for clinical diagnostic purposes. For that reason, we have applied a decoupled FSI between commercially available solvers. Using this approach, we were able to obtain an accurate estimation of the flow patterns, WSS and von Mises wall stresses on a relatively short-term basis. The current method is quite efficient for providing fairly rapid results. The reconstruction of the FEM volumes based on the patient specific CT-scan data (MMS Inc.) takes about 2–3 h on a moderate PC workstation. The fluid flow analysis takes about 1 h, while the wall stress analysis takes about 5 h on a single node of a computational cluster (The BuMPer cluster; http://bumps.ugent.be/bumper). This demonstrates that a dedicated finite element modelling is a valuable tool for a more accurate diagnosis and prediction of rupture risk in patient specific AAA.

5. Conclusions

We seek a more accurate prediction of risk for rupture then the standard diagnostic clinical practice which is based on diameter (5.5 cm) as a sole predictor for AAA rupture. Our analysis provides mapping of the actual stress distribution within the wall, indicating areas of stress concentration that may be prone to rupture (or their absence). This is achieved by modelling the vessel wall and its interaction with the flow patterns generated within a patient specific AAA lumen in the presence and absence of ILT. A decoupled FSI approach was applied between commercially available fluid and solid solvers. While this represents only the peak
systolic flow conditions, using this approach, we were able to obtain an accurate estimation of the flow patterns, WSS and the resultant von Mises wall stresses distribution within the AAA wall. A fully coupled dynamic FSI simulation may yield slightly different results. We have demonstrated that the inclusion of ILT in the stress analysis of patient specific AAA is important and would likely increase the accuracy of predicting AAA rupture. Compared to the current clinical practice of predicting the risk of rupture by either the maximal diameter of the AAA, or by a statistic analysis between rupture risk and aneurysm diameter, the FSI approach analyses the deformations in the aneurysm wall and provides a detailed mapping of the ensuing stresses. This can directly point to specific stress concentration regions, which may lead to a much better predictor for risk of rupture of the aneurysm based on the inherent variability among patients. While we have applied an advanced hyperelastic model to characterise better the wall mechanics, we recognise that a diseased wall of AAAs is not necessarily uniformly hyperelastic and is potentially embedded with plaques and segments of calcified tissue. We will incorporate such complexities in our numerical models accordingly, for the purpose of calculating more accurately wall stress in a diseased aorta, thereby increasing the predictive value of this method for AAA risk of rupture. This study is motivated by the need of helping the clinician to determine whether the risks involved in an elective surgery to repair the AAA are outweighed by its risk of rupture. Having this improved technology in hand will allow decisions whether surgical repair of the AAA is warranted with a greater degree of confidence.

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