The role of thrombosis in abdominal aortic aneurysms

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Abstract

An abdominal aortic aneurysm (AAA) is a life threatening expansion of the abdominal aorta, which, if left untreated, will continue to enlarge until rupture. In clinical practice, the diameter of the aneurysm is the indicator used to evaluate rupture risk.

Clinically, 75% of AAAs includes thrombosis, so it is important to understand the role of the thrombosis inside AAA.

The motivation for this research is to understand how the existence of thrombosis inside aneurysm affects its stress distribution, and from that knowledge to develop a more reliable method to identify those aneurysms at risk of rupture.

The keywords: Abdominal aortic aneurysm, thrombosis, modeling.

Introduction

Abdominal Aortic Aneurysm (AAA) is a cardiovascular disease occurring when the aorta becomes weak and develops a balloon expansion in its wall. This balloon diameter can reach sizes up to 4 times the normal aortic diameter, with the diameter enlarging at rates of 0.2–1.0 cm/year. Ruptured aneurysm leads to death in 78% – 94% of diseased aortas (Finol,2003).

More than 8.8% of the US populations above 65 years old have this disease and 100,000 people die each year in the United Kingdom because of this disease. AAA rupture is considered the 13th leading cause of death in the United States (Finol,2003). The mean age of patients with AAA is 67 years and men are affected more than women by a ratio of 4:1 (Finol,2003). Mortality rates for ruptured AAA are still high even with the current advances in medical surgeries and technologies. Aneurysm rupture is a biomechanical event that occurs when the mechanical stresses in the wall of the aorta exceed the failure strength of the aortic tissue (Lombardi,2004; Heller, 2000; Speelman,2007).

In medical practice, when the maximum diameter of AAA exceeds 5 cm it is considered at risk of rupture. Surgical repair is usually not considered until the diameter reaches at least 5 cm. However, this is a simple indicator; it is actually only one indicator of many more that are to complex to be assessed, such as bulge shape, wall thickness, flow dynamics and vessel mechanical properties (Finol,2001).

It is frequently observed that AAAs with diameters less than 4 cm can rupture which raise the need of finding a more reliable method to assess rupture risk (Marra et al. 2003;Buth et al. 2004).

Attention has been focused on aneurysm diameter, because Laplace's Law states that stress is proportional to the applied pressure and diameter of a pressure vessel, and inversely proportional to wall thickness. However, biomechanical analysis of blood dynamics could also be used in combination to maximum diameter as an indicator for rupture risk of the aneurysm. Accurate calculations of the fluid stress on the internal wall of the aneurysm may indicate when a particular aneurysm will rupture (Wang,2001; Raghavan et al. 2002).

Researchers are trying to develop a better indicator of risk of rupture by patient-specific modeling of aneurysms with the effects of the internal blood flow, and thereby predict the actual stress in the aneurysm wall. Most researches neglected the existence of thrombosis which is found in more than 75% of aneurysms for simplifications reasons.

Thus, the motivation for this research is to understand how the existence of thrombosis inside aneurysm affects its stress distribution, and from that knowledge to develop a more reliable method to identify those aneurysms at risk of rupture.

Method

Two fusiform asymmetric three-dimensional (3D) aneurysm models were constructed based on a previously published used geometries (Elger et al. 1996) with representative dimensions. Thus the dimensions of the model, illustrated in Fig. 1 are: inlet diameter, d = 2.0 cm; maximum aneurysm diameter, D = 5.0 cm. Figure 1(A)

In one of the two models, a layer of thrombosis was created attached to the internal wall of the aorta with a maximum thickness of 4.4 mm. Figure 1(B)

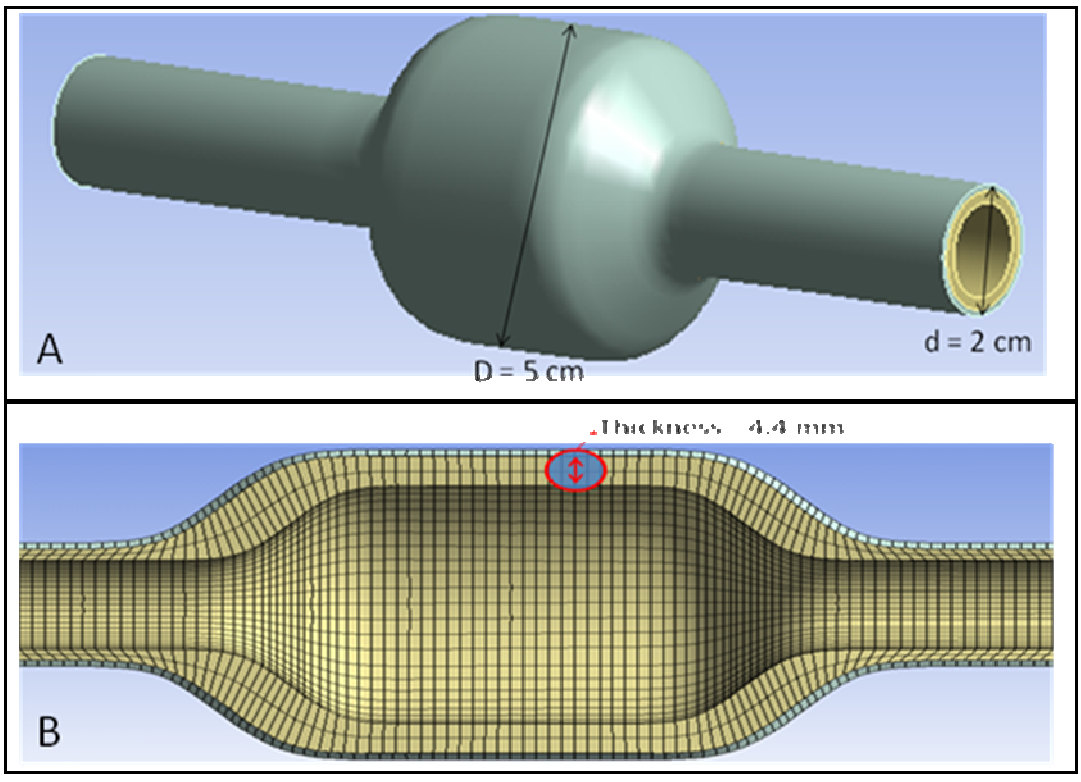

Figure 1: (A) The AAA model with dimensions. (B) The thrombosis layer inside

The finite element method (FEM) was used for all the computational studies reported in this work.

The AAA walls are assumed to have a uniform thickness of t = 2.0 mm the wall material are modeled as linearly elastic isotropic (Poisson's ratio, n = 0.3).

A homogeneous pressure of 145 mmHg was applied to the internal surface of the wall with and without the presence of the thrombosis. This value represents the peak systolic pressure of a 'normal' patient. All measurements of stresses were taken on the middle layer of the shell elements of the wall. All finite element stress analyses reached convergence.

Results and discussion

Figures 2 illustrate the Von Mises stress distribution in the wall of the two models (with and without thrombosis) obtained from static simulations of the peak systolic pressure.

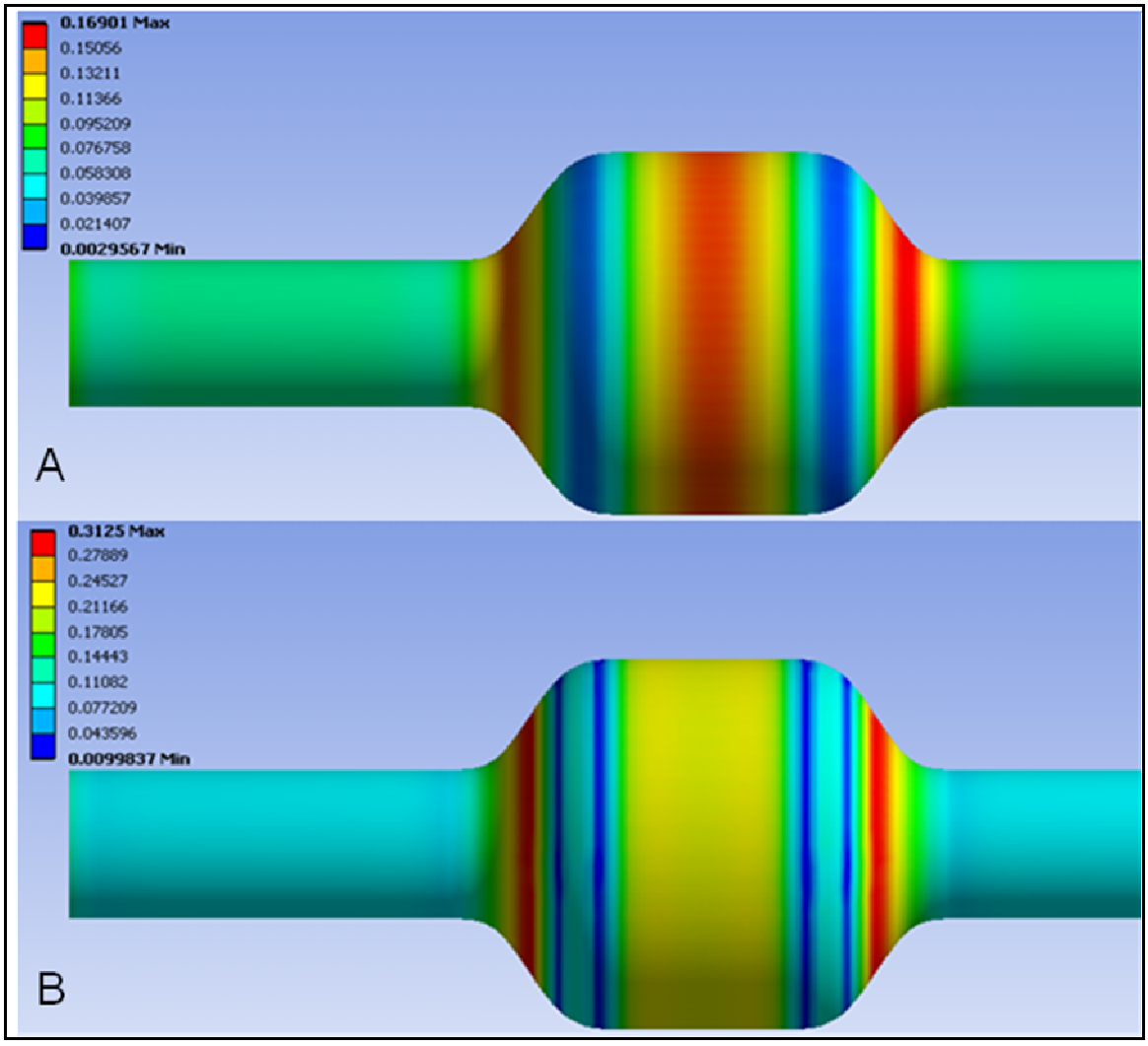

Figure 2: Von Mises stress distribution of the models (A) with thrombosis (B) without thrombosis.

Based on the two aneurysms studied here, it appears from Figure 2 that stresses dropped to almost half magnitude in the presence of thrombosis – maximum is 0.31 MPa without thrombosis and 0.169 MPa

with the presence of thrombosis – which very clearly show the role of thrombosis in decreasing the stresses on arterial wall. The presence of thrombosis also changes the distribution of maximum stresses as it could be noticed on figure 2.

More investigations on the role of the thrombosis were performed by varying its thickness and it was found that the stresses on the wall decrease when the thickness of the thrombosis was increased.

These findings support the results of (Speelman L.et al 2008) that thrombosis reduces AAA wall stress and as a result play a positive role in supporting the wall and decreasing the chance of AAA rupture. The effect is linearly related to the relative thrombosis volume.

The conclusions of this experiment seem to be in conflict with some clinical researches (Schurink G.W.H. et al 2000) which conclude that thrombosis may increase the chance of AAA rupture.

The conclusion of this report that from mechanical point of view, thrombosis work as a cushion to protect the AAA wall, but in the same time from biological point of view it may play an important role in the pathology and natural history of AAA which.

More investments which can match the mechanical and the biological sides can give more understanding the role of the thrombosis within AAA.

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