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PREDICTION OF THROMBUS-PRONE REGIONS IN ABDOMINAL AORTIC ANEURYSMS

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INTRODUCTION

Intraluminal Thrombus (ILT) is present in almost all abdominal aortic aneurysms (AAAs), with several studies trying to clarify if it increases or decreases the risk of rupture: increases local proteolytic activity and causes wall hypoxia leading to wall weakening, or buffers against wall stress? In the meantime, the prediction of its growth might be of value, as clinical studies have demonstrated that growth of the ILT might indicate AAA rupture risk.

Thrombus formation and growth is a complicated process, with several biological and hemodynamic factors to participate in a dynamic cascade. In brief, the formation is initiated by activation of platelets, which is determined by a combination of factors, including high shear stress and time exposure to such stresses. Activated platelets may next accumulate in recirculation and stagnation zones of the flow field, where they are attached usually at the non-endothelialized surface (thrombogenic), where low WSS and high residence time promote their adherence. This dynamic and multifactorial process of thrombus formation and evolution makes it difficult to model and predict its deposition.

In an effort to evaluate if hemodynamic parameters are promoting ILT formation, the Lagrangian approach is usually considered for the evaluation of coherent vortex formation and the estimation of platelet activation occurrence, a process that requires a large computational time. However, since many studies show that AAAs possess the necessary hemodynamic conditions for coagulation and activation of platelet clotting mechanisms, the AAA sac most probably is filled with activated platelets ready to attach to a thrombogenic surface if the hemodynamic conditions permit it. Therefore, near-wall hemodynamics (wall shear stress) is most probably the major determinant for ILT deposition. Our hypothesis is that with a statistically sufficient patient cohort, a co-mapping of flow dynamic conditions at the AAA wall with the information of ILT deposition distribution will allow the construction of a statistical model that will predict thrombus-prone regions.

In the present study we compute the wall shear stress (WSS) distribution in two patient specific AAAs that were thrombus-free at the first scan. The luminal surface is then divided into patches. In each patch the WSSs are averaged and the information of thrombus deposition is stored. A logistic regression with a clustering by patient method is then performed to test the statistical significance of the association between hemodynamics and thrombus location.

METHODS

Data acquisition

Two CT scans of AAAs (and their follow-ups) were obtained. The 3D lumen and external wall surface of the sac was reconstructed with ITK-SNAP and smoothed with the Taubin algorithm in vascular modeling tool kit (vmtk). The AAA surfaces were co-registered using the aorta at the level of the renal arteries (± 5 mm) and the aortic/renal bifurcations as registration features. In detail, the wall and lumen surfaces of the follow-up scan were best fit aligned to the respective surfaces of the first scan at these registration features.

Meshing and computational fluid mechanics

Flow extensions were added to the luminal surface of the first scan, and a pure hexadehral mesh was constructed using ANSA (BETA CAE Systems *S.A.*, Greece) consisting of approximately 800,000 elements. In order to account for the shear thinning properties of blood, the Herschel-Bulkley viscosity model was considered. Simulations were performed using Fluent (ANSYS Inc.) with default convergence criteria set to 10^{-5}. A pulsatile profile was prescribed at the inlet with Re_{mean}=330 and Re_{peak}=1800, and results were collected

after all transient effects were washed out. The time-average WSS was computed and its distribution was mapped on the surface.

Surface patching

Because the initial lumen The luminal surface of the first scan (with the hemodynamics information) and the luminal surface of the second scan (with the thrombus thickness information) were patched using vmtk (Figure 1).

RESULTS

DISCUSSION

This study investigated the relationship between two hemodynamic factors and ILT deposition. A significant relation was determined between areas with low WSS and increased RRT and thrombus deposition. While either of these two variables alone can be used to predict thrombus prone regions, a model including both factors demonstrated a significantly higher prediction value.

However, high RRT and low WSS cannot be considered as a sufficient condition for thrombus deposition since platelet activation and biochemical processes such as inflammation also influence clotting mechanisms. However, once the clotting process is triggered, the ILT is likely to form in regions of high RRT and low WSS. We are therefore, not proposing a new hemodynamic mechanism for ILT formation, but rather a convenient method for predicting it in AAAs. More patient-specific AAA cases with thrombus-free baseline sacs are currently monitored to further improve the statistical model and potentially obtain threshold values for these hemodynamic markers of thrombus deposition.

In conclusion, RRT values and WSS distribution emerge as an appropriate tool for identifying the possible regions of thrombus formation.

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