A Blood Flow based model for Platelet Activation in Abdominal Aortic Aneurisms

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Introduction

Thrombus formation is the physiological response to vascular injury, it prevents loss of blood and permits wound healing, however, it is also associated with pathological conditions like hypoxia, anoxia and infarction [1]. Consequently, thrombus development must be carefully modulated to avoid uncontrolled growth, which in turn could lead to organ malfunctions. Specifically, an Intra-Luminal Thrombus (ILT) is found in almost all larger (clinically relevant) Abdominal Aortic Aneurysms (AAAs) and multiple biochemical [2] and biomechanical [3] implications on the underlying wall tissue have been reported. Despite the dominant role played by the ILT in AAA disease little is known regarding its development, and hence, the present study investigates ILT formation with particular emphasis on platelet activation triggered by biomechanical and biochemical field variables.

Method

The proposed model assumes that platelet activation is defined by a single field variable $\theta$ representing the accumulation of mechanical [4] and chemical [5] factors as the platelet moves along its path line. Platelet activation is given as soon as $\theta$ overcomes a certain threshold thought to be a constitutive property of blood. Specifically, the rate of the activation variable is determined by the maximum shear stress and the local concentrations of agonists and antagonists. To implement the model the fluid mechanical problem was solved in (COMSOL, COMSOL AB) and a particle tracking analysis (MATLAB, The MathWorks) was applied as a post processing step. The flow in a circular tube and the Backward Facing Step (BFS) problem under varying initial conditions were used for a basic investigation of the model and to relate its predictions to available data in the literature. Finally, platelet activation in patient specific AAAs was predicted and related to ILT development, which was estimated from Computer Tomography-Angiography (CT-A) data recorded from patient follow-up studies.

Results and Conclusions

The platelet activation variable $\theta$ is complex distributed (highly heterogeneous) in the flow field, where, specifically, at the boundary of vortexes [6] and in the boundary layer of the non-endothelialized wall highest values were predicted. Continuous release of antagonists from the endothelialized wall lowers $\theta$ in its vicinity, and hence, despite the high shear stress platelet activation
is prevented. The proposed model links biomechanical and biochemical mechanisms of platelet activation and is able to predict the onset of thrombus formation of the BFS problem. The model is also able to predict some features of ILT development in the AAA, however, the change in luminal geometry is a cumulative effect of ILT growth, wall growth and their mechanical interactions, and hence, data recorded form patient follow-up studies needs to be analyzed carefully when validating the present model.

References

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