COMPUTATIONAL SIMULATION OF PATIENT-SPECIFIC BLOOD COAGULATION IN STENT THROMBOSIS

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Introduction

The efficacy of stents to treat coronary artery stenosis is related to the hemodynamic environment. The struts of these devices disturb the local flow field and induce flow recirculation zones and endothelial damage [1]. The aim of this study is to improve understanding of the impact of injury induced blood coagulation on stent thrombosis and to identify risk factors that contribute to it, with the ultimate goal of improving stent designs. This will be studied analyzing local hemodynamics together with patient-specific clot formation using computational fluid dynamics (CFD).

Methods

A biophysical model of a fibrin-rich clot is developed based on the framework presented in Bouchnita et al. [2]. A system of coupled convection-diffusion-reaction (CDR) equations is used to solve the concentration of factor IX and X, prothrombin, thrombin, fibrinogen, fibrin, and fibrin polymer. The production or consumption of each of these agonists is incorporated into the source terms in the CDR equations. The computational domain consists of a 2D channel including a stent strut. A section of the bottom wall serves as a surface flux boundary condition representing a tissue factor (TF) coated surface. The clot is modeled as a porous medium with the porosity depending on the concentration of fibrin polymer. The model is implemented into FLUENT 2021 R1. A set of cases are studied with 3 different TF patch locations, with both average healthy and hypercoagulant coagulation parameters for a physical time of 30 minutes [3].

Results

When comparing the impact of the location of the TF patch, we observed that in the healthy case the clot was formed earlier and appeared to be smaller but denser proximal to the stent strut compared to the clot formed distal to the strut (Fig. 1A-D). When the TF patch was located after the recirculation zone no clot was formed (Fig. 3E-F), even in the case of hypercoagulant plasma (Fig. 3K-L). At the other TF locations, clot formation occurred at a significantly faster rate and resulted in a larger clot in the hypercoagulant case compared to the healthy case (Fig. 3G-J). Also in the hypercoagulant case the proximal clot (Fig. 3I-J) was denser than the distal clot in which the fibrin was completely spread downstream of the recirculation zone (Fig. 3G-H).

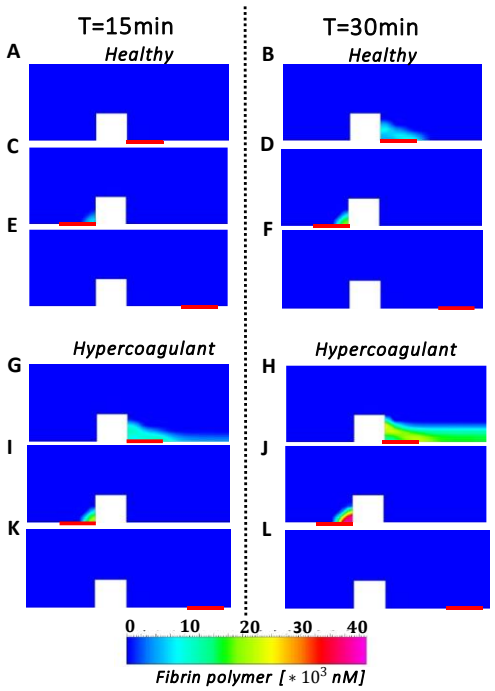

Figure 1: Results of the simulation after 15min. (left) and 30min. (right) for different tissue factor patch locations indicated in red, with (A-F) healthy plasma and (G-H) hypercoagulant plasma.

Discussion

Results demonstrated that the largest clots were formed when the wall was damaged right after the stent strut, probably due to the presence of the recirculation zone which allows for prolonged interactions between coagulation factors. More concentrated clots were formed proximal to the strut, likely due to the confinement of coagulation factors by the stent strut. The clot growth rate and size were increased in a hypercoagulant case compared to a healthy case, which highlights the importance of incorporating patient-specific parameters. We are currently working on investigating the effects of various stent designs and flow conditions on coagulation dynamics.

References

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