

EFFECTS OF SHEAR STRESS-INDUCED THROMBUS BREAKDOWN ON THROMBOSIS IN AORTIC DISSECTION

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Introduction

False lumen (FL) thrombosis is a key factor in assessing prognosis in type B aortic dissection (TBAD) patients. Several computational models have been developed to predict thrombus formation and growth in TBAD [1,2]. Thrombosis is a complex and dynamic process involving both thrombus formation and breakdown which can occur simultaneously. While most existing models consider the role of low shear stress in the initiation and growth of thrombus, they often ignore the effect of thrombus breakdown induced by high shear stress. In this study, a new shear stress-induced thrombus breakdown function is proposed and implemented in our previous thrombosis model [1]. The performance of the refined model is assessed by comparing predicted thrombosis in a TBAD geometry with follow-up CT scans. The effect of thrombus breakdown on thrombus growth is also quantified.

Method

A new parameter τ is introduced into the thrombosis model to account for local shear stress experienced by each thrombus element, defined in Equation 1. Thrombus is represented by the variable bound platelets (BP) and governed by Equation 2.

$$\tau = \sqrt{\frac{1}{6} \sum (\sigma_{ii} - \sigma_{jj})^2 + \sum \sigma_{ij}^2}, i = x, y, z \quad (1)$$

$$\frac{\partial BP}{\partial t} = K_{BP} \phi_c \phi_{RT} \phi_{\dot{\gamma}} [AP] - K_{breakdown} \phi_{BP} \frac{\tau_{local}^2}{\tau_{local}^2 + \tau_{breakdown}^2} \quad (2)$$

Where τ is the total scalar shear stress calculated based on the stress tensor (σ), K_{BP} is a kinetic constant accounting for thrombus growth (12 s^{-1}), ϕ_i is a switching function used to turn on and off thrombosis depending on the local concentration of each variable (coagulant, shear rate and residence time), $K_{breakdown}$ is the rate of thrombus breakdown (300 s^{-1}), and $\tau_{breakdown}$ is the shear stress threshold (0.3 Pa). Compared to our original model, the new model allows formed thrombus to detach when τ_{local} exceeds $\tau_{breakdown}$.

The refined thrombus model was applied to a patient-specific TBAD model (Figure 1A) – a computational mesh was generated in ICEM (v21.2). Simulations were performed using Ansys CFX (v21.2) and were run until thrombus volume plateaued. Blood was treated as a non-Newtonian fluid described by the Bird-Carreau model. A pulsatile waveform was applied at the inlet, with 3-element Windkessel models at each outlet [1].

Results

As shown in Figure 1, locations of thrombus formation predicted by the refined model matched the 3-year follow-up geometry better than the original model which did not account for thrombus breakdown. At location 1, the FL above the right renal artery was completely thrombosed, which was captured by the refined model. At location 2, partial thrombosis in the FL was captured by both models, but the original model overpredicted thrombus growth. Figure 2 shows change in thrombus volume as a function of simulation time. Improvements were achieved in both computational time and final predicted volume.

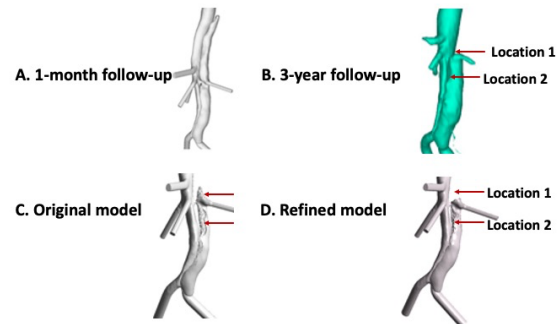


Figure 1: A, B: Reconstructed geometries at 1-month and 3-year follow-up. C, D: Lumen surface following thrombosis using the original and refined models.

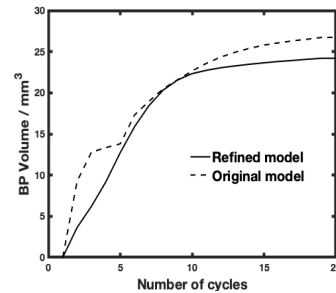


Figure 2: Change in thrombus volume over time.

Discussion

This study shows the influence of shear stress-induced thrombus breakdown on predicted thrombus growth in a patient-specific TBAD. Comparisons with the follow-up CT scan and original model prediction demonstrated that accounting for thrombus breakdown not only improved the accuracy of predicted thrombus, but also reduced computational time by stabilising growth more quickly. The refined thrombosis model can replace our previous model for future patient-specific applications.

References

1. CH Armour et al, Journal of Endovascular Therapy, 2020
2. Tobin N, Manning KB. Int J Numer Meth Biomed Eng 38.10 (2022): e3638

