MULTISCALE MULTIPHYSICS MODEL OF THROMBUS BIOMECHANICS IN AORTIC DISSECTION

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Aortic dissection is a life threatening event; it is responsible for significant morbidity and mortality in individuals ranging in age from children to young and older adults. When a dissection communicates with the true lumen and forms a so-called false lumen within the aortic wall, this false lumen may remain patent or become either partially or completely thrombosed. *Increasing clinical evidence suggests that a completely thrombosed false lumen results in an improved prognosis whereas a partially thrombosed false lumen may render the wall more vulnerable to further dissection or rupture.* There is currently little understanding of why certain dissections thrombose while others do not. We submit that data-driven mathematical modeling has promise to increase our understanding of this important clinical condition.

There are, however, no current models that can predict the development, growth, or arrest of an intramural thrombus within the dissected aortic wall. Similarly, there are no models of either the potential remodeling of an intramural thrombus that is well away from the following blood or the adverse remodeling of the remnant aortic wall leading to frank rupture. To address these gaps, we will develop the first data-driven, multiscale, multiphysics model of the biomechanics of intramural thrombus in aortic dissection.

During the first year of this study, we have focused on initial model development and data collection. Specifically, we are extending two complementary multiscale models (i.e., flow-thrombus model of Karniadakis and thrombus-wall remodeling model of Humphrey) that will be coupled in year two to create a novel multiscale, multiphysics computational framework. This coupled model will span scales from platelet dynamics to continuum flows and from fibrin / collagen fiber remodeling to whole wall stress analysis. We are also collecting longitudinal data using the most widely accepted animal model of dissecting aortic aneurysms (continuous subcutaneous infusion of angiotensin-II in the apolipoprotein null mouse) to refine parameter values within our coupled multiscale model and to validate the predicted time-courses and extents of accumulation of thrombus and remodeling of the wall. We will present initial modeling and experimental advances during the annual IMAG meeting.

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