Multiscale model of platelet translocation and collision

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Abstract
The tethering of platelets on the injured vessel surface mediated by glycoprotein Ibα (GPIbα) - Von Willebrand factor (vWF) bonds, as well as the interaction between flowing platelets and adherent platelets, are two key events that take place immediately following blood vessel injury. This early-stage platelet deposition and accumulation triggers the initiation of hemostasis, a self-defensive mechanism to prevent the body from excessive blood loss. To understand and predict this complex process, one must integrate experimentally determined information on the mechanics and biochemical kinetics of participating receptors over very small time frames (1-1000 μs) and length scales (10-100 nm), to collective phenomena occurring over seconds and tens of microns. In the present study, a unique three dimensional multiscale computational model, platelet adhesive dynamics (PAD), was applied to elucidate the unique physics of (i) a non-spherical, disk-shaped platelet interacting and tethering onto the damaged vessel wall followed by (ii) collisional interactions between a flowing platelet with a downstream adherent platelet. By analyzing numerous simulations under different physiological conditions, we conclude that the platelet's unique spheroid-shape provides heterogeneous, orientation-dependent translocation (rolling) behavior which enhances cell-wall interactions. We also conclude that platelet-platelet near field interactions are critical for cell-cell communication during the initiation of microthrombi. The PAD model described here helps to identify the physical factors that control the initial stages of platelet capture during this process.