

Medical device thrombosis

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Blood-contacting medical devices, such as vascular grafts, stents, heart valves and catheters are frequently used in cardiovascular diseases. In the United States alone, 650-700,000 stents are implanted each year. Approximately 1-2% of the implanted stents become occluded due to thrombus formation, resulting in myocardial infarction or death in the majority of these patients. Artificial surfaces promote clotting through a number of mechanisms including protein adsorption, adhesion of blood cells, and thrombin generation. And although there have been considerable improvements in biomaterials, patients with stents, mechanical heart valves and other medical devices require treatment with antiplatelet drugs or anticoagulants to prevent thrombosis. Several clinical studies have shown a higher risk of thrombosis in patients with medical devices treated with fondaparinux compared to low molecular weight heparin, or for dabigatran compared to vitamin K antagonists. This suggests that general inhibition of coagulation is preferred over specific targeted inhibition at the level of thrombin. Activation of the contact system of coagulation is a major driver of thrombus formation induced by medical devices. Targeted inhibition of proteins of the contact system was found to be very efficacious both in in vitro studies and in a recent study of patients undergoing elective knee arthroplasty. In the presentation, I will discuss the processes involved in medical device-induced thrombosis, and highlight the advances that have been made in our knowledge on ways to prevent thrombosis on blood-contacting medical devices.