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Commentary

# Acute Coronary Stent Thrombosis in Modern Era

**Adam Gerhart***

*Department of Thrombosis, NYU Grossman School of Medicne, New York, USA*

## DESCRIPTION

Stent thrombosis is a catastrophic complication of Percutaneous Coronary Intervention (PCI) associated with significant morbidity and mortality. Technological advances from balloon angioplasty to bare metal stent placement and drug-eluting stent placement have reduced the incidence of stent thrombosis. Reliable management and precautions can still be ignored. Here we describe two cases of definitive subacute stent thrombosis of the right coronary artery with peritonitis after right coronary artery surgery and very late left anterior descending artery thrombosis. In both cases, inhibition of platelet aggregation by clopidogrel showed excellent compliance. Therefore, after successful PCI in both cases, we switched from clopidogrel to potentially potent antiplatelet drugs such as ticagrelor to reduce the incidence of future stent thrombosis.

Stent thrombosis can be caused by a variety of mechanisms. Patient-related factors, pharmacological factors, lesion and procedure-related factors, and post-procedure factors can play a role. Overall, the development of blood clots in the lumen of the stent can be the result of one or more of the following: Activation of the extrinsic coagulation cascade upon contact with the subcutaneous tissue, stent struts, or polymer coating, Inadequate inhibition of platelet activation, activation of the endogenous coagulation cascade by low shear stress from slow coronary flow or presence of thrombus-promoting states.

From the beginning, ST has been a source of trouble for stent placement. The first experience of wall tents in the late 1980s was overshadowed by the ST rate approaching 24%. Postoperative antithrombotic therapy at the time often consisted of aspirin in combination with oral anticoagulant therapy. Double antiplatelet therapy with aspirin and thienopyridine thiclopidine and switching from bailout to selective stenting significantly reduced ST to less than 2%. Previous oral administration of antiplatelet drugs and glycoprotein IIb/IIIa antagonists further reduced ST. It provides an overview of ST reported in recent studies on BMS and DES, ranging from 0.1% to 3.1%. The wide range of ST incidence in studies using both BMS and DES is explained by differences in definition, follow-up period, antithrombotic drugs, and patient and lesion complexity.

The first significant concern about ST over 30 days arose with the use of intracoronary brachytherapy. Stenting in the same setting as intracoronary brachytherapy is associated with a sustained ST risk of more than 30 days in 5% to 10% of patients. This risk was mitigated by extending DAPT from the standard 30 days to 3 or 6 months. Therefore, when clinical trials for Drug-Eluting Stents (DES) were designed, late ST was well known and DAPT was planned accordingly. Interestingly, in randomized DES clinical trials, there was no difference in ST rates between DES and Bare Metal Stents (BMS) at any point in time.

Stent thrombosis is a truly confusing clinical entity, and reducing its incidence is important for both clinical safety and patient and physician safety. Given that STEMI patients are one of the groups with the highest risk of developing stent thrombosis after primary PCI; this could be an ideal place to focus on research activities, especially randomized controlled trials, in the future. Appropriate patient selection based on rigorous screening protocols, if circumstances permit, allows physicians to select the appropriate stent in each case, complications associated with underlying medical conditions, socioeconomic factors, and drug non-compliance helps to minimize illness. However, as a whole, in the clinical context, stents can be used to derive a diverse and multifaceted approach to coronary blood circulation reconstruction that focuses on both patient-related and procedural factors using current data. It has the potential to provide the greatest long-term benefits in a world where thrombosis is difficult.

**Correspondence to:** Adam Gerhart, Department of Thrombosis, NYU Grossman School of Medicne New York, USA, E-mail: gerhart_adam@tcu.edu

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