# Diagnosis and Evaluation of Stent Thrombosis with Optical Coherence Tomography

Alessio Mattesini, MD, Benedetta Bellandi, MD,
Serafina Valente, MD, Guido Parodi, MD, PhD, FESC*

**KEYWORDS**

• Stent thrombosis • OCT • Intravascular imaging • PCI

**KEY POINTS**

- Malapposition and stent underexpansion have been identified as predictors of stent thrombosis (ST) that can be evaluated by optical coherence tomography (OCT) at the time of stent implantation to reduce the risk of subsequent ST.
- During follow-up, OCT may identify high-risk intrastent morphology such as neoatherosclerosis.
- OCT-guided management seems to be a feasible, safe, and appropriate approach to assess the efficacy of coronary thrombus removal and to detect the prevalent stent-related factor that caused the ST.
- Owing to the multifactorial nature of ST and the heavy contribution of platelets to the acute event, intracoronary imaging and platelet reactivity evaluation may help to fully understand the cause of ST.

## INTRODUCTION

Percutaneous coronary intervention (PCI) with stent implantation is an effective invasive strategy for the treatment of coronary artery disease. The most feared complication related to coronary stent placement is stent thrombosis (ST). This complication is relatively rare, particularly with second-generation drug-eluting stents (DES), occurring in 0.5% to 1% of patients within 1 year after then procedure. When it does occur, ST is associated with substantial morbidity and mortality. It most commonly presents as acute myocardial infarction (MI)[1] requiring emergent repeat PCI with optimal reperfusion occurring only in two-thirds of patients.[2] As a result, ST has been associated with high 30-day mortality rates (range, 25%–30%) as well as with high rates of recurrent ST. In fact, about 20% of patients with a first episode of ST experience a recurrence within 2 years. The mechanisms underlying ST are multifactorial, including patient, procedural and postprocedural treatment characteristics. By virtue of its high resolution and the characteristics of imaging with light, optical coherence tomography (OCT) represents a unique tool for ST diagnosis. Moreover, it can be used to improve the understanding of the particular mechanism of ST in a particular patient, and may thereby guide the most appropriate intervention. Moreover, when used as guidance for complex PCI procedures, OCT may play a role in minimizing the risk of ST. In this review, we provide an update on ST with a particular focus on OCT for its diagnosis and prevention.

## DEFINITION AND CLASSIFICATION OF STENT THROMBOSIS

The lack of consensus on the definition of ST among clinical trials has led to disparities in

The authors have no disclosure to declare related to this article.
Department of Cardiology, Careggi Hospital, Largo Brambilla n 3, Florence 50100, Italy
* Corresponding author. Department of Cardiology, Careggi Hospital, Viale Pieraccini 17, Florence I-50134, Italy.
*E-mail address:* parodiguido@gmail.com

Intervent Cardiol Clin 4 (2015) 295–307
http://dx.doi.org/10.1016/j.iccl.2015.02.006
2211-7458/15/$ – see front matter 

reports of ST and, in particular, has prevented comparison of the rates of ST between studies. To address this issue, the Academic Research Consortium definition of ST has been established.[3] In this definition, ST is categorized currently according to the timing after initial PCI and the objective evidence of ST.

### Timing of Stent Thrombosis

ST is considered to be acute when occurring between 0 and 24 hours after stent implantation; subacute between 24 hours and 30 days; late between 30 days and 1 year; and very late after 1 year. The term 'early' ST can also be used to refer to acute and subacute ST.

### Definite Stent Thrombosis

Definite ST requires the presence of an angiographic confirmation of ST (the presence of a thrombus that originates in the stent or in the segment 5 mm proximal or distal to the stent) and must be associated with at least 1 of the following criteria within a 48-hour window: acute onset of ischemic symptoms at rest, new ischemic electrocardiographic changes that suggest acute ischemia or typical increase and decrease in cardiac biomarkers, or the presence of a pathologic confirmation of ST (evidence of recent thrombus within the stent determined at autopsy or via examination of tissue retrieved after thrombectomy). The incidental angiographic documentation of stent occlusion in the absence of clinical signs or symptoms is not considered a confirmed ST (silent occlusion).

### Probable Stent Thrombosis

Probable ST is defined as the presence of any unexplained death within the first 30 days after stent implantation, or in the presence of any MI related to documented acute ischemia in the territory of the implanted stent without angiographic confirmation of ST and in the absence of any other obvious cause, irrespective of the time after the index procedure.

### Possible Stent Thrombosis

Possible ST is defined as any unexplained death from 30 days after stenting until the end of follow-up.

## PATHOPHYSIOLOGY OF STENT THROMBOSIS

The pathophysiology of ST is complex and involves several different factors. Platelet function and platelet reactivity on antiplatelet therapy, coagulation factors, and inflammation play a pivotal role in the mechanism of ST. In addition to these elements, ST can be triggered by patient clinical characteristics and risk factors such as the following:

- Chronic renal failure;
- Diabetes;
- Left ventricular dysfunction or type of stent implanted (bare metal stent, first- or second-generation DES, biovascular scaffold);
- Treated lesion characteristics (long, complex lesion, bifurcation, chronic total occlusion);
- Acuity of the index clinical syndrome preceding stenting (stable angina, unstable angina, non-ST elevation MI or ST-elevation MI);
- Procedure-related factors; and
- Other unknown factors (**Box 1**).[1,4,5]

Different temporal pathophysiologic mechanisms are involved in each category of ST (acute, subacute, late, and very late). Procedure- and mechanical-related factors seem to play a major role in acute/subacute ST, whereas impaired reendothelization, stent malapposition or fracture, hypersensitivity reaction to the polymer used in DES, inflammation, or de novo plaque rupture have important roles in late and very late ST (**Box 2**).[6,7] It is currently unknown what extent of lack of stent coverage can be considered unsafe. Effective dual antiplatelet therapy is able to mitigate the early risk related to uncovered stent struts. Early discontinuation of dual antiplatelet therapy, as described in the Mechanism Of Stent Thrombosis (MOST) study, or ineffective platelet inhibition owing to poor responsiveness to therapy[8,9] allows the uncovered metallic stent strut to trigger the thrombotic event.[10] The number of uncovered stent struts is a chronic trigger factor, requiring activated platelets to develop thrombosis. Taking into consideration the small amount of available data, it seems that, in the case of a slight lack of stent strut coverage, the occurrence of ST can be provoked by concomitant high on-treatment platelet reactivity, especially when associated with nonresponsiveness to aspirin therapy.[11] Tailored antiplatelet therapy based on platelet function test results was investigated in the Assessment by Double Randomization of a Conventional Antiplatelet Strategy versus a Monitoring-guided Strategy for Drug-Eluting Stent Implantation and of Treatment Interruption versus Continuation One Year after Stenting (ARTIC) and Gauging Responsiveness with A VerifyNow Assay - Impact on Thrombosis And Safety (GRAVITAS) trials showed