JACC: CARDIOVASCULAR INTERVENTIONS
VOL. 7, NO. 10, 2014

ISSN 1936-8798/$36.00
PUBLISHED BY ELSEVIER INC.
http://dx.doi.org/10.1016/j.jcin.2014.05.016

STATE-OF-THE-ART REVIEW

Stent Thrombosis

A Clinical Perspective

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JACC: CARDIOVASCULAR INTERVENTIONS CME

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CME Objective for This Article: At the completion of this article, the learner should be able to: 1) define stent thrombosis; 2) compare antithrombotic therapies and strategies in patients receiving coronary artery stents to decrease the risk of stent thrombosis; and 3) assess the risk of the development of stent thrombosis in a patient undergoing percutaneous coronary intervention.

CME Editor Disclosure: *JACC: Cardiovascular Interventions* CME Editor Olivia Hung, MD, PhD, has received research grant support from NIH T32, Gilead Sciences, and Medtronic Inc.

Author Disclosures: Dr. Jaffer has received research grants from Merck, Kowa, and Siemens. Dr. Mehran has received institutional research grant support from The Medicines Company, Bristol-Myers Squibb/Sanofi-Aventis, Lilly/Daichi Sankyo, Regado Biosciences, and STENTYS; is a consultant for Abbot Vascular, AstraZeneca, Boston Scientific, Covidien, CSL Behring, Janssen Pharmaceuticals, Merck, and Sanofi-Aventis; and has equity in and is a shareholder of Endothelix, Inc. Dr. Piek is on the Advisory Board of Abbott Vascular; and is a consultant for Miracor. Dr. Dangas is a consultant for Medtronic and The Medicines Company; is on the Advisory Board of AstraZeneca; has received institutional research grants from Eli Lilly and The Medicines Company; his spouse is on the Advisory Board of Boston Scientific, Abbott Vascular, and Bristol-Myers Squibb/Sanofi-Aventis. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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Issue Date: October 2014
Expiration Date: September 30, 2015

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Manuscript received April 8, 2014; revised manuscript received May 12, 2014, accepted May 14, 2014.

JACC: CARDIOVASCULAR INTERVENTIONS VOL. 7, NO. 10, 2014
OCTOBER 2014:1081–92

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ABSTRACT

The invention of intracoronary stents greatly increased the safety and applicability of percutaneous coronary interventions. At this time, >1 million coronary stent implantations are performed each year in the United States. But together with the growing use of stents, stent thrombosis, the most feared complication after stent implantation, has emerged as an important entity to understand and prevent. Adjunct pharmacological therapy, stent design, and deployment technique have been adjusted ever since to reduce its occurrence. The current clinical overview of stent thrombosis ranges from its pathophysiology to current state-of-the-art technical and pharmacological recommendations to avoid this complication. (J Am Coll Cardiol Intv 2014;7:1081–92) © 2014 by the American College of Cardiology Foundation.

The safety and efficacy of percutaneous coronary intervention (PCI) improved dramatically after the introduction of the coronary artery stent. A severe and common early complication of balloon angioplasty alone was abrupt vessel closure, which was associated with significant morbidity and mortality and the need for emergency coronary artery bypass surgery (1). Coronary artery stents significantly reduced the incidence of abrupt vessel closure and also reduced restenosis by essentially eliminating arterial recoil and negative remodeling after angioplasty (2). From the very beginning, thrombosis of the metal endoprosthesis, or stent thrombosis (ST), was recognized as an important complication and adjunct pharmacological therapy, and stent technique and technology have been adjusted to reduce its occurrence.

DEFINITION OF STENT THROMBOSIS

The sensitivity and specificity of the definition of ST depends on the level of certainty required (a better judgment can be made in the presence of angiography or autopsy studies), but also on the accuracy of data available during adjudication (e.g., events occurring in remote locations and long after the index procedure cannot be easily adjudicated due to many coinciding factors, interval interventions, and other clinical conditions treated by unrelated medical teams). Until 2007, a large variety of ST definitions were in use, limiting the possibility to consistently evaluate ST rates among studies. A standardized definition of ST was proposed by the Academic Research Consortium (ARC) (**Table 1**) (3). The ARC definition acknowledges these issues by establishing a gradation of certainty (definite, probable, and possible ST), and standardized the timing of ST (acute, subacute, late, and very late ST), which may have different pathophysiological mechanisms and clinical implications.

SCOPE OF THE PROBLEM

At the very beginning of intracoronary stenting, with low-pressure inflation and single-antiplatelet therapy, bare-metal stents (BMS) were associated with high rates of ST, ranging from 20% in early reports (4–6) to around 3% to 5% (2,7) with the use of aggressive anticoagulation therapy, which in turn was associated with hemorrhagic complications. Colombo et al. (8) first introduced stenting with dual-antiplatelet therapy (DAPT) instead of warfarin, accomplished by attaining adequate stent expansion using high-pressure balloon inflation. This implantation technique achieved an acceptable 1.6% rate of angiographically documented ST at 6-month follow-up and was universally accepted from then on (9,10).

The incidence of ST up to 1 year follow-up seems similar for DES and BMS and ranges from 0.6% to 3.2% for BMS and 0.6% to 3.4% for DES, depending on patient and lesion characteristics (11–14). Before the introduction of DES, ST was perceived as a complication occurring early after stent implantation. In 2004, McFadden et al. (15) described 4 cases of late and very late ST in first-generation DES (sirolimus-eluting stent, Cypher, Cordis, Warren, New Jersey, and paclitaxel-eluting stent, Taxus, Boston Scientific, Natick, Massachusetts). Subsequently, data from a large all-comers registry suggested that very late ST may occur steadily at an annual rate of 0.4% to 0.6% after first-generation DES implantation (16). These