Mechanisms of Very Late Stent Thrombosis After Drug-Eluting Stent Implantation

Findings From Coronary Angioscopy and Optical Coherence Tomography

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Drug-eluting stents (DES) are now widely used for patients with coronary artery disease undergoing percutaneous coronary interventions. A current major concern of using DES is very late stent thrombosis (VLST) that may occur beyond 1 year after DES implantation (1). VLST is an infrequent, albeit catastrophic complication, which can lead to myocardial infarction or sudden cardiac death (2). However, the underlying mechanisms of VLST remain unclear. In the current report, we show 3 cases of VLST after sirolimus-eluting stent (SES) implantation. Findings from coronary angioscopy (CAS) and optical coherence tomography (OCT) suggested 3 differential mechanisms of VLST in each case.

In the first case (Fig. 1), CAS and OCT showed uncovered and malapposed stent struts at the culprit lesion. Late stent malapposition is caused by stent underexpansion and thrombus resolution or by positive vessel remodeling. The later mechanism is characteristic of DES (3). In this case, CAS findings revealed yellow plaque, which might reflect inflammatory reaction. Delayed neointimal coverage over stent struts and late stent malapposition are ordinarily thought to be associated with VLST in patients treated with DES.

In the second case (Fig. 2), coronary rupture was observed in the yellow plaque which completely covered stent struts. Rupture of thin-cap fibroatheroma is a common cause of acute coronary syndrome. It has been reported that neointimal atherosclerotic change (neoatherosclerosis) occurred with DES earlier than with a bare-metal stent; and unstable features of neoatherosclerosis were identified after shorter implantation duration then with DES (4). Enhanced inflammation after DES implantation may promote atherosclerosis and exaggerate vulnerability of the plaque.

In the third case (Fig. 3), ruptured plaque was observed under the stent struts. The rupture continued to a large cavity behind the completely covered stent struts. To our knowledge, this is the first report of such a case. This is likely associated with delayed arterial healing following DES implantation in a patient with acute myocardial infarction (AMI). The underlying plaque in this patient might have been lipid-rich necrotic plaque, which was not covered by thick neointima following stent implantation because of the drug effect. This may have created another thin-cap fibroatheroma, which finally ruptured.
A 70-year-old man underwent a 2.5 mm × 28 mm sirolimus-eluting stent (SES) implantation in the mid-left anterior descending artery (LAD) for acute myocardial infarction (AMI). Aspirin (100 mg/day) with clopidogrel (75 mg/day) was prescribed, but clopidogrel was stopped because of drug-induced liver injury 1 month after SES implantation. Thirty-four months after SES implantation, the patient suddenly suffered from recurrence of angina and was admitted to our hospital. Emergent coronary angiography (CAG) showed thrombus-like shadow in SES (A). After aspiration thrombectomy (B), optical coherence tomography (OCT) and coronary angioscopy (CAS) were performed. OCT and CAS revealed no coverage of neointima over stent struts in the distal and proximal portion of stent (a). At thrombus sites, OCT revealed malapposition and red and white thrombus on the stent struts which were not covered by neointima (b, c and d). CAS revealed malapposition and yellow plaque under stent struts (b, c and d, yellow arrow).

A 68-year-old man underwent 2 SES implantations in the mid-LAD for AMI (2.5 mm × 18 mm and 2.5 mm × 18 mm). Aspirin (100 mg/day) with ticolipidine (200 mg/day) was prescribed. Aspirin and ticlopidine were stopped 1 year after SES implantation by a self-judgment. Fifty-four months after SES implantation, the patient suddenly suffered from recurrence of angina on exertion and was admitted to our hospital. Emergent CAG revealed total occlusion at proximal SES in the mid LAD (A). After balloon angioplasty (B), OCT and CAS were performed. OCT and CAS revealed neointimal coverage over stent in the distal and proximal portion of stent (a). OCT revealed cavity formation (white arrow) over stent struts (b, c). CAS revealed yellow plaque rupture (C, yellow arrow) and cavity formation (b, c, red arrow) over stent struts. Abbreviations as in Figure 1.
REFERENCES


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Figure 3. Rupture Continued to Lipid Core Behind Covered Stent Struts

A 70-year-old man underwent a 2.5 mm × 28 mm SES implantation in the mid-LAD for AMI. Aspirin (100 mg/day) and ticlopidine (200 mg/day) was prescribed thereafter. Ticlopidine was stopped 6 months after SES implantation. Fifty-nine months after SES implantation, the patient suddenly suffered from recurrence of angina and was admitted to our hospital. Emergent CAG showed total occlusion at the site of SES (A). After aspiration thrombectomy (B), OCT and CAS was performed. In the distal and proximal portion of stent, OCT and CAS revealed neointimal coverage over stent struts and no stent underexpansion (a). OCT revealed mixed thrombus, plaque rupture (b), and cavity formation behind neointima-covered stent (c, d, white arrow). CAS revealed ruptured yellow plaque under stent struts (b, c, yellow arrow). The stent struts were floating over the ruptured plaque (b, red arrow). Abbreviations as in Figure 1.