Optical Coherence Tomography Findings in Very Late (4 Years) Paclitaxel-Eluting Stent Thrombosis

Arend F. L. Schinkel, MD, PhD,* Peter Barlis, MD,* Heleen M. M. van Beusekom, MD, PhD,† Patrick W. Serruys, MD, PhD, FACC,* Evelyn Regar, MD, PhD*

Rotterdam, the Netherlands

The mechanisms of late coronary stent thrombosis, a complication with high morbidity and mortality (1), are poorly understood. Late thrombosis in drug-eluting coronary stents has been associated with delayed endothelialization of the stent struts or systemic and intrastent hypersensitivity reaction (2). We present in vivo findings in a patient with very late stent thrombosis. In 2003, a 60-year-old man with a history of stable angina and smoking underwent elective placement of 2 overlapping paclitaxel-eluting stents (3.0 × 16 mm and 2.75 × 28 mm, Taxus stent, Boston Scientific, Natick, Massachusetts) and post-dilation with a noncompliant balloon, for a significant stenosis of the left anterior descending artery. The discharge medication consisted of aspirin, clopidogrel (12 months), beta-blockers, and statins. Four years after stenting, the patient presented with acute anterior myocardial infarction caused by late stent thrombosis while under chronic aspirin therapy (Fig. 1). After thrombus aspiration, intravascular ultrasound and intravascular optical coherence tomography were performed showing incomplete coverage of 21% of the stent struts, which may reflect delayed vascular healing (Figs. 2 and 3). Pathology of the thrombotic material showed a recent multilayered thrombus (Fig. 4). Post-procedure, the patient was treated with ReoPro (Centocor, Leiden, Netherlands) and was discharged with sustained aspirin and clopidogrel therapy.
Figure 2. Intracoronary Imaging After Thrombus Aspiration

(Upper panels) Intravascular ultrasound (Atlantis; Boston Scientific); (lower panels) optical coherence tomography (LightLabImaging, Boston, Massachusetts; cross-sectional and longitudinal view). (A) Distal reference showing eccentric plaque in 11 to 4 o’clock position. (B) Distal stent portion showing a well-expanded and apposed stent with thin tissue coverage by optical coherence tomography that is missed by intravascular ultrasound. (C) Irregular lumen borders with intraluminal remnants of the thrombus. (D) Proximal stent edge with incomplete apposition of 5 stent struts (arrows) against the vessel wall. The distance to the vessel wall is 200 μm. Optical coherence tomography shows tissue around the stent struts (differential diagnosis thrombus, neointima). Incomplete stent apposition may be caused by initial malaposition or acquired malposition caused by a vascular response to the stent. C = catheter artifact; GWA = guidewire artefact.

Figure 3. Spectrum of Tissue Coverage as Seen by OCT in This Stented Segment

Tissue coverage was not visible in 21% (n = 110) of the struts, while 79% (n = 411 of 521) of the struts showed tissue coverage of various thickness. (A) No visible coverage; (B) Very thin coverage (40 μm); (C) Maximal coverage (140 μm). OCT = optical coherence tomography.
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Reprint requests and correspondence: Dr. Arend F. L. Schinkel, Department of Cardiology, Thoraxcenter Room Ba316, Erasmus Medical Center, Dr. Molewaterplein 40, 3015 GD, Rotterdam, the Netherlands. E-mail: arendschinkel@hetnet.nl.

REFERENCES

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