Two Cases of Coronary Stent Thrombosis Very Late After Bare-Metal Stenting

Masamichi Takano, MD,* Masanori Yamamoto, MD,† Kyoichi Mizuno, MD*
Tokyo, Japan

Medical literature has recently focused on very late stent thrombosis (VLST) after drug-eluting stent implantation, while its mechanistic issue was not fully explored in the bare-metal stent (BMS) era. The first case is a 59-year-old man presenting with inferior non–ST-segment elevation myocardial infarction, 4 years after BMS implantation (NIR 3.5/18 mm, Boston Scientific, Galway, Ireland) for a chronic total occlusion lesion in the proximal right coronary artery. Coronary angiograms showed Thrombolysis In Myocardial Infarction (TIMI) flow grade 1 and filling defects in the BMS deployed previously, and massive red thrombi attaching to uncovered stent struts were found by angioscope (Fig. 1, Online Video 1). Thrombectomy and adjunctive balloon angioplasty were performed based on the angioscopic findings, and TIMI flow grade 3 was obtained. The second case is a 71-year-old man who was admitted for a diagnosis of inferior non–ST-segment elevation myocardial infarction 10 years after a treatment with BMS (gfx 3.0/18 mm, Applied Vascular

Figure 1. Findings of Coronary Angiography and Angioscopy
(A) Coronary angiograms show Thrombolysis In Myocardial Infarction (TIMI) flow grade 1 with filling defects in the stent segment. In this segment, occlusive red thrombi (arrows) and uncovered stent struts (arrowhead) are identified by angioscope. (B) Angiograms show TIMI flow grade 3 and haziness in the stent segment (arrowheads). Ruptured yellow plaque (arrow) and thrombi are found by angioscope. Any stent struts are not visible in this angioscopic view. *Guidewire.
Engineering, Santa Rosa, California) for the culprit lesion of stable angina pectoris in the distal right coronary artery. Angiographic haziness in the BMS segment was seen despite of TIMI flow grade 3. Any progressive lesions on angiogram were not seen in other segments. Angioscopic observation for the stent segment demonstrated absence of the uncovered struts. Remarkably, ruptured yellow plaque accompanied by thrombi occupied the lumen (Fig. 1, Online Video 2). Direct stenting was consequently performed for sealing the ruptured plaque. Although both cases were definite VLST standardized by the Academic Research Consortium (1), lumen appearance of direct visualization by angioscope was quite different. Previous autopsy studies showed that plaque disruption outside the BMS with extensive prolapse could lead to thrombosis (2). For the first time, angioscopic findings in the second case propose strong evidence that atherosclerotic plaque disruption inside the BMS may be one potential trigger of thrombosis. However, persistent uncovered struts in the first case may lead to VLST, as well as those of the drug-eluting stent (3). The present images cannot generalize VLST to all cases. However, contrastive angioscopic images suggest that various pathogeneses may contribute to the occurrence of definite VLST after BMS implantation, and different interventional strategies for VLST may be chosen.

Reprint requests and correspondence: Dr. Masamichi Takano, Division of Cardiology, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8602, Japan. Email: takanom@nms.ac.jp.

REFERENCES

APPENDIX
For accompanying videos, please see the online version of this article.