Intraprocedural Stent Thrombosis Detected on Coronary Angioscopy

Toru Miyoshi, MD; Hideo Kawakami, MD; Akira Oshita, MD; Hiroshi Matsuoka, MD

Intraprocedural stent thrombosis (IPST) during percutaneous coronary intervention (PCI) is a rare complication leading to poor outcome.\(^1\)\(^-\)\(^3\) but, to date, there have been few reports on coronary angioscopy (CAS) of IPST.

A 60-year-old man was admitted to hospital due to chest pain. Four years previously he had undergone PCI for acute coronary syndrome, in the proximal left anterior descending coronary artery with 2.75×33-mm everolimus-eluting stent (EES), and in the OM with 2.5×12-mm EES. He had the coronary risk factors type 2 diabetes mellitus (HbA1c, 6.6%), and dyslipidemia (low-density lipoprotein cholesterol, 106 mg/dl), and was on aspirin 100 mg, losbastatin 5 mg, nicorandil 15 mg, bisoprolol 5 mg, famotidine D 20 mg, and ethyl icosapentate 1,800 mg. Electrocardiogram showed ST elevation in leads II, IIIaVF. Emergency coronary angiography showed no significant stenosis in the left coronary artery, but the proximal right coronary artery (RCA) was totally occluded (Figure 1-I). The patient was treated with dual antiplatelet therapy (clopidogrel 300 mg, loading dose), followed by aspiration of red thrombus (Figure 1-II). Next, on optical coherence tomography (OCT)
Intraprocedural Stent Thrombosis on CAS

the lumen profile was almost 3.0 mm, with multiple plaque ruptures and a large amount of red thrombus (Figure 1A–C). EES (3.0×24 mm) was deployed in the proximal RCA (Figure 2-III), but in-stent haziness developed gradually, which was thought to be due to IPST (Figure 2-IV). Activated coagulation time was 288 s. Next, the stent was dilated with a 3.5×15-mm non-compliant balloon with rated burst pressure 18 atm during 60 s (total), until the IPST disappeared. This site was then assessed on OCT and CAS. On CAS, a high yellow color-grade plaque was seen to be protruding from the stent struts, with some of the struts covered by this higher yellow plaque, and various thrombi at the stent site. (Lower A–D) Optical coherence tomography showing attenuated thrombus in the stent, and the stent strut buried in these materials.

The lumen profile was almost 3.0 mm, with multiple plaque ruptures and a large amount of red thrombus (Figure 1A–C). EES (3.0×24 mm) was deployed in the proximal RCA (Figure 2-III), but in-stent haziness developed gradually, which was thought to be due to IPST (Figure 2-IV). Activated coagulation time was 288 s. Next, the stent was dilated with a 3.5×15-mm non-compliant balloon with rated burst pressure 18 atm during 60 s (total), until the IPST disappeared. This site was then assessed on OCT and CAS. On CAS, a high yellow color-grade plaque was seen to be protruding from the stent struts, with some of the struts covered by this higher yellow plaque, and various thrombi at the stent site. (Upper A–D) Coronary angioscopy showing high yellow color-grade plaque protruding from the stent struts, with some of the struts covered by this higher yellow plaque, and various thrombi at the stent site. (Lower A–D) Optical coherence tomography showing attenuated thrombus in the stent, and the stent strut buried in these materials.

In the present case, multiple ruptures and a large amount of red thrombus were detected on OCT. Absolutely yellow plaque and neointima on CAS were associated with various types of thrombosis, therefore yellow dense plaque may be associated with the cause of thrombosis. Another possible factor is poor effect of antiplatelet therapy. In the present case the patient received clopidogrel at admission, and also prior to PCI, but he did not develop stent thrombosis. This suggests a low potential for clopidogrel resistance.

IPST may be associated with early phase of stent thrombosis. It was reported that the incidence of major adverse cardiac events, target lesion revascularization, 5-year mortality in early stent thrombosis and late stent thrombosis was higher than in very late thrombosis. The present case suggests that IPST is related to dense yellow plaque, and to thrombosis. If IPST is seen on coronary angiography, anti-coagulation therapy and aggressive lipid lowering therapy should be continued.

References