Very Late Sirolimus-Eluting Stent Thrombosis Due to Stent Fracture and Late-Acquired Incomplete Stent Apposition Detected on Multislice Computed Tomography

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Figure. (A) Baseline coronary angiography (CAG) demonstrating a significant stenosis in the left anterior descending coronary artery. (B) Post-procedural CAG with 2 overlapped sirolimus-eluting stents (proximal, 3.5x23 mm; distal, 3.0x18 mm). (C) Post-procedural intravascular ultrasound at the site of arrow in (B) showing good expansion and apposition of the stent. (D) Multi-planar reconstruction image and (E) cross-sectional image 5 years after the procedure showing the late-acquired incomplete stent apposition (arrow-heads). (F) 3-D stent image showing complete separation of stent strut (arrow) and (G) cross-sectional image without stent strut, indicating stent fracture. (H) CAG 5 years after the procedure demonstrating total occlusion at the proximal segment of the stents. (I) Intravascular ultrasound at the site of the arrow in (H; identical to cross-section in C) showing positive vessel remodeling, absence of stent strut, indicating stent fracture, and low-intensity mass in the lumen suggesting thrombus. (J) Optical coherence tomography at the site of arrow in (H) demonstrating a high-backscattering mass with signal attenuation, protruding into the lumen, implying red thrombus.
A 71-year-old man with hypertension, hyperlipidemia, and diabetes mellitus was referred for coronary angiography (CAG) following an episode of chest pain on exertion. CAG showed a significant stenosis in the middle segment of his left anterior descending (LAD) coronary artery, which was treated with 2 overlapped sirolimus-eluting stents (SESs; 3.5×23 mm and 3.0×18 mm) on 25 August 2005 (Figures A, B). Intravascular ultrasound (IVUS) immediately after SES implantation confirmed a well-expanded stent that was not in incomplete apposition (Figure C).

At 5 years after stenting, the patient was admitted for surgery of an unruptured abdominal aortic aneurysm. Although 64-slice multislice computed tomography (MSCT) before the operation (on 8 October 2010) showed no significant stenosis in the coronary arteries, contrast enhancement outside the stent indicated late-acquired incomplete stent apposition (ISA; Figures D, E, arrow heads). In addition, complete separation of stent struts was seen on 3-D imaging (Figure F, arrow), and absence of stent struts due to stent fracture was seen on the cross-sectional image (Figure G).

Both aspirin and ticlopidine were discontinued for 7 days before the operation, and then re instituted 2 days after surgery. At 5 days after surgery (on 23 October 2010), the patient developed acute chest pain and electrocardiogram showed an acute anterior-wall ST elevation myocardial infarction. Emergency CAG showed in-stent occlusion in the proximal LAD (Figure H), followed by revascularization therapy. IVUS and optical coherence tomography (OCT) were performed following revascularization with balloon dilatation. IVUS demonstrated absence of stent struts, indicating stent fracture, and a low-intensity mass in the lumen, suggesting thrombus (Figure I). In addition, OCT also demonstrated a high-back-scattering mass with signal attenuation protruding into the lumen implying red thrombus (Figure J), comparable to findings of late stent thrombosis (LST).

LST after DES implantation is one of the problems of the drug-eluting stent era. While the mechanism of LST is yet to be completely understood, several factors have been suggested as contributors to LST. In a study with IVUS, late-acquired ISA was highly prevalent in patients with LST after DES implantation. Previous pathological analysis reported that stent fracture had a significant impact on the occurrence of stent thrombosis. An IVUS study has proposed a relationship between late-acquired ISA and stent fracture. Some types of late-acquired ISA are caused by positive vessel remodeling due to biological reactions to the DES, which may allow motion and/or kinking of the stent, leading to stent fracture. Alternatively, stent fracture can lead to local mechanical irritation of the vessel, which may lead to ISA. Therefore, late-acquired ISA and/or stent fracture could set the stage for LST. LST might arise by combination of such structural changes of the vessel and discontinuation of anti-platelet therapy.

Intravascular imaging modalities such as IVUS and OCT are useful for assessment of coronary arteries with implanted stents, but these modalities, as a diagnostic procedure, are invasive. Currently, MSCT is widely used for the evaluation of coronary artery disease as a less invasive imaging technique. In the present case, MSCT just before the onset of LST was able to visualize not only late-acquired ISA but also stent fracture. We should consider alternative anticoagulant therapy such as heparin in cases involving these MSCT findings requiring discontinuation of anti-platelet therapy.

Disclosure
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References