Simultaneous Two-Vessel Very-Late Stent Thrombosis of Everolimus-Eluting Stents

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ABSTRACT: Very late stent thrombosis is a rare complication. Several factors related to the patient, procedure, and premature discontinuation of antiplatelet therapy are known to contribute to its occurrence. This report describes the case of a 40-year-old patient with simultaneous two-vessel stent thrombosis (left anterior descending and right coronary arteries) 31 months after everolimus-eluting stent implantation.

Drug-eluting stents (DESs) are designed to deliver drugs and to suppress in-stent restenosis. Binary restenosis rate has been reduced to less than 10% after DES implantation and second-generation DESs fared even better in terms of deliverability. However, stent thrombosis, a potentially fatal event due to acute vessel closure, is one of the most feared complications after percutaneous coronary intervention (PCI). Stent thrombosis is multifactorial, including procedure-related factors such as incomplete expansion, multiple stents, dissection, and late stent malapposition due to resolution of thrombus on the vessel wall. Patient-related factors include diabetes mellitus, advanced age, renal dysfunction, acute coronary syndrome, and bifurcation or ostial lesions. Discontinuation of antiplatelet therapy is one of the most powerful predictors of stent thrombosis. Very late stent thrombosis is a rarer complication, occurring more than 1 year after the index procedure in patients treated with DESs. The second-generation DESs appear to have reduced the incidence; however, it still remains a dreaded entity. It is, however, even rarer to have a simultaneous two-vessel very late stent thrombosis with the second-generation DESs. We report the case of a patient who experienced simultaneous two-vessel very late stent thrombosis 31 months after everolimus-eluting stent implantation following accidental discontinuation of his aspirin.

Case Description
A 40-year-old man presented to the emergency department with severe chest pain within 30 minutes after onset of symptoms. His past medical history included dyslipidemia, smoking, and strong family history of ischemic heart disease. He had undergone cardiac catheterization 31 months prior for an anterior ST-segment elevation myocardial infarction (STEMI) and a 3.0 x 23 mm Xience V DES (Abbott Vascular) was implanted in his mid left anterior descending (LAD) coronary artery and postdilated with a 3.5 x 12 mm balloon. He also had a right coronary artery (RCA) lesion, which was stented 5 days later with a 2.5 x 23 mm Xience V DES and postdilated with a 2.75 x 12 mm balloon. His time between the index procedures and now was unremarkable; he received dual-antiplatelet therapy with aspirin and clopidogrel for the initial 6 months and was subsequently continued on aspirin 100 mg only as well as statin and a beta-blocker. The patient accidentally stopped his aspirin about 5 days prior to this event, citing difficulty in procuring them from the pharmacy.

In the emergency department, the patient’s electrocardiogram showed ST-segment elevation in the inferior leads and Q-waves in the anterior wall. His physical examination was unremarkable, with a pulse rate of 65 beats/min and a blood pressure of 120/80 mm Hg. A 600 mg loading dose of clopidogrel was given and the patient was transferred to the catheterization laboratory for primary percutaneous coronary intervention. Coronary angiography revealed 100% occlusion of the mid-segment of the LAD within the stent and 100% occlusion of the RCA within the stent, consistent with LAD and RCA stent thrombosis (Figures 1 and 2). In view of the emergent situation, the RCA was immediately wired using balloon support as it was difficult to traverse the thrombotic segment through the stent on its own. Subsequently, we used an Export catheter...
(Medtronic, Inc) to aspirate the thrombus. Intracoronary abciximab followed by intravenous infusion was administered. The vessel looked a bit larger in caliber than the 2.75 mm final size achieved previously; therefore, we deployed a 3.0 x 23 mm Xience V stent to cover the previous stent as well as the stent inlet proximally, and postdilated the proximal part of the stent with a 3.5 x 12 mm NC Balloon, giving a good end result (Figure 3). We started with a bolus and followed with an infusion of abciximab.

We proceeded to PCI for the LAD lesion, which again looked bigger than the final 3.5 mm caliber that was achieved previously. The vessel was wired; however, a balloon would not cross the stent, so it was presumably behind the struts of the stent. Thus, we rewired with a larger bend on the wire and managed to pass through the stent. The lesion was predilated with the 2.0 x 12 mm Trek balloon to establish TIMI 2 flow. Intravascular ultrasound showed late stent malapposition, with a large amount of thrombus detected and positive coronary vessel remodelling around the stent. The stent diameter at the proximal edge was 3.5 mm, whereas the vessel diameter was 4.0 mm (Figure 4). Subsequently, we used a 4.0 x 12 mm balloon to postdilate the entire stent and the final angiographic result showed TIMI 3 flow with the patient free from pain (Figure 5). The patient was kept on an intra-aortic balloon pump overnight. Peak plasma troponin I was >50.0 ug/L (reference range, 0.0-0.03 ug/L) and peak plasma creatine kinase was 2682 ng/mL (reference range, 25-175 ng/mL). No renal dysfunction was detected. An echocardiogram was performed and showed a mild-to-moderately dilated left ventricle with severe global impairment of systolic function and an ejection fraction of 30%. The patient was discharged 3 days later on dual-antiplatelet therapy to be continued for life.

Discussion
To the best of our knowledge, this is the first reported case of simultaneous two-vessel very-late stent thrombosis occurring 31 months after second-generation everolimus-eluting stent implantation. Recently, two case reports were published that also had simultaneous two-vessel very-late stent thrombosis of first-generation DESs. In our case, intravascular ultrasound clearly identified stent malapposition with positive remodeling of the vessel wall. Resolution of trapped thrombus between struts and vessel wall, and late hypersensitivity reactions are described as some of the the mechanisms for late stent thrombosis and several reports have shown eosinophilic infiltration in the vascular wall that leads to a positive remodeling after DES implantation. The factors leading to this event included the cessation of dual-antiplatelet therapy; however, the simultaneous very-late stent thrombosis of the two-vessel leads to a possibility of late stent malapposition and hypersensitivity reaction to the DES. As the penetration of second-generation DESs becomes even more widespread, follow-up imaging using either non-invasive multi-slice computed tomography, or angiography with intravascular ultrasound/optical coherence tomography may be indicated to screen for very-late stent malapposition.

References
Figure 1. 100% occlusion of the mid-segment of the left anterior descending coronary artery within the stent.

Figure 2. 100% occlusion of the right coronary artery within the stent.
Figure 3. Right coronary artery final result.

Figure 4. Left anterior descending coronary artery final result.
Figure 5. Intravascular ultrasound of left anterior descending coronary artery showing late malapposition.