Late Stent Thrombosis Associated with Late Stent Malapposition after Drug-Eluting Stenting: A Case Report

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ABSTRACT

We report here on one patient who suffered from acute myocardial infarction that was due to late stent thrombosis, and this was associated with late stent malapposition very late (21 months) after the deployment of a paclitaxel-eluting stent and shortly after (7 days) the discontinuation of the aspirin therapy. The intravascular ultrasound examination revealed that the late stent thrombosis was accompanied by late stent malapposition. This is a report on late stent thrombosis associated with late stent malapposition after the successful implantation of a paclitaxel-eluting stent (PES). (Korean Circulation J 2006;36:472–475)

KEY WORDS : Stent ; Thrombosis.

Introduction

Drug-eluting stents (DES) have a benefit over bare metal stents (BMS) because they reduce the need for later revascularization and they also reduce the risk of adverse cardiac events. Although the safety profiles of DESs did not seem to differ from those of BMSs in many trials, concerns have arisen about the potential for developing late stent thrombosis (LST) that is related to delayed endothelialization and hypersensitivity. Although LST is a rare finding, its clinical outcome is generally catastrophic. Moreover, the risk factors related to LST in a DES is not well understood. The incidence of late stent malapposition (LSM) after DES implantation is reported to be higher than that for BMS implantation, but its clinical significance remains uncertain. We report here on one case of acute myocardial infarction (AMI) that was due to LST, and it was associated with LSM after deployment of a Paclitaxel-eluting stent (PES).

Case

A 44 year-old male patient presented with severe chest pain that had lasted for 1 hour. The patient had history of hypertension and he was a 15 pack-year smoker. Twenty-one months ago, he was admitted the hospital due to effort-related chest pain. A diagnostic coronary angiogram showed the anomalous origin of the right coronary artery (RCA) and there was significant stenosis of the distal RCA (Fig. 1A). Percutaneous coronary intervention (PCI) was successfully performed in the distal RCA with deployment of a 3.0 × 16 mm Taxus stent (Boston Scientific, Minnesota, USA) (Fig. 1B). The intravascular ultrasound (IVUS) examination revealed adequate expansion and apposition without any complications (Fig. 1C) after the Taxus stent implantation. The patient was then discharged and he received continuous combined antiplatelet medications of aspirin and clopidogrel. At the 6-month follow-up, the angiogram revealed good patency of the stent without restenosis (Fig. 1D). After 3 months of this combined antiplatelet treatment, the patient was switched from combined antiplatelet treatment with aspirin and clopidogrel to treatment with aspirin alone. At 21 months after stent deployment and 7 days after the discontinuation of aspirin, the patient was stricken with severe chest pain for 1 hour and he then visited our emergency room. The cause of the patient not taking his aspirin was not non-cardiac surgery or preparation for other procedures, but rather, it was non-compliance. ST-segment elevation was noted on the inferior leads, and so primary PCI was done. The coronary angiogram demonstrated haziness in the distal segment of the stent and at the distal side of the stent with spontaneous reperfusion (Fig. 2A). IVUS examination revealed that a huge thrombus was accompanied with late stent malapposition at the distal segment and distal side of the stent (Fig. 2B-D). Per-
formance of balloon angioplasty (Sprinter, 3.0 × 20 mm) and thrombectomy with a PercuSurge aspiration system (Medtronic AVE, Santa Rosa, CA, USA) was successfully done. The patient received triple antiplatelet medications of aspirin, clopidogrel and cilostazol, and he was discharged without complication.

**Discussion**

Stent thrombosis is rare, but very serious complication after performing coronary stent placement, and it usually occurs before the endothelialization has been completed. For a BMS, this process takes a few weeks, but a DES delays the endothelialization process. For this reason, patients who have DESs implanted are prescribed aspirin and clopidogrel therapy for at least 6 months. The incidence of subacute stent thrombosis, which defined as stent thrombosis within 30 days of deployment, has been reduced to <0.5-2% as a result of the improved deployment techniques that fully appose the stent to the vessel wall, and with the use of antiplatelet agents.\(^{12}\) Stent thrombosis has been shown to be increased for the patients who undergo emergency stenting because of AMI or unstable angina that is due to the presence of intracoronary thrombus.\(^{9}\) Premature discontinuation of antiplatelet therapy is the most common precipitating factor of stent thrombosis.\(^{10}\) Long and multiple stents,\(^{12}\) stent under-expansion,\(^{4}\) stent malapposition,\(^{4}\) residual dissection,\(^{2}\) resistance to aspirin and clopidogrel, and platelet polymorphism\(^{5}\) have been suggested as the other possible other causes of stent thrombosis. LST (>30 days after PCI) is uncommon with using BMSs except after brachytherapy; however, delayed endothelialization associated with the implantation of a DES may increase the risk of LST. The incidence of LST has been reported to be at least 0.35% or possibly up to 0.72% after implanting a DES, but the “real-world” incidence of LST may be substantially higher than the rates reported for the clinical trials. Several mechanisms of LST have been postulated; these include a local drug effect that delays endothelialization or the formation of a dysfunctional endothelium, a hypersensitivity to the polymer and the development of neointimal hyperplasia with occlusive thrombus formation as an acute event.\(^{7}\) According to the recent data, the most independent predictor of LST was the premature discontinuation of antiplatelet therapy.\(^{8}\) Yet, the most appropriate duration of treatment with antiplatelet therapy that is required to prevent this complication is still controversial. In this case, AMI developed at 7 days after the discontinuation of aspirin. Our report shows that stent thrombosis can arise very late (>21 months after...
stenting) after the uncomplicated placement of a paclitaxel-eluting stent when the short-term antiplatelet therapy is discontinued.

When the stent is incompletely apposed or in the presence of a flow-limiting dissection of the vessel wall, the dominant initial stimulus for an acute and subacute stent thrombosis is the existence of an area of low flow between the stent and the vessel wall. Stent underexpansion and incomplete apposition is associated with stent thrombosis for DESs, but there is little data on the clinical results of LST. Hong et al. have recently reported that there was no significant difference in the MACEs between the LSM and non-LSM groups during three years follow-up after BMS implantation. The incidence of LSM ranged from 4% to 5.4% for BMS implantation. On the other hand, the reported incidence of LSM was 8.7% in the SIRIUS study and 1.1% in the TAXUS IV trial. However, this finding was not associated with any adverse clinical events at 1 year in those trials. Virmani et al. have recently reported on a case of LST associated with LSM, and this was due to the possible localized hypersensitivity to the polymer. In the present case, the huge thrombus occurred at the LSM site and at the distal side of the stent. The precise pathogenesis of LST in this case is uncertain. The initial PCI was a simple procedure of 16 mm single stent implantation, and the post-procedural IVUS findings revealed that adequate expansion and apposition was achievable without edge dissection and residual stenosis. We think that the major cause of the LST in this case was discontinuation of antiplatelet therapy; in addition, the low flow between the stent and the vessel wall at the LSM site may have acted as a stimulus for stent thrombosis under the condition of no antiplatelet medication.

We suggest that the potential risk of stent occlusion should be seriously considered when contemplating discontinuation of antiplatelet therapy for the patients treated with DESs. A further study is required to reveal the true incidence and predictive factors of LST in DESs.

REFERENCES


