A Case of Extremely Very Late Stent Thrombosis 8 Years after Implantation of Drug-Eluting Stent Observed by Intravascular Ultrasound

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Drug-eluting stents (DES) are now widely used for patients with coronary artery disease undergoing percutaneous coronary interventions. The current major agenda for using DES is very late stent thrombosis (VLST) that occurs beyond 1 year after DES implantation. Although VLST is rare, it is a serious complication that can result in sudden death or myocardial infarction. Until now, there have been only a few case reports of VLST within 7 years. We report a case of a 78-year-old man who presented with an ST segment elevation myocardial infarction due to extremely very late stent thrombosis resulting from a mal-apposed stent and delayed neointimal coverage that occurred 8 years after stent implantation after the cessation of antiplatelet agents for 10 days.

Keywords: Coronary artery stent thrombosis; Very late stent thrombosis; Drug-eluting stent; Percutaneous coronary intervention

INTRODUCTION

Stent thrombosis is an abrupt thrombotic occlusion of a previously inserted patent stent. Very late stent thrombosis (VLST) is defined as occlusion that occurs beyond 1 year after stent insertion. Up until now, there have not been any reports regarding the incidence and prevalence of VLST in South Korea. VLST occurs in 0.4% to 0.6% of cases per year [1]. Stent thrombosis is a relatively rare complication with a malignant nature that can result in acute myocardial infarction (AMI) or sudden death. Therefore, due to its malignant nature, further studies to disclose its mechanism and prevent thrombosis are needed.

Several mechanisms of VLST have been revealed, which are associated with mal-apposition of the stent struts after under-expansion, delayed neointimal coverage and coronary plaque rupture in thin or thick covered stent struts. In a search for these mechanisms, intravascular ultrasound (IVUS) has made a great contribution to the outcomes of the investigations. Until now, there have been only a few case reports of VLST within 7 years. However we report a case of a thrombosis free interval of 8 years, which exceeds the prior case, and with the use of IVUS, we have confirmed the cause of this extremely very late stent thrombosis.

CASE REPORT

A 78 year-old male visited the emergency room presenting with left chest pain. The onset of pain was 30 minutes prior to the visit to the emergency room and the character of the pain was persistent and squeezing. The patient was a 30 pack-year ex-smoker with a history of admission to the division of neurology due to a transient ischemic attack in the year 2003 and since then, after being diagnosed with hypertension, the patient had been taking losartan.

In May 2005, the patient underwent percutaneous coronary intervention (PCI) due to an AMI, and a 95% stenosis was identified in the proximal to the middle left anterior descending artery (LAD). TAXUS 3.0 × 32 mm and TAXUS 2.75 × 20 mm stents were implanted in the proximal LAD and middle LAD respectively (Fig. 1). He underwent coronary angiography due to dyspnea in March 2012, which showed patent flow at the site of the previous stent insertion (Fig. 2A). The patient received dual antiplatelet therapy (DAPT) for 7 years, and due to aspirin intolerance, which was found in 2012, clopidogrel monotherapy was maintained ever since. Even after sufficient antiplatelet therapy, chest pain developed shortly after cessation of the antiplatelet agent.

The patient’s initial vital signs were blood pressure 136/90 mm
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Fig. 1. (A) In May 2005, coronary angiography showed 95% stenosis of proximal to middle left anterior descending artery (LAD) and (B) improved coronary artery blood flow after intervention with two TAXUS stent implantation in proximal LAD and middle LAD, respectively.

Fig. 2. (A) In March 2012, coronary angiography shows patent flow at the previous stent implantation site. (B) Stent thrombosis was presented with total occlusion of the coronary artery at the same site of the previous stent implantation in July 2013. (C) Coronary blood flow improved after intervention with balloon angioplasty.

Hg, pulse rate 80 beats/min, respiration rate 16 breaths/min and body temperature 36.3°C. Laboratory findings showed serum creatinine kinase 102 IU/L (range, 56 to 244 IU/L), creatine kinase-myocardial band 8.9 ng/mL (range, 0.0 to 5.0 ng/mL), troponin-I 0.5 ng/mL (range, 0.00 to 0.16 ng/mL) and myoglobin 431 ng/mL (range, 0 to 72 ng/mL). Electrocardiography showed ST segment elevation of 2 mm in the V3, V4, and V5 leads, and hence, he was diagnosed with ST segment elevation myocardial infarction (STEMI). We performed coronary angiography, which showed total occlusion of the proximal to the middle LAD at the previous stent insertion site (Fig. 2B). Successful percutaneous coronary artery angioplasty (PTCA) with balloon angioplasty and thrombus aspiration was done (Fig. 2C). To ascertain the cause of the stent thrombosis, the patient underwent IVUS, and delayed neo-intimal coverage and mal-apposed stent struts were found (Fig. 3). After PTCA, the patient was free of symptoms and was discharged.
DISCUSSION

Drug-eluting stents (DES) are now widely used for patients with coronary artery disease undergoing PCI because they have a low risk of in-stent restenosis and associated clinical events, by inhibiting neointimal proliferation, compared with bare metal stents (BMS). The current major concern of using DES is stent thrombosis due to the prolonged exposure of the stent to blood. As a result, DES patients present more frequently with STEMI compared with BMS: 60% vs. 22%, respectively [2,3].

VLST is an infrequent but fatal complication causing AMI and sudden death. Several mechanisms of VLST have been revealed in previous case studies with coronary angioscopy and optical coherence tomography [4,5]. Firstly, delayed neointimal coverage [6] and mal-apposed stents after underexpansion during PCI may cause VLST. The second mechanism is coronary rupture after neoaatherosclerosis of the intima. The third mechanism is delayed healing of a ruptured atheroma under stent struts and the covering by a thin neointima because of the drug effect, with finally rupture of the thin-capped fibroatheroma. In this case, on IVUS, we found an uncovered and mal-apposed stent strut from the intima which is consistent with the mechanisms mentioned above. Even though a follow-up coronary angiography in 2012 showed patent flow, at that time, the stent was probably mal-apposed. This stent thrombosis was finally triggered by discontinuation of the antiplatelet therapy, and that was done for a tooth extraction.

Nowadays before and after stent deployment, IVUS is performed in order to confirm the lesion characteristics, neointimal coverage, presence of stent underexpansion and stent edge dissection of coronary vessel. Additionally we recommend that, if possible, when follow-up coronary angiography is to be done, attempts should be made to visualize the stent strut and coronary anatomy with IVUS in order to correctly appose the stent and identify the status of the covered neointima because there are many cases presented with AMI accompanying vasospasm causing malposition and technically limitation of confirm the stent sturts status.

The period requiring DAPT is longer with DES, due to the delayed neointimal coverage. Currently, the optimal duration of DAPT after stent implantation is controversial. Most of the evidence suggests that 12 months of DAPT is a reasonable goal. We recommend to make a decision of optimal duration of DAPT by individual IVUS findings and stent type. Incidence of stent thrombosis is different from stent type. In two meta-analysis reports, there was no significant difference in rates of stent thrombosis comparing first generation DES to BMS, but the risk of stent thrombosis with everolimus eluting stent is lower than first generation DES [7,8],

![Fig. 3. Intravascular ultrasound (IVUS) revealed concentric fibrofatty plaques at the proximal reference vessel (A). At the site of the developed stent thrombosis, we found an uncovered stent (B) and incomplete apposition of the stent (C). Using IVUS, we identified diffuse hyperplasia of the neointima at the distal part of the stent (D).](http://jsms.sch.ac.kr)
Due to advancement in stent structure, polymer, drug, there are expert’s suggest that using DAPT less than a year is sufficient. Furthermore, unnecessary long period of DAPT use can cause adverse effects including bleeding. In this view, we should carefully decide treatment duration of DAPT by individual findings of IVUS and stent type.

To the best of our knowledge, there have been only a few case reports of VLST caused by neointimal rupture within 7 years of a successful stent implantation [9,10]. In our article, we report a case of VLST 8 years after implantation of the DES (756 days longer than the above case), which makes it significantly unique from other cases. Noticeably, the mechanism of occlusion was a mal-apposed stent and delayed neointimal coverage that occurred as late as 8 years after stent implantation. Taking into consideration the above facts, we recommend follow-up coronary angiography and IVUS to confirm stent under-expansion and make a decision of optimal duration of DAPT by individual IVUS findings and stent type.

REFERENCES