Very Late Bare Metal Stent Thrombosis Presenting with Acute Myocardial Infarction: 14 Years after Coronary Stenting

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Abstract

Fourteen years previously, a 67-year-old man underwent percutaneous coronary intervention (PCI) for proximal left anterior descending artery lesion with a bare metal stent (BMS) for acute myocardial infarction (AMI) and attained an excellent result. Ticlopidine (200 mg) was administered for one month and 100 mg of aspirin was daily has been continued. One year after PCI, coronary angiography showed no restenosis. However, 14 years after PCI, he suffered from AMI due to stent thrombosis. Intracoronary aspiration thrombectomy and implantation of a drug-eluting stent were successful. This report demonstrates evidence of a very late case of stent thrombosis with the use of BMS.

Key words: bare metal stent, late stent thrombosis, acute myocardial infarction

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Introduction

Coronary stenting has revolutionized the practice of interventional cardiology. It has dramatically improved upon the acute procedural success and also reduced the restenosis rates observed with balloon angioplasty alone (1).

Although rare, the incidence of stent thrombosis is associated with a high frequency of major adverse clinical consequences. Its estimated 30-day mortality ranges from 20 to 48% and can cause myocardial infarction in 60-70% of patients (2).

The most recently accepted definition established by the Academic Research Consortium classifies stent thrombosis as: early (occurring within 30 days), late (30 days to 1 year), or very late (after 1 year). Very late stent thrombosis has been reported following drug-eluting stent implantation with rates up to 0.3-0.6% per year and has been attributed to delayed strut endothelialization (2, 3). It is distinctly unusual with the use of BMS, because the stent endothelialization is considered to be completed four weeks after the intervention (4).

Case Report

A 67-year-old male with hypertension and hyperlipidemia was referred for anterior acute myocardial infarction (AMI). Coronary angiography (CAG) showed a significant stenotic lesion in the proximal-segment of the left anterior descending coronary artery (LAD) (Fig. 1A). He had undergone percutaneous coronary intervention (PCI) for proximal LAD lesion with a bare metal stent (Palmaz-Shatz stent 3.0×15 mm) and had an excellent angiographic result (Fig. 1B). The peak creatine kinase value was only 564 IU/L at this first infarction. The left ventricular ejection fraction at this time was 64% by ultrasound cardiogram, and anterior wall motion was well preserved, suggesting that the viability of the infarcted region affected by first attack was maintained. Ticlopidine (200 mg) was administered for one month and 100 mg daily of aspirin was daily continued. Other medications were 10 mg of pravastatin and 20 mg of nifedipine. His drug compliance was satisfactory, and the level of blood pressure was controlled within normal range. Six months later, and one year later after implantation of the stent, CAG showed no restenosis (Fig. 1C). On quantitative coronary ar-

Figure 1. Coronary angiography. (A) Baseline coronary angiography, 14 years previously, showing a significant lesion in the proximal-segment of the left anterior descending (LAD) coronary artery. (B) Postprocedural coronary angiography after percutaneous coronary intervention (PCI) with a bare metal stent in the LAD. (C) Coronary angiography 12 months after the PCI showing no significant stenosis. (D) Coronary angiography 14 years after the PCI showing total occlusion at the proximal edge of the stent. (arrow) (E) Postprocedural coronary angiography after the recent PCI with the drug eluting stent in the LAD.

Figure 2. On admission after the second MI attack, ECG showed ST elevation in V1-4 leads.

terangiography (QCA), the % diameter stenosis (%DS) of the proximal stent edge was 24%. Fourteen years later after implantation of stent, he developed acute chest pain and his resting ECG showed ST elevation in V1-4 leads (Fig. 2). He was immediately referred for angiography the same day. On

admission, the level of LDL cholesterol was 136 mg/dL and the level of highly sensitive C-reactive protein (hs-CRP) was 1.2 mg/dL. CAG showed total occlusion at the entrance of the proximal stent in the LAD lesion (Fig. 1D, arrow). After positioning of a 6 French FL 3.5 guiding catheter (Boston

Figure 3. After stent implantation, the ST elevation of chest leads normalized immediately, and proper ST resolution was obtained.

Figure 4. Intravascular ultrasound image. IVUS image confirmed a tight fibrotic in-stent hyperplasia and thrombus.

Scientific, Watertown, MA), a 0.014 in. Runthrough Guidewire (Terumo, Tokyo) was passed across the occlusion. An intravascular ultrasound (IVUS) catheter (CVIS-device, Boston Scientific) was then employed to reveal the underlying cause of this occlusive lesion. The IVUS catheter was advanced distal to the lesion, and imaging was performed retrograde through the proximal reference artery at a pullback speed at 0.5 mm/s automatically. IVUS image confirmed a tight fibrotic in-stent hyperplasia and thrombus (Fig. 4). Then, intracoronary aspiration thrombectomy was successful. After that he was treated with balloon angioplasty and a drug-eluting stent (TAXUS Liverte stent 3.0×28 mm, Boston Scientific) for LAD, and TIMI 3 coronary flow was obtained (Fig. 1E). After stent implantation, the ST elevation of chest leads was normalized immediately, in other words, proper ST resolution was achieved (Fig. 3). Few days later, the pathological findings revealed that the aspiration material was fresh thrombus. We described a patient with acute myocardial infarction who had late stent thrombosis 14 years after stent implantation.

Discussion

Stent thrombosis is a catastrophic complication that frequently presents as an acute myocardial infarction and/or sudden cardiac death. Stent thrombosis has in large part been overcome by the use of high-pressure inflation and the aggressive use of antiplatelet agents such as aspirin and either ticlopidine or clopidogrel (5).

Because bare metal stent endothelialization is considered to be complete after four weeks of the intervention, late stent thrombosis is distinctly unusual with the use of BMS (4).

On the other hand, late stent thrombosis is increasingly being recognized as a complication of drug-eluting stents where it may be related to delayed endothelialization (6). Brachytherapy trials have also identified the phenomenon of late stent thrombosis, because brachytherapy caused slow progression of pathophysiologic process of the neointimal hyperplasia (7).

It is reported that late stent thrombosis with drug-eluting stent occurs with an incidence of 0.3% to 1.0% (2, 3). Some recent reports indicated that the incidence of stent thrombosis did not differ significantly between patients with drug-eluting stents and those with bare-metal stents (7-9). Independent predictors of stent thrombosis were premature antiplatelet therapy discontinuation, renal failure, bifurcation lesions, diabetes, and a lower ejection fraction (2, 5).

The incidence of definite very late stent thrombosis after BMS implantation seems to be very low, as only 21 cases could be identified in the Medline literature, despite the deployment of millions of BMS over the past decade. To the best of our knowledge, very late thrombosis of BMS has been reported within 10 years after stent implantation (8).

Our report demonstrated evidence of the latest case of stent thrombosis with the use of BMS.

The pathophysiology of very late stent BMS thrombosis is poorly defined. Of course we should consider the potential existence of conditions, which may facilitate the formation of thrombus, because the present case experienced acute coronary syndrome twice. However, he has no factor to fa-

cilitate thrombus formation, such as collagen disease, abnormality of coagulation, dehydration, renal insufficiency. We could not deny the possibility of arrhythmia, such as paroxysmal atrial fibrillation. Presumed causes of bare metal stent thrombus, both early and late, include noncompliance with antiplatelet agents, exercise-induced procoagulant state, brachytherapy, small stent size, and underdeployment of stent (9).

In the present case, IVUS image confirmed a tight fibrotic in-stent hyperplasia and thrombus. By the pathological finding, the aspiration material was fresh thrombus. These data suggest that BMS restenosis was the main pathologic mechanism leading to abrupt thrombotic vessel closure. The insufficient control of LDL cholesterol might lead to the rupture of the lipid-rich plaque. Further, the level of hs-CRP of this case was elevated at the time of second MI attack. Kimura et al described that pathological analysis of the stented human coronary arteries demonstrated heavy infiltration of lipid-laden macrophages around the struts beyond 5 years after coronary stenting, which indicating chronic inflammatory reactions. This chronic inflammation might induce late atherosclerotic progression of the treated le-

sions (10). In the present case, this chronic inflammation might also lead to the luminal renarrowing beyond 10 years and plaque rupture. In-stent restenosis has been commonly believed to be a benign process due to its stable clinical presentation, as a result of slow and progressive neointimal proliferation and smooth muscle cell migration. However, as a matter of fact, in-stent restenosis presents as acute myocardial infarction in 3.5 to 19.4% of patients, and as such is not always a benign process (8, 11).

We described a case of late coronary stent thrombosis which manifested as AMI 14 years after stenting. This case is, to our knowledge, the latest case of stent thrombosis with the use of BMS.

Conclusion

To the best of our knowledge, the most delayed case of BMS thrombosis is ten years after stent implantation. In the present case, late thrombosis occurred about fourteen years after implantation of bare metal stent implantation. Our report demonstrated evidence of the latest case of stent thrombosis with the use of bare metal stent.

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