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A Case of Very Late Simultaneous Bare Metal Stent Thrombosis with Complete Atrio-Ventricular Block

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Abstract

Percutaneous Coronary Intervention (PCI) is the most commonly used revascularization method in treatment of Coronary Artery Disease. Stent thrombosis is a feared complication of PCI yet. Very late stent thrombosis is defined as angiographically confirmed stent thrombosis occurring more than 1 year. In our report we described a rare case of simultaneous very late double-arterial stent thrombosis following bare metal stent implantation, presented as acute anterior and inferior myocardial infarction with complete atrio-ventricular block, which was treated with temporary pacemaker and angioplasty.

Keywords: Very late stent thrombosis, bare metal stent, simultaneous.

Introduction

The invention and usage of stents has made percutaneous coronary intervention (PCI) a safe, effective, and feasible revascularization method. Stent thrombosis (ST), a man made disease, is the Achilles tendon of PCI. According to the Academic Research Consortium, ST is considered as acute when occurring between 0 and 24 hr after stent implantation, subacute between 24 hr and 30 days, late between 30 days and 1 year, and very late after 1 year (1).

Case Report

A 64-year-old male presented with new onset angina and the diagnosis was acute anterior myocardial infarction (MI). The patient had a history of hypertension and hyperlipidemia. The coronary angiogram revealed presence of three-vessel disease with total occlusion of left anterior descending coronary artery (LAD) at the level of first diagonal branch, and severe mid right coronary artery (RCA). Immediate PCI of LAD lesion was made with 3.0x15 mm Ephesos bare metal stent (BMS) (Alvimedica, Istanbul, Turkey) at 10 atm and TIMI III flow was restored. He was taken to the coronary care unit (CCU) and intravenous heparin and tirofiban infusion was given for 24 hours because of the high thrombus burden. Echocardiogram showed moderate left ventricular systolic depression with an ejection fraction of 42%. He was discharged with dual antiplatelet therapy (Aspirin 300 mg/day and Clopidogrel 75 mg/day). Four months later, 2.5x13 mm and 3.5x15 mm Ephesos stents was electively deployed to the lesions before crux and before RV branch of RCA, respectively.

12 months later he presented to emergency room with anterior MI and he was immediately taken to catheterization laboratory and coronary angiogram revealed LAD in-stent thrombosis. Intracoronary tirofiban bolus was performed. Balloon dilatation did not improve

arterial patency, and then 3.0x13mm Lekton Motion BMS (Biotronik, Switzerland) was deployed at 20 atm with successful patency. Clopidogrel resistance assessment was not an option so clopidogrel dosage was doubled.

The patient did well for 3 years until he presented to emergency room with severe crushing retrosternal chest pain, nausea and vomiting. Electrocardiogram revealed complete atrio-ventricular (AV) block and diffuse ST segment elevations on anterior and inferior derivations without right ventricular involvement. His arterial blood pressure and heart rate were 90/70 mmHg and 40 bpm, respectively. Physical examination showed a 1–2/6 grade systolic murmur auscultated in the apical region, otherwise normal. Because of the unstable hemodynamics of the patient, he was emergently transferred to catheterization laboratory and a temporary pacemaker was implanted via the right femoral vein. Coronary angiogram showed dual in-stent occlusion of the LAD and RCA. Although he was on clopidogrel and aspirin treatment, 600 mg clopidogrel was loaded and 300 mg aspirin was chewed. LAD lesion was passed with floppy guidewire and dilated with Biotronik 3.25x20 mm balloon at 20 atm with an optimal angiographic result. Then RCA lesion was passed with floppy guidewire and dilated with Biotronik 3.25x20 mm balloon at 16 atm. Intravenous heparin and intracoronary tirofiban bolus was given and TIMI III flow was obtained on both coronary arteries. The patient was monitored at CCU and tirofiban infusion was continued 48 hours. His rhythm was turned to sinus rhythm with right bundle branch block and temporary pacemaker was removed after 24 hours. The patient had an uncomplicated hospital course and four days after admission patient was discharged with the prescription of aspirin 300 mg/day, clopidogrel 150 mg/day, cilostazol 100 mg/day, atorvastatin 20 mg/day, perindopril 10 mg/day and carvedilol 25 mg/day.

Discussion

Stent thrombosis is a rare but fatal complication following coronary stent implantation, with an incidence of 0.6% to 1.2% (2). In general the incidence of late and very late ST was estimated at 0.5% to 1.5% during the first year and 0.5% per every following year (3) with a 6-month mortality of 20-25% and with a 60% risk of nonfatal myocardial infarction (4,5). Causes of bare metal ST can be grouped into four categories: patient related factors (acute coronary syndrome as indication for index PCI, heart failure, low ejection fraction, renal failure, insulin dependent diabetes mellitus, active malignancy); lesion related factors (total stent length, multiple stents, bifurcation lesion, small vessel); procedural factors (undersizing, underexpansion, malapposition, dissection, stent overlap, lack of intravascular ultrasound guidance); drug related factors (discontinuation of antiplatelets, nonresponse to antiplatelets) (3).

In our case, the patient was compliant to his double anti-platelet medications. The angiographic result of the index intervention showed no residual stenosis or dissection, decreasing the possibility of inadequate stent apposition because of several post-stent dilatations with high pressure. During the ST event, the patient was hemodynamically unstable, could be causing secondary thrombosis of one stent due to hypotension from an initial single ST. According to optical coherence tomography (OCT) findings, Bennett et al. suggested that atheromatous neointimal degeneration, followed by plaque rupture, exhibits an important role in very late ST in BMS (6).

But as we did not perform intravascular ultrasound (IVUS) or OCT before coronary angioplasty, we could not comment on the exact mechanism of ST. Nevertheless, more ST elevation in inferior leads and AV block may suggest us the responsible lesion for the patient's current status was RCA ST. We performed PTCA but did not implant stent because stents were well expanded and no residual dissection was present; and several studies have demonstrated that implanting an additional coronary stent at the time of the ST is associated with an increased risk of recurrent ST (7,8). We used tirofiban because of the protective effects against ST recurrence, as glycoprotein IIb/IIIa therapy during PCI for ST is strongly recommended (8,9). Prasugrel or ticagrelor was not available in our country. The addition of cilostazol to standard dual antiplatelet therapy appears to reduce angiographic restenosis and target lesion revascularization rate after coronary stent implantation without increasing the risk of bleeding (10).

In conclusion, although very late ST seems to be a complication of DES, it is an increasingly encountered complication that can also occur with BMS. Our case highlighted that the use of multiple stents in multiple coronary artery lesions should be undertaken cautiously. During (IVUS and OCT guidance) and after stent implantation (double-antiplatelet therapy and diabetes control) precautions should be taken to prevent ST because of the catastrophic outcomes of simultaneous stent thrombosis in multiple vessels.

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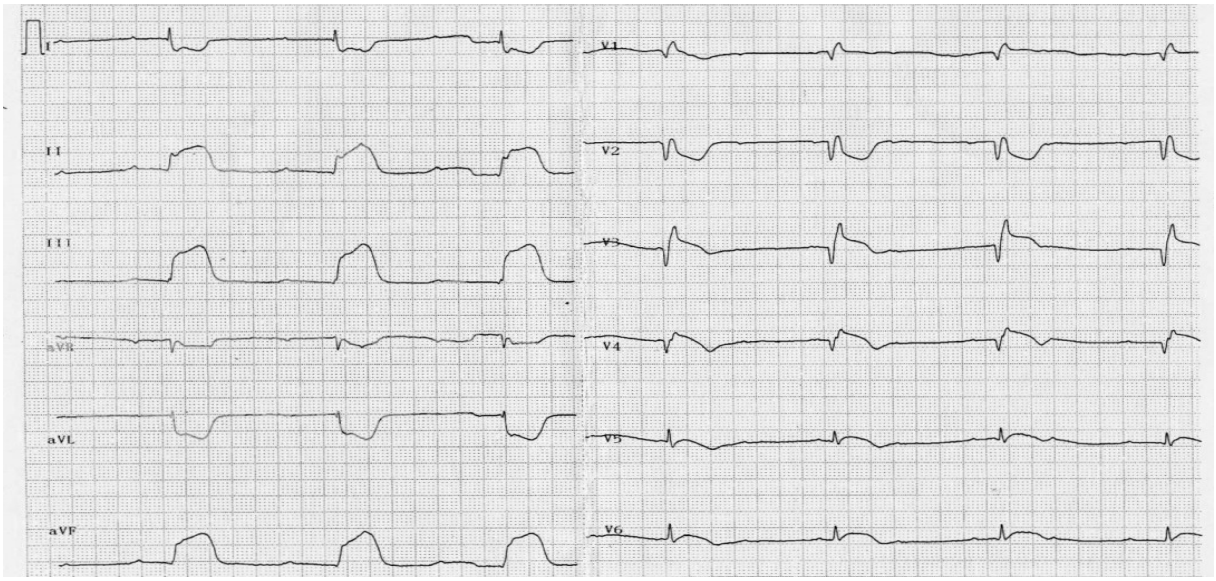

Fig. 1. Electrocardiography showing ST-segment elevation in Leads II, III, aVF, and V3–V6 and complete atrio-ventricular block suggesting inferior and anterior acute myocardial infarction.

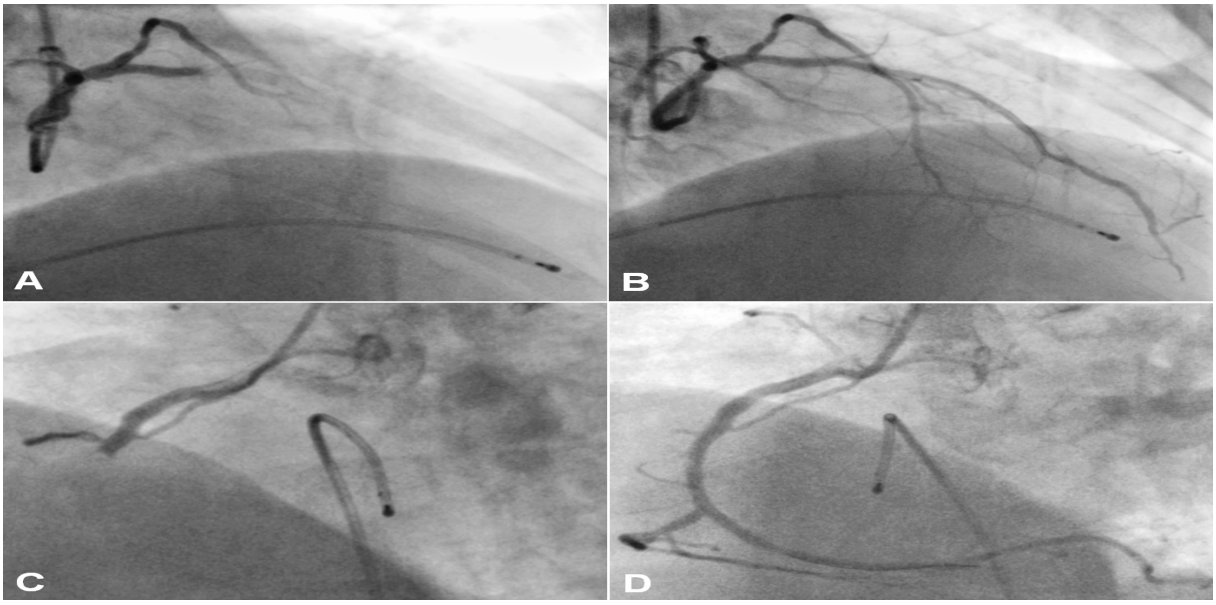

Fig. 2. Coronary angiogram showing total simultaneous occlusion of bare metal stents in left anterior descending artery (A) and right coronary artery (C). Post-procedural angiographic results after balloon angioplasty in left anterior descending artery (B) and right coronary artery (D).

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