Late bare metal stent thrombosis

Geç düz metal stent trombozu

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Late stent thrombosis is very rare in bare metal stents. We report a 72-year-old male patient who developed late thrombosis of a bare metal stent implanted in the left main coronary artery (LMCA). The patient presented with cardiogenic shock 350 days after the first stent implantation. Coronary angiography showed total occlusion of the stent. Following the first balloon predilatation of the lesion, a flow in the LMCA was observed, but there was no flow in the left anterior descending (LAD) artery. Then, a bare metal stent was implanted into the LAD. Although the flow was maintained and all inotropic support continued, hypotension persisted. Angiography of the right coronary artery demonstrated 90% stenosis at the same location which had been observed as a noncritical lesion during the first percutaneous coronary intervention. As the patient was in shock, the right coronary artery was also stented and TIMI 3 flow was obtained. However, the patient developed cardiac arrest and died despite repeated efforts of cardiopulmonary resuscitation. It was learned that the patient had undergone an urological operation for bladder stone nine days before, for which both aspirin and clopidogrel were discontinued six days before the operation. Only aspirin was reinitiated three days after the procedure. He then presented to our hospital with cardiogenic shock on his first day after discharge.

Key words: Angioplasty, transluminal, percutaneous coronary; platelet aggregation inhibitors/therapeutic use; stents; thrombosis/etiology.

Geç stent trombozu düz metal stentlerde oldukça nadirdir. Bu yazıda, sol ana koroner artere takılan düz metal stentte geç tromboz gelişen 72 yaşında bir erkek hasta sunuldu. Hasta ilk stent yerleştirilmesinden 350 gün sonra kardiyojenik şokla yatırıldı. Koroner anjiyografide stentin tamamen tıkalı olduğu görüldü. Lezyon bölgesinin balonla genişletilmesinden sonra sol ana koroner arterde akım sağlandı. Sol ön inen arterde akım görülmemesi üzerine bu lezyon için de düz metal stent takıldı. Sürekli akım sağlanmış olmasına ve inotropik desteğin sürdürülmesine rağmen hastanın hipotansiyonunda düzelme olmadı. Bunun üzerine yapılan sağ koroner arter anjiyografisinde, ilk perkütan koroner girişim sırasında kritik olarak değerlendirilmeyen bir lezyon yerinde %90 daralma görüldü. Hasta şokta olduğundan, sağ koroner artere de stent takıldı ve TIMI 3 akımın sağlandığı görüldü. Bu çabalara rağmen hastanın kalbi durdu ve tekrarlayan kardiyopulmoner canlandırma girişimlerine yanıt alınamayarak hasta kaybedildi. Daha sonra, hastanın dokuz gün önce mesane taşı için bir üroloji ameliyatı geçirdiği ve bu işlemden altı gün önce almakta olduğu aspirin ve klopidogrel tedavisinin kesildiği öğrenildi. Bu ameliyattan üç gün sonra sadece aspirine yeniden başlanmıştı. Bunu izleyen süreçte, hasta taburcu olduktan sonraki ilk gün kardiyojenik şokla hastanemize getirilmişti.

Anahtar sözcükler: Anjiyoplasti, translüminal, perkütan koroner; trombosit agregasyon inhibitörü/terapötik kullanım; stent; tromboz/etyoloji.

Stent thrombosis is a serious complication of coronary stents that often results in myocardial infarction or death. Six-month mortality rates after stent thrombosis range from 9 to 21 percent with bare metal stents (BMS).[1,2] The occurrence of late stent thrombosis is rare with BMS.[3] Late stent thrombosis has received considerable attention in recent years because of its association with high morbidity and mortality rates and its unpredictable nature in the absence of risk factors.

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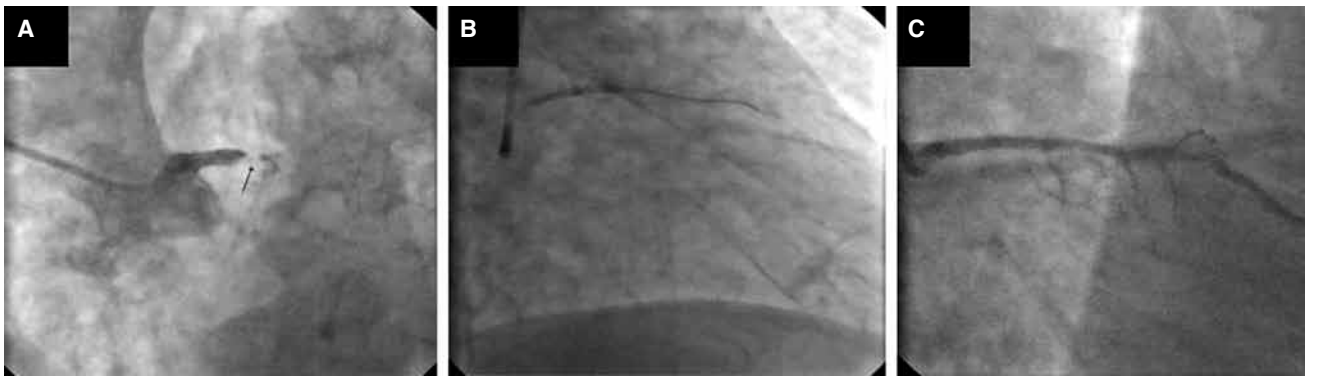

Figure 1. Coronary angiograms during the first coronary intervention. **(A)** Right anterior oblique caudal view demonstrating total occlusion of the left main coronary artery. **(B)** After crossing the lesion with a soft guidewire, balloon angioplasty was performed. **(C)** Implantation of a bare metal stent resulted in TIMI 2-3 flow.

We report a case of late BMS thrombosis that presented as cardiogenic shock 350 days after implantation for left main coronary artery (LMCA) disease. The reason for stent thrombosis was cessation of antiaggregant medications for a noncardiac surgery.

CASE REPORT

In April 2006, a 72-year-old male presented with severe angina of four-hour duration, dyspnea, and diaphoresis. The patient had hypertension for 25 years and was a smoker (35 packs/year). He had no history of angina or a previously diagnosed coronary artery disease. On admission, he was confused, dyspneic, and orthopneic. His blood pressure was 85/55 mmHg, heart rate was 130 beats/min with a regular rhythm, respiratory rate was 32/min, and arterial oxygen saturation was 75%. On auscultation, inspiratory crackles were heard at both lung bases and the middle zones, and expiration was rather prolonged. An S_3 gallop was heard. The electrocardiogram demonstrated ST-segment elevation of 2 mm in leads V2-6. Following administration of 300 mg aspirin, 600 mg clopidogrel, and 5000 U heparin, the patient was transferred immediately to the catheterization laboratory under inotropic support. Coronary angiography with a 7 French JL 4.0 guiding catheter (Cordis, Johnson & Johnson, Miami, USA) showed total occlusion of the LMCA and there was no antegrade flow (Fig. 1a). There was only a retrograde flow of TIMI 0-1. A 0.014'' soft guidewire was used to pass through the left anterior descending (LAD) artery, but we could not succeed in passing through the circumflex artery. Balloon angioplasty with a 2.5 x 20 mm Sprinter balloon (Medtronic, Minneapolis, USA) inflated at 10 atm failed to restore the antegrade flow. Then, the same balloon was reinflated at 8 atm for the second and third times in the proximal and mid LAD, respectively. With these redilatations, an antegrade flow of TIMI 2 was restored. The circumflex artery could not be protected due to failure to pass the guidewire. A 3.0 x 28 mm BMS (Lekton Motion stent, Biotronik, Switzerland) was implanted and inflated at 14 atm between the mid LMCA and mid LAD (Fig. 1b), resulting in TIMI 2-3 antegrade flow (Fig. 1c). Angiogram of the right coronary artery documented a dominant coronary artery with a noncritical lesion. Following a bolus administration, tirofiban infusion

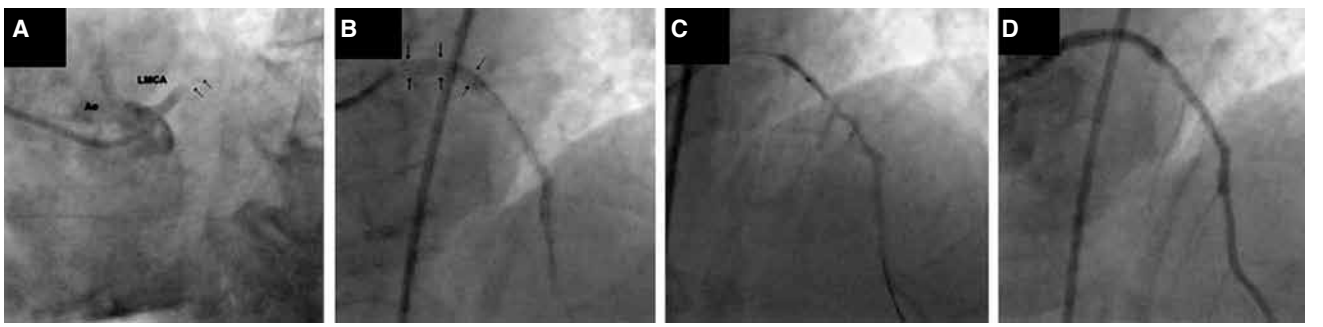

Figure 2. Coronary angiograms during the second coronary intervention. **(A)** Right anterior oblique cranial view demonstrating thrombosis of the stent previously implanted (arrows). **(B)** Following the first balloon predilatation of the lesion, a flow in the left main coronary artery was restored, but there was no flow in the left anterior descending (LAD) artery. **(C)** A bare metal stent was implanted into the LAD and **(D)** TIMI 2 flow was obtained.

was initiated postprocedurally and was continued for 48 hours. On the third day of admission, there was no requirement for further inotropic support. His predischarge echocardiogram demonstrated an ejection fraction of 30-35%. He was discharged on the eighth day on dual antiplatelet therapy. As he had first-degree AV block with a PR interval of 0.26 seconds, beta-blocker could not be used. As a possible clopidogrel resistance would lead to life-threatening complications, we preferred to start clopidogrel at a dosage of 150 mg per day for the first month. Aspirin was initiated at a dosage of 300 mg per day. On his first outpatient visit one month later, his functional capacity was NYHA I-II and he had no angina. Clopidogrel dose was lowered to 75 mg per day and carvedilol 6.25 mg per day was started. The patient remained asymptomatic for six months, and on his last outpatient visit, myocardial perfusion scintigraphy showed no ischemia. He was recommended to continue dual antiplatelet therapy.

In April 2007, 350 days after BMS implantation, the patient presented again with cardiogenic shock. His blood pressure was 60/40 mmHg and his electrocardiogram demonstrated ST-segment elevation in leads V2-6. He was immediately intubated and taken to the catheterization laboratory. After implantation of an intraaortic balloon pump, coronary angiography with a 7 French JL 4.0 guiding catheter (Cordis, Johnson and Johnson) showed total occlusion of the stent previously implanted (Fig. 2a). Following the first predilatation of the lesion with a 2.5 x 20 mm Maverick balloon (Boston Scientific, MA, USA) inflated at 10 atm, a flow in the LMCA was observed, but there was no flow in the LAD (Fig. 2b). Then, a 2.5 x 25 mm BMS (Lekton Motion stent, Biotronik) at 10 atm was implanted into the LAD (Fig. 2c, d). Although the flow was maintained and all inotropic support continued, hypotension persisted. Angiogram of the right coronary artery demonstrated a 90% stenosis at the same location which had been observed as a noncritical lesion on his first angiogram. As the patient was in shock, a 3.0 x 18 mm BMS (Lekton Motion stent, Biotronik) at 14 atm was also successfully implanted into the right coronary artery and TIMI 3 flow was maintained. However, the patient developed cardiac arrest and died despite repeated efforts of cardiopulmonary resuscitation.

It was later learned that the patient had undergone an urological operation for bladder stone nine days before. Despite preoperative cardiac evaluation, both aspirin and clopidogrel were discontinued six days before the operation because of concerns about the possibility of uncontrollable perioperative bleeding. Only aspirin was reinitiated three days after the procedure. He then presented to our hospital with cardiogenic shock on his first day after discharge.

DISCUSSION

Stent thrombosis is a feared complication of coronary stents that often results in myocardial infarction or death. Depending on the time of occurrence after percutaneous coronary intervention and implantation, stent thrombosis is classified as acute (within 48 hours), subacute (between 2-30 days), late (after 30 days), and very late (after the first year). About 80% of BMS thromboses occur within the first two days and, to a lesser degree, in the first month of the implantation. The use of dual antiplatelet therapy has decreased this incidence to quite acceptable levels. Clinical trials on the use of BMS in mostly noncomplex lesions reported the incidence of stent thrombosis as less than 1% with the use of dual antiplatelet therapy and high-pressure postdilation, with increases to higher rates (2% to 3%) in more complex patients and lesions.[1] Late stent thrombosis is very rare in BMSs.[3] An angioscopic study demonstrated that reendothelization was almost totally completed in all BMSs within 3 to 6 months.[2]

The most important risk factor for stent thrombosis still appears to be premature cessation of antiplatelet therapy.[4] Based upon data from the PCI-CURE trial, it has been recommended that, if not contraindicated, clopidogrel should not be discontinued for one year.[5] The most common reasons for premature cessation of dual antiplatelet therapy seem to be noncompliance, bleeding, allergic reactions, and noncardiac surgery.

Before noncardiac surgery, the evaluation of patients bearing coronary stents is vital. Besides the clinical condition of the patient, the type of the stent and the time of implantation should be considered carefully. The problem is great especially in patients undergoing noncardiac surgery within the first month of intervention. The clinicians should balance the risk for stent thrombosis associated with discontinuation of the antiaggregants against the risk for excessive intraoperative and postoperative bleeding related with the use of antiplatelets.

In patients who are candidates of noncardiac surgery, the risk for stent thrombosis should be minimized by reserving percutaneous coronary intervention for only high-risk patients. Low-risk patients should be directed to noncardiac surgery just following stabilization with optimal medical therapy.[4] Drug-eluting stents should not be preferred in patients for whom noncar-

diac surgery is planned within 12 months, as dual antiplatelet therapy should not be discontinued ideally for one year.[6] If noncardiac surgery is planned between 6 to 12 weeks of stent implantation, a BMS should be preferred as cessation of antiplatelets after the sixth week seems to be safe for BMS.[7] Dual antiplatelet therapy should not be interrupted within the first month of any coronary intervention and in anytime in high-risk patients.[8] In case of cessation of antiplatelets, short-acting intravenous glycoprotein IIb/IIIa inhibitors may be used perioperatively as bridge therapy and all antiaggregants should be reinitiated postoperatively as soon as possible.[7] Duration of discontinuation of antiplatelets should be limited to 7 to 10 days.

A variety of additional risk factors for stent thrombosis have also been identified. Patient-related risk factors can be listed as diabetes mellitus, low ejection fraction, renal failure, and stenting during acute myocardial infarction. Lesion-related risk factors include persistent dissection, suboptimal stent deployment (underexpansion), multiple stenting, small vessel caliber, long lesion, small final minimal lumen area, brachytherapy, and neointimal hyperplasia.[9]

Delayed endothelization seems to be the major predisposing mechanism of late stent thrombosis for drug-eluting stents, whereas neointimal hyperplasia is a more prominent risk factor for BMS implantation.[10] In our case, excessive neointima formation may be the reason of failure in the second intervention.

We cannot exclude the possibility of suboptimal stent deployment in the baseline procedure for the development of late stent thrombosis in our case. Besides, a stent with a diameter of 3 mm may be undersized for the LMCA. Intravascular ultrasonography was necessary for the exclusion of the possibility of underexpansion; however, it was not available at that time.

The most important predictor of stent thrombosis is premature cessation of antiplatelet therapy. Although the duration of dual antiplatelet therapy is clearly defined in current guidelines, each patient should also be evaluated individually. The duration of dual antiplatelet therapy may be prolonged in patients with additional risk factors such as low ejection fraction, diabetes mellitus, or stenting during acute myocardial infarction. Thus, if noncardiac surgery is considered, preoperative evaluation of stented patients is crucial. All clinicians should be aware of this important problem.

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