

Late stent thrombosis after paclitaxel-eluting stent placement in a patient with essential thrombocytosis

Esansiyel trombositozlu bir hastada paklitaksel salınlı stent yerleştirme sonrası gelişen geç stent trombozu

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We report on a case of late stent thrombosis after drug-eluting stent placement in a patient with essential thrombocytosis. A 51-year-old male patient with a three-month history of paclitaxel-eluting stent placement to the left anterior descending artery presented with a complaint of severe retrosternal chest pain. A high platelet count (1,063,000/mm³) was detected two months prior to presentation, which was interpreted as essential thrombocytosis. He was on standard dual antiplatelet therapy (aspirin and clopidogrel). The electrocardiogram showed ST-segment elevation in leads V1-V6. Emergent coronary angiography revealed thrombotic total occlusion at the location of the paclitaxel-eluting stent. Balloon angioplasty was performed yielding a satisfactory result and TIMI 3 flow. Following the procedure, there was no chest pain. His platelet count was 388,000/mm³. He was discharged on medical therapy following an uneventful hospital course. Patients with essential thrombocytosis may not be eligible for drug-eluting stent placement.

Key words: Angioplasty, balloon, coronary; paclitaxel; stents; thrombocytopenia, essential/complications; thrombosis/etiology.

The introduction of coronary stents in 1987 has been the most important advancement in percutaneous coronary interventions. The development of drug-eluting stents (DES) is a major breakthrough as a potential solution for the restenosis problem. However, the problem of stent thrombosis associated with DES has emerged as a potential limitation of these stents, resulting from delayed endothelialization and enhanced platelet aggregation after DES implantation.

Essential thrombocytosis (ET) is an acquired myeloproliferative disorder characterized by sustained el-

Bu yazıda, esansiyel trombositozlu bir hastada ilaç salınlı stent yerleştirme sonrası gelişen geç stent trombozu sunuldu. Üç ay önce sol ön inen artere paklitaksel salınlı stent yerleştirilen 51 yaşındaki erkek hasta şiddetli retrosternal göğüs ağrısı yakınmasıyla başvurdu. İki ay öncesinde hastanın trombosit sayısı yüksek (1 063 000/mm³) bulunmuş ve durumu esansiyel trombositoz olarak yorumlanmıştı. Hasta standart ikili antitrombosit tedavi (aspirin ve klopidogrel) görmekteydi. Elektrokardiyografide V1-V6 derivasyonlarında ST-segment yükselmesi izlendi. Acil koroner anjiyografide paklitaksel salınlı stent yerinde trombotik tam tıkanıklık gözlemlendi. Balon anjiyoplasti uygulanan hastada başarılı sonuç alınarak TIMI 3 akım elde edildi. İşlem sonrasında hastanın göğüs ağrısı yakınması kayboldu. Trombosit sayısı 388 000/mm³ idi. Sorunsuz bir işlem sonrasında medikal tedavi verilerek hasta taburcu edildi. Esansiyel trombositozlu hastalarda ilaç salınlı stent kullanımı uygun olmayabilir.

Anahtar sözcükler: Anjiyoplasti, balon, koroner; paklitaksel; stent; trombositemi, esansiyel/komplikasyon; tromboz/etioloji.

evation of platelet number with a tendency for thrombosis and hemorrhage. Vascular occlusive events include major thrombotic events involving the cerebrovascular, coronary, and peripheral arterial circulation.

We describe a case of essential thrombocytosis that presented with late stent thrombosis after paclitaxel-eluting stent placement.

CASE REPORT

A 51-year-old, normotensive, nondiabetic male patient with a three-month history of paclitaxel-eluting stent

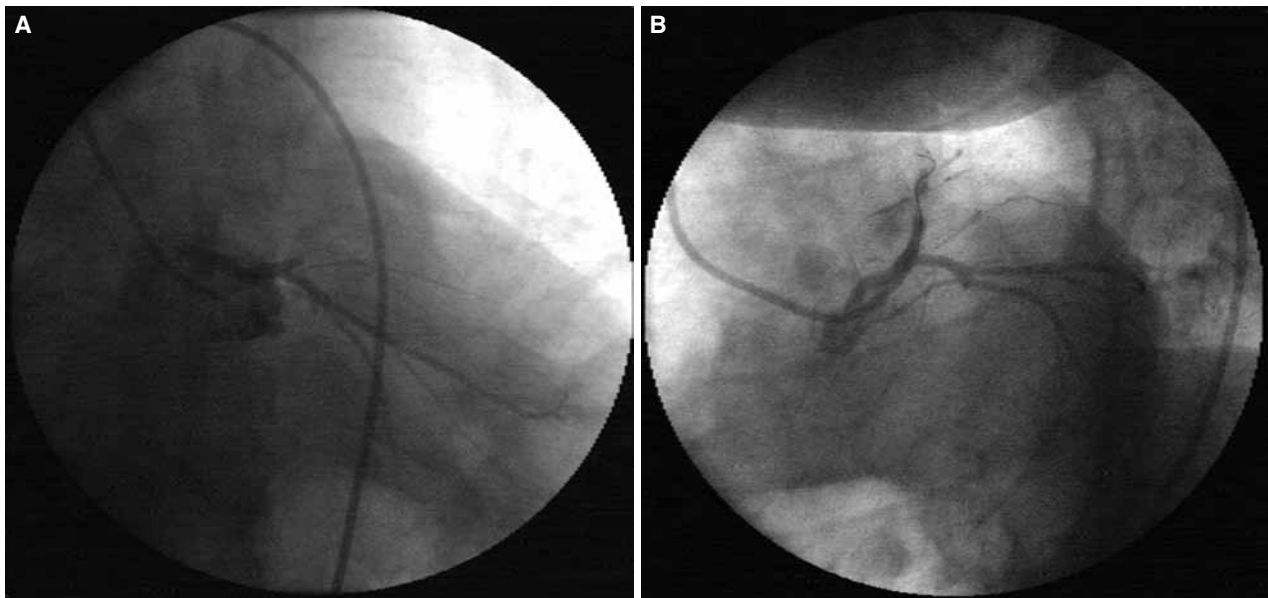


Figure 1. (A) Baseline angiography showing a thrombotic total occlusion of the proximal left anterior descending artery. **(B)** Crossing the occlusion with a floppy guide wire.

placement to the left anterior descending (LAD) artery in another hospital presented with a complaint of severe retrosternal chest pain within the past two hours. His medical history was significant for a high platelet count ($1,063,000/\text{mm}^3$) two months prior to presentation, which had been interpreted as essential thrombocytosis. He was given anagrelide for this disease. The patient was on standard dual antiplatelet therapy (aspirin and clopidogrel). On physical examination, he was diaphoretic, his blood pressure was 135/85 mmHg and pulse rate was 98 bpm. His electrocardiogram showed persistent anterior ST-segment elevation in leads V1-V6. The patient underwent emergent coronary angiography which revealed a thrombotic total occlusion of the proximal LAD artery at the location of the paclitaxel-eluting stent (Fig. 1a). The left main coronary artery was selectively cannulated using a 6 Fr Judkins left catheter. The proximal LAD in-stent occlusion was crossed with a 0.014-inch floppy guide wire (Fig. 1b). Balloon angioplasty was performed with a 3.0x20 mm balloon inflated to 8 atm. The angiographic result was satisfactory, with a residual stenosis of <20% (Fig. 2) and TIMI 3 flow was obtained upon completion of the procedure. The patient tolerated the procedure well and his chest pain was relieved completely. He was given tirofiban for 24 hours, followed by dual antiplatelet therapy (aspirin and clopidogrel) and initiation of metoprolol, lisinopril, and atorvastatin.

Postprocedure echocardiography showed mildly to moderately decreased left ventricular function with

an estimated ejection fraction of 40% and moderate hypokinesis involving the anterior, septal, and apical segments. His platelet count was $388,000/\text{mm}^3$. Electrolytes, creatinine level, liver function tests, activated protein C, lupus anticoagulant, anticardiolipin antibody, and homocysteine levels were in normal range. He was discharged from the hospital three days later following an uneventful hospital course.

DISCUSSION

When angioplasty was first developed in the 1970s, symptoms frequently recurred within six months of the procedure, indicating restenosis.^[1] Restenosis that once occurred in approximately 30% to 60% of patients within six months remains to be the Achilles' heel of coronary angioplasty. Stenting has effectively reduced restenosis rates to approximately 15% to 30% through prevention of elastic recoil and negative remodeling. However, stent implantation contributes to the development of neointimal hyperplasia, which acts as the main mechanism of in-stent restenosis.^[2] Bare metal stents (BMS) are associated with greater lumen losses in the late period compared to balloon dilatation alone. However, they provide greater acute gains in lumen diameter, which prevents neointimal production resulting in lower restenosis rates. The incidence of restenosis has been markedly reduced with the use of BMS and DES. Drug-eluting stents, in particular, provide localized therapy to the target lesion without systemic toxicity.^[3] However, in-stent restenosis continues to be a major problem for both BMS and DES,

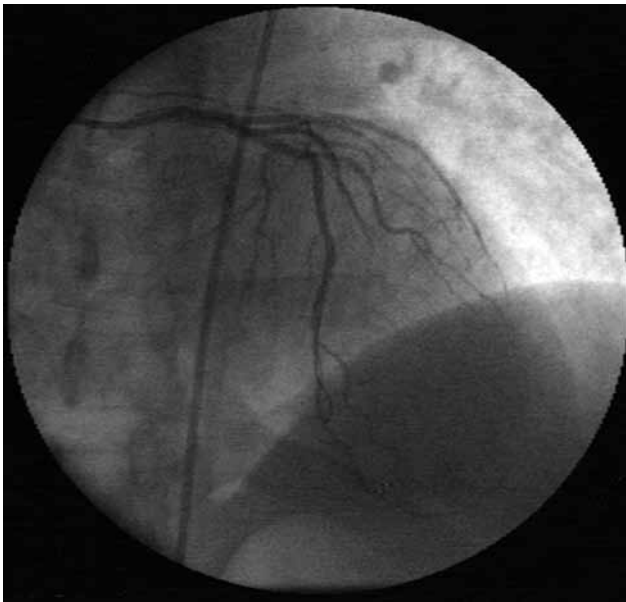


Figure 2. Final angiography showing a residual stenosis of less than %20.

with similar frequencies in early and late period. The overall incidence of stent thrombosis with DES at 9 to 12 months ranged from 0.5% to 0.7% in clinical trials,^[4] while registries reported higher rates (2 to 3 fold).^[5,6] In addition, thrombotic events occur more frequently with DES beyond 12 months (very late thrombosis).^[7] Although several patient-, lesion-, and procedure-related factors exist, the strongest independent predictor of stent thrombosis seems to be premature discontinuation of dual antiplatelet therapy.^[5-7]

Essential thrombocytosis is an acquired myeloproliferative disorder characterized by sustained elevation of platelet number, $>600.000/\mu\text{l}$ according to the Polycythemia Vera Study Group,^[8] with a tendency to thrombosis and hemorrhage.^[9] Some patients with ET are asymptomatic, others may experience vasomotor, thrombotic, or hemorrhagic disturbances. Vascular occlusive events include major thrombotic events involving the cerebrovascular, coronary, and peripheral arterial circulation. Thromboses of large arteries represent a major cause of mortality or may result in severe neurological disabilities, cardiac problems, or disturbances of peripheral arteries. The incidence of

coronary artery disease in ET has been reported as 9.4% in patients 40 years or older with a high incidence of acute myocardial infarction.^[8]

To our knowledge, this is the first report of late stent thrombosis after DES placement in a patient with ET. Patients with ET may be more susceptible to stent thrombosis; therefore, they may not be eligible for DES placement.

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