

Extremely late stent thrombosis after more than 7 years (2691 days) of sirolimus-eluting stent implantation 🧠

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Introduction

Stent thrombosis (ST) is a rare but potentially fatal complication of percutaneous treatment of coronary artery disease.

According to the Academic Research Consortium criteria and classification, ST can occur either acutely (within 24 h), subacutely (within 1-30 days), late (within 1-12 months), or very late (beyond 1 year) after stent implantation (1). The use of a new term “extremely late stent thrombosis” was suggested for cases of stent thrombosis that occur ≥ 5 years after stent implantation (2). Very late stent thrombosis (VLST) occurs more frequently with first-generation DES than with BMS, and the majority of VLST occur within 1-4 years of stent implantation. VLST is extremely rare after 5 years of stent implantation, with the first case being reported in 2009 (3). Few cases have been reported since 2009 until now. The longest reported intervening period between stent implantation and acute coronary event secondary to stent thrombosis is 11 years (4). The underlying pathophysiology of VLST is not completely understood and because duration of dual antiplatelet therapy is undetermined. Here we report the first case of an extremely late stent thrombosis presenting as a non-ST-elevation myocardial infarction (NSTEMI) from Turkey, which occurred 2691 days after implantation of a first-generation DES and 3 months after discontinuation of clopidogrel therapy.

Case Report

A 63-year-old male patient presented to our hospital with NSTEMI in August 2017. In March 2010, he underwent coronary

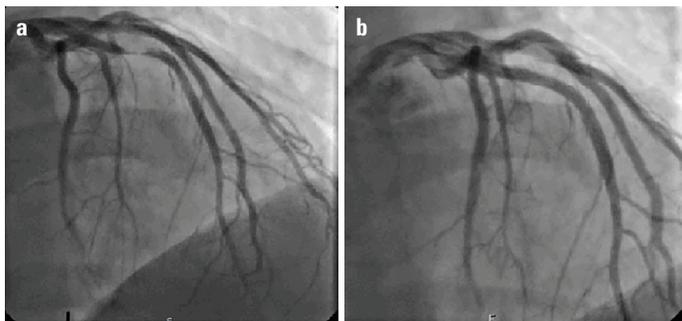


Figure 1. (a) Coronary angiography showing thrombotic occlusion in left coronary artery. (b) A drug-eluting stent (CYPHER) was deployed on the left coronary artery

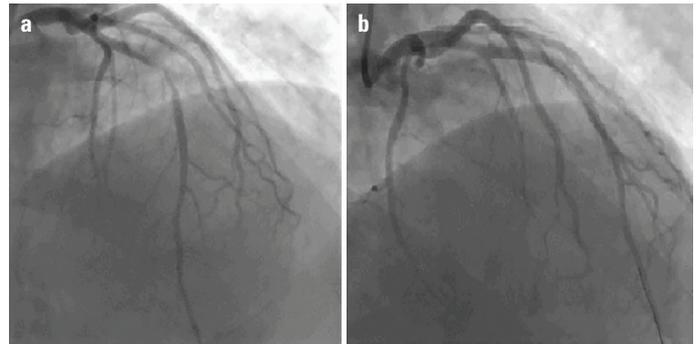


Figure 2. (a) A very late stent thrombosis causing subtotal occlusion distal to the drug-eluting stent in the left coronary artery. (b) Percutaneous coronary intervention was performed for stent thrombosis on the left coronary artery

angiography due to NSTEMI, which revealed normal left main and left circumflex artery, noncritical stenosis on the right coronary artery, and a critical thrombotic lesion on the mid left anterior descending artery (LAD) (Fig. 1a, Video 1). A 3.5×28-mm CYPHER sirolimus-eluting stent (C-SES) had been implanted on LAD after balloon angioplasty (Fig. 1b, Video 2). The patient had been discharged with dual antiplatelet therapy with 100 mg acetylsalicylic acid and 75 mg clopidogrel. He had continued this therapy for 7 years and remained asymptomatic.

In May 2017, he was subjected to a treadmill test under Bruce protocol for routine evaluation. The test was maximally negative and the patient was asymptomatic. After this evaluation, dual antiplatelet therapy was converted to monotherapy with acetylsalicylic acid.

At the current presentation, 3 months after the cessation of clopidogrel therapy, the patient was admitted to the emergency department with severe chest pain and was diagnosed with NSTEMI. He was treated with 180 mg ticagrelor and taken to the catheterization laboratory. Coronary angiography revealed a subtotal stenosis due to stent thrombosis on LAD (Fig. 2a, Video 3). New-generation drug-eluting stent (PROMUS element, 3.0×32 mm stent) was implanted into the lesion. Final coronary angiography showed TIMI-3 distal flow (Fig. 2b, Video 4). The patient recovered uneventfully and was discharged with a strict recommendation of dual antiplatelet therapy with acetylsalicylic acid and ticagrelor.

Discussion

VLST is an infrequent but clinically important sequela of stent implantation (5). The mechanism of VLST is not fully understood. Delayed arterial healing, ongoing vessel inflammation, neoatherosclerosis, and late stent malapposition are some of the mechanisms which are thought to play a role in VLST (6). According to Usui et al. (7) neointimal erosion is another potential cause of VLST.

In early experience with C-SES, incomplete neointimal coverage and insufficient expansion of the stent struts were reported by investigators (8). Specifically, compared with paclitaxel-eluting stents, SESs tend to be associated with more rapid neoatherosclerotic changes perhaps because of a difference in the polymer coating on the stent strut surface. SESs have been shown to promote the formation of lipid-rich yellow neointima, which is associated with unstable plaques that have a higher potential of rupture and thrombotic sequelae (9).

Importantly, the discontinuation of dual antiplatelet therapy in itself has not been shown to be a risk factor for VLST (10). However, our case questions this statement because VLST occurred >7 years after DES implantation and 3 months after discontinuation of clopidogrel therapy in our case.

Conclusion

In conclusion, should we recommend lifelong dual antiplatelet therapy in the absence of any contraindication for first-generation DESs (especially C-SESs)?

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Video 1. Coronary angiography revealing thrombotic occlusion in the left coronary artery.

Video 2. A drug-eluting stent (CYPHER) was deployed on the left coronary artery.

Video 3. Very late stent thrombosis causing subtotal occlusion distal to the drug-eluting stent in the left coronary artery.

Video 4. Percutaneous coronary intervention was performed for stent thrombosis on the left coronary artery

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Successful treatment of a renal arteriovenous fistula with pulmonary hypertension occurring 38 years after nephrectomy

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Introduction

Iatrogenic arteriovenous fistula (AVF) is a rare cause of pulmonary hypertension (PH) and high-output heart failure (HOHF). We report a case with PH and 15-years heart failure history secondary to an iatrogenic renal AVF due to nephrectomy performed 38 years ago who underwent closure.

Case Report

A 61-year-old female patient has applied to an external healthcare center with a complaint of dyspnea. Upon detection of PH by transthoracic echocardiography (TTE), the patient was referred to our clinic for further evaluation of PH. The patient with progressive dyspnea for 15 years has had World Health Organization (WHO) class III symptoms, orthopnea, and leg edema