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CASE REPORT

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Simultaneous subacute thrombosis of bare metal coronary stents in two different arteries early after clopidogrel cessation

Uğur Canpolat, Hikmet Yorgun, Enver Atalar

Department of Cardiology, Hacettepe University, Ankara, Turkey

Abstract

Coronary stent thrombosis is a rare event leading to ST segment elevation myocardial infarction and death. Although early thrombosis of drug-eluting and bare metal stents is relatively frequent, simultaneous thrombosis of bare metal coronary stents has rarely been reported. Here, we present a case of simultaneous subacute thrombosis of two bare metal stents in different coronary arteries early after clopidogrel cessation. (Cardiol J 2012; 19, 3: 309–313)

Key words: bare metal stent, thrombosis, myocardial infarction

Introduction

Coronary artery stent thrombosis is a rare complication, mostly presenting as ST segment elevation myocardial infarction or sudden cardiac death [1]. Stent thrombosis occurs in approximately 1% of bare metal stents (BMS) [2]. However, simultaneous multivessel stent thrombosis is very rare and can have catastrophic results. Several factors have been associated with coronary stent thrombosis including patient comorbidities, procedural factors, non-compliance with anti-platelet agents and resistance to anti-platelet medications [2–4]. Of these, early cessation of dual anti-platelet therapy is the strongest predictor of stent thrombosis [3].

We present the case of a 56 year-old woman who underwent percutaneous coronary intervention (PCI) using BMS in two separate coronary vessels. Seven days after coronary stenting, simultaneous thrombosis of two BMSs was observed on coronary angiography. Although strict adherence to medical treatment was advised before hospital discharge, the patient did not take clopidogrel but acetylsalicylic acid (ASA). We want to highlight the importance of strict adherence to anti-platelet medications to prevent coronary stent thrombosis.

Case report

A 56 year-old woman was admitted to our cardiology department complaining of exertional chest pain of three months' duration which was squeezing in character and radiating to the left arm. She had had hypertension for six years, was a smoker (35 pack years), and had a history of transient ischemic attack and peripheral artery disease with critical stenosis at both carotid arteries. Family history was unremarkable for any cardiovascular illness. Physical examination was within normal limits except for a mild apical systolic murmur. Complete blood count and serum biochemistry were within normal limits. Coronary angiography showed critical stenosis at the middle segment of the left anterior descending artery (LAD) and proximal major obtuse marginal (OM) artery (Fig. 1). Successful PCI to the mid LAD was performed with BMS (Multi Link Vision, 2.75 × 15 mm, Abbott Vascular, Abbott Park, Illinois, USA) and another BMS (Multi Link Vision, 2.75 × 12 mm, Abbott Vascular, Santa Clara, California, USA) to proximal major OM. Final angiography showed TIMI 3 distal flow without residual stenosis or dissection at both coronary arteries (Fig. 2).

Address for correspondence: Uğur Canpolat, Hacettepe University Faculty of Medicine, Department of Cardiology, Sihhiye-06100, Ankara, Turkey, tel: 090 312 305 1780, fax: 090 312 305 4137, e-mail: dru_canpolat@yahoo.com

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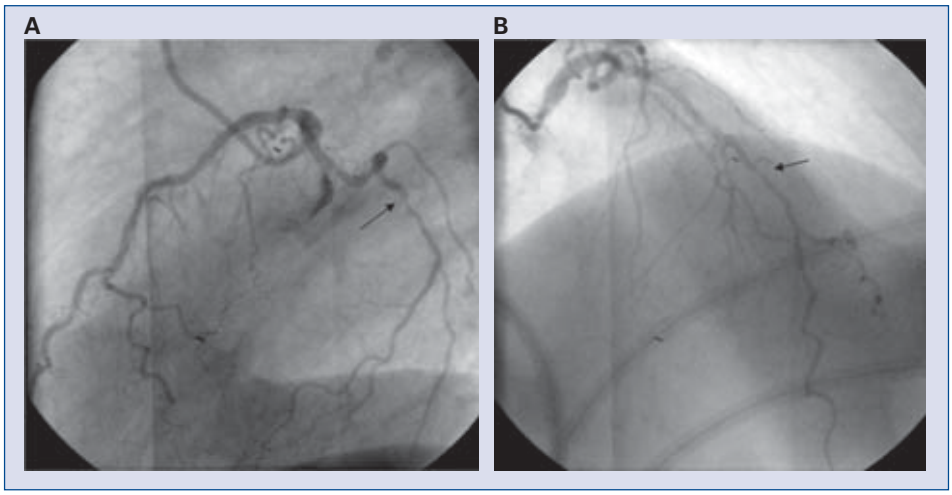

Figure 1. Coronary angiography showing critical coronary stenosis at major obtuse marginal artery in the left anterior oblique cranial projection (**A**) and mid left anterior descending artery in right anterior oblique cranial projection (**B**) (arrows).

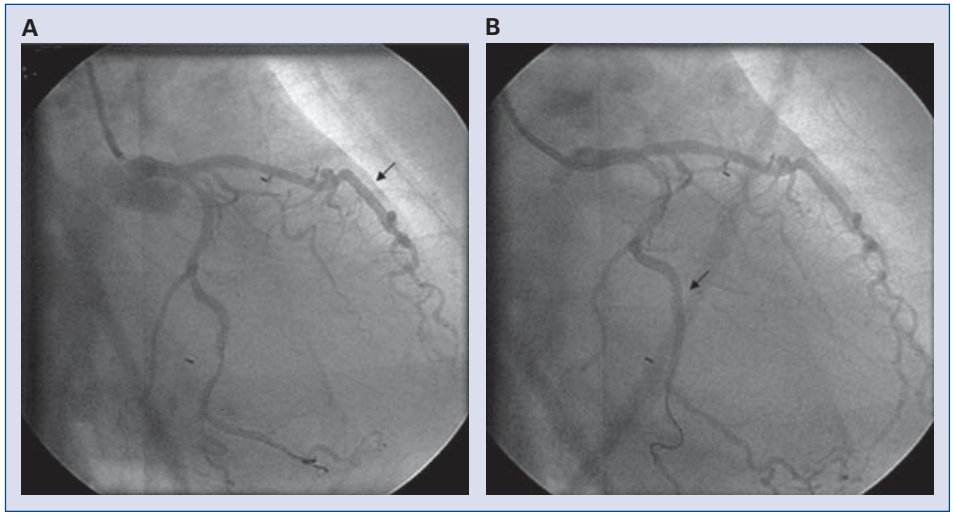

Figure 2. A bare metal stent (Multi Link Vision, 2.75 × 15 mm) was deployed to mid left anterior descending artery without residual lesion (**A**) (arrow). Afterwards, another bare metal stent (Multi Link Vision, 2.75 × 12 mm) was implanted to proximal major obtuse marginal artery stenosis without residual lesion or dissection (**B**) (arrow).

The patient was discharged with ASA, clopidogrel, ramipril and metoprolol medications three days after stent implantation. Four days after hospital discharge, the patient was readmitted to the emergency department with severe chest pain. Electrocardiography revealed both anterior and inferolateral ST segment elevation (Fig. 3). Treatment with ASA (300 mg), clopidogrel (600 mg), intravenous unfractioned heparin, metoprolol and atorvastatin was immediately initiated. She was hemodynamically stable. The patient was taken to the catheter laboratory for primary PCI which revealed simultaneous stent thrombosis at LAD and major OM stents (Fig. 4). The LAD mid occlusion was treated with semi-compliant balloon angioplasty (Sprinter Legend RX Semi-compliant Balloon, 2.5 × 30 mm, Medtronic, Minnesota, USA) followed by a second BMS (Multi Link Vision, 2.75 × 8 mm) placed proximal to the initial stent because of dissection after repeated balloon inflations (Fig. 5). The major OM occlusion was also treated with balloon angioplasty (Biotronik AG, 2.5 × 20 mm, Bulach, Switzerland). Final angiography revealed TIMI 3 flow distal to the coronary stents. After the intervention, tirofiban therapy was given for 36 hours. During the hospital stay, complete blood count, serum biochemistry, hypercoagulable

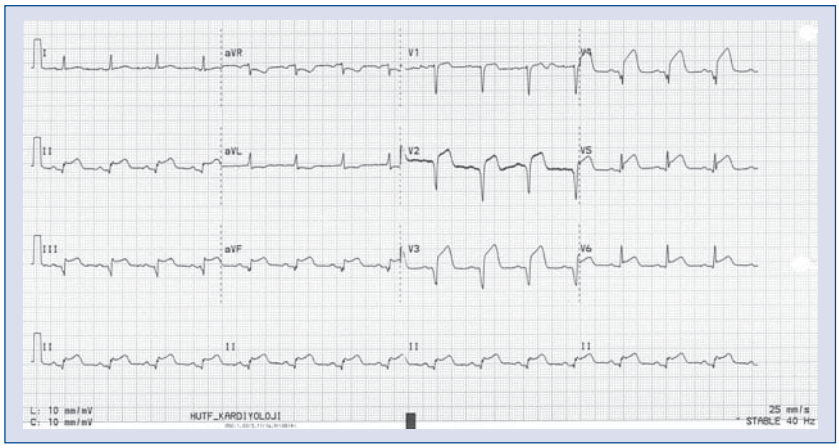

Figure 3. Electrocardiography revealed ST segment elevation on both anterior and inferior leads.

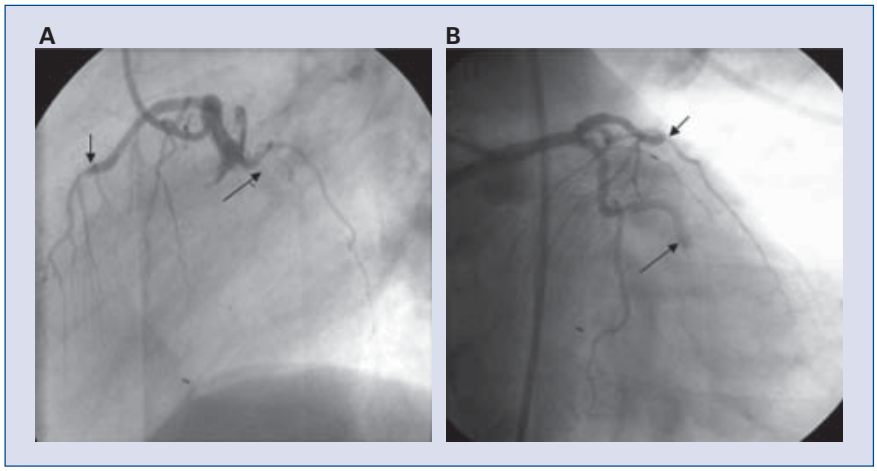

Figure 4. Simultaneous coronary stent thrombosis causing complete occlusion proximal to bare metal stent in left anterior descending artery (**A**) (arrows) and major obtuse marginal artery (**B**) (arrows).

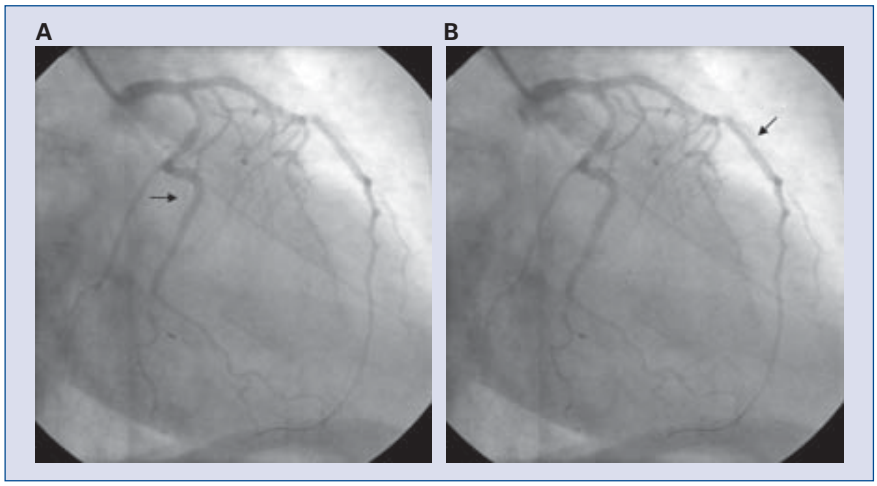

Figure 5. After balloon angioplasty with balloon (BiotronicElect 2.5 × 20 mm), TIMI 3 flow was observed (**A**) (arrow). For left anterior descending artery lesion, after balloon angioplasty (Sprinter Legend Rx 2.5 × 30 mm), coronary dissection was observed proximal to left anterior descending artery stent, so a bare metal stent (Multi Link Vision 2.75 × 8 mm) was implanted (**B**). Final coronary angiography showed TIMI 3 distal flow without residual stenosis or dissection.

blood tests including prothrombin time, activated partial thromboplastin time, international normalized ratio, protein C activity, protein S (total and free), antithrombin III, anticardiolipin antibodies, homocysteine and factor V Leiden mutation were evaluated and found to be within normal limits. To rule out clopidogrel resistance, a test was carried out for adenosine diphosphase (ADP)-induced platelet aggregation utilizing the VerifyNow $P2Y_{12}$ point-of--care assay (VerifyNow $P2Y_{12}$ test cartridge system, Accumetrics, San Diego, California, USA). This revealed a normal response. She was observed as an inpatient for four days and discharged with a strict recommendation of dual anti-platelet therapy for at least one year. A recent three month follow-up found no significant symptoms and she has complied with her dual anti-platelet medication.

Discussion

Despite the beneficial role of coronary stent implantation for obstructive coronary artery lesions, stent thrombosis has remained a serious complication of percutaneous interventions either with a BMS or a drug-eluting stent (DES). Although simultanous subacute stent thrombosis after the implantation of two DESs and with one BMS and one DES have previously been reported, to the best of our knowledge this is the first case report of simultaneous subacute thrombosis of two BMSs in two separate coronary arteries.

Most cases of stent thrombosis occur within the first month after deployment, irrespective of the stent type [5]. In a pooled analysis of six coronary stenting studies, stent thrombosis mostly occurred within two days of BMS implantation [2]. Several factors related to the patient, coronary lesion and percutaneous intervention predispose to stent thrombosis. Incomplete stent expansion, stent malposition, stent length, small vessel caliber, bifurcation lesion, in-stent restenosis lesion, intermediate ($\geq$ 50% to $<$ 70% stenosis) coronary artery disease proximal to the culprit lesion and residual thrombus or persistent dissection after stent placement are the leading procedural, lesion and stent characteristics predisposing to coronary stenosis [2, 3, 6, 7].

Acute coronary syndrome, older age, diabetes mellitus, left ventricular dysfunction, prior brachytherapy, malignancy, chronic kidney disease, hypercoagulable state, disorders in the metabolism of anti-platelet agents and adherence to medications were the main factors related to our patient. Importantly, the most important predictor for early and late stent thrombosis is non-adherence to clopidogrel therapy [3].

Adherence to anti-platelet agents plays a key role in the management of patients after PCI. Dual anti-platelet therapy with aspirin and clopidogrel is standard to prevent stent thrombosis. Discontinuation is associated with a marked increase in the risk of stent thrombosis. Patient non-adherence, side effects like bleeding, allergic reactions and the need for invasive or surgical procedures are the main factors for premature cessation of anti-platelet therapy. Furthermore, a patient's educational level and the cost of drugs may have an impact on adherence to optimal medical therapy, especially in developing countries. Beyond those, the results of the REGINA survey indicated a dramatic heterogenity as to the management of anti-platelet theapy discontinuation and reintroduction among practitioners interested in anti-platelet therapy, from dentists to cardiologists, highlighting the urgent need to improve practitioners' basic knowledge [8].

Simultaneous stent thrombosis can be due to several factors including cessation of anti-platelet agents, anti-platelet resistance, systemic inflammatory and thrombogenic milieu due to acute coronary syndromes, or secondary thrombosis due to systemic hypotension after one stent thrombosis.

However, our patient did not have an acute coronary syndrome which can cause stent thrombosis. She was hemodynamically stable on admission, so hypotension-induced stent thrombosis was not considered as the cause. The possible etiology of clopidogrel resistance was excluded by an ADP-induced platelet aggregation test which revealed a normal response. Hypercoagulable disorder was not found after standard coagulation tests. Periprocedural coronary dissection or stent malposition was not considered because the patient's repeat catheterization did not reveal any dissection. An intravascular ultrasound was not performed because of uneventful intervention with successful angiographic results after post-dilatation to high pressure with a semi-compliant balloon.

In our patient, the single most important factor predisposing to simultaneous BMS thrombosis seems to have been non-adherence to clopidogrel therapy.

In conclusion, this case report highlights the importance of strict adherence to anti-platelet medications after coronary stent implantation. Physicians should be aware of the factors that contribute to clopidogrel cessation and patients should be educated about the dangers of anti-platelet drug discontinuation.

Conflict of interest: none declared

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