International Journal of the Cardiovascular Academy 2 (2016) 96–97

Contents lists available at ScienceDirect

International Journal of the Cardiovascular Academy

journal homepage: www.elsevier.com/locate/ijcac

Short communication

Warfarin treatment in stent thrombosis☆,☆☆

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ARTICLE INFO

Article history:
Received 27 February 2016
Received in revised form 5 May 2016
Accepted 8 May 2016
Available online 13 May 2016

Keywords:
Stent thrombosis
Warfarin
IVUS

ABSTRACT

A 62 year-old male patient was admitted to the hospital due to anterior ST segment elevated myocardial infarction. Thrombotic complication developed three times during the hospitalization after primary percutaneous coronary intervention (PCI). Instent thrombus was identified despite the optimal medical therapy, new antiplatelet agents and intravascular ultrasound guided PCI. Therefore, symptoms were controlled with warfarin + clopidogrel treatment. At the third month of warfarin + clopidogrel treatment, the patient was asymptomatic and no active electrocardiographic changes were observed. Stent thrombosis developing under new antiplatelet agents constitutes a great problem and there are no clear data for the solution. We described an interesting case in which we achieved symptomatic control by warfarin + clopidogrel in the stent thrombosis developing during the treatment with dual antiplatelet therapy.

Introduction

Current guidelines recommend timely applied primary percutaneous coronary intervention (PCI) for all patients with ST segment elevation myocardial infarction (STEMI).[1] Despite the developments in medical treatment and intervention methods, stent thrombosis (ST) has still been a great challenge. There are limited data, in particular, about what to do after ST developed under the treatment with new antiplatelet agents. We have identified an interesting case without complications under warfarin + clopidogrel treatment that did not have hereditary thrombophilia and had recurring thrombotic complications despite the use of prasugrel, ticagrelor and intravascular ultrasound (IVUS) guided PCI.

Case report

A 62 year old male patient without any additional risk factor except hypertension was admitted to the hospital due to acute anterior ST segment elevated myocardial infarction (STEMI). In the coronary angiography (CAG), LAD was found to be occluded in the proximal segment and there were no any significant lesions in RCA and LCX. A 3.5 × 9 mm bare metal stent (BMS) was implanted to proximal LAD and a 3,5 × 28 mm BMS was implanted to mid-region. Post-dilatation was applied to both stents with 3,5 × 15 mm noncompliant balloon of Simpass (NCB) at nominal pressure. 180 mg ticagrelor, 300 mg acetyl salicylate and intravenous abciximab were administered during the procedure. The patient was taken to post-MI follow-up and anginal complaints developed again two days after PCI. In the patient's electrocardiography (ECG), there were 2 mm ST segment elevation in the anterior leads, and CAG was applied to the patient again. It has been found that proximal stent was occluded with thrombosis. The lesion was reached with floppy guidewire (FGW) and it was dilated with 4 × 18 mm NCB. Trofiban was started as intracoronary and systemic, ticagrelor discontinued and 60 mg prasugrel was administered. After the patient was delivered to the intense care unit in a stable condition, tirofiban infusion was interrupted one day later. Because of chest pain and 1 mm ST segment elevation in anterior leads, the patient was referred to CAG again one day later. It was found that the proximal stent was occluded with thrombus and the lesion was dilated with 4 × 18 mm NCB at 18 atm pressure, following 1.5 × 15 mm balloon and 3.5 × 20 mm BMS at nominal pressure was implanted. The examinations were sent in terms of thrombosis tendency. No disease or condition leading to thrombosis was identified. IVUS was planned for the patient. The expansion of stent to lumen was evaluated with IVUS. It was observed that proximal stent expanded insufficiently (Fig. 1). It was dilated again with 4.5 × 15 mm NCB. It was checked with IVUS and seen that the stent apposition was optimal. Due to presence of thrombus during the IVUS (Fig. 2), thrombectomy was performed by thrombus aspiration catheter. Intravenous tirofiban infusion was initiated and the patient was referred to the intense care unit. However, angina and ECG changes

☆ Prior publication: This article is an original case report and it has not been published or submitted for publication elsewhere, in whole or in part, before submission to the journal.
☆☆ Funding and support: Nothing to declare.
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Peer review under responsibility of The Society of Cardiovascular Academy.

http://dx.doi.org/10.1016/j.ijcac.2016.05.001

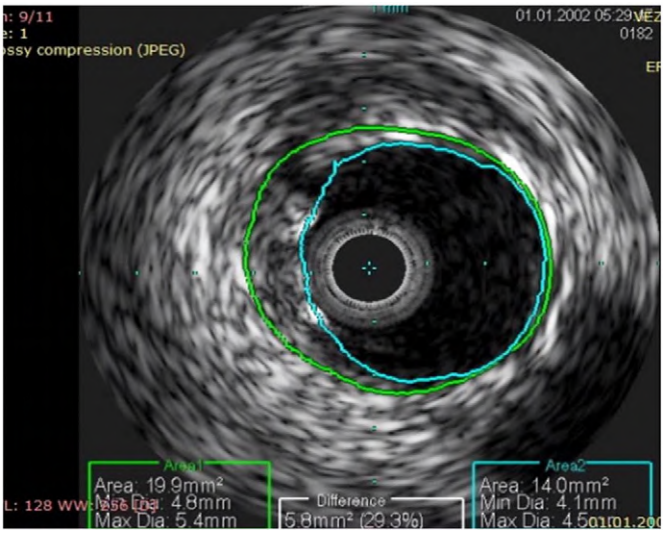

Fig. 1. IVUS image demonstrated that proximal stent expanded insufficiently.

were developed again 8–10 h later following the discontinuation of tirofiban. After readministration of intravenous tirofiban infusion the patient's chest pain was regressed. ST was observed in LAD during control CAG. Because of presence of TIMI 3 flow, we decided to start warfarin plus clopidogrel 75 mg daily and prasugrel was discontinued. No angina developed and no ECG changes were found during the monthly follow-up visits. We aimed the target INR value between 2.0 and 3.0 and the last two INR values of patients were 2.3 and 2.5. The patient was discharged after three days without symptoms. At the third month of the follow-up, the patient was asymptomatic, and there were no changes in the ECG compared to the ECG examination during discharge.

Discussion

PCI in the treatment of acute coronary syndromes is the most significant and efficient treatment option for morbidity and mortality.

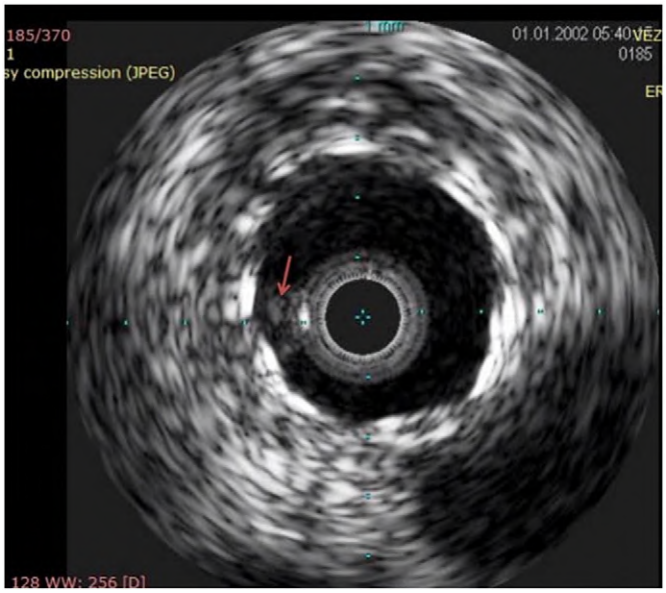

Fig. 2. IVUS image reveals optimal appositiona and thrombus formation (red arrow).

However, ST is still a significant problem in terms of mortality. We described an interesting case having thrombotic complications three times during the hospitalization that referred to the hospital due to anterior MI. ST is a rare but usually catastrophic event that complicates the stent implantation and it is associated with considerable mortality and adverse outcomes due to abrupt vessel closure.[2] It was defined as the occlusion originating in the peri-stent region or presence of a thrombus originating in the peri-stent region in the presence of acute onset ischemic symptoms, or the presence of new ECG changes indicating acute ischemia or the presence of typical rise and fall in cardiac biomarkers.[3] ST etiology is multifactorial and it is associated with the procedural factors, lesion and patient charecteristics.[4] Although a significant decrease was reached in ST with the use of DES, ST has still been a significant mortality and morbidity problem. In our case, the stents implanted were BMS. However, due to a large stent diameter, it may not be an appropriate approach to associate ST to the stent type used.[5]

During the first stent implantation, dual antiplatelet therapy with ticagrelor was used, and the treatment was replaced with prasugrel after stent thrombosis. However, ST developed again, considering that hereditary thrombophilia was not found in our case. ST development for 3 times makes the case quite complicated. It is known that IVUS guided PCI decreases ST prominently. IVUS enables us to get information about ST and to evaluate the stent underexpansion, arterial remodeling, neointimal structure and distribution.[6] In our case, we applied IVUS after second ST. After IVUS examination, we observed that the proximal stent was underexpanded. Then, the stent diameter, which was 3,5 mm, was dilated with 4,5 mm NCB. It was observed that the stent expanded to the lumen completely. However, intraluminal thrombus was still found (Fig. 2). Thrombectomy was applied and TIMI III flow was provided. Upon the detection of intrastent thrombus in the control angiography performed two days later, warfarin + clopidogrel treatment was initiated as the maintenance therapy. No anginal complaint occurred during this treatment, and no active ECG changes were observed. After the discharge from hospital, it was seen that the symptom and ECG did not change at 1st and 3rd months of follow-up.

The thrombotic process of patients continued although treatment with IVUS guided PCI, glycoprotein 2b/3a antagonists and new antiplatelet agents. No chest pain and active ECG change were observed under warfarin + clopidogrel treatment. There has been no clear data in the guides yet about the treatment method in case that ST develops during treatment with new antiplatelet agents. As in our case, warfarin usage may provide solution in such cases.

Conflict of interest

The authors have no commercial, financial, and other relationships in any way related to the subject of this article that might create any potential conflict of interest.

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