

Simultaneous multivessel acute myocardial infarction by double-stent thrombosis

Rozległy ostry zawał serca spowodowany jednoczesną zakrzepicą
w dwóch stentach

Sofía Calero Nuñez, Miguel Corbí-Pascual, Juan Gabriel Córdoba-Soriano,
Arsenio Gallardo-López, María Isabel Barrionuevo Sánchez, Jesús Jiménez Mazuecos

Department of Cardiology, Albacete University General Hospital, Albacete, Spain

Abstract

We report a case of a patient presenting with an anterior and inferior ST-elevation myocardial infarction due to double-arterial coronary stent thrombosis in the left anterior descending artery and right coronary artery, despite the administration of prasugrel and acetylsalicylic acid. Stent thrombosis is an uncommon, but serious complication, usually manifested by sudden death (20–40%) or acute ST-elevation myocardial infarction (50–70%). It is a multifactor phenomenon, which involves other factors of the patient. Understanding of its histopathology and risk factors becomes necessary to try to prevent it, and we should keep in mind, that the deployment of a drug-eluting stent could be unsafe if it is not supported by a suitable evidence-based clinical guideline.

Key words: neoatherosclerosis, antiplatelet therapy

Folia Cardiologica 2018; 13, 2: 163–165

Introduction

Stent thrombosis is an uncommon, but serious complication, manifested mostly by sudden death or acute ST-elevation myocardial infarction. We report a rare case of a patient presenting with an anterior and inferior ST-elevation myocardial infarction due to very late double-arterial coronary stent thrombosis occlusion. We should keep in mind that the deployment of a drug-eluting stent could be unsafe if it is not supported by a clear clinically oriented pathway, that considers the overall condition of the patient.

Case report

A 67-year-old man, smoker and suffering from diabetes, who had undergone implantation with four drug-eluting stents (DES, everolimus) placed in the proximal (3.0 × 12 mm) and

medium (2.5 × 12 mm) left anterior descending artery (LAD), and in the medium (3.0 × 12 mm) and distal (2.5 × 12 mm) right coronary artery (RCA), due to non-ST-elevation myocardial infarction (NSTEMI), was asymptomatic at 12 months follow-up, and was still treated with dual antiplatelet therapy with acetylsalicylic acid (ASA) and prasugrel. Subsequently he was admitted to our hospital with an anterior and inferior ST-elevation myocardial infarction (STEMI) (Figure 1A). Coronary angiography (CA) showed simultaneous DESs thrombosis occlusion of the proximal RCA and LAD (Figure 1B; see supplementary files, [video 1'](#) and [video 2'](#), on-line version on the journal website). He was successfully treated with thrombus aspiration and balloon angioplasty on both vessels (Figure 1C). Four months later he developed a new episode of NSTEMI, CA showed an in-stent restenosis (ISR) in the proximal LAD stent (Figure 2; see supplementary file, [video 3'](#), on-line version on the journal website). After discussing the

Address for correspondence: Dra Sofía Calero Nuñez, Albacete University General Hospital, C/Hermanos Falcó 37, 02006 Albacete, Spain,
e-mail: sofia.caleron@gmail.com

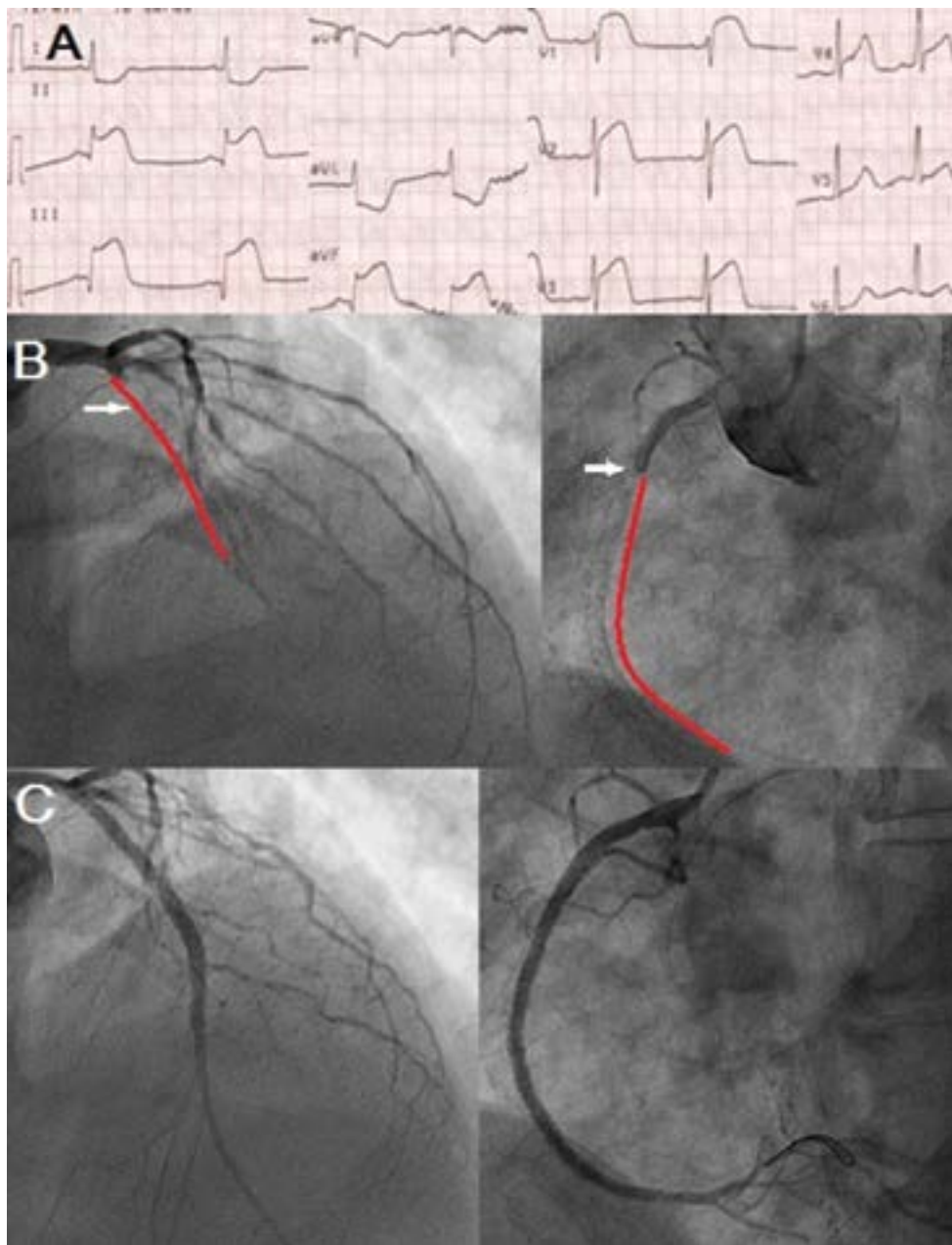


Figure 1A. Electrocardiography (ECG) showing ST-elevation on inferior and anterior leads; **B.** Simultaneous stent thrombosis (ST) of the left anterior descending artery (LAD) and the right coronary artery (RCA) (arrow); **C.** Reopening of the LAD artery and the RCA

patient's data within the heart team it was decided, that coronary-artery bypass grafting (CABG) was necessary. One single internal mammary artery coronary bypass to the LAD was performed, but the patient had a torpid postoperative course and he died from refractory septic shock.

Discussion

Double simultaneous ST in two different coronary arteries causing a double STEMI is a rare complication, that was already described in the literature, but our case is the first

in a patient with second generation DES and new antithrombotic therapies (prasugrel). It is possible, that the first ST may lower blood pressure rapidly provoking the second ST in another vessel due to the reduced flow. ST is probably related to an ongoing prothrombotic vascular inflammatory response; therefore patients undergoing stent implantation within an acute coronary syndrome (ACS) suffer ST more often than those with stable coronary artery disease. It is generally thought, that lack of stent strut coverage and late malapposition may be responsible for very late ST (occurrence > 360 days after stent implantation). However, it is

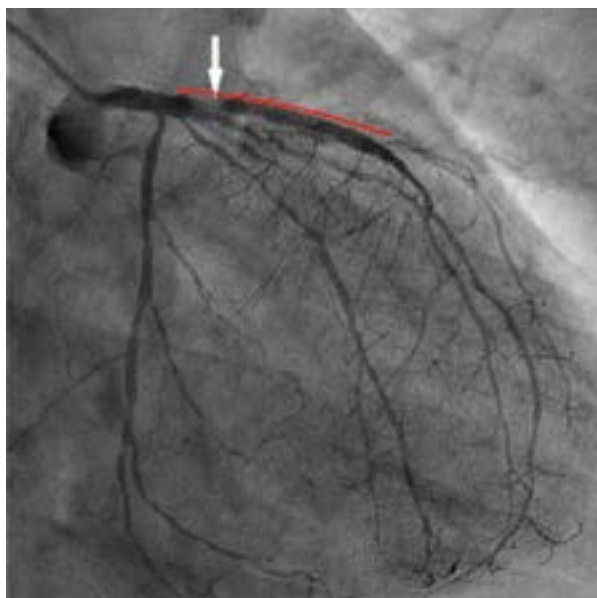


Figure 2. Restenosis of the proximal left anterior descending artery (LAD) stent

a multifactor phenomenon, which also involves other such factors related to the patient (primary stenting for acute myocardial infarction (AMI), diabetes mellitus, left ventricular dysfunction, hypersensitivity to the polymer or metal, renal failure, premature antiplatelet therapy discontinuation, non-responsive to ASA or clopidogrel). Our patient had several of these factors. The advent of high-resolution intracoronary imaging, especially optical coherence tomography and intravascular ultrasound, has shown that in many cases of late stent failure neoatherosclerotic change within the stented segment represents a final common pathway for both thrombotic and restenotic events. Many strategies have been employed for the treatment of ST, although recent developments focus on strategies, which circumvent the need for chronically indwelling stents – such as drug-coated balloons or fully bioresorbable stents – more data are needed before a wider use of these therapies could be advocated.

Conflict of interest(s)

The authors declare that there is no conflict of interest.

Streszczenie

W pracy opisano przypadek pacjenta z zawałem ścian przedniej i dolnej serca z uniesieniem odcinka ST spowodowanym zakrzepicą w dwóch stentach wieńcowych umieszczonych w gałęzi międzykomorowej przedniej i prawej tętnicy wieńcowej, która wystąpiła mimo stosowania prasugrelu i kwasu acetylosalicylowego. Zakrzepica w stencie jest rzadkim, lecz poważnym powikłaniem, zwykle prowadzącym do nagłego zgonu (20–40%) lub ostrego zawału serca z uniesieniem odcinka ST (50–70%). Powikłanie to ma podłoże wieloczynnikowe. Poznanie cech histopatologicznych i czynników ryzyka zakrzepicy w stencie jest konieczne, aby można było zapobiec wystąpieniu tego powikłania. Ponadto należy pamiętać, że wszczepienie stentu uwalniającego lek może być niebezpieczne, jeśli nie jest zgodne z wytycznymi opartymi na dowodach naukowych.

Słowa kluczowe: *neoatherosclerosis*, terapia przeciwplateletowa

Folia Cardiologica 2018; 13, 2: 163–165

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