

Infarct-related artery filled with thrombus cleared completely after tirofiban therapy

Rezolucja skrzepliny wypełniającej tętnicę odpowiedzialną za zawał po leczeniu tirofibanem

Serkan Bulur¹, Hakan Özhan¹, Yasin Türker¹, Şule Bulur²

¹Department of Cardiology, Duzce University, Duzce Medicine Faculty, Turkey

²Department of Physiology, Selcuk University, Meram, Turkey

Post Kardiol Interw 2012; 8, 1 (27): 75–77

DOI: 10.5114/pwki.2012.28074

Abstract

The treatment of acute coronary syndrome patients with high thrombus burden is difficult. Percutaneous coronary interventions increase the frequency of distal embolization in these patients. Glycoprotein IIb/IIIa inhibitors are effective anti-platelet agents and they can be very helpful in such cases. Herein, we present the impact of tirofiban infusion on an infarct-related artery with high thrombotic burden. The thrombus cleared completely without coronary angioplasty.

Key words: acute coronary syndrome, glycoprotein IIb/IIIa inhibitor

Streszczenie

Leczenie ostrych zespołów wieńcowych u pacjentów, u których obecna jest duża ilość skrzeplin, jest skomplikowane. Przeszkórne interwencje wieńcowe zwiększają częstość występowania dystalnej embolizacji u tych chorych. Inhibitory glikoproteiny IIb/IIIa są skutecznymi lekami przeciwplateletowymi i mogą być bardzo pomocne w takich sytuacjach. Prezentujemy wpływ wlewu tirofibanu na tętnicę odpowiedzialną za zawał wypełnioną materiałem zakrzepowym. Skrzepliny uległy całkowitej rezolucji bez konieczności wykonywania angioplastyki wieńcowej.

Słowa kluczowe: ostry zespół wieńcowy, inhibitor glikoproteiny IIb/IIIa

Introduction

Coronary atherosclerosis is the most frequent cause of ischaemic heart disease, and plaque disruption with superimposed thrombosis is the main cause of acute coronary syndrome (ACS) [1]. The clinical manifestation of an ACS thrombotic event is determined by the balance between the propensity for thrombus formation and the efficacy of the endogenous thrombolytic processes [2]. An invasive strategy of coronary angiography, with revascularization when appropriate, is recommended for high-risk patients who have an acute coronary syndrome [3].

Tirofiban is a non-peptide tyrosine derivative which belongs to the class of glycoprotein (GP) IIb/IIIa inhibitors (GPI). By preventing the binding of fibrinogen and von Willebrand factor to the GP IIb/IIIa receptor on the surface

of the platelet, GPIs are currently regarded as the most potent inhibitors of platelet aggregation [4].

Herein, we present the effect of tirofiban infusion on an infarct-related artery with high thrombotic burden.

Case report

A 73-year-old woman was admitted to our hospital with fluctuating crushing chest pain. The electrocardiogram (ECG) revealed normal sinus rhythm and negative T waves on V1-V2 and aVL. Her medical history revealed hypertension. She was taken to the catheterization laboratory for percutaneous coronary intervention with the diagnosis of acute coronary syndrome. Coronary angiography showed that the right coronary artery was filled with thrombus (Figure 1). The left anterior descending and circumflex arter-

Corresponding author/Adres do korespondencji:

Dr. Serkan Bulur, Duzce Universitesi, Düzce Tıp Fakültesi 81620, Konuralp Düzce, Turkey, tel: +90 533 3863984, fax: +90 380 541 41 05, e-mail: drserkanbulur@yahoo.com

Praca wpłynęła: 24.11.2011, przyjęta do druku: 30.01.2012.

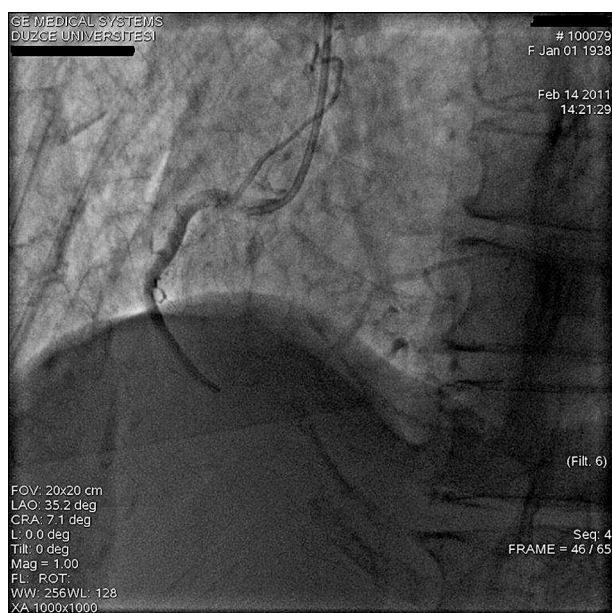


Fig. 1. Right coronary artery before tirofiban treatment

Ryc. 1. Prawa tętnica wieńcowa przed leczeniem tirofibanem

ies were normal. The culprit lesion could not be identified. Therefore, tirofiban infusion 0.40 µg/kg/min for the first 30 min plus 0.10 µg/kg/min infusion was started and continued for 48 h. In addition, aspirin, clopidogrel and low molecular weight heparin were given for ACS therapy. At the end of the infusion, the coronary angiogram was repeated. The right coronary artery was completely cleared (Figure 2). Creatine kinase (CK), CK-MB and troponin values did not increase during hospital follow-up. The patient was symptom-free after discharge from the hospital and was doing well at her 6-month follow-up visit with aspirin 100 mg, metoprolol 50 mg, clopidogrel 75 mg, atorvastatin 10 mg and perindopril 10 mg/day therapy.

Discussion

The presence of angiographically detected thrombosis in patients presenting with ACS is associated with a higher incidence of in-hospital and long-term adverse cardiac events [5]. In the setting of ACS or when faced with a thrombotic lesion for which percutaneous coronary intervention (PCI) is being considered, adjunctive pharmacological therapy delivered intravenously or via the intracoronary route should aim at the prevention of thromboembolic events associated with PCI, and the reversal of such events if they occur. The interventional cardiologist is faced with the hard task of managing lesions with high thrombotic burden, which is more prevalent when earlier catheterization is performed [6]. The mechanical manipulation of such lesions is associated with a high incidence of periprocedural complications at least in part due to a high incidence of distal embolization [7]. Intravenous

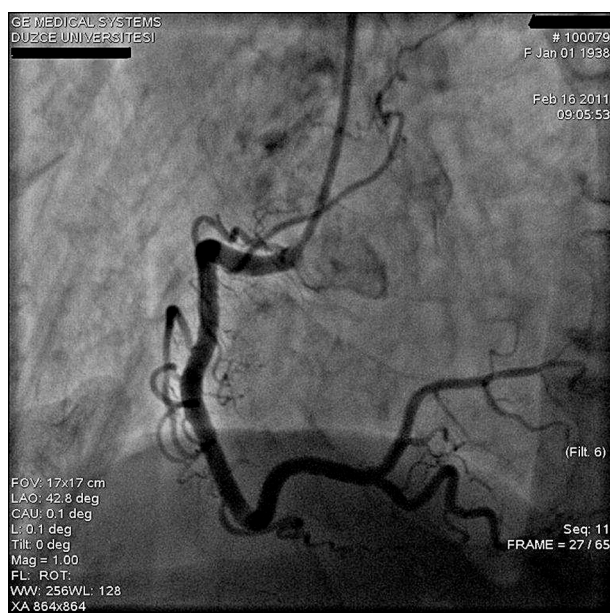


Fig. 2. Right coronary artery after tirofiban treatment

Ryc. 2. Prawa tętnica wieńcowa po leczeniu tirofibanem

antiplatelet GP IIb/IIIa inhibitors are frequently administered to patients with ACS undergoing PCI, a strategy supported by several randomized clinical trials [3]. In our index patient, PCI was delayed due to very high thrombus burden and mismatch of ECG changes with the coronary anatomy. However, aggressive antiplatelet treatment with tirofiban, a potent inhibitor of GP IIb/IIIa receptors, in combination with low molecular weight heparin was administered. The result was a perfectly cleared coronary artery without a need for PCI. Therefore, invasive cardiologists should always keep in mind that GP IIb/IIIa receptor blockers may avoid unnecessary percutaneous coronary intervention in patients with acute coronary syndrome complicated with very high intracoronary thrombus burden. In such cases “cooling” the lesion with appropriate antithrombotic therapy may help to achieve better results and even prevent unnecessary interventional procedures.

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