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A case of acute stent thrombosis treated successfully with intracoronary tirofiban

Ostra zakrzepica w stencie skutecznie leczona dowieńcowym podaniem tirofibanu

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Abstract

Acute stent thrombosis (AST) is occasionally observed during percutaneous coronary intervention in patients with acute coronary syndrome (ACS). It may jeopardize hemodynamic status. Currently, there is no adequate solution for this problem. We report our experience with an ACS patient who developed AST associated with cardiogenic shock after percutaneous coronary stent deployment. Intracoronary administration of tirofiban immediately restored the coronary flow of the target vessel, and the disastrous condition was reversed. Our experience suggests that intracoronary administration of tirofiban can be considered as an option in cases of AST during percutaneous coronary intervention.

Key words: acute stent thrombosis, intracoronary tirofiban

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INTRODUCTION

The risk of acute stent thrombosis (AST) is increased in patients with acute coronary syndrome (ACS) because of increased systemic and intracoronary thrombogenicity and inflammation. ACS and thrombotic lesions are known risk factors for AST. Stent thrombosis persists at a rate of 6% in patients with acute coronary syndromes. During percutaneous coronary intervention (PCI) in patients with acute coronary syndrome, AST may jeopardize the hemodynamic status in these patients, which is complicated by cardiogenic shock in 26.4% of cases. Moreover, the mortality rate is 16.5% at 30 days. The current treatment strategy is not well defined. In this context, it is well-recognized that the principal objective must be to obtain effective reperfusion as quickly as possible [1]. We present a patient with ACS who developed stent thrombosis associated with shock after percutaneous coronary stent deployment. We aim to show the impact of intracoronary tirofiban injection on AST and discuss the result in the light of literature data.

CASE REPORT

A 46 year-old man was referred to our department complaining of squeezing chest pain during the previous three hours. On admission, his blood pressure was 110/80 mm Hg, and his heart rate was 85 beat/min. Physical examination was normal. His medical history was unremarkable. Electrocardiography showed a 3 mm ST-segment elevation in leads V1–V4. A diagnosis of acute anterior myocardial infarction was made and he was referred to the cardiac catheterization laboratory for primary coronary angioplasty. Medical treatment was started with 300 mg aspirin, 600 mg clopidogrel, intravenous nitrate infusion and bolus injection of 5,000 units unfractioned heparin. Coronary angiography revealed intraluminal-filling defects due to a massive thrombus, resulting in partial vessel occlusion in the proximal segment of the left anterior descending (LAD) coronary artery (Fig. 1).

PCI for LAD was performed using a 6 Fr Tip JL4 guiding catheter and a floppy guide wire via a transfemoral approach. A bare metal coronary stent (Ephesos stents, Nemed Corpo-

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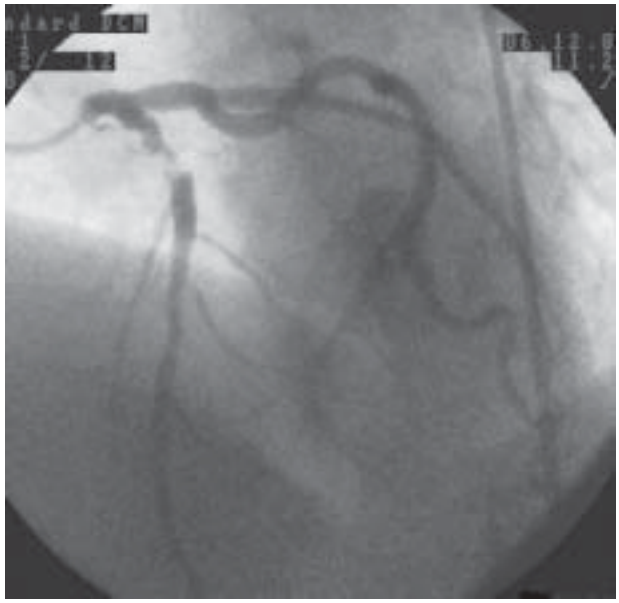

Figure 1. Left anterior oblique projection of selective left coronary angiogram shows the culprit lesion with 90% stenosis and fresh thrombus inside the lesion at the proximal part of left anterior descending artery

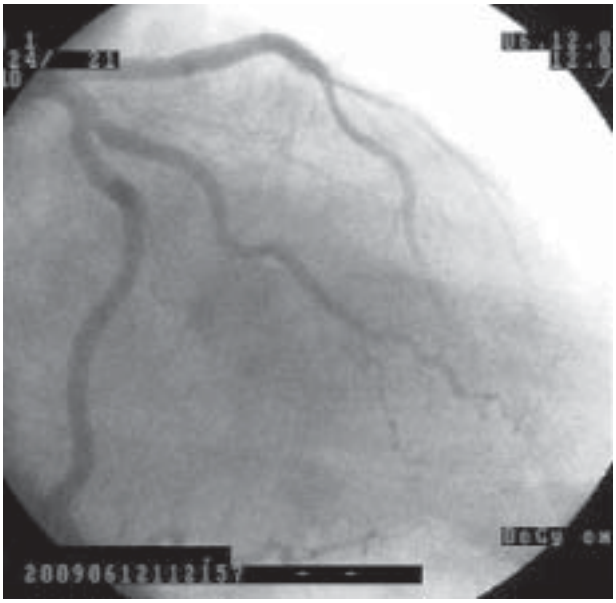

Figure 3. Right anterior oblique projection with caudal angulation of selective left coronary angiogram; distal flow was restored after intracoronary tirofiban administration

ration, Turkey) 3.5 × 18 mm was deployed to the lesion at 12 bars. Unfortunately, the subsequent coronary angiography showed total occlusion due to stent thrombosis distal to the lesion site (Fig. 2). We attempted to inflate the balloon four times at 8–10 bars for 10–15 seconds sequentially from inside the stent to its distal segment. However, the total occlusion due to stent thrombosis persisted, despite our attempt to fragment the thrombus using balloon dilatation. The patient complained of dyspnea and a squeezing chest pain. The systolic blood pressure began to decrease to 60 mm Hg. Tirofiban 750 μg (10 μg/kg) was administered intracoronary for 2–3 minutes through the guiding catheter. Four to five minutes after being administered tirofiban, the patient had relief from his angina and dyspnea and then the blood pressure returned to normal (> 100 mm Hg systolic). Selective coronary angiography immediately after intracoronary administration of tirofiban showed a distal TIMI-2 flow, without identification of the thrombus (Fig. 3). This patient was transferred to the intensive care unit for further observation after the procedure. Intravenous tirofiban infusion after PCI was maintained at a rate of 0.15 μg/kg/min for 24 hours. This patient was discharged without complications four days after PCI, and was followed-up at the outpatient clinic uneventfully.

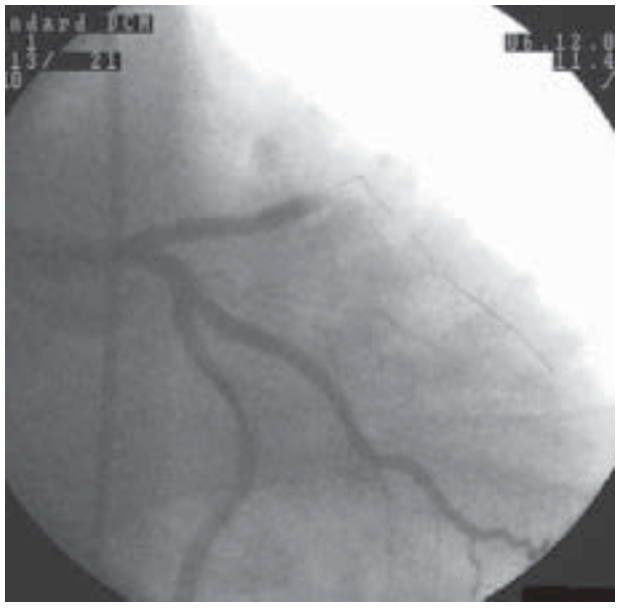

Figure 2. Right anterior oblique projection with caudal angulation of selective left coronary angiogram; acute stent trombosis after stent deployment

DISCUSSION

The risk of stent thrombosis is increased in patients with ACS because of heightened systemic and intracoronary thrombogenicity and inflammation. ACS and thrombotic lesions are known risk factors for ST. Stent thrombosis persists in 6% of patients with ACS [1]. Clinical trials in which the thrombus was aspirated, along with autopsy studies, have demonstrated different thrombus composition in patients with stent thrombosis compared to patients with *de novo* ST elevation myocardial infarction (STEMI) [2]. Indeed, the thrombus is almost totally composed of platelets and contains very low fibrin material in the case of stent thrombosis, which may

impact the efficacy of thrombolysis for this indication. The pathophysiology of the thrombus formation and composition differs in patients with *de novo* STEMI who usually develop a thrombus due to ruptured plaque; in patients with stent thrombosis, it is more likely the result of a lack of healing that leads to the occlusion of the stent. The current treatment strategy is not well defined, but the principal objective of stent thrombosis treatment is to obtain effective reperfusion as quickly as possible. Although emergency PCI (balloon dilatation or new stent implantation) is commonly employed, the procedure is not always successful [3]. A few small retrospective studies with < 30 patients have shown that thrombus aspiration for stent thrombosis permits a reperfusion success rate of up to 90% [4].

There is possibly a favorable effect of the glycoprotein IIb/IIIa antagonist for the treatment of stent thrombosis. Abciximab is a glycoprotein IIb/IIIa receptor antagonist which determines a potent inhibition of platelet aggregation and thrombus formation. These properties seem to prevent not only thrombus formation, but also to promote (at higher drug concentration) lysis of fresh thrombus. The local administration of abciximab at the site of coronary thrombosis may enhance, by increasing its local concentration, the binding to both platelet and endothelium receptors. The results of several angiographic studies assessing the effect of intracoronary abciximab administration support on clinical grounds its adoption in patients with fresh coronary thrombosis, and better post-angioplasty coronary flow has been achieved, compared to the intravenous, systemic, administration of a drug bolus [5]. Tirofiban is another IIb/IIIa antagonist. The role of tirofiban in the setting of acute STEMI treated with primary coronary angioplasty remains controversial [6]. Tirofiban has been found to be less effective than abciximab in platelet inhibition within 60 min of intravenous administration; with venous administration, a long time is needed before the drug reaches the lesion, and thus the drug could be metabolized, bound to protein, or diluted by the body pool. Only a limited amount of drug reaches the coronary artery and has an effect on the target lesion. Instead, intracoronary administration provides an instantaneous and high local drug concentration focused at the target lesion that effectively cleaves the thrombus.

Preliminary data suggests that intracoronary administration of tirofiban offers greater therapeutic effects than intravenous administration [7]. In this single patient experience, tirofiban did restore the coronary blood flow promptly and effectively when AST occurred. Because tirofiban blocks the final common pathway of platelet aggregation, and administration of tirofiban quickly resolved the AST, our experience is consistent with previous studies suggesting that platelets play a major role in AST.

CONCLUSIONS

Our experience with this patient showed that intracoronary administration of tirofiban may restore coronary flow in patients with ACS who have developed AST after percutaneous coronary stent implantation. However, whether this option is effective will depend on the results of further studies.

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