Successful recanalization of an occluded coronary artery by percutaneous coronary intervention, systemic administration of tirofiban, a glycoprotein IIb/IIIa inhibitor, and intracoronary thrombolysis with alteplase

Erfolgreiche Rekanalisation einer verschlossenen Koronararterie durch perkutane Koronarintervention, systemische Gabe von Tirofiban, einem Glykoprotein-IIb/IIIa-Inhibitor und intrakoronare Thrombolyse mit Alteplase


Summary A 51 year-old male was admitted to our institution with subacute inferior myocardial infarction. Coronary angiography showed thrombotic occlusion of the right coronary artery. Percutaneous coronary intervention including the delivery of 3 stents was unsuccessful (TIMI grade 0 flow). In addition to an ongoing systemic administration of tirofiban, a glycoprotein IIb/IIIa inhibitor, the patient received intracoronary thrombolysis (ICT) with alteplase (recombinant tissue type plasminogen activator, rt-PA). There was complete reperfusion on control angiography the following day (TIMI grade 3 flow); 7 months later, there was still TIMI grade 3 flow. To our knowledge, this is the first report on systemic administration of tirofiban combined with ICT.

Schlüsselwörter Myokardinfarkt – perkutane Koronarintervention – Glykoprotein-IIb/IIIa-Inhibitor – Tirofiban – intrakoronare Thrombolyse – Alteplase

Key words Myocardial infarction – percutaneous coronary intervention – glycoprotein IIb/IIIa inhibitor – tirofiban – intracoronary thrombolysis – alteplase

Introduction Intracoronary thrombolysis (ICT) was a frequently performed procedure for acute myocardial infarction or complicated percutaneous coronary intervention in the 1980s and the first half of the 1990s. Subsequently, systemic thrombolysis was found to be a superior treatment for acute myocardial infarction that was readily available in most institutions. Furthermore, the introduction of coronary stenting and gly-
coprotein IIb/IIIa blockade into interventional cardiology decreased the rate of bail out situations significantly; meanwhile, these advancements were also found to improve outcome in acute myocardial infarction in experienced centers. Consequently, nowadays ICT is used rarely in clinical practice. However, our report indicates that ICT might deserve reconsideration in the setting of massive intracoronary thrombosis as an adjunct to percutaneous coronary intervention and glycoprotein IIb/IIIa blockade.

Case report

A 51 year-old male was admitted to the cardiology department because of a 5 day history of chest pain with suspected myocardial infarction.

The patient was completely well until 5 days prior to this admission when he had acute chest pain with radiation to the neck, and diaphoresis. He received an unknown therapy by his general practitioner that included some type of injection but symptoms persisted. There was a history of hypertension, and treatment consisted of captopril 12.5 mg twice daily. The patient had quit smoking 12 months earlier. His father had suffered a myocardial infarction.

On admission, blood pressure was 170/110 mmHg, heart rate was 72/min. Body height was 173 cm, and he weighed 85 kg (body mass index 28.4 kg/m²). Physical examination was otherwise unremarkable, there were no signs of heart failure.

The ECG was consistent with subacute inferior myocardial infarction: It showed sinus rhythm with a rate of 90/min, there were significant Q-waves in leads II, III and aVF as well as descending ST-segment depression in leads I, aVL, and V2 (Fig. 1). On echocardiography, the left ventricle was found to be of normal size with symmetric hypertrophy (interventricular septum 13 mm, left ventricular posterior wall 13 mm). There was hypokinesia of the basal segments of the septum, the inferior wall, the posterior wall and the lateral wall. The left ventricular ejection fraction was 50%. Laboratory tests indicated subacute myocardial infarction (Fig. 2).

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**Fig. 1** A 12 lead ECG on admission (panel 1), after PCI (panel 2), and after intracoronary thrombolysis (panel 3). "A" indicates leads I, II, III, aVR, aVL, and aVF, "B" indicates leads V1 to V6 (from top to bottom)

**Fig. 2** Laboratory values are depicted on a logarithmic scale as a factor in relation to the upper limit of normal (CK: 3.15 μmol/l*h, aPTT: 40 s) or lower limit of normal (HCT: 40%, PLT: 150,000/μl). CK creatine kinase, aPTT activated partial thromboplastin time, HCT hematocrit, PLT platelets