Several reports have recently opened a new therapeutic window for the use of platelet glycoprotein (GP) IIb/IIIa receptor blockade as an adjunct to thrombolytic therapy in acute myocardial infarction.1,2 Because of the different protocols and the different agents that were used, as well as the relatively small number of patients included in these studies, one cannot draw any definitive conclusions about the efficacy of platelet GP IIb/IIIa receptor blockers as an adjunct to thrombolysis. Nevertheless, one could also make a case for the use of GP IIb/IIIa receptor blockers even as a monotherapy for acute myocardial infarction, as in the following case.

A 36-year-old, previously healthy male who was a heavy smoker was admitted with intermittent chest pain of 4 hours’ duration and ECG findings compatible with an acute anterior wall myocardial infarction (Figure 1). The patient was treated with aspirin (325 mg) and underwent emergency coronary angiography. This demonstrated multiple filling defects consistent with thrombotic occlusions involving the left main (Figures 2 and 3), the proximal left anterior descending, and the right (Figure 4) coronary arteries. At this stage, intravenous heparin (5000 U) was administered, achieving an activated clotting time of 265 seconds. Standard-dose, weight-adjusted abciximab was administered as a bolus, and continuous infusion was subsequently started for 12 hours, together with heparin, maintaining an activated partial thromboplastin time between 60 and 80 seconds. A few minutes after abciximab bolus injection, chest pain was relieved and gradual resolution of ST-segment elevation was apparent. Over the following 4 days, the patient remained asymptomatic. He developed a non–Q-wave myocardial infarction (Figure 5), with an increase in creatine kinase to 579 IU/L and 18% MB fraction. Echocardiography demonstrated mild septoapical hypokinesis. Repeat angiography on day 5 revealed normal coronary arteries with no evidence of residual thrombus or coronary narrowing (Figures 6 and 7). Laboratory workup revealed that the patient is heterozygous for a mutation in factor V known as activated protein C resistance (APCR), a mutation that results in an abnormal resistance to degradation by APC (frequently called factor V Leiden) and an increased tendency to thrombosis, particularly in patients who are homozygous for this mutation.3 This was detected and confirmed by a polymerase chain reaction–based test. The patient was discharged on oral therapy with aspirin and warfarin and remained symptom-free for the next 4 months.
References

