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How Innocent is the Restenosis of the Infarct-related Coronary Artery After Successful Initial Recanalization?

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ABSTRACT

The present case report describes a patient who sustained an acute inferior wall myocardial infarction, but initially remained clinically stable, then he underwent a successful coronary angioplasty and stenting procedure of a totally occluded right coronary artery, subsequently developing a dramatic clinical course with cardiogenic shock and cardiac arrest due to acute stent thrombosis which was successfully managed with repeat coronary angioplasty. We attributed this discrepant clinical manifestation of acute coronary occlusion to coronary collaterals, initially being present and then disappearing following the recanalization procedure, as being responsible for the dramatic clinical picture following the stent thrombosis.

CASE REPORT

A 59-year-old gentleman was transferred to our hospital for coronary angiography after an acute myocardial infarction. He had developed chest discomfort and one hour later he had been admitted to the local hospital. The admitting physician diagnosed acute inferior wall and right ventricular acute myocardial infarction. Thrombolysis was administered immediately and the patient's symptoms abated. The patient was subsequently transferred to our hospital to undergo coronary angiography. Upon admission cardiac troponin I levels were elevated to 8.18 ng/ml.

The following day he underwent coronary angiography which demonstrated a total proximal occlusion of the right coronary artery. Adhoc coronary angioplasty and stenting was performed successfully. After the procedure, the patient was transferred to the cardiology ward in stable condition without any symptoms. However, a few hours later, the patient developed acute chest pain and the ECG showed elevation of the ST segment localized to the inferior wall leads. The patient's status deteriorated quickly into cardiogenic shock, and the ECG showed ventricular flutter. A DC shock with 300 joules restored sinus rhythm and the patient required the use of vasopressor and inotropic agents (dopamine and dobutamine) for hemodynamic support. In view of the presence of right ventricular involvement, IV volume repletion was also initiated with use of normal saline. The patient was then transferred immediately to the catheterization laboratory for emergent repeat coronary angiography.

Coronary angiography demonstrated acute reocclusion at the site of the stent implantation (acute stent thrombosis).The patient underwent successful coronary

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Address for correspondence: Nikolaos Patsouras E-mail: nikospatsouras@pat.forthnet.gr angioplasty and was moved to the coronary care unit for close monitoring. However, he remained hemodynamically unstable with signs of pulmonary congestion. Supplemental oxygen via mask was used to improve arterial oxygenation and the patient promptly received IV diuretics. Patient's symptoms were related to dyspnea without chest discomfort.

Due to the prolonged period of hypotension, liver enzymes were elevated. An episode of atrial flutter occurred which was initially reverted to sinus rhythm with the intravenous use of amiodarone but half an hour later atrial flutter recurred. An echocardiographic examination revealed dilation of the right ventricle with reduced systolic function and moderate to severe tricuspid regurgitation.

Over the next four days the patient's condition improved and there was gradual reduction in the dose of dopamine and dobutamine. Right heart catheterization revealed pulmonary artery saturation 61% and arterial saturation 96%. The patient was subsequently improved clinically and hemodynamically and finally discharged home on the 14th day of hospitalization with mild symptoms. One year later the patient remains asymptomatic.

DISCUSSION

This case report illustrates that acute in-stent thrombosis might result in an unpredictable dramatic course despite an initial clinical stability in the presence of an occluded coronary artery. We suggest that rapid disappearance of collaterals is a possible underlying pathophysiological mechanism that might have had a great impact on the clinical manifestation in this case of reocclusion of the infarct related coronary artery.