ELSEVIER

Cardiovascular Revascularization Medicine 9 (2008) 56–61

CARDIOVASCULAR REVASCULARIZATION MEDICINE

The pathogenesis and treatment of no-reflow occurring during percutaneous coronary intervention

Mohammad-Reza Movahed*, Samuel M. Butman

Department of Medicine, Sarver Heart Center, University of Arizona Medical Center, Tucson, AZ 85724, USA

Received 21 August 2007; accepted 28 August 2007

Abstract No-reflow is one of the major causes of postinterventional rise of cardiac enzyme and myocardial infarction (MI). This complication is associated with substantial morbidity and mortality after percutaneous coronary intervention (PCI). During and after a no-reflow episode, the patient can suffer from severe chest pain, hypotension, bradycardia, hemodynamic collapse, MI, congestive heart failure, and death. Every effort should be taken to reduce the incidence of this complication. The distal embolic protection device has been shown to decrease this risk in saphenous vein graft (SVG) interventions but not in native coronaries. On the other hand, the use of glycoprotein IIb/IIIa receptor antagonists have been effective in reducing the occurrence of no-reflow during PCI of native coronaries but not during SVG interventions. The treatment of no-reflow is based on the intracoronary administrations of medications that induce maximal vasodilatation in small distal coronary vasculature. The most commonly used drugs in this setting are adenosine, nitroprusside, and verapamil. The goal of this study was to review the pathogenesis and treatment of no-reflow in patients undergoing PCI.

Keywords: No-reflow; PCI; Stenting; PTCA; Balloon angioplasty; Percutaneous coronary intervention; Complications

1. Case report

The patient was a 71-year-old male with a history of CABG and angioplasty presenting with unstable angina. Cardiac catheterization revealed chronic occluded circumflex (CFX) and left anterior descending (LAD) arteries. Right coronary artery (RCA) had 90% proximal and 70% mid-RCA lesions (Fig. 1). Saphenous vein grafts (SVGs) to LAD and CFX were patent, but SVG to RCA was 100% occluded. The decision was made to proceed with percutaneous coronary intervention (PCI) of RCA. RCA was wired using a high-torque floppy wire (Guidant Inc.). Two 2.5×16 mm Taxus (Boston Sci. Inc.) stents were advanced and placed in the proximal and mid-RCA. Poststenting, there was a remaining high-grade lesion between the two stents in the mid-RCA (Fig. 2), which was stented using a 2.5×16 Taxus stent followed by stenting of the RCA ostium using a 2.5×16 Taxus stent. Poststenting, the patient suffered from severe chest pain, ST elevation, hypotension, and no-reflow (Fig. 3). Multiple doses of intracoronary nitroprusside and verapamil were given, which gradually resolved the no-reflow (Fig. 4). The patient recovered from the acute episode but had significant rise in the postprocedural CPK.

2. Epidemiology

As the number of PCIs rises each year, complications remain a major cause of morbidity and mortality in these

* Corresponding author. Division of Cardiology, Department of Medicine, University of Arizona Sarver Heart Center, 1501 North Campbell Ave., Tucson, AZ 85724, USA. Tel.: +1 520 626 2000; fax: +1 949 400 0091.
E-mail addresses: rmovahed@email.arizona.edu, rmova@aol.com (M.-R. Movahed).

1553-8389/08/$ – see front matter © 2008 Elsevier Inc. All rights reserved.
doi:10.1016/j.carrev.2007.08.005

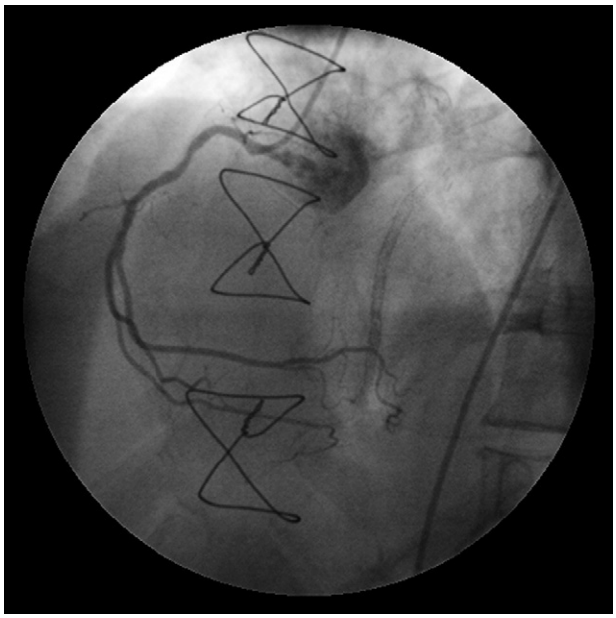

Fig. 1. High-grade mid-RCA and proximal RCA stenosis.

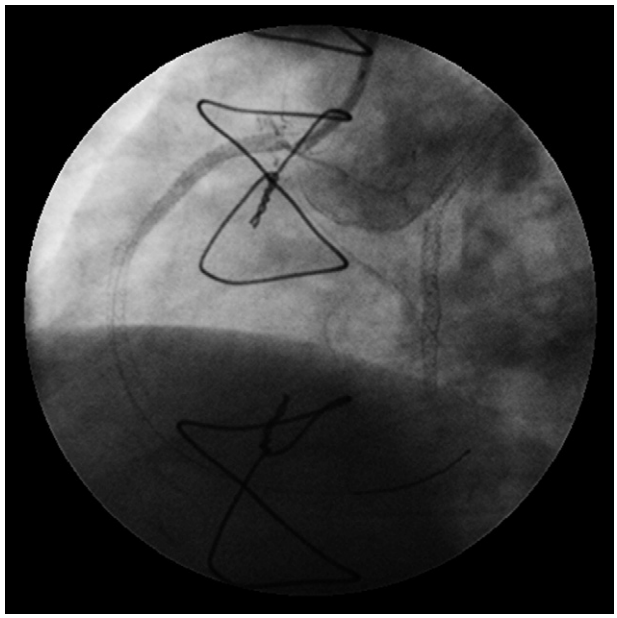

Fig. 3. No-reflow occurring after stenting of mid-RCA.

patients. No-reflow is defined as a failure to restore antegrade normal coronary flow despite appropriate treatment of coronary obstruction [1,2]. The prevalence of this complication occurs in 0.6% to 5% of PCIs [3,4]. The incidence of no-reflow appears to be highest in patients undergoing PCI of SVGs, during acute myocardial infarction (MI) or during rotational atherectomy [5]. It can occur in as high as 50% of PCI cases involving the treatment of thrombus-containing lesions [6]. Furthermore, a history of diabetes mellitus, or the absence of preinfarct angina, was found to increase the risk of no-reflow [7].

3. Pathophysiology

The cause of no-reflow is complex and multifactorial. It appears to be related to damage to microvascular structures but is not completely understood. Multiple mechanisms have been suspected as the cause of no-flow, such as

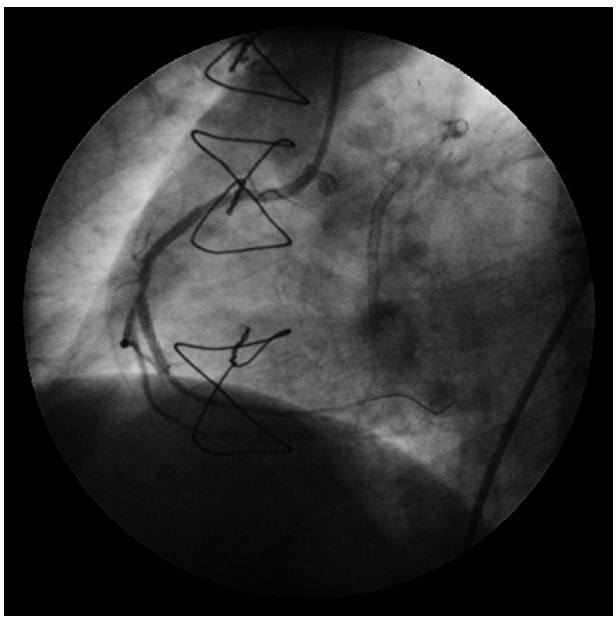

Fig. 2. Residual mid-RCA stenosis after two stents.

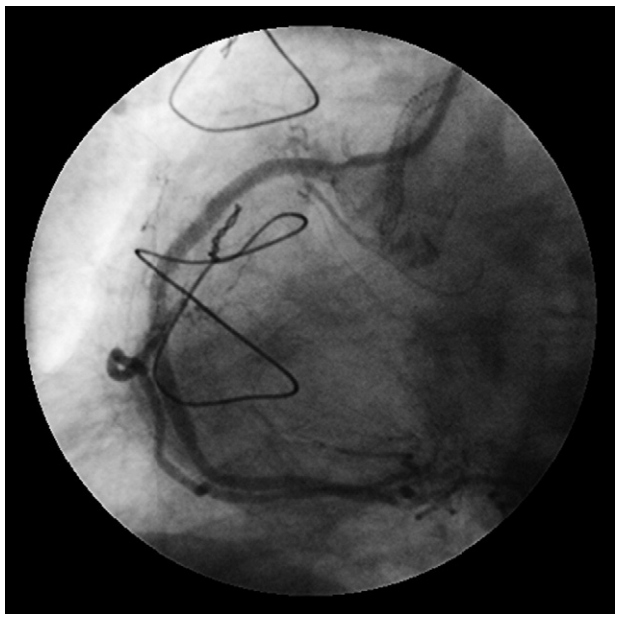

Fig. 4. Resolution of no-reflow after intracoronary verapamil and nitroprusside administration.