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CASE REPORT

Development and Management of No-Reflow in a Patient Performed Coronary Angiography for Acute Coronary Syndrome: A Case Report

Akut Koroner Sendrom nedeniyle Koroner Anjiyografi Yapılan Bir Hastada No-Reflow Gelişimi ve Yönetimi: Olgu Sunumu

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Abstract

Despite eliminating epicardial coronary artery stenosis or occlusion, insufficient myocardial perfusion is called "no-reflow." While the no-reflow phenomenon is defined as coronary flow below increased thrombolysis (TIMI) 3 in myocardial infarction in some sources, TIMI is defined as 0–1 flow in some sources. In addition, some publications refer to TIMI 2 flow as slow coronary flow. However, the general view is that TIMI 0–1 coronary flow without severe residual stenosis, spasm, thrombus, or dissection is called no-reflow. The no-reflow phenomenon is a condition in which blood flow is significantly reduced in the ischemic myocardium despite percutaneous coronary intervention. So far, no standard treatment has been established to cure this condition. In this case, we present an interesting case that demonstrates a practical approach to the no-reflow phenomenon.

Keywords: Coronary artery disease; No-reflow phenomenon; Percutaneous transluminal angioplasty

A 58-year-old male patient with no known history of coronary artery disease was admitted to the emergency department with typical complaints of chest pain and fatigue. Electrocardiography (ECG) of the patient at admission was sinus rhythm, and no pathological condition was observed (Fig. 1). In the physical examination, the general condition was good, conscious, oriented, and cooperative; blood pressure was 132/83 mmHg; and heart rate was 83 min^{-1}; respiratory rate was 23 min^{-1}. No other pathology was found in the physical examination. He had a history of smoking for 10 years. In the first cardiac enzyme results of the patient, troponin I was 432 (0–45), while creatine kinase-MB was normal. In echocardiographic evaluation, left ventricular ejection fraction was 55%, and there was trace mitral regurgitation; however, there were no wall motion abnormalities.

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The patient was admitted to the coronary intensive care unit with the prediagnosis of non-ST-elevation myocardial infarction (NON-STEMI). Anticoagulant (enoxaparin 0.6 2 × 1 sc), antiplatelet (clopidogrel 300 mg p.o., acetylsalicylic acid 100 mg p.o.), anti-ischemic (metoprolol 50 mg 1 × 1 p.o.), antihyperlipidemic, and antihypertensive treatments were applied to the patient. Coronary angiography (CAG) was performed approximately 12 h after hospitalization. CAG showed 90% lesion in the proximal segment of the left anterior descending artery (LAD). The circumflex artery was seen as plaque in the mid-region, and the right coronary artery was normal (Fig. 2).

Percutaneous transluminal angioplasty was decided for the lesion in the LAD. The left main coronary ostium was cannulated with a left guiding catheter. The lesion was crossed with a guidewire, predilatation was achieved with a 2.00 × 15 mm balloon, and a 3.5 × 12 mm drug-eluting stent was implanted in the lesion. Afterward, when the stent was not fully opened, postdilatation was performed with a 4.0 × 12 mm noncompliant (NC) balloon to achieve optimal patency. After the NC balloon was made, no-reflow developed during the procedure, and the distal flow in the LAD was observed as TIMI 0 (Fig. 3). The balloon was withdrawn from the coronary. Dosages of 4 cc nitroprusside and 2 cc diltiazem were administered intracoronary immediately. Then, the left guiding catheter was maneuvered slightly backward from the coronary. Bradycardia was not observed during the monitor follow-up, the patient' s blood pressure was stable, the LAD was redisplayed later during the procedure, and TIMI 3 flow was observed (Fig. 4). The patient was taken to the coronary intensive care unit and followed up. No wall motion defect was observed in the echocardiography performed after the procedure. The patient, who had no ECG changes in the follow-ups, did not describe chest pain, and whose troponin value decreased, was discharged and called for outpatient polyclinic control.

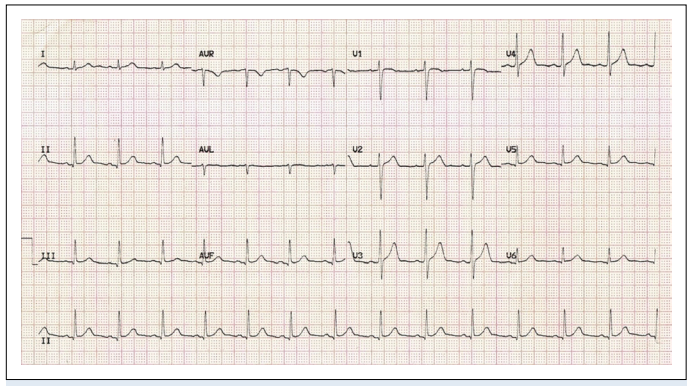

Figure 1. Electrocardiography of the patient at the first admission.

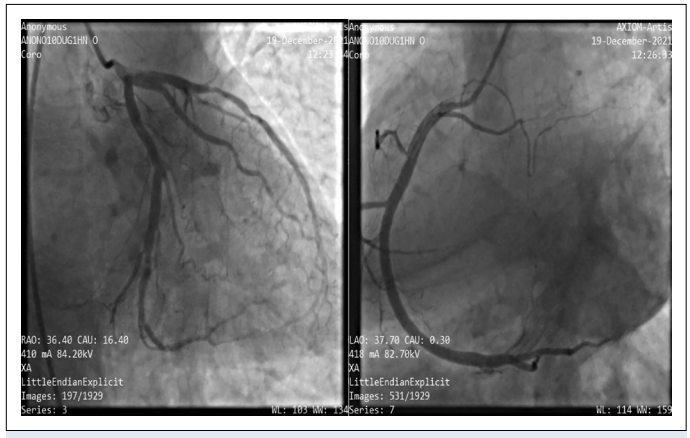

Figure 2. Coronary angiographic image of the patient before percutaneous transluminal angioplasty.

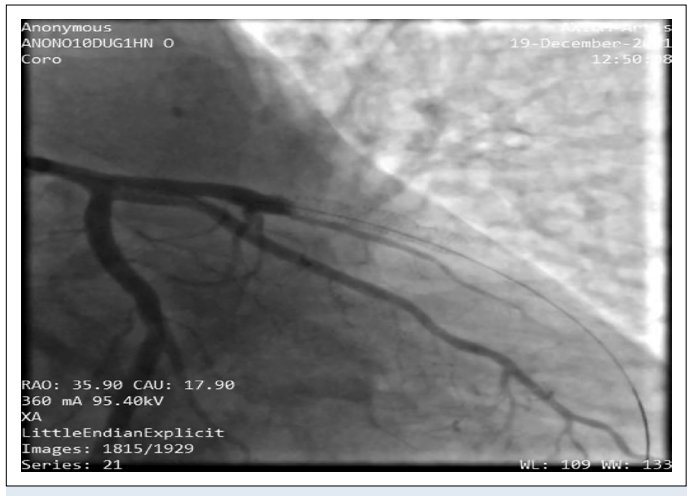

Figure 3. No-reflow development in the left anterior descending artery after percutaneous transluminal angioplasty.

Discussion

In the case of NON-STEMI, we experienced a case of a no-reflow phenomenon that occurred after stent placement in the LAD. We successfully managed this condition with the intracoronary injection of nitroprusside and diltiazem and gentle withdrawal of the catheter. There is no established treatment with universally accepted evidence for the no-reflow phenomenon, and various pharmacological treatments (adenosine, anisodamine, diltiazem, nicorandil, nitroprusside, urapidil, and verapamil) have been reported.[1,2] Results of meta-analyses showed that intracoronary injection of nitroprusside was beneficial in preventing the no-reflow phenomenon, improving TIMI flow rate and left ventricular ejection fraction.[3] It also reduces adverse reactions in patients after PCI and rehospitalization due to cardiovascular events.[4,5]

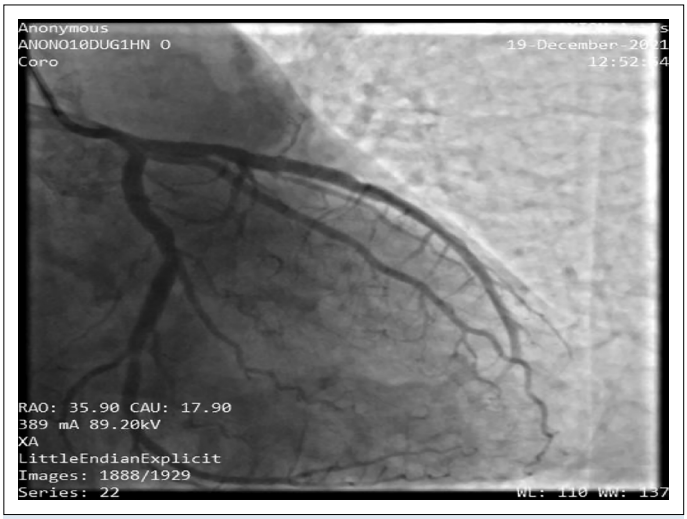

Figure 4. Improvement of no-reflow after intravenous nitrate and diltiazem injection and after catheter manipulation.

Since nitroprusside causes coronary hyperemia through dilation of coronary microvessels, intracoronary nitroprusside is likely to have a beneficial effect on the treatment and prevention of the no-reflow phenomenon.[6] Zhao et al.[4] administered intracoronary nitroprusside infusion using the thrombectomy catheter for the no-reflow phenomenon. In our case, we did not use any thrombectomy catheter. We reasoned that in the no-reflow/slow-flow phenomenon, drugs could not reach the distal microvessels efficiently from the guiding catheter and that nitroprusside had a risk of lowering systemic blood pressure when leaking from the coronary artery into the aorta. An animal study showed that after prolonged myocardial ischemia, intra-aortic balloon pump (IABP) assistance begins only 10 min before reperfusion increases coronary blood flow, and the no-reflow phenomenon limits the size and extent of the limited infarct.[7] As the benefit of IABP in patients with acute myocardial infarction is still controversial, we did not use it in our case. The total trial, a large randomized trial on primary PCI with or without routine manual thrombectomy, showed no benefit for the routine use of thrombectomy.[8,9] Thrombectomy is likely beneficial for patients with very large thrombi although this has not been robustly proven by randomized controlled evidence. Routine thrombus aspiration is also not recommended in the ESC guideline.[10] As a result, we did not use this maneuver due to the risk of stopping the coronary blood flow again and the current risk of stroke. We thought that this was due to impaired blood flow in microvessels rather than thrombus obstruction in the epicardial coronary lesion. Therefore, we tried to provide blood flow by strong vasodilation with the intracoronary injection of nitroprusside and diltiazem. In conclusion, we think that intracoronary injection of nitroprusside and diltiazem and withdrawal of the left guiding catheter for the no-reflow phenomenon may be beneficial in the treatment of the no-reflow phenomenon during PCI for NON-STEMI.

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Informed Consent: Written informed consent was obtained from patients who participated in this study.

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Conflict of Interest: None declared.

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