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Case Report

A Triple Disaster Phenomenon For the Coronary Artery An Impressive Case of No-reflow and Complication Management

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Abstract

Background: Coronary no-reflow is characterized by very slow or absent epicardial blood flow. It results in inadequate myocardial perfusion and is an independent predictor of mortality due to malignant arrhythmias and heart failure.

Case Illustration: This case describes a patient who underwent angiography due to a reperfused inferior myocardial infarction. Following the diagnostic procedure, a decision was made to perform a primary percutaneous intervention on the Circumflex (Cx) artery. When the stent balloon was inflated to the optimal pressure, it burst. The control image revealed the development of no-reflow after the Left Main Coronary Artery (LMCA). This potentially fatal condition was successfully treated with the administration of Intracoronary (IC) adrenaline.

Conclusions: No-reflow is a significant factor affecting mortality. The occurrence of this phenomenon in multiple vessels further increases this risk, but it can be effectively managed with IC adrenaline, and multiple-vessel no-reflow can be treated

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Introduction

Coronary no-reflow phenomenon is defined as very slow or absent distal flow in the absence of angiographic findings of vascular occlusion, spasm, or dissection in epicardial arteries.[1] It complicates ST Elevation Myocardial Infarction (STEMI) cases, increasing the incidence of malignant arrhythmias and heart failure, and is an independent predictor of mortality.[2-3] Balloon rupture may occur during percutaneous coronary interventions when the inflation pressures determined by the manufacturer are exceeded and may lead to severe arterial damage, perforation, or slowing of coronary blood flow.[4] In this case report, we describe a patient with acute coronary syndrome who underwent Primary Percutaneous Coronary Intervention (PPCI) with IC adrenaline treatment of no-reflow after proximal rupture of the stent balloon.

Case Illustration

A 53-year-old male patient with no known history of chronic disease and a smoking history of 40 pack/year was admitted to the emergency department of an external center with the complaint of chest pain that started in the early morning and awakened him from sleep. With the diagnosis of acute inferior Myocardial Infarction (MI) based on Electrocardiography (ECG) (Figure 1), the patient was given 300 mg acetylsalicylic acid, 180 mg ticagrelor, and 5000U heparin and referred to our hospital for primary PCI. The patient's door-to-lab time was 65 minutes. Therefore, thrombolytic therapy was not administered to the patient. Despite not receiving thrombolytic therapy, the patient's ECG showed normalization of the ST segment in the inferior leads (Figure 2). The patient was admitted to the coronary angiography laboratory with suspected reperfused inferior MI. During this

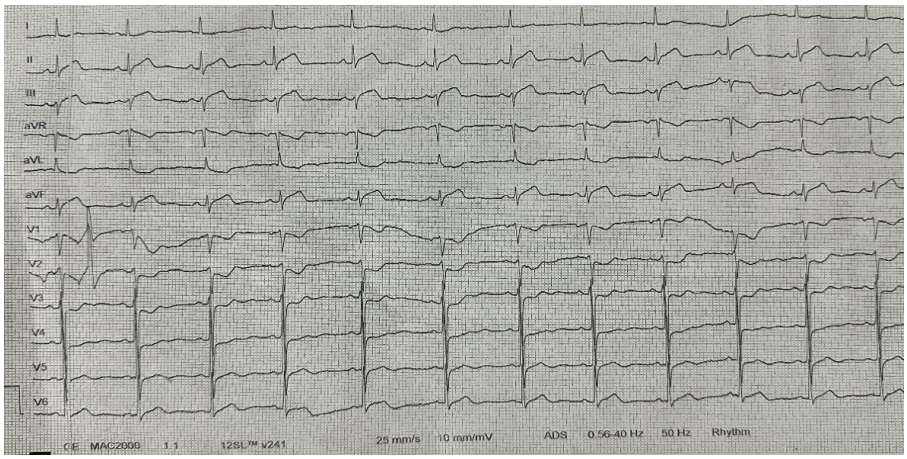

Figure 1. ECG of the patient on admission to an external center.

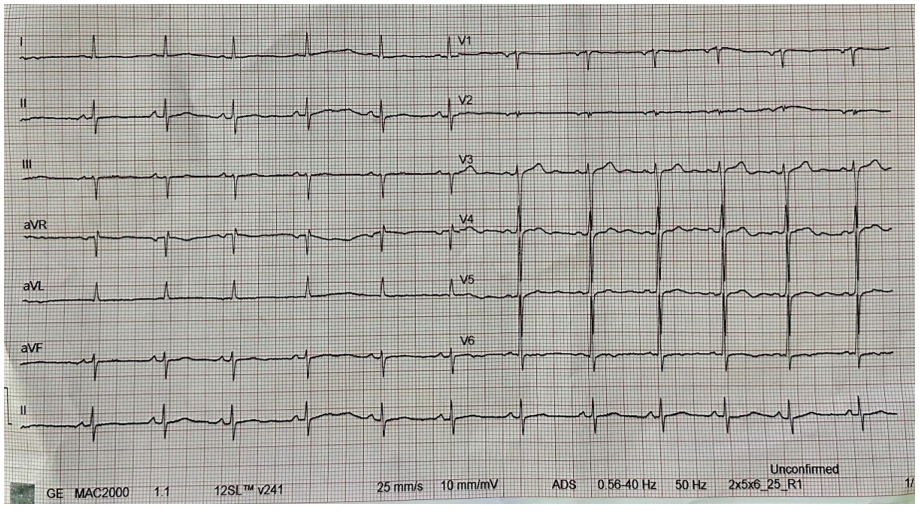

Figure 2. ECG of the patient on admission to our clinic.

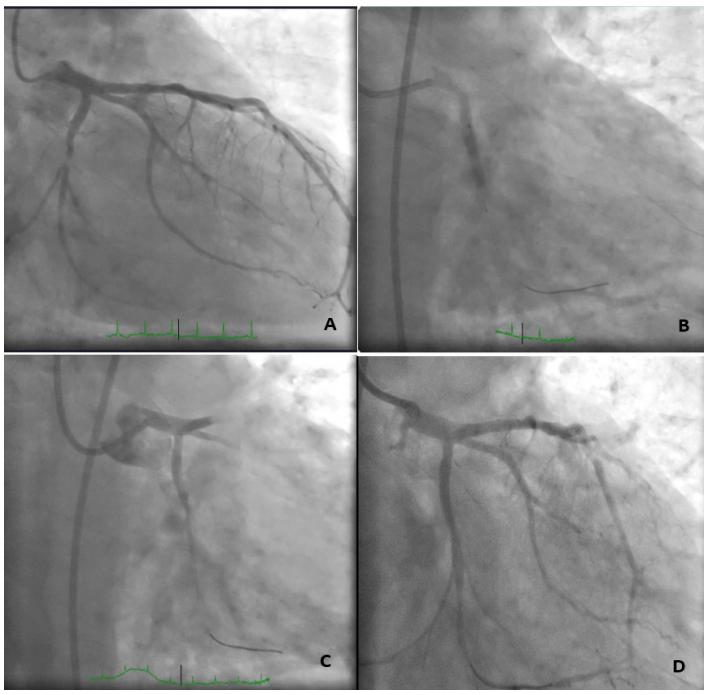

Figure 3. A. 95% critical stenosis in the mid segment of the Cx on coronary angiography, B. proximal rupture of the stent balloon during stent implantation, C. no-reflow after LMCA on control image, D. recovery of no-reflow after intracoronary 250 μg adrenaline.

process, the patient's hemodynamics were stable, chest pain had decreased, and blood pressure was 130/80 mmHg.

Coronary angiography showed a 95% critical stenosis in the mid-segment of the Circumflex (Cx) artery, and PPCI of the Cx was decided. The lesion was crossed with a floppy guidewire, and predilatation was performed with a 2.0 × 18 mm Noncompliant (NC) balloon. A 2.75 × 24 mm Drug-Eluting Stent (DES) was to be implanted in the lesion site. The stent's nominal pressure was 10 atmospheres (atm), and its burst pressure was 18 atm. The stent balloon burst when inflated to 14 atm. This was thought to be due to a calcified spicule proximal to the Cx lesion. Subsequent imaging revealed no-reflow developing after the Left Main Coronary Artery (LMCA), with distal flow in the left system being Thrombolysis In Myocardial Infarction-0 (TIMI-0). The patient developed acute chest pain, blood pressure started to drop, and confusion and dizziness started. The follow-up monitoring showed the development of ST-segment elevation. The patient, who was not bradycardic, immediately received an Intracoronary (IC) 300 μg adenosine bolus and an IC abciximab bolus at a weight-appropriate dose. In the control image obtained, it was observed that the image was consistent with no reflow. Subsequently, 250 μg of adrenaline was administered IC into the LMCA through the left guiding catheter. When preparing this solution, a 1 mg ampoule containing 1 mg/1 ml of adrenaline was used. 1 ml of the ampoule was diluted with 10 ml of physiological saline. 1 ml of this solution was taken, and the same solution was prepared in another syringe. The defibrillator was set up to counter the risk of ventricular arrhythmia, and vasodilator agents were kept on hand. No-reflow resolved, and the control image revealed distal flow in the left coronary system as TIMI-3 (Figure 3), and the stent implanted in the Cx was found to be patent. No arrhythmia developed. The patient's chest pain resolved, and normotension was achieved. The monitor showed a regression in ST-segment elevation.

Hemodynamic stabilization was achieved. It was decided to continue abciximab treatment and follow-up in the intensive care unit. Control angiography was planned after abciximab infusion, and the procedure was terminated.

Subsequent control angiography revealed that the Cx stent was patent, with distal flow in the Cx and other vessels classified as TIMI-3. Troponin levels decreased. The left ventricular ejection fraction (LVEF) was found to be 50%. The patient, whose complaints had subsided and whose ECG showed no signs of ischemia, was discharged after a clinic follow-up, and medical treatment was arranged.

When the patient returned to the clinic one month later, he stated that he had no active complaints. His medication was reviewed, and medical follow-up continued.

Discussion

Development of no-reflow may lead to important outcomes such as malignant ventricular arrhythmias, death, recurrent MI, decreased left ventricular ejection fraction, heart failure, and cardiac rupture.[5] A meta-analysis showed that factors such as advanced age, smoking history, diabetes mellitus, hypertension, lesion length, multiple vessel disease, and reference lumen diameter were associated with the risk of no-reflow in patients with STEMI.[6]

Cases resulting in balloon rupture during coronary interventions have been reported. Sofidis et al. reported that no-reflow developed in the index vessel due to distal embolization as a result of rupture of the semicompliant balloon when the inflation pressure was exceeded during PCI.[7] In our case, the calcific lesion was prepared with an NC balloon, but the stent balloon was semicompliant, resulting in balloon rupture despite inflation at optimal pressure. Moreover, because the balloon rupture was proximal, the balloon contents were not only embolized distal to the index vessel but also to other vessels in the left coronary system, resulting in no-reflow in all vessels after the LMCA. STEMI patients represent a high-risk group in terms of no-reflow. Our case demonstrates that a procedure-related complication during PPCI may put not only the occluded vessel but also other vessels at risk of no-reflow, and suggests that intravascular imaging methods may be used to evaluate lesions in patients with reperfused STEMI.

The treatment of no-reflow is based on maximal vasodilation of small distal coronary arterioles, and the use of glycoprotein IIb/IIIa receptor antagonists is also effective in preventing no-reflow during PCI.[8] Verapamil, adenosine, or nitroprusside is frequently used as an IC to eliminate this phenomenon.

In a related case report, Simoni et al. showed that a no-reflow phenomenon developing in the index vessel in a patient undergoing PCI with a diagnosis of Non-STEMI was reversed with 100 μg IC adrenaline in first-line treatment due to the development of hypotension.[9] In our case, IC adrenaline was not used as first-line treatment due of the absence of hypotension. However, coronary flow could not be established in the left coronary system despite primary treatment. With the development of hypotension, 250 μg of adrenaline was administered, and the no-reflow phenomenon outside the index vessel resolved, restoring flow in the left coronary system. This case not only demonstrated the efficacy of adrenaline on the no-reflow phenomenon occurring in multiple vessels, but also prevented a potentially fatal course of the patient by restoring flow in a system that supplies blood to a very important part of the heart.

At the receptor level, the vessels supplying the heart contain alpha (a) and beta (b) receptors. Adrenaline causes vasodilation by activation of β2 receptors in coronary arterioles and has inotropic and chronotropic effects by activation of β1 receptors.[10] However, despite these, if administered at high doses, it causes malignant arrhythmias or coronary spasm as a result of activation of a1 receptors.[10] In this case, we have demonstrated that multi-vessel no-reflow was safely corrected with a dose of 250 μg of adrenaline, and we report achieving hemodynamic stabilization without any arrhythmia in the patient. However, further and larger studies are needed to determine safe and effective limits regarding the dose of adrenaline and the protocol for its administration in no-reflow.

Conclusion

No-reflow causes impaired myocardial perfusion, especially in patients presenting with acute coronary syndromes, and adversely affects the mortality or morbidity of patients in the acute or chronic period. The most important treatment for the physician will be to prevent its development. In patients with reperfused STEMI who are hemodynamically stable, especially in the absence of chest pain, the use of intravascular imaging methods to determine the character of the lesion in the responsible vessel may be useful in the primary prevention of the no-reflow phenomenon, which may occur during PCI due to procedure-related complications, complicating the case.

The use of IC adrenaline in patients with multivessel no-reflow phenomenon should be considered as an effective method to restore flow in all vessels with no-reflow. More studies are needed to determine the standardization of no-reflow treatment in general.

List of Abbreviations

Cx	Circumflex
DES	Drug Eluting Stent
ECG	Electrocardiography
IC	Intracoronary
LMCA	Left Main Coronary Artery
NC	Noncompliant
PPCI	Primary Percutaneous Coronary Intervention
STEMI	ST Elevation Myocardial Infarction
TIMI	Thrombolysis In Myocardial Infarction

Ethical Clearance

Not applicable.

Publication Approval

All authors approve the manuscript for publication.

Authors' Contributions

EÇ was the main author who conceptualized the case, provided clinical supervision, and made critical contributions to the manuscript's writing and final revision. ÇK and KY assisted with literature searching and manuscript writing. EÇ, ÇK, and KY provided valuable clinical input, reviewed the manuscript, and offered additional insights on the case details and clinical implications discussed in the manuscript.

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Conflict of Interest

The authors declare no conflicts of interest.

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Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

There is no AI tools involved in the writing process of this manuscript. Manuscript were drafted manually. Proofread and spell-check were also performed manually by authors.

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