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Coronary No-Reflow Following Percutaneous Intervention

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ABSTRACT

Normally, coronary revascularization with percutaneous coronary intervention (PCI) leads to restoration of normal coronary blood flow through the coronary arteries, with reperfusion of the myocardium. However, there is a group of patients who do not benefit from prompt restoration of antegrade flow, as they fail to show resolution of the indirect signs of ischemia such as electrocardiographic (ECG) changes and improvements in perfusion abnormalities. This condition is referred to as "no-reflow phenomenon." In this case report, we will discuss the case of a patient who developed coronary no-reflow immediately following PCI.

Keywords

Coronary interventions, Blood flow, Hypertension.

Introduction

Primary percutaneous coronary intervention (PPCI) is the gold standard of treatment of ST segment elevation myocardial infarction (STEMI). PPCI restores thrombolysis in myocardial infarction flow 3 (TIMI 3) perfusion in over 90% of patients [1]. However, in rare instances, coronary revascularization does not lead to coronary reperfusion; instead there is a further decrease of coronary blood flow. The mechanism of this phenomenon is not well understood, but there are several theories for its pathophysiology which will be investigated. Initial theories suggested that prolonged ischemia and extensive myocardial damage led to microvascular damage, which subsequently causes incomplete reperfusion. In summary, the cause of no-reflow can be classified into four main pathogenetic components: distal atherothrombotic embolization, ischemia-related and/or reperfusion-related injury, and the susceptibility of coronary microcirculation to injury. Although the exact mechanism of no-reflow remains unknown, it is most likely complex and multifactorial [2]. The no reflow phenomenon can happen during elective or primary percutaneous coronary intervention [3].

There are multiple predictors, both modifiable and non-modifiable risk factors, for no-reflow: age, smoking, time-to-treatment interval, left ventricular ejection fraction (LVEF), previous myocardial infarction, Killip class, serum creatinine, C-Reactive Protein (CRP), B-type natriuretic peptide (BNP), baseline TIMI flow grade, elevated blood glucose, long lesion, higher reperfusion time, presence of collateral circulation, higher thrombus burden prior to PCI, intra-aortic balloon pump (IABP) placement prior to PCI, and initial perfusion defect [4].

The purpose of this case report is to show early and clear identification of the no-reflow and follow its management. It can occur in up to 10% of cases of primary PCI and is associated with an increased 30-day mortality if not adequately treated (32% vs. 2.8%, p<.0.001) [3].

Case Report

A 76-yearr-old female presented to the our hospital with new-onset substernal chest pain over 3 days. Her primary care physician initially diagnosed her with gastroesophageal reflux disease (GERD), but then her chest pain acutely worsened and she developed dyspnea on exertion as well. She has a past medical history of hypertension, hyperlipidemia, type 2 diabetes mellitus, Raynaud's disease, rheumatoid arthritis, degenerative joint disease, and Sjögren's disease. Her electrocardiogram (EKG) on admission showed an anterolateral wall ST-elevation myocardial infarction (STEMI) for which she had to be taken emergently for left heart catheterization. Her vitals were the following: blood pressure 52/44 mmHg, pulse of 122 beats/min, respiratory rate of 20 breaths/min, temperature 36.8 C, and SpO_2 of 100% on room air. On physical exam, her findings were unremarkable.

However, the patient had a very complex hospital course following her PCI during left heart catheterization. Coronary angiography via a right radial approach showed no significant stenosis of the right coronary artery and an acute occlusion of the left anterior descending coronary artery (LAD) from the ostium (Figure 1).

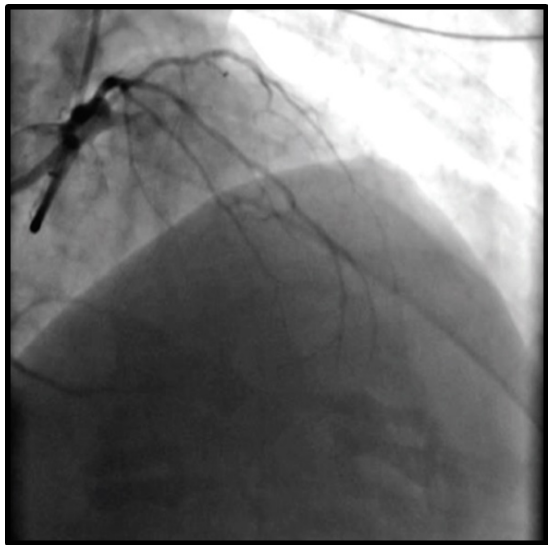

Figure 1: Acute occlusion of the left anterior descending coronary artery (LAD) from the ostium.

Due to significant thrombus burden, she had a drug-eluting stent (DES) placed to a 100% lesion of the proximal LAD but there was no-reflow following placement. Afterwards flow in the LAD was scored at Thrombolysis in Myocardial Infarction (TIMI) 1 (Figure 2).

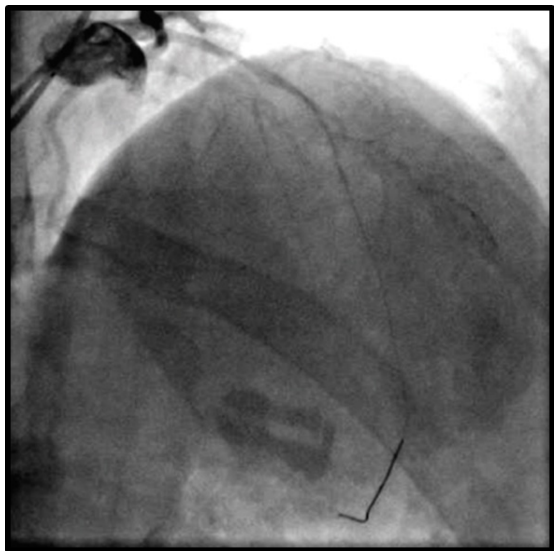

Figure 2: Drug-eluting stent (DES) placed to 100% lesion of the proximal LAD but there was no reflow following placement. Afterwards flow in the LAD was scored at Thrombolysis in Myocardial Infarction (TIMI) 1.

Subsequently, the patient became bradycardic, hypotensive, and agonal; within a few minutes she had two episodes of cardiac arrest with return of spontaneous circulation (ROSC) achieved with CPR, epinephrine, and atropine. She then had to be sedated and intubated and started on a norepinephrine drip for hypotension.

Left heart catheterization revealed a left ventricular end-diastolic pressure of 40 mmHg and the estimated left ventricular ejection fraction by ventriculography was 20-25%. There was subsequent placement of IABP for stabilization of hemodynamics and upfront left ventricular unloading for cardiogenic shock [4]. This was then upgraded to an Impella via the right common femoral artery. An Impella was placed via the right common femoral artery. An intracoronary injection of nicardipine, adenosine, and eptifibatide with a micro-catheter resulted in mild improvement in TIMI flow, at the distal LAD, to TIMI 2 (Figure 3).

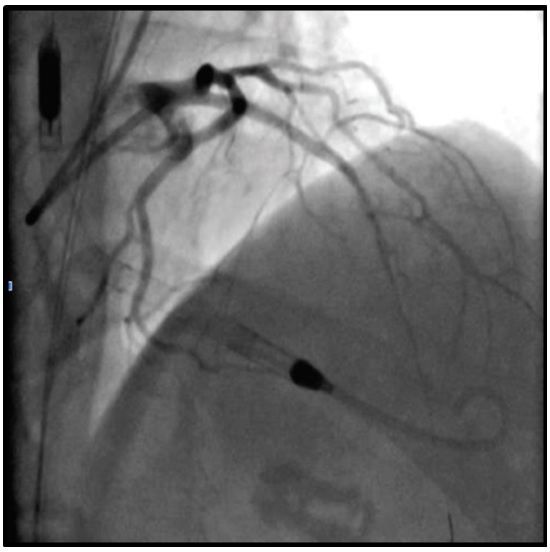

Figure 3: Intracoronary injection of nicardipine, adenosine, and eptifibatide with a micro catheter resulted in mild improvement in TIMI flow at the distal LAD, to TIMI 2 (Figure 3).

Unfortunately, there was then subsequent development of Impella® site bleeding and the development of a right groin hematoma. On day 2 of hospitalization, the patient's mean arterial pressure (MAP) dropped below 65 mmHg with increasing vasopressor requirements. Hemoglobin dropped to 5.5 mg/dL following 8 units of packed red blood cells, which was concerning for acute blood loss. On re-examination, the patient's right groin hematoma was expanding to the abdominal wall and down her right thigh. At this point, the patient was urgently transferred to a tertiary care center for further evaluation.

Discussion

For our patient, PCI did not lead to the usual and expected restoration of coronary blood flow. The patient had evidence of angiographic no-reflow phenomenon characterized by evidence of slow-flow in the affected vessel (TIMI flow equal to or less than 2) and lack of contrast uptake "blush" by the subtended myocardium, leading to a potential dissociation between coronary revascularization and myocardial perfusion in STEMI [2]. She also had several risk factors, both modifiable and non-modifiable, that were predictors of no reflow: age, smoking, time-to-treatment

interval, serum creatinine, baseline TIMI flow grade, elevated blood glucose, and higher thrombus burden prior to PCI [4].

There are several treatment options for no reflow, although it is not known which treatment is best; this will require more in-depth study in the future. Current medical treatment options are the following: injection of adenosine (dilator of arteries and arterioles), nitroprusside sodium (relaxes arteries and veins), or verapamil (smooth muscle dilator) [3]. Our patient was given an adenosine injection when the no-reflow was recognized. Mechanical support devices can also be used in addition to medical management, although routine manual thrombus aspiration has not been shown to improve cardiovascular death, recurrent MI, cardiogenic shock, or heart failure, but a theoretical benefit to thrombectomy persists for improving thrombus burden and preventing microvascular plugging in selected cases of high thrombus burden [2]. Also, an IABP increases coronary blood flow, decreases myocardial oxygen demand, and can mitigate the no reflow; left ventricular unloading improves coronary flow and reduces myocardial oxygen demand. Thus, the Impella® should be considered upfront in patients with elevated left ventricular end-diastolic pressure, low ejection fraction, and early cardiogenic shock, and with persistent symptoms, as our patient experienced [2].

The impact of no-reflow should not be underestimated. The development of no-reflow phenomenon is a poor prognosticator because it is associated with considerable reduction of the myocardial salvage by primary PCI in patients with STEMI. Because reduced myocardial salvage results in larger myocardial necrosis, no-reflow subsequently influences left ventricular function and mortality [4]. One-year mortality was 16.7 percent in patients with no-reflow versus 5.5 percent in patients with normal flow. Six-month follow-up angiography on patients with no-reflow showed that only 20 percent continued to have slow TIMI flow, with normalization of TIMI flow in the majority (80 percent) of patients. Recently, long-term prognostic data have also been published and confirmed the persistent poor prognostic effect of no-reflow causing an increase in five-year mortality from 9.5 to 18.2 percent [2]. Our patient decompensated acutely and very rapidly, requiring multiple vasopressors and intubation for support. Within minutes of the no-reflow, our patient went into cardiac arrest and required very aggressive resuscitation.

Conclusion

This is not the first occurrence of coronary no-reflow. However, the prognosis and presentation of patients can vary. There are no randomized, controlled trials large enough to demonstrate hard clinical endpoint benefits from a single pharmacological or mechanical agent in treating the no-reflow phenomenon [2]. For the last few decades, treatment of no-reflow has been mostly pharmacotherapy-based, without much success. This case depicts why it is important to recognize coronary no-reflow early and to continue exploring treatment options.

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