

Multifocal Plaque Rupture and Diffuse Pan-Coronary Instability Manifesting as Simultaneous Two-Vessel Myocardial Infarction: Case Report

López Marroquín Brenda Citlalic*¹, Quintero Pánuco Jair Alexander²

^{1,2} Hospital General de Zona No.1 Dr. Luis Ernesto Miramontes Cárdenas, Instituto Mexicano del Seguro Social; Tepic, Nayarit.

ABSTRACT

ST-segment elevation myocardial infarction (STEMI) secondary to the acute, simultaneous thrombotic occlusion of two or more major epicardial coronary arteries constitutes a catastrophic and rare cardiovascular emergency (incidence 1.7%–4.8%) associated with a state of diffuse pan-coronary instability and cardiogenic shock. We present the case of a 57-year-old male patient with a medical history of hypertension and type 2 diabetes who presented to the emergency department of a secondary-care hospital with acute coronary syndrome. The initial electrocardiogram confirmed an acute anterior STEMI. Following failed pharmacological thrombolysis, the patient developed subsequent ST-segment elevation in the inferolateral leads, prompting urgent coronary angiography. Angiography demonstrated complex multivessel coronary artery disease. Urgent ad hoc percutaneous coronary intervention was performed on the culprit vessels via the deployment of two overlapping drug-eluting stents in the left anterior descending artery and one stent in the distal left circumflex artery, achieving angiographic success with final TIMI 2/3 flow and adequate myocardial perfusion while staging the right coronary artery lesion. In conclusion, simultaneous thrombotic occlusion of two major vessels is a highly lethal entity that underscores the phenomenon of multifocal plaque rupture. In the presence of electrical and hemodynamic instability, urgent primary percutaneous coronary intervention of the culprit vessels represents the most effective rescue therapeutic strategy, proving feasible and successful in secondary care centers equipped with cardiac catheterization laboratories.

KEYWORDS: ST-Elevation Myocardial Infarction; Multivessel Coronary Occlusion; Percutaneous Coronary Intervention; Coronary Thrombosis; Plaque Rupture, Atherosclerotic.

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I. INTRODUCTION

Acute coronary syndrome, specifically ST-segment elevation myocardial infarction (STEMI), is pathophysiologically driven in the vast majority of cases by the rupture or erosion of a single vulnerable atherothrombotic plaque, culminating in the occlusion of a single epicardial vessel (1, 2). However, the clinical presentation of STEMI secondary to the acute, simultaneous thrombotic occlusion of two or more major coronary arteries represents an extraordinary diagnostic and therapeutic challenge in contemporary cardiology (3). This clinical entity, characterized by the synchronous formation of high-grade thrombi, is a catastrophic scenario that doubles the myocardial area at risk and has historically been associated

with drastically elevated in-hospital and short-term mortality rates (4, 5).

From an epidemiological standpoint, simultaneous multivessel occlusion is an uncommon but critically underestimated phenomenon. Modern angiographic registries, cohort studies, and retrospective meta-analyses index its estimated incidence between 1.7% and 4.8% of all patients undergoing primary percutaneous coronary intervention (3, 6). Despite its low global prevalence, it accounts for up to 10% of cases presenting with extreme hemodynamic instability (4). Varied analyses based on international registries, such as the Korea Acute Myocardial Infarction Registry, demonstrate that this entity carries a significantly higher probability of cardiogenic shock at presentation, need for mechanical circulatory support, cardiac

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arrest, and lethal ventricular arrhythmias compared to single-vessel culprit infarctions (5, 6).

Clinical studies have identified a specific phenotypic and risk profile in patients who develop multifocal thrombotic occlusion. A strong association with the male sex has been documented, representing up to 85% of the analyzed cohorts (3, 4). Furthermore, traditional cardiovascular risk factors—including heavy active smoking, arterial hypertension, and severe dyslipidemia—serve as potent endothelial catalysts (6, 8). Notably, type 2 diabetes mellitus and heightened chronic inflammatory states demonstrate a critical correlation, as they promote a procoagulant microenvironment and diffuse endothelial dysfunction that predisposes to synchronous instability across the coronary vasculature (8, 9).

Pathophysiologically, this phenomenon transcends the conventional "single culprit lesion" model to introduce the concept of diffuse pan-coronary instability and multifocal plaque rupture (7). Evidence from intravascular ultrasound (IVUS) and optical coherence tomography confirms that STEMI patients frequently harbor multiple vulnerable ruptured or eroded plaques remote from the primary infarct site (7, 8). This process is triggered by an exacerbated acute systemic inflammatory response and a massive surge in adrenergic tone (9). The initial occlusion of a first vessel generates severe myocardial hypoxia, hypotension, and a critical reflex coronary spasm; this increased hemodynamic and shear stress mechanically induces the destabilization and rupture of a second adjacent vulnerable plaque (3, 9). The subsequent activation of the coagulation cascade and extreme platelet reactivity promote the simultaneous formation of occlusive thrombi (3, 7).

Accurate diagnosis constitutes a major challenge due to the complexity of electrocardiographic vector manifestations. Clinically, patients present with sudden dyspnea, high-intensity crushing chest pain, and hemodynamic collapse (4). Electrocardiographically, simultaneous involvement may manifest as ST-segment elevation in concurrent or non-contiguous leads, reflecting ischemia across multiple myocardial territories (4, 6). However, the injury vector of one wall can occasionally mask or attenuate the changes of the other, forcing the clinician to maintain a high index of suspicion in the presence of persistent shock or combined ECG findings (1, 6). Definitive confirmation remains strictly angiographic within the catheterization laboratory, demonstrating multiple filling defects compatible with high-grade thrombi (TIMI thrombus grade 3) and absent or critically diminished antegrade flow (TIMI 0/1) (3, 10).

Regarding therapeutic management, current clinical practice guidelines from the American College of Cardiology/American Heart Association (ACC/AHA) and the European Society of Cardiology (ESC) are definitive (1, 2). In the presence of multiple culprit lesions presenting with acute occlusion and hemodynamic instability or cardiogenic shock, immediate urgent complete revascularization via

primary PCI of all involved vessels during the index procedure is formally indicated (1, 10). Optimal treatment requires the restoration of effective epicardial flow and myocardial perfusion (final TIMI 3 flow and TMP grade 3), combined with aggressive antiplatelet and anticoagulant therapy (1, 10). The present case report describes the intraprocedural management and successful resolution of a patient presenting with an anterior and inferolateral STEMI driven by the simultaneous thrombotic occlusion of both the left anterior descending (LAD) and left circumflex (LCx) arteries, treated via primary multivessel percutaneous intervention at a secondary care hospital.

II. CASE PRESENTATION

A 57-year-old male patient with a medical history of hypertension and type 2 diabetes mellitus presented with a clinical syndrome classic for acute coronary syndrome. He sought medical attention at the emergency department of a secondary care hospital. An electrocardiogram performed at 17:49 hours confirmed an acute anterior ST-segment elevation myocardial infarction (Figure 1). Pharmacological thrombolysis was promptly administered. However, a subsequent ECG performed at 21:50 hours revealed new-onset ST-segment elevation in the inferolateral leads (Figure 2), prompting urgent referral for diagnostic coronary angiography.

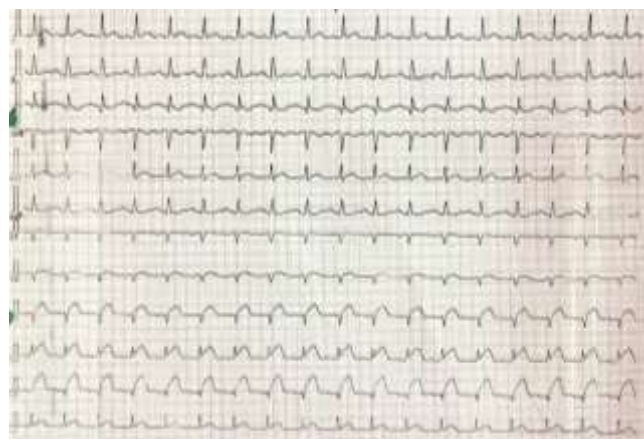


Figure 1. Anterior ST-segment elevation myocardial infarction

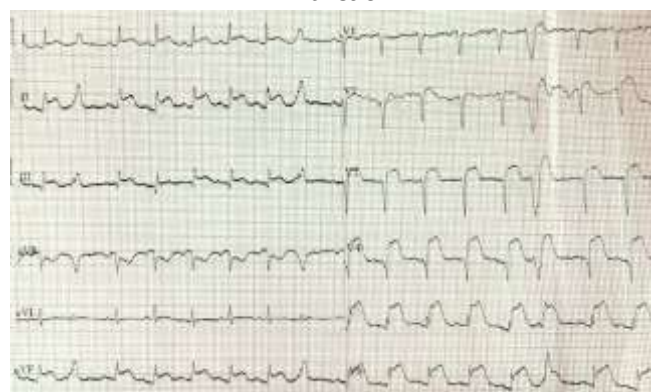


Figure 2. New-onset ST-segment elevation in the inferolateral leads

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Coronary angiography demonstrated multivessel coronary artery disease involving the left anterior descending (LAD), left circumflex (LCx), and right coronary artery (RCA) territories:

Left Anterior Descending Artery: Ruptured plaque at the proximal-to-mid segment junction associated with a TIMI thrombus grade 3, resulting in a total occlusion and distal TIMI 0 flow.

Left Circumflex Artery: Distal segment demonstrating a ruptured plaque with an associated TIMI thrombus grade 3 and a 70% stenosis.

Right Coronary Artery: Distal segment exhibiting an 80% stenosis proximal to the crux cordis.

Based on the clinical presentation, multimodal diagnostic findings, evolving electrocardiographic changes showing subepicardial injury in both the anterior and inferolateral walls, and the angiographic findings, the heart team decided to perform urgent ad hoc percutaneous coronary intervention targeting the LAD and LCx arteries.

For the LAD artery, a 3.5×12 mm Abluminus drug-eluting stent (DES) and a 3.5×16 mm Abluminus DES were successfully deployed overlapping from the mid-to-proximal segments, achieving a final TIMI flow grade 2 and a TIMI myocardial perfusion (TMP) grade 2 post-PCI (Figure 3). For the LCx artery, a 3.5×20 mm Abluminus DES was successfully placed in the distal segment, yielding a final TIMI 3 flow and a TMP grade 3 (Figure 4).

The significant lesion in the distal right coronary artery was left untreated at this stage, with plans for staged revascularization during a subsequent procedure; intervention during the index event was strictly restricted to the culprit vessels responsible for the acute coronary syndrome.

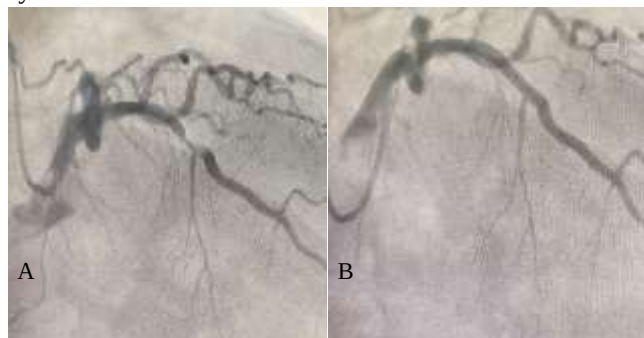


Figure 3. A. Proximal-to-mid left anterior descending artery occlusion with a TIMI thrombus grade 3. B. Post-PCI: final TIMI flow grade 2 and TMP grade 2.

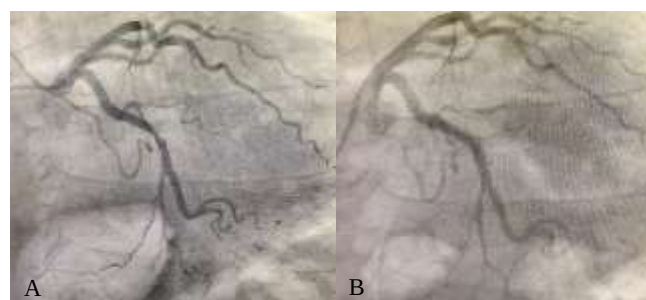


Figure 4. A. Distal segment of the left circumflex artery occlusion with a TIMI thrombus grade. B. Post-PCI: final TIMI flow grade 3 and TMP grade 3.

III. CONCLUSIONS

Acute simultaneous thrombotic occlusion of two major coronary arteries is an extraordinarily rare angiographic phenomenon, documented in fewer than 5% of catheterizations for acute myocardial infarction, making it one of the most critical and atypical scenarios in interventional cardiology. Finding synchronous thrombosis within two of the heart's primary vessels represents a true vascular catastrophe that doubles the myocardial area at imminent risk, triggering extreme electrical instability and fulminant cardiogenic shock. This case highlights that such a rare occurrence stems from a mechanism of multifocal plaque rupture and diffuse pan-coronary instability, directly challenging the classic single-culprit-lesion dogma. Despite the exceedingly high technical complexity required to simultaneously revascularize both occluded territories in a critically ill patient, immediate ad hoc percutaneous coronary intervention proved to be a highly successful rescue strategy. This report underscores that, through timely clinical suspicion and operator expertise, it is entirely feasible to successfully manage these exceptionally rare and complex scenarios within secondary care hospitals equipped with a cardiac catheterization laboratory, dramatically improving the patient's ultimate prognosis.

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