

The False Culprit: A Chronic Total Occlusion Mimicking the Infarct-Related Artery During Primary PCI

Oualid Kerrouani MD^{1,2}✉, Mohamed Sarsari MD^{1,2}, Jamila Zarzur PhD^{1,2} and Mohamed Cherti PhD^{1,2}

¹Mohammed V University, Rabat, Morocco

²Cardiology B Department, Ibn Sina University Hospital Center, Rabat, Morocco

Corresponding author: Oualid Kerrouani MD, Department of Cardiology B, Ibn Sina University Hospital Center, Rabat, Morocco. E-mail: Oualid.kerrouani12@gmail.com.

Received: 11 August 2026; **Revised:** 10 October 2026; **Accepted:** 15 October 2026; **Published:** 20 October 2026

Academic Editor: Dr. Neeraj Aggarwal

Abstract

Background: Accurate identification of the infarct-related artery may be challenging in patients with ST-segment elevation myocardial infarction, especially when multivessel coronary disease is present. This challenge is further compounded by the presence of chronic total occlusions (CTOs) or complex coronary anatomy, which may produce atypical or misleading ECG patterns.

Case Presentation: We report the case of a 50-year-old male who presented with simultaneous anterior and inferior ST-elevation myocardial infarction (STEMI). Urgent coronary angiography revealed a thrombotic stenosis of the right coronary artery with distal thromboembolization, along with a complete occlusion of the mid left anterior descending artery. The angiographic features, together with the inability to pass a work horse guidewire across the LAD lesion, strongly supported a chronic total occlusion rather than an acute thrombotic event. We propose that the acute compromise of the RCA, which served as the collateral donor vessel, critically reduced perfusion to the territory supplied by the chronically occluded LAD. This led to acute anterior wall ischemia and an ECG pattern resembling an acute LAD occlusion. This case shows how an acute occlusion of a collateral donor artery in the context of a chronic CTO can mask the true culprit lesion during primary PCI.

Conclusion: Careful integration of ECG findings, angiographic lesion characteristics, collateral circulation, and procedural observations—including guidewire behavior when intracoronary imaging is unavailable—can facilitate accurate culprit vessel identification and guide the optimal revascularization strategy.

Keywords: Culprit lesion; Chronic total occlusion; Percutaneous coronary intervention; Acute myocardial infarction; Coronary artery disease

Introduction

In 10-15% of patients with myocardial infarction, chronic total occlusion of a non-infarct-related artery occurs [1]. Its co-existence with an acute thrombosis during primary PCI creates a significant diagnostic dilemma. While coronary angiography is considered the gold standard to identify the site of the obstruction, it can still be difficult to distinguish between acute event and chronic occlusion.

Angiographic features such as tapered stumps, severe calcification, and well-developed collateral circulation are suggestive of chronic coronary disease. In contrast, acute thrombotic occlusions of coronary arteries are characterized by abrupt occlusion of the vessel and a intraluminal filling defect [2]. However, significant overlap between these features may complicate the interpretation of coronary angiography and the identification of the culprit vessel. Simultaneous thrombotic occlusion of two distinct coronary arteries is a recognized yet exceptionally rare phenomenon, and its pathophysiological mechanisms remain incompletely elucidated [3]. In our case, the underlying mechanism involves destabilization of the donor vessel, which subsequently precipitated acute ischemia in the collaterally supplied CTO territory ; a cascade that may obscure the primary culprit lesion.

Case Presentation

We report the case of a 50-year-old man presenting with acute chest pain that had been evolving over five hours. His medical history was notable for diabetes mellitus and active smoking, with no prior cardiovascular events. The patient initially presented to the emergency department of a peripheral hospital, where a 12-lead electrocardiogram (ECG) demonstrated atrial fibrillation with ST-segment elevation involving the anterior and inferior leads, consistent with an acute ST-elevation myocardial infarction (Figure 1 A). He received a loading dose of dual antiplatelet therapy (aspirin 300 mg and clopidogrel 300 mg) and was subsequently transferred to our institution for emergency coronary angiography and primary percutaneous coronary intervention (PCI). Upon admission, the patient was free of chest pain and hemodynamically stable. A repeat ECG showed spontaneous conversion to sinus rhythm, with partial resolution of ST-segment elevation. Frequent premature ventricular complexes were also noted (Figure 1 B).

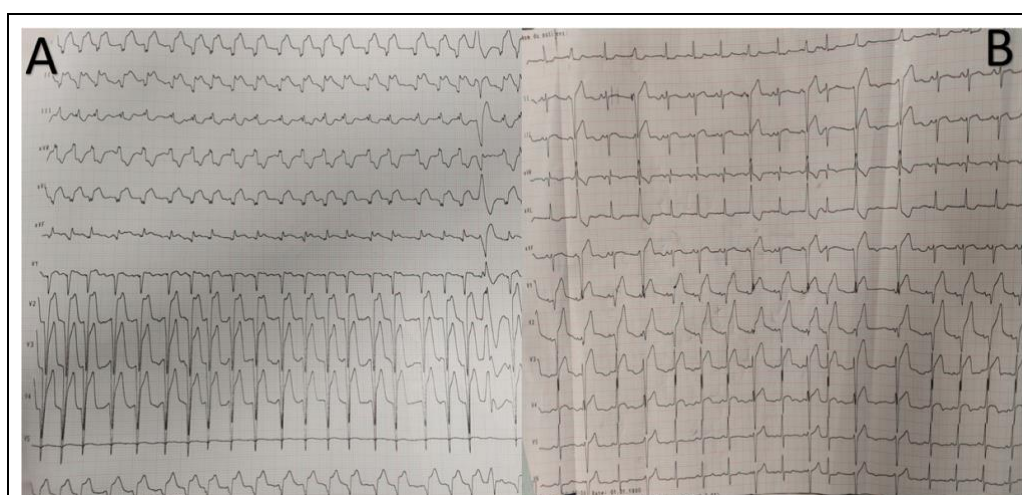


Figure 1: 12 leads ECG at admission. **(A):** Initial ECG at referring hospital demonstrating acute myocardial infarction with ST-segment elevation involving both the extensive anterior and inferior leads with atrial fibrillation. **(B):** 12 leads-ECG at admission shows sinus rhythm conversion with ST-segment resolution and ventricular ectopy.

Coronary angiography was performed via the right radial approach. The left coronary system revealed mid-left anterior descending artery occlusion with TIMI 0 antegrade flow (Figure 2 A). The right coronary artery demonstrated a significant lesion in the mid-segment, with occlusion of the distal segment (Figure 2 B). At this stage, identification of the culprit vessel remained challenging. While the ECG demonstrated ST-segment elevation in both anterior and inferior leads, and transthoracic echocardiography revealed inferior wall motion abnormalities, angiographic findings suggested a thrombotic-appearing lesion in the mid-RCA, whereas the LAD was occluded with no collateral circulation. The angiographic appearance of the LAD including diffuse atherosclerosis, heavy calcification, and a tapered, ambiguous termination of contrast was suggestive of a longstanding occlusion.

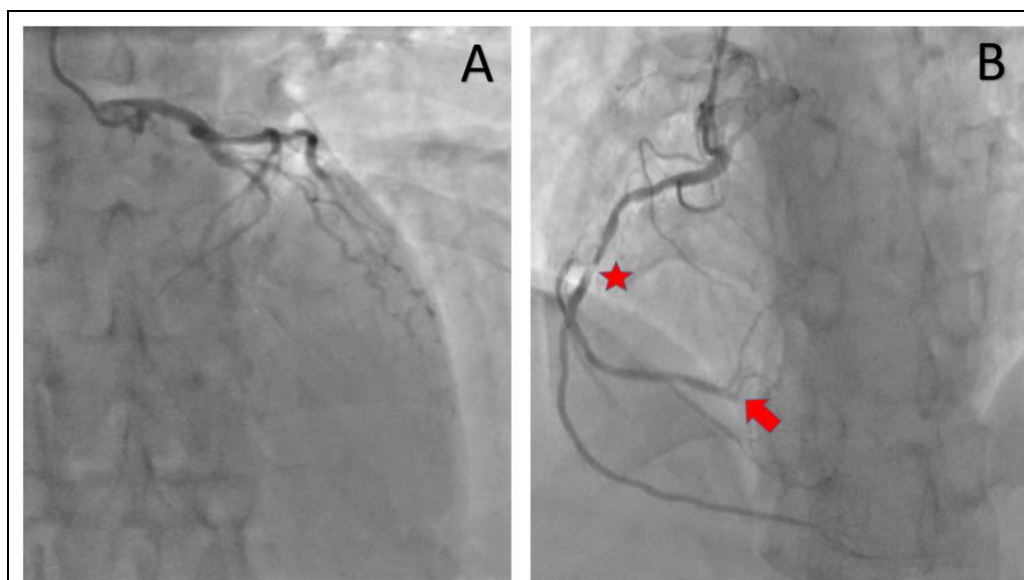


Figure 2: Coronary angiography of left and right arteries. **(A):** Coronary angiography showing total occlusion of the mid-left anterior descending artery (LAD) in the cranial 40° projection. **(B):** LAO Cranial view showing thrombotic-appearing stenosis (asterisk) of the mid right coronary artery with distal contrast cutoff (arrow).

The presence of a blunt, cup-shaped contrast termination in the distal segment raised suspicion for a recent occlusive event, although definitive differentiation between acute and chronic phases could not be established on angiography alone. Intracoronary imaging was unavailable. To resolve this diagnostic ambiguity, multiple attempts to cross the LAD occlusion using a workhorse guidewire were performed, all of which proved unsuccessful, strongly suggesting the presence of a chronic total occlusion rather than an acute thrombotic event (Figure 3 A). In contrast, the RCA was easily crossed and successfully stented within the same procedural setting (Figure 3 B). A decision was made to defer angioplasty of the LAD to a staged procedure.

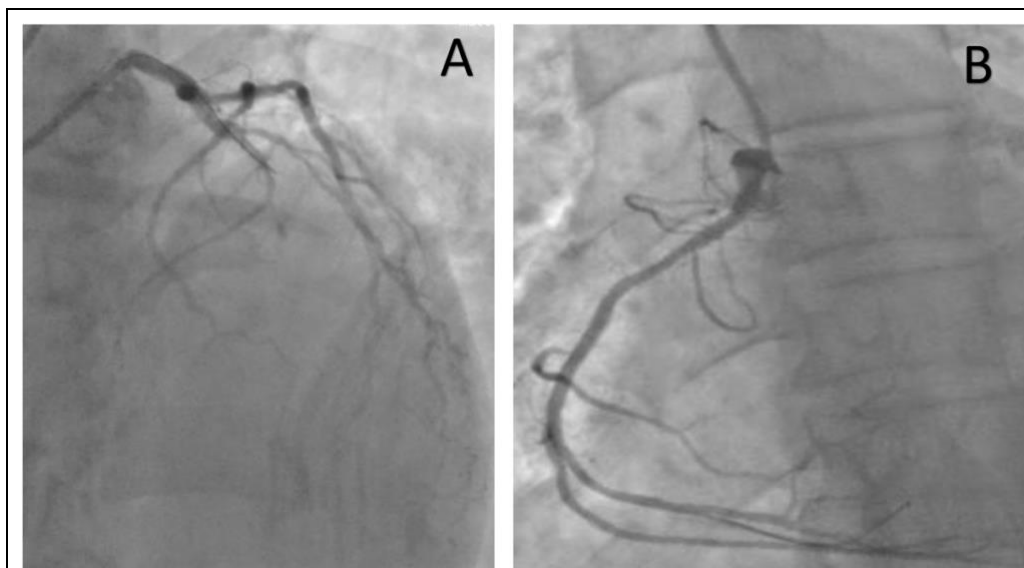


Figure 3: Failed recanalization of the occluded LAD and successful PCI of the RCA. **(A):** Attempted crossing of the left anterior descending artery (LAD) occlusion using work horse guidewires, without success. **(B):** Coronary angiography demonstrating successful PCI of the RCA after predilation and DES implantation, with easy crossing of the RCA and restored distal flow, consistent with an acute lesion.

Discussion

When two vessel occlusions are identified on coronary angiography performed during the acute phase of myocardial infarction, typically only one represents an acute event, while the other constitutes a chronic total occlusion [4]. The coexistence of a CTO in a non-infarct-related artery (non-IRA) at the time of STEMI presentation is a relatively uncommon yet clinically significant finding, reported in approximately 10-15% of patients undergoing primary PCI [1]. The pathophysiology of simultaneous ST-segment elevation in multiple coronary territories is complex and remains incompletely understood. One proposed mechanism is the concurrent rupture or erosion of atherosclerotic plaques in different coronary arteries, potentially promoted by a systemic inflammatory or prothrombotic state. Other possible mechanisms include multivessel coronary vasospasm and microvascular dysfunction, although these remain less well documented. When the IRA serves as the donor vessel supplying collateral blood flow to the CTO territory, its acute occlusion may simultaneously jeopardize both the IRA-dependent myocardium and the collaterally perfused CTO area, a phenomenon referred to as the "double-jeopardy" effect [5]. Accurate identification of the IRA in acute coronary syndromes (ACS) is essential for appropriate management and carries important diagnostic and prognostic implications. Although the 12-lead ECG remains the first-line tool for rapid triage and guidance of reperfusion therapy, its interpretation may be misleading particularly in the setting of complex multivessel coronary artery disease. From a hemodynamic perspective, the presence of a concomitant non-IRA CTO has been associated with a higher incidence of cardiogenic shock, reduced left ventricular ejection fraction, and increased short- and long-term mortality compared to STEMI patients with single-vessel disease [6]. In contrast with the typically severe clinical presentations described in the literature, our patient remained hemodynamically stable despite simultaneous involvement of two major coronary vessels. This relative stability may be explained by several complementary mechanisms. First, chronic occlusion of the LAD may have induced ischemic preconditioning of the dependent myocardium, a phenomenon that is particularly frequent in patients with diabetes mellitus. Second, spontaneous reperfusion of the RCA potentially facilitated by early antiplatelet therapy is suggested by the rapid resolution of ST-segment elevation observed upon arrival in the catheterization laboratory. While coronary angiography remains the gold standard for coronary artery assessment, differentiating an acute thrombotic occlusion from a CTO on angiographic grounds alone can be challenging. Features suggestive of chronicity include a tapered proximal cap, severe calcification, and well-developed collateral circulation, whereas intraluminal thrombus or abrupt vessel cut off is more consistent with an acute coronary event [7]. Definitive lesion characterization may remain difficult in the absence of intracoronary imaging. In our patient, ST-segment elevation in both inferior and anterior leads suggested ischemic involvement of two myocardial territories. The heterogeneous mid-RCA lesion and associated distal occlusion, potentially due to distal embolization, suggested an acute coronary event. This interpretation was supported by easy guidewire passage through the RCA lesion, followed by prompt restoration of distal flow. In contrast, repeated attempts to cross the LAD occlusion with conventional guidewires were unsuccessful, supporting the diagnosis of a chronic total occlusion (CTO). We hypothesize that the acute occlusion of the donor RCA may have compromised perfusion of the LAD territory, considered the recipient network, precipitating ischemic destabilization and explaining the anterior ECG changes. This case highlights the importance of integrating ECG findings, angiographic features, and procedural observations to identify the acute culprit lesion and guide revascularization during primary PCI.

Conclusion

This case underscores the diagnostic challenge posed by the coexistence of an acute thrombotic occlusion and a chronic total occlusion during primary PCI. When ECG findings and angiographic appearance prove insufficient to identify the true culprit lesion, a stepwise interventional approach — using guidewire crossing attempts as a pragmatic diagnostic tool — can be decisive. Accurate culprit identification remains essential to ensure appropriate revascularization and optimize clinical outcomes in this high-risk subset of STEMI patients.

Availability of Data and Materials

Data sharing is not applicable to this article as no data sets were generated or analysed during the current study.

Acknowledgements

The authors have no acknowledgments to declare.

Conflict of Interest

The Author (s) declare(s) that there is no conflict of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

Consent Statement

Written informed consent was obtained from the patient for the publication of this case report, including the accompanying images.

Authors Contributions

Kerrouani Oualid: Study concept, Data collection, Data analysis, Writing the paper.

Sarsari Mohamed: Data collection, Data analysis.

Zarzur Jamila: Supervision and data validation.

Cherti Mohamed: Supervision and data validation.

REFERENCES

1. Henriques JPS, Hoebbers LP, Råmunddal T, et al. Percutaneous Intervention for Concurrent Chronic Total Occlusions in Patients with STEMI. *Journal of the American College of Cardiology*. 2016; 68: 1622-1632. <https://doi.org/10.1016/j.jacc.2016.07.744>.
2. Dash D. Coronary Chronic Total Occlusion Intervention: A Pathophysiological Perspective. *Indian Heart Journal*. 2018; 70: 548-555. <https://doi.org/10.1016/j.ihj.2018.01.021>.
3. Pollak PM, Parikh SV, Kizilgul M, et al. Multiple Culprit Arteries in Patients with ST Segment Elevation Myocardial Infarction Referred for Primary Percutaneous Coronary Intervention. *The American Journal of Cardiology*. 2009; 104: 619-623. <https://doi.org/10.1016/j.amjcard.2009.04.053>.
4. Maagh P, Wickenbrock I, Schrage MO, et al. Acute Simultaneous Proximal Occlusion of Two Major Coronary Arteries in Acute Myocardial Infarction: Successful Treatment with Percutaneous Coronary Intervention. *J Interv Cardiol*. 2008; 21: 483-492. <https://doi.org/10.1111/j.1540-8183.2008.00394.x>.
5. Scholz M, Meyer T, Maier LS, et al. Infarct-Related Artery as a Donor of Collaterals in ST-Segment–Elevation Myocardial Infarction with Concomitant Chronic Total Occlusion: Challenge of the Double-Jeopardy Thesis. *JAHA*. 2023; 12: e028115. <https://doi.org/10.1161/JAHA.122.028115>.
6. Watanabe H, Morimoto T, Shiomi H, et al. Chronic Total Occlusion in a Non-infarct-related Artery is Closely Associated with Increased Five-year Mortality in Patients with ST-segment Elevation Acute Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention (From the CREDO-Kyoto AMI registry). *Euro Intervention*. 2017; 12: e1874-1882. <https://doi.org/10.4244/EIJ-D-15-00421>.
7. Galassi AR, Werner GS, Boukhris M, et al. Percutaneous Recanalisation of Chronic Total Occlusions: 2019 Consensus Document From the EuroCTO Club. *EuroInterv*. 2019; 15: 198-208. <https://doi.org/10.4244/EIJ-D-18-00826>.