### Case Report

## Double Culprit in A Patient who Presented in Coma: A Perlexing but Rewarding Case.

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#### Abstract

Acute thrombotic occlusion of more than one coronary artery causing STEMI is a rather rare finding in coronary angiography during primary angioplasty in myocardial infarction (PAMI). We present a case of a 72 year old male, with no known comorbidities who presented with acute anterior wall myocardial infarction (AWMI) in comatose condition. Coronary angiography showed thrombotic occlusion in Left anterior descending (LAD) and circumflex (LCx) arteries. Clinical condition improved after percutaneous transluminal coronary angioplasty (PTCA) of both arteries. Given this unique presentation, the best management is not standardized. However, quick decision making and proper timely revascularisation helps patients to achieve a full and excellent recovery post revascularisation. We further emphasise need for guideline directed therapy in patients presenting with such multiple culprit vessel lesions in STEMI.

KEY WORDS: Double culprit vessels, Acute myocardial infarction, coma.

#### Introduction

In spite of availability of therapeutic and preventative interventions, acute myocardial infarction (MI) with or without ST-segment elevation is one of the global leading causes of mortality. ST-elevation myocardial infarction (STEMI) causes epicardial coronary artery occlusion leading to localized infarction and myocardial necrosis due to atherothrombosis.1 Evidence has shown that STEMI typically occurs with a single culprit vessel; however, multi-vessel thrombosis is a rare condition associated with poor outcomes and a high fatality rate.2 Only 2.5% of STEMI patients encountered during percutaneous coronary intervention (PCI) are noted to have multiple acute coronary artery thromboses.3 Prevalence of dual culprit lesions in left coronaries during an ACS is a rarity reported in literature.

In prior studies, the most frequent angiographic outcome involved simultaneous thrombosis in both the left anterior descending (LAD) and right coronary arteries (RCA), treated with PCI and an intra-aortic balloon pump.2 Multi vessel coronary thrombosis can arise secondary to coronary vasospasm.4 Although most often cause remain unidentifiable. Currently, no guideline define the best management of STEMI with multiple culprit lesions.

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In STEMI patients with hyperacute MI, and rapidly worsening hemodynamics, urgent PTCA to the culprit artery becomes necessary after an angiogram to save the patient's life. Similar patients presenting with shock, LVF, arrhythmias and sometimes with coma/ syncope may have multiple responsible culprit arteries. Detection of this possibility of multivessel thrombosis is important. We present a rare case of acute MI due to multiple thrombotic occlusions highlighting the presentation, management, and outcome of the patient.

#### **Case Report**

72 Year old male with no comorbidities, was brought to ER in a state of unresponsiveness since 1 hour. Initial evaluation revealed normal vitals signs, GCS: E1V1M1, GRBS of 180mg/dL. ABG was also normal. He was intubated for airway protection. ECG changes (FIG1) were present as shown in the figure. ECHO done showed apical hypokinesia with LVEF 20%. He underwent a CT brain which revealed no acute infarct/ bleed. Troponin was elevated. He was taken up for primary PCI which revealed LAD and LCx having 100% thrombotic occlusions (FIG 2) which were successfully treated by 1X DES in LAD and 1xDES in LCx achieving TIMI 3 flow (FIG3) . Patient was extubated the following day as he regained consciousness with no end organ damage or shock. Timely intervention and inclusion of two arteries as culprit vessels, with such peculiar presentation as well as the ECG changes makes it a case of rarity. It also signifies the importance of treating the condition on an emergent basis to aid in complete recovery.





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# FIG 2 : CORONARY ANGIOGRAM SHOWIN LAD AND LCX 100% THROMBOTTIC OCCLUSION

#### FIG 3: POST PCI



#### Discussion

The primary mechanism of STEMI is the rupture of the susceptible plaque with thrombus development. STEMI is often triggered by a single artery thrombosis and rarely caused by more than one culprit artery. In 50% of STEMI patients, this mechanism was discovered during an autopsy examination.6 However, plaque rupture with thrombus formation is seen only in 1.7 to 4.8% cases in STEMI patients undergoing PAMI.3 A possible explanation for this disparity is that majority of patients developing STEMI due to multiple culprit arteries die because of massive MI and related complications before reaching the hospital. Above incidence is not observed in patients undergoing PAMI. This may partly be due to clinicians' unawareness leading to missing culprit lesions in other coronaries or tiny plaque ruptures causing inconspicuous angiographic findings. Kanei et al determined heightened inflammatory response with catecholamine surge due to acute blockage in one coronary artery leading to plaque rupture in other coronaries.7 Other possible mechanisms are coronary spasm, hypercoagulable state, coronary embolism or hemodynamic instability.6 Similar to the various factors explored by Mahmoud et al., our case contributes to the STEMI literature by presenting a scenario involving multiple coronary thromboses.2 Our case had multiple risk factors such as DM, hypertension, and smoking. Moreover, in our case, our patient had LVF associated with CHB which further accounted for simultaneous coronary thrombosis. Overall, acute MI cases are discovered to have multivessel obstructive coronary artery disease, and recommendations have addressed the benefits of culprit lesion alone PCI versus total revascularization of non infarct related arterial lesions.5 A complete revascularization guided by fractional flow reserve significantly lowered the risk of future events in STEMI patients with multivessel disease when compared to initial PCI without invasive procedures. Due to no differences in all-cause mortality, patients could safely have all lesions treated during the index hospitalization to prevent further revascularization.8 Multiple culprit lesions are not addressed in the 2015 ACC guidelines on primary PCI for STEMI; instead, it states that PCI of non-culprit lesions is reasonable in the STEMI. Additionally, hemodynamically stable multivessel diseases can be managed during primary PCI or as a planned stage procedure.5

In conclusion, our case study highlights the importance of complete revascularisation in cases with multiple vessel athero-thrombosis in order to provide optimal clinical outcomes to our patients with STEMI.

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