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Case Report

A tale of two culprits: a case report

Ajinkya V. Mahorkar*, Virag U. Mahorkar, Uday K. Mahorkar, Avanti A. Vidhale, Suresh J. Sarwale

Department of Cardiology, Avanti Institute of Cardiology, Nagpur, Maharashtra, India

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***Correspondence:**
Dr. Ajinkya V. Mahorkar,
E-mail: ajinkyamahorkar1@gmail.com

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ABSTRACT

Acute thrombotic closure of more than one coronary artery causing STEMI is an infrequent finding in coronary angiography during primary angioplasty in myocardial infarction (PAMI). We presented a case of 37 years old male with acute inferior wall myocardial infarction (IWMI) with complete heart block (CHB) with left ventricular failure (LVF). Coronary angiography showed thrombotic occlusion in right coronary artery (RCA) and circumflex arteries. Clinical condition improved only after percutaneous transluminal coronary angioplasty (PTCA) of both arteries. Given this unique presentation, the best management is not standardized. Further studies are needed to better predict outcomes and guide future management.

Keywords: ST- elevation myocardial infarction, Acute coronary syndrome, Multiple culprit lesions

INTRODUCTION

Despite the 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 blockages advancing to localized infarct, necrosis particularly 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 with percutaneous coronary intervention (PCI) developed multiple acute coronary artery thromboses.[3]

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] Though many times causes remain unidentifiable. Currently, no guidelines define the best management of STEMI with multiple culprit lesions. The American College of Cardiology (ACC) and the European Society of Cardiology (ESC) guidelines addresses culprit lesion management with only PCI versus complete revascularization of non-infarct related lesions.[5]

In STEMI patients with hyperacute MI, and rapidly worsening hemodynamics, urgent PTCA to the culprit artery becomes necessary before an angiogram to save the patient's life. Similar patients with shock, LVF, and arrhythmias 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

A 37 years old male was admitted to the hospital with complaints of typical severe chest pain, profuse sweating and restlessness. He had type 2 diabetes mellitus, hypertension, smoking, and obesity as risk factors. On examination, his pulse was 35/min regular, BP was 90/60 mmHg, cold extremities, SPO_2 was 90% and had bilateral basal crepitations. Electrocardiogram showed narrow

QRS, complete heart block (CHB) with heart rate 35/min, and changes suggestive of hyperacute infero-posterior wall MI (Figure 1). The 2D echocardiograph showed severe hypokinesia of non-LAD territory with an ejection fraction 30% and no evidence of any mechanical complications. The RCA was the strongly suspected culprit vessel. He was immediately transferred to Cath lab, and suspected RCA culprit artery was cannulated with JR 3.5 guiding catheter through the radial route. The angiogram showed 100% thrombotic occlusion of distal RCA. RCA angioplasty was performed using drug eluting stent 2.25×22 mm distally and another 2.5×26 mm in mid-RCA with excellent results (Figure 1 and 2). However, the chest pain, ST elevation, CHB, and hypotension persisted. The angiogram also showed normal left main, mild irregularities in proximal LAD, and 100% thrombotic occlusion of the left circumflex artery (LCX). The LCX turned out to be a non-dominant but large vessel. PTCA to LCX was performed. Post PTCA to LCX, CHB, and chest pain disappeared and BP started improving (Figure 3). The patient was shifted to ICCU. He then slowly improved over the next 2 days and was discharged on day 5 in stable condition.

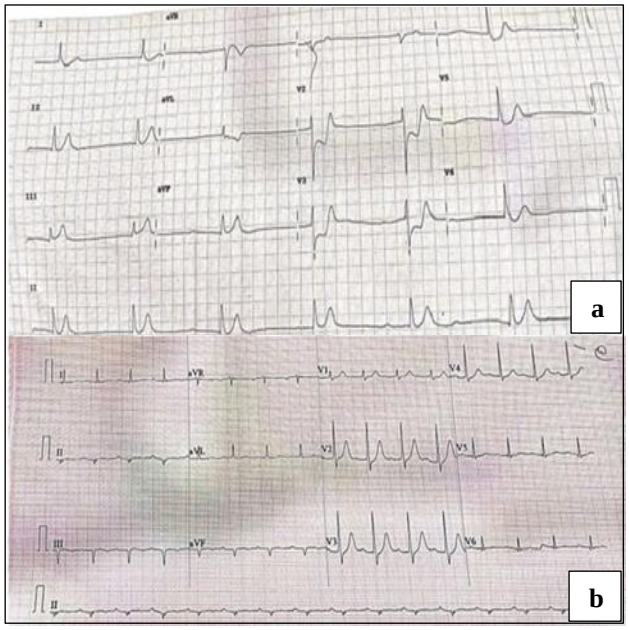

Figure 1: ECG (a) before PCI- ST segment elevation in III, avF and ST depression with upright T waves in V2, V3 with complete atrio- ventricular dissociation; and (b) after PCI- Q waves in III, avF and sinus rhythm.

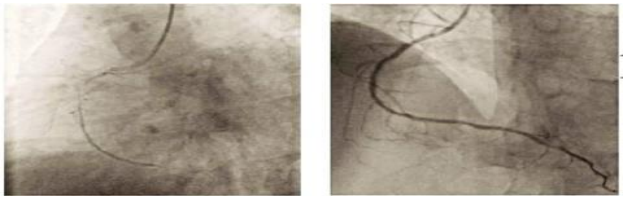

Figure 2: RCA before and after stenting with two DES in mid and distal RCA.

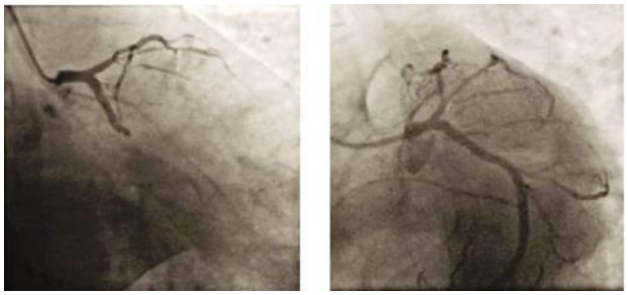

Figure 3: LCX before and after stenting with one DES in mid proximal part.

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, clinical experience in STEMI patients undergoing PAMI plaque rupture with thrombus formation is seen only in 1.7 to 4.8% cases.[3] A possible explanation for this disparity is the 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 clinician's unawareness leading to missing culprit lesions in other coronaries or tiny plaque ruptures causing inconspicuous angiographic findings.

Kanei et al determines 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, they merely state that PCI of non-culprit lesions is reasonable in the STEMI. Additionally, hemodynamically stable multivessel diseases should be

managed during primary PCI or as a planned stage procedure.[5]

In our case, the exact cause remained undetected. Main clue for other culprit artery was no improvement after PTCA to RCA and due to acute block in left circumflex. Non- resolution of ST segment and persistence of CHB, pointed towards the possibility of 2nd culprit lesion. After the thrombectomy, coronary stents were effectively implanted to treat both RCA and LCX arteries.[9] Currently, the ECG-based deep learning are among novel diagnostics can both identify STEMI and forecast culprit vascular occlusion.[10]

CONCLUSION

An uncommon form of STEMI with multi-vessel coronary thrombosis is characterized by high incidence of cardiac complications. Our case describes STEMI with two culprit vessels remarkably improving clinical outcomes after PAMI procedure to both vessels. Hence, we strongly recommend performing PTCA on both culprit vessels in the same setting, particularly if the initial culprit vessel has not improved following the initial PCI procedure

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