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CASE SERIES

Multiple Plague Rupture in Setting of Acute Myocardial Infarction

Muhammad Hashim, Muhammad Naeem Mengal, Naveedullah Khan, Syed Nadeem Hassan Rizvi & Tahir Saghir
National Institute of Cardiovascular Diseases (NICVD), Karachi-Pakistan.

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Corresponding Author Email:
drhashim25@gmail.com

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Abstract

Background: ST-segment elevation myocardial infarction (STEMI) due to the simultaneous formation of thrombi in multiple arteries, known as multiple culprits, is an infrequent angiographic finding in clinical practice. Current guidelines for managing STEMI patients with multiple culprits are not very clear. However, various studies reported the beneficence of complete revascularization in such patients.
Case Presentation: We presented a series of five cases presented with STEMI with multiple culprits who underwent complete revascularization.
Management: The successful intervention of multiple culprits with closed contrast monitoring leads to a good outcome and a short hospital stay. Although, the optimal management strategy for the simultaneous multiple culprit lesions has yet to be established.
Conclusion: Acute myocardial infarction caused by simultaneous multi-vessel coronary occlusion is rarely reported. The successful intervention of multiple culprits with closed monitoring of the contrast leads to a good outcome and short hospital stay. Although, the optimal management strategy for the simultaneous multiple culprit lesions has yet to be established.

Keywords

ST-segment elevation myocardial infarction, STEMI, multiple plague rupture, management

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Introduction

ST-segment elevation myocardial infarction (STEMI) due to the simultaneous formation of thrombi in multiple arteries, known as multiple culprits, is a rare angiographic finding in clinical practice[1-5]. Angiographic evidence of multiple culprits in a patient undergoing primary percutaneous coronary intervention (PCI) was reported in 2.5% [3]. The occurrence of multiple acutely thrombosed coronary arteries is associated with poor prognosis and often associated with fatal outcomes[1,2,5]. Pathophysiology of multiple occlusions is not well established; various mechanisms behind the vulnerable plaques rupture like catecholamine action, inflammation, or increased sympathetic tone are likely to be involved. Current guidelines for managing STEMI patients with multiple culprits are not very clear[6]. However, various studies reported the beneficence of complete revascularization in such patients[1,4,5]. We presented a series of five cases presented with STEMI with multiple culprits who underwent complete revascularization.

Cases Summary

Case 1

50-year-old female haying hypertension for two years on medical treatment with no past medical history of diabetes, ischemic heart disease, connective tissue disorder, or hematological disorder, presented to the emergency department of National Institute of Cardiovascular Disease (NICVD), Karachi, with sudden onset of severe chest heaviness of approximately one-hour duration. Physical examination showed a pulse of 105 beats/min, blood pressure of 90/60 mmHg, respiratory rate of 24 cycles/min, and the patient was in painful distress and diaphonic. Pulse Oximeter showed an oxygen saturation of 94% on two liters of oxygen. Cardiovascular examination was normal. There was bilateral basal crept on lung auscultation. Initial 12-lead ECG findings showed sinus tachycardia with ST elevation in the lead I, aVL, V5, and V6, and there were diffused ST depressions in all other leads (Figure 1).

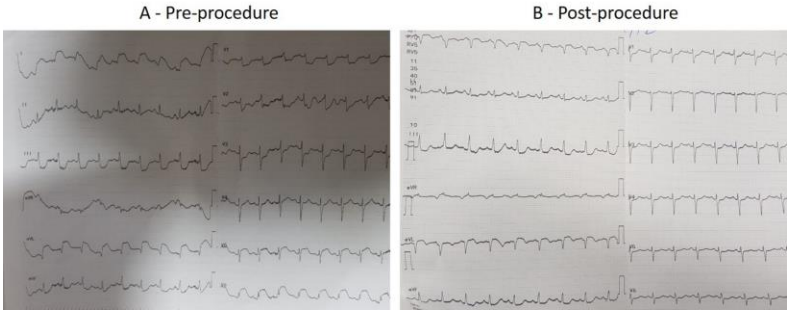

Figure 1: A) ECG at the time of presentation. B) ECG after PCI for Case 1

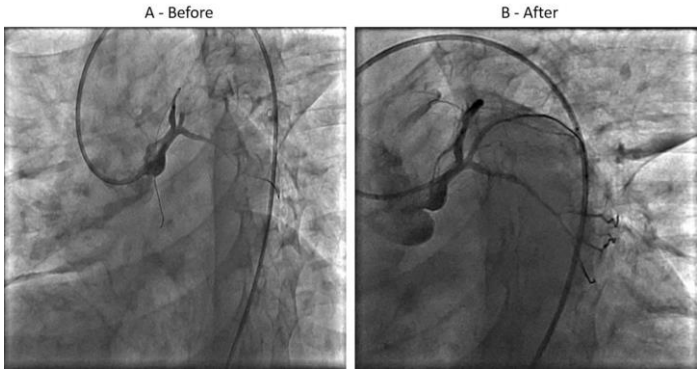

Figure 2: A) Angiogram of the left system showing total occlusion of LAD, Remus intermedius, and LCX (LAO caudal view). B) Post PCI angiogram of the left system with a resolution of obstruction in LAD, LCX, and Remus intermedius

Her initial troponin level was 0.041 ng/ mL. Chest x-ray showed bilateral basal infiltrate. An assessment of high literal ST-segment elevation myocardial infarction was made, and she was preloaded with antiplatelet and Heparin in the emergency room (ER). The patient underwent emergent cardiac catheterization after informed written consent, which showed total thrombotic occlusion of the left anterior descending artery (LAD), proximal left circumflex artery, and Remus intermedius (Figure 2) and evidence of thrombus in the distal right coronary artery (RCA) with TIMI flow III (Figure 3). It was decided to deal with the occlusion of LAD first, and after thrombectomy, successful PCI with drug-eluting stent (DES) (3.0x15) was performed, and TIMI III flow was achieved (Figure 4). After that, thrombectomy was performed in LCX, and TIMI III flow was achieved due to the small vessel size stent was not deployed in LCX (Figure 5). After dealing with LCX and achieving TIMI III flow, the pain of the patient was persistent, so it was decided to perform thrombectomy of Remus intermedius, and successful PCI was done with DEC (2.75x14), and TIMI III flow was achieved (Figure 6).

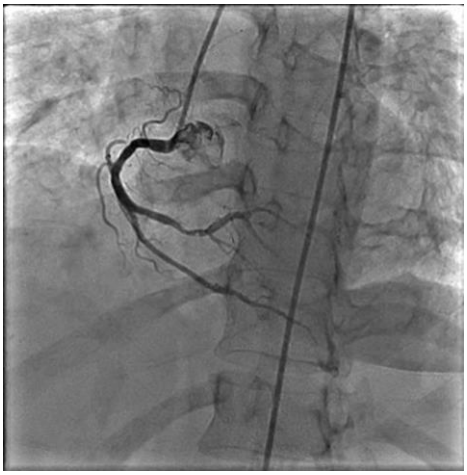

Figure 3: Coronary angiogram of right coronary artery showing thrombus in distal RCA with TIMI III flow.

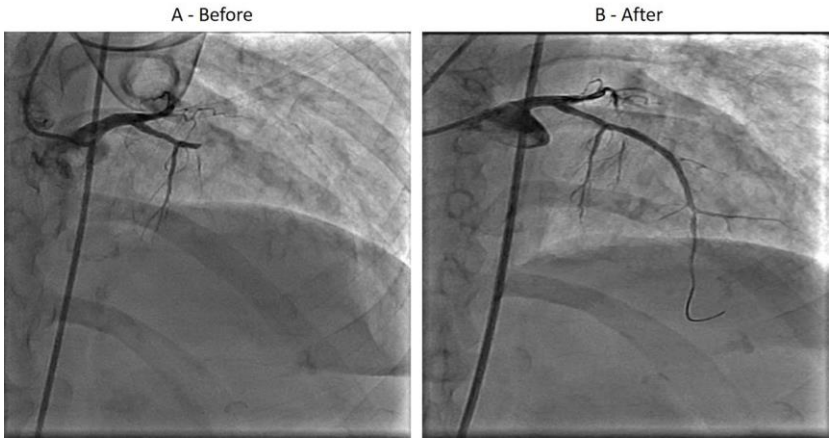

Figure 4: A) Preprocedure coronary angiogram showing total (100%) occlusion of LAD and LCX (AP cranial view). B) Coronary angiogram after PCI of LAD

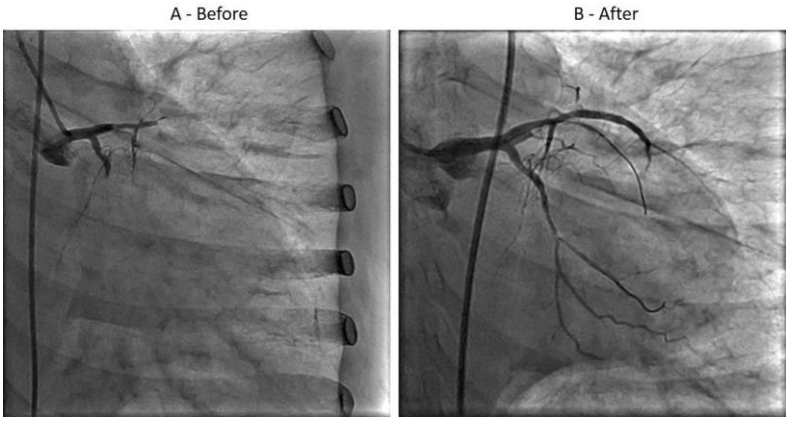

Figure 5: A) Angiogram of the left system showing 100% occlusion of LCX (RAO caudal view). B) Post PCI angiogram of the left system showing resolution of occlusion with TIMI III flow

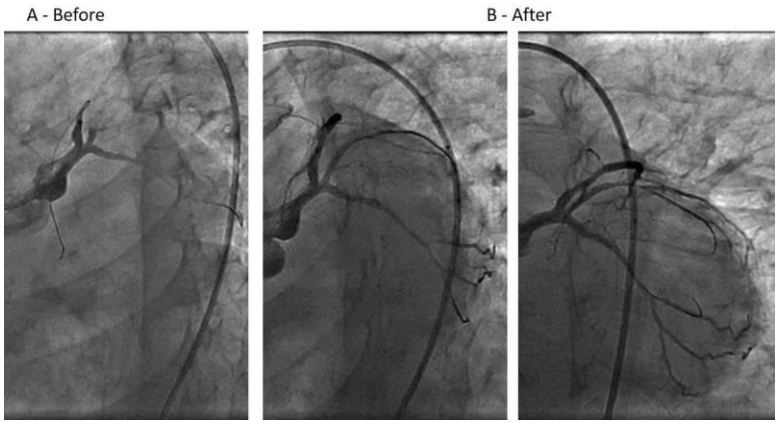

Figure 6: Final shot for case 1

The pain of the patient was settled down, and an on-table ECG was performed, which showed the settlement of ST Elevation. The patient shifted to the coronary care unit (CCU), labs were done at 24 and 48 hours, which were normal, and the patient was discharged after 72 hours of the procedure, and stage PCI of RCA was planned.

Case 2:

A 60-year-old male known case of hypertension, on medical treatment for three years, and current smoker with no past medical history of ischemic heart disease, and the connective tissue or hematologic disease presented with sudden onset of chest pain for two hours of duration. On physical examination, he was hemodynamically stable, with an oxygen saturation of 95% on room air. Cardiovascular examination was normal, and the chest was clear bilaterally. ECG shows sinus tachycardia with ST elevation in the lead II, III aVF, and V3-V6 while ST depression in the lead I and aVL (Figure 7).

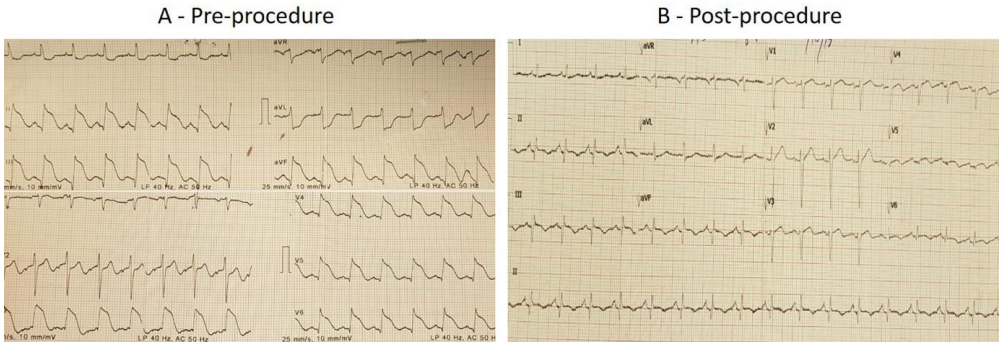

Figure 7: A) ECG at the time of presentation. B) ECG after PCI for Case 2

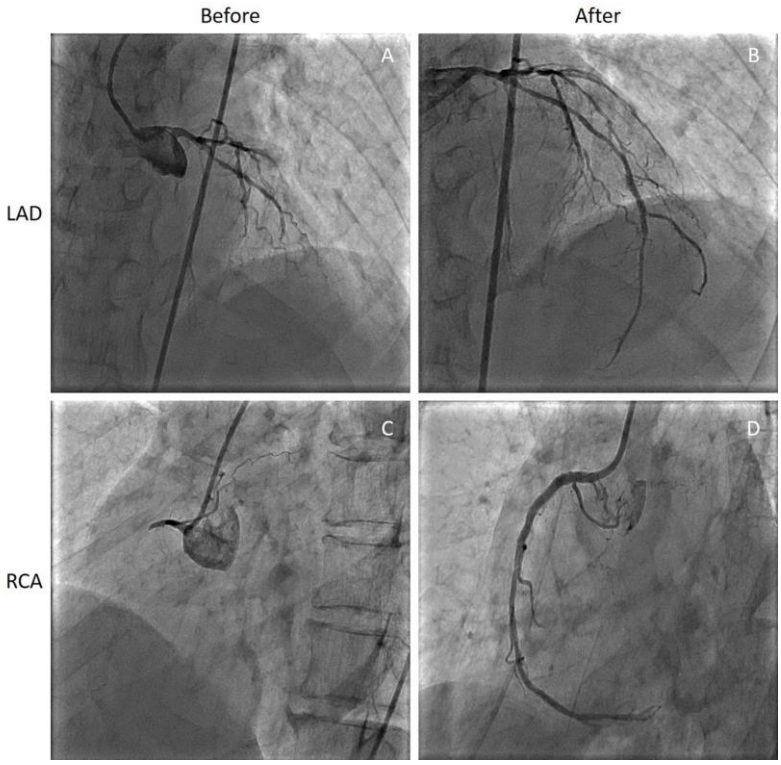

Figure 8: A) Pre-procedure coronary angiogram showing total (100%) occlusion of LAD. B) Coronary angiogram after PCI of LAD. C) Pre-procedure coronary angiogram showing 100% occlusion of RCA. D) Coronary angiogram after PCI of RCA

Initial troponin was within normal range, and assessment of anterio-inferior wall MI was made; he was preloaded with antiplatelet and Heparin in ER, and the patient underwent cardiac catheterization after written informed consent, which showed 100% thrombotic occlusion of LAD and RCA with moderate to severe LV dysfunction with EF of 30%. We decided to deal with RCA; first, PCI to RCA was done with DES (3.0x30), and after that, PCI to LAD was done with DES (2.75x18). TIMI III flow was achieved in both vessels. And the patient was discharged after 48 hours of the procedure (Figure 8).

Case 3:

A 52-year-old male known case of hypertension, on medical treatment for six years, with no past medical history of ischemic heart disease, and the connective tissue or hematologic disease presented with sudden onset of chest pain for six hours of duration. On physical examination, he was hemodynamically stable with oxygen saturation of 93% on room air. Cardiovascular examination was normal, and the chest was clear bilaterally. ECG shows sinus rhythm with a rate of 80 bpm, with ST elevation in lead V1–V4, and II, III, and aVF while ST depression in the lead I and aVL (Figure 9).

Initial troponin was 18 ng/dL, and initial assessment of anterio-inferior wall MI was made; was preloaded with antiplatelet and Heparin in ER. The patient underwent cardiac catheterization after written informed consent. Patient showed 95-99% thrombotic occlusion of LAD and Remus intermedius (Figure 10), non-dominant small RCA with server diseases in mid-segment (Figure 11), with mild to moderate LV dysfunction with EF of 40%. We decided to deal with LAD; first, PCI to LAD was done with DES (3.5x34), and after that, PCI to Remus intermedius was done with DES (3.0x15). TIMI III flow was achieved in both vessels. And the patient was discharged after 48 hours of the procedure (Figure 10).

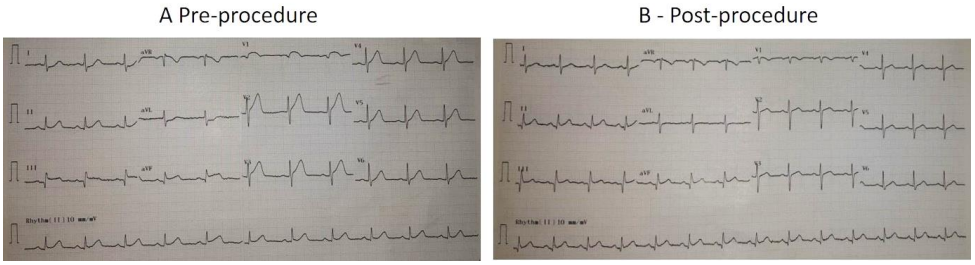

Figure 9: A) ECG at the time of presentation. B) ECG after PCI for case 3

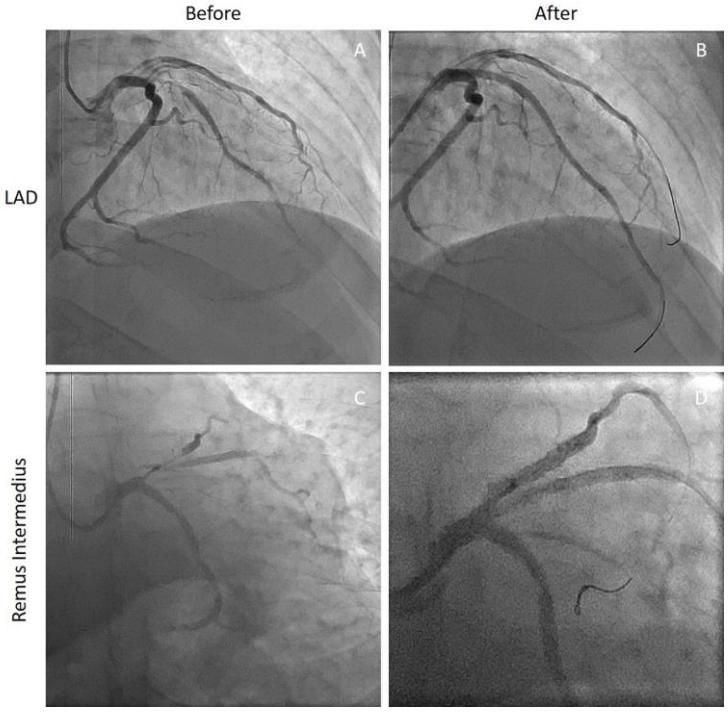

Figure 10: A) Pre-procedure coronary angiogram showing 95-99% occlusion of LAD. B) Coronary angiogram after PCI of LAD. C) Pre-procedure coronary angiogram showing 95-99% occlusion of Remus intermedius. D) Coronary angiogram after PCI of Remus intermedius

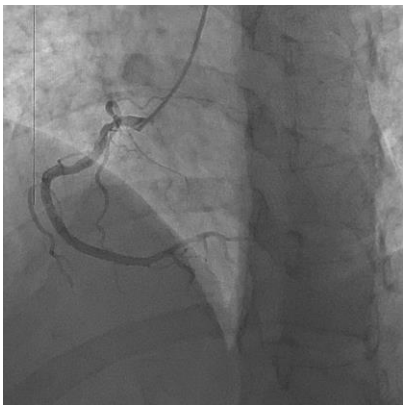

Figure 11: Coronary angiogram of the right coronary artery

Case 4:

A 47-year-old male current smoker with no past medical history of ischemic heart disease. The connective tissue or hematologic disease presented with sudden onset of chest pain for four hours of duration. On physical examination, he was hemodynamically stable, with an oxygen saturation of 96% on room air. Cardiovascular examination was normal, and Chest x-ray showed bilateral basal crepts. ECG showed sinus rhythm with the rate of 90 bpm and global ST elevation with ST depression in lead aVR (Figure 12).

Initial troponin was within normal range, and the patient was diagnosed as a case of acute MI. And he was preloaded with antiplatelet and Heparin in ER, and the patient underwent cardiac catheterization after written informed consent, which showed 100% thrombotic occlusion of LAD and LCX with moderate to severe LV dysfunction with EF of 30% and severe disease in the proximal RCA (Figure 13-14).

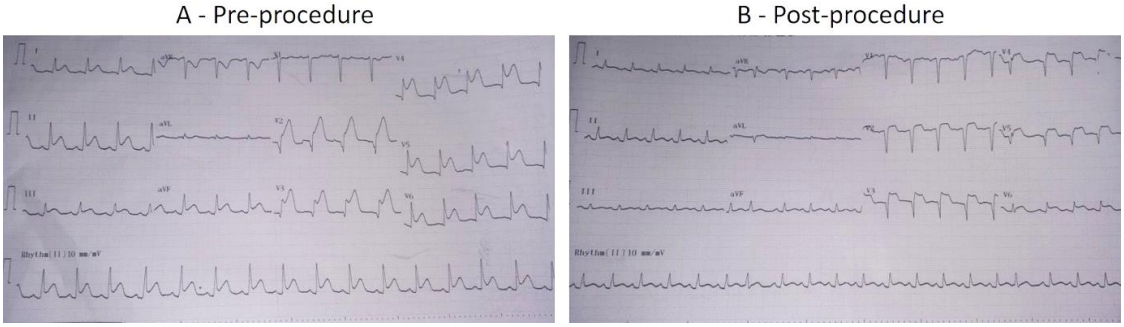

Figure 12: A) ECG at the time of presentation. B) ECG after PCI for Case 4

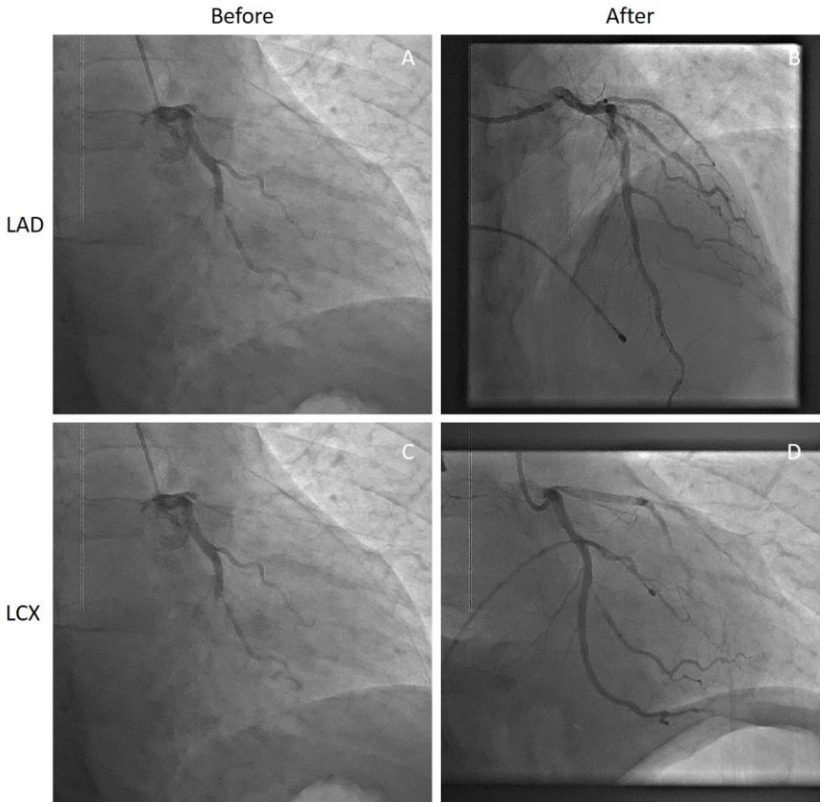

Figure 13: A) Pre-procedure coronary angiogram showing 95-99% occlusion of LAD. B) Coronary angiogram after PCI of LAD. C) Pre-procedure coronary angiogram showing 95-99% occlusion of LCX. D) Coronary angiogram after PCI of LCX

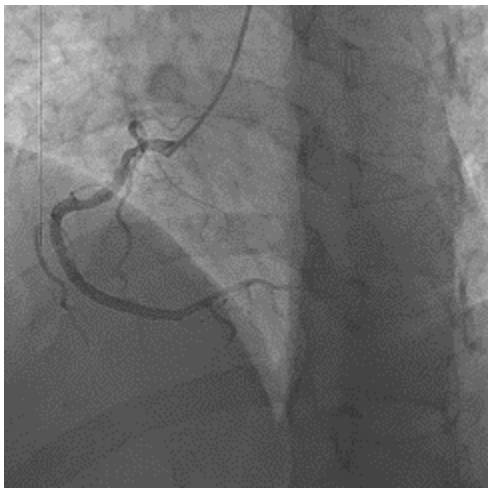

Figure 14: Coronary angiogram of right coronary artery showing thrombus in proximal RCA

We decided to deal with LAD first, PCI to LAD done with DES (3.5x15) after LAD stenting patient went into VFib, successfully defibrillated BP drops to 90/60, an intra-aortic balloon pump (IABP) pump was placed, and the patient was intubated during resuscitation after recovery of spontaneous circulation PCI to LCX was made with DES (3.5x26). TIMI III flow was achieved in both vessels. The patient was extubated the next day IABP was removed after 48 hours and discharged after four days of the procedure (Figure 15).

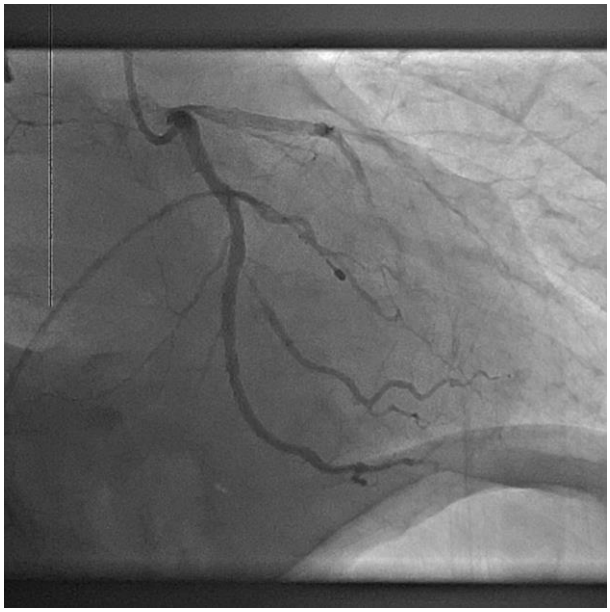

Figure 15: Coronary angiogram of the left coronary artery.

Case 5:

A 42-year-old male addicted to chewing tobacco with no past medical history of ischemic heart disease and connective tissue or hematologic disease presented with sudden onset of chest pain for four hours of duration. On physical examination, BP was 100/60 mmHg and a pulse rate of 40 bpm with an oxygen saturation of 92% on room air. Cardiovascular examination was normal. ECG showed sinus rhythm with the rate of 90 bpm and global ST elevation with ST depression in lead V1 to V6, II, III, and aVf (Figure 16).

The patient was diagnosed with a case of acute MI. And he was preloaded with antiplatelet and Heparin in ER, and the patient underwent cardiac catheterization; after written informed consent, right femoral access was taken, as the patient was in temporary bradycardia pacemaker (TPM) was passed, and then coronary angiography was performed, which showed 100% thrombotic occlusion of LAD and RCA with moderate to severe LV dysfunction with EF of 30%. We decided to deal with LAD; first, PCI to LAD done with DES (3.0x30) and post-dilated with NC balloon (3.5x12) and PCI to RCA was done with DES (3.5x34) and post-dilated with NC balloon (3.75x12) (Figure 17). TIMI III flow was achieved in both vessels. The patient regained his normal sinus rhythm, TPM was removed after 24 hours, and the patient was discharged after 48 hours.

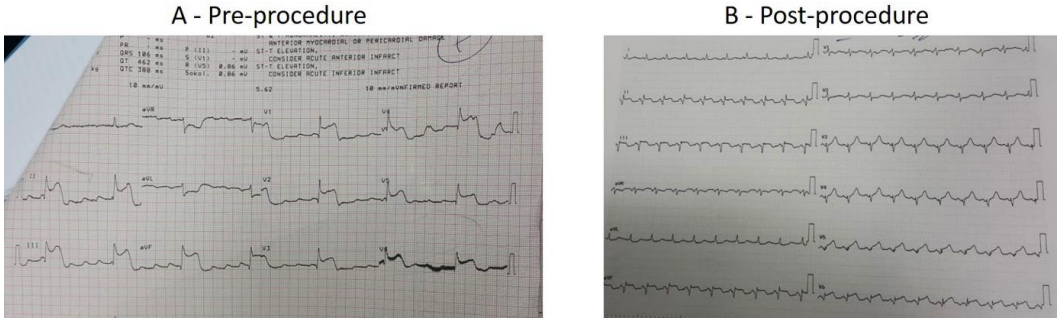

Figure 16: A) ECG at the time of presentation. B) ECG after PCI for Case 5

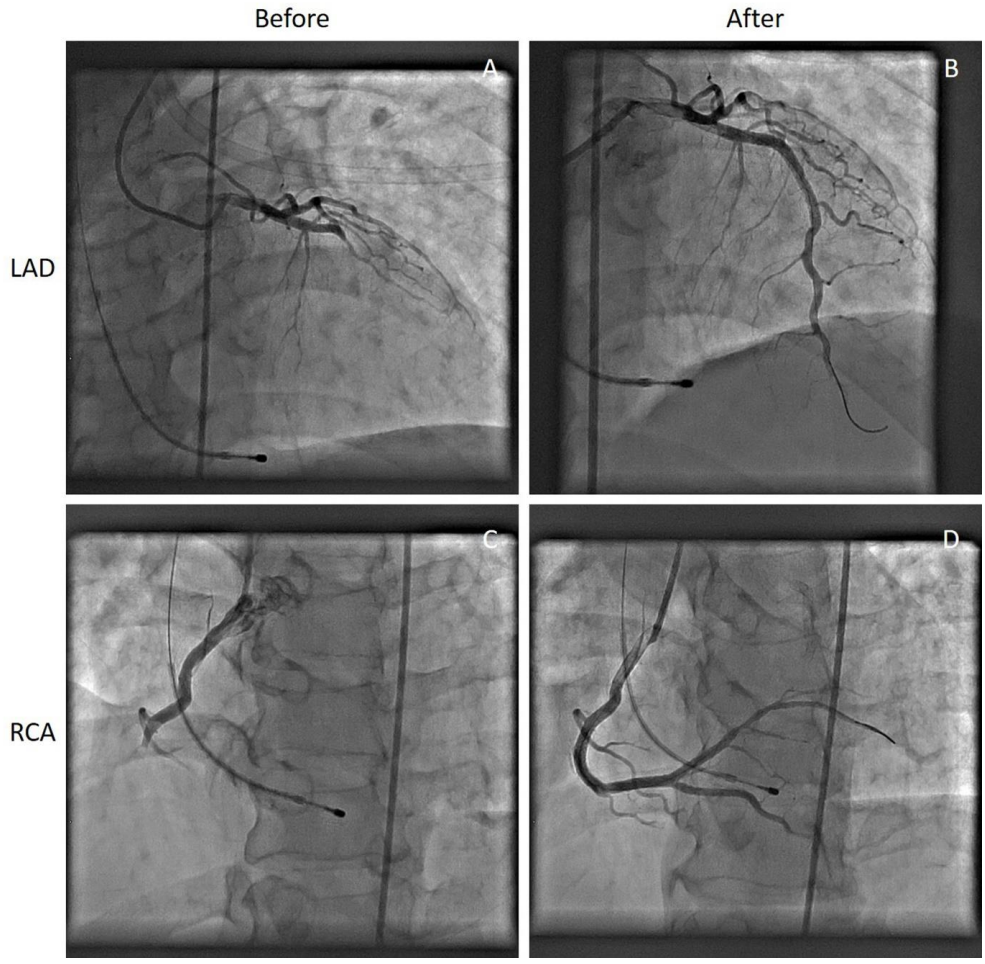

Figure 17: A) Pre-procedure coronary angiogram showing 100% occlusion of LAD. B) Coronary angiogram after PCI of LAD. C) Pre-procedure coronary angiogram showing 100% occlusion of RCA. D) Coronary angiogram after PCI of RCA

Discussion

Multivessel disease in ST-elevation MI (STEMI) is a common angiographic finding. It is observed that patients with multivessel disease in STEMI have a worse prognosis than patients with the single-vessel disease[7]. Occlusion of two or more coronary arteries simultaneously in STEMI is rare and difficult to diagnose before coronary angiography and associated with a very high rate of complications (i.e., cardiogenic shock, arrhythmia, and heart failure)[3]. The cases that we present in this case series are worthy since all three patients had STEMI with multiple culprits in all of them were dealt with primary revascularization with no adverse outcome. None of these factors like multivessel spasms, connective tissue disorder, and hypercoagulability are known factors associated with simultaneous coronary occlusion were present in our cases. Even though the definite etiology of multivessel coronary thrombosis is not fully understood[4].

Up till now, primary PCI remains the best reperfusion strategy in ST-elevation MI if performed on time [8]. Although, in patients with stable hemodynamic non-culprit vessel PCI at the time of the index, PCI is not recommended according to the current STEMI guidelines[3]. However, due to the extreme rarity of this clinical scenario and perceived insufficient clinical data of multivessel culprits in the setting of acute MI, optimal medical management is not very clear.

Various causes have been reported with successful thrombus aspiration and stenting in the setting of STEMI with multiple culprit lesions [2-4,9,10].

Conclusion

Acute myocardial infarction caused by simultaneous multi-vessel coronary occlusion is rarely reported. The successful intervention of multiple culprits with closed monitoring of the contrast leads to a good outcome and short hospital stay. Although, the optimal management strategy for the simultaneous multiple culprit lesions has yet to be established.

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References

1. Kilit C, Şen T, Doğan A, Amasyalı B, Özgeyik M. Myocardial reinfarction with simultaneous occlusions of two major coronary arteries one of which is due to the early stent thrombosis. Int J Cardiovasc Acad. 2016; 2(1): 21-4.
2. Choudhary S, Jain A, Choudhary R. Acute myocardial infarction with simultaneous involvement of right coronary artery and left anterior descending artery: A case report. J Indian Coll Cardiol. 2016; 6 :28-31.
3. Pollak PM, Parikh SV, Kizilgul M, Keeley EC. Multiple culprit arteries in patients with ST segment elevation myocardial infarction referred for primary percutaneous coronary intervention. Am J Cardiol. 2009; 104(5): 619-23.
4. Ifedili IA, Bob-Manuel T, Bolorunduro O, Askari R, Ibebuogu UN. Complete Revascularization of Simultaneous Multiple Culprit Lesions in a Septuagenarian with ST-Elevation Myocardial Infarction. Am J Case Rep. 2016; 17: 997.
5. Matte BD, Araujo GN, Valle FH, Krepsky AM. Multiple Culprit Coronary Artery Thrombosis in a Patient with Coronary Ectasia. Case Rep Cardiol. 2018;1-4.
6. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, Chambers CE, Ellis SG, Guyton RA, Hollenberg SM, Khot UN. 2015 ACC/AHA/SCAI focused update on primary percutaneous coronary intervention for patients with ST-elevation myocardial infarction: an update of the 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention and the 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction. J Am Coll Cardiol. 2016 ;67(10): 1235-50.
7. Batra MK, Rasool SI, Solangi BA, Khan N, Karim M, Hassan RS. Multivessel Disease As A Prognostic Marker In Patients Presenting For Primary Percutaneous Coronary Intervention. J Ayub Med Coll Abbottabad. 2018; 30(4): 534-8.
8. Andersen HR, Nielsen TT, Rasmussen K, Thuesen L, Kelbaek H, Thayssen P, Abildgaard U, Pedersen F, Madsen JK, Grande P, Villadsen AB. A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. N Engl J Med. 2003; 349(8): 733-42.
9. Lanaro E, Pereira Júnior EC, Falcão FJ, Barbosa AH. Simultaneous thrombosis in two epicardial coronary arteries during acute myocardial infarction. Rev Bras Cardiol Invas. 2012; 20(4): 431-4.
10. Ananthakrishna R, Wang LJ, Zhao LP, Tan HC. Double jeopardy in acute ST-segment elevation myocardial infarction. Singapore Med J. 2017; 58(4): 225-227.