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Complete Revascularization of Simultaneous Multiple Culprit Lesions in a Septuagenarian with ST-Elevation Myocardial Infarction

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Female, 74
Final Diagnosis: Multiple culprit lesions in ST-elevation myocardial infarction
Symptoms: Chest pain • shortness of breath
Medication: —
Clinical Procedure: Cardiac catheterization
Specialty: Cardiology

Objective: Unusual clinical course





Background: ST-elevation myocardial infarction (STEMI) is usually caused by rupture of unstable plaque with thrombus formation and abrupt cessation of blood flow through a single coronary artery that is deemed the culprit. The simultaneous thrombotic occlusions of multiple coronary arteries in the setting of STEMI is a rare occurrence with implications for patient management and outcome not fully addressed in the current STEMI guidelines, although more recent studies suggest a benefit of complete revascularization compared to culprit vessel-only treatment in the setting of STEMI.

Case Report: A 74-year-old female presented with STEMI. Coronary angiography revealed simultaneous multiple coronary thrombotic occlusions involving the right coronary, left circumflex, and ramus intermedius arteries successfully treated with primary percutaneous revascularization at the same setting with good outcome and short hospital length of stay.

Conclusions: Although the most appropriate timing to treat simultaneous multiple culprit lesions has yet to be definitively defined, multi-vessel percutaneous coronary intervention in the setting of a STEMI with multiple culprit lesions is feasible with good outcome as shown by our index case.

MeSH Keywords: Acute Coronary Syndrome • Angioplasty, Balloon, Coronary • Anterior Wall Myocardial Infarction

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/900849>

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Background

The rupture of coronary atherosclerotic plaque resulting in thrombus formation and vessel occlusion is the underlying pathogenesis of ST-elevation myocardial infarction (STEMI) [1]. Although for the most part this occlusion involves a single coronary artery, simultaneous thrombotic occlusions in multiple coronary arteries in the setting of STEMI are rare, poorly understood, and difficult to diagnose from a 12-lead electrocardiogram (EKG) [2]. The reported incidence of multiple thrombotic coronary occlusions reached 50% in an autopsy series of patients who died from sudden cardiac death, suggesting that the incidence is low in clinical practice because most of the patients die before medical contact [1]. Although, the specific mechanisms that trigger plaque disruption and occlusive thrombus formation in a single coronary artery are not fully understood, the guidelines are clear with regards to the percutaneous treatment of STEMI patients with a single culprit lesion, but less so with regards to simultaneous multiple coronary occlusions [3]. We describe a rare case of simultaneous multiple coronary thrombotic occlusions in a septuagenarian who presented with STEMI.

Case Report

A 74-year-old African-American female with a past medical history of coronary artery disease status after percutaneous coronary intervention (PCI), non-insulin dependent diabetes mellitus, hypertension, chronic obstructive pulmonary disease, prior cerebrovascular accident with residual right hemiparesis, and no prior history of hematologic or connective tissue disease, who presented to the emergency room with sudden-onset, severe, constant, left-sided chest pressure of approximately 1 h duration, which radiated to her left arm. There was associated shortness of breath and nausea. On physical examination, the patient was in moderate painful distress and diaphoretic with a blood pressure of 161/108 mmHg, heart rate of 78 beats/min, and respiratory rate of 18 cycles/min. Oxygen saturation was 99% on 2 liters of oxygen by nasal cannula. She had normal S1 and S2 heart sounds with no murmur, rubs, or gallops. Her lungs were clear to auscultation bilaterally. She had right-sided hemiparesis from her prior stroke. Initial 12-lead EKG findings showed a right bundle branch block with inferior ST elevation and reciprocal anterolateral ST depressions (Figure 1), which was new compared to an old EKG (Figure 2). Her initial troponin level was 0.062 ng/mL with a ProBNP of 229 pg/ml. Chest x-ray showed basilar airspace disease, most notably on the left. An assessment of

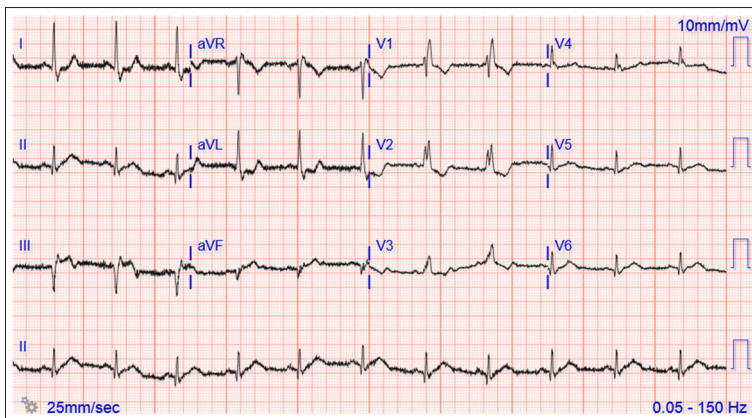


Figure 1. Admission electrocardiograph.

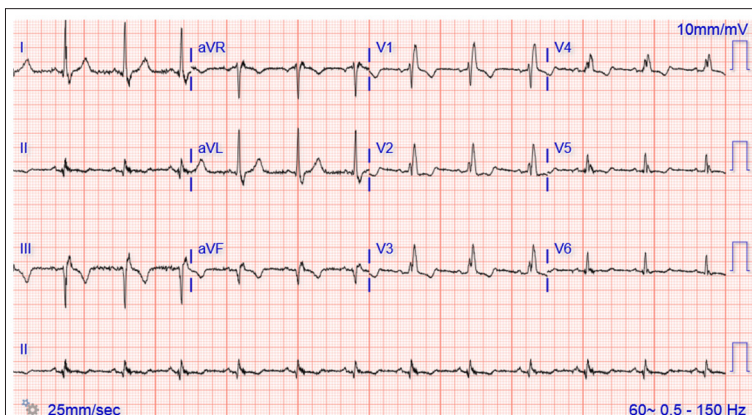


Figure 2. Old preadmission electrocardiograph.



Figure 3. Pre-PCI coronary angiogram showing 100% in-stent thrombotic occlusion of the PDA branch of the RCA (white arrow). Left anterior oblique view with cranial angulation.



Figure 5. Pre-PCI coronary angiogram showing 99% high-grade in-stent restenosis of proximal ramus intermedius (white arrow). Left anterior oblique view with caudal angulation.

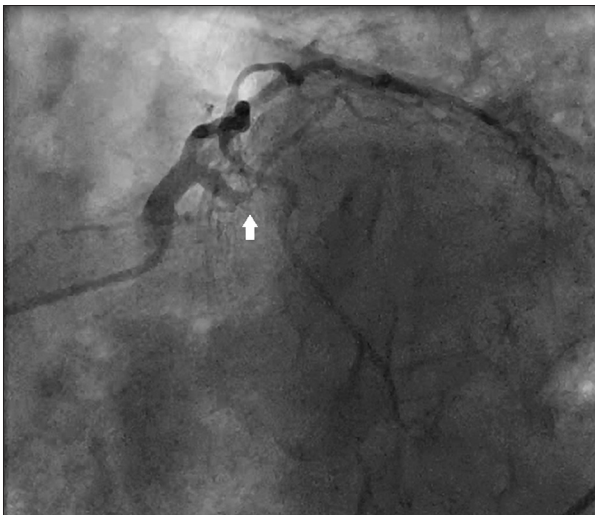


Figure 4. Pre-PCI coronary angiogram showing 100% thrombotic occlusion of the proximal LCX (white arrow). Left anterior oblique view with caudal angulation.

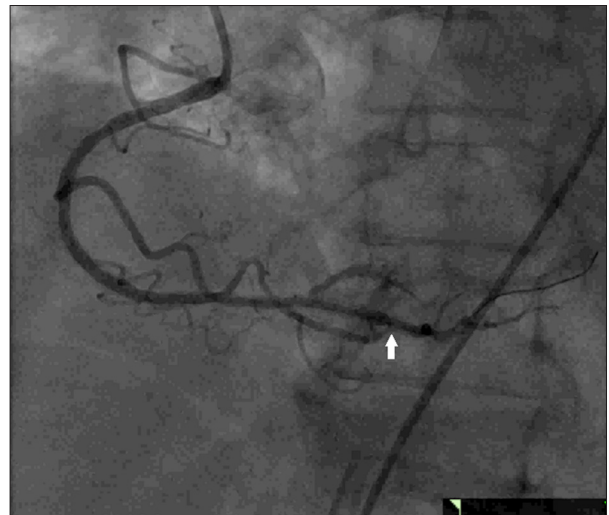


Figure 6. Coronary angiogram after PCI of PDA branch of the RCA showing resolution of occlusion (white arrow). Left anterior oblique view with caudal angulation.

inferior ST-segment elevation myocardial infarction was made and the patient underwent emergent cardiac catheterization, which showed 100% thrombotic occlusion of the posterior descending artery (PDA) branch of the right coronary artery (RCA), 100% thrombotic occlusion of the proximal left circumflex artery (LCX), and a 99% in-stent thrombotic lesion of the proximal ramus intermedius (RI) (Figures 3–5). A left ventriculogram revealed an ejection fraction of 45% with no apical filling defect. She was preloaded with ticagrelor. Given inferior ST elevation on EKG, and review of her old cardiac catheterization films, which showed RCA dominance for PDA, we decided to tackle the occlusion of the PDA first with a successful PCI and

balloon angioplasty with restoration of thrombolysis in myocardial infarction (TIMI) 3 flow (Figure 6) while ensuring very minimal contrast use. However, the patient continued to experience severe chest pain and remained diaphoretic, so we decided to immediately intervene on the occluded LCX with successful PCI and drug-eluting stent implantation and restoration of TIMI 3 flow (Figure 7). Chest pain improved but did not completely resolve, necessitating intervention to the subtotally occluded ramus intermedius with balloon angioplasty restoring TIMI 3 flow (Figure 8). Her symptoms eventually resolved, with resolution of EKG changes (Figure 9). The patient was discharged less than 48 h post-PCI, with normal kidney

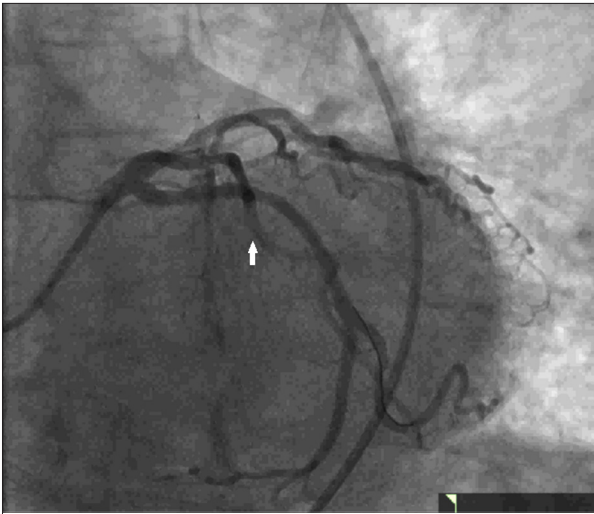


Figure 7. Coronary angiogram after PCI of LCX showing resolution of occlusion (white arrow). Left anterior oblique view with caudal angulation.

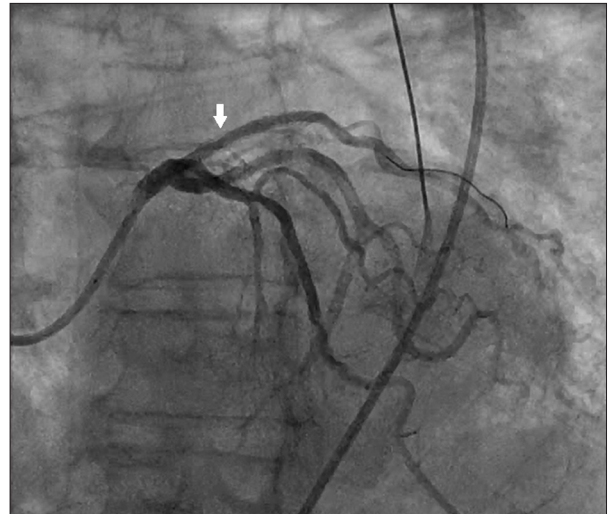


Figure 8. Coronary angiogram after PCI of ramus intermedius showing resolution of occlusion (white arrow). Left anterior oblique view with caudal angulation.

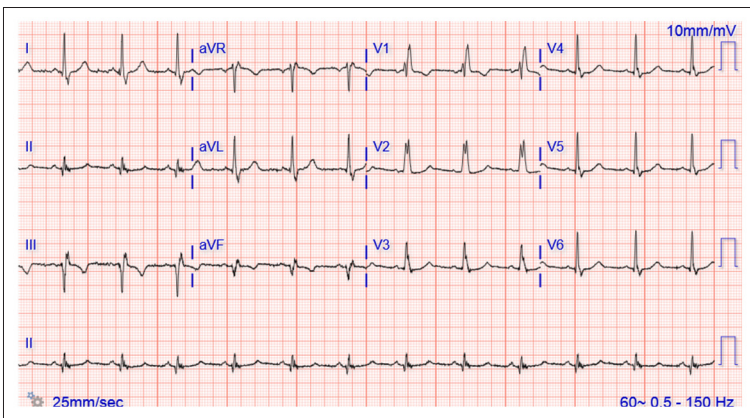


Figure 9. Post-intervention electrocardiograph.

function and no post-PCI complications despite complete revascularization of multiple coronary culprit lesions and her advanced age.

Discussion

In about 40% of STEMI there is presence of multiple non-occlusive coronary plaques [4], leading to the distinction of culprit versus non-culprit coronary artery plaques to describe lesions causing the ST elevation changes and those present but not causing an acute process. STEMI with simultaneous occlusion of 2 or more coronary arteries is very rare and difficult to diagnose before coronary angiography, with consequent high rate of arrhythmia, heart failure, and cardiogenic shock [5]. This case is worthy of note since this was an elderly STEMI patient with simultaneous thrombus formation in 3 coronary arteries treated with primary revascularization with no untoward adverse outcome. None of the known factors associated with

simultaneous coronary occlusion, such as multi-vessel spasm, hypercoagulability, and connective tissue disorder, were present in our patient. Although the etiology of multi-vessel coronary thrombosis is not fully understood, we propose that simultaneous rupture of unstable plaque is the likely etiology in our patient given her long-standing history of diabetes mellitus. Primary percutaneous coronary intervention remains the accepted reperfusion strategy in STEMI if performed in a timely manner [6]. Patients with multi-vessel non-occlusive disease at the time of primary coronary intervention tend to have worse long-term outcomes compared to those with less extensive coronary disease [7]. Although in patients without hemodynamic compromise, non-culprit vessel PCI at the time of the index PCI is contraindicated according to the STEMI guidelines [2], more recent studies show that primary intervention to a non-infarct artery with major stenotic lesions at the time of primary PCI leads to better outcome [8,9]. In the setting of multi-infarct STEMI with multiple culprit lesions, as seen in our patient, there are no randomized clinical trials elucidating

the optimal management strategy due to the extreme rarity of this clinical scenario and the perceived insufficient clinical equipoise to even undertake such a trial, hence the reliance on best clinical judgment. One of the main reservations about performing multiple interventions at the same setting is due to the concern for renal dysfunction and its impact on hospital length of stay and mortality. However, recent trials have shown this concerns to be unfounded and that multi-vessel percutaneous coronary intervention in STEMI patients is safe [9,10]. In our patient, careful attention was paid to remaining within the recommended threshold for total contrast amount as well as optimally hydrating the patient. This resulted in stable kidney function and a short hospital length of stay. This invariably lends credence to the school of thought suggesting that complete revascularization in the setting of STEMI is superior to performing a staged PCI. In inferior STEMI, various algorithms have been proposed in the prediction of the culprit artery based on EKG changes [11]. These algorithms could not be applied to our patient given that both the RCA and LCX were completely occluded, resulting in an EKG pattern variation that may be the sum total of long-standing ischemic heart disease and repeat interventions causing dependent collateral circulations that affect observed changes on EKG – in this instance, inferior versus lateral ST-segment elevations.

References:

1. Davies MJ, Thomas A: Thrombosis and acute coronary-artery lesions in sudden cardiac death. *N Engl J Med*, 1984; 310: 1137–40
2. 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: 619–23
3. Levine GN, Bates ER, Blankenship JC et al: 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: 1235–50
4. Goldstein JA, Demetriou D, Grines CL et al: Multiple complex coronary plaques in patients with acute myocardial infarction. *N Engl J Med*, 2000; 343: 915–22
5. 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: 619–23
6. Andersen HR, Nielsen TT, Rasmussen K et al, DANAMI-2 Investigators: A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. *N Engl J Med*, 2003; 349: 733–42
7. Sorajja P, Gersh BJ, Cox DA et al: Impact of multivessel disease on reperfusion success and clinical outcomes in patients undergoing primary percutaneous coronary intervention for acute myocardial infarction. *Eur Heart J*, 2007; 28: 1709–16
8. Wald DS, Morris JK, Wald NJ et al., PRAMI Investigators: Randomized trial of preventive angioplasty in myocardial infarction. *N Engl J Med*, 2013; 369: 1115–23
9. Villablanca PA, Briceno DF, Massera D et al: Culprit-lesion only versus complete multivessel percutaneous intervention in ST-elevation myocardial infarction: A systematic review and meta-analysis of randomized trials. *Int J Cardiol*, 2016; 220: 251–59
10. Engström T, Kelbæk H, Helqvist S et al., DANAMI-3 – PRIMULTI Investigators: Complete revascularisation versus treatment of the culprit lesion only in patients with ST-segment elevation myocardial infarction and multivessel disease (DANAMI-3 – PRIMULTI): An open-label, randomised controlled trial. *Lancet*, 2015; 386: 665–71
11. Vales L, Kanei Y, Schweitzer P: Electrocardiographic predictors of culprit artery in acute inferior ST elevation myocardial infarction. *J Electrocardiol*, 2011; 44: 31–35

Conclusions

Simultaneous multiple culprit lesions in the setting of STEMI as compared to a single culprit lesion with major stenotic lesions in non-infarct coronary artery are extremely rare. The successful coronary intervention of multiple culprit lesions with close attention to contrast volume in an elderly STEMI patient, leading to good outcome and short hospital length of stay, shows that complete revascularization can be achieved in this setting with no untoward adverse consequence. Although the most appropriate timing to treat simultaneous multiple culprit lesions has yet to be definitively established, given the low frequency of these events, it is critical that the interventionalist integrate assessment of lesion severity, patient clinical characteristics, and risk of peri-procedural complications in order to determine the optimal PCI strategy in STEMI patients with multiple culprit lesions.

Conflict of Interest

None.