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Case Report

Hemodynamic impairment of double culprit ST elevation myocardial infarction, double the trouble: a case report

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Abstract

Background: Multiple culprit artery involvement is rare (2.5%) among ST-segment elevation myocardial infarction (STEMI) patients undergoing primary coronary intervention (PCI). It can occur due to multiple factors and reflects a widespread pathophysiologic process. Most patients present with unstable hemodynamics and cardiogenic shock (CS), which results in a high mortality rate. Currently, there are no guidelines or consensus on the management of multiple culprit arteries in STEMI patients.

Case Illustration: A 51-year-old man with chest pain for the past 16 hours was referred to the National Cardiovascular Center, Harapan Kita. ECG at presentation revealed sinus rhythm with ST elevation in the inferior, posterior, and right leads. He was diagnosed with late-onset infero-posterior STEMI + right ventricle infarction, Killip IV, and thrombolysis in myocardial infarction 6/14, then was prepared for early PCI due to ongoing chest pain and CS. The patient underwent complete revascularization with drug-eluting stents and thrombus aspiration due to the high thrombus burden of the lesion in the right coronary artery and first obtuse marginal artery. After early PCI, his hemodynamic condition improved, and epigastric pain was his only complaint. However, on the following day, the patient experienced acute pulmonary edema and rhythm conversion to total AV block. He was managed conservatively with heparinization, inotropes, vasopressors, diuretics, and noninvasive ventilation. After 14 days of hospitalization, the patient was discharged without any complaints.

Conclusion: Double culprit STEMI is rare and associated with catastrophic hemodynamic impairment, including CS, at presentation. Individualized treatment with early and aggressive revascularization yields relatively good results.

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Introduction

In cases of acute ST-segment elevation myocardial infarction (STEMI), usually, only one epicardial coronary artery is completely occluded by thrombus, referred to as the "culprit" artery, and the rate of having multiple culprit arteries is only 2.5% among patients undergoing emergent primary percutaneous coronary intervention (PCI).[1] In contrast, from an autopsy series, it was found that multiple culprit artery thrombotic occlusions occurred in up to 50% of patients.[2] This discrepancy may be explained by the fact that patients with multiple culprit STEMI are more likely to experience sudden cardiac death and not survive long enough to undergo angiography.

Multiple acute coronary thromboses can occur due to multiple factors, including plaque rupture, erosion, coronary artery dissection, coronary vasospasm, cocaine abuse, hypercoagulability, or coronary embolism; however, in most cases, the underlying mechanisms remain unclear.[3] At least in some cases, acute coronary events reflect widespread pathophysiologic processes, which may lead to multifocal plaque instability coupled with clinical instability at multiple locations.[4] One certain thing is that multiple culprit STEMI cases are related to unstable hemodynamics and cardiogenic shock (CS), which results in a high mortality rate.[1-2]

Currently, there are no guidelines or consensus on the management of multiple culprit arteries in STEMI patients. This case report aims to present the hemodynamic impairment of a patient with late-onset STEMI who presented with simultaneous total occlusion of two major epicardial coronary arteries treated with immediate complete revascularization.

Case Illustration

A 51-year-old male was referred to the National Cardiovascular Center Harapan Kita emergency department with a complaint of chest pain accompanied by nausea and sweating in the past 16 hours. The patient had a history of uncontrolled hypertension and was a smoker. He was referred with a diagnosis of inferior STEMI (Figure 1) complicated by CS and total AV block (TAVB) and had received 320 mg aspirin, 300 mg clopidogrel, 5000 IU heparin continued with 1000 IU/24 hours, 40 mg atorvastatin, and hemodynamic support using dobutamine, dopamine, and norepinephrine.

Upon arrival at our hospital, his blood pressure was 88/61 mmHg, and his heart rate was 110 beats per minute (bpm) with support of dobutamine 7 mcg/kg/min, dopamine 5 mcg/kg/min, and norepinephrine 0.05 mcg/kg/min. His lung sounds were vesicular with bilateral 1/3 basal rales. ECG at 16 hours after onset revealed sinus tachycardia with a rate of 107 bpm, left axis deviation, a normal P wave, a PR interval of 160 ms, pathological Q waves in II, III, avF, V3R-V4R, and V7-V9, a QRS duration of 90 ms, ST elevation in III, avF, and V8-V9, ST depression in I, avL, and V2-V4, and left ventricle hypertrophy (Figure 2). Laboratory tests revealed reduced renal function (urea 42 mg/dL, creatinine 2.37 mg/dL, eGFR 33 mg/mmol) and elevated high-sensitivity troponin T (4314 ng/mL). Bedside echocardiography revealed a reduced left ventricular ejection fraction (LVEF) (18%), reduced tricuspid annular plane systolic excursion (TAPSE) (7 mm), and regional wall motion abnormalities seen as akinetic inferolateral and inferior segments, while other segments were hypokinetic. His hemodynamics profiles were elevated estimated right atrial pressure (15 mmHg), low cardiac output (CO) (1.6 L/min), and high systemic vascular resistance (SVR) (3150 dynes/sec/cm^{-5}). He was then diagnosed with late-onset (16 hours) inferoposterior STEMI + right ventricle infarction, Killip IV, and thrombolysis in myocardial infarction (TIMI) 6/14, with acute kidney injury dd/acute on chronic kidney disease. The patient was then prepared for early PCI. Dopamine was stopped, dobutamine was increased to 10 mcg/kg/min, and another loading of 300 mg of clopidogrel was given.

Coronary angiography during early PCI revealed total occlusion of the proximal right coronary artery (RCA) (Figure 3A) and proximal first obtuse marginal artery (OM1) (Figure 3B) with thrombus grade V and TIMI 0 flow. Balloon predilatation and thrombus aspiration were initially performed at the RCA. A 3.5x33 mm Xience Xpedition (Abbott Vascular, USA) drug-eluting stent (DES) was implanted, and the final angiographic evaluation of the RCA revealed TIMI 3 flow with no residual thrombus or stenosis and no perforation (Figure 3C). For the OM1 lesion, balloon predilatation was performed initially, followed by stent implantation with a 2.5x20 mm Promus Premiere (Boston Scientific, USA). Because the angiographic evaluation showed TIMI 2 flow with a hazy appearance, it was decided to perform thrombus aspiration once, which resulted in TIMI 3 flow with residual thrombus, no residual stenosis, and no perforation (Figure 3D). The patient is then transferred to the intensive cardiovascular care unit (ICVCU).

After the procedure, the patient experienced no more chest pain but epigastric pain with a VAS score of 2/10. Hemodynamic echocardiography revealed improvement with dobutamine 5 mcg/kg/min as the patient's only hemodynamic support. However, one day after the early PCI, the patient experienced respiratory distress with signs of acute pulmonary edema and reduced SpO_2 to 92-93% on 3 3-liter per minute nasal cannula. His ECG changed to TAVB with a junctional escape rhythm, and his heart rate decreased from 110-120 to 80-90 bpm without any sign of new ischemic changes (Figure 4). Hemodynamic echocardiography revealed reduced CO from 3.8 to 2.6 L/min and increased SVR from 1410 to 1907 dynes/sec/cm^{-5}. His laboratory test also revealed worsening renal function due to hypoperfusion (creatinine 3.0 mg/dL).

For the TAVB, 5 mcg/kg/min dopamine was added, and his heart rate increased from 80 to 100 bpm. His hemodynamics also improved, with CO increasing to 3.5 L/min and SVR to 1440 dynes/sec/cm-5. For breathing problems, oxygen supplementation was escalated to noninvasive ventilation (NIV) with 5 mmHg positive end-expiratory pressure, 5 mmHg pressure support, and 70% FiO_2. His blood gas analysis showed improvement (lactate decreased from 3.4 mg/dL to 2.0 mg/dL), and his SpO_2 increased to 99%. A bolus of 40 mg intravenous furosemide was also administered and continued at 5 mg/hour. Heparinization with UFH continued until 5 days after early PCI. His urine output target (1.5 cc/kg/hour) was achieved the next morning, and his creatinine level improved to 1.8 mg/dL.

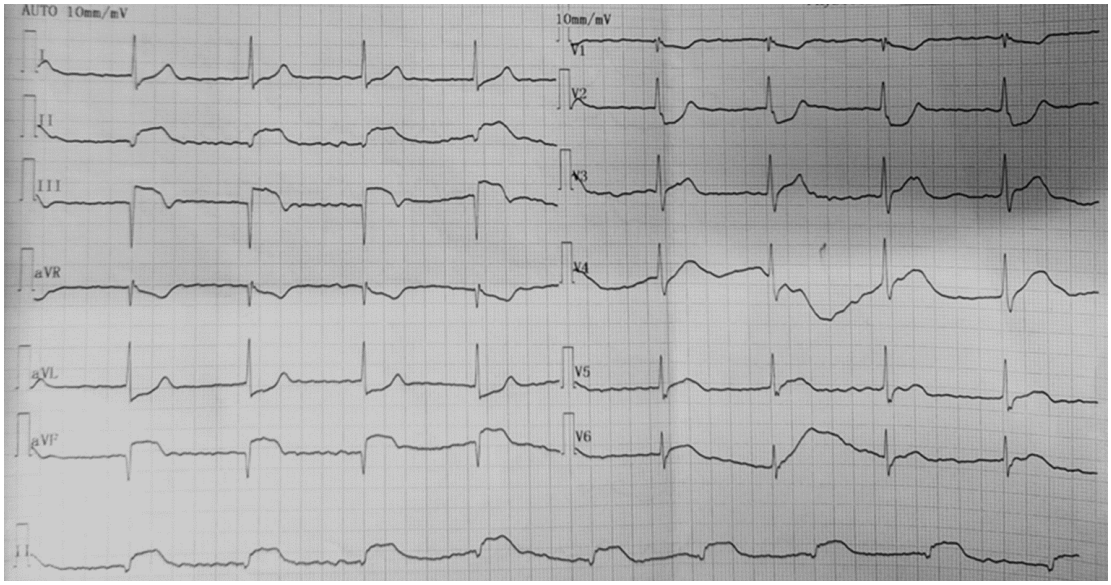

Figure 1. ECG at 5 hours after onset.
Total AV block with atrial fibrillation, inferior leads ST-Elevation.

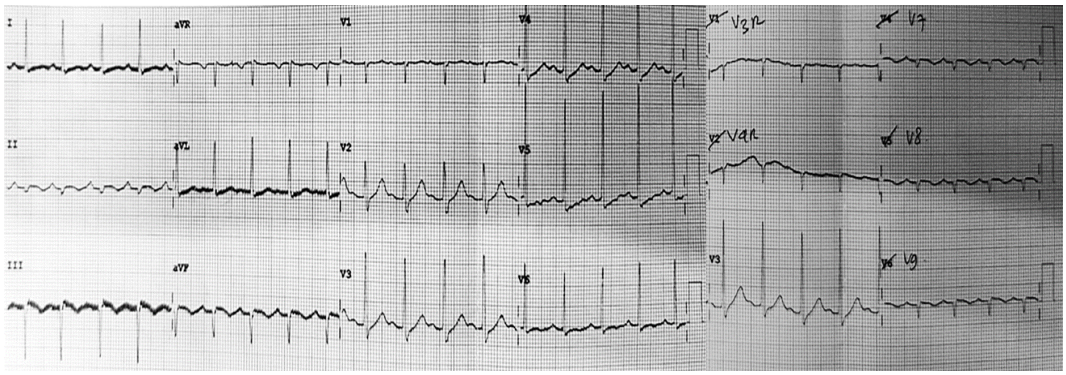

Figure 2. ECG at 16 hours after onset.
Sinus tachycardia with inferoposterior and right leads ST-Elevation.

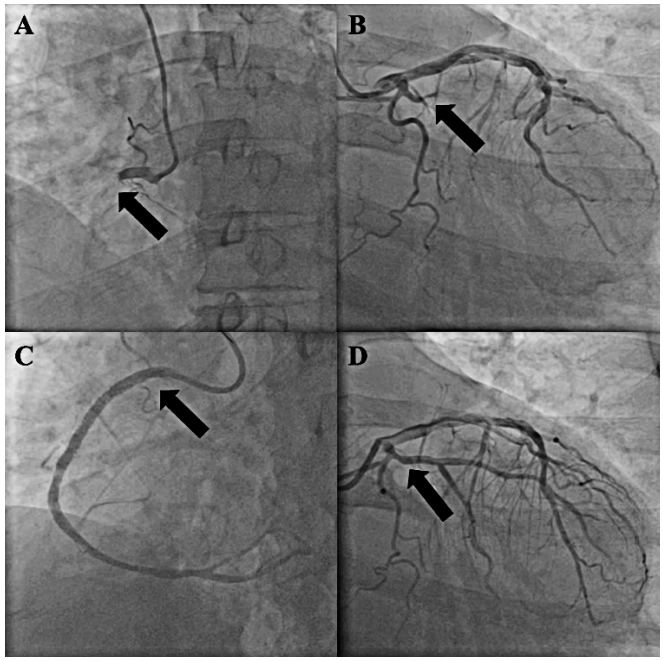

Figure 3. Early percutaneous coronary intervention.
Initial angiography showing lesions in A) right coronary artery and B) first obtuse marginal artery; final angiography of C) right coronary artery and D) first obtuse marginal artery.

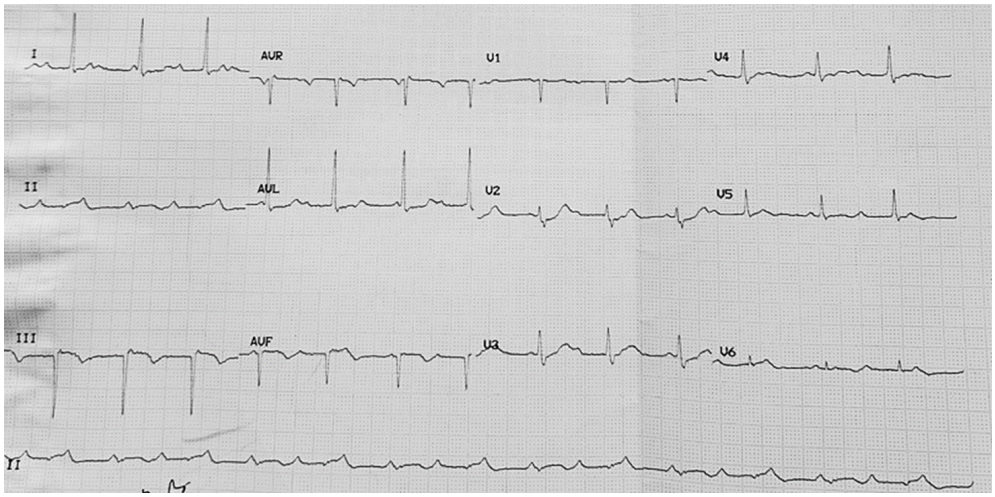

Figure 4. ECG one day after early percutaneous coronary intervention.
TAVB with accelerated junctional escape rhythm, no new ischemic changes.

After 7 days in the ICVCU, his rhythm returned to sinus, and after 10 days, he was able to weaned from all support and moved to the general ward. His medication was changed to oral medication, and after 14 days of hospitalization, he was discharged without any complaints with medication of 1x80 mg aspirin, 2x90 mg ticagrelor, 1x20 mg atorvastatin, 1x0.6125 mg bisoprolol, 1x10 mg ramipril, 1x25 mg spironolactone, and 2x40 mg furosemide. Predischarge echocardiography showed improvement in his LVEF (42%) and TAPSE (18 mm). Summary of the case timeline presented in Figure 5.

Discussion

The term culprit lesion refers to the coronary lesion considered to be responsible for Acute Coronary Syndrome (ACS). According to the 2020 European Society of Cardiology (ESC) guidelines on ACS presenting without ST-segment elevation, at least two of the following morphological features suggestive of acute plaque rupture should be present: (1) intraluminal filling defects consistent with thrombi; (2) plaque ulceration; and (3) plaque irregularity, dissection, or impaired flow.[5] In our patient, we observed intraluminal filling defects consistent with thrombi showing

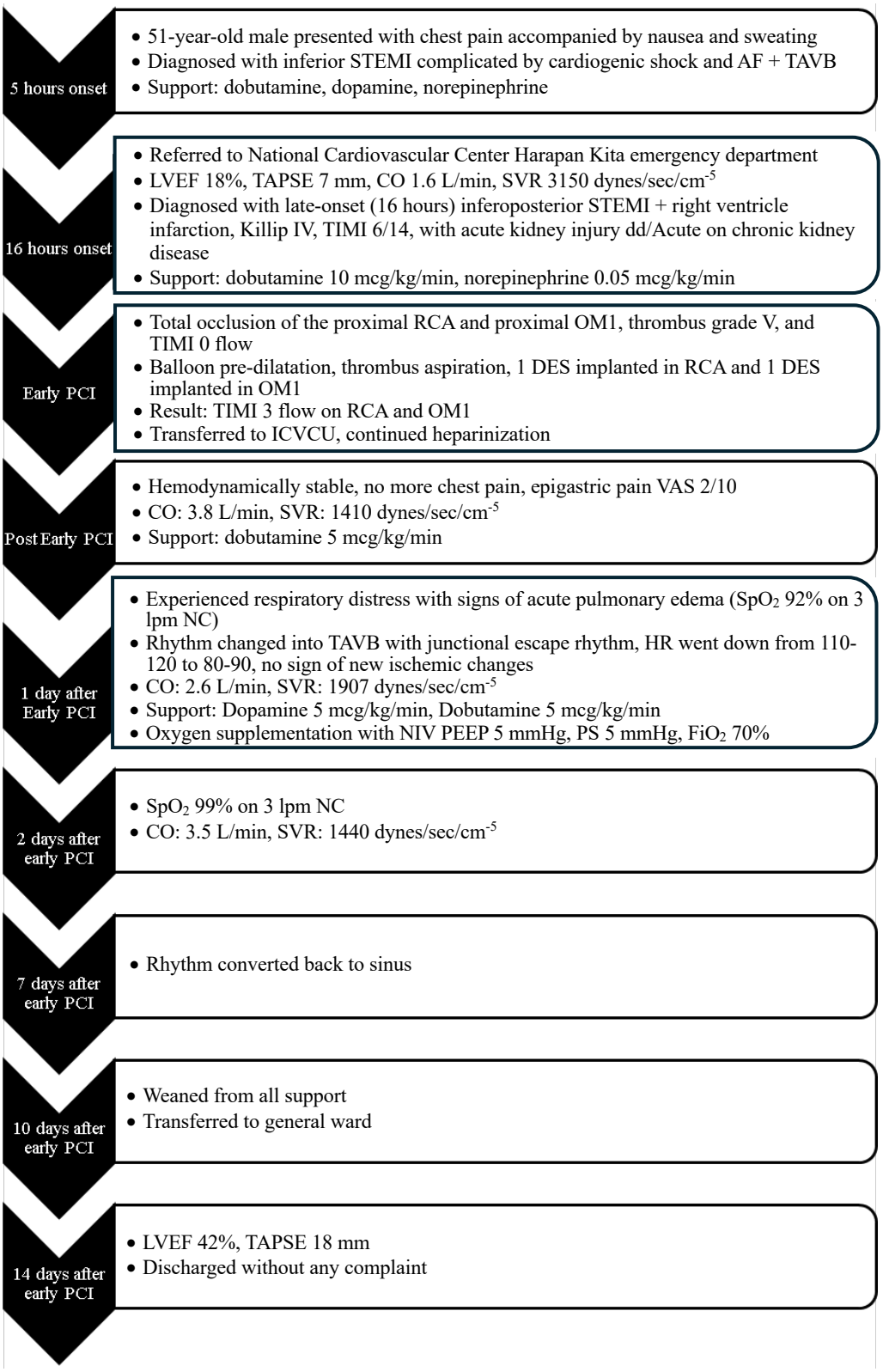

Figure 5. Case timeline.

STEMI: ST-elevation myocardial infarction; AF: atrial fibrillation; TAVB: total AV bock; LVEF: left ventricle ejection fraction; TAPSE: tricuspid annular plane systolic excursion; CO: cardiac output; SVR: systemic vascular resistance; TIMI: thrombolysis in myocardial infarction; PCI: percutaneous coronary intervention; RCA: right coronary artery; OM1: first obtuse marginal artery; DES: drug-eluting stent; ICVCU: intensive cardiovascular care unit; NC: nasal cannula; HR: heart rate; NIV: non-invasive ventilation; PEEP: positive end-expiratory pressure; PS: pressure support.

acute occlusion abruptly ending with a squared-off or convex margin with impaired flow in two major epicardial coronary arteries, the RCA and OM1. Furthermore, contrast staining consistent with the TIMI thrombus burden classification was also observed for OM1. We can also note that after PCI, angiography of the RCA revealed no collaterals to the LCx, which confirmed that the occlusion was of acute origin. Therefore, it is concluded that the patient had a double culprit lesion.

A systematic review by Mahmoud et al.[2] on simultaneous multivessel coronary thrombosis in patients with STEMI found that the mean age of the patients was 59 years; most were male (88%), were current smokers (59%), and had a history of hypertension (50%). Atrial fibrillation (AF) at presentation or a history of AF was not common (5%). CS was the most common clinical presentation, occurring in 41% of the patients, followed by ventricular arrhythmias (25%). The most common territory of ST-segment elevation seen on ECG is in the inferior leads (29%). Meanwhile, from coronary angiography findings, the most common coronary artery with a thrombus burden was the RCA + left anterior descending artery (50%), followed by the RCA + left circumflex artery (LCx) (32%). Our patient had similar clinical characteristics to those reported in the literature. He was a male with a history of uncontrolled hypertension and was a current smoker. He presented with CS and ST elevation in the inferior leads. His coronary angiography result was the second most common finding, which involved the RCA + LCx.

Currently, there are no guidelines or consensus on the management of multiple culprit arteries in STEMI patients. The available guidelines or trials are limited to patients with multivessel disease (MVD) who present with ACS and are related to the decision on when to revascularize another significant non-Infarct-Related Artery (IRA) stenoses. Nevertheless, complete revascularization is recommended for STEMI patients with MVD either during the index procedure or within 45 days due to better mortality outcomes.[6] For patients presenting with STEMI and CS, according to the SHOCK[7] and CULPRIT-SHOCK[8] trials, immediate revascularization of the IRA only was advised. Table 1 provides insight into several case reports showing different interventions for double culprit STEMI. However, due to the large territory of the myocardium jeopardized by double culprit STEMI, all reports have shown early and aggressive attempts at complete revascularization that yielded relatively good results.

Our patient arrived on late-onset (16 hours) compared to other case reports (<6 hours) and beyond the ideal time frame for primary PCI (<12 hours).[15] However, due to the presence of CS and ongoing chest pain, our patient was scheduled for early PCI. We decided to perform complete revascularization because both lesions were deemed to be culprit lesions. Thrombus aspiration was performed because, after predilation in the RCA and placement of the stent in the LCx, there was still residual thrombus. Although the TOTAL[16] trial revealed that routine thrombus aspiration did not improve outcomes and was associated with an increased rate of stroke in patients with a high thrombus burden, current ESC guidelines on ACS still recommend it in patients with a large residual thrombus burden after opening the vessel with a guide wire or balloon.[15] Anticoagulants should generally be discontinued immediately after PCI, except in specific clinical circumstances.[15] However, in our case, the patient was still given heparinization for 5 days because there was still a hazy appearance in the acute marginal branch and OM1 according to angiography. We found that the patient's clinical condition (epigastric pain) and rhythm were also improved after heparinization, which was consistent with the observed improvement in ischemia. After heparinization, the patient's P2Y12 inhibitor was also escalated from clopidogrel to ticagrelor.

Because the patient's condition deteriorated due to rate problems, we decided to administer dopamine for his TAVB. However, it was decided not to rush the use of a temporary pacemaker because the escape QRS complex was still narrow, suggesting a supra-Hisian block that might respond to medication.[17] Meanwhile for the use of mechanical circulatory support, we planned to use an intra-aortic balloon pump (IABP) because although there was significant improvement in our patient's hemodynamics with dopamine, his CO (from 2.6 to 3.5 L/min) and SVR (from 1907 to 1440 dynes/sec/cm^{-5}) still exhibited borderline values, with a cardiac index of 1.9 L/min/m^2. Nevertheless, the current guidelines from the IABP-SHOCK II study do not suggest the routine use of the IABP in patients without mechanical complications due to the lack of improvement in mortality.[15,18] In the end, we only used inotropic/vasopressor drugs, and fortunately, our patient responded well.

Table 1. Various case reports on double culprit STEMI

Author	Onset	Intervention	Outcome
Hage et al.[9]	3 hours	• GP IIb/IIIa • Thrombus aspiration (LAD and RCA) • LMWH (4 days)	The patient was discharged on day 5, with decreasing troponin, normal kidney function, and no hemorrhagic complications. OCT evaluation → thrombus (-)
Turgeman et al.[10]	3 hours	• IABP • GP IIb/IIIa • Thrombus aspiration + 125,000 units of urokinase (LAD and RCA)	Seven days post-admission, repeat coronary angiography showed near-normal coronary vessels
Caliskan et al.[11]	No data	• PCI of RCA and LAD	Resolution of chest pain, ST-elevation, and hemodynamic stabilization
Saito et al.[12]	40 mins	• TAVB → TPM • Thrombus aspiration → DES (RCA and LAD) • Impella CP (3 days)	On the 12th day, the patient had a stroke → discharged on the 57th day of hospitalization with NYHA class II HF
Nordkin et al.[13]	1.5 hours	• PCI of LCx and RCA	On day six, the patient was discharged in good medical condition without signs of congestive heart failure (CHF).
Ginanjar et al.[14]	2 hours	• Thrombus aspiration and DES in LAD and LCx	Improvement of patient clinical condition, ST elevation, and hemodynamic status

Conclusion

A rare case of double culprit STEMI has been presented. It is usually associated with catastrophic hemodynamic impairment, including CS at presentation, due to a large area of myocardium being jeopardized. The underlying mechanism in most patients remains unclear, with no specific guidelines or consensus on its management. However, individualized treatment with early and aggressive revascularization yields relatively good results.

List of Abbreviations

AF Atrial Fibrillation
CO Cardiac Output
CS Cardiogenic Shock
DES Drug-eluting Stent
ECG Electrocardiogram
HR Heart Rate
ICVCU Intensive Cardiovascular Care Unit
LVEF Left Ventricle Ejection Fraction
NC Nasal Cannula
NIV Non-invasive Ventilation
OM1 First Obtuse Marginal Artery
PCI Primary Percutaneous Coronary Intervention
PEEP Positive End-expiratory Pressure
PS Pressure Support
RCA Right Coronary Artery
STEMI ST-segment Elevation Myocardial Infarction
SVR Systemic Vascular Resistance
TAPSE Tricuspid Annular Plane Systolic Excursion
TAVB Total AV Block
TIMI Thrombolysis in Myocardial Infarction
UFH Unfractionated Heparin
VAS Visual Analogue Scale

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

MA: Conception, case management, data collection, drafting, and critical revision of the manuscript; JEA: Case management support, literature review, drafting of clinical details, and

manuscript editing; SSD: Senior supervision, critical revision of intellectual content, and final approval of the version to be published.

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Conflict of Interest
The authors declared that they have no competing interests.

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