JACC: CASE REPORTS VOL. 3, NO. 18, 2021

CASE REPORT

INTERMEDIATE

CLINICAL CASE

Double Culprit Lesions in a Patient With ST-Segment Elevation Myocardial Infarction

Should I Stent or Should I Go?

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ABSTRACT

We describe the case of simultaneous acute thrombotic occlusions in 2 major coronary arteries in a young patient admitted for extensive myocardial infarction. We highlight the importance of intravascular imaging to determine the underlying putative mechanism and to optimize treatment decisions. (**Level of Difficulty: Intermediate.**)
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HISTORY OF PRESENTATION

A 53-year-old man was admitted to our hospital for an extensive ST-segment elevation myocardial infarction (STEMI). Three hours before admission, he reported sudden onset of acute chest pain at rest, associated with right arm weakness. Heart rate was 67 beats/min; blood pressure was 142/73 mm Hg; SpO_2 was 90%. On physical examination, heart and lung sounds were normal. The patient did not present any sign of cardiogenic shock.

Initial 12-lead electrocardiogram showed sinus rhythm with extensive anterior and inferior ST-segment elevation and ST-segment depression in leads aVR and aVL (Figure 1). He was preloaded with 250 mg aspirin intravenously, 180 mg ticagrelor orally, and 4,000 IU of unfractionated heparin intravenously. In addition, 12 mg of morphine was administered for pain relief.

LEARNING OBJECTIVES

- To recognize this life-threatening presentation of myocardial infarction.
- To learn how to rule out mechanism of acute coronary artery thrombosis.
- To highlight the importance of intravascular imaging guidance to optimize treatment decisions.

MEDICAL HISTORY

He was a current smoker without any other cardiovascular risk factor or significant past medical history. There was no history of illicit drug abuse.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received April 1, 2021; revised manuscript received July 14, 2021, accepted July 21, 2021.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2021.07.028

DIFFERENTIAL DIAGNOSIS

In this case, our main goal was to determine the origin of multiple coronary artery thrombi. There was no evidence of illicit drug abuse or prior coagulopathy. Initial angiogram did not show any sign of prolonged vasospasm or evidence of spontaneous coronary artery dissection. Different investigations were also used to rule out a cardioembolic origin. Transthoracic echocardiography showed moderate left ventricular dysfunction (45%) with apical akinesis. There was no evidence of intracardiac thrombus or valvular abnormalities. Agitated saline contrast injection to detect intracardiac shunt was also negative. Cardiac computed tomography angiogram showed no evidence of intracardiac thrombus including a normal left atrial appendage free of thrombus. Forty-eight hours of telemetry did not show any arrhythmia. Testing for hereditary hypercoagulable state was not done.

INVESTIGATIONS

Emergent coronary angiogram with a left transradial approach revealed a large thrombus in the proximal left anterior descending artery (LAD) with distal emboli in the distal LAD and the first diagonal branch (**Figure 2A**, Video 1). Another large thrombus was present in the mid-right coronary artery (RCA) with distal emboli in a posterolateral branch. Except for embolic occlusion of the distal branches, TIMI flow grade was normal in both LAD and RCA arteries (**Figure 3A**, Video 2). Moreover, the angiogram revealed coronary arteries free of atheromatous lesions.

MANAGEMENT

We decided to use an intensive antithrombotic regimen with a combination of dual-antiplatelet therapy (aspirin 75 mg/d, ticagrelor 90 mg twice a day), low-molecular weight heparins (enoxaparin 6,000 international units twice a day), and glycoprotein IIb/IIIa receptor inhibitors (tirofiban IV based on weight). Aspiration thrombectomy using a 7-F AP Export catheter (Medtronic) in LAD failed, and no thrombus was retrieved (Video 3). Balloon angioplasty of the distal LAD using a 2.5 × 12 mm mini Trek compliant balloon (mini TREK, Abbott) did not restore distal flow (Video 4). To avoid worsening distal embolizations, balloon angioplasty of the proximal LAD was not performed. The patient remained stable during the procedure. Due to similar culprit lesion characteristics and failed percutaneous coronary intervention on the LAD, we decided to treat the RCA conservatively.

The patient was monitored in our intensive care unit. Chest pain progressively resolved in a few hours but electrocardiogram remained unchanged. We did not find evidence of diabetes mellitus (HbA1C = 5.2%) nor hypercholesterolemia (low-density lipoprotein cholesterol = 1.22 g/L). Peak troponin I was 93 ng/mL (normal <0.16 ng/mL). His initial laboratory test results were as follow: hemoglobin 15.4 g/dL, white blood cells 11,200/mm^3, serum creatinine 64 μmol/L, C-reactive protein 4 mg/L, and fibrinogen 2.98 g/L.

ABBREVIATIONS AND ACRONYMS

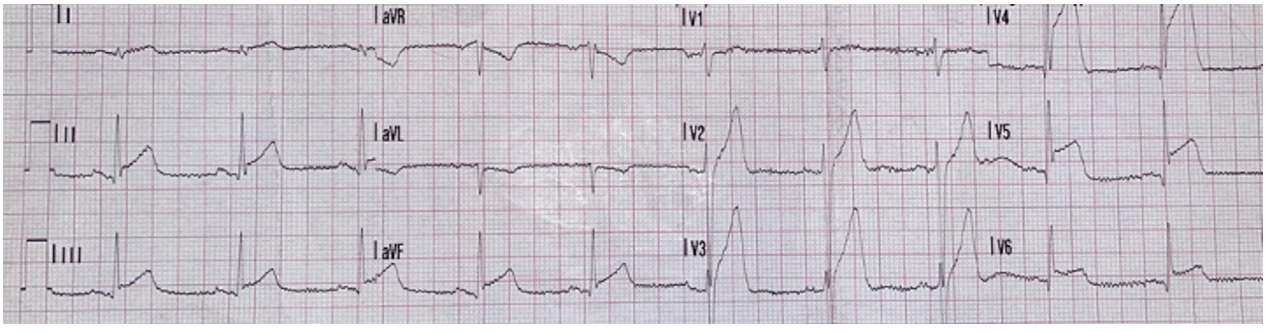
LAD = left anterior descending artery

OCT = optical coherence tomography

RCA = right coronary artery

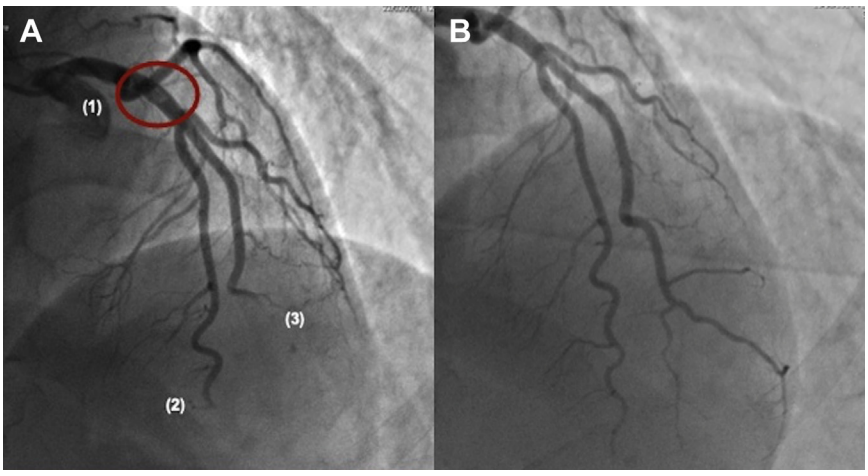
STEMI = ST-segment elevation myocardial infarction

FIGURE 1 Electrocardiogram at Presentation



Initial electrocardiogram shows an extensive anterior and inferior ST-segment elevation.

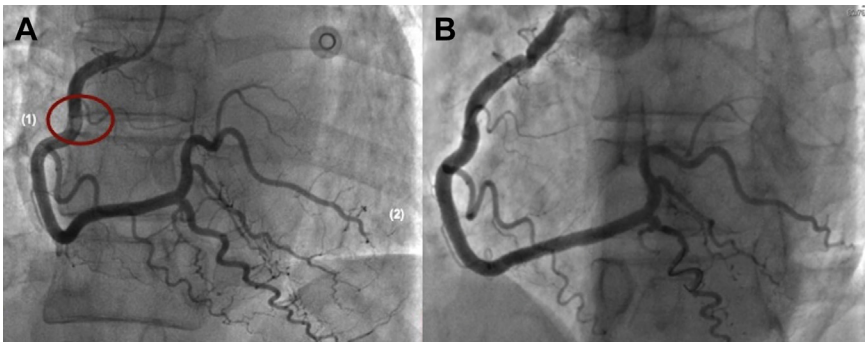
FIGURE 2 Left Coronary Artery



Left coronary artery before **(A)** and after **(B)** medical therapy. Initial left coronary angiogram: the **red circle** shows a large thrombus in the proximal left anterior descending artery (LAD) **(1)** with distal occlusions of the LAD **(2)** and the first diagonal branch **(3)**. Day 4 left coronary angiogram shows complete resolution of proximal LAD thrombus and restored flow in distal artery.

After 48 hours of glycoprotein IIb/IIIa receptor inhibitors and 4 days of low-molecular weight heparin, we performed a new coronary angiogram with optical coherence tomography (OCT) (Dragonfly, Abbott) to understand the putative mechanism. The 2 initial large thrombi were not visualized anymore without any evidence of distal emboli in the 2 involved coronary arteries (**Figures 2B and 3B**, Videos 5 and 6). OCT performed in both proximal LAD and mid-RCA identified plaques erosions with residual non-occlusive thrombi adhered to a thickened intima. There was no evidence of dissection or hematoma (**Figures 4 and 5**, Videos 7 and 8). We adopted a medically based strategy consisting of a dual-antiplatelet therapy (aspirin 75 mg/d and ticagrelor 90 mg/twice a day for 12 months), beta-blockers, angiotensin-converting enzyme inhibitors, proton pump inhibitors, high-intensity statin therapy, and

FIGURE 3 Right Coronary Artery



Right coronary artery before **(A)** and after **(B)** medical therapy. Initial right coronary angiogram: the **red circle** shows a large thrombus in the mid-right coronary artery (RCA) **(1)** with distal occlusion of a posterolateral branch **(2)**. Day 4 right coronary angiogram shows complete resolution of mid-RCA thrombus and restored flow in posterolateral branch.

nicotine replacement therapy. The patient was discharged at day 5, with decreasing troponin, normal kidney function, and no hemorrhagic complications.

DISCUSSION

Acute STEMI usually results from atheromatous plaque rupture or erosion with a subsequent thrombotic occlusion of 1 single coronary artery (1). In few cases, multiple thrombotic occlusions can occur in more than 1 major coronary artery. A 2-center experience suggests that their prevalence is estimated to be 2.5% (2). However, it must be pointed out that, due to selection bias associated with catastrophic outcomes, their real incidence is probably significantly underestimated (3,4).

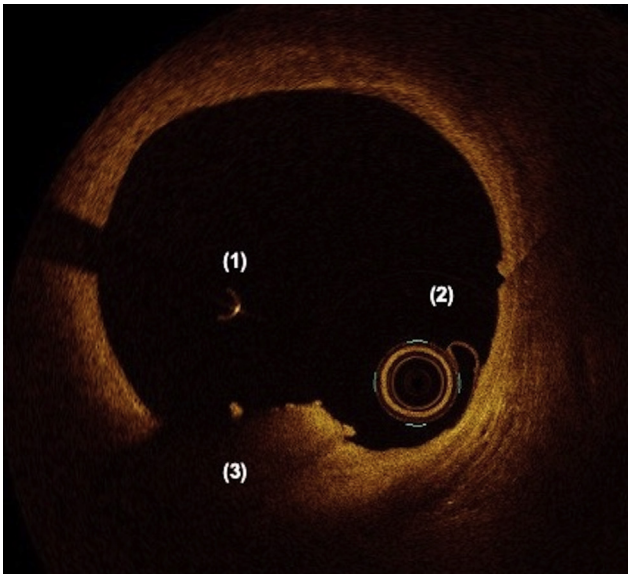
As in our case, a systematic review found that the simultaneous thrombosis of the LAD and RCA was the most common angiographic finding (3). Our first strategy was a balloon angioplasty on the distal arteries to restore a normal coronary flow. We also decided to perform an aspiration thrombectomy on the proximal LAD culprit lesion. Both attempts failed. Due to a high risk of worsening thrombus embolization, we decided to treat conservatively.

There are many reasons for multiple acute coronary thrombosis: plaque rupture, erosion, spontaneous coronary artery dissection, prolonged vasospasm, cocaine abuse, hypercoagulable states, or cardiac embolization. In our case, we could not determine the definite mechanism on the initial angiogram. Moreover, an extensive cardiac work-up did not find any identifiable cause of cardiac embolization. To understand the underlying mechanism, intracoronary imaging was performed.

Due to its 10 times greater resolution, we thought that OCT was more suited than intravascular ultrasound to detect thrombus and to characterize the intima (5). However, OCT timing is challenging. With major thrombus on the initial angiogram, we preferred to postpone OCT imaging for a few days after aggressive antithrombotic therapy to limit the risk of adverse events such as thrombus embolization or no reflow while manipulating the OCT lens.

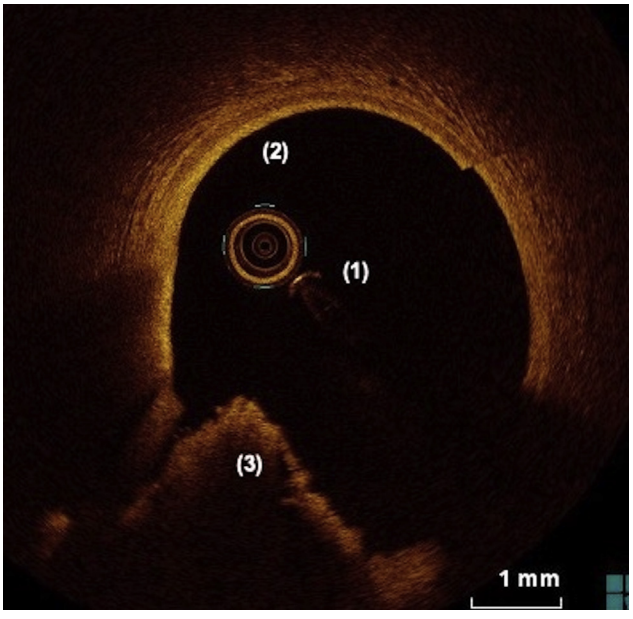
OCT identified plaques erosions with small pedunculated thrombi adherent to the vessel wall in both culprit lesions. The patient had clinical and laboratory parameters favoring this underlying mechanism (6). OCT confirmed the diagnosis (7) and was crucial to determine the best treatment strategy by avoiding unnecessary stenting. With residual diameter stenosis <70% in the setting of plaque erosion, conservative treatment may be a safe option (8), with good long-term prognosis (9).

FIGURE 4 OCT Imaging of LAD



Optical coherence tomography (OCT) imaging shows the proximal left anterior descending artery (LAD) lumen with the guidewire **(1)**, the OCT lens **(2)** and a plaque erosion with nonocclusive thrombus adherent to the vessel wall **(3)**.

FIGURE 5 OCT Imaging of RCA



OCT imaging shows the mid-RCA lumen with the guidewire **(1)**, the OCT lens **(2)**, and a plaque erosion with nonocclusive thrombus adherent to the vessel wall **(3)**. Abbreviations as in Figures 3 and 4.

JACC: CASE REPORTS, VOL. 3, NO. 18, 2021
DECEMBER 15, 2021:1906–1910

FOLLOW-UP

At 3 months follow-up, the patient was in a healthy condition without any ischemic recurrence.

CONCLUSIONS

Simultaneous thrombotic occlusions of 2 major coronary arteries is rare in patients with STEMI. Ruling out thromboembolic causes is crucial. Diagnostic confirmation usually requires intravascular imaging to identify the underlying mechanisms and guide treatment decisions.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS coronary angiography, imaging, myocardial infarction

APPENDIX For supplemental videos, please see the online version of this paper.