Acute Myocardial Infarction with Simultaneous Thrombosis of Multiple Coronary Arteries

Tae Hoon Yim, Jee Seon Kim, Byung Chul Kim, Hak Ro Kim, Tae Jin Kim, Young Bok Kim
Department of Internal Medicine, Pohang Sunlin Hospital, Pohang, Korea

Simultaneous thrombosis of multiple coronary arteries in acute myocardial infarction is very rare in clinical settings. Its mechanism is not yet clear, but patients displaying multivessel simultaneous thrombosis tend to have poor clinical outcomes. Hence, it is important to recognize this condition and provide timely and proper management. We report a case of simultaneous thrombosis involving multiple coronary arteries in a patient with ST-segment elevation myocardial infarction.

**Keywords:** Simultaneous; Coronary occlusion; Myocardial infarction

**INTRODUCTION**

Acute myocardial infarction (AMI) simultaneously involving multiple coronary arteries is rare [1,2]. Many patients with multivessel thrombosis in AMI are clinically ill and have a poor prognosis, including death [3]. Therefore, it is very important to recognize this condition and provide prompt and appropriate management for patients. We report a case of documented multivessel thrombosis in ST-segment elevation myocardial infarction (STEMI).

**CASE REPORT**

A 41-year-old male came to our emergency room (ER) with tight anterior chest pain lasting for several hours. His vital signs showed his blood pressure to be 100/70 mmHg, with a heart rate of 80/min and a respiration rate 26/min. The oxygen saturation in the room air was 100%. The rest of the physical examination, including the breathing sound and chest X-ray, was unremarkable. Electrocardiography (ECG) revealed ST-segment elevation in the inferior leads (II, III, and aVF) and anterior leads (V3-V5), and ST-segment depression in the lateral leads (I and aVL) (Fig. 1). Findings detected in portable echocardiography in the ER were severe left ventricular (LV) systolic dysfunction with left ventricular ejection fraction (LVEF) of 25% to 35% and akinesia of left anterior descending artery (LAD) territory. Initial laboratory tests showed: troponin-I 10.23 ng/mL, creatinine kinase (CK)-myocardial band (MB) isoenzyme 4.90 ng/mL, CK 164 IU/L, glucose 274 mg/dL, aspartate aminotransferase 64 IU/L, alanine aminotransferase 74 IU/L, cholesterol 210 mg/dL, low density lipoprotein cholesterol 136.6 mg/dL, high density lipoprotein cholesterol 46 mg/dL, and white blood cell 15.2 × 10^3/μL. He has had hypertension and type 2 diabetes mellitus and has been smoking half a pack of cigarettes per day for 20 years.

We moved him to a coronary angiography (CAG) room for intervention, just after seeing his ECG and portable echocardiographic findings. When arriving at the CAG room, he collapsed with dropped blood pressure at 80/50 mmHg, and his consciousness changed to a drowsy state. We began resuscitation with intravenous dopamine at a rate of 2.2 μg/kg/min, and then performed his coronary angiogram. Its findings were multiple coronary artery thrombotic occlusions; total occlusions of the proximal LAD, right coronary artery (RCA), and left circumplex artery (LCX) (Fig. 2A-C). In addition, we could not find any collateral flow to LAD, RCA, and LCX territories. Fortunately, the flow to the ramus intermedius artery (RI) and obtuse marginal artery (OM) were preserved with thrombosis in myocardial infarction (TIMI) flow 2. We first performed the intervention on the proximal LAD which we thought...
was the culprit vessel for this STEMI. The LAD lesion was passed smoothly with a 0.03556 cm guidewire (Runthrough; Terumo, Tokyo, Japan). A Xience prime stent (Abbott Vascular, Sana Clara, CA, USA) 3.5 × 33 mm was deployed after ballooning, resulting in TIMI flow 3 and weak collateral flow from LAD to LCX territory (Fig. 3A). However, his chest pain was becoming aggravated, and his blood pressure (BP) dropped to 60/50 mmHg, despite the successful coronary angioplasty on the proximal LAD. Immediately, we were able to maintain systolic BP above 90 mmHg with volume infusion and intracoronary epinephrine injection. We moved to the second intervention of the proximal RCA. Wire-passing using 0.03556 cm guidewire (Runthrough, Terumo, Tokyo, Japan) to the lesion at RCA was also smooth. Another Xience prime stent (Abbott Vascular) 3.5 × 38 mm was well positioned after ballooning. After successful intervention on the proximal RCA, the flow of RCA was good (TIMI flow 3) and the patient’s chest pain was relieved (Fig. 3B). Furthermore, we could not find any collateral flow from the RCA to LCX territory.

We also tried to perform the third intervention on the proximal LCX using 0.03556 cm guidewire (Runthrough, Terumo), but we failed to pass through the lesion (Fig. 3C). Moreover, the BP of the patient was unstable, dropping again to 80/60 mmHg with hemodynamic support, including inotropics and intravenous volume infusion. We decided to defer the intervention on the proximal LCX and moved the patient to the intensive care unit.

The day after the intervention, the echocardiography revealed
LV systolic dysfunction with EF of 28%, akinesia of inferior and septal walls, including apex, and hypokinesia of the anterior wall from base to mid LV. The serial cardiac enzyme reached the peak level, troponin-I 146.74 ng/mL and CK-MB isoenzyme 530.75 ng/mL, and then began to decrease. He was discharged after an in-hospital treatment of 6 days. The EKG before discharge showed ST-segment resolution (Fig. 4). He has visited our out-patient clinic regularly without any symptoms for several months.

**DISCUSSION**

Multiple coronary thromboses involving two or more epicardial coronary vessels in previously normal coronary arteries are very rare [1,2]. In pathological studies, multiple coronary thromboses were found in about 10% of patients who died from AMI [4]. The patients with multiple coronary artery thromboses tend to have more severe clinical outcomes, including cardiogenic shock and...
Pathophysiologic conditions causing multiple coronary thromboses are unclear. However, several possible mechanisms for multiple coronary artery thromboses have been suggested, including the thrombus due to multivessel spasm [7], the state of hypercoagulability [8], the decreased blood flow to other coronary arteries after AMI [2], and diabetes mellitus (DM) [9]. Smoking and alcohol are also related to increased risks of coronary artery thrombosis by accelerating platelet aggregation and adhesion and by impairing endothelial dependent vasodilation [10]. In our case, the patient had a risk factor of coronary artery disease, including type 2 DM, hypertension, and smoking, which might be involved in the development of acute simultaneous occlusion of multiple coronary arteries by causing a hypercoagulable state.

We speculate that both LAD and RCA total occlusions were caused by simultaneous thrombosis of coronary arteries because there was no collateral circulation from other vessels to LAD and RCA territories, and both LAD and RCA lesions were passed easily by guide wire. However, LCX lesions might be considered as CTO, as it had the collateral flow from LAD to LCX territories after intervention on the proximal LAD, and it was too firm for the guide wire to pass through, despite several attempts.

In conclusion, AMI involving multiple coronary arteries is a very rare but life-threatening condition. Moreover, it is not easy to differentiate multivessel coronary thrombosis from AMI in the ER. If we recognize such a condition and provide timely and appropriate management, we might exert a good influence of the prognosis of patients with multivessel coronary artery thrombosis.

REFERENCES