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Simultaneous double coronary thrombosis in a 47-year-old male patient with acute 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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Patient: Male, 47
Final Diagnosis: Acute myocardial infarction
Symptoms: Chest pain
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course

Background: Double myocardial infarction involving two culprit major vessels is a rarely reported presentation with high incidence of mortality.

Case Report: In this study, we report 47-year-old male patient who had an attack of chest pain associated with ST-segment elevation in the antero-lateral leads. Pharmaco-invasive reperfusion approach was adopted with full dose tissue plasminogen activator, followed by transferring the patient to a specialized heart center for Percutaneous Coronary Intervention (PCI). Coronary angiography showed a fresh thrombus totally occluding Left Anterior Descending (LAD) and another thrombus causing distal total occlusion of a dominant Right Coronary Artery (RCA). Two Bare metal stents were placed in both lesions with Thrombolysis in Myocardial Infarction (TIMI) 3 flow post dilatation, but the patient, unfortunately, went into Ventricular Fibrillation (VF) followed by asystole and died 35 minutes later.

Conclusions: Acute double vessel coronary artery thrombosis is a serious event that requires prompt diagnosis and management to prevent its complications.

Key words: multiple plaques rupture • double coronary thrombosis • Coronary occlusion

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Background

Acute ST-segment elevation myocardial infarction (MI) usually presents by an occluding thrombus in 1 artery that is the "culprit" lesion, but on rare occasions this culprit lesion can be found in more than 1 artery. This is extremely rare, and is estimated to occur in 2.5% of all primary PCI patients [1]. Most of these patients have evidence of hemodynamic instability, with 28% being in cardiogenic shock, 22% having life-threatening ventricular arrhythmias, and 22% requiring intra-aortic balloon pumps [1]. One of the postulated reasons for such low incidence is that most of the patients die from sudden cardiac death (SCD) before coming to the hospital, and the incidence is much higher in autopsies of sudden cardiac death patients, reaching up to 50% of cases [2]. Although the cause of simultaneous acute thrombosis of multiple coronary arteries is still unclear, many theories are postulated, including the presence of a hyper coagulable state or increased catecholamine surge and inflammatory response caused by the occlusion of the first artery causing thrombosis of the other [1]. In this study we present a rare case of double coronary thrombosis myocardial infarction and discuss our management of the case, the possible causes of double infarction, and the outcomes of this high-risk presentation.

Case Report

Our patient was a 47-year-old man of Indian origin, who was diabetic, a heavy smoker, and without family or previous history of coronary artery disease (CAD). He reached the emergency 2 hours after feeling a severe crushing retro-sternal chest pain associated with diaphoresis. At the emergency department, the patient was hemodynamically stable, with blood pressure of 139/103 mmHg, equal in both arms, pulse of 125 beats per minute (bpm) and SpO2 of 98% on room air. His chest was clear to auscultation and heart examination results were normal. Electrocardiograph ECG showed extensive antero-lateral ST-segment elevation myocardial infarction (Figure 1). Because the anticipated time from first medical contact to PCI (percutaneous coronary intervention) was >120 min, a pharmaco-invasive strategy was adopted, and the patient received aspirin 300 mg, clopidogrel 300 mg, and heparin 4000 units IV bolus stat, followed by continuous IV infusion of heparin by 1000 unit/hour. Tissue plasminogen activator (TPA), reteplase administered IV stat in 2 doses (10 units each), separated by a 30 min interval according to the protocol. High sensitive troponin T results were positive (105 ng/L and normal level 3–14 ng/L). No thrombocytosis was found. Just prior to the transfer of the patient to the specialized heart center, his general condition began to deteriorate, requiring elective intubation for impending respiratory failure. The patient was transported from the emergency department to a PCI-capable facility in

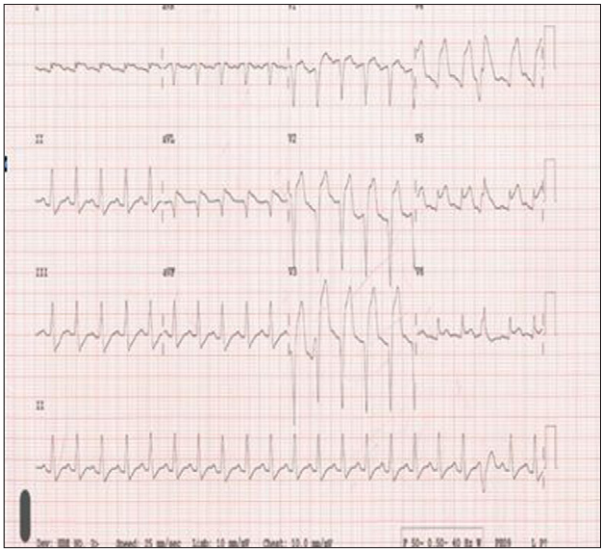

Figure 1. An ECG of the patient (on arrival to the emergency) showing ST-segment elevation in all precordial leads (V1 through V6), aVL and I, with ST depression in inferior leads, indicating extensive anterior MI.

60 min. The patient's coronary angiography showed total occlusion by a fresh thrombus to both proximal LAD (Figure 2A) and distal RCA (Figure 2C). The LAD lesion was crossed using a guide wire, with achievement of TIMI 3 flow immediately after passing it (Figure 2B). This was followed by deployment of a bare metal stent (BMS), with good angiographic results (Figure 3A). Attention was shifted then to RCA, and the distal lesion was crossed using another guide wire and TIMI 3 flow was established (Figure 2D). This was followed by balloon dilatation, then a BMS was deployed, with TIMI 3 flow post-deployment (Figure 3B). Unfortunately, immediately after the procedure, the patient developed VF and received 1 direct current shock, followed by cardio-pulmonary resuscitation (CPR), which lasted for 35 minutes, but the patient died.

Discussion

Acute myocardial infarction caused by simultaneous multi-vessel coronary occlusion is a rarely reported event that needs immediate management owing to the high rate of complications such as arrhythmia, heart failure, and cardiogenic shock. Incidence of double-vessel coronary thrombosis may account for 1.7% to 4.8% of all primary PCIs [1] and it is believed that this incidence is far below the actual value. The incidence of occlusion in more than 1 epicardial artery reached up to 50% of an autopsy series of patients who died from SCD [2]. Thus, such low incidence reflects a selection bias because most of the patients die before reaching the hospital. The study by Pollak et al. [1], despite its limitations, has provided some information about the critical nature of this pathology, reporting

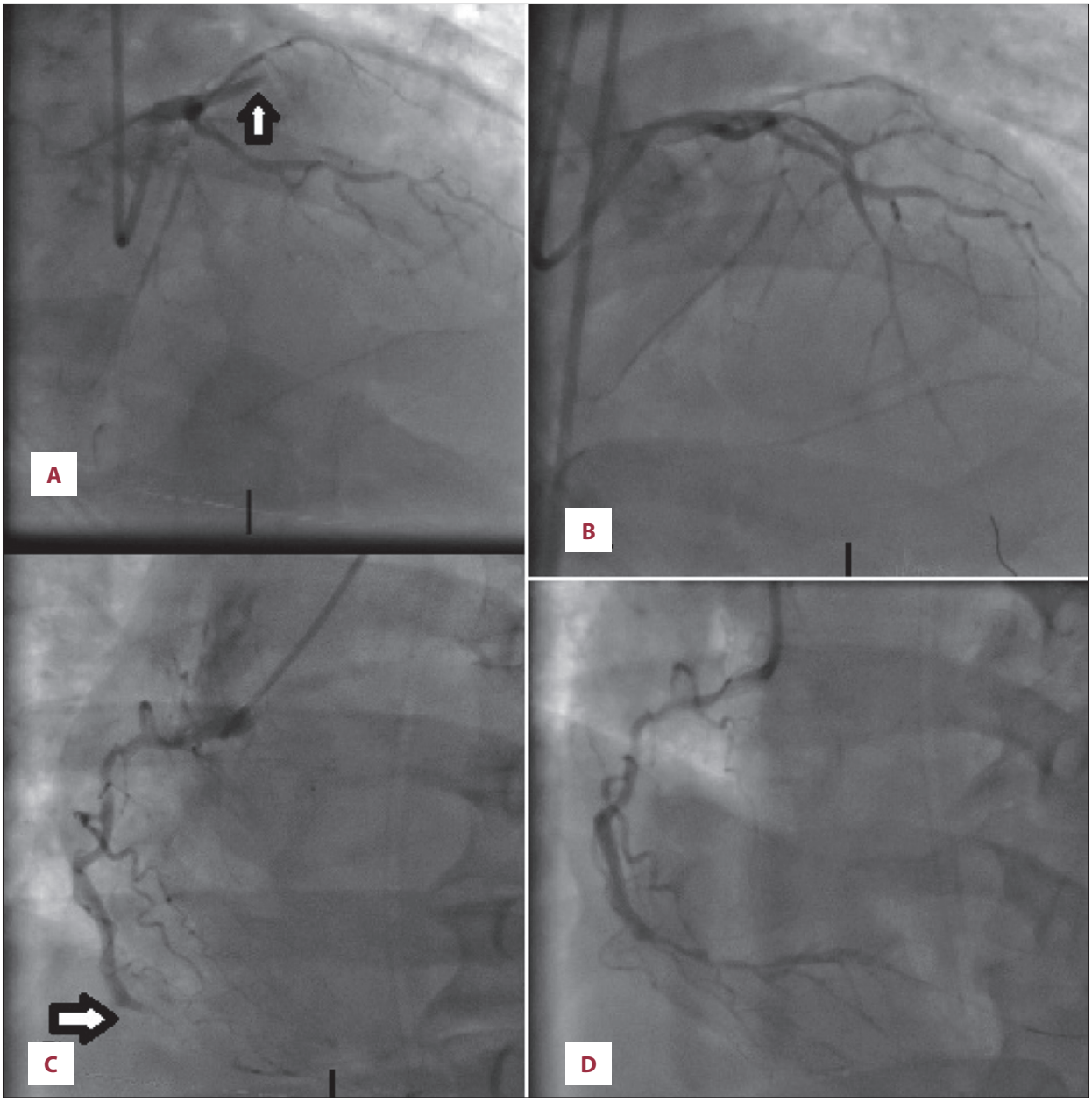

Figure 2. (**A**) LAD injection showing total occlusion of proximal part by a fresh thrombus (arrow). (**B**) LAD injection after passing the BMW wire. (**C**) RCA injection showing total occlusion of distal part by a fresh thrombus (arrow). (**D**) RCA injection after passing the BMW wire.

that nearly one-third of patients are presented in cardiogenic shock, and nearly one-forth required intra-aortic balloon pump or have a life-threatening arrhythmia. It appears that multivessel coronary thrombosis trends to be more common in heavily smoking males with dyslipidemia [1,3], such as this patient. Pollak et al. [1] found that the percentage of males was 85% and history of current tobacco use reached 49%; the percentage of diabetics was 21%; history of previous CAD, MI or PCI, was 15% (indicating that most patients had first-diagnosis ST elevation MI); and the mean age was 54 (±14). Most of these characteristics were found in our patient. Pollak et al also reported that the most common ECG finding is inferior ST elevation (30%), followed by inferior and anterior ST elevation (19%), then anterior and lateral ST elevation (15%), as in our patient [1]. Angiographically, RCA and LAD acute occlusion (49%) was the commonest presentation (as in our patient), followed by RCA and left circumflex (28%) [1]. There were several factors we had to consider regarding the management of our patient. The first was which strategy we should use for reperfusion. We had 3 options: to start thrombolysis by tissue

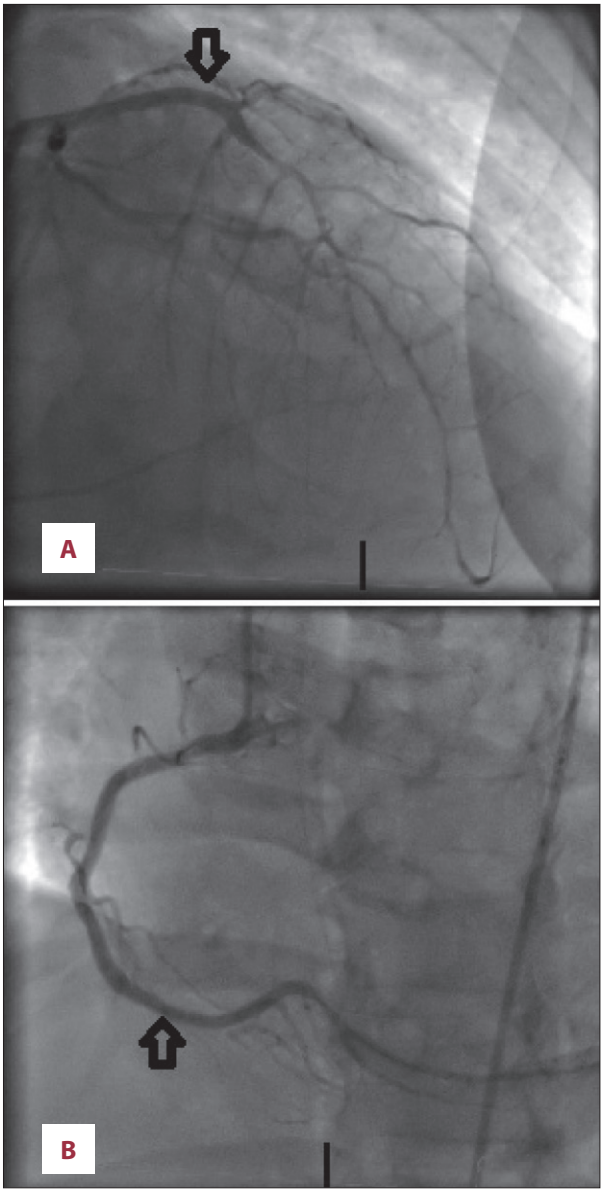

Figure 3. (**A**) LAD injection post-stenting showing a diffusely diseased LAD with TIMI III flow. The stent is illustrated by the arrow. (**B**) RCA injection post-stenting showing adequate TIMI III flow. The stent is illustrated by the arrow.

plasminogen activator (tPA) alone, to transfer the patient to a specialized heart center for primary PCI, or to give tPA then transfer for PCI as soon as possible (the "pharmaco-invasive" approach). The patient was had been experiencing chest pain for only 2 hours and his ECG result and clinical picture were suggestive of extensive anterior MI with left ventricular failure. We also anticipated that the time from first medical contact (FMC) to PCI would take >120 minutes. Thus, we started thrombolysis, as recommended by the current European Society of Cardiology (ESC) and American Heart Association (AHA) guidelines (class IA) [4,5], followed by the transfer of the patient to the catheterization laboratory as soon as possible to do PCI (Pharmaco-invasive reperfusion). This approach was shown to reduce mortality and morbidity 30 days post-MI [6]. The second consideration was whether we should place an intra-aortic balloon pump (IABP) before starting the PCI. It was obvious that the patient had an extensive myocardial infarction with total double-vessel acute occlusion, and we saw that advantage of starting the intervention to achieve a swift reperfusion to the ischemic myocardium, which outweighed the benefit of placing a pump that would require a new arterial access and would further delay the PCI. This was supported by the evidence of several studies that showed the benefit of PCI diminishes with time, with increased 30-day mortality [7,8]. On the other hand, the benefit of IABP is a matter of debate, with many trials showing contradictory evidence and even no mortality benefit [9]. Thus, our decision was to begin with the PCI followed by insertion of an IABP soon after the procedure. Unfortunately, the patient went into VF followed by asystole and died before pump placement.

The etiology of multivessel coronary thrombosis is still not fully understood. Many of case reports published in the literature have obvious predisposing factors such as coronary artery vasospasm [10,11], idiopathic thrombocytopenic purpura [12], antithrombin III deficiency [13] and thrombophilia [14,15]. Other cases were reported following cocaine abuse [16] and during puerperium [17]. In a few other cases, the cause was unclear [18,19]. We believe that the pathogenesis of multiple coronary occlusive lesions may be explained by the simultaneous rupture of multiple plaques during the progression of myocardial infarction. Rioufol et al. [20] reported that 19 acute coronary syndrome patients out of 50 had ruptured plaques in more than 1 epicardial coronary artery, diagnosed by intravascular ultrasound (IVUS). However, other studies illustrated that plaque rupture can also be found in chronic stable angina patients or even asymptomatic patients [21,22], and the clinical picture depends on other variables such as thrombus formation and lumen size [24]. In consideration of the above factors, it appears that a high thrombus burden and/or a small lumen area are necessary to have multiple occlusive lesions causing ST-elevation myocardial infarction and simultaneously ruptured plaques.

Conclusions

Acute double-vessel coronary artery thrombosis is a serious event that requires prompt diagnosis and management to prevent complications. Further study on acute double coronary vessel thrombosis is required to clarify the underlying mechanisms and outcomes.

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