Double Vessel Acute Myocardial Infarction Showing Simultaneous Total Occlusion of Left Anterior Descending Artery and Right Coronary Artery

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Simultaneous double vessel acute myocardial infarction (AMI) is extremely rare and usually has poor clinical outcomes. Management of this complicated condition is challenging and time-limited. The case of a 46-year-old Taiwanese man with simultaneous anterior and inferior wall AMI is reported. Rapid deterioration of clinical condition with ventricular fibrillations (VF), cardiogenic shock and asystole developed before catheterization. Coronary angiogram revealed simultaneous total occlusion of left anterior descending (LAD) and right coronary arteries (RCA). Frequent VF attack was still noted after diagnostic catheterization. After cardiopulmonary resuscitation, immediate percutaneous coronary intervention of the LAD and RCA, and intra-aortic balloon counterpulsation was inserted. Due to intractable heart failure and cardiogenic shock, extracorporeal membrane oxygenation was performed. Rhabdomyolysis with acute renal failure was also noted with hemodialysis treatment. Thirty-one days after hospitalization, he was discharged with a New York Heart Association functional class III heart failure, without hemodialysis. (Circ J 2008; 72: 1034–1036)

Key Words: Double vessel myocardial infarction; Extracorporeal membrane oxygenation; Intra-aortic balloon counterpulsation; Percutaneous coronary intervention

A 46-year-old Taiwanese man was admitted with simultaneous acute anterior and inferior myocardial infarction. His risk factors for coronary artery disease were smoking and being a male older than 40 years of age. A 12-lead surface electrocardiogram (ECG) at a clinic showed acute inferolateral myocardial infarction (Fig 1A). About 30 min later, he was referred to our hospital. His blood pressure, pulse rate and respiratory rate were 95/64 mmHg, 110 beats/min and 16 breaths/min, respectively, when he was referred to our emergency department. A chest X-ray revealed bilateral interstitial pulmonary edema. A 12-lead surface ECG showed simultaneous acute inferior and antero-lateral myocardial infarction (Fig 1B) and a monitor ECG showed 2nd degree Mobitz type I atrio-ventricular block (Fig 1C). Right-side ECG revealed no right ventricular infarction. Laboratory data showed troponin-I 0.843 ng/ml (AMI cutoff value: 0.5 ng/ml), creatine kinase (CK) 2.014 IU/L, CK-MB isoenzyme 104 IU/L and serum creatinine 1.2 mg/dl. While waiting for emergent percutaneous coronary intervention (PCI), aspirin, heparin and glycoprotein IIb/IIIa receptor antagonist tirofiban were given as the primary PCI protocol. VF and then asystole were found in our catheterization room before diagnostic catheterization. After endotracheal intubation and cardiopulmonary resuscitation (CPR), coronary angiograms revealed total occlusion of the middle LAD artery below the septal branch, total occlusion of the distal RCA (Figs 2A, B) and insignificant stenosis of the non-dominant left circumflex artery. Frequent VF attack was still noted after diagnostic catheterization. The LAD lesion was soon and easily passed through using a 0.014”LS floppy guidewire (Boston). After balloon angioplasty, thrombus was found and Thrombolysis In Myocardial Infarction (TIMI) grade II flow was noted. After stenting for LAD, TIMI grade III flow was observed and no more VF attacks were discovered, but shock and sinus bradycardia with a heart beat 50 beats/min were still noted. A temporary transvenous pacemaker was then inserted. No collateral circulation from the LAD to RCA was noted after PCI for LAD. Thereafter, the RCA lesion was also soon and easily passed through using a 0.014”LS floppy guidewire (Boston) and a 2.5×20 mm Sprinter balloon catheter (Metronic) was inflated up to 12 atm, and then a 2.75×24 mm Micro-Driver stent (Metronic) was deployed smoothly. TIMI grade III flow of the LAD and RCA were observed after PCI (Figs 2C, D). The blood pressure was 90/60 mmHg after PCI, when an intra-aortic balloon counterpulsation (IABP) device was inserted.
The patient regained consciousness after CPR and PCI. Follow-up echocardiography showed hypokinesis over the apical septum, mid-anterior wall, apical inferior wall and inferior wall with a left ventricular ejection fraction of 32%. Despite judicious medical treatment and the insertion of the IABP device, shock and refractory pulmonary edema were still noted and extracorporeal membrane oxygenation (ECMO) device was inserted thereafter. Rhabdomyolysis, acute renal failure with serum creatinine level up to 8.4 mg/dl and bacteremia occurred during hospitalization, and were treated adequately. Hemodialysis was performed for acute renal failure. The patient was weaned-off the ECMO and IABP devices in a stepwise manner and then hemodialysis was discontinued. Thirty-one days later, he was discharged with a New York Heart Association functional III heart failure status and serum creatinine 3.3 mg/dl.

**Discussion**

Atheromatous plaque rupture with subsequent thrombus formation and then vessel occlusion is the most common cause of AMI, but myocardial infarction may also occur in other rare conditions without atherosclerosis. AMI with simultaneous total occlusion of 2 or 3 coronary arteries is extremely rare, and usually with a poor prognosis, including mortality. Coronary artery dissection with extension to the non-culprit vessel, and total occlusion of the culprit coronary artery with impaired coronary blood flow and subsequent thrombus formation (stagnation thrombosis) in non-culprit coronary artery were reported for developing multiple vessel myocardial infarction. Multiple ruptured plaques with thrombus formation occurs frequently in patients with acute coronary syndrome, and it has been reported in more than 10% of autopsied cases, but it usually is not recognized clinically because of its rapid and fatal course. The cause for our case presented here is most likely due to the rupture of multiple plaques. Initial ECG at a clinic showed acute infero-lateral myocardial infarction. So, the ECG at our hospital should be interpreted as acute inferior and antero-lateral myocardial infarction. Because the septal branch of...
LAD was spared, no ST elevation was noted in V1, V2. Furthermore, the LAD lesion was soft with thrombus and the RCA lesion was also soft and easily passed using a very soft guidewire. After PCI for LAD, no collateral circulation from LAD to RCA was found. So, the RCA lesion was not a chronic total occlusion lesion and we are sure this is a case of simultaneous acute anterior and inferior myocardial infarction.

The mortality rate of cardiogenic shock complicating AMI remains very high, up to 70%, despite aggressive treatment with emergency PCI and use of IABP. Applications of mechanical support devices, such as ECMO and left ventricular assist device, have revolutionized treatment and improved the outcome of complicated AMI. Theoretical first, we should insert IABP first before PCI. Due to frequent VF and CPR, we had no time to prepare IABP on this occasion. Because we thought that PCI for LAD could be easily and quickly accomplished, we chose PCI for LAD first. In this patient, we just used IABP after PCI, and then followed with ECMO due to persistent cardiogenic shock and pulmonary edema thereafter. Reviewing the whole course of hospitalization, we thought that IABP should be inserted before PCI if possible and earlier ECMO insertion maybe needed immediately after PCI due to the high probability of intractable heart failure after multi-vessel myocardial infarctions.

Conclusion

Multi-vessel myocardial infarctions are associated with high mortality and result in complicated hospitalization courses. A combination therapy of judicious medical treatment, efficient primary PCI and early mechanical support devices, especially EMCO, is crucial to improving the survival rate of this high mortality rate disease.

References