ACUTE MYOCARDIAL INFARCTION WITH SIMULTANEOUS INVOLVEMENT OF RIGHT CORONARY ARTERY AND LEFT ANTERIOR DESCENDING ARTERY: A CASE REPORT

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Acute myocardial infarction is usually caused by rupture of unstable plaque and involves a single coronary artery. Simultaneous occlusions of multiple coronary arteries in patients with ST elevation myocardial infarction are uncommon and lead to a fatal outcome. We report a 75-year-old male presenting with persistent chest pain complicated by ventricular fibrillation. After defibrillation and cardiopulmonary resuscitation, an emergency coronary angiogram showed total occlusion of the right coronary artery, and thrombus in the proximal left anterior descending artery. Both coronary arteries underwent successful balloon inflation and stenting. The patient finally survived under ventilatory support. This rare case suggests that aggressive reperfusion therapy and even mechanical support to improve poor clinical outcome are suggested in high risk patients with multivessel occlusions.

Key Words: acute myocardial infarction, coronary occlusion, percutaneous coronary intervention

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CASE PRESENTATION

A 75-year-old man presented with persistent chest pain for 2 hours and came to our emergency department. He was a heavy smoker, with a past medical history of hypertension and gouty arthritis. He had no history of a hypercoagulation disorder including autoimmune and hematologic disease. On arrival, his blood pressure was 105/65 mmHg, and heart rate was 67 beats/min. Immediately, consciousness was lost and the ECG monitor showed ventricular fibrillation. He received advanced cardiopulmonary resuscitation and defibrillation before restoration of spontaneous circulation. The post-resuscitation ECG revealed a bizarre wide QRS complex initially (Figure 1A) and serial follow-up ECG after 1 hour showed ST elevation over leads III and aVF, and pathologic Q waves in leads II, III...
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and aVF (Figure 1B). The laboratory studies revealed a white cell count of 12.3 × 10^9/L, platelet count of 177 × 10^9/L, creatine kinase (CK) of 2.3 μkat/L, creatinine kinase MB (CK-MB) of 0.15 μkat/L (CK-MB/CK ratio = 6.4%), troponin-I of 95 ng/L, total cholesterol of 2.85 mmol/L, and low density lipoprotein of 1.63 mmol/L. Aspirin and clopidogrel were given and heparin was used for anticoagulation. Follow-up data of cardiac enzymes revealed CK of 44.71 μkat/L, CK-MB of 6.05 μkat/L (CK-MB/CK ratio = 13.5%), and troponin-I of 3,818 ng/L. An emergency coronary angiogram demonstrated total occlusion of the RCA (Figure 2A) and a thrombus in the proximal LAD (Figure 2B). A glycoprotein IIb/IIIa inhibitor was then given for the large thrombus burden. Initially, we tried to manage the total occlusion of the RCA, with the lesion easily located followed by successful balloon expansion and insertion of a bare metal stent (Figure 2C). Then the LAD lesion also successfully underwent balloon inflation and stenting (Figure 2D). Thrombolysis in Myocardial Infarction III flow of the LAD and RCA were observed after percutaneous coronary intervention. During the procedure, intra-aortic balloon counterpulsation (IABP) was applied for low blood pressure. Left heart ventriculography showed global hypokinesia, and left ventricular ejection fraction was only 29%. After percutaneous coronary intervention, the patient was transferred to our coronary care unit for further intensive care.

In the coronary care unit, the patient gradually regained consciousness and his hemodynamics became more stable. Therefore, we removed the vasopressor and the IABP. He was treated initially with dual anti-platelet therapy, and then an angiotensin-converting

Figure 1. (A) Post-resuscitation electrocardiogram revealed a bizarre wide QRS complex (suspect intraventricular conduction disturbance related to defibrillation and myocardial infarction). (B) One hour later, follow-up electrocardiogram showed ST elevation over leads III and aVF and pathologic Q waves in leads II, III and aVF.
enzyme inhibitor and a beta-blocker were added under stable hemodynamics. Twenty days later, he was transferred to the respiratory care center as he was dependent on ventilatory support.

**DISCUSSION**

Rupture of coronary artery plaque resulting in thrombotic occlusion is a common cause of AMI. However, simultaneous multivessel occlusions have been reported in the literature despite being uncommon. Multiple ruptured plaques with thrombus formation have been reported in more than 10% of autopsied cases. These cases were not usually recognized clinically because of their rapid course and high mortality [1]. In addition, AMI patients with multiple complex plaques are likely to have poor clinical outcomes [2].

Using coronary angiography, Goldstein et al [2] found additional unstable lesions other than a single culprit lesion in 21% of patients with AMI. They suggested that plaque instability is not merely a random “vascular accident”, but perhaps may reflect a “pan-coronary” process. Asakura et al [3] also reported that in patients with AMI, all three major coronary arteries were widely diseased and had multiple yellow plaques by angioscopic study. These findings suggest that AMI may represent the pan-coronary process of vulnerable plaque development.

Further evidence supporting a generalized process throughout the complete coronary tree is the presence of endothelial apoptosis. Valgimigli et al [4] found that serum from patients with acute coronary syndrome induced apoptosis of human umbilical vein endothelial cells, and Fas and Bcl-2 gene expression increased. However, the rate of apoptosis was markedly reduced and no longer different from that of healthy subjects after 1 year in stable conditions. They concluded that serum from patients with acute coronary syndromes displays a pro-apoptotic effect on human endothelial cells and contributes to the pathophysiological etiology of the pan-coronary syndrome.

According to limited past case reports, there are different causes of multivessel thrombosis, such as systemic thrombocytosis [5], intravenous cocaine abuse [6], coronary spasm [7], high catecholamine concentrations [8], and diabetes mellitus [9]. However, our case did not have these associations, and was an elderly...
man with hypertension and a history of heavy smoking. These factors might be the contributing causes in this case.

The 12-lead ECG is an important tool for the diagnosis and initial evaluation of patients with AMI. It enables physicians to localize the infarct-related coronary artery and optimize treatment. However, its use might be limited in patients with simultaneous coronary occlusions. In our case, the initial ECG after defibrillation revealed ST elevation over leads II, III, aVF, and V1-4, and intraventricular conduction disturbance was also suspected. These findings might be related to defibrillation and resuscitation. ECG follow-up 1 hour later revealed no further ST elevation over precordial leads, but was still noted over inferior leads with pathologic Q waves. Multivessel occlusions could not be easily recognized according to the follow-up ECG.

Aggressive reperfusion therapy and medical treatment are very important in cases with multivessel occlusions because the added complication of cardiogenic shock is common in these high risk patients. Our patient was treated aggressively with a glycoprotein IIb/IIIa inhibitor, dual anti-platelet therapy, and stenting in both the RCA and LAD. Vasopressors and IABP are also used for severe cardiogenic shock. An intravenous glycoprotein IIb/IIIa inhibitor is very useful in these situations in preventing further thrombus formation during percutaneous coronary intervention [10]. The strategies of intervention were also somewhat different in the limited case reports. In most reported cases with multivessel occlusions, both right and left coronary arteries were treated with stents [9–11]. However, Turgeman et al reported a case treated with balloon angioplasty and thrombus aspiration [12] and Hosokawa et al described a case treated with stent placement in the RCA and intracoronary thrombolysis for a thrombus in the LAD [13]. Because ventricular arrhythmia and cardiogenic shock usually occur in such high risk patients, further mechanical support with IABP or even an extracorporeal membrane oxygenation device is also necessary [12].

Our patient survived despite requiring ventilatory support. Several factors, such as old age, poor left ventricular systolic function, and pneumonia were thought to be related to ventilator dependency in our case. Early infection control and optimal medical treatment are important and might be helpful to decrease the rate of ventilator dependency.

To conclude, simultaneous multivessel occlusions in patients with AMI are uncommon in the literature. Because of their rapid and fatal course, these patients need aggressive reperfusion therapy and even mechanical support to improve the poor clinical prognosis.

REFERENCES

急性心肌梗塞同时涉及右冠状动脉与左前降冠状动脉－个案报告

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急性心肌梗塞通常起因於不穩定的血管斑塊破裂，而且多影響單一冠狀動脈。於 ST 段上升之心肌梗塞病患，同時發生多條冠狀動脈阻塞是不常見的，而且易導致致命性的預後。我們報告一位 75 歲男性病患，以持續胸痛作為表現且發生心室顫動。經過電擊與心肺復甦術後，緊急心導管呈現右冠狀動脈全部阻塞與左前降枝血栓形成。兩條冠狀動脈皆成功地被氣球擴張且放置了支架。病患最後存活下來儘管仍須呼吸器維持。這個罕見的個案提醒我們，在多條冠狀動脈阻塞的高危險病患，建議需要積極的再灌流治療甚至是機械性輔助來改善其不好的臨床預後。

關鍵詞：急性心肌梗塞，同時冠狀動脈阻塞，經皮冠狀動脈介入治療
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