Two Consecutive Episodes of Acute Myocardial Infarction Occurring in Different Coronary Arteries of a Single Patient with Sepsis

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Although recurrent ST-segment elevation myocardial infarction (STEMI) in the same coronary artery due to acute stent thrombosis has been reported in the literature, there have been no reported cases discussing consecutive STEMI recurring in different coronary arteries in the same patient in one day. Herein, we report an elderly male patient initially suffering from STEMI over the inferior wall who subsequently had another episode of STEMI over the anterior wall within several hours. Despite primary percutaneous coronary intervention being performed over both the right coronary artery and the left anterior descending artery, the patient eventually expired notwithstanding intensive care. This case should caution physicians that consecutive STEMI in different coronary arteries is an extremely rare but still possible medical phenomenon, and could lead to catastrophic clinical outcome.

Key Words: Myocardial infarction ● Primary percutaneous coronary intervention ● Recurrent ST-elevation

INTRODUCTION

Recurrent ST-segment elevation myocardial infarction (STEMI) due to acute stent thrombosis (ST) has been previously reported in the literature.[1,2] However, no literature has discussed consecutive STEMI recurring in different coronary arteries within one day. Herein we reported an elderly male patient who initially suffered from STEMI over the inferior wall, and subsequently had another episode of STEMI over the anterior wall within just a few hours. The most probable mechanism of rapid coronary thrombosis in a non-culprit vessel is progression of inflammatory process, which is related to urosepsis with disseminated intravascular coagulation (DIC), increased C-reactive protein (CRP) level, and ongoing shock status.

CASE REPORT

A 77-year-old male had suffered from acute chest pain and nausea in the morning and was brought to our emergency department (ED) for first aid. Tracing back the patient's history, he had hypertension controlled by regular medication with no history of smoking, dyslipidemia, or malignancy. On arrival at our ED, his pulse rate was 69 beats/min and blood pressure was 77/31 mmHg. Electrocardiogram (ECG) initially showed ST elevation over lead II, III, and aVF (Figure 1A). Dual antiplatelet (aspirin and clopidogrel) were loaded and vasopressor was given for shock status. Under the impression of STEMI over the inferior wall complicated with cardiogenic shock, primary percutaneous coronary in-

tervention (PCI) was arranged. The patient's right coronary angiogram showed subtotal occlusion over the proximal right coronary artery (RCA) (Figure 2A), and left coronary angiogram showed both left anterior descending artery (LAD) and left circumflex artery with 50% stenosis (Figure 2B). Then, coronary angioplasty with stenting was performed over RCA and TIMI 3 flow was restored (Figure 2C). Due to unstable hemodynamics of the patient, we gave intravenous fluid hydration continuously and a dopamine pump (under beta effect) was used to maintain blood pressure. The patient was then transferred back to our ED for further care, and waited for cardiac care unit (CCU) admission.

During the patient's ED stay, central venous pressure was inserted to evaluate the fluid status; the level of central venous pressure was 11 cmH_2O. His blood pressure also gradually improved (up to 90/48 mmHg) under fluid hydration and vasopressor use. However, the patient still suffered from pulseless ventricular tachycardia twice and defibrillation was immediately performed. Endotracheal intubation was performed and intra-aortic balloon pumping (IABP) was also inserted to address the patient's unstable hemodynamic. Surprisingly, follow-up ECG revealed ST elevation over lead

V_2-V_6 (Figure 1B). Emergent coronary angiography was again arranged under the preliminary diagnosis of STEMI over the anterior wall. Follow-up right coronary angiogram showed patent RCA without in-stent restenosis or stent thrombosis, but left coronary angiogram revealed middle LAD total occlusion (Figure 2D). Therefore, further thrombus aspiration with stenting was performed over LAD and TIMI 3 flow was restored. Then, the patient was transferred to our CCU for intensive care.

Despite primary PCI being performed over both RCA and LAD, unstable hemodynamics were still noted in the patient even under IABP and multiple vasopressor use. Follow-up laboratory data showed cardiac enzymes: creatine kinase-MB: 423.7 U/L, troponin I: > 100 ng/mL. Other data were as follows: increased white blood cell count (WBC: 30100/ul), CRP: 158 mg/L, D-Dimer level (3.4 mg/L of fibrinogen equivalent units), partial thromboplastin time and prothrombin time level (> 120 second and 16.9 second, respectively); and decreased platelet count (78000/ul).Urinary analysis also revealed pyuria. Under the impression of cardiogenic shock in combination with urosepsis and DIC, empirical antibiotic (piperacillin/tazobactam) was also given for infection
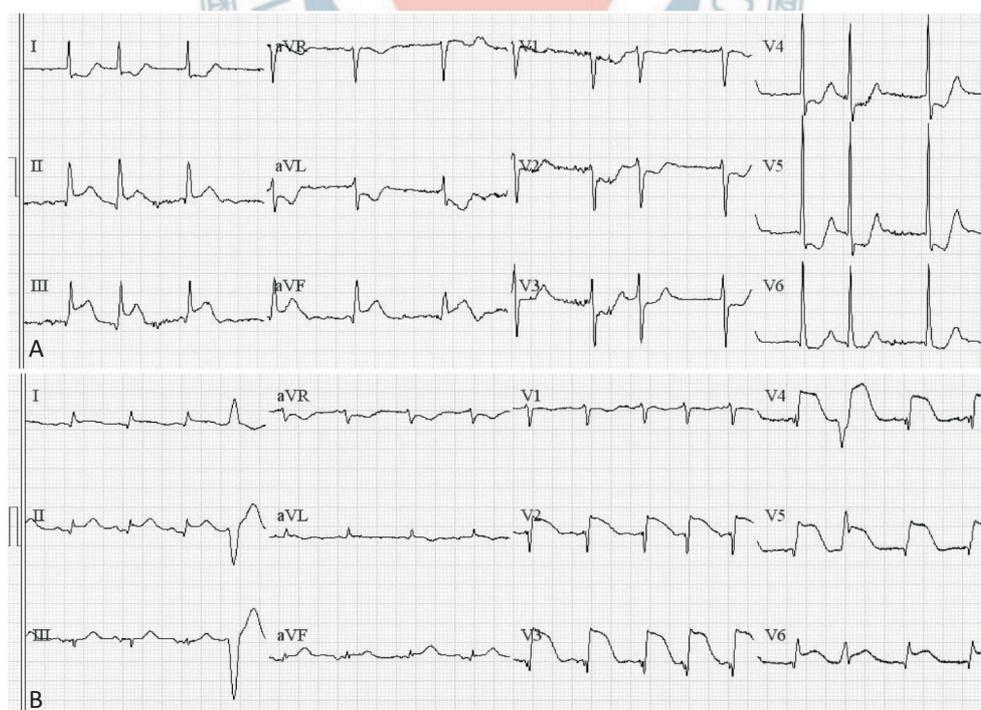
Figure 1. *Electrocardiogram (ECG) initially showed ST elevation over leads II, III, and aVF (A). Follow-up ECG revealed ST elevation over leads V2-V6 (B).*
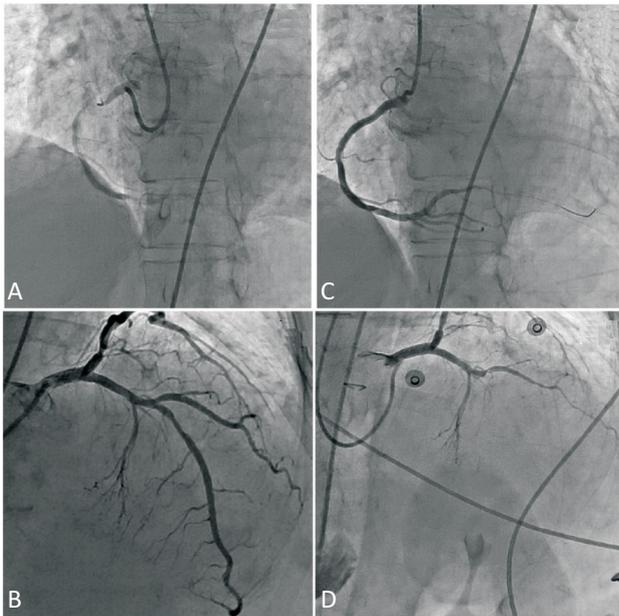
Figure 2. (A) Right coronary angiogram showed subtotal occlusion over proximal right coronary artery (RCA). (B) Left coronary angiogram showed both the left anterior descending artery (LAD) and the left circumflex artery with 50% stenosis. (C) Coronary angioplasty with stenting was performed over RCA and TIMI 3 flow was restored. (D) Follow-up left coronary angiogram revealed middle LAD total occlusion.

control. However, the patient expired within one day because of profound shock status.

DISCUSSION

STEMI is usually caused by rupture of unstable plaque and involves a single coronary artery. Acute ST is a possible cause of recurrent STEMI which occurred within one day of a previous episode.[1,2] It is an uncommon but fearful complication, and is associated with high mortality.[3] Most cases of ST occur in a single coronary vessel, but there were still some cases where simultaneous ST in multiple coronary vessels were reported.[3,4] Furthermore, simultaneous occlusion of multiple coronary arteries in patients with STEMI is also uncommon and often leads to poor clinical outcome.[5-7] However, there are no prior reports in the literature involving consecutive STEMI recurring in a different coronary artery within one day. To our knowledge, our case should be the first report with this finding, suggesting that vulnerable plaques in a non-culprit coronary artery rapidly progressed and led to coronary total occlusion within a few hours after the first STEMI episode.

Patients with acute myocardial infarction (AMI) may have multiple complex coronary plaques that are not limited to the culprit lesions. Lee et al. reported that little angiographic change occurred during 6 months of follow-up of AMI in the non-culprit complex plaques.[8] However, histopathologic study has suggested acute coronary disease might result from thrombosis and possibly other biochemical reactions, superimposed on chronic, rather than on recent, ulcerated plaques.[9] Hong et al. once conducted a study of 3-vessel intravascular ultrasound to evaluate the incidence and predictors of single and multiple plaque ruptures in patients of AMI and stable angina. They found plaque ruptures of non-infarct-related artery in 17% patients of AMI. In addition, the only independent clinical predictor of plaque rupture in AMI patients was an elevated CRP level.[10] Many intrinsic and extrinsic factors can precipitate fibrous cap weakness and finally result in plaque rupture, such as inflammation, intraluminal mechanical forces modulated by sympathetic tone and catecholamines. In addition, DIC was also reported to be associated with AMI and coronary thrombosis. According to the laboratory data of our patient, our case might also have sepsis-related DIC due to increased D-Dimer and decreased platelet count. Therefore, in our case, the most likely mechanism of rapid coronary thrombosis in non-culprit vessel was progression of inflammatory process that was related to urosepsis with DIC, increased CRP level, and ongoing shock status.

CONCLUSIONS

This case reminds us that consecutive STEMI recurring in a different coronary artery is an extremely rare but possible scenario and could lead to catastrophic clinical outcome. Physicians should not only treat coronary lesions but also initially evaluate the infectious status of the patient, and consider early empirical antibiotic use to avoid possible poor prognosis.

CONFLICT OF INTERESTS

None declared.

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