CASE REPORT

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Immediate Rupture of Another Coronary Plaque After PCI of the Culprit Lesion in ST Segment Elevation Myocardial Infarction

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ABSTRACT ST-segment elevation myocardial infarction (STEMI) is usually the result of thrombosis in the single coronary artery. The incidence of multiple coronary artery thrombosis in acute phase is very low. The etiology of multivessel coronary thrombosis is still unclear. Observational studies have shown that plaque instability underlying thrombosis is not due to local vascular causes alone. These pathophysiological processes such as inflammation, sympathic activity and catecholamines discharge act across the entire coronary network and cause multiple plaque instability.[1,2] We think that the pathogenesis of multiple coronary occlusive lesions may be explained by multiple atherosclerotic plaque rupture during the acute phase of myocardial infarction. In our case, the first percutaneous coronary intervention was performed on the left anterior descending artery due to anterior STEMI. At the second hour of follow-up, inferior STEMI developed and percutaneous coronary intervention was performed on optus marginalis.

Keywords: Recurrent myocardial infarction; percutaneous coronary intervention; atherosclerosis

CASE REPORT

A 49 year-old man without any history of heart disease presented to the emergency department with squeezing chest pain of 2h duration, which was radiating to his back and neck. His physical examination findings on the arrival were as follows: blood pressure: 124/73 mmHg, pulse rate: 110/min, respiratory rate: 13/min, temperature: 36.8°C and no evidence of heart failure. His past medical history was significant for hypertension and smoking. On admission, the electrocardiography (ECG) showed ST-segment elevation in leads V1-4 and ST segment depression in leads II, III and aVF. He was diagnosed with anterior ST-segment elevation myocardial infarction (STEMI) and was given 180 mg of ticagrelor, 300 mg of acetylsalicylic acid, and 5000 IU of IV heparin. Then, he was taken to the catheterization laboratory and immediate coronary angiogram showed the total occlusion of left anterior descending (LAD) from mid-region (Figure 1 A), 50% stenosis in the proximal portion of the left circumflex artery (LCX) and 60% stenosis in the optus marginal (OM) (Figure 1 B). Right coronary artery was normal. A drug-eluding stent was im-

planted in the LAD mid region (Figure 1 C). Two hours later he experienced chest pain again and the ECG showed ST segment elevation in leads II, III and aVF. Control coronary angiogram showed that the LAD stent was patent (Figure 2 A) and the OM was totally occluded (Figure 2 B). Then, OM was also stented. The further course was uneventful.

DISCUSSION

In patients who died due to acute myocardial infarction, multiple coronary artery thrombosis was reported to be approximately 40%.[1] Clinical results of this patient have showed that plaque instability is not only a local vascular event and it also reflects pathophysiological processes spoiling the stabilization of all atherosclerotic plaques throughout the coronary artery. Our case shows that patients with acute myocardial infarction may have multiple coronary plaques causing adverse clinical outcomes. Catecholamine increase, increased inflammatory response, hypotension and cardiogenic shock after occlusion of the vessel, prolonged coronary vasospasm, malignancy and related hypercoagulability, thrombocytosis, hyperhomocysteinemia, cocaine use, atrial fibrillation and human immunodeficiency virus can cause acute thrombosis of multiple coronary arteries.[2-4]

In a study conducted by James et al., in the 253 patients with acute myocardial infarction; those with multiple complex plaques were found to have

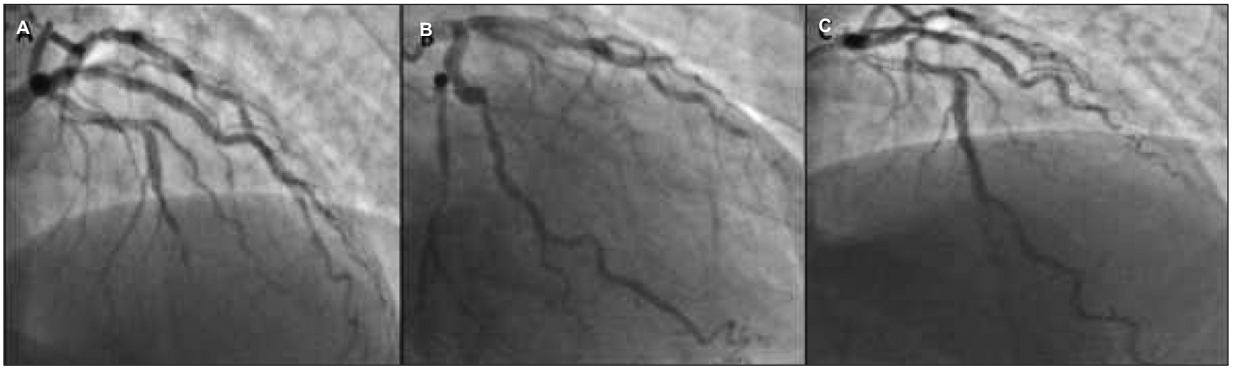

FIGURE 1: AP cranial view showing LAD total occlusion **(A)**; RAO caudal view showing OM lesion on the original angiogram **(B)**; AP cranial view showing LAD after stenting **(C)**.
LAD: Left anterior descending; OM: Optus marginal.

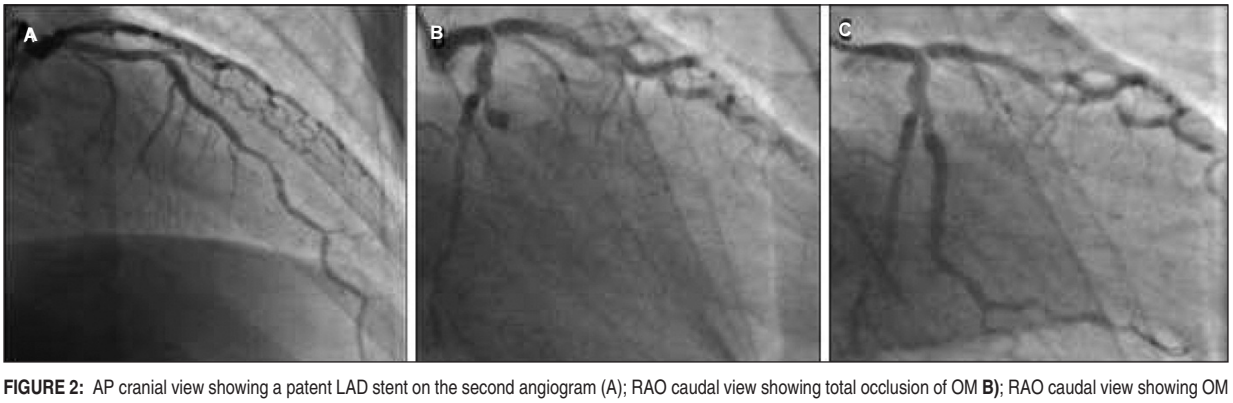

FIGURE 2: AP cranial view showing a patent LAD stent on the second angiogram (A); RAO caudal view showing total occlusion of OM **B)**; RAO caudal view showing OM after stenting.
LAD: Left anterior descending; OM: Optus marginal.

a higher need for staged multivessel angioplasty.[5] In addition, in the autopsy after sudden cardiac death, multiple coronary thrombus and multicentric plaque rupture have been shown multifocal plaque instability.[1,6] The main cause of plaque instability is the weakening of the plaque protective fibrous hood. Compared with stable plaques,unstable plaques are characterized by active inflammation of the fibrosis cover.[2] Inflammatory cells activate matrix metalloproteinases that disrupt the extracellular matrix. Atherosclerotic coronary plaques are commonly affected by the toxic effects of oxidized low density lipoproteins (LDL), viral triggers or other factors.[3] Intrinsic plaque instability may develop due to changes in lipid metabolism. In addition, intraluminal mechanical forces act along the entire coronary network and increase plaque rupture risk.[4] Therefore, in all of the coronary vessels, an increase in the thrombogenicity of erosion-affected plaques is expected.

Clinical observations support that systemic processes affect plaque instability. Patients who had acute myocardial infarction have systemic inflammatory findings. The increase of serologic markers in macrophage activity has been associated with the presence of multiple complex plaques in unstable angina patients.[7] Systemic treatments to stabilize plaque, the reduction in myocardial infarction provide an additional contribution to the fact that unstable coronary artery disease is a multifocal process affected by systemic factors.[8] At least in some patients, in acute coronary syndrome, these pathologic, angiographic, and clinical observations reflect a common pathophysiological process leading to multifocal plaque instability and rapid plaque progression in association with clinical instability.[9] In our case, it is the most possible cause of the rupture of the plaque at OM region is as much as possible to have inflammation and increased sympathetic efficacy.

In conclusion, the presence of multiple complex coronary plaques in coronary angiography shows an increased risk of recurrent ischemia even after the beginning of acute percutaneous interventions.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

All authors contributed equally while this study preparing.

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