CLINICAL CASE REPORTS

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Survival from Acute Occlusion of the Left Main Coronary Artery with Preexisting Collateral Vessels—A Case Report

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Abstract

A thirty-two-year-old man suffered from evolving acute myocardial infarction caused by total occlusion of the left main coronary artery, which was 95% stenosed before the onset. Nevertheless, he had a good clinical course. The myocardium may have been protected by well-developed preexisting collateral vessels as evidenced by serial coronary angiograms.

Introduction

Acute complete occlusion of the left main coronary artery (LMCA) induces serious myocardial ischemia, including fatal myocardial infarction (MI) and sudden death.[1-3] Although acute MI (AMI) with this lesion has occasionally been reported, information concerning the coronary artery stenosis before the onset has been very limited.[4-8] We present here a patient with effort angina that evolved into AMI by LMCA occlusion. His serial coronary angiograms and electrocardiograms together with clinical profiles are reported.

Case Report

A thirty-two-year-old businessman had had an oppressive feeling in the chest on moderate exercise in January, 1987, and visited our outpatient clinic on February 14. His resting electrocardiogram appeared normal. Exercise test by treadmill with a modified

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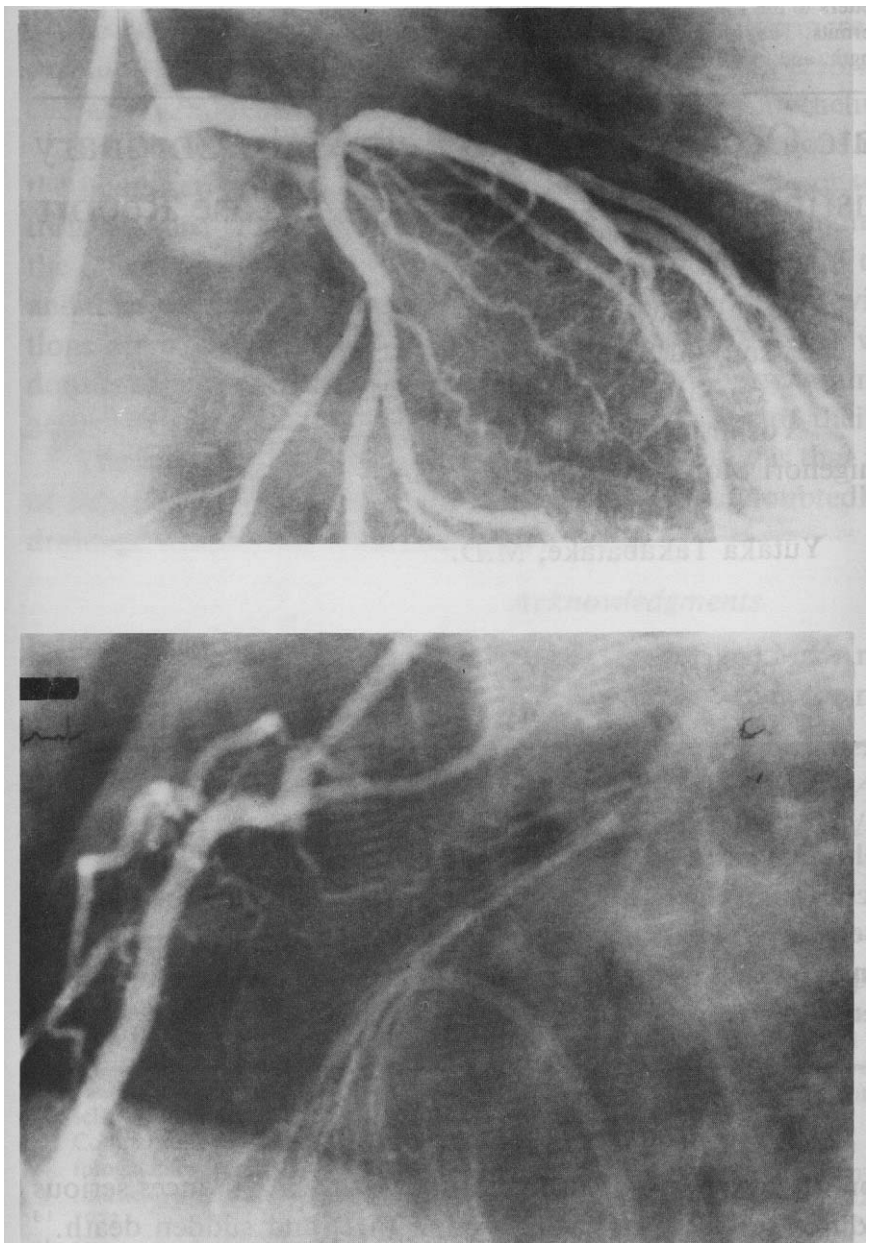

FIG. 1. Coronary angiography on February 23, 1987. (Top) There were a 95% stenosis at the left main coronary artery and a 90% stenosis at segment 7 of the left anterior descending coronary artery. Right anterior oblique view. (Bottom) Right coronary angiography showed well-developed collateral vessels to the left anterior descending coronary artery. Left anterior oblique view.

Bruce protocol for five minutes induced a downsloping 4 mm ST segment depression in lead V_5 and a typical anginal pain. He was hospitalized for further evaluation on February 20. He had smoked three packs of cigarette a day for fifteen years. His father had a history of AMI at the age of fifty-three.

On admission, his blood pressure was 112/50 mmHg, heart rate 68/minute and regular. Heart sounds were normal. On chest x-ray, the cardiothoracic ratio was 44%. Laboratory tests revealed a decreased serum high density lipoprotein level of 25 mg/dL. Cardiac catheterization on February 23 showed no abnormalities. Coronary angiography by Jud-

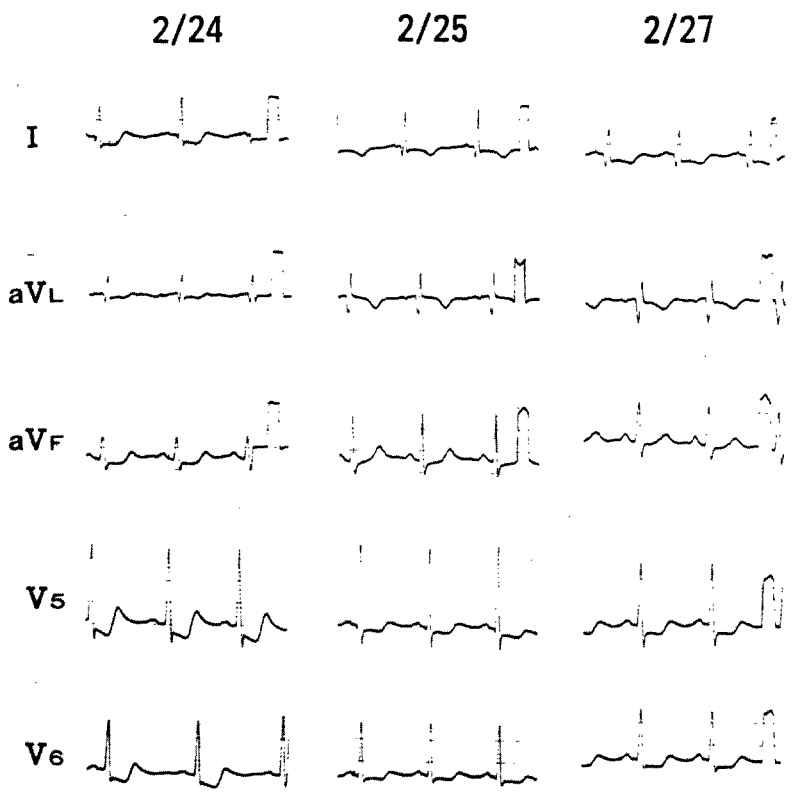

FIG. 2. Time course of the electrocardiograms during the acute myocardial infarction from February 24 (left), 25 (middle), to 27 (right).

kins technique demonstrated a 95% stenosis of the LMCA and a 90% stenosis at the proximal portion of the left anterior descending (LAD) coronary artery (Figure 1-Top). The right coronary artery (RCA) had no significant stenosis and supplied collateral vessels to the LAD artery (Figure 1-Bottom).

Ten hours after the catheterization, he had an anginal attack with ST segment depression. Twenty-nine hours later, he had another prolonged chest pain at 6 PM on February 24. The electrocardiogram revealed marked ST segment depression in precordial leads (Figure 2, left). A nitrate agent infused intravenously was ineffective and so 960,000 IU urokinase was administered one hour later. Serum creatine phosphokinase (CPK) increased to 2,150 IU/L and CPK-MB to 204 IU/L, twenty-two hours after the onset. A small q wave appeared in lead $_aV_L$ (Figure 2, middle). Pulmonary congestion was transiently observed but improved in the next five days. An electrocardiogram on February 27 showed moderate ST segment depression in lead V_5 (Figure 2 right).

Coronary angiography on March 23 showed complete LMCA occlusion (Figure 3-Top). Collateral vessels from the RCA became more prominent and opacified the left coronary artery (Figure 3-Bottom). Since a reversible perfusion defect in the left ventricular anterior wall was seen on the exercise ^{201}Tl scintigraphy with a 4 mm ST segment depression in lead V_5 and chest pain, an aortocoronary graft was placed to the LAD coronary artery on April 6. His final coronary angiography, thirty-nine days after the surgery, showed a patent graft and disappearance of the collateral vessels. The patient has been asymptomatic for three years.

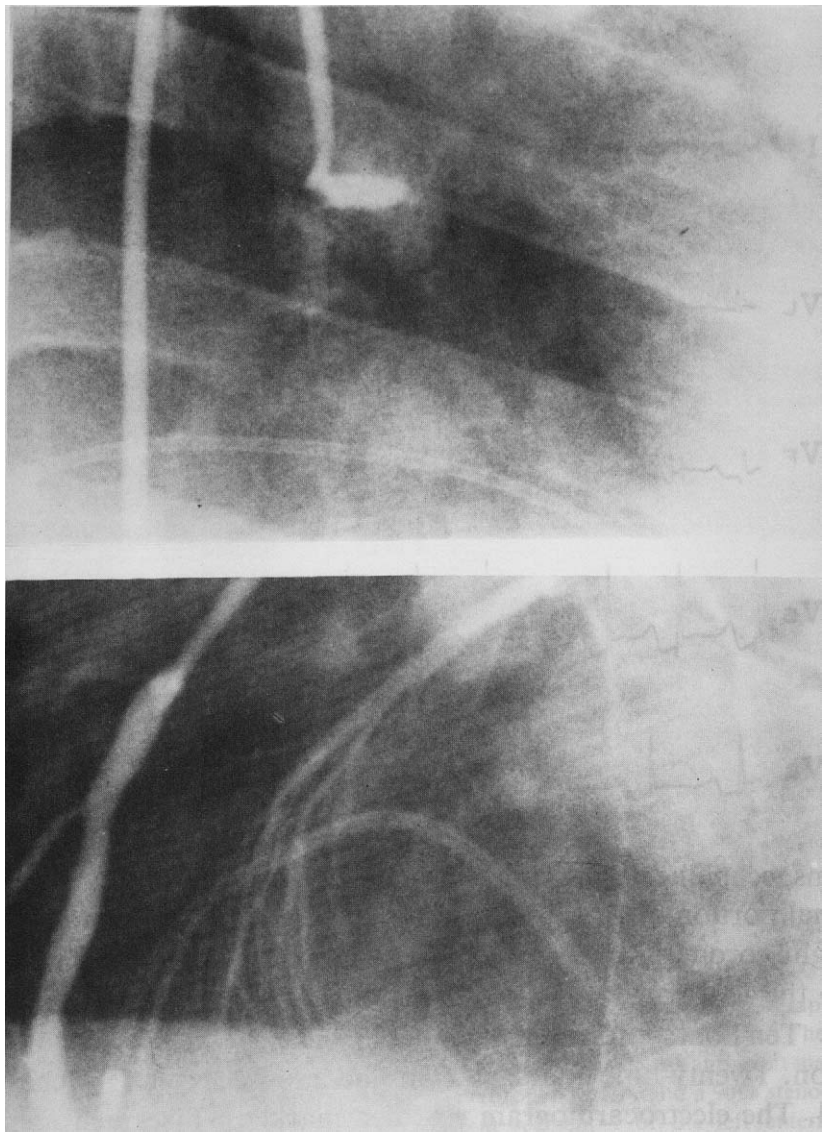

FIG. 3. Coronary angiography on March 23, 1987. (Top) There was a total occlusion of the left main coronary artery. Right anterior oblique view. (Bottom) Right coronary angiogram revealed further development of the collateral vessels with retrograde opacification of the left main coronary artery. Left anterior oblique view.

Discussion

Occlusion of the LMCA is found in 0.02% to 0.3% of diagnostic cardiac catheterizations.[1,2] Case reports showing the progression from high-grade LMCA stenosis to complete occlusion have been few. As far as we know, only 5 cases have been documented with the angiographic feature before the total LMCA occlusion.[4-8] One reported case with a 90% LMCA stenosis sustained an AMI six hours after the coronary angiography[4] and another 2 cases did so during coronary angiography.[5,7] None of these 5 patients had collateral vessels before the occlusion. Our case is unique since the collateral vessels from the RCA were clearly observed before the total LMCA occlusion. His survival from the following AMI largely depended upon this well-developed collateral circulation. Absence of a typical ST segment elevation during the AMI can be explained by these preexisting collateral vessels.[9]

Frequent episodes of myocardial ischemia could promote new vessel formation. In canine experiments, repeated occlusions of the coronary artery for two minutes at thirty-two minute intervals induced collateral circulation in several days.[10] In patients with vasospastic angina, collateral vessels appeared transiently in 44% of ergonovine-induced spasms of the LAD coronary artery.[11] Although the cause of acute LMCA occlusion was not clear in our case,[12] we should pay attention to collateral vessel formation in patients with crescendo angina pectoris.

Conclusion

Survival from evolving MI in this case stressed the significance of the collateral vessels established before the complete LMCA occlusion.

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