Spontaneous Intimal Dissection in a Patient with Post-Infarct Angina: Identification with Intravascular Ultrasound and Treatment with Coronary Stenting

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SUMMARY

A 45-year-old Turkish male patient was admitted to our hospital for an acute myocardial infarction. He had suffered two previous acute myocardial infarctions 5 and 6 years earlier. Coronary angiography performed after the previous two acute myocardial infarctions had shown normal coronary arteries and coronary vasospasm had been suspected. The patient was treated with thrombolytic therapy (rt-PA) during the last coronary event. Five days after the current admission, the patient had postinfarct angina and underwent coronary angiography, showing only a nonobstructive lesion with irregular ulcerated edges in the left anterior descending artery after the first diagonal branch. Subsequent intravascular ultrasound confirmed the presence of an atherosclerotic lesion with plaque dissection. Stenting of this lesion was performed with successful relief of on-going chest pain.

Intravascular ultrasound can provide important diagnostic information in patients presenting with acute coronary syndromes in the absence of severe angiographic stenosis. Identification of plaque dissection at mildly stenotic lesions provides pathophysiologic insights that may have therapeutic implications. However, the optimal treatment for such lesions is not known. (Jpn Heart J 2003; 44: 557-564)

Key words: Acute myocardial infarction, Intravascular ultrasound, Plaque dissection, Vulnerable plaque, Coronary stent

Approximately 70% of patients suffer an acute myocardial infarction (MI) as the first symptom of coronary artery disease. Most acute MIs are caused by rupture or superficial erosion of an atherosclerotic plaque and subsequent thrombotic occlusion of the coronary artery. The size of lesions underlying the thrombus is variable. Angiographic studies in patients with acute myocardial infarction have shown that the culprit lesion is frequently not severely stenotic in the
months before the event. Similarly, the extent of residual stenosis after successful thrombolysis is not often very severe.

The occasional finding of a normal angiogram at the time of presentation with acute coronary syndromes, including acute myocardial infarction, is well known as syndrome X. In these patients, prolonged coronary vasospasm and hypercoagulable states with coronary thrombosis or embolism were thought to be the responsible mechanism. In the patient presented here, Syndrome X had been accused for pathophysiologic causes of previous MIs because there was no risk factor for coronary heart disease in the past.

CASE REPORT

A 45-year-old Turkish male patient visited our emergency department with a 30-minute history of chest pain radiating to the left arm. The patient was looking pale and confused. His arterial blood pressure was 100/50 mmHg with a heart rate of 122 bpm. Subsequently, a 12-lead ECG demonstrated ST-segment elevation in leads V1 to V5 and an old q-wave in the anterolateral leads (Figure 1A). Initial cardiac markers, including CK-MB, ALT, LDH, and Tr-T, were not high. Based on the clinical and electrographic findings, an acute anterior myocardial infarction (MI) was diagnosed and the patient was treated with an intravenous infusion of rt-PA at a dose of 100 mg as a thrombolytic agent. Within 10 minutes after the rt-PA infusion, his arterial blood pressure improved and the chest pain resolved completely.

The values of CK-MB and troponin-T 6 and 12 hours after admission significantly increased (45 IU at 6 hours, 260 IU at 12 hours; 0.1 ng/mL at 6 hours, 1.2 ng/mL at 12 hours, respectively). A 12-lead ECG after 24 hours showed Q waves and inverted T-waves in leads V1-V5 (Figure 1B).

This was the patient's third MI and was nearly 5 years after the second MI attack. He was hospitalized in 1995 and 1996 for acute anteroseptal and anterolateral myocardial infarctions, respectively. He underwent coronary angiography after each of these acute MIs, and both coronary angiographies showed normal coronary anatomy (Figure 2). Coronary vasospasm or embolic events had been considered as possible causes of the previous MIs because there was no risk factor for coronary heart disease. The patient had been kept on medical therapy, including long-acting nifedipine and aspirin.

Five days after the current acute MI, the patient started to suffer from recurrent chest pain (post-MI angina) and therefore underwent coronary angiography. The only abnormal finding was a nonobstructive lesion with border irregularity in the proximal left anterior descending artery (Figure 3). In order to further evalu-
Figure 1. (A) A 12-lead electrogram, showing acute anterior myocardial infarction, recorded at the time of present admission, and (B) the second electrogram recorded after thrombolytic therapy.
ate examination of the left anterior descending artery using intravascular ultrasound (IVUS) (30 MHz, 3.2 French UltraCross catheter, Boston Scientific/
Scimed, Inc., Maple Grove, Minnesota) showed an atherosclerotic plaque with interruption and dissection (Figure 4A and 4B). These IVUS findings are consistent with vulnerable plaque. We implanted a stent (17 mm and 3.0 mm, Devon Stent, Hamburg, Germany) into the dissected plaque. The patient was asymptomatic during a 6-month follow-up period after stenting.

Figure 4. (A) Intravascular ultrasound images from the proximal part of the lesion in the LAD [lumen cross-sectional area (CSA) 4.2 mm²; vessel CSA 11.8 mm²] (B) the distal part of the lesion in the LAD (lumen CSA 5.2 mm²; vessel CSA 11.3 mm²).
DISCUSSION

The patient presented was followed up previously with no thought of the presence of any plaque in the vessels although he had had two prior episodes of myocardial infarction. However, the presence of associated changes in the arterial wall had been incompletely investigated. Although more than 70% of culprit lesions in patients with unstable angina have characteristic angiographic findings, including eccentricity or irregular ulcerated edges, coronary angiography provides only the planar silhouette of the vessel lumen that can miss plaque ulceration and rupture-prone “vulnerable” plaques.\textsuperscript{8,9} IVUS has demonstrated a varying degree of dissociation between luminal stenosis and plaque size in most patients with coronary artery disease.\textsuperscript{10} In a prospective study by Yamagishi, \textit{et al}, plaque that ultimately caused an acute coronary syndrome exhibited a large volume and eccentric shape distribution even though the lumen area could be preserved at the time of coronary angiography, causing insensitivity to vulnerable plaque.\textsuperscript{11} An important reason that coronary angiography is not sensitive in identifying rupture-prone plaque is arterial remodeling.\textsuperscript{12} Because of the compensatory vessel enlargement at the site of growing plaques, rupture frequently develops in vessel segments without high-grade stenosis.\textsuperscript{13} Recent studies demonstrate that these early lesions may be particularly prone to rupture.\textsuperscript{14,15} It is conceivable that temporary thrombotic occlusion after rupture of such a lesion could cause an acute coronary syndrome in the absence of angiographic stenosis.

The present patient had suffered two prior episodes of acute MI. Because the first two coronary angiographies were normal, the patient had been treated for assumed coronary vasospasm. It is unclear if vasospasm occurred superimposed on a preexisting but angiographically undetected atherosclerotic lesion and if a relation with plaque rupture could have been established with IVUS. Another study by Yamagashi, \textit{et al} showed that focal vasospasm occurred at the site of atherosclerosis, which was the most likely source of local hyperreactivity and could not be demonstrated by conventional coronary angiography. Also, they proposed that spasm itself might contribute to accelerating the progression of atherosclerosis.\textsuperscript{16}

This case report of a nonobstructive culprit lesion in a patient with postinfarct angina exemplifies the complex interaction between the lumen and vessel wall of atherosclerotic lesions. It demonstrates that IVUS can provide important diagnostic information in patients presenting with acute coronary syndromes in the absence of severe angiographic stenosis. Identification of plaque rupture at mildly stenotic lesions provides pathophysiologic insights that may have therapeutic implications in patients with acute coronary syndrome. This is particularly true as recent studies show that lesions with evidence of vulnerability or rupture
are frequently found distant from the culprit lesion in patients with acute MI.\textsuperscript{17,18)} However, the best treatment for such lesions is not known. It would be logical to assume that medical management including antithrombotic therapy and aggressive early risk-factor modification may be beneficial.\textsuperscript{19)} The role of stenting such lesions is incompletely understood.\textsuperscript{20)}

\textbf{REFERENCES}
