Ruptured Atherosclerotic Plaque Distant from Maximal Stenosis in Acute Myocardial Infarction

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Abstract

We describe a patient with acute myocardial infarction who showed ruptured plaque distant from the maximally stenotic lesion. In a 54-year-old male patient with acute antero-lateral myocardial infarction, coronary angiography showed a resolution of occlusive lesion with residual stenotic lesion in the middle portion of the left anterior descending artery (LAD) following t-PA administration. One month later, coronary angiography again disclosed significant stenosis of the middle LAD. Intravascular ultrasound revealed ruptured plaque that was located proximal to the maximally stenotic site which is generally considered as the culprit lesion. In this case, transient vessel occlusion occurred at the maximally stenotic site probably associated with plaque rupture distant from this lesion.

Key words: acute coronary syndrome, intravascular ultrasound, unstable plaque, plaque rupture

Case report

A 54-year-old man was admitted to our clinic within one hour after the onset of persistent chest pain. Electrocardiogram on admission showed ST elevation in leads VI to V4, and poor R wave in the leads I and aVL, consistent with an evolving antero-lateral myocardial infarction (Fig. 1). After transvenous injection of t-PA (800,000 U) the patient was taken to the cardiac catheterization laboratory. After administration of nitroglycerin (0.25 mg) coronary angiography disclosed resolution of total occlusion and significant stenosis in the left anterior descending (LAD) coronary artery with TIMI III flow (Fig. 2). Therefore, subsequent coronary angioplasty was not performed at that time. Under these conditions, this stenotic lesion was considered to be responsible for the occurrence of acute myocardial infarction, although there were minor luminal irregularities proximal to this stenosis (Fig. 2A). The patient was treated with intravenous heparin (15,000 U/day) and peak CPK level was 4,286 IU/l.

One month after the emergency catheterization, the patient underwent coronary angiography again. The morphology of the maximally stenotic site in the LAD was relatively unchanged from that shown in the acute phase although stenosis became somewhat greater than that in the acute phase (Fig. 3). Intravascular ultrasound which was performed before angioplasty did not show any ruptured lesion within the stenotic segment (Fig. 3A) but a ruptured plaque was found proximal to the most stenotic lesion (Fig. 3B and 3C). There was disease continuity from the stenotic segment to the ruptured site and there was slight positive remodeling with 1.055 as an index. The lumen areas of these ruptured sites were well preserved by both angiography and intravascular ultrasound. It was interesting that the site with plaque rupture had angiographically minimal luminal irregularities in the acute phase. No other complex lesion was found in the LAD by both angiography and IVUS. The patient had balloon angioplasty and stent deployment that covered not only the most stenotic lesion but also the proximal ruptured site.

Discussion

The most important finding of this case is that plaque rupture occurred at a site distant from the maximally stenotic lesion in acute myocardial infarction. Acute coronary syndrome, such as unstable angina and acute myocardial infarction, has been considered to be the result of disruption of vulnerable plaque and the subsequent thrombus formation at the ruptured site. Pathological studies support this hypothesis based on the post mortem evaluation of patients...
who died of acute coronary events (1-4). However, new modalities such as intravascular ultrasound systems and coronary angioscopy systems enable visualization of the coronary arteries from inside the vessels and show new aspects of acute coronary syndrome (5, 6), because angiography shows only a silhouette of coronary arteries and sometimes leads to misinterpretation of disease morphology. Goldstein et al reported the evidence of multiple complex angiographic lesions in almost 30% of patients presenting with acute coronary event (7). This suggests that most stenotic lesions are not always responsible for plaque rupture with or without thrombosis. Indeed, discrepancy of the sites between the most stenotic coronary sites and ruptured plaques has been reported by Maehara et al who preliminarily reported that approximately 74% of ruptured plaque exists at a site different from the maximally stenotic lesion (8).

Considering that plaque rupture can occur at any site of the coronary segments where underlying atherosclerosis is present (5), it should not be surprising that the rupture of plaques occurs at a different site from the most stenotic lesion as observed in the present patient. It was possible that in the present patient the thrombus that was produced at the ruptured site occluded the most stenotic site at the onset of myocardial infarction, because occlusive lesion was resolved by the administration of t-PA without angioplasty. There seemed to be an echolucent area at the most stenotic site, suggesting that this site could be also vulnerable. However, this echolucency was derived from the attenuation of ultrasound intensity. Therefore, the most stenotic site itself was not vulnerable in comparison with the proximal site.

The present case provides several important implications. First, when coronary intervention is indicated for a patient with acute coronary syndrome, it is important to carefully examine not only the most stenotic sites but also the adjacent sites where plaque disruption occurred. Otherwise, another event associated with these remote lesions can occur after interventional procedures. Secondly, in addition to the interventional treatment of angiographically culprit lesions, the subsequent treatments such as plaque stabilizing therapy by aggressive lipid lowering should be done to the lesions which coexist with the most stenotic lesions and may or may not be responsible for future recurrence of acute coronary event (9, 10). For these purposes, intravascular ultrasound

Figure 1. Electrocardiogram at the time of admission showed ST elevation in leads VI to V4, and poor R wave in leads I and aVL.

Figure 2. Coronary angiography after administration of t-PA in the acute phase. There was 75% stenosis in right (A) and left (B) anterior oblique views. Note that there were also minor luminal irregularities proximal to the stenosis (arrow in A). At that time, no further angioplasty procedure was performed because of maintaining distal coronary flow.
Figure 3. Coronary angiography (upper) and intravascular ultrasound (IVUS, lower) images one month after the onset of myocardial infarction. By angiography, stenosis became somewhat increased to be 90%. At the stenotic lesion, IVUS did not show any sign of plaque rupture (arrow A). Proximal to the stenosis, ruptured plaques were noted on IVUS (B and C, arrows).

should be considered to identify the true culprit lesion and/or coexistence of another unstable plaque. Further follow-up study may demonstrate whether the ruptured plaque can repeatedly be responsible for acute coronary events.

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References