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Reocclusion of Coronary Artery during Coronary Thrombolysis in Patients without Severe Residual Stenosis

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Abstract We present 4 cases of acute reocclusion of a coronary artery. They had total occlusion at the time of acute myocardial infarction, and had been recanalized with intracoronary urokinase infusion with a residual lesion of less than 50%. Reocclusion occurred by thrombus formation at the same location, where total occlusion was seen in acute myocardial infarction. These cases may suggest that thrombus formation is a major cause for acute occlusion in some patients without significant obstructive coronary artery disease, as well as the coronary artery wall abnormality is.

Key Words : Acute myocardial infarction, Coronary thrombolysis, Reocclusion

Introduction

Coronary thrombolytic therapy early in acute myocardial infarction can restore myocardial blood flow.[1-5] Restoration of coronary blood flow may then reduce the extent of myocrdial infarction and preserve left ventricular function that might otherwise be lost. Yet, the reooclusion after recanalization may negate these beneficial effects. There has been reports on reocclusion in the follow-up catheterization after coronary thrombolysis for acute myocardial infarction[6-12]. However, the factors influencing reocclusion after coronary thrombolysis has not been amply reported.

In this study, we observed the reocclusion of widely recanalized coronary artery during coronary thrombolysis, and discussed the factors influencing reocclusion and the mechanism of acute myocardial infarction.

Material and Methods

One hundred and five patients received intracoronary urokinase infusion for acute myocardial infarction. All patients had 960,000 to 1,440,000 units of urokinase within 6 hours after the onset of infarction. Intracoronary nitroglycerin was routinely administered before urokinase infusion. All patients recieved intravenous 5000 IU of heparin before catheterization. All patients had total or subtotal occlusion of the infarct related artery before nitroglycerin or urokinase infusion was started. Recanalization was defined as prompt opacification of the entire distal coronary artery. Coronary angiograms were reviewed by at least 2 experienced angiographers. The severity of the residual stenosis

was graded by qualitative visual inspection, and consensus as less than 50% or 50% or more luminal reduction. The angiographic criteria for residual thrombus included : a long intraluminal filling defect extending distally from the site of occlusion ; a round or oval filling defect surrounded by a ring of contrast ; embolization of the proximal "clot in lysis" to the distal artery ; and "thumb-printing," in which the segment of coronary artery is poorly opacified except for a marginal rim of contrast[12]. These criteria for residual thrombus were "learned" by observing progressive lysis of the clot during streptokinase infusion as well as by disappearance of the findings above during follow-up angiography of patients with persistent patency of the infarct related artery.

In the consecutive 105 patients who received coronary thrombolysis for acute myocardial infarction, 32 patients had a widely recanalized coronary artery with a lumen reduction of less than 50%, where total occlusion was seen in the initial injection of contrast media. Four of 32 patients were reoccluded during coronary thrombolysis (Fig.1—2). Reocclusion occurred immediately after recanalization during cardiac catheterization. The locations of the reocclusion was same as those of total occlusion seen in acute myocardial infarction.

Case 1. A 73-year-old man had angina on exertion for 1 week before acute myocardial infarction. He was referred to the hospital with severe chest pain during bathing in the evening. ECG showed acute anteroseptal myocardial infarction. Coronary thrombolysis was performed 2 hours after the onset of attack. The occluded left anterior descending coronary artery was recanalized

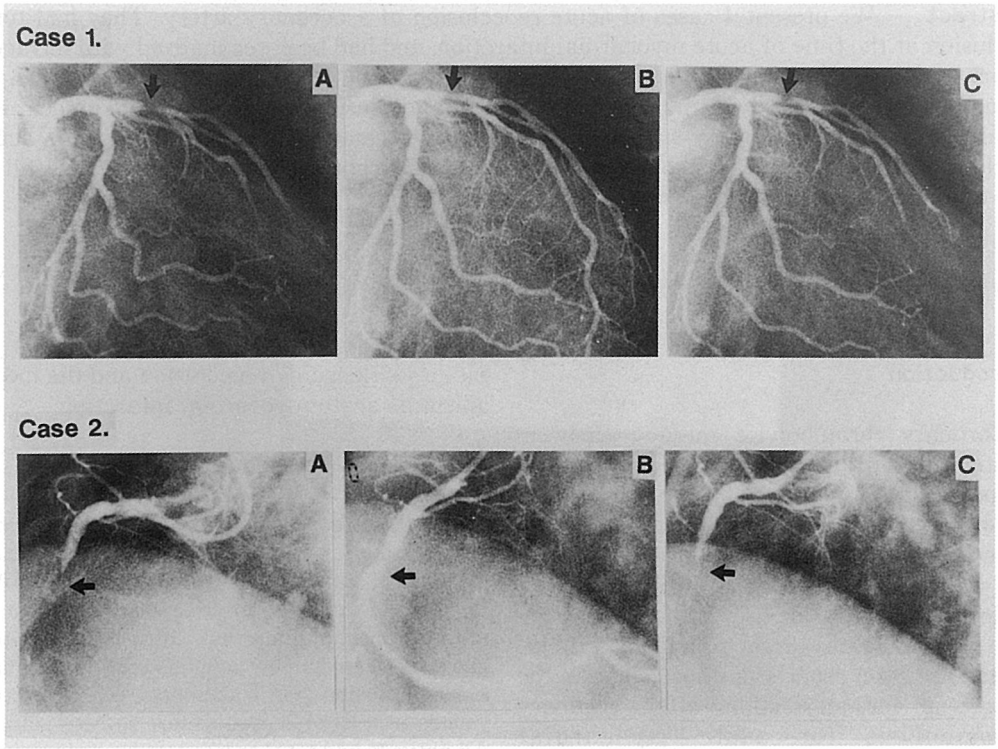

Fig. 1.
Case 1. The left coronary artery before (A) and during coronary thrombolysis (B, C). The proximal left anterrior descending coronary artery was recanalized without significant residual stenosis (B), which was reoccluded to the subtotal occlusion (C).
Case 2. The right coronary artery before (A) and during coronary thrombolysis (B, C). The right coronary artery was fully patent with urokinase infusion, which was reoccluded after recanalization.

with intracoronary urokinase infusion. The chest pain subsided and the ST-segment elevation in the precordial leads improved. However, the widely patent left anterior descending coronary artery was reoccluded at the same location, where the occlusion was seen initially. The further infusion of the intracoronary urokinase again recanalized the left anterior descending occlusion. With the subsequent heparinization after cardiac catherization, the patient had no more chest pain and ST-segment elevation.

Case 2. A 53-year-old man admitted to the hospital with sudden onset of chest pain at rest in the evening. He was diagnosed acute inferior myocardial infarction. He had no angina before myocardial infarction. Coronary thrombolysis was performed 4 hours after the onset of chest pain. He smoked 2 packs per day for 30 years.

Case 3. A 64-year-old man had angina on exertion for one year before acute myocardial infarction. He had a sudden onset of cheat pain in the morning, and admitted to the hospital with the diagnosis of acute anteroseptal myocardial infarction. Coronary thrombolysis was done 1 hour after the onset of attack. No significant risk factors were

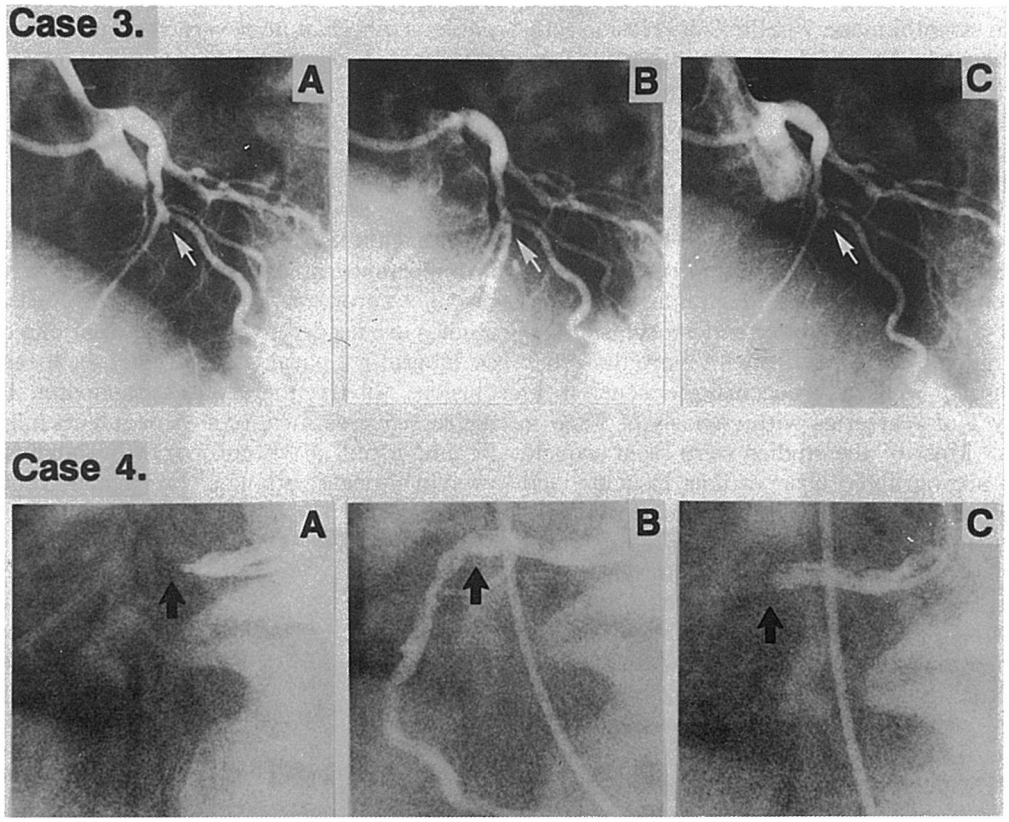

Fig. 2.
Case 3. The left coronary artery before (A) and during coronary thrombolysis (B, C). The left anterior descending coronary artery was recanalized. No significant residual stenosis was seen just distal to the major septal perforator. The left anterior descending coronary artery was reoccluded at the same site of the initial occlusion.
Case 4. The right coronary artery before (A) and during coronary thrombolysis (B, C). The right coronary artery was recanalized without significant residual stenosis. The later right coronary angiograms revealed reocclusion (C).

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Case 4. A 64-year-old man had a sudden onset of chest pain at rest in the morning, and admitted to the hospital under the diagnosis of acute inferior myocardial infarction. He underwent coronary thrombolysis 3 hours after the onset of chest pain. He had only a risk factor of hypertension.

Discussion

Factors influencing reocclusion after coronary thrombolysis for acute myocardial infarction have been amply investigated[11,12]. Harrison et al[11]. studied that in 24 patients who had undergone successful reperfusion with streptokinase, 7 patients had lesion rethrombosed. As assessed by quantitative coronary angiography, 7 of 13 patients with minimal luminal cross-sectional areas of less than 0.4 mm^2 had rethrombosis. None of the 11 patients with lumens greater than 0.4 mm^2 had rethrombosis. Gash et al.[12] reported that among 26 patients which acute thrombolytic recanalization, the reocclusion rate was 57% among infarct arteries containing residual clot after streptokinase, and reocclusion occurred in 5 of 17 arteries with more than 75% stenosis, whereas reocclusion occurred in only 1 of 7 arteries with stenosis of 75% or less. Most of the studies were from sequential angiographic observations in acute and chronic phases, and resulted in that reocclusion of the vessels were in part related to the size of the residual lesion after recanalization. The present study reported the reocclusion during coronary thrombolysis patients who had a widely recanalized coronary artery by intracoronary urokinase infusion. At present, it would be assumed that several of the causes ; such as spasm, abnormal hemostasis, plaque repture, hemorrhge into a plaque, and severe progressive atherosclerosis are interacting to produce the end results-coronary thrombosis and myocardial infarction[13]. Serruys et al[8]. reported that in 31% of patients a diameter stenosis of less than 50% was found whereas one of 70% or more was seen in only 19%, with streptokinase infusion. In the infarct angiograms, Brown et al[10]. also observed that the original stenosis was less than 50% in 10 of 32 (31%) acutely thrombosed coronary segments, and in 21 (66%) it was less than 60%. The majority of these coronary segments that became acutely occluded were mildly or moderately narrowed by atherosclerosis. Thus, the thrombosis dose have a major role in the genesis of myocardial infarction, as well as vessel wall abnormality or coronary atherosclerosis dose[13-15]. This study is a retrospective angiographic observation of rethrombosis in patients recieving coronary thrombolysis. The onset of acute myocardial infarction was reproduced by the reocclusion of the infarct related artery during angiographic study. Reocclusion was observed in patients who had no severe residual stenosis after intracoronary urokinase infusion. The formation of thrombus may be a dominant cause in the genesis of acute occlusion of the ocronary artery. The location of reocclusion was same as that of total occlusion seen in acute myocardial infarction, which may suggest that the local arterial wall abnormality is important, although it has no significant organic coronary aftery stenosis. Further studies are needed to determine what clinical or laboratory parameters predict each reocclusion. In the therapeutic standpoint, the aggressive treatment of anticoagulants might be important after coronary thrombolysis, even in patients with widely recanalized coronary artery.

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