Case Report

Arteriographic Demonstration of Coronary Artery Spasm during Thrombolysis

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A 60-year-old man with an acute inferior-wall myocardial infarction was noted, on arteriography, to have an occluded distal right coronary artery. The vessel was reopened with intravenous tissue plasminogen activator; during resolution of the clot, spasm was observed arteriographically but was successfully treated with intracoronary nitroglycerin. Follow-up arteriography showed a normal right coronary artery.

The significance of this case lies in the fact that we were able to document the occurrence of spasm during coronary thrombolysis; such documentation supports the hypothesis that spasm may be a factor in the initiation of coronary thrombosis. ***(Texas Heart Institute Journal 1988;15:52-54)***

Occlusion of a fixed stenotic lesion by coronary thrombus is widely acknowledged to play a dominant role in the pathologic sequence that leads to myocardial infarction. This concept has gained acceptance because of the results of angiographic studies performed during the first few hours of evolving myocardial infarction.[1,2]

In the foregoing case, the occurrence of coronary spasm during coronary thrombolysis was demonstrated arteriographically, thus raising the possibility that the thrombosis itself may have been initiated by coronary spasm.

Case Report

A 60-year-old man was admitted to our hospital with a 2-hour history of cardiac chest pain and dyspnea. He had no previous history of exertional or resting angina. His cardiac risk factors included a 60 pack/yr history of cigarette smoking. Upon admission, the electrocardiogram showed 1-mm ST-segment elevation in leads II, III, and aV_F, with 1-mm ST-segment depression in leads V_1 to V_4. The patient was enrolled in the thrombolysis in myocardial infarction (TIMI) protocol, which consisted of immediate cardiac catheterization, with coronary arteriography and left ventriculography, as well as the administration of tissue plasminogen activator, since the infarct-related artery was totally occluded and did not respond to intracoronary nitroglycerin.

Left ventricular angiography disclosed posterobasal akinesia and lateral wall hypokinesia. Coronary arteriography showed the left circumflex artery to be small and nondominant. The distal right coronary artery was totally occluded beyond the origin of the posterior descending artery (Fig. 1). Because intracoronary administration of 200 μg of nitroglycerin produced no change in the vessel, tissue plasminogen activator was given, at the rate of 60 mg during the first hour and 20 mg/hr during the next 2 hours. Sixty minutes after this treatment was started, reperfusion was established, with 25% patency in the involved segment (Fig. 2). At 90 minutes, however, the vessel became reoccluded; 1 minute later, it reopened spontaneously but continued to have 99% stenosis. Administration of 200 μg of nitroglycerin into the right coronary artery relieved the spasm and restored the luminal status to 25% patency.

The creatine kinase value peaked at 946 U/ml, and the myocardial band was 92 U/ml at 24 hours. The LDH I and SGOT levels peaked at 448 and 182 U/ml, respectively, at 32 hours.

Key words: *Coronary artery spasm; coronary thrombosis; thrombolysis*

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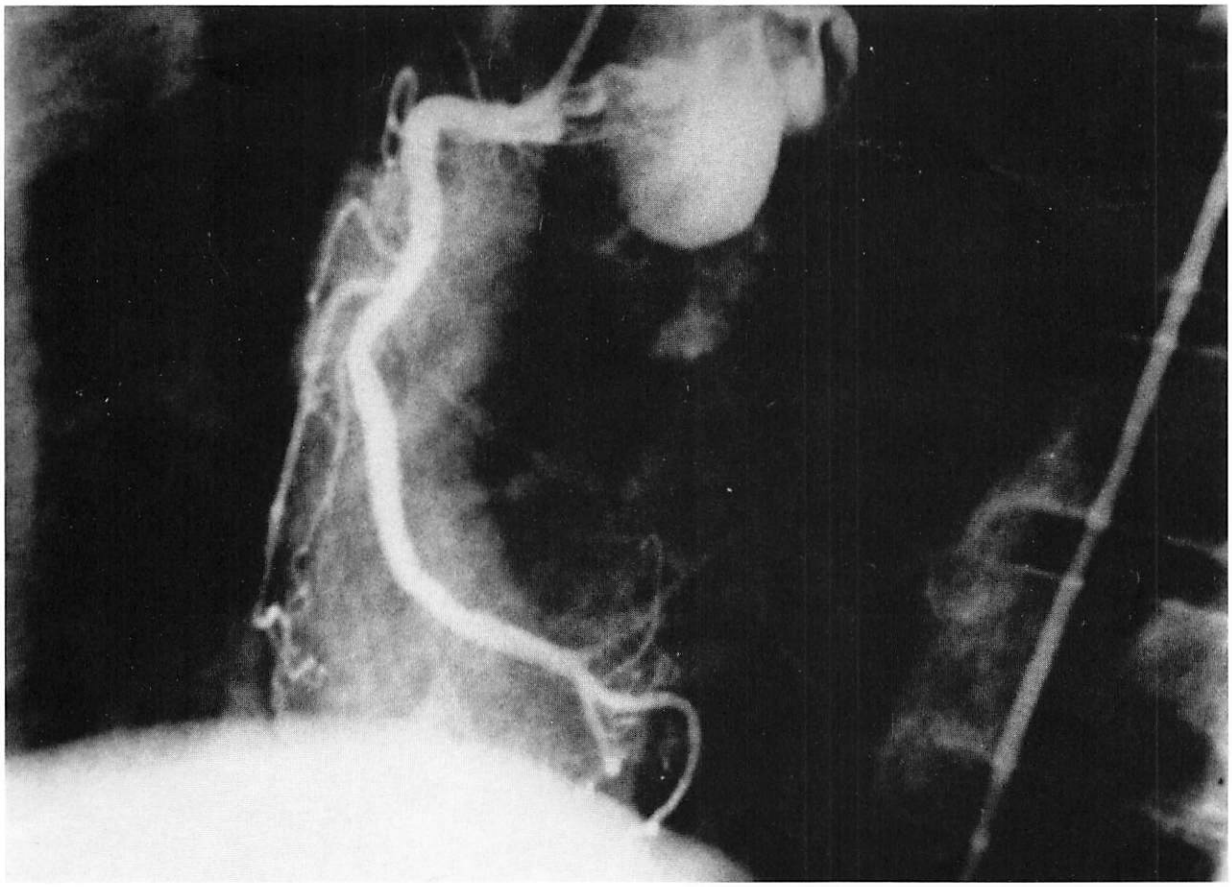

Fig. 1 *Right anterior oblique view of the right coronary artery, showing distal occlusion.*

During follow-up arteriography on day 8, the spasmodic right coronary segment was seen to be without significant plaque or narrowing. A plaque was detected further proximally, however, in the mid right coronary artery. Hypokinesia of the posterobasal wall was also present.

Discussion

In this case, the occurrence of coronary spasm during coronary thrombolysis raised the question of whether the thrombosis itself might have been initiated by coronary spasm, either at the site of a normal coronary artery or within an atherosclerotic lesion too small to be visualized arteriographically. This hypothesis is supported by the fact that the coronary artery occlusion did not respond to sublingual nitroglycerin but did respond to tissue plasminogen activator. During resolution of the clot, recurrent spasm was observed angiographically and was successfully treated with sublingual nitroglycerin.

Angiographic studies in patients with chest pain and electrocardiographic ST-segment elevation without Q waves have revealed that, in the vast majority of cases, the artery that supplies the area of evolving infarction is totally occluded.[1,2] Moreover, the intravenous administration of thrombolytic agents into the occluded coronary artery produces patency in a high percentage of cases.[3]

Dalen[4] has suggested that, in cases in which coronary thrombus has been documented by arteriography in a patient with evolving myocardial infarction, such thrombus may be due to coronary spasm. He further suggested that, by causing stasis owing to complete or near-complete obstruction of an atherosclerotic coronary artery, persistent coronary spasm may produce in situ thrombosis proximal to the site of obstruction. In a study of preinfarction angina by Maseri and associates,[5] one patient in whom spasm was observed at the outset of infarction died 6 hours later. At postmortem examination, a fresh laminar thrombus was found at the site of the spasm. In the same study, complete thrombotic occlusion of the branch shown to undergo vasospasm was documented angiographically in two patients.

In canine experiments, Gertz and colleagues[6] have shown that, after partial coronary artery ligation resulting in a 40% to 50% reduction in luminal diameter, platelet aggregation and endothelial damage occur proximal to, and at the site of, constriction. The endothelial damage may be caused by the

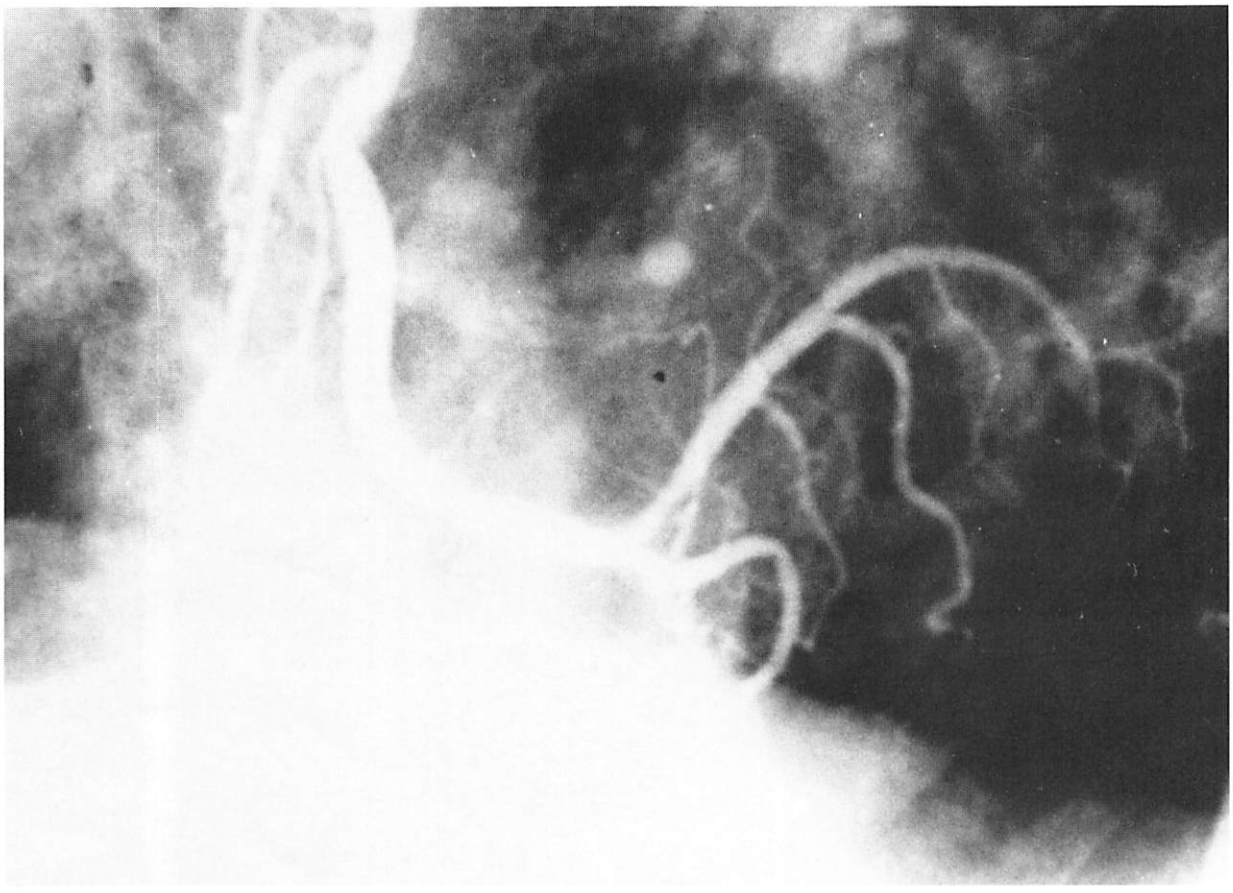

Fig. 2 *Right anterior oblique view of the right coronary artery after the intravenous administration of tissue plasminogen activator.*

increased shear rate associated with partial fixed obstruction or spasm. Thromboxane A_2, which is synthesized by platelets from membrane arachidonic acid, is a powerful vasoconstrictor that may produce spasm. Increased synthesis of thromboxane A_2 by aggregating platelets, cholesterol-related sensitization of the arteries (promoting constriction), and decreased generation of prostacyclin by atherosclerotic plaques may therefore make the site of the lesion especially vulnerable to spasm.

Interestingly, our patient had a recurrence of spasm during the lysis of thrombus; this fact supports the notion that platelet degradation products originating from thrombus may promote spasm in a predisposed area of the arterial wall. In patients in whom successful recanalization of the infarct-related artery has been achieved by thrombolytic therapy, such spasm may play a role in re-occlusion of the same artery in the hours following the therapy. One way to prevent this from happening may be to continue the patient on intravenous nitroglycerin infusion in addition to intravenous heparinization during the first few days following thrombolytic therapy.

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