Case Report

Arteriographic Demonstration of Coronary Artery Spasm during Thrombolysis

Amin H. Karim, MD
William Bennett, MD
Saul J. Silver, MD
Neal S. Kleiman, MD
Dennis Goodman, MD
Jacques Heibig, MD

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*

From: *The Department of Medicine, Section of Cardiology, Baylor College of Medicine, Houston, Texas*

Address for reprints: *Jacques Heibig, MD, Director, Cardiac Catheterization Laboratory, Veterans' Administration Medical Center Hospital, Houston, TX 77211*

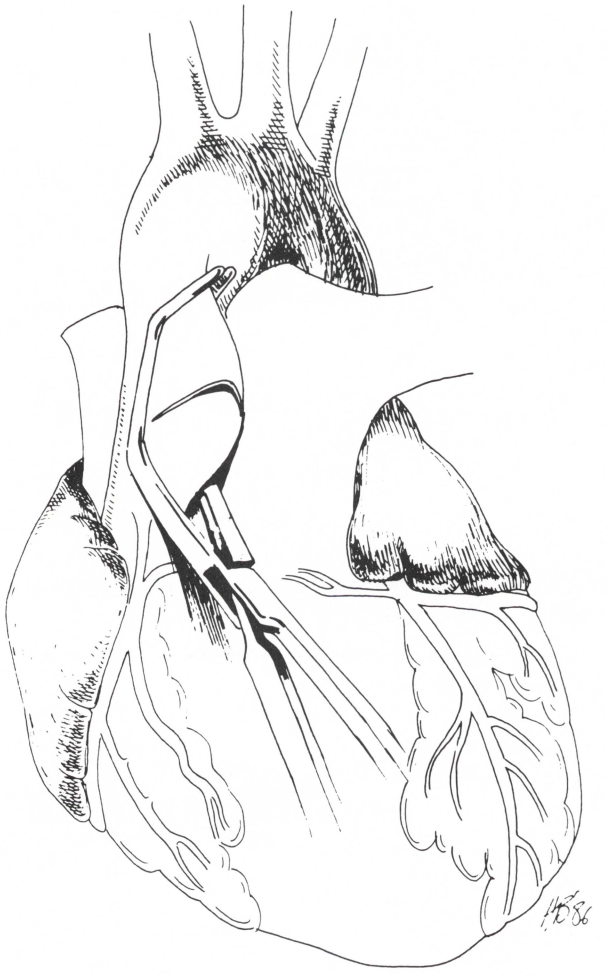

Fig. 2 *Hemostasis was achieved with a partial occlusion clamp, thus allowing adequate cardiac output.*

have been possible, since these injuries have involved incomplete transection of the aortic wall, and sufficient adventitia has been left to support the aortic blood column. In these cases, varying amounts of blood have been encountered in the pericardium at the time of exploration, but cardiac arrest has not ensued.

Unless immediate cardiopulmonary bypass is available, control and repair of the aorta after free rupture can be accomplished only if the tear is partial and if the hemorrhage can be controlled with a partial occlusion clamp. Moreover, an adequate aortic lumen must exist behind the clamp, to allow an adequate stroke volume for the reestablishment of blood pressure and tissue perfusion (Fig. 2).

The foregoing case indicates that long-term survival is indeed possible after this type of trauma and that resuscitative efforts should not be abandoned once the serious nature of the injury is established.

Acknowledgment

The authors wish to thank Haluk Boneval, MD, for providing the illustrations and Ms. Carmella Rebuck for her patience in typing this manuscript.

References

1. Parmley LF, Mattingly TW, Marrion WC, Jahnke EJ Jr. Nonpenetrating traumatic injury of the aorta. Circulation 1958; 17:1086-1101.
2. Sevitt S. The mechanisms of traumatic rupture of the thoracic aorta. Br J Surg 1977; 64:166.
3. Symbas PN, Tyras DH, Ware RE, Diorio DA. Traumatic rupture of the aorta. Ann Surg 1973; 178:6-12.
4. Charles KP, Davidson KG, Miller H, Caves PK. Traumatic rupture of the ascending aorta and aortic valve following blunt chest trauma. J Thorac Cardiovasc Surg 1977; 73:208-211.
5. Cleveland JC, Cleveland RJ. Successful repair of aortic root and aortic valve injury caused by blunt chest trauma in a patient with prior aortic dissection. Chest 1974; 66:447.
6. Cuadros CL, Hutchinson JE, Mogtader AH. Laceration of a mitral papillary muscle and the aortic root as a result of blunt trauma to the chest. J Thorac Cardiovasc Surg 1984; 88:134-140.
7. Lundell CJ, Quinn MF, Finck EJ. Traumatic laceration of the ascending aorta: Angiographic assessment. AJR 1985; 195:715-719.
8. Passaro E, Pace WB. Traumatic rupture of the aorta. Surgery 1959; 46:787.
9. Kirsch MM, Orringer MB, Behrendt DM. Management of unusual traumatic ruptures of the aorta. SGO 1978; 146:365-370.
10. Vasko JS, Raess DH, Williams TE. Nonpenetrating trauma to the thoracic aorta. Surgery 1977; 82:403-406.
11. Beel T. Traumatic rupture of the thoracic aorta. Ann Emerg Med 1980; 9:483-486.
12. Reyes LH, Rubio PA, Korompai FL, Guinn GL. Successful treatment of transection of the aortic arch and innominate artery. Surgery 1975; 19:468-471.

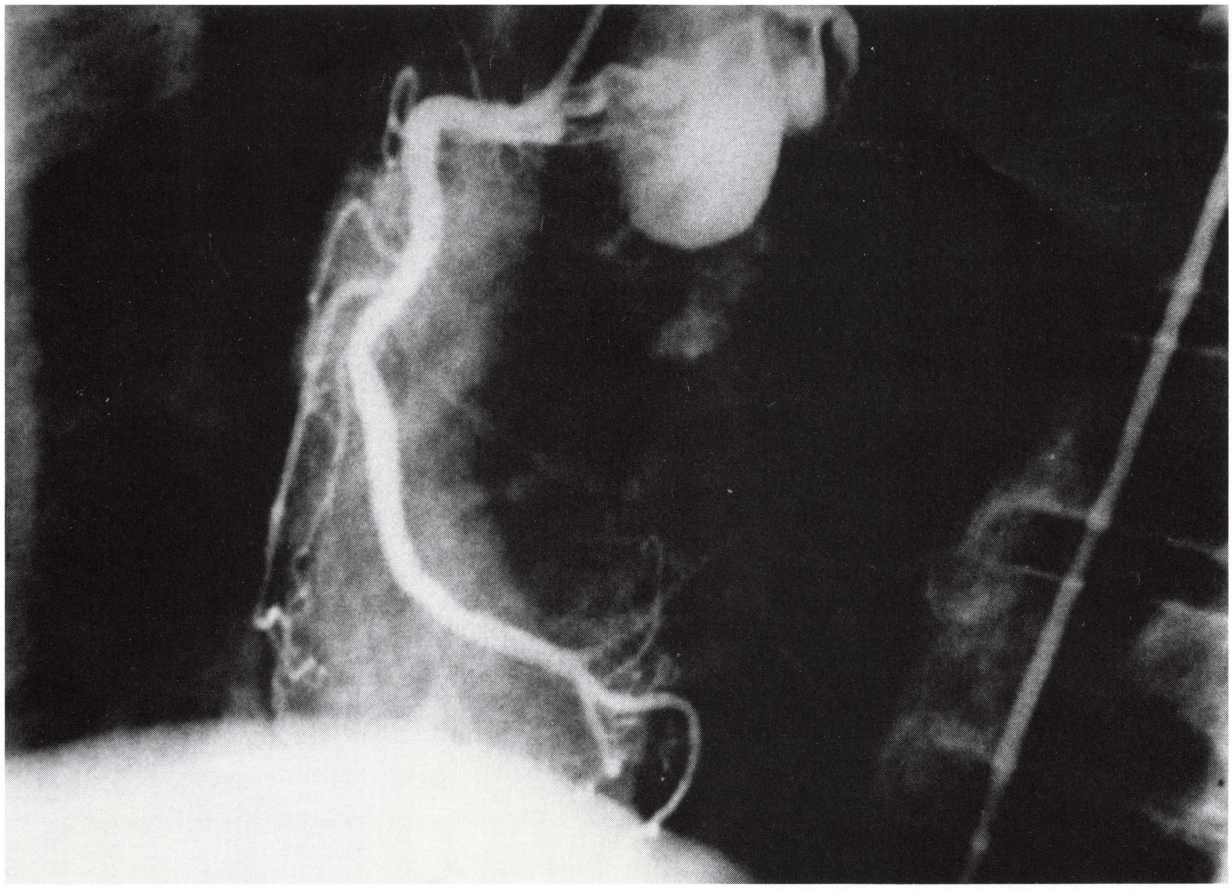

Fig. 1 *Right anterior oblique view of the right coronary artery, showing distal occlusion.*

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

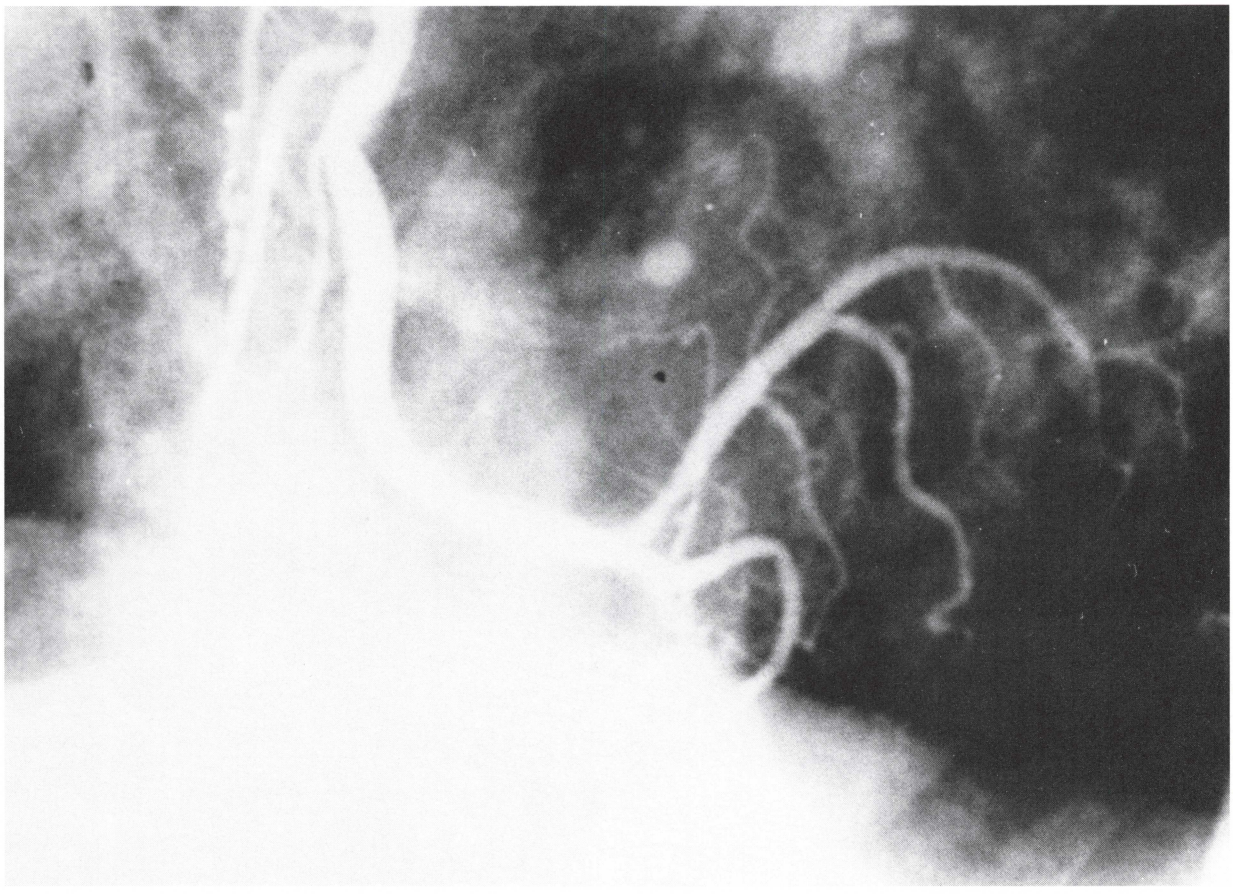

Fig. 2 *Right anterior oblique view of the right coronary artery after the intravenous administration of tissue plasminogen activator.*

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.

References

1. Oliva PB, Breckinridge JC. Arteriographic evidence of coronary arterial spasm in acute myocardial infarction. Circulation 1977; 56:366.
2. DeWood MA, Spores J, Notske R, Mouser LT, Burroughs R, Golden MS, Lang, HT. Prevalence of total coronary occlusion during early hours of transmural myocardial infarction. N Engl J Med 1980; 303:897.
3. Rentrop P, Blanke H, Karsch KR, Kaiser H, Kosterling H, Leitz K. Selective intracoronary thrombolysis in acute myocardial infarction and unstable angina pectoris. Circulation 1981; 63:307.
4. Dalen JE, Ockene IS, Alpert JS. Coronary spasm, coronary thrombosis, and myocardial infarction: A hypothesis concerning the pathophysiology of acute myocardial infarction. Am Heart J 1982; 104:1119.
5. Maseri A, L'Abbate A, Baroldi G, Chierchia S, Marzilli M, Ballestra AM, Severi S, Parodi O, Biagini A, Distante A, Pesola A. Coronary vasospasm as a possible cause of myocardial infarction. N Engl J Med 1978; 299:1271-1277.
6. Gertz SD, Merin G, Pasternak RC, Gotsman MS, Blaumanis OR, Nelson E. Endothelial cell damage and thrombosis following partial coronary arterial constriction: Relevance to the pathogenesis of myocardial infarction. Isr J Med Sci 1978; 14:384.