Acute Coronary Thromboembolization in Unstable Angina Without Subsequent Myocardial Infarction

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A 58 year old man underwent coronary arteriography for unstable angina. During the catheterization, distal embolization of a nonoccluding intracoronary thrombus formed at the site of a subtotal atherosclerotic occlusion of the left anterior descending coronary artery and was documented angiographically. This event, which appears unique, was not associated with subsequent myocardial infarction or any apparent morbidity.

Coronary embolization is an unusual event and has been reported to cause severe angina, myocardial infarction and death (1,2). It has been described in patients with infective endocarditis, mitral valve prolapse, aortic stenosis and cardiomyopathy and in patients undergoing cardiac catheterization (1,3). Most often the source of the embolus is outside the coronary circulation itself and different from the coronary thrombi associated with myocardial infarction. In this case, however, the origin of the embolus seemed clearly intracoronary and the embolization did not cause any apparent morbidity.

Case Report

A 58 year old man presented for cardiac catheterization with new daily effort angina. He had had an inferior myocardial infarction 7 years earlier. A 12 lead electrocardiogram revealed small Q waves in leads II, III, aVF, V₆ and V₉. A treadmill exercise test using the Bruce protocol 1½ months before hospital admission revealed that the patient exercised 9 minutes and developed 1.5 mm upsloping ST segment depression with typical angina.

Coronary angiography. During a first left coronary injection using the Judkins technique, a subtotal proximal left anterior descending artery narrowing with an intraluminal filling defect just distal to the obstruction was well outlined (Fig. 1A). Several minutes later, a repeat coronary injection demonstrated the persistence of this filling defect (Fig. 1B), which then was dislodged and traveled down the left anterior descending artery (Fig. 1C). At the end of this same injection of contrast dye, the thrombus became lodged at the distal bifurcation of the diagonal artery (Fig. 1D). A repeat injection of the left coronary artery revealed that the previously noted filling defect at the atherosclerotic lesion had disappeared (Fig. 1E).

The patient did not complain of angina during the angiogram and no ST segment change was noted. The patient was then followed up in the coronary care unit for 3 days with serial electrocardiograms and serum cardiac enzyme determinations demonstrating no evidence of acute myocardial infarction. He subsequently underwent coronary artery bypass surgery for his severe angina and triple vessel disease and is currently doing well.

Discussion

The finding of an occluding intracoronary thrombus in association with acute transmural myocardial infarction is well documented (4) and has prompted the increasing use of streptokinase to reestablish coronary flow. In patients with unstable angina and nontransmural myocardial infarction, this has been a much less consistent finding. Vetrovec et al. (5) studied 129 patients with a history of unstable angina of 1 month or less and found only 8 patients (6%) with angiographic findings consistent with intracoronary thrombus. In one patient, dislodgment and passage of the clot into the left circumflex coronary artery was noted in association with evidence of myocardial infarction.

Source of coronary filling defect. We considered several explanations for the filling defect seen in our patient, other than its representing an endogenously formed intracoronary thrombus. First, an air embolus from the coronary catheter was a possibility, but because the filling defect persisted in the same location between two coronary injections performed several minutes apart, this is highly un-
Figure 1. Coronary angiograms of the left coronary artery in right and left anterior oblique projections (A through E). The arrow in A indicates an intraluminal coronary filling defect in the left anterior descending artery distal to a subtotal stenosis. The arrows in C and D show the progression of the embolus and its lodging in a distal diagonal branch (D). In E, the previous coronary filling defect has disappeared.
likely. Second, a clot might have been injected into the coronary artery from the catheter itself, although as the coronary artery filled, the filling defect was initially noted distal to a very tight stenosis. A catheter-induced clot of this size probably would not have passed through such a tight narrowing without impacting therein and totally occluding the vessel. Finally, we wondered whether the clot truly became spontaneously dislodged, or whether the force of the dye injection and any related changes in vascular tone might have caused this event.

Lack of myocardial necrosis. We are uncertain why the clot, which became lodged in and totally occluded a distal branch of the left anterior descending coronary artery, did not cause detectable myocardial damage. It is possible that after embolization the clot underwent spontaneous lysis, allowing flow to be reestablished. In addition, the area of myocardium supplied by that small terminal epicardial branch may have received adequate blood supply from collateral vessels or surrounding capillary networks, or both, such that viability was maintained. Finally, there may have been a very small area of myocardial necrosis that failed to produce sufficient electrocardiographic or enzymatic abnormalities to be detected.

Clinical implications. In view of the increased frequency of manipulation of coronary obstructions and stenoses, we believe that our findings are significant. Often during streptokinase infusions, an intracoronary thrombus is seen to diminish in size and then suddenly disappear (6). It is possible that this "disappearance" is caused by distal embolization. It is impossible to assess additional damage caused by such an event because it always occurs in patients who have associated electrocardiographic and serum enzyme evidence of a myocardial infarct in that distribution. In patients undergoing percutaneous transluminal coronary angioplasty, where there is most often no infarct demonstrable in the distribution of the affected vessel, the possible danger of distal embolization of arterial "debris" may be an issue.

Finally, the mechanisms by which stable angina progresses to unstable angina and myocardial infarction are still not completely understood. A recent report (7) described a patient with normal coronary arteries who developed severe spasm, thrombus formation and myocardial infarction. Our patient may well represent the opposite end of the spectrum, with a severe atherosclerotic narrowing and associated intraluminal thrombus that did not cause myocardial infarction, presumably because flow past this lesion complex was not interrupted.

References