*A 47-year-old woman was transferred to Harper Hospital for recurrent, severe post-AMI angina refractory to maximal medical therapy, 4 days after sustaining a non-Q-wave inferior AMI associated with a diminutive increase in CK to 300 IU. On the day of transfer, she had recurrent rest angina associated with new horizontal ST-segment depression and upright T waves in leads $V_2$ through $V_5$, believed to represent "remote" anterior subendocardial ischemia or recurrent anterior non-Q-wave AMI (Fig. 2, top left). Blood pressure was 110/70 mm Hg, pulse 72 beats/min and lungs were clear. Urgent coronary angiography showed the left coronary system to be normal. Right coronary artery injection disclosed a high-grade stenotic lesion (at least 95% luminal diameter narrowing) involving the midportion of the vessel (Fig. 2, top right). Serial inflations with a 2.0-mm Simpson Robert balloon catheter dilated the right coronary artery stenosis (Fig. 2, bottom left). Precordial ST-segment depression abated and abnormal tall R waves did not evolve (Fig. 2, bottom right).*

Recently, we described the early electrocardiographic findings of posterior AMI in 27 patients randomized to the Diltiazem Reinfarction Study of MB-CK-confirmed non-Q-wave AMI.[5] All patients initially showed a pattern of precordial ST-segment depression with upright T waves, which was believed to represent "anterior" non-Q-wave AMI; however, before discharge, abnormal right precordial R waves indicative of posterior AMI evolved in all patients. Coronary angiography was not performed on these study patients by trial design.

We have shown 2 contrasting examples of early posterior AMI due to isolated high-grade 1-vessel disease of a circumflex and right coronary artery, respectively. Each case showed significant early precordial ST-segment depression in the absence of left anterior descending coronary artery disease. These findings emphasize that precordial ST-segment depression with upright T waves due to circumflex or right coronary artery obstruction may produce an early electrocardiographic "current of injury," which is projected as reciprocal precordial ST-segment depression (posterior ST-segment elevation). Such patients may have occluded or subtotally occluded infarct-related coronary arteries, and should not be considered ineligible for acute thrombolytic therapy because of the absence of electrocardiographic ST-segment elevation.

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# Dissolution of Angiographically Detected Intracoronary Thrombus for Unstable Angina Pectoris After Aspirin Therapy

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Atherosclerotic plaque disruption with nonocclusive coronary artery thrombosis plays a major role in the pathogenesis of unstable angina pectoris.[1] Progression to cardiac death or nonfatal acute myocardial infarction (AMI) occurs in more than 10% of these patients within 3 months[2] and is not prevented by conventional medical treatment with nitrate, $\beta$-blocking or calcium antagonist drugs. Two multicenter, randomized, double-blind, placebo-controlled trials have shown a 50% reduction in mortality and nonfatal AMI rates with aspirin therapy,[2,3] presumably from inhibition of platelet-dependent thrombus formation. This report provides arteriographic evidence of thrombus dissolution in patients with unstable angina whose condition improved on aspirin therapy.

***Case 1:*** *A 62-year old man had recurrent postinfarction angina after a non-Q-wave inferior wall AMI. Angina did not recur after aspirin was added to the antianginal regimen of isosorbide dinitrate, metoprolol and diltiazem. Cardiac catheterization revealed a 90% proximal right coronary artery diameter reduction with associated thrombus (Fig. 1). Repeat catheterization 1 month later revealed a 40% ulcerated stenosis without thrombus.*

***Case 2:*** *A 67-year-old man had a 15-minute episode of chest pain associated with inferior ST-segment elevation 10 days after a non-Q-wave inferior wall infarction. Angina did not recur after aspirin was added to the antianginal regimen of isosorbide dinitrate and diltiazem. Cardiac catheterization revealed a 90% middle right coronary artery diameter stenosis with associated thrombus (Fig. 2). Repeat catheterization 1 month later showed a 20% ulcerated stenosis without thrombus.*

Antiplatelet, anticoagulation and fibrinolytic agents are new medical therapies for unstable angina. These treatment strategies are based on the theory that unstable angina is 1 end of a continuum of acute ischemic syndromes including subendocardial AMI, transmural AMI and ischemic sudden death, which result from atherosclerotic plaque disruption and arterial thrombosis.[1] Which syndrome develops may depend

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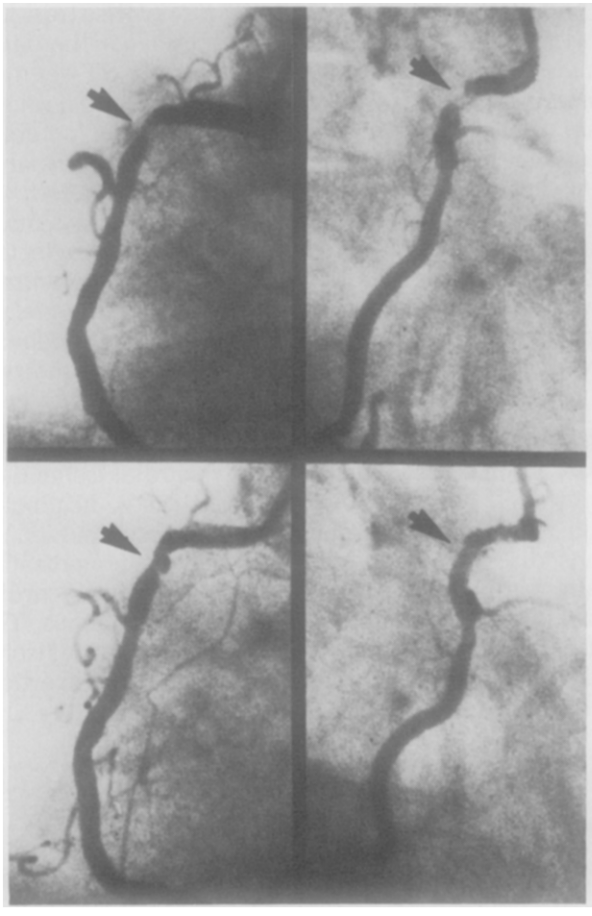

**FIGURE 1. Left anterior oblique (*left*) and right anterior oblique (*right*) views of a right coronary artery with intracoronary thrombus before treatment (*top*) and after 1 month of aspirin therapy (*bottom*). An ulcerated 40% stenosis without thrombus persists.**

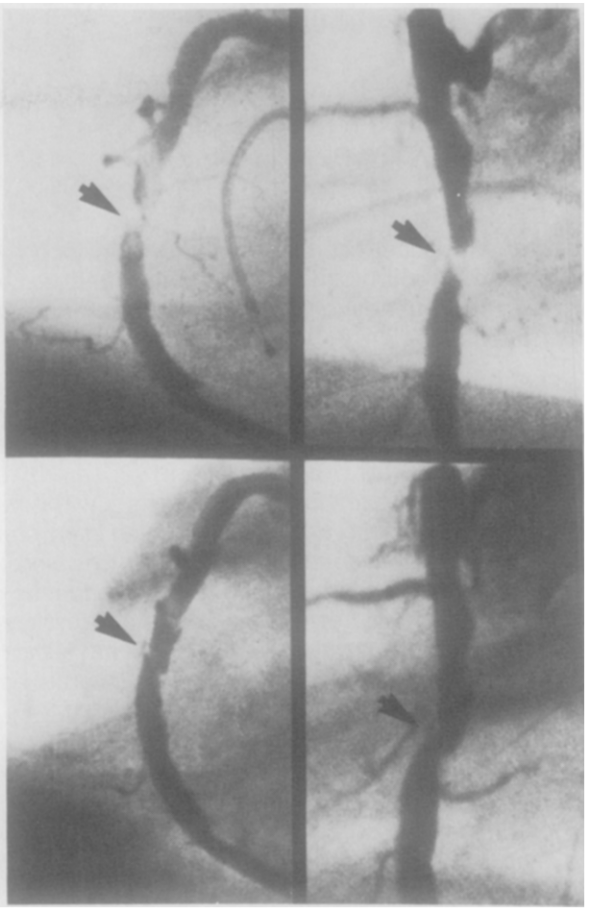

**FIGURE 2. Left anterior oblique (*left*) and left posterior oblique (*right*) views of a right coronary artery with intracoronary thrombus before treatment (*top*) and after 1 month of aspirin therapy (*bottom*). An ulcerated 20% stenosis without thrombus persists.**

on the suddenness, completeness and duration of blood flow deprivation caused by platelet thrombi.[1] Dramatic decreases in mortality and nonfatal AMI have been seen in patients with unstable angina treated with aspirin.[2,3] An explanation is that coronary artery thrombosis often occurs adjacent to atherosclerotic plaques of less than 60% diameter stenosis.[4] Because the ischemic syndromes in the 2 patients in this report stabilized on aspirin therapy, we avoided the risks of angioplasty in the setting of angiographically detected intracoronary thrombus.[5] Cardiac catheterization performed 1 month later in each patient revealed resolution of the thrombus and a widely patent artery. Thus, neither emergency nor elective angioplasty was performed.


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