Relation of Acute Myocardial Infarction to Cocaine Abuse

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It is estimated that 10 million Americans have used cocaine and that 5 million use it regularly.[1] Although cocaine was first introduced in 1884 as a local anesthetic, little information appeared on the cardiovascular effects of cocaine use until recently. Cocaine abuse has been associated with tachycardia, systemic hypertension, ventricular arrhythmias, acute myocardial infarction (AMI) and sudden death.[2-7] Coleman et al[2] reported the first case of AMI associated with illicit use of cocaine; however, the patient was presumed to have underlying coronary artery disease, although he did not undergo coronary angiography. Kossowsky and Lyon[3] described 6 patients with AMI attributed to cocaine abuse. Four of their patients had some degree of atherosclerotic narrowing of the coronary arteries by angiography or autopsy and 2 did not have cardiac catheterization. Schachne et al[4] described another patient whose coronary arteries were normal. We describe another cocaine user who had AMI.

A 38-year-old white man had been well until age 32 years (April 1979), when he had a documented inferior wall AMI. After an uncomplicated course, he was transferred to the Bronx Veterans Administration Medical Center 3 weeks later. Blood pressure on admission was 120/80 mm Hg and serum cholesterol was normal. Cardiac catheterization and selective coronary angiography with left ventriculography was performed. The coronary arteries and the left ventricle were normal. Provocative maneuvers did not produce spasm. After discharge, he was asymptomatic but continued to use cocaine and heroin sporadically. Before AMI, he had increased his cocaine use to 3 times per week. He was readmitted to the coronary care unit 6 months later (November 1979) with an extensive anterior lateral wall AMI. Chest pain occurred 45 minutes after intranasal administration of cocaine. Selective coronary angiography again showed normal coronary arteries. The left ventricular ejection fraction was 68%. The infarctions were believed to have been induced by coronary artery spasm, but this could not be demonstrated by repeat ergonovine administration. He has been followed up by the cardiology service for the past 6 years. He has not used drugs for the past 5 years, although he continues to smoke cigarettes heavily and is a heavy alcohol binge drinker. He has not had chest pain or other cardiovascular symptoms since his last AMI. His blood pressure is 130/80 mm Hg. Left ventricular function by gated blood pool scintigraphy in August 1984 showed an ejection fraction of 53%.

Our patient is similar to the case reported by Schachne et al.[4] He also shares a common feature with several of the patients described by Kossowsky and Lyon[3] in that he was a mixed-substance abuser. Cocaine is a potent vasoconstrictor. It constricts blood vessels and prevents its own absorption.[5] AMI in any young patient who does not have risk factors should raise the suspicion of the possibility of coronary spasm related to recreational cocaine use. The prognosis for a patient who survives the initial infarction and has good left ventricular function appears to be excellent if cocaine usage is discontinued.

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Coronary Arterial Findings After Accidental Death Immediately After Successful Percutaneous Transluminal Coronary Angioplasty

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The mechanism by which percutaneous transluminal coronary angioplasty (PTCA) achieves its results remains minimally documented. Plaque compression and redistribution was the initial postulate for the success of arterial dilatation in peripheral atherosclerotic disease. Subsequent animal and human cadaver experiments[1] and human postmortem examinations[2,3] suggest plaque splitting and disruption with stretching of the nonatherosclerotic arterial wall. We present the pathologic findings in a patient who died immediately after a technically successful PTCA after an inadvertent intracoronary injection of lidocaine.

A 65-year-old woman with a 6-year history of angina pectoris was admitted with prolonged ischemic chest pain. Anteroseptal ST-segment depression was present, but no Q waves developed despite a moderate increase in cardiac en-

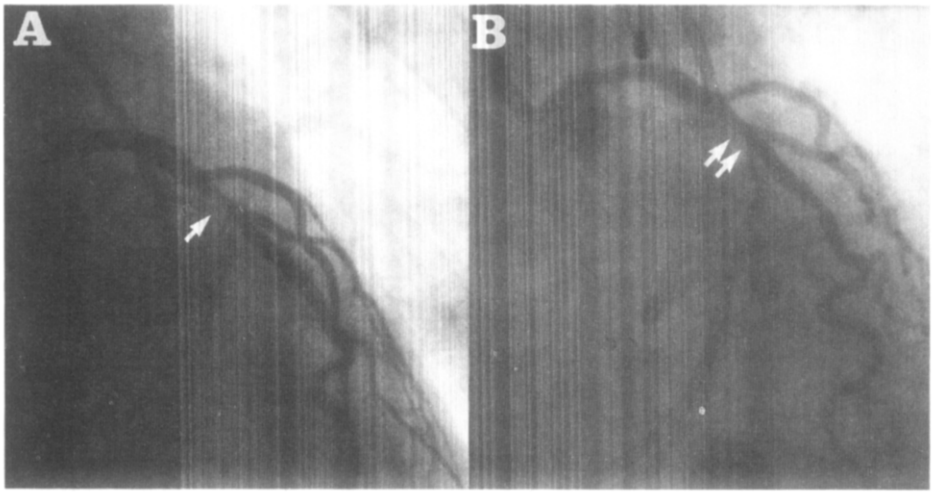

FIGURE 1. Left anterior descending coronary artery stenosis (**arrows**) (right anterior projection). **A,** before angioplasty; **B,** after angioplasty.

zyme levels. Cardiac catheterization after 10 days of continuing unstable angina revealed that the right coronary artery had an 85% narrowing of luminal diameter and the left circumflex artery was completely occluded. The left anterior descending artery (LAD) was narrowed 65% as seen in the right anterior oblique projection (Fig. 1A). The inferior left ventricular wall was moderately hypokinetic.

We decided to proceed to PTCA of the LAD and right coronary artery. PTCA of the LAD was accomplished using a 20–35 Meditech balloon and steerable guidewire system. Three dilatations to a maximal inflation pressure of 8 atm and total occlusion time of 135 seconds reduced the mean transstenotic gradient from 70 to 29 mm Hg. The postdilatation angiogram showed a reduction in diameter narrowing to 40% (Fig. 1B). In attempting a left anterior oblique view, 7 to 10 ml of a nonradiopaque fluid was injected into the left coronary artery. This had been mistaken for contrast medium and was later identified as 2% lidocaine. After 2 minutes of increased PR interval and QRS width, complete atrioventricular block developed, with no subsidiary escape rhythm. Resuscitative measures were unsuccessful.

Postmortem angiography confirmed a satisfactory angiographic result of PTCA. At autopsy an inferior infarct was found that was about the same age as the admission infarct. At the PTCA site, a split extended through the atheromatous plaque to the media with dissection and lifting of the plaque from the media (Fig. 2). A hemorrhagic area within the plaque opposite the split had an appearance that suggested that its occurrence predated PTCA.

In this patient, technically successful PTCA was achieved by splitting of the atheromatous plaque, and the patient died from an unrelated cause. Of the few previous reports of the pathologic findings in patients who died early after PTCA, plaque splitting was a frequent finding. However, most patients died from complications of acute myocardial infarction initiated by the PTCA.[2–4] Pathologic changes observed late after PTCA have been variable. An intense fibrocellular proliferation almost occluding the LAD lumen was found in 1 patient who died during repeat PTCA.[5]

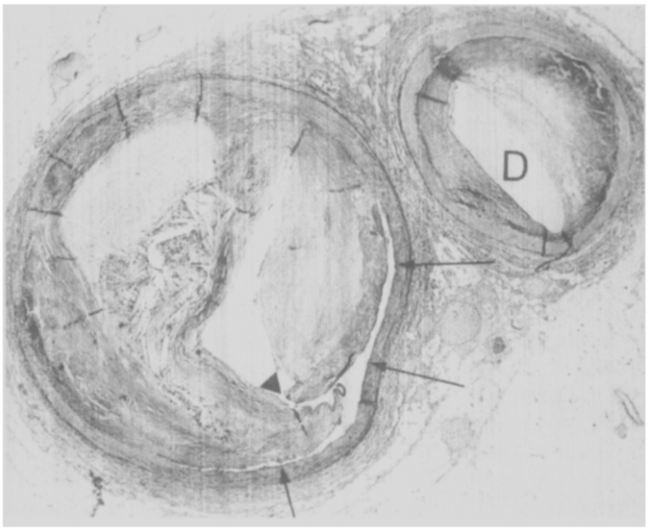

FIGURE 2. Cross section through the proximal part of the left anterior descending artery and adjacent diagonal branch (D). An atherosclerotic plaque with hemorrhage is narrowing the lumen. The plaque opposite shows disruption and splitting (**arrowhead**) and is dissected and lifted from the media (**arrows**). Apart from an atherosclerotic narrowing of the lumen, the diagonal branch does not show other changes.

Three other patients[4] showed no pathologic changes attributable to PTCA, although these were technically successful, and only 1 patient had recurrence of mild angina.

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