CASE REPORT

Coronary Spasm, Variant Angina, and Recurrent Myocardial Infarctions

ALLEN D. JOHNSON, M.D., AND JOHN H. DETWILER, M.D.

SUMMARY  A 24-year-old male student had three myocardial infarctions, one prior to and two following the angiographic documentation of normal coronary arteries. A spontaneous episode of variant angina prompted repeat coronary angiography, during which intravenous ergonovine caused spasm of the left anterior descending coronary artery, transient ST-segment elevation, and ischemic chest pain; the previously normal right coronary artery was found to be occluded proximally. This constellation of clinical and angiographic findings suggests that coronary spasm can cause acute myocardial infarction as well as variant angina.

THE ROLE OF CORONARY SPASM in the genesis of acute myocardial infarction is at present uncertain. Variant angina, coronary arterial spasm, and acute myocardial infarction have each been reported in patients with angiographically normal coronary arteries;** the possible interrelationships among these phenomena remain speculative and are a continuing focus for clinical investigation.

We report the case of a young man with recurrent myocardial infarctions, angiographically normal coronary arteries, variant angina, and coronary arterial spasm. The coexistence of these several features of the clinical spectrum of myocardial ischemia in the setting of normal coronary arteries lends support to a role for coronary arterial spasm as an important underlying pathophysiologic mechanism in these disorders.

Case Report

On August 12, 1974, a 24-year-old male student was admitted to the San Diego Veterans Administration Hospital complaining of two hours of substernal aching chest discomfort which began at rest. The family history was negative for coronary artery disease. The patient had smoked less than one half pack of cigarettes per day sporadically. Physical examination disclosed amputation of the right leg above the knee secondary to a war wound and healed shrapnel wounds of the left lower extremity and both upper extremities; the cardiovascular examination was normal except for an S2 gallop. A high lateral acute myocardial infarction was documented on electrocardiogram and was substantiated by serum enzyme abnormalities (fig. 1, table 1). In the hospital the patient had transient ventricular irritability, which responded to quinidine therapy. Anticoagulation was begun with warfarin sodium, and the patient was discharged three weeks after admission after a 12-hour ambulatory ECG recording revealed one premature ventricular systole and no ST-segment changes.

The patient was asymptomatic until March 1, 1975, when recurrent substernal chest discomfort occurred at rest, persisted for 30 min, and was accompanied by nausea and diaphoresis. A three day hospitalization revealed no ECG or enzyme changes. On March 26, 1975, the patient underwent elective cardiac catheterization and coronary angiography, using the brachial approach. Coronary angiography was accomplished prior to cine left ventriculography. The left ventricular cineangiogram demonstrated akinesis in the high lateral area (fig. 2). Ejection fraction was 51% and left ventricular end-diastolic pressure was 14 mm Hg. Atrial pacing for five minutes at a rate of 130 beats/min caused no chest pain, ECG changes, or elevation of left ventricular end-diastolic pressure. Coronary angiograms without prior administration of nitroglycerin or atropine (fig. 3) demonstrated no abnormalities. A glucose tolerance test, serum cholesterol, and triglyceride levels, and lipoprotein electrophoresis were all normal. He was discharged, and maintained on anticoagulant therapy and instructed in the use of sublingual nitroglycerin.

On August 12, 1975, while at rest, the patient experienced three successive episodes of chest discomfort which radiated to both arms. On admission to the hospital, an acute inferior subendocardial myocardial infarction was documented with T wave inversions in lead II, III, and aVF (fig. 1), and a typical enzyme pattern for acute myocardial infarction evolved (table 1). The patient had no further symptoms until November 4, 1975, when he was readmitted after he had had one and one-half hours of chest pain at rest. Serial electrocardiograms and myocardial enzymes revealed no evidence of recurrence of a myocardial infarction. Over the next five months, substernal chest discomfort occurred several times a month, always occurring at rest, usually following, but never during exercise or emotional stress. The pain was always relieved by sublingual nitroglycerin. He noted no nocturnal or early morning chest pain.

On April 1, 1976, the patient had an episode of substernal chest discomfort associated with nausea and diaphoresis occurring at rest following swimming; the chest discomfort persisted for one hour. He reported to a local hospital complaining of severe chest discomfort. Lead I of an electrocardiogram revealed marked ST-segment elevation (fig. 4). An intravenous line was put in place and 10 mg of morphine sulfate administered for relief of the pain prior to comple-
tion of the electrocardiogram. When taken, the ECG was unchanged from previous tracings. Serial cardiac enzymes and electrocardiograms revealed no evidence for an acute myocardial infarction.

Cardiac catheterization by the brachial technique was repeated on April 2, 1976. Left ventricular end-diastolic pressure was 13 mm Hg, the left ventricular ejection fraction was 66%, and the persistence of the high lateral wall motion abnormality was noted. Coronary arteriography performed prior to cine left ventriculography, without the administration of nitroglycerin or atropine, revealed that the right coronary artery had a narrow, irregular lumen, the distal branches of which filled by means of collateral vessels from the left coronary system; the left coronary artery was normal (fig. 3). Ergonovine maleate 0.05 mg was then administered intravenously;3 three minutes later the patient complained of his usual substernal chest discomfort; marked ST-segment elevation appeared in leads I and aVL. Injection of the left coronary artery at this time revealed intense spasm of the proximal left anterior descending coronary artery with very faint filling of the distal vessel (fig. 3). The patient's chest discomfort and ST-segment elevation resolved within 3 min of sublingual and intravenous administration of nitroglycerin. Subsequent coronary artery injections revealed resolution of the narrowed segment of the left anterior descending coronary artery but persistence of the abnormalities in the right coronary artery.

Following cardiac catheterization, treatment was initiated with chewable isosorbide dinitrate taken every four hours, and nitroglycerin ointment applied to the precordium at bedtime. The patient maintained strict and compulsive adherence to this regimen, and noted no episodes of chest discomfort for three months.

On June 23, 1976, the patient noted chest discomfort occurring at rest following an episode of working with a shovel in his garden, and was readmitted to hospital. Serial cardiac enzymes (table 1) and electrocardiograms (fig. 1) confirmed an acute anterior myocardial infarction. The patient had an uncomplicated hospital course, and subsequently was begun on oral Nifedipine,4 10 mg six times daily. Ischemic chest discomfort occurring at rest and relieved by nitroglycerin has recurred on two occasions over a three month period, with no changes in the electrocardiogram or cardiac enzymes.

### Discussion

The role of coronary spasm in the variant angina syndrome has been well established and documented in several recent reports.3,4 Since there have been no reports of coronary angiograms documenting coronary spasm during the

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**Table 1. Serum Enzyme Determinations**

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<th>DATE</th>
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Abbreviations: Serum creatinine phosphokinase (CPK) in international units, normal range less than 130; serum glutamic oxaloacetic transaminase (SGOT) in units, normal range less than 40.

*Bay-a-1040, Delbay Pharmaceutical Co., New Jersey.*
evolution of an acute myocardial infarction, the association between coronary spasm and acute myocardial infarction is not clear.\(^1\) However, reports of coronary spasm occurring in patients withdrawn from chronic industrial nitroglycerin exposure who subsequently experienced chest discomfort and acute myocardial infarction provided inferential evidence that coronary artery spasm might be responsible for some cases of acute myocardial infarction.\(^7\) The report of a patient who had catheter-induced coronary artery spasm complicated by acute myocardial infarction, and angiographically normal coronary arteries three months subsequently, suggested that spasm could be responsible for acute myocardial infarction.\(^6\) Spontaneous spasm of a dominant left circumflex coronary artery during coronary angiography has been reported in a patient who had variant angina following a subendocardial inferior myocardial infarction;\(^3\) coronary spasm was suggested to have caused the myocardial infarction in this case.

The clinical course in our patient strongly suggests that coronary arterial spasm was responsible for the myocardial infarctions and variant angina attacks. Three months following the demonstration of induced, reversible spasm in

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**Figure 2.** Left ventriculogram, PA projection, of March 25, 1975, revealing a high lateral wall motion abnormality (arrow).

**Figure 3.** Selected views from coronary angiograms of 3/26/75 and 4/2/76. Apparent spasm of the proximal left anterior descending coronary artery occurred following ergonovine administration, 0.05 mg i.v. (upper right) accompanied by ST-segment elevation in ECG lead I. Note that the right coronary artery was normal on 3/26/75, but revealed proximal occlusion and retrograde collateral filling of the posterior descending and distal right coronary arteries (arrow) on 4/2/76.
an otherwise normal left anterior descending coronary artery, associated with ST-segment elevation and chest discomfort, our patient sustained an anterior myocardial infarction. Acute inferior myocardial infarction occurring five months following demonstration of a normal right coronary artery, with angiographic documentation of total occlusion of the right coronary artery seven months following this infarct, suggests that coronary spasm might lead to permanent obliteration of the lumen of a coronary vessel. Because myocardial infarctions recur in spite of rigorous therapy with sublingual nitrates, our patient was begun on oral Nifedipine, a potent coronary vasodilator not in the nitrate family.10, 11

This case demonstrates many features of the spectrum of spasm of otherwise normal coronary arteries: recurrent myocardial infarctions, spontaneous episodes of variant angina, an induced episode of coronary artery spasm accompanied by chest pain and ST-segment elevation, and progression of a previously angiographically normal coronary artery to occlusion. The occurrence of these phenomena in our patient illustrates the potential contribution of coronary arterial spasm to the pathogenesis of myocardial ischemia in some individuals.

References

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