A 62-YEAR-OLD MAN ADMITTED INTO THE CORONARY CARE UNIT WITH PROGRESSIVE CHEST PAIN

Gary Gerstenblith, MD*

BACKGROUND

The patient was a 62-year-old man who first developed angina in 1987. He initially responded well to an intensive therapeutic regimen, but underwent coronary bypass surgery in 1994 because of symptom progression. Recurrent symptoms required a secondary coronary bypass procedure in March 2001. At that time, a left internal mammary artery (LMA) graft was placed to the left anterior descending (LAD) artery, and a Y saphenous vein graft to the distal right coronary and second LAD diagonal vessels. His usual angina pattern occurred once per week. During the several days prior to this admission, he experienced rest pain associated with dyspnea. On the night prior to admission and on the morning of admission, he rated the pain as 10 out of 10. He presented to an outside hospital and was transferred.

MEDICAL HISTORY

The patient had a history of hypertension, mixed hyperlipidemia, prior right carotid angioplasty with stent placement, paroxysmal atrial fibrillation, renal insufficiency, and one-half block bilateral claudication. His activity was usually limited by the claudication, rather than dyspnea or chest pain.

CURRENT MEDICAL TREATMENT

The patient's outpatient regimen included aspirin 325 mg per day, clopidogrel 75 mg per day, sotalol 80 mg twice a day, rabeprazole 20 mg twice a day, warfarin 5 mg per day, nitroglycerin patch 0.4 mg per hour (12 hours on, 12 hours off), sublingual nitroglycerin 0.4 mg as needed, erythropoietin 2500 units per week, fenofibrate 145 mg per day, lisinopril 40 mg per day, and amlodipine 10 mg per day. He also had taken a high-dose statin, but recently stopped because of myalgias.

FAMILY HISTORY

The patient's mother experienced the onset of coronary disease at age 58. His father did not have known coronary disease. He had 1 brother and 1 sister, both of whom had hypertension and elevated cholesterol but no known coronary artery disease.

SOCIAL HISTORY

The patient smoked 2 packs of cigarettes a day for 40 years and stopped smoking 2 years ago. He consumed alcohol occasionally.

PHYSICAL EXAMINATION

The patient had continued chest discomfort on arrival. His vital signs were: temperature, 97.2°F; respiration rate, 18 respirations per minute; pulse, 64
beats per minute; and blood pressure, 141/66 mm Hg. Further examination revealed fair dentition, and no xanthelasmas, edema, or elevated central venous pressure. There were bilateral carotid bruits and endarterectomy scars, a 2/6 systolic murmur, and diminished left-sided pedal pulses. The patient’s chest was clear. His abdomen was soft without tenderness. The neurologic exam was grossly within normal limits.

**REVIEW OF SYSTEMS**
A review of systems was negative.

**LABORATORY FINDINGS**
Initial laboratory tests were as follows: potassium, 5.4 mEq/L; creatinine, 1.8 mg/dL; international normalized ratio, 1.4; peak troponin level, 4.2; peak creatinine kinase myocardial band level, 29; low-density lipoproteins, 120; high-density lipoproteins, 36; and triglycerides, 257. Other laboratory values were within normal limits. An electrocardiogram (ECG) demonstrated sinus bradycardia at 58 beats per minute with an inferior myocardial infarction pattern, right bundle branch block, ST elevation in the inferior leads, and ST depression in the lateral leads. The ECG demonstrated moderate inferior hypokinesis with overall normal systolic function. There was moderate concentric left ventricular hypertrophy and mild aortic valve sclerosis and thickening.

**IMPRESSION AND DIAGNOSIS**
The patient had multiple risk factors and known coronary artery disease. His symptoms were consistent with acute coronary syndrome (ACS) with increased risk because of rest pain on an already adequate regimen, prior aspirin use, dynamic ECG changes, and elevated cardiac markers.

ACS can present with new or changed ischemic chest pain. It can be further characterized as unstable angina—in which case a myocardial infarction is subsequently ruled out—or as ST-elevation myocardial infarction, or non–ST-elevation myocardial infarction (NSTEMI). Because it is not possible to know at the time of presentation what the subsequent cardiac enzyme values will be, the unstable angina and NSTEMI patients have traditionally been studied as 1 group in clinical trials. Thus, guidelines for their care are concordant. In patients presenting with increased risk factors and suitable anatomy, outcomes are usually superior if they receive an early invasive strategy consisting of catheterization and revascularization.

**TREATMENT PLAN**
The patient was taken to the Cardiac Catheterization Laboratory, where the LIMA graft was determined to be patent and inserted distal to the first diagonal vessel. Ninety percent stenoses were found in the first diagonal vessel and left circumflex. The right coronary had a total proximal lesion. However, the second portion of the saphenous Y graft to the distal right coronary artery was patent, as was the first portion of the saphenous Y graft, which went to the first LAD diagonal vessel. The patient underwent successful percutaneous intervention of the first diagonal and circumflex lesions.

The patient’s subsequent hospital course was unremarkable. He ambulated without difficulty. The use of a statin was rediscussed with him. In retrospect, he thought that the myalgias he had experienced might have been related to claudication, and he agreed to try a low-dose statin. When last seen in follow-up, the patient was doing well without recurrent ischemic symptoms and was tolerating the medical regimen without difficulty.