

Angina pectoris, dyspnea, fatigue, and edema after a non-ST-segment–elevation myocardial infarct

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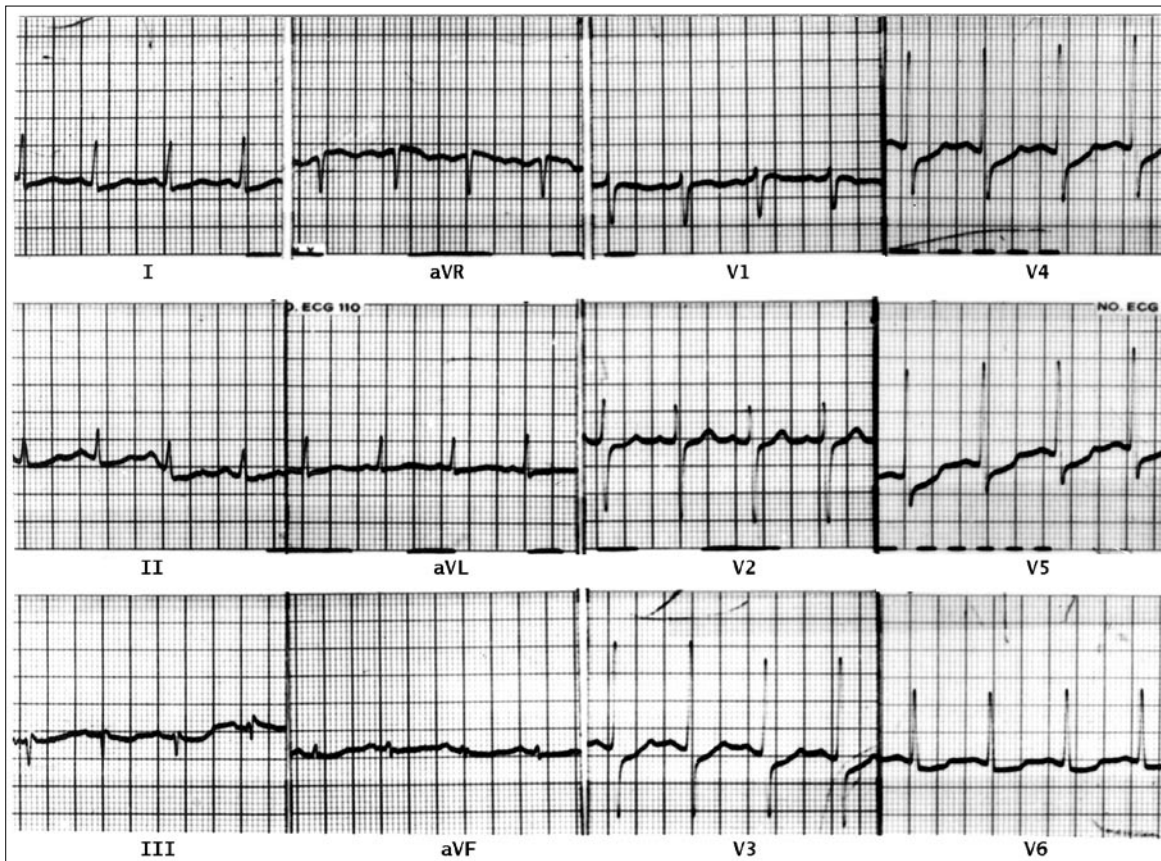


Figure 1. Electrocardiogram recorded on the patient's first admission. See text for explication.

A 65-year-old man came to the hospital because of retrosternal chest pain, and an electrocardiogram was recorded (Figure 1). It showed sinus tachycardia and ST-segment depression in 8 leads (I, II, aVL, V₂–V₆) with slight reciprocal ST-segment elevation in lead aVR, findings of severe subendocardial ischemia and/or injury (1, 2). Serum markers confirmed non-ST-segment–elevation myocardial infarction. Despite the development of a systolic cardiac murmur, the patient had an uneventful recovery.

Over the ensuing 8 months, the patient had angina pectoris for the first time and gradually developed exertional dyspnea, fatigue, orthopnea, and marked peripheral edema. He returned to the hospital, and another electrocardiogram was recorded (Figure 2). This one was quite different from the first electro-

cardiogram. Widespread ST-segment depression was no longer seen. The QRS axis in the frontal plane had shifted from approximately +10 degrees to about +75 degrees. The S wave in lead V₁ had shrunk, while the S waves in leads I, aVL, V₅, and

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The patient described in this report is case 8 in the study cited in reference 3. The electrocardiograms and chest radiographs have not been published before.

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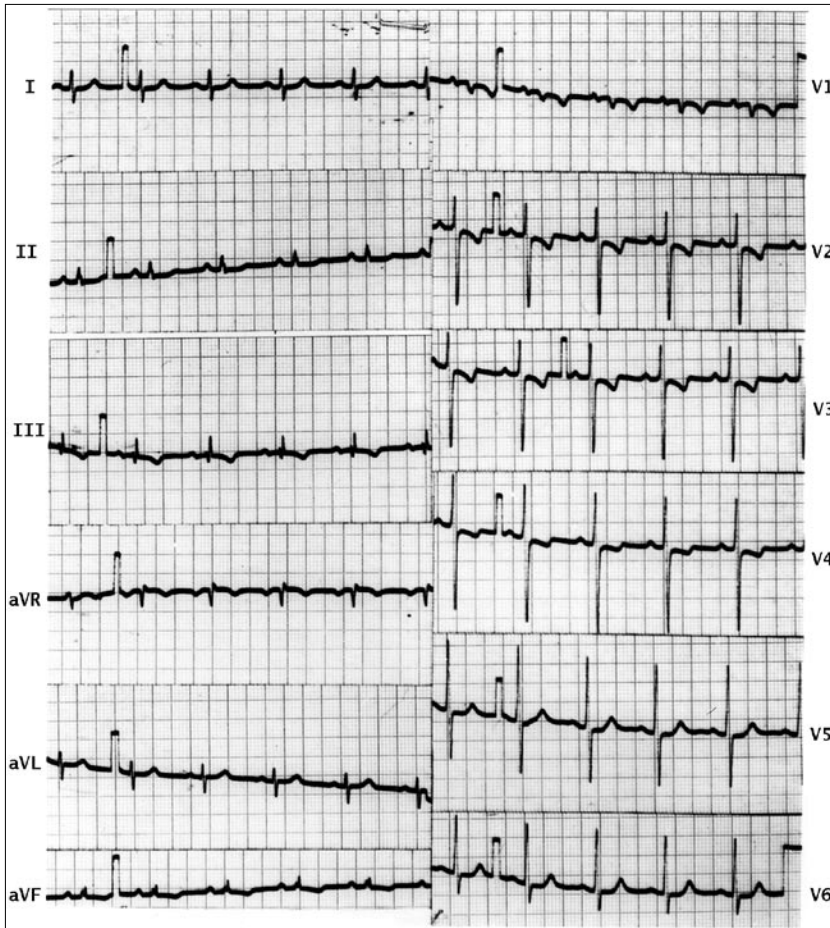


Figure 2. Electrocardiogram recorded on the patient's second admission. See text for explanation.

V_6 had increased. T waves were now inverted in leads V_1 to V_4 . These are signs of right ventricular enlargement.

What is the cause of the right ventricular enlargement? The most likely pulmonary cause of such a change in 8 months would be pulmonary embolic disease. The chest radiograph, however, showed pulmonary plethora rather than oligemia, and all of the cardiac chambers were large, including the left atrium (Figure 3). Furthermore, a loud apical systolic murmur of mitral regurgitation had a definitely decrescendo quality, suggesting a large left atrial v wave with significant left atrial and, consequently, pulmonary arterial hypertension. This supposition was confirmed by pressures (in mm Hg) measured at cardiac catheterization: pulmonary arterial wedge mean of 35 with v waves of 60 and pulmonary arterial pressure of 95/40 with a mean of 60. A left ventriculogram showed severe mitral regurgitation and normal left ventricular systolic function (3).

At operation, a fibrotic posteromedial papillary muscle was found to be the cause of the severe mitral regurgitation. The valve was replaced, and the patient improved symptomatically

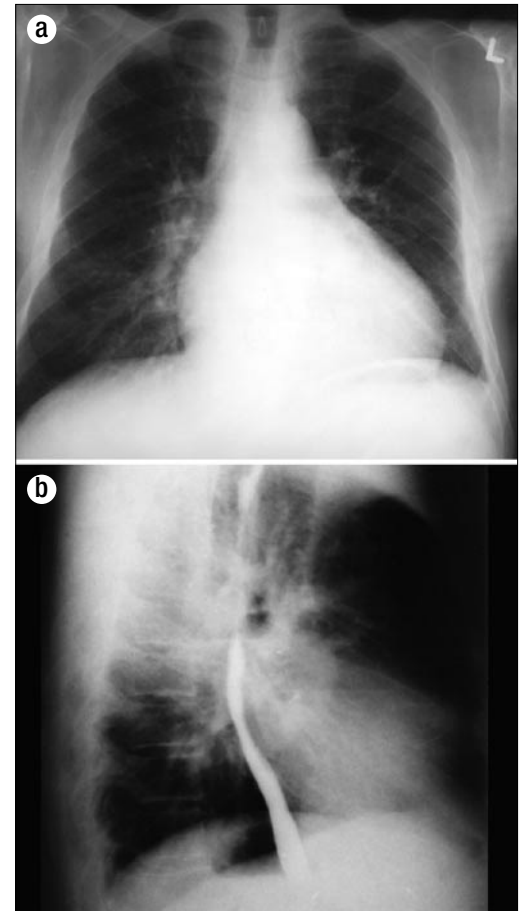


Figure 3. Chest radiographs taken during the patient's second admission. (a) The posteroanterior view shows plethoric lungs, prominent pulmonary arteries, and generalized cardiomegaly. (b) The lateral view shows indentation of the barium-filled esophagus by the enlarged left atrium.

and hemodynamically (3). Thus, although the patient underwent operation 35 years ago without the benefit of myocardial revascularization, he was part of a study demonstrating that left ventricular function is the most important determinant of outcome in patients with ischemic mitral regurgitation.

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2. Barrabes JA, Figueras J, Moure C, Cortedellas J, Soler-Soler J. Prognostic value of lead aVR in patients with a first non-ST-segment elevation acute myocardial infarction. *Circulation* 2003;108:814-819.
3. Glancy DL, Stinson EB, Shepherd RL, Itscoitz SB, Roberts WC, Epstein SE, Morrow AG. Results of valve replacement for severe mitral regurgitation due to papillary muscle rupture or fibrosis. *Am J Cardiol* 1973;32:313-321.