MARC WILLIAMS, MD, EDITOR

CURTIS M. RIMMERMAN, MD
Head, Section of Clinical Cardiology.
Department of Cardiology, Cleveland Clinic

The Clinical Picture

A 46-year-old man with dyspnea

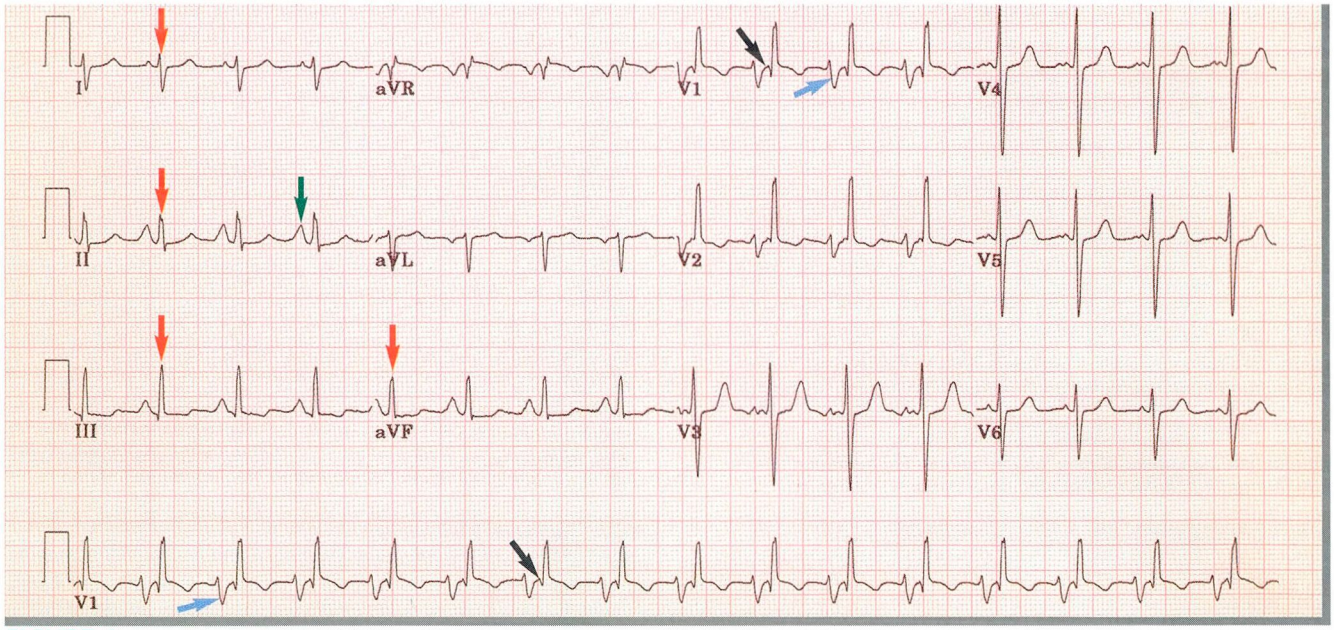

FIGURE 1

Q: A 46-YEAR-OLD MAN came to the emergency department because of progressive recumbent and exertional dyspnea, which began 1 month previously. During this time he had gained approximately 25 pounds, with lower-extremity swelling that impaired his ability to tie his shoes. Before this, he had been very active without limitations.

As a child he had contracted a poorly characterized febrile illness that required bed rest and home confinement for approximately 1 month. Otherwise, there was nothing of note in his history. He was taking no medications and had not seen a physician for a physical examination in many years.

Physical examination. The patient was afebrile, pulse 90, respirations 28 (using accessory muscles to breathe), blood pressure 116/78 mm Hg. His jugular venous pressure was elevated at least to the angle of his jaw at 45 degrees, with prominent V waves easily seen. Wet rales were heard in the lower half of each lung field. Cardiac examination disclosed a readily palpable parasternal right ventricular heave, a loud opening snap, and a diastolic rumble heard best in the fifth intercostal space at the midclavicular line. The lower extremities demonstrated 3+ pitting edema to the level of the knees.

Chest radiography showed bilateral moderate-sized pleural effusions, enlarged main pulmonary arteries, an enlarged cardiac silhouette suggesting right ventricular and left atrial prominence, and pulmonary edema.

The electrocardiogram (FIGURE 1) shows all of the following except which one?

- ❑ Right-axis QRS complex deviation
- ❑ Left atrial abnormality
- ❑ Right atrial abnormality
- ❑ Left ventricular hypertrophy with secondary ST-T changes
- ❑ Right ventricular hypertrophy with secondary ST-T changes

A: This electrocardiogram does not indicate left ventricular hypertrophy with secondary ST-T changes, as the QRS complex voltage in leads V_5 and V_6 is not elevated. However, it does demonstrate:

- Normal sinus rhythm (the P wave axis is normal and the atrial rate is slightly less than 100 per minute)
- Right axis deviation (the QRS complex vector is negative in lead I and positive in leads II, III, and aVF—red arrows)
- Left atrial abnormality (a deep, terminally negative P wave is present in lead V_1—blue arrow)
- Right atrial abnormality (the P wave in lead II is 120 ms in duration and 3 mm in amplitude—green arrow)
- Right ventricular hypertrophy with secondary ST-T changes and severe pulmonary hypertension (a minuscule R wave is seen in lead V_1 followed by a small Q wave and large R′ deflection—black arrow—coupled with the right atrial abnormality, QRS complex right-axis deviation, and a normal QRS complex duration).

The clinical history, physical examination, and electrocardiogram are most consistent with advanced rheumatic mitral stenosis. In retrospect, his childhood febrile illness most likely was acute rheumatic fever. The valvular sequelae that variably follow acute rheumatic fever most often become clinically manifest decades later. Pathologically, rheumatic valvular heart disease is felt to represent the end result of an autoimmune response.

Rheumatic mitral stenosis is suspected on the basis of the clinical history and physical examination. Exertional dyspnea, the most common presenting symptom, reflects an increase in pulmonary venous pressure due to the exertional increase in the mitral transvalvular gradient. Typical physical findings may include an elevated jugular venous pressure, a parasternal right ventricular heave, a palpable S_1 and P_2, increased intensity of S_1, an opening snap, and a low-pitched diastolic rumble best heard between the left lower sternal border and left anterior axillary line. Atrial fibrillation may be present.

Less likely diagnostic considerations include chronic recurrent pulmonary emboli and cor pulmonale, primary pulmonary hypertension, congenital heart diseases such as an unrepaired atrial septal defect and Eisenmenger syndrome, advanced chronic obstructive pulmonary disease, and pulmonic stenosis.

The electrocardiographic and physical signs—prominent bi-atrial abnormality and right ventricular hypertrophy with secondary ST-T changes, coupled with elevated jugular venous pressure, a right ventricular heave, an opening snap, and a diastolic rumble—support the diagnosis of long-standing severe mitral stenosis and severe pulmonary hypertension. The diagnosis was confirmed by echocardiography and by direct examination of the valve at the time of mitral valve replacement. A preoperative right heart catheterization documented severe pulmonary hypertension, with a pulmonary artery systolic pressure of 110 mm Hg.

This electrocardiogram is consistent with long-standing severe mitral stenosis and reflects this patient's advanced clinical presentation. The markedly elevated R′ in lead V_1 reflects severe pulmonary hypertension. Commonly, patients with mitral stenosis present with symptoms earlier in the disease course with a lesser degree of pulmonary hypertension.

SUGGESTED READING

Abrams J. Mitral stenosis. In: Essentials of Cardiac Physical Diagnosis. Philadelphia: Lea and Febiger; 1987:275–306.

Chou T. Electrocardiography in Clinical Practice. Philadelphia: W.B. Saunders Company; 1991:55–63.

Rimmerman C, Jain A. Interactive Electrocardiography CD-ROM with Workbook. Lippincott Williams and Wilkins, 2001.

Waller BF, Howard J, Fass S. Pathology of mitral stenosis and pure mitral regurgitation. Part I. Clin Cardiol 1994; 17:330–336.

ADDRESS: Curtis M. Rimmerman, MD, Department of Cardiology, F15, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, OH 44195; e-mail rimmerc@ccf.org.

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