Electrocardiogram in a woman with cor pulmonale

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Figure. Electrocardiogram recorded on hospital admission. See text for explication.

A 53-year-old woman with a 3-year history of exertional dyspnea, a diagnosis of interstitial pulmonary fibrosis, no history of smoking, and home use of oxygen by nasal cannula was admitted to our hospital for the first time because of worsening shortness of breath over the prior 2 months. Physical examination showed jugular venous pulsations to the level of her ear lobes when she was upright and edema of her legs despite 40 mg of furosemide per day. Crackles were heard at the base of her lungs. The right ventricle was prominent to palpation, and auscultation revealed a systolic murmur and a loud pulmonic closure sound.

A chest radiograph showed diffuse interstitial pulmonary disease, which subsequently was confirmed by a computed tomogram. Arterial blood gases performed with a fraction of inspired oxygen of 28% revealed hypoxia and hypercarbia with a pH of 7.46, a partial pressure of carbon dioxide of 52 mm Hg, a partial pressure of oxygen of 78 mm Hg, a bicarbonate of 37 mEq/L, and an oxygen saturation of 97.3%. Ventilatory

function testing indicated severe restrictive lung disease with a vital capacity of 0.67 L.

A two-dimensional transthoracic echocardiographic and Doppler study demonstrated a cavernous right atrium, dilatation of the right ventricle with impaired systolic function, flattening of the ventricular septum in systole and diastole suggesting right ventricular pressure and volume overload, severe tricuspid regurgitation, mild to moderate pulmonic regurgitation, a dilated inferior vena cava without inspiratory collapse indicating severe right atrial hypertension, a pulmonary arterial pressure judged by Doppler to be >75/30 mm Hg, Doppler evidence of an elevated right ventricular end-diastolic pressure,

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and normal left-sided valves and chambers with a left-ventricular ejection fraction of >55%. Thus, the diagnosis of severe cor pulmonale was confirmed.

The patient's admission electrocardiogram (*Figure*) was abnormal but not nearly as abnormal as the echocardiogram. The sinus rhythm was normal, with little to suggest right atrial enlargement; the P wave axis was normal (+35°), not to the right of +75°, and the height of the P waves was at or below the upper limit of normal in leads II, III, and aVF (2.5 mm) and in V₁ and V₂ (1.5 mm) (1). Ikeda et al have pointed out that, in contrast to patients with severe obstructive lung disease, in those with severe interstitial fibrosis, such as our patient, the P waves are usually normal with a normal axis (2).

The QRS axis was vertical (+91°), which is unusual for a person this age, but not definitely abnormal on its own. However, in conjunction with R/S >1 in lead V₁, SV₁ ≤ 2 mm, onset of the intrinsicoid deflection in V₁ of 0.035 to 0.055 seconds, RV₅ or RV₆ <5 mm, and the presence of S waves in V₅ and V₆, the vertical axis signifies right ventricular hypertrophy (3–6).

As is the case with electrocardiographic criteria for left ventricular hypertrophy, virtually no electrocardiogram meets all of the myriad criteria for right ventricular hypertrophy (3–7), and in patients with lung disease and anatomically documented right ventricular hypertrophy, any single electrocardiographic criterion for right ventricular hypertrophy is more specific than sensitive (3–7). Milliken et al estimated that individual criteria have a sensitivity for diagnosing right ventricular hypertrophy >90% in patients with congenital heart disease, ~65% in those

with mitral stenosis, and ~30% in patients with chronic cor pulmonale (5). Others agree that the sensitivity of electrocardiographic criteria for diagnosing isolated right ventricular hypertrophy is highest in congenital heart disease (7) and lowest in cor pulmonale (7–9).

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