Type I second-degree atrioventricular block and intermittent left posterior fascicular block in a 26-year-old woman with an inferoposterior acute myocardial infarct

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Figure 1. Electrocardiograms recorded the night of admission. See text for explication.

A 26-year-old woman had been dyspneic for a week and intermittently dizzy for several days before presenting to the emergency department where an electrocardiogram showed sinus tachycardia; type I second-degree atrioventricular block with 6:5 Wenckebach periods; right axis deviation; and ST-segment elevation in leads II, III, aVF, and V₆ with ST-segment depression in leads I, aVL, and V₁–V₄ (Figure 1). The initial troponin I level was 7.27 ng/mL (reference, <0.05), and subsequent values were progressively lower. Thus, the patient had an inferoposterior acute myocardial infarct with atrioventricular nodal ischemia.

Subsequent electrocardiograms continued to show subtle, but definite, ST-segment changes of acute inferoposterior injury and second-degree atrioventricular block with conduction ratios of 7:6, 6:5, 3:2, and 2:1 (Figure 2). The right axis deviation of the QRS complex, however, came and went and when present had a pattern of left posterior fascicular block, i.e., rS in leads I and aVL with qR in leads III and aVF. Limb-lead misplacement is a common cause of a marked change in the QRS axis in the frontal plane. Two things are against that in this patient. First, the leads would have to have been alternately misplaced and then properly placed not once, but twice. Second, no matter what the frontal-plane axis, leads II, III, and aVF showed ST-segment elevation, and leads I and aVL showed ST-segment depression. Therefore, although left posterior

fascicular block is the least common conduction defect seen with acute myocardial infarction (1–3), it has been described in the condition, as well as transiently with myocardial ischemia (4), and appears to have been intermittent in our patient.

Acute ST-segment-elevation myocardial infarction is distinctly uncommon in persons this young, and in our experience two groups comprise the majority of such patients. In one there are relatively few risk factors, and coronary arteriography reveals no abnormality, suggesting that coronary arterial spasm, or arterial occlusion by a clot that forms on a nonocclusive plaque and subsequently lyses, may play a major role (5). In the second group there are multiple risk factors including diabetes mellitus and smoking (6). Our patient was in the latter category with obesity (body mass index 31.2, reference 20.0–24.9), diabetes mellitus (hemoglobin A_{1c} 14.0%, reference 4.7–6.4), cigarette smoking, hyperlipidemia (total serum cholesterol 261 mg/dL, reference <200; triglycerides 291 mg/dL, reference <150; low-density lipoprotein cholesterol 166 mg/dL, reference <130; high-density lipoprotein cholesterol 37 mg/dL, reference

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Figure 2. Electrocardiograms recorded (a) 10 minutes later, and (b) the following morning and (c) afternoon. All show sinus tachycardia; type I second-degree atrioventricular block with conduction ratios of 6:5 and 7:6, 3:2, and 2:1, respectively; and ST-segment changes of acute inferoposterior injury. Figure 1 and Figure 2b show left posterior fascicular block; Figures 2a and 2c have normal QRS axes.

40–59), systemic arterial hypertension, and a mother who died in her 40s of an acute myocardial infarct. Our patient's urine toxicology screen was negative for cocaine. Arteriography revealed a 60% ostial narrowing of the left anterior descending coronary artery and an 80% narrowing of the midportion of the right coronary artery, and the latter, the culprit lesion, was successfully stented (*Figure 3*).

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Figure 3. (a) Right coronary arteriogram in the left anterior oblique projection after administration of sublingual and intracoronary nitroglycerin shows severe narrowing proximally and lesser narrowings distally. (b) After stenting with a 2.5 × 15-mm bare metal stent, the proximal artery is widely patent.