A 70-year-old man came to the Access to Primary Care Clinic at the Interim LSU Public Hospital because he had been told at another hospital that he needed a kidney doctor. The patient had a history of high blood pressure, type 2 diabetes mellitus, long-standing kidney disease, an above the knee amputation on the left, gout, a possible coronary stent procedure five years ago, and recently poor appetite and inability to care for himself. He had a long history of medical noncompliance and was taking no medications when he came to the hospital. He denied all cardiac symptoms, including chest discomfort. He was admitted to hospital because of a blood pressure of 240/110 mmHg, a serum creatinine of 6.0 mg/dL, and an estimated glomerular filtration rate of 11 mL/min – i.e., chronic kidney disease, stage V. His electrocardiogram was read by the computer as normal (Figure 1).

Figure 1: Electrocardiogram recorded the day of admission. See text for explication.

What is your diagnosis?

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DIAGNOSIS: Sinus rhythm; long PR interval (0.22s); broad P waves (0.12s) in leads II, III, and aVF suggesting left atrial enlargement; in leads I, V₃–V₆ U waves are inverted – consequently, the ECG is abnormal and indicative of heart disease.

U waves are generally small (≤ 0.2 mV) waves occurring after the T wave with a vector similar to it. The normal U wave occasionally may be negative in leads III and aVF; is usually isoelectric in leads I, aVR, and aVL; and should be positive in all other leads. The U wave typically begins at the time of the second heart sound when the ventricles are beginning to relax, and this timing suggests a mechanoelectrical origin of the U wave.

A negative U wave in leads I, II, aVL, or any of the standard precordial leads, is always abnormal and is associated with heart disease in 98% of patients and with other ECG abnormalities in 95%. Negative U waves are most helpful clinically when, as in this patient’s admission ECG, no other significant abnormality is present. Unfortunately, negative U waves are rarely recognized. The single most common cause of negative U waves is systemic hypertension, but negative Us may occur with left ventricular hypertrophy of any cause or with myocardial ischemia. With right ventricular hypertrophy, negative U waves may occur in leads facing the enlarged right ventricle (V₄R, V₅–V₆).

As is the case with left ventricular hypertrophy, negative U waves associated with clinical ischemia are usually found in leads I, aVL and/or V₄ – V₆ and have > 95% specificity for significant coronary arterial narrowing, most often of the proximal left anterior descending or the left main coronary artery, whether the ischemia occurs spontaneously or is induced by exercise. In patients with posterior ischemia due to obstruction of blood flow in the right or left circumflex coronary artery, upright U waves may become more prominent in the precordial leads, presumably the reciprocal of inverted U waves posteriorly.

Transient U wave inversion may occur with ischemia, with a temporary rise in blood pressure in hypertensive patients during Prinzmetal’s variant angina and preceding or during the early stages of myocardial infarction. When the infarct is anterior, the inverted U waves are seen in the anterior precordial leads, and when the infarct is inferior, the inverted U waves are in leads III and aVF.

Our hypertensive patient came with a significant spike in his blood pressure that could account for the U wave inversion in the lateral leads, and his minor serum troponin elevations (0.12, 0.14, and 0.14 ng/ml on samples drawn at approximately eight-hour intervals; reference < 0.04) with normal creatine kinase and creatine kinase MB levels could have been ascribed to the pressure rise and his kidney failure. On the other hand, he was 70 years old and had hypertension, diabetes mellitus, kidney failure, peripheral arterial disease, and dyslipidemia (serum total cholesterol level, 184; LDL cholesterol, 122; HDL cholesterol, 35; and triglyceride, 135 mg/dL) with a high total serum cholesterol/HDL cholesterol level of 5.3 (reference, < 5.0). Thus, significant coronary arterial disease was likely. A

Figure 2: Electrocardiogram recorded the day after admission. See text for explication.
repeat electrocardiogram the morning after admission showed that the inverted U waves had virtually disappeared but were replaced by striking terminal T-wave inversions in the anterolateral leads (Figure 2). Thus, as suspected, coronary ischemia, although painless, was a major cause of the initial U wave inversions, and the proximal left anterior descending coronary artery was almost certainly severely narrowed. By the fourth hospital day, the T-wave inversions had largely disappeared.

The patient’s blood pressure has been controlled; he is now on hemodialysis; and coronary arteriography is being considered.

REFERENCES


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