A 73-year-old man with mild coronary artery disease and a dilated cardiomyopathy presented to the emergency room with a hemodynamically stable wide-QRS tachycardia. His 12-lead ECG revealed episodes of ventriculoatrial block, and a diagnosis of ventricular tachycardia (VT) was made (Figure 1). Intravenous procainamide restored sinus rhythm. Tachycardia recurred, and a second bolus of intravenous procainamide again restored sinus rhythm. The patient was started on concomitant amiodarone 800 mg/d.

The next day, the patient had significant prolongation of the QT interval with prominent U waves (Figure 2). He continued to have slower episodes of monomorphic VT on combination therapy. After 5 days of intravenous procainamide and oral amiodarone, he developed sustained polymorphic VT (Figure 3), suffered a cardiac arrest, and required defibrillation to restore sinus rhythm. His procainamide and N-acetylprocainamide levels were 5.6 μg/mL and 9.4 mg/mL near the time of the arrest. Electrolytes, magnesium, BUN, and creatinine were all within normal limits.

Procainamide was discontinued. After arrest, the patient continued to have short runs of polymorphic VT (compatible with torsade de pointes, Figure 4) that resulted in no hemodynamic compromise. These episodes gradually diminished. Despite this apparent stability, a routine ECG 2 days after arrest revealed profound QT prolongation and dramatic T-wave alternans (Figure 5). These changes gradually resolved with reduction of his amiodarone dose.

An implantable cardioverter-defibrillator (ICD) was placed before hospital discharge. After 10 months of follow-up, he has been clinically stable (requiring no ICD therapies). His QT interval (and QTc) was 460 ms.

Figure 1. During presenting wide-QRS tachycardia, there is intermittent loss of ventriculoatrial conduction (arrows). V1, II, V5 surface ECG leads.
Figure 2. A 12-lead ECG after intravenous procainamide demonstrates sinus rhythm with QT prolongation and prominent U waves. I, II, III, aVR, aVL, aVF, V_1 through V_6 surface ECG leads.

Figure 3. On intravenous procainamide and oral amiodarone, increasing ventricular ectopy was followed by ventricular fibrillation.

Figure 4. Short runs of torsade de pointes gradually ceased after procainamide was stopped.
Figure 5. Dramatic QT prolongation and T-wave alternans. These changes occurred "paradoxically" after tachyarrhythmias stabilized and most likely reflect complex (multitarget) electrophysiologic and antiarrhythmic effects of amiodarone. I, II, III, aVR, aVL, aVF, V₁ through V₆ surface ECG leads.