Atrial Repolarization Causing Pseudoprolongation of the QT Interval

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electrocardiogram; atrial arrhythmia; repolarization

A 73-year-old African-American male with a history of hypertension, diabetes, obstructive sleep apnea, and end-stage renal disease presented for evaluation of worsening dyspnea on exertion and fatigue. His medications included aspirin, atorvastatin, amlodapine, sodium bicarbonate, and neurontin. One month prior, his beta-blocker medication had been discontinued after a low heart rate was noted during dialysis.

On physical examination, the patient was a well appearing elderly male in no acute distress. A 12-lead electrocardiogram revealed sinus rhythm with blocked atrial bigeminy and a regular ventricular rate of 45 bpm. In the initial ECG (Fig. 1), the diagnostic statement of the Marquette 12 SL computer program was "Long QT." The QT was measured as 590 ms (QTc = 506 ms). The patient's ECG from 3 weeks earlier (Fig. 2) showed a normal sinus rhythm with a ventricular rate of 50 and a QT interval of 454 ms (QTc = 413 ms). Serum electrolyte levels around the time of both EKG's were unremarkable. (Sodium 140, potassium 5–5.2, calcium 8.8, and magnesium 2.0).

The premature atrial complexes in the inferior leads (arrow in Fig. 1) have an axis of -90° [low atrial]. The impact of the positive repolarization wave of these ectopic beats augments, by superpositioning, both the amplitude and duration of the T wave in these leads.

In most instances, atrial repolarization has a very minimal if any impact on the surface EKG. In normal sinus rhythm with a very long P-R interval, or complete AV block, atrial repolarization can be observed as a negative T_a wave that is shallow and often invisible.¹ It can, however, impact on inferior leads in exercise tests.² In contrast, the T_a wave of low atrial ectopic rhythm or retrograde P waves is positive and has a significantly greater voltage.³ It may cause ST elevation that can mimic an early acute inferior myocardial infarction. In our case, we speculate that the position of the T_a wave seemingly prolongs ventricular repolarization. This "pseudoprolongation" of the QT interval has not been previously reported in the literature.

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Figure 1. Twelve-lead ECG showing sinus rhythm with blocked atrial bigeminy (arrow). The QT interval appears prolonged and falsely measures 590 ms.

Figure 2. Twelve-lead ECG 3 weeks earlier shows normal sinus rhythm. The QT interval measures 454 ms.