Giant T-Wave Inversions and Extreme QT Prolongation
Marc A. Miller, MD; Sammy Elmariah, MD; Avi Fischer, MD

An 84-year-old woman presented with hypoglycemia-induced altered mental status. Presentation vital signs were notable for a cuff blood pressure of 150/102 mm Hg but otherwise were unremarkable. Her neurological examination after dextrose was unremarkable and a CT of the brain showed no evidence of infarction or hemorrhage. Her initial ECG (Figure A) demonstrated normal sinus rhythm (rate, 84 bpm), left ventricular hypertrophy, left anterior hemi-block, asymmetrically inverted T waves (leads I, AVL, V5, V6), and a mildly prolonged QT interval (QTc, 480 ms). Four hours after presentation, hypertensive urgency developed (224/117 mm Hg) accompanied by mild shortness of breath, but no chest pain or mental status changes were present. Troponin I levels drawn thereafter were consistent with myocardial infarction (peak troponin I, 4.5 ng/mL). Coronary angiography was notable for 80% occlusion of the left main coronary artery and 90% occlusion of the proximal left anterior descending artery. An ECG 24 hours after initial presentation (Figure B) revealed giant global T-wave inversions and extreme QT prolongation (QTc, 680 ms) in the absence of any neurological deficits or cardiovascular complaints. Potassium, magnesium, and calcium levels were all within normal limits. By hospital day 3, the ECG demonstrated “canyon T waves” (Figure C) with prolonged albeit improved QT intervals (QTc, 500 ms). The QT interval eventually returned to near baseline values by hospital day 4 (Figure D).

In the setting of an episode of hypertensive urgency, our patient had a non-ST-elevation myocardial infarction as a

Figure. Successive ECGs demonstrating the development and recovery of giant global T-wave inversions and extreme QT prolongation in the setting of a non-Q-wave myocardial infarction. A, normal sinus rhythm, left ventricular hypertrophy, asymmetrically inverted T waves (leads I, AVL, V5, V6) and a mildly prolonged QT interval (QTc = 480 msec). B, ECG 24 hours after initial presentation with giant global T-wave inversions and extreme QT prolongation (QTc = 680 msec). C, ECG on hospital day 3 demonstrating “canyon T waves.” D, Return of QT interval to near baseline value by hospital day 4.

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consequence of obstructive coronary artery disease. Prominent T-wave inversions and QT-segment prolongation are well recognized electrocardiographic signs that can occur in acute coronary syndrome; however, our case demonstrates an extreme example of this phenomenon. Although the exact mechanism responsible for infarct-related prolongation of the QT segment remains controversial, it may be related to the electric heterogeneity of the ventricular myocardium, which is composed of 3 distinct cell types with varying electrophysiological properties.\(^1\)\(^2\) The M cell, located within the midmyocardium, exhibits a significantly longer action potential duration than the epicardial and endocardial cell types and coincides with the end of the T wave.\(^2\) In the absence of injury, the electronic coupling of the M cell to the other cell layers minimizes these inherent differences in action potential duration.\(^3\) However, after an insult, such as a subendocardial infarction, uncoupling of the M cells from the adjacent cell layer removes these electrotonic influences and permits the M-cells to express their intrinsic properties, which is manifested on the surface ECG as QT prolongation.\(^4\)

Alternative etiologies for dramatic QT prolongation, such as myocardial ischemia as a direct consequence of percutaneous coronary intervention or due to the use of ionic contrast agents, is not applicable to our patient. The onset of giant T-wave inversions and extreme QT prolongation occurred before coronary angiography was performed; a percutaneous coronary intervention was not performed, and a nonionic contrast agent was used for the diagnostic angiography.

**Disclosures**

None.

**References**


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