Atypical Behavior of QTc and ST-T Intervals in a Patient With the Brugada Syndrome

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INTRODUCTION
Electrocardiographic abnormalities in patients with the Brugada Syndrome may be transient, and ajmaline or flecainide tests can uncover such abnormalities.\(^1\),\(^2\) These tests may, however, cause conduction disorders and arrhythmias.\(^3\),\(^4\)

We report electrocardiographic findings in a patient who presented variable QTc prolongation and T-wave alternans.

CASE REPORT
A 56-year-old man with no history of cardiovascular disease was admitted to the emergency service after receiving an electric shock. The electrocardiogram (ECG) showed a J point and ST segment elevation of up to 5 mm in leads V1 to V3, which normalized in 24 hours. The ajmaline test caused elevation of the J point and of the ST segment up to 12 mm in leads V1 to V3, QTc lengthening, and QTc and T wave alternans. These results denoted alterations in the duration of myocardial action potentials, a common finding in patients with Brugada syndrome and long QT syndrome.

Key words: Brugada syndrome. Long QT syndrome. Ajmaline test.

DISCUSSION
The Brugada syndrome can coexist with hereditary long QT syndrome (LQT3) and patients’ ECGs can show both afflictions.\(^5\) The Brugada syndrome appears
when the heart rate is faster and the QTc interval is smaller, whereas LQT3 presents at slower heart rates and a longer QTc interval.\(^7\)

Sodium channel blockers lead to a decrease in phase 0 and 1 amplitudes in the epicardium, with loss of the action potential dome and action potential shortening. This creates an electrical gradient between the epicardium and the endocardium, leading to a marked ST elevation in the ECG.\(^6\) However, when the effect wears off, variations in the duration and amplitude of the subepicardial action potentials appear. Moreover, these variations are independent of heart rate.

\[\text{Figure 1.} \quad \text{A: ECG at admission to hospital, showing “saddle shaped” ST and J-point elevation in leads V1 to V3. B: After 24 hours, a prominent J wave is observed only in leads V1 and V2.}\]

\[\text{Figure 2.} \quad \text{A: J wave in leads V1 and V2. B: ST elevation after ajmaline administration (greatest in lead V2 [C]).}\]

\[\text{Figure 3.} \quad \text{A: Prolongation of the QTc interval in lead V2 after 5 minutes (after the second beat) accompanied by T wave inversion. B: after 11 minutes, V1 and V2 show uneven QTc prolongation and T-wave alternans. C: after 12 minutes, QTc in lead V1 and V2 have a stable prolongation and the T waves are negative. D: all alterations disappear after intravenous administration of isoproterenol.}\]
rate. Thus, the QTc intervals in the ECG are unevenly prolonged and T-wave alternans appears.

Administration of isoproterenol shortens the duration of the subepicardial action potentials, improves intramyocardial conduction and normalizes the QTc interval.

REFERENCES


