**INTRODUCTION**

Electrocardiographic abnormalities in patients with the Brugada Syndrome may be transient, and ajmaline or flecainide tests can uncover such abnormalities. These tests may, however, cause conduction disorders and arrhythmias.

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 prominent J-wave 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. 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

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**Figure 1.** 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.

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

**Figure 3.** 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