BRIEF REPORT

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

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We present a 56-year-old man who was admitted to an emergency service after receiving an electric shock. The 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.*

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Comportamiento atípico del QTc y del ST-T en un caso con síndrome de Brugada

Un paciente de 56 años ingresó en el servicio de urgencias después de una descarga eléctrica. El electrocardiograma (ECG) mostró un supradesnivel de 5 mm del punto J y del segmento ST de V1 a V3, que desapareció a las 24 h. La prueba de ajmalina provocó un supradesnivel del punto J y del segmento ST de 12 mm de V1 a V3, prolongación del QTc, alternancia del QTc y de T. Estos cambios expresan variaciones en la duración de los potenciales de acción miocárdicos característicos de los síndromes de Brugada y del QT largo.

Palabras clave: *Síndrome de Brugada. Síndrome de QT largo. Prueba de ajmalina.*

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 prominent J-wave and ST-segment elevation of up to 5 mm in leads V1 to V3 (Figure 1A). Total creatine kinase (CK) increased but the echocardiogram was normal. After 24 hours, the prominent J wave was still observed unchanged in leads V1 and V2, but it had disappeared from lead V3 (Figure 1B and 2A).

The patient was suspected of having the Brugada syndrome,[5] so an intravenous ajmaline test was performed (50 mg). After 60 seconds, J-point and ST-segment elevation in leads V1 to V3 were seen (Figure 2B). The maximum elevation of 12 mm occurred in V2 (Figure 2C). After 5 minutes, the QTc intervals were prolonged from 372 to 539 ms and the T wave became negative (Figure 3A), followed by T-wave alternans (Figure 3B). Finally, the QTc interval stabilized at 539 ms (Figure 3C).

The ECG returned to normal after administration of isoproterenol (Figure 3D). During one year of follow-up, the QTc values were normal and the ECG traces were similar to that of Figure 2A.

DISCUSSION

The Brugada syndrome can coexist with hereditary long QT syndrome (LQT3) and patients' ECGs can show both afflictions.[6] The Brugada syndrome appears

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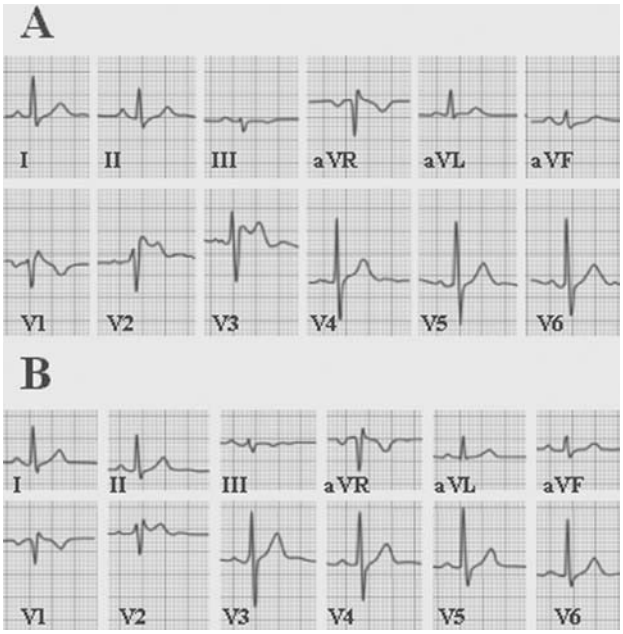

Figure 1. A: ECG at admission to hospital, showing "saddle shaped" ST and J-point elevation in leads V1 to V3. B: After 24 hours, a prominent J wave is observed only in leads V1 and V2.

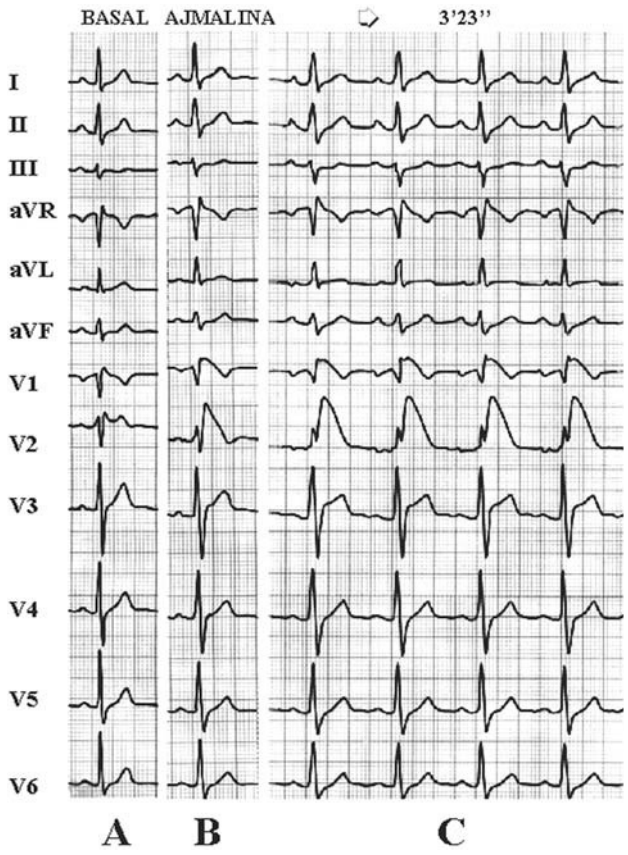

Figure 2. A: J wave in leads V1 and V2. B: ST elevation after ajmaline administration (greatest in lead V2 [C]).

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

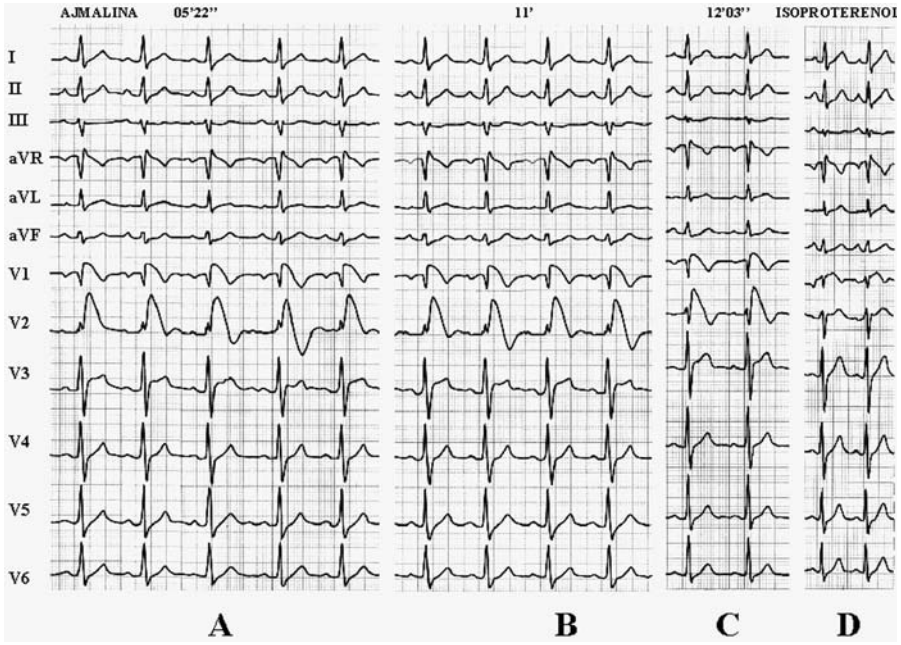

Figure 3. A: Prolongation of the QTc interval in lead V2 after 5 minutes (after the second beat) accompanied by T wave inversion. B: after 11 minutes, V1 and V2 show uneven QTc prolongation and T-wave alternans. C: after 12 minutes, QTc in lead V1 and V2 have a stable prolongation and the T waves are negative. D: all alterations disappear after intravenous administration of isoproterenol.

rate.[6] 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.

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