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CASE REPORT

A Case of Asymptomatic Brugada Electrocardiographic Pattern Incidentally Unmasked During the Recovery Phase of an Exercise Stress Test

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ABSTRACT

The interest about Brugada syndrome, an inherited channelopathy associated with sudden cardiac death in individuals without structural heart disease, is exponentially increasing lately. Similarly to chameleon, the electrocardiographic (ECG) signal of the disease fluctuates over time, is often concealed, and may be unmasked under certain conditions. Recently, emergence of the characteristic ST-segment elevation during exercise stress test (EST) has been reported, probably resulting from an alteration of the autonomic tone in the different stages of exercise. We present the case of a 43-year-old, otherwise healthy male with an asymptomatic Brugada ECG pattern incidentally unmasked during the recovery phase of an EST.

KEY WORDS: *Brugada syndrome; Brugada ECG pattern; exercise stress test; channelopathies; sudden cardiac death*

List of Abbreviations
ECG = electrocardiogram
EPS = electrophysiology study
EST = exercise stress test
SAECG = signal-averaged ECG
SCD = sudden cardiac death
VF = ventricular fibrillation
VT = ventricular tachycardia

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INTRODUCTION

The interest in Brugada syndrome, an inherited channelopathy with an abnormal electrocardiogram (ECG) associated with sudden cardiac death (SCD) in individuals without structural heart disease, is exponentially increasing lately. Three ECG patterns of ST elevation in leads $V_{1\text{-}3}$ have been described. Type 1 ECG pattern with pronounced (>2 mm/ 0.2 mV) elevation of the J point, a coved-type ST segment, and an inverted T wave in $V_{1\text{-}3}$. Type 2 pattern has a saddleback ST-segment elevated by >1 mm. Type 3 pattern (coved or saddleback) has the ST segment elevated < 1 mm. Type 1 ECG pattern is considered diagnostic of Brugada syndrome. However, similarly to a chameleon, the ECG signal of the disease may be dynamic, fluctuating over time, and often concealed. Administration of various pharmaceutical agents and other conditions, such as electrolyte abnormalities, febrile state, alcohol and cocaine intoxication, are able to unmask or accentuate the ST-segment elevation. In addition, the ECG may fluctuate over time depending on the autonomic balance.[1,2] We present the case of a 43-year-old male who developed typical type-1 Brugada-like ECG pattern during the recovery phase of an exercise stress test (EST).

Conflict of interest / financial disclosure: none declared

CASE REPORT

A 43-year-old gentleman presented in the outpatient clinic for a routine EST. The patient was totally asymptomatic and his past medical history was uneventful. He had a history of arterial hypertension treated with amlodipine, hydrochlorothiazide and ramipril, and a history of hypercholesterolaemia managed with dietary and lifestyle modification therapy. There was no history of smoking, alcohol or drug abuse. No family history of SCD or unexplained syncope was reported. The patient is a father of a daughter and a son who are both healthy with normal resting ECGs. General medical and cardiovascular physical examination was unremarkable.

The 12-lead ECG showed an incomplete right bundle branch block, and apart from a slight ST-segment elevation in lead V1 no other significant changes in the ST-T segment in leads V1-V3 were noted (Fig. 1a). The patient underwent an EST using the Bruce treadmill exercise protocol, during which he completed 11 minutes and 16 seconds. The test was interrupted because the target heart rate was achieved without any symptoms or ECG changes on exercise. On the sixth minute of the recovery phase however, a shift of the ECG to type 1 Brugada pattern was noted with accentuated J wave, pronounced coved-type ST-segment elevation followed by inverted T waves in the precordial leads V1 and V2 (Fig. 1b). The patient remained hemodynamically stable and asymptomatic and no ventricular extrasystolic activity was documented. In the next few minutes, the ECG normalized gradually without any therapeutic intervention.

The patient was referred to the electrophysiology service for further evaluation. Hematological and biochemical tests were normal. Echocardiography showed only mild left ventricular hypertrophy. Left and right ventricular functions were normal. A pharmacological challenge test was subsequently performed. Patient was challenged with procainamide in the recommended dose of 10 mg/kg over 10 min by slow intravenous infusion under continuous ECG monitoring.[1] A shift to the diagnostic type 1 Brugada ECG pattern with pronounced elevation of the J point, a coved-type ST segment and an inverted T wave in V1-V2 (Fig. 1c) were observed and the test was interrupted.[1,2] A signal-averaged ECG (SAECG) was performed, which did not detect late potentials. Subsequently, an electrophysiology study (EPS) was performed. Baseline conduction interval measurements were within normal limits. In addition, programmed ventricular stimulation, from the right ventricular apex and the right ventricular outflow tract with three premature extrastimuli, failed to induce ventricular tachycardia (VT) or ventricular fibrillation (VF). After obtaining informed consent from the patient, his daughter and his son, molecular genetic analysis was performed, showing no mutations in the SCN5A gene. SCN5A mutations are detected in only 20-30% of cases, however, negative results do not exclude Brugada syndrome.[1,3] Both patient's children had pharmacological challenge tests with procainamide, revealing a type 1 Brugada pattern in the son.

As the patient was asymptomatic, had no family history of SCD, and developed type 1 Brugada ECG only after pharmacological challenge and had no ventricular arrhythmias during EPS, he was classified as being at minimal or no risk of SCD.[1,2] Subsequently, no other intervention was decided apart from close follow-up in the outpatient clinic. Up to this date he remains well and asymptomatic without any changes in his health status, receiving only his antihypertensive medication. No ventricular arrhythmias have been recorded. Repeat ECGs over the 9-month follow-up period revealed no ST-T abnormalities.

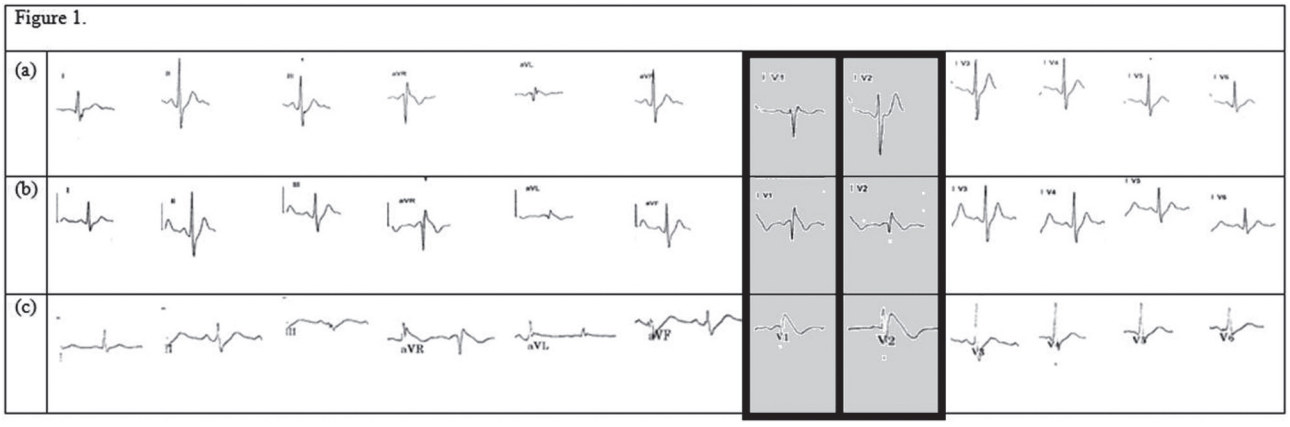

FIGURE 1. 12-lead electrocardiogram recorded during the resting phase (a), at the 6th minute of the recovery period of the exercise stress test (b), and during the pharmacologic challenge with intravenous procainamide (c).

DISCUSSION

Fluctuation of the ECG signal during exercise in the Brugada syndrome has been described, probably depending on the autonomic balance.[1,4] Adrenergic stimulation decreases ST-segment elevation while vagal stimulation worsens it. Additionally, ventricular tachyarrhythmias are commonly bradycardia-related and thus increased vagal activity may contribute to the higher incidence of SCD during sleep or rest.[1] On the other hand, enhancement of ST-elevation during physical activity is rare.[2] Despite the widespread use of EST in everyday practice, to the best of our knowledge, this is only the third case in the literature of a Brugada ECG pattern unmasked during the recovery phase of an EST.[5,6] Grimster et al reported recently the case of a 33-year-old symptomatic male with positive family history of SCD, who demonstrated an ECG shift from type 2 to type 1 pattern, during the recovery phase of an EST.[5] Ozeke et al reported a 59-year-old male who developed VT with a left bundle branch block during an EST at the recovery phase of which ECG changed from type 2 to typical type 1.[6]

Further investigation with EPS, pharmacological challenge, SAECG and genetic testing established the diagnosis and determined a low risk for SCD in our patient. The predictive value of the EPS as well as its role in the risk assessment for SCD is debated in Brugada syndrome.[1] Some authors suggested that VT/VF inducibility during programmed ventricular stimulation is a strong predictor for SCD in asymptomatic subjects,[7] whereas others reported a poor predictive value in both symptomatic and asymptomatic subjects.[2,8] Currently, the test is recommended in asymptomatic patients with type 1 Brugada ECG (spontaneously or after pharmacologic challenge) and positive family history of SCD.[1] It is also justified in cases of asymptomatic patients with negative family history of SCD when spontaneous type 1 Brugada ECG changes are manifested.[1]

Implantation of an automatic cardioverter defibrillator is considered as the only effective treatment for the disease and is reserved for symptomatic patients and asymptomatic patients with inducible VT/VF during EPS, who are at highest risk for SCD.[1] No such intervention was necessary in our case.

It can be speculated that although ECG changes and arrhythmias are suppressed during exercise, parasympathetic-vagal exacerbation during recovery has a dominant role. Physicians should be aware of this phenomenon, especially in symptomatic patients with syncope or aborted sudden death or those with a family history of SCD. Exercise testing is a simple, easily accessible, low cost test widely used in clinical practise, and has become a useful tool in the assessment of many arrhythmias lately. A review of the literature even revealed suggestions for possible diagnostic implications of EST in the investigation of patients with type 2 ECG pattern who are symptomatic or have a family history of SCD.[5] In any case, among other more common ECG changes that may potentially be observed during the test, the physician should be able to recognize emergence of a typical type 1 Brugada ECG in individuals with type 2 or type 3 pattern.

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