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Journal of Electrocardiology 45 (2012) 195 – 198

JOURNAL OF Electrocardiology

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Coexisting early repolarization pattern and Brugada syndrome: recognition of potentially overlapping entities ☆

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Received 8 September 2011

Abstract The Brugada type 1 electrocardiographic (ECG) pattern and the early repolarization pattern (ERP) are 2 ECG patterns characterized by the appearance of J waves. Although Brugada type 1 ECG pattern in the context of the Brugada syndrome (BrS) is well known for predisposing to life-threatening ventricular arrhythmias, it has only recently come to light that ERP, which was previously believed to be benign, may also be a marker for arrhythmogenic potential. ERP and BrS share many remarkable cellular, ionic, and ECG similarities and behave comparably in terms of their response to heart rate, pharmacologic agents, and neuromodulation. The extent to which ERP and BrS may overlap remains unclear.

Here, we present an illustrated case of a symptomatic patient whose ECG signature evolved spontaneously from ERP alone to ERP with a concomitant Brugada type 1 ECG pattern over a short number of days. This case lends further strength to the notion that these 2 ECG patterns may be more closely related than had been initially thought.

Keywords: Early repolarization pattern; Brugada syndrome; Ventricular repolarization; Sudden cardiac death

Introduction

The Brugada type 1 electrocardiographic (ECG) pattern and the early repolarization pattern (ERP) are 2 ECG patterns characterized by the appearance of J waves. Although Brugada type 1 ECG pattern in the context of the Brugada syndrome (BrS) is well known for predisposing to life-threatening ventricular arrhythmias, it has only recently come to light that ERP, which was previously believed to be benign, may also be a marker for arrhythmogenic potential.[1] Early repolarization pattern and BrS share many remarkable cellular, ionic, and ECG similarities and behave comparably in terms of their response to heart rate, pharmacologic agents, and neuromodulation.

Early repolarization pattern is a common ECG finding that affects 1% to 9% of the general population and is more common in African Americans and athletes. Although no consensus definition exists, it has previously been defined by elevation of the J point (QRS-ST junction) of at least 1 mm (0.1 mV) above baseline in at least 2 contiguous leads, in the inferior leads (II, III, and aVF), lateral leads (I, aVL, and V_4 to V_6), or both. It can manifest either as "slurring" of the QRS (a smooth transition from the QRS segment to the ST segment) or "notching"—a positive J deflection inscribed on the S wave. Early repolarization pattern was initially thought to be entirely benign, but recent evidence suggests otherwise.[1,2]

Brugada syndrome is a clinical ECG entity predisposing to ventricular arrhythmia and sudden cardiac death (SCD). It is defined electrocardiographically by a characteristic pattern including J point and ST-segment elevation 2 mm or greater, followed by a negative T wave in the right precordial leads. The ECG of patients with BrS can change over time between type 1 "coved" and types II and III "saddleback" patterns and even show transient normalization. The BrS pattern has been closely linked to *SCN5A* gene mutations affecting sodium channel function.[3]

The extent to which ERP and BrS may overlap remains unclear.[4] Both ERP and BrS have been described simultaneously in different members of the same family,[5] and some have even proposed classifying them as part of a spectrum of disease.[6] Here, we present what is, to the best of our

☆ Conflicts of interest and financial disclosures: none.

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0022-0736/$ – see front matter © 2012 Elsevier Inc. All rights reserved.
doi:10.1016/j.jelectrocard.2011.10.008

knowledge, the first illustrated case of a symptomatic patient whose ECG signature evolved spontaneously from ERP alone to ERP with a concomitant Brugada type 1 ECG pattern over a short number of days. This case lends further strength to the notion that these 2 ECG patterns may be more closely related than had been initially thought.

Clinical case

A 20-year-old Brazilian male professional soccer player presented for assessment, complaining of 2 presyncopal episodes and 1 syncopal episode in the preceding 30 days. The 2 presyncopal episodes occurred coinciding with defecation. Both episodes were associated with lightheadedness, diaphoresis, and nausea. The syncopal episode occurred at rest with no prodromal symptoms. His medical history was unremarkable; he had successfully passed 2 prior precompetitive clinical ECG screenings.

The patient's family history was significant for the sudden, unexplained death of a paternal uncle at the age of 35 years. The patient volunteered that his brother was being followed up by a cardiologist because of "cardiac arrhythmia" but was unable to provide further detail.

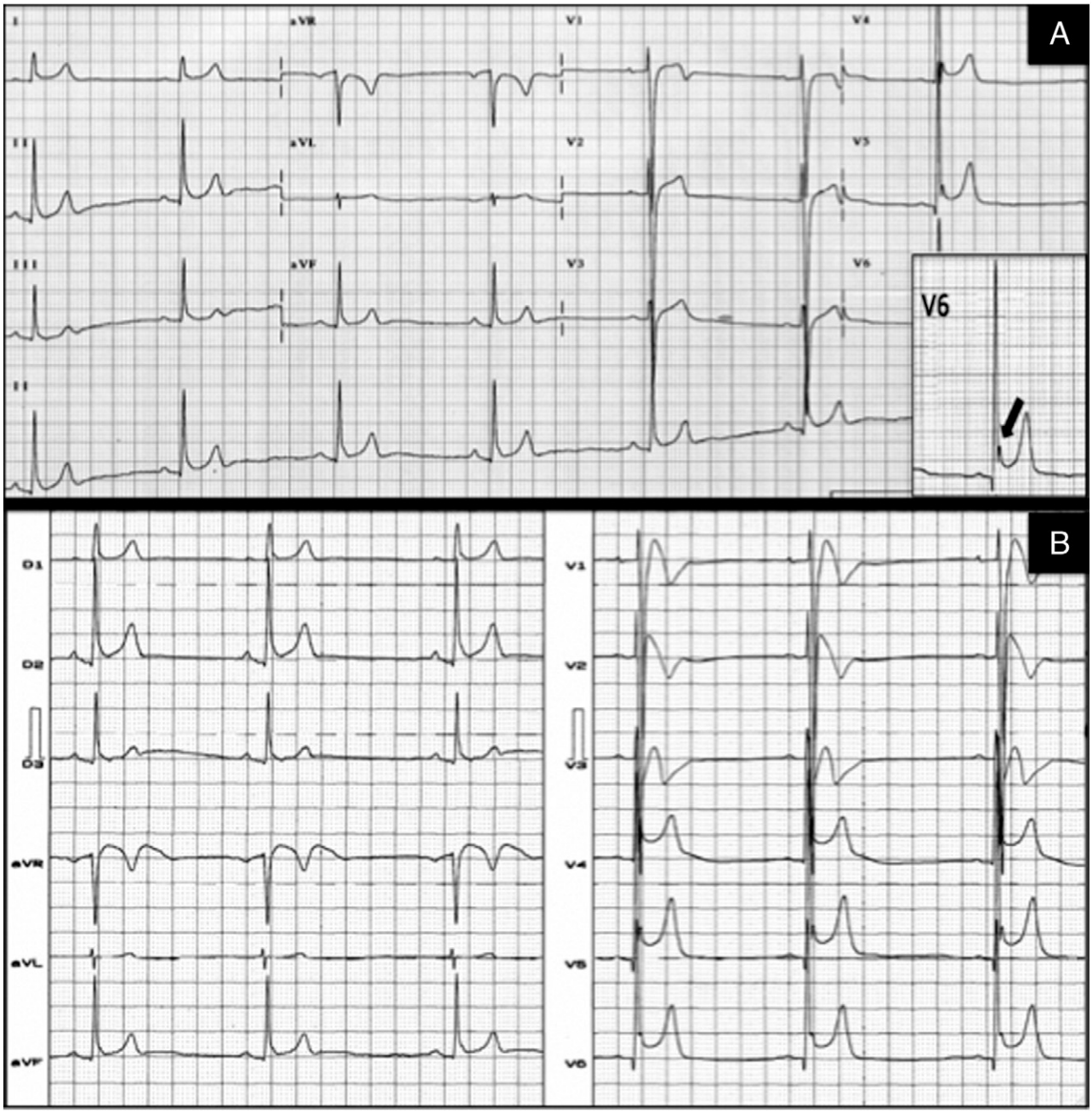

Fig. 1. Panel A, Twelve-lead ECG from a 20-year-old man, showing sinus bradycardia, J waves (arrow) with concave up ST-segment elevation in leads V_4 to V_6 and the inferior leads. These features are considered to be consistent with ERP. Panel B, Twelve-lead ECG from the same 20-year-old man, recorded 72 hours later. The ERP persists, and there is now sinus bradycardia with a Brugada type 1 ECG pattern (coved type) in leads V_1 to V_3. The ST-segment elevation seen in lead aVR has been identified as a potential high-risk marker for ventricular arrhythmia in patients with BrS.