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Review

Brugada ECG patterns in athletes

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Abstract Brugada syndrome is responsible for up to 4% of all sudden cardiac deaths worldwide and up to 20% of sudden cardiac deaths in patients with structurally normal hearts. Heterogeneity of repolarization and depolarization, particularly over the right ventricle and the outflow tract, is responsible for the arrhythmogenic substrate. The coved Type I ECG pattern is considered diagnostic of the syndrome but its prevalence is very low. Distinguishing between a saddle back Type 2 Brugada pattern and one of many "Brugada-like" patterns presents challenges especially in athletes. A number of criteria have been proposed to assess Brugada ECG patterns. Proper precordial ECG lead placement is paramount. This paper reviews Brugada syndrome, Brugada ECG patterns, and recently proposed criteria. Recommendations for evaluating a Brugada ECG pattern are provided.

Keywords: Brugada pattern; High lead ECG; Athletes

Introduction

Distinguishing a Brugada ECG pattern from a "Brugada like" pattern can be challenging. This paper aims to briefly review Brugada syndrome (BrS), Brugada ECG patterns (BrEP), and criteria for evaluating screening ECGs in athletes.

Background: Brugada syndrome

The ECG pattern comprised of a coved-type rSr' pattern, ST-segment elevation, and inversion of the terminal portion of the T waves in the right precordial leads, has been the hallmark of BrS since 1992 [1–3]. BrS is responsible for up to 4% of all sudden cardiac deaths (worldwide) and up to 20% of sudden cardiac deaths in patients with structurally normal hearts [4,5]. Manifestations include syncope, atrial arrhythmias, ventricular arrhythmias, and sudden cardiac death in the absence of myocardial ischemia or overt heart disease. The vulnerability to sudden cardiac death seems to occur mostly during sleep or when vagal tone is increased. Inheritance is variable but 15%–30% of cases are attributable to pathogenic variants in the sodium channel gene SCN5A [6–8]. It predominantly affects males and has a predilection for men under 40 in Southeast Asia [5,8,9]. Increased vagal tone, fever, electrolyte disturbances, and sodium channel blocking medications can accentuate the ECG pattern. Class I antiarrhythmic agents, such as ajmaline, flecainide, and procainamide, can "provoke" the BrEP and are used in evaluation of suspected BrS patients. Isoproterenol (isoprenaline) and quinidine can normalize the pattern [8]. The pathophysiology behind the BrEP is ascribed to two main hypotheses [10,11]. The first implicates abnormal repolarization: loss of Na current due to mutations in SCN5A results in "unopposed" outward K current by I_{to}, creating a voltage gradient (from dispersion of refractoriness) between the epicardium and endocardium in the right ventricle (RV) and RV outflow tract, where there are fewer M cells, causing the BrEP changes [12]. The second hypothesis, and not necessarily mutually exclusive of the first, implicates delayed depolarization and structural alterations in the RV and RV outflow tract [5,12,13]. A recent small but intriguing study utilizing electrocardiographic imaging (ECGI) showed that both repolarization and conduction (or depolarization) abnormalities are present in BrS patients [11]. The heterogeneity of repolarization and depolarization generates the arrhythmogenic substrate for polymorphic ventricular tachycardia or ventricular fibrillation.

Brugada ECG pattern

While diagnosing BrS should include both clinical history and ECG findings, the Type 1 BrEP alone is now considered diagnostic [12,14]. Classically, the Type 1 BrEP has a *coved*

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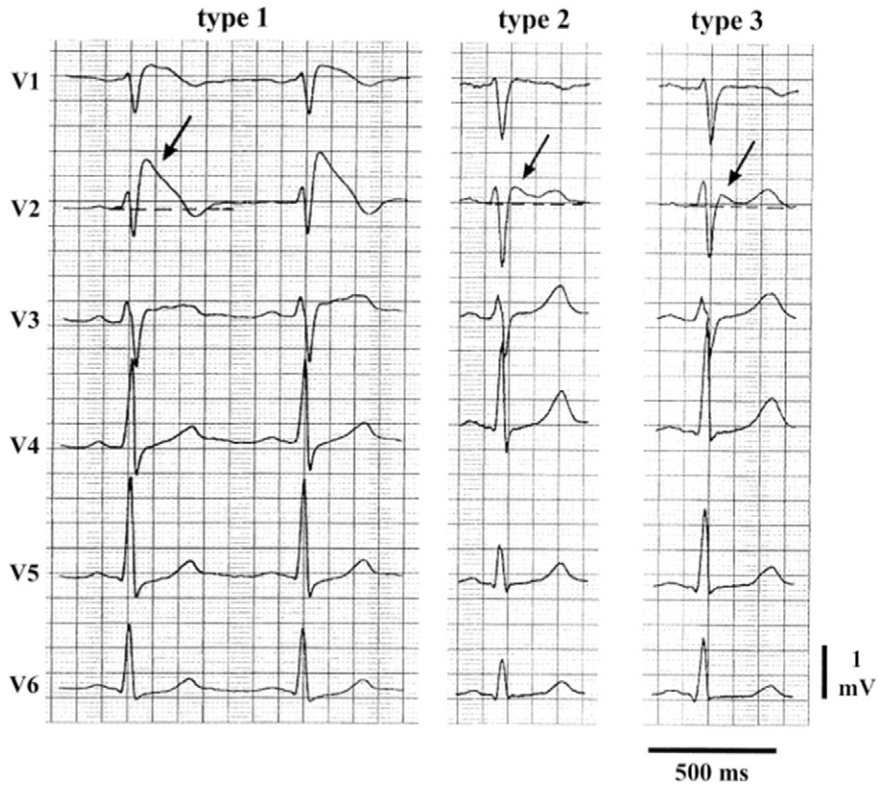

Fig. 1. Classic Brugada ECG patterns. Type 1 is considered diagnostic of Brugada syndrome. Types 2 and 3 were grouped together as Type 2 in a recent consensus report. From Ref. [7].

ST segment elevation ≥2 mm, negative T wave and no isoelectric separation of T wave (Fig. 1); Type 2 BrEP has a *saddleback* appearance with an ST segment elevation of ≥2 mm, trough that is still ≥1 mm ST elevation and a positive or biphasic T wave; Type 3 BrEP is also *saddleback* but has an ST segment elevation of <1 mm. A consensus report combined Type 2 and 3 as Type 2 [12]. Another recent expert consensus statement states: "[Brugada syndrome] is definitively diagnosed when a Type 1 ST-segment elevation is observed either spontaneously or after intravenous administration of a sodium channel blocking agent (e.g. ajmaline, flecainide, pilsicainide, or procainamide) in at least one right precordial lead (V_1 and V_2), which are placed in a standard or a superior position (up to the 2nd intercostal space)"[15]. The prevalence of the BrEP is very low: in a cohort of approximately 12,000 apparently healthy Europeans, only 23 (0.19%) had any BrEP; of these, only 2 had the Type 1 pattern [16].

Distinguishing and evaluating "Brugada-like" ECGs

Distinguishing between a BrEP and "Brugada-like" (also called rSr', non Type 1, Type 2 Brugada type) pattern can be challenging, especially in athletes. A recent term, "Brugada phenocopy", has been proposed to describe a BrEP, in a person without true BrS, provoked by agents or clinical situations not expected to unmask true BrS, such as

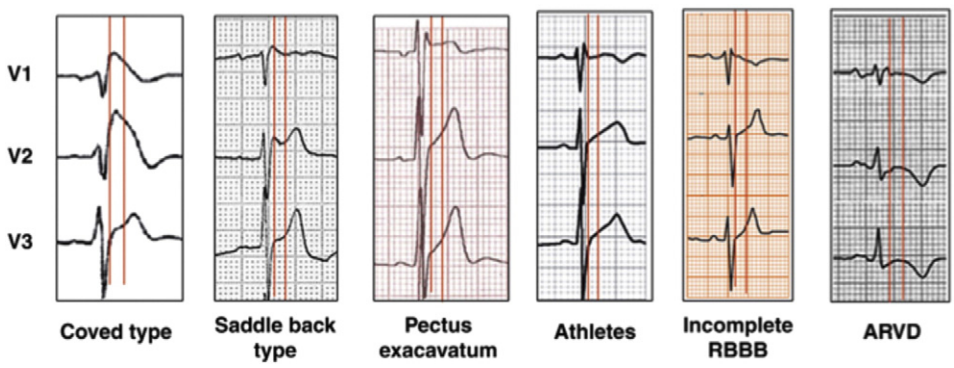

Fig. 2. Electrocardiographic differential diagnosis for Brugada-like ECG patterns. From Ref. [12].