IJC Heart & Vasculature 18 (2018) 58–64

Contents lists available at ScienceDirect

IJC Heart & Vasculature

journal homepage: http://www.journals.elsevier.com/ijc-heart-and-vasculature

High risk electrocardiographic markers in Brugada syndrome

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ARTICLE INFO

Article history:
Received 19 January 2018
Received in revised form 27 February 2018
Accepted 1 March 2018
Available online xxxx

Keywords:
Brugada syndrome
Electrocardiogram
Sudden cardiac death

ABSTRACT

Several clinical, electrocardiographic (ECG) and electrophysiological markers have been proposed to provide optimal risk stratification in patients with Brugada syndrome (BrS). Of the different markers, only a spontaneous type 1 ECG pattern has clearly shown a sufficiently high predictive value. This review article highlights specific ECG markers based on depolarization and/or repolarization that have been associated with an increased risk of arrhythmic events in patients with BrS.

1. Introduction

The Brugada syndrome (BrS) is considered a primary arrhythmogenic disorder associated with increased risk of sudden cardiac death due to polymorphic ventricular arrhythmias in patients without overt structural cardiac abnormalities [1,2]. The syndrome is responsible for 4–12% of all sudden deaths and at least 20% of deaths in patients with structurally normal hearts [3].

There are two main, not necessarily mutually exclusive, mechanisms on the pathophysiologic basis of BrS: the depolarization and the repolarization hypotheses [4,5], with much insights derived from pre-clinical animal models [6–10]. According to the depolarization hypothesis, the delayed depolarization of the right ventricular outflow tract (RVOT) creates a potential difference between it and the right ventricle. The repolarization model is related to the higher level of transmural dispersion of repolarization, driven by the loss of the spike and dome action potential morphology at right ventricular epicardium, involving in local and transmural repolarization alterations leading to phase 2 re-entry [2,11].

Although the diagnosis of asymptomatic BrS patients may be achieved relatively easily through ECG, the risk stratification of these patients has still been one of the most challenging and - up to now - unresolved clinical problems. Currently, guidelines provide clear recommendations for the management of symptomatic patients [1]. On the contrary, there is no consensus for the asymptomatic patients and the management depends on evaluation of different parameters. Several clinical, ECG and electrophysiological markers have been proposed to provide optimal risk stratification [1]. This review article briefly describes current knowledge on the assessment of the risk of arrhythmic events in patients with BrS based on ECG markers.

2. Brugada syndrome diagnosis and ECG pattern

The diagnosis of BrS is based on the characteristic coved-type ST-segment elevation in at least one of the right precordial leads V1 and V2 positioned in the 2nd, 3rd or 4th intercostal space (Fig. 1). The diagnostic type 1 electrocardiogram (ECG) may occur spontaneously or after drug challenge with a sodium channel blocker (ajmaline, flecainide, procainamide or pilsicainide) which can convert type 2 or type 3 to type 1 ECG pattern [1]. Type 2 ECG pattern shows a high take-off ≥2 mm and a saddleback ST-configuration ≥1 mm, while type 3 pattern is characterized by J-point elevation of <2 mm and either a saddleback or coved-type ST-segment elevation of ≤1 mm.

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https://doi.org/10.1016/j.ijcha.2018.03.001

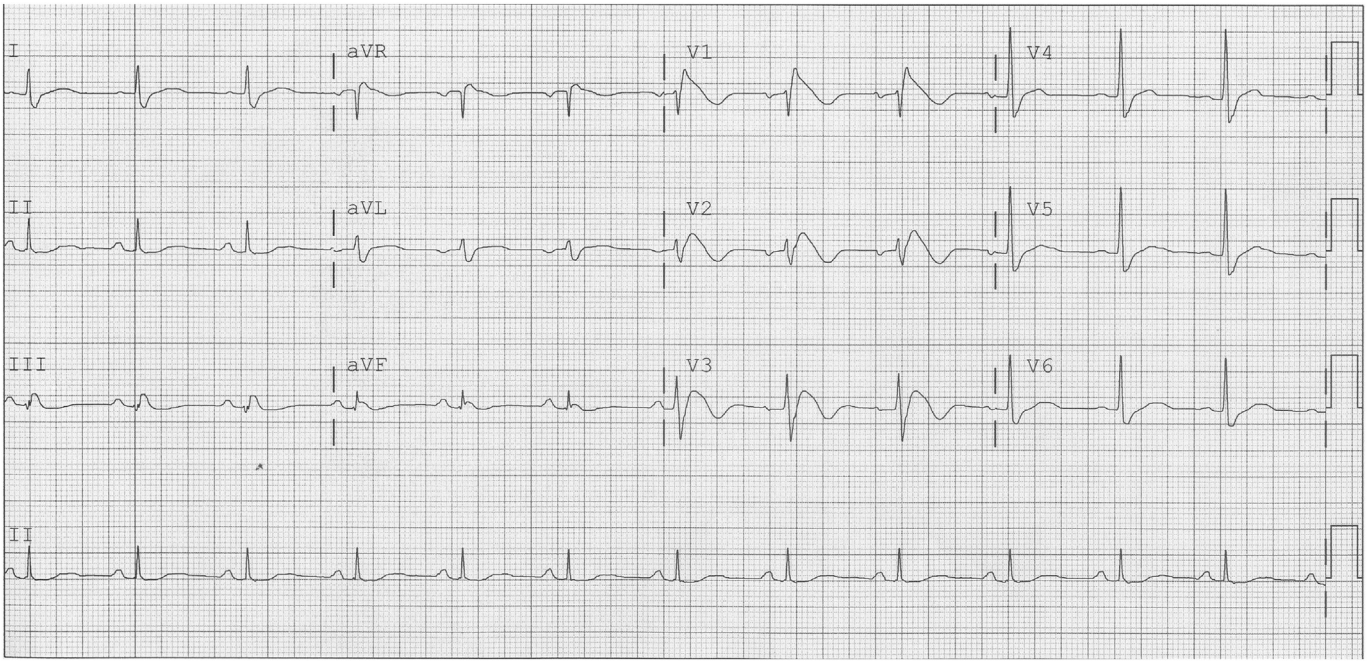

Fig. 1. Spontaneous type 1 ECG pattern of BrS in lead V1.
Reproduced from [69] with permission.

Although the type 1 ECG pattern is diagnostic for BrS, the type 2 pattern requires diagnostic distinction between true BrS and different clinical entities (myocardial ischemia, pulmonary embolism, electrolyte abnormalities), the so called Brugada *phenocopies* [12], which may develop a similar ECG pattern in mimicking conditions such as coronary artery dissection [13], myocardial infarction [14], hyperkalaemia [15], or pulmonary embolism [16]. Therefore, a careful assessment of the ECG is required in order to make an accurate diagnosis. The use of the β-angle (≥58°) (Fig. 2) and base of the triangle (Fig. 3) in type 2 Brugada ECG pattern may distinct a true Brugada ECG from other conditions with high sensitivity and specificity [17,18].

3. High risk ECG markers in Brugada syndrome

There has been evidence that subjects with spontaneous coved type ECG pattern are at higher risk than those with drug-induced ECG for arrhythmic events [19,20]. A meta-analysis showed that the presence of a spontaneous type 1 Brugada ECG predicts a more malignant natural history exhibiting a 3-fold to 4-fold increased risk of adverse events compared to those with a drug-induced Brugada ECG pattern [21]. A recent meta-analysis reported that the prevalence of a type 1 pattern was higher in male, Asians, adults, and fever subjects [22]. Of the different markers, only the presence of a spontaneous type 1 ECG pattern has clearly shown sufficiently high risk predictive value. Nevertheless, as reviewed previously [23], fragmented QRS complexes and early

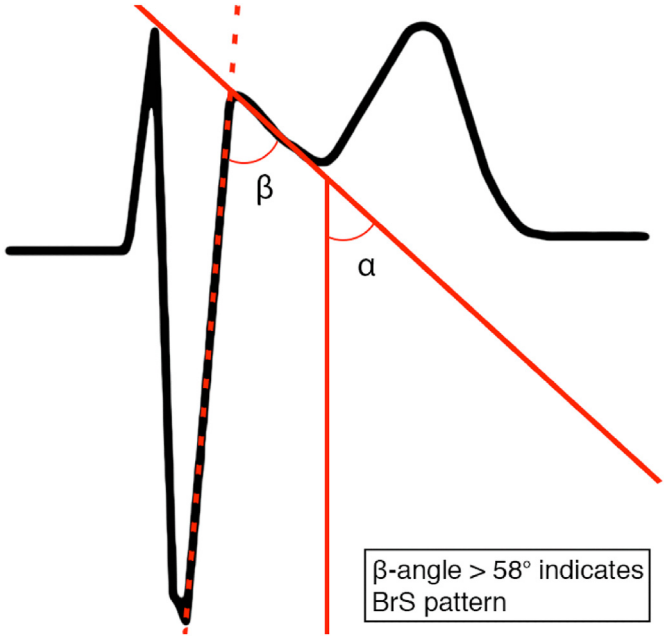

Fig. 2. Beta angle helps distinguish type 2 Brugada pattern from Brugada phenocopies.

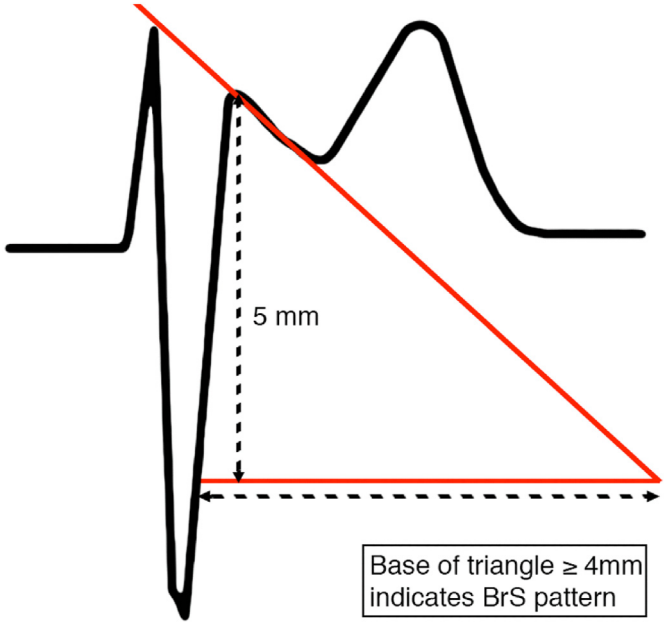

Fig. 3. Base of triangle helps distinguish type 2 Brugada pattern from Brugada phenocopies.