CLINICAL ARRHYTHMIA
Case Report

https://doi.org/10.24207/jca.v35i1.3468

Type 1 Brugada Pattern Unmasked During the Recovery Phase of Treadmill Test

Guilherme Dagostin de Carvalho[1,*], Luciana Vidal Armaganijan[1], Eduardo Giovanini[1], César Henrique Morais Alves[1], Vinícius Motta Machado de Aguiar[1], Silvio Zampieri Ribeiro[1], Luadir Gasparotto Júnior[1], Amanda Vanessa Demarchi[1], Marcos Vinícius de Oliveira Montesi[1], Paulo de Tarso Jorge Medeiros[1]

ORCID ID

Carvalho GD https://orcid.org/0000-0001-8587-875X
Armaganijan LV https://orcid.org/0000-0003-3100-3557
Giovanini E https://orcid.org/0000-0001-8656-8980
Alves CHM https://orcid.org/0000-0001-8191-8240
Aguiar VMM https://orcid.org/0000-0001-7824-3043
Ribeiro SZ https://orcid.org/0000-0001-5889-958X
Gasparotto Júnior L https://orcid.org/0000-0001-8411-9356
Demarchi AV https://orcid.org/0000-0003-3430-2253
Oliveira Montesi MVO https://orcid.org/0000-0001-5889-958X
Medeiros PTJ https://orcid.org/0000-0002-5938-3208

ABSTRACT

We described a case of a 39-year-old asymptomatic patient presenting a coved type 1 Brugada pattern during the recovery phase of treadmill testing. Electrophysiological study resulted in induced polymorphic ventricular tachycardia and implantable cardioverter defibrillator was indicated.

KEYWORDS: Brugada syndrome; Exercise test; Electrophysiologic techniques, cardiac.

INTRODUCTION

Brugada syndrome (BrS) is associated with increased risk of sudden cardiac death in young patients. Treadmill test may unmask BrS pattern in patients with suspected but non-diagnostic electrocardiogram (ECG) especially during the recovery phase due to increased vagal tone.

CASE REPORT

A 39-year-old asymptomatic patient was referred for treadmill testing in order to initiate physical activities. Medical history was remarkable for controlled systemic hypertension with losartan. Neither other comorbidities nor family history of sudden death were reported. There were no abnormalities on physical examination.

Baseline 12-lead ECG showed sinus rhythm, normal axis, PR and corrected QT intervals, and ST elevation of 1.5 mm in precordial leads suggestive of non-type 1 Brugada pattern. During exercise, no significant changes

1. Dante Pazzanese Institute of Cardiology – São Paulo (SP), Brazil.
***Correspondence author::** dagostinguilherme@gmail.com
Received: Jan 2, 2022 | **Accepted:** Mar 7, 2022

in ventricular repolarization were seen with increased heart rate. In the recovery phase, however, coved-type ST elevation > 2 mm, followed by negative T wave, was noted, compatible with type 1 Brugada pattern (Fig. 1). No ischemia and no arrhythmias were reported. ECG showed preserved biventricular function and no wall motion abnormalities.

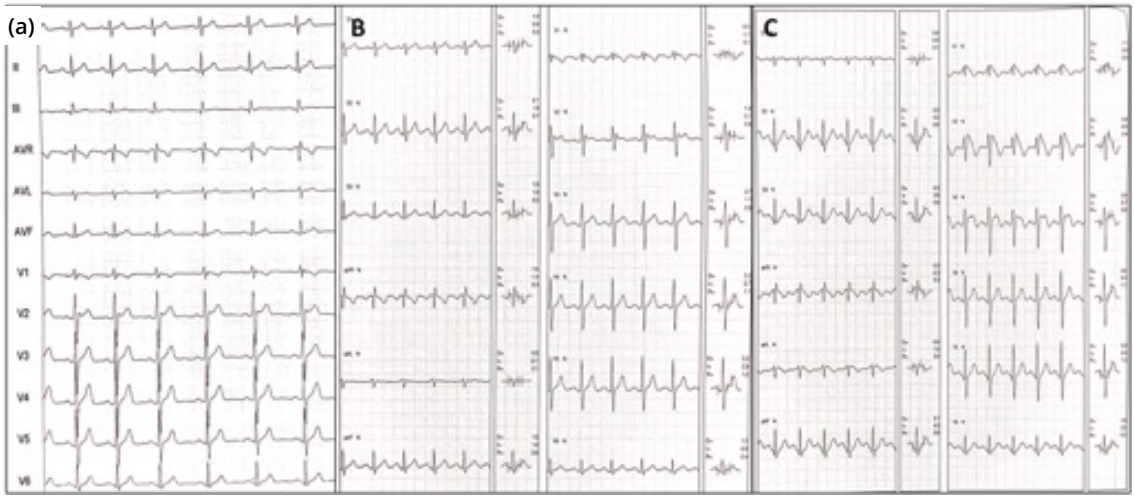

Figura 1. (a) ST elevation of 1.5 mm in precordial leads suggestive of non-type 1 Brugada pattern on baseline electrocardiogram, and (b) no significant changes in ventricular repolarization during exertion. (c) Coved-type ST elevation > 2 mm, followed by negative T wave, compatible with type 1 Brugada pattern in the recovery phase (d).

Given the spontaneous type 1 Brugada pattern during treadmill test, electrophysiological study was indicated. Sustained polymorphic ventricular tachycardia (VT) with degeneration to ventricular fibrillation (VF) was induced with programmed ventricular stimulation (basic cycle length of 430 ms and 3 extrastimuli) (Fig. 2). Implantable cardioverter defibrillator was then indicated.

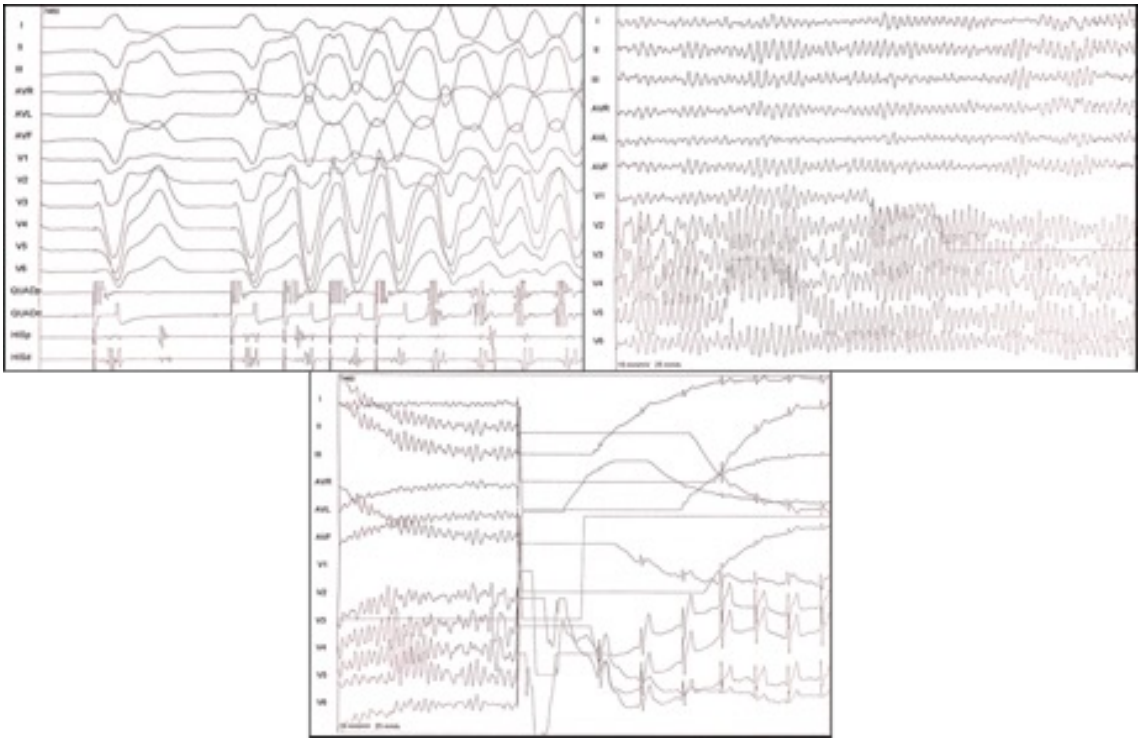

Figura 2. Sustained ventricular tachycardia with degeneration to ventricular fibrillation on electrophysiological testing. Termination with shock.

DISCUSSION

We described a case of BrS unmasked during the recovery phase of a treadmill test. The current diagnosis of BrS is based on the demonstration of type 1 morphology on the ECG, characterized by ST-segment elevation ≥ 2 mm in one or more right precordial leads (V1 to V3) positioned in the second, third or fourth intercostal spaces, spontaneously or after provocative testing with intravenous administration of sodium channel blockers (ajmaline, flecainide, procainamide or pilsicainide)[1].

The prevalence of BrS ranges from 1:2,000 to 1:5,000 individuals. The incidence of the electrocardiographic pattern varies from 0.12 to 0.8% in the literature. The syndrome accounts for 4 to 12% of all sudden cardiac deaths (SCD), and it can reach 20% of them in patients with structurally normal hearts. It predominantly affects males, with prevalence of two to 10 times higher in men than women[2].

Patients with BrS may present syncope, seizures, and agonizing nocturnal breathing due to polymorphic VT or VF. Malignant arrhythmias usually occur during rest or sleep, suggesting an association with bradycardia or vagal tone. Febrile episodes were also related to the appearance of the characteristic electrocardiographic pattern of the syndrome. Diagnosis is usually made in adulthood, with a mean age presentation of 41± 15 years old, although it can also occur in children and the elderly[3,4].

Placing the electrodes in the cranial positions increases the diagnostic sensitivity of the ECG in some patients due to the variable anatomical correlation between the right ventricular outflow tract and the standard position of V1 and V2[5]. The identification of spontaneous type 1 pattern in the upper intercostal spaces provided a similar prognosis for individuals with typical alterations found with the usual placement[6].

Accurate stratification of individuals at high risk for SCD is one of the main challenges in the assessment of patients with BrS. Syncope, spontaneous type 1 pattern on ECG, ventricular effective refractory period < 200 ms and QRS complex fragmentation are predictors of arrhythmic events[7]. Traditionally, the electrophysiological study is used for risk stratification in asymptomatic patients with spontaneous type 1 ECG pattern, and the induction of VF, especially with one or two extrastimuli, is a risk factor in patients with the pathology[8].

Other non-invasive markers of arrhythmogenic risk in BrS have also been suggested and include the ST-segment elevation during recovery on exercise stress testing, the presence of atrial fibrillation (which occurs spontaneously in up to 30% of patients), and early repolarization in inferior and/or lateral leads (which may be present in 10 to 15% of patients with BrS)[9-11].

There are few studies in the literature about the use of exercise testing in BrS. The largest sample on the subject comes from the cohort of Makimoto et al.[12], who analyzed the incidence and clinical significance of ST-segment elevation during the exercise recovery phase. A total of 93 patients with previous diagnosis of BrS were included, 91 (98%) were male, and among them 22 had a documented history of VF, 35 had an episode of syncope and 36 were previously asymptomatic. These, in turn, were compared to 102 healthy controls.

An increase in ST-segment amplitude ≥ 0.05 mV was most frequently reported in leads V1 to V3 in the BrS group, and this finding was associated with the occurrence of VF during the mean follow-up of 76 months (44% versus 17%, p = 0.004). ST elevation was an independent predictor for major arrhythmic events, especially in those with a previous history of VF and syncope, but also in asymptomatic individuals. These data point to a potential applicability of exercise testing for risk stratification in patients with BrS.

The expression of electrocardiographic changes usually becomes more characteristic during vagal predominance. Exercise testing, in turn, causes, in the initial recovery phase, an immediate parasympathetic activation with subsequent gradual adrenergic inhibition. When performing the recovery phase passively, that is, without movement and in horizontal dorsal decubitus, initial vagal stimulus hyperactivation may unmask the Brugada pattern in susceptible individuals[4].

In the present case, BrS was unmasked during treadmill test. Increased vagal tone during the recovery phase may help the diagnosis in patients with suspected but non-diagnostic ECG.

AUTHORS' CONTRIBUTION

Conceptualization: Carvalho GC, Armaganijan LV; **Writing - original draft:** Carvalho GC, Giovanini E, Alves CHM, Aguiar VMM, Ribeiro SZ, Gasparotto Jr L, Demarchi AV, Montesi MV; **Writing - review and editing:** Carvalho GC, Armaganijan LV, Medeiros, PTJ

DATA AVAILABILITY STATEMENT

Not applicable.

FUNDING

Not applicable.

CONFLICT OF INTERESTS

The authors declare no competing interests.

ACKNOWLEDGMENTS

Not applicable.

REFERÊNCIAS

1. LPriori SG, Blomström-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC) Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J. 2015;36(41):2793-867. https://doi.org/10.1093/eurheartj/ehv316

2. Quan X-Q, Li S, Liu R, Zheng K, Wu X-F, Tang Q. A meta-analytic review of prevalence for Brugada ECG patterns and the risk for death: Medicine. 2016;95(50):e5643. https://doi.org/10.1097/MD.0000000000005643

3. Michowitz Y, Milman A, Sarquella-Brugada G, Andorin A, Champagne J, Postema PG, et al. Fever-related arrhythmic events in the multicenter Survey on Arrhythmic Events in Brugada Syndrome. Heart Rhythm. 2018;15(9):1394-401. https://doi.org/10.1016/j.hrthm.2018.04.007

4. Antzelevitch C, Brugada P, Borggrefe M, Brugada J, Brugada R, Corrado D, et al. Brugada Syndrome: Report of the Second Consensus Conference: Endorsed by the Heart Rhythm Society and the European Heart Rhythm Association. Circulation. 2005;111(5):659-70. https://doi.org/10.1161/01.CIR.0000152479.54298.51

5. Nagase S, Hiramatsu S, Morita H, Nishii N, Murakami M, Nakamura K, et al. Electroanatomical Correlation of Repolarization Abnormalities in Brugada Syndrome. J Am Coll Cardiol. 2010;56(25):2143-5. https://doi.org/10.1016/j.jacc.2010.06.050

6. Miyamoto K, Yokokawa M, Tanaka K, Nagai T, Okamura H, Noda T, et al. Diagnostic and prognostic value of a type 1 Brugada electrocardiogram at higher (third or second) V1 to V2 recording in men with Brugada syndrome. Am J Cardiol. 2007;99(1):53-7. https://doi.org/10.1016/j.amjcard.2006.07.062

7. Priori SG, Gasparini M, Napolitano C, Della Bella P, Ottonelli AG, Sassone B, et al. Risk stratification in brugada syndrome. J Am Coll Cardiol. 2012;59(1):37-45. https://doi.org/10.1016/j.jacc.2011.08.064

8. Sroubek J, Probst V, Mazzanti A, Delise P, Hevia JC, Ohkubo K, et al. Programmed ventricular stimulation for risk stratification in the brugada syndrome: a pooled analysis. Circulation. 2016;133(7):622-30. https://doi.org/10.1161/CIRCULATIONAHA.115.017885

9. Takagi M, Aonuma K, Sekiguchi Y, Yokoyama Y, Aihara N, Hiraoka M. The prognostic value of early repolarization (J wave) and ST-segment morphology after J wave in Brugada syndrome: Multicenter study in Japan. Heart Rhythm. 2013;10(4):533-9. https://doi.org/10.1016/j.hrthm.2012.12.023

10. Kawata H, Morita H, Yamada Y, Noda T, Satomi K, Aiba T, et al. Prognostic significance of early repolarization in inferolateral leads in Brugada patients with documented ventricular fibrillation: A novel risk factor for Brugada syndrome with ventricular fibrillation. Heart Rhythm. 2013;10(8):1161-8. https://doi.org/10.1016/j.hrthm.2013.04.009

11. Subramanian M, Prabhu MA, Harikrishnan MS, Shekhar SS, Pai PG, Natarajan K. The utility of exercise testing in risk stratification of asymptomatic patients with type 1 brugada pattern: risk stratification in type 1 brugada pattern. J Cardiovasc Electrophysiol. 2017;28(6):677-83. https://doi.org/10.1111/jce.13205

12. Makimoto H, Nakagawa E, Takaki H, Yamada Y, Okamura H, Noda T, et al. Augmented ST-segment elevation during recovery from exercise predicts cardiac events in patients with brugada syndrome. J Am Coll Cardiol. 2010;56(19):1576-84. https://doi.org/10.1016/j.jacc.2010.06.033