Rev Port Cardiol. 2015;34(4):287.e1–287.e7

Revista Portuguesa de
Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

CASE REPORT

Fever-induced type 1 Brugada pattern☆

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Received 29 December 2013; accepted 10 October 2014

KEYWORDS
Brugada syndrome;
Fever;
Syncope

Abstract Brugada syndrome, first described over 20 years ago, is characterized by a typical electrocardiographic pattern with coved-type ST-segment elevation in the right precordial leads and a high risk of sudden death in otherwise healthy young adults.

The electrocardiographic pattern is sometimes intermittent, and fever is a possible trigger. The authors present the case of a 68-year-old woman who came to the emergency department with fever and syncope. A diagnosis of community-acquired pneumonia was made. The electrocardiogram performed when the patient had fever revealed a type 1 Brugada pattern, which disappeared after the fever subsided. After other causes of Brugada-like pattern were excluded, Brugada syndrome was diagnosed and a cardioverter-defibrillator was implanted.

This case demonstrates that this entity can be diagnosed at more advanced ages and highlights the usefulness of electrocardiography in a febrile state.

PALAVRAS-CHAVE
Síndrome de Brugada;
Febre;
Síncope

Padrão de Brugada tipo 1 induzido pela febre

Resumo A síndrome de Brugada, descrita há cerca de 20 anos, caracteriza-se eletrocardiograficamente por uma elevação convexa do segmento-ST nas derivações precordiais direitas e pelo elevado risco de morte súbita em jovens aparentemente saudáveis.

Este padrão eletrocardiográfico é, por vezes, intermitente, sendo a febre um possível fator precipitante. Os autores apresentam o caso clínico de uma doente de 68 anos que recorre ao serviço de urgência por febre e síncope. Feito o diagnóstico de pneumonia adquirida na comunidade. O eletrocardiograma realizado em contexto de febre revelou um padrão de Brugada

☆ Please cite this article as: Madeira M, Caetano F, Providência R, et al. Padrão de Brugada tipo 1 induzido pela febre. Rev Port Cardiol. 2015. http://dx.doi.org/10.1016/j.repc.2014.10.002

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tipo 1, que desapareceu após resolução do quadro febril. Excluídas outras causas de padrão *Brugada-like* foi confirmado o diagnóstico de síndrome de Brugada e realizada implantação de cardioversor-desfibrilhador.

Este caso ilustra a possibilidade do diagnóstico desta entidade poder ser feito numa faixa etária já avançada e reforça a utilidade da realização de um eletrocardiograma em contexto febril.

Introduction

Brugada syndrome (BrS), first described as a clinical entity in 1992,[1] is an autosomal dominant disease associated with a high risk of sudden cardiac death in otherwise healthy young adults.[2]

BrS is rare, with an estimated prevalence of 5/10 000, and affects both sexes but is eight times more common in males.[2] It is characterized by a typical electrocardiographic pattern with coved-type ST-segment elevation in the right precordial leads (V1–V3).[2] Although this pattern is characteristic, it is sometimes intermittent, which can hinder diagnosis. Fever is a known triggering factor.[3]

The authors present the case of a patient diagnosed with BrS during a febrile state.

Case report

A 68-year-old woman with a history of hypertension, dyslipidemia and smoking was medicated as an outpatient with amlodipine, indapamide and rosuvastatin. She came to the emergency department with fever of one day's duration, pleuritic-type pain in the anterior right hemithorax radiating to the back, non-productive cough and an episode of loss of consciousness without prodrome, of short duration and with spontaneous recovery, and no postcritical period. The patient had no history of previous syncope or nocturnal agonal respiration or family history of sudden death.

On admission she was subfebrile (37.4 °C) and hemodynamically stable (blood pressure 130/65 mmHg and heart rate 88 bpm). Physical examination revealed no abnormalities on cardiac or pulmonary auscultation and there were no indirect signs of deep vein thrombosis.

Laboratory tests showed elevated inflammatory parameters (leukocytes 16 800 $\times 10^3/\mu l$ and C-reactive protein 19.6 mg/dl) but no elevation of D-dimers or troponin I. The chest X-ray revealed a round alveolar opacity in the lower half of the right pulmonary field.

In view of her symptoms and the laboratory and imaging findings, empirical antibiotic therapy was begun for community-acquired pneumonia (amoxicillin/clavulanic acid and azithromycin).

An electrocardiogram (ECG) performed to investigate the episode of loss of consciousness revealed first-degree atrioventricular block, incomplete right bundle branch block (RBBB) and ST-segment elevation, down-sloping in V1 and V2 and horizontal in V3, together with T-wave inversion (Figure 1).

A diagnosis of acute coronary syndrome was excluded, despite the existence of multiple cardiovascular risk factors, since the patient presented with non-typical chest pain and no elevation in myocardial necrosis markers was seen in serial assessments. The possibility of pulmonary embolism suggested by the patient's symptoms was ruled out by normal D-dimer levels.

A diagnosis of BrS (fever-induced type 1 electrocardiographic pattern and syncope) was assumed and the patient was admitted to the cardiac intensive care unit (CICU) for monitoring of arrhythmias.

As the infection resolved, the ECG evolved to a type 3 Brugada pattern (Figure 2). However, an ECG with leads V1 and V2 placed in the third intercostal space continued to show a type 1 Brugada pattern (Figure 3).

There were no arrhythmic events during the patient's stay in the CICU.

Transthoracic echocardiography ruled out structural or functional heart disease. In the light of a definitive diagnosis of BrS in a patient with syncope, an implantable cardioverter-defibrillator (ICD) was implanted, in accordance with the European Society of Cardiology guidelines (class IIa recommendation).[4]

The patient was referred for genetic study but no mutation was found. She has two children, who are asymptomatic and have normal ECG.

At 36-month ICD follow-up no arrhythmic events had been detected.

Discussion

BrS is a hereditary channelopathy with autosomal dominant transmission and incomplete penetrance (approximately 16%, but this figure varies in different families).[5] It is a relatively common cause of sudden cardiac death (4% of all sudden deaths) and is responsible for up to 20% of sudden deaths in patients without structural heart disease.[6] The clinical phenotype is eight times more common in men than in women.[7] The first arrhythmic event occurs at a mean age of 40.[7]

The case presented here is somewhat atypical, since the first clinical manifestation (syncope) occurred in a female patient in her late 60s.