

# Fever in a patient with common cold and Brugada syndrome, what should we do?

**Keywords:** brugada syndrome, potential triggers, psychotropic drugs, anesthetic agents, cocaine, excessive alcohol, fever, shock, consciousness, headache, coughing, sneezing, malaise, autosomal dominant inherited disorder, ventricular arrhythmias, diagnosis, arrhythmia unit, propranolol, sodium channels, output potassium, electric ICD

## Introduction

Brugada syndrome is characterized by a coved type ST-segment of  $\geq 2$  mm with a negative or flattened T wave in the precordial leads (V1-V3) and an increased risk of potentially fatal ventricular tachyarrhythmia's episodes.<sup>1,2</sup> Several potential triggers of ventricular arrhythmias have been described, such as the use of some psychotropic drugs, anesthetic agents, cocaine, excessive alcohol intake or fever.<sup>3</sup>

## Clinical case

A 27-years-old male patient, diagnosed with Brugada syndrome (Figure 1) clinically debut with cardiac arrest in ventricular fibrillation. In 2009 an implantable cardioverter defibrillator (ICD) was placed in a context of secondary prevention. In a 3 years period it has adequately addressed several episodes of malignant ventricular arrhythmias

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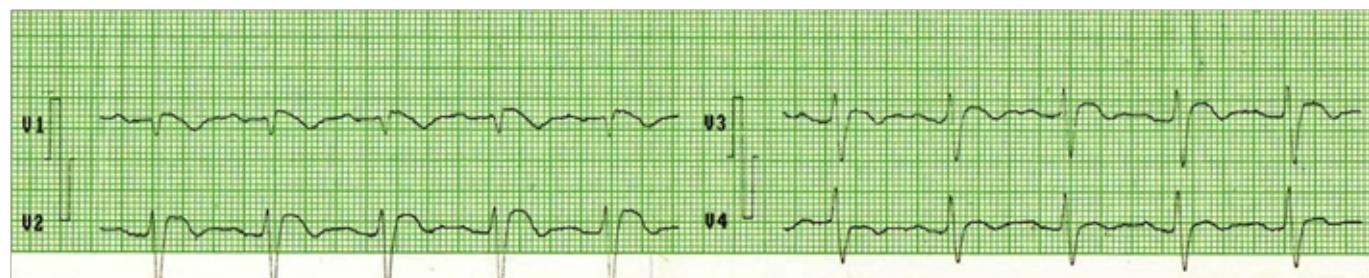
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(ventricular fibrillation and polymorphic ventricular tachycardia). In the last 3 years he has not presented events registered by the device and he has remained under treatment with quinidine and propranolol. Two days ago he begins to present some symptoms related to common cold (headache, coughing, sneezing, malaise), with a temperature of 38.5 °C. Approximately one hour later, he had palpitations, felt an electric ICD shock and suddenly lost consciousness with complete recovery within a few seconds. When he was valued in an arrhythmia unit, the diagnosis of appropriate ICD shock was made.



**Figure 1** Electrocardiogram showing the Brugada sign (coved pattern) in the right precordial leads (V1-V3).

## Discussion

The Brugada syndrome is an autosomal dominant inherited disorder with incomplete or variable penetrance. Linked to mutations in 19 different genes especially in the SCN5A.<sup>4</sup> These mutations cause a function loss of ion current input especially inactivating sodium channel (INa) which further enhance the ionic currents output potassium.<sup>2</sup> Inactivating sodium channel has also been associated with changes in body temperature and in several studies have demonstrated the association between febrile conditions and the appearance of its characteristic electrocardiographic pattern, increased ST segment elevation or even the presence of ventricular arrhythmias.<sup>5-7</sup> As mentioned above the current literature recommend early and aggressively treatment of any febrile episode in patients with this syndrome.<sup>3-8</sup>

## Acknowledgements

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## Conflict of interest

The author declares there is no conflict of interest.

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