

IMAGES IN MEDICINE

Fever-Induced Type 1 ECG Pattern of Brugada Syndrome

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ABSTRACT

A surface electrocardiogram (ECG) on admission in a 38-year-old lady performed during a febrile state (38.8°C) revealed the typical coved type 1 ECG pattern of Brugada syndrome. Two hours later, when the patient was afebrile, a repeat ECG failed to show any abnormality. A febrile state may unmask Brugada syndrome but has also been reported to even precipitate ventricular fibrillation. Thus, fever should be vigorously treated with antipyretics in these patients.

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KEY WORDS: *Brugada syndrome;
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A 38-year-old female with a history of tobacco use presented to the emergency department with symptoms and signs of an upper respiratory infection. ECG on admission performed during febrile state (38.8°C) revealed the diagnostic type 1 ECG pattern of Brugada syndrome, a pattern characterized by coved type ST-segment elevation > 2 mm followed by negative T waves in the right precordial leads (V₁ and V₂) (Fig. 1). Two hours later, a new ECG performed without fever failed to show any of these abnormalities (Fig. 2). Serum electrolytes and biochemical markers of myocardial damage were normal. Transthoracic echocardiography demonstrated normal wall motion of both ventricles with an estimated left ventricular ejection fraction of 70%. Ambulatory Holter ECG monitoring demonstrated low heart rate variability without any arrhythmias. Her past medical history was unremarkable and negative for syncope or family history of Brugada syndrome. The patient is under a close follow-up and she was counselled to use antipyretics whenever a fever develops as well as to avoid certain drugs that unmask the Brugada ECG pattern.¹



The Brugada syndrome is an arrhythmogenic entity that typically manifests with syncope or cardiac arrest due to ventricular fibrillation in individuals with structurally normal hearts.^{2,3} The *SCN5A* gene encoding for the cardiac sodium channel (Nav1.5) was the first gene linked to BS, found in 11% to 28% of Brugada syndrome probands.^{3,4} Mutations in *CACNA1C* (Cav1.2), *CACNB2b* (Cavβ2b), glycerol-3-phosphate dehydrogenase 1-like enzyme gene (*GPD1L*), *SCN1B* (β1 subunit of sodium channel), *KCNE3* (MiRP), and *SCN3B* (β3 subunit of sodium channel) are much more rare.⁴

The ECG features of Brugada syndrome are often concealed. Sodium channel blockers, vagotonic agents, α-adrenergic agonists, β-adrenergic blockers, tricyclic

ABBREVIATIONS

ECG = electrocardiogram

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FEVER UNMASKING BRUGADA SYNDROME

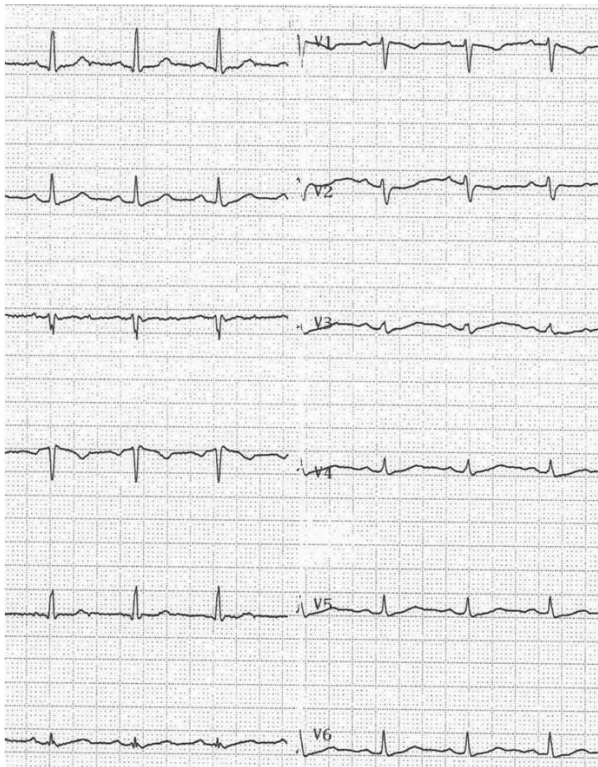


FIGURE 1.



FIGURE 2.

antidepressants, glucose-induced insulin secretion, fever, hyperkalemia, hypokalemia, hypercalcemia, alcohol and cocaine toxicity have been shown to unmask or modulate the characteristic ST-segment elevation in right precordial leads.^{3,4} Previous reports have demonstrated that febrile state unmask or accentuate the ECG pattern of Brugada syndrome and precipitate ventricular fibrillation occurrences.⁵⁻⁸ At a molecular level, the ionic mechanisms responsible for the ECG pattern of Brugada syndrome have been shown to be temperature dependent. Dumaine et al reported an accelerated inactivation of the sodium current carrying the T1620M missense mutation at high temperatures.⁹ Keller et al. identified a novel SCN5A mutation, F1344S, in a patient with Brugada syndrome and fever-induced ventricular fibrillation. The mutation resulted in loss of sodium channel function due to shift of activation.¹⁰ Due to the increased risk of ventricular arrhythmias during febrile state regardless of the existence of a predisposing genetic base,¹¹ fever should be vigorously treated with antipyretics in these patients.

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