

# Does fever make difference?

## Abstract

Brugada syndrome is increasingly recognized as a cause of cardiac death. Many of these patients do not get diagnosed due to dynamic and often hidden nature. We have come a long way in understanding the disease process, and its electrophysiology appears to be intimately linked with sodium channel mutations or disorders. The cardiac rhythm in these patients can deteriorate into fatal ventricular arrhythmias. This makes it important for the clinician to be aware of the condition in which arrhythmogenicity of Brugada syndrome is revealed or even potentiated. We present such an instance where our patient's Brugada syndrome was unmasked by fever.

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## Case report

22 years Old male presented to ER with history of dizzy spells associated with lack of concentration and being mentally inattentive for short periods. He described it as if, he is parting from his surroundings; these episodes are short lived and usually follow extreme exertion and heavy meals. His most recent episode was preceded with upper respiratory tract infection with high fever of 38 degrees. He denied history of loss of consciousness, syncope, seizure, chest pain or palpitations. There was no family history of sudden cardiac death. There was no abusing drugs and denied use of any new medication. He was hemodynamically stable T: 38, BP: 120/80.

PSO:97% RA RR: 16 P: 90 B/M.

Chest: bilateral equal air entry no added sound.

CVS: S1, S2, no add sound or murmur.

His initial ECG (Figure 1), showed sinus rhythm, RSR pattern in V1 and V2 with ST-elevation in V1 and V2 ECG. We were asked to see the patient as the ECG changes were interpreted as acute myocardial infarction. The ECG was later interpreted by cardiology staff to be typical changed of type 1 brugada syndrome, then asked to do ECG with higher position of v1,v2 (Figure 2) shows more pronounced changes(coved ST elevation) and (Figure 3) demonstrate the effect of normalization of body temperature with complete resolution of the ECG changes. The patient was admitted to cardiology monitored bed started on antipyretic medication as well as antibiotics, later he developed more fever and had recurrence of his ECG changes (Figure 4). He was counselled regarding the need for electrophysiological study, genetic counselling and need for defibrillator.

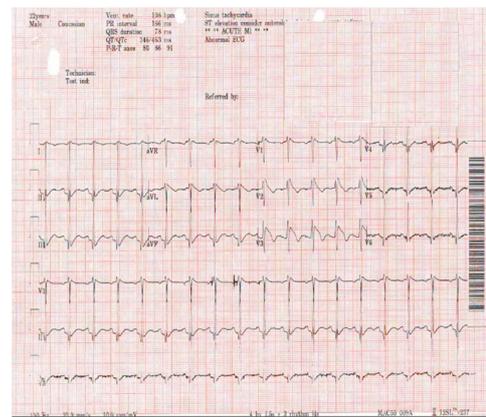


Figure 2 ECG with higher v1, v2.

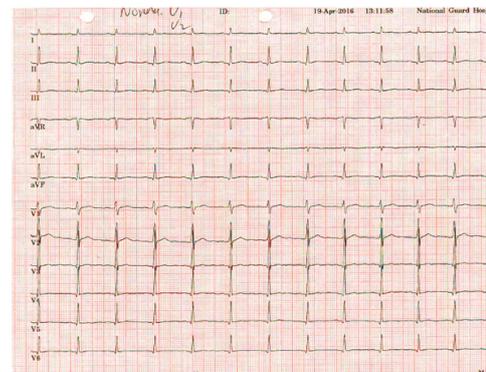


Figure 3 ECG after 2 hour when t: 36, 7.

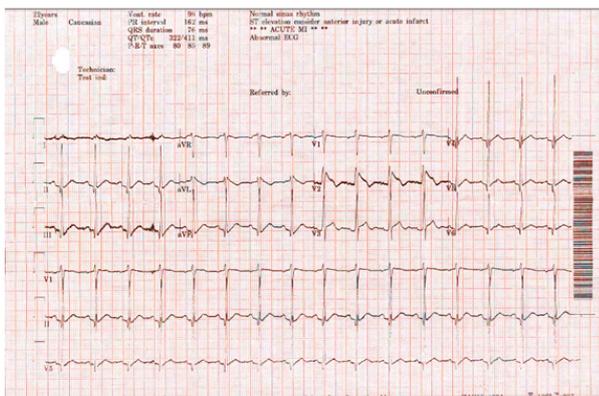


Figure 1 Initial ECG.

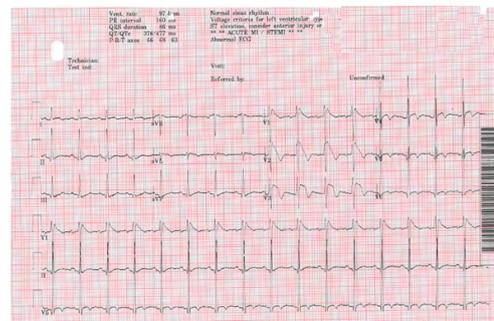


Figure 4 To keep patient apyrexia and ABS was given and full discussion with patient about genetic counseling and EPS then at night pt. developed fever again so ECG at that time.

## Discussion

Brugada syndrome is a genetic disorder with a characteristic ECG finding of persistent or transient ST segment elevation in the right precordial leads (V1, V2) with or without right bundle branch block. These ECG changes are dynamic, often hidden, and may reveal themselves in the presence of triggers like fever, intoxication (alcohol, cocaine, or cannabis), vagal stimulation, electrolyte imbalance, anesthetics (propofol, bupivacaine), psychotropic agents (amitriptyline, lithium), and sodium channel blockers.<sup>1</sup> Fever-induced Brugada syndrome is becoming a well-known entity. In a study by Amin et al.<sup>2</sup> it was noted that fever was the precipitating factor for about 18% of the cardiac arrests in patients with symptomatic Brugada syndrome. Adler et al.<sup>3</sup> in their study found that a type I Brugada ECG pattern was 20 times more likely to occur in febrile patients than febrile patients. This translated to a prevalence of 2%, whereas the estimated prevalence of asymptomatic Brugada syndrome in the general population is 0.05%.<sup>4</sup> They also found the patients to be typically in an age group of 30 to 60 years, and 87% were of the male gender,<sup>4</sup> a finding that was seen in other studies as well.<sup>5</sup> This brings up an interesting possibility of an androgen hormone relationship to these patients with potential fatal ventricular arrhythmias.<sup>4</sup> If our patient was female, it is possible that her postmenopausal state contributed to the appearance of the Brugada ECG in this setting. Brugada syndrome has three different ECG patterns.<sup>5</sup>

- Type 1 has a coved type ST elevation with at least 2 mm (0.2 mV) J-point elevation a gradually descending ST segment followed by a negative T-wave.
- Type 2 has a saddle back pattern with a least 2 mm J-point elevation and at least 1 mm ST elevation with a positive or biphasic T-wave. Type 2 pattern can occasionally be seen in healthy subjects.
- Type 3 has either a coved (type 1 like) or a saddle back (type 2 like) pattern with less than 2 mm J-point elevation and less than 1 mm ST elevation. Type 3 pattern is not rare in healthy subjects.

The pattern seen on the ECG is persistent ST elevations in the electrocardiographic leads V1-V3 with a right bundle branch block (RBBB) appearance with or without the terminal S waves in the lateral leads that are associated with a typical RBBB. A prolongation of the PR interval (a conduction disturbance in the heart) is also frequently seen. The ECG can fluctuate over time, depending on the autonomic balance and the administration of antiarrhythmic drugs. Adrenergic stimulation decreases the ST segment elevation, while vagal stimulation worsens it. (There is a case report of a patient who died while shaving, presumed due to the vagal stimulation of the carotid sinus massage.) The administration of class Ia, Ic and III drugs increases the ST segment elevation, as does fever.<sup>6</sup> Exercise decreases ST segment elevation in some patients but increases it in others (after exercise when the body temperature has risen). The changes in heart rate induced by atrial pacing are accompanied by changes in the degree of ST segment elevation. When the heart rate decreases, the ST segment elevation increases and when the heart rate increases the ST segment elevation decreases. However, the contrary can also be observed. The cause of sudden death in Brugada syndrome is ventricular fibrillation (VF). The average age of death is 41. According to clinical reports, sudden death in people with Brugada syndrome most often happens during sleep. The episodes of syncope (fainting) and sudden death (aborted or not) are caused by fast polymorphic ventricular tachycardias or ventricular fibrillation. These

arrhythmias appear with no warning. While there is no exact treatment modality that reliably and totally prevents ventricular fibrillation from occurring in this syndrome, treatment lies in termination of this lethal arrhythmia before it causes death. This is done via insertion of an implantable cardioverter-defibrillator (ICD), which continuously monitors the heart rhythm and will shock the wearer if ventricular fibrillation is sensed. Patients with BrS who develop F-type1 are at risk of arrhythmic events. F-type1 appears to develop through a more complex mechanism as compared with drug-induced type I ECG.

## Conclusion

In the past few years, a number of case reports described that fever triggers the clinical manifestations of BrS. Hence, based on these studies and the present work, BrS should be considered in any patient with syncope during febrile state. The genetic background of BrS is heterogeneous. In the small population of fever-susceptible cases presented here, two patients out of four did not carry any mutation in SCN5A. Additional investigations are therefore needed to identify the genetic bases of this disorder. Finally, as illustrated by our findings, the molecular and cellular mechanisms underlying the fever-dependent manifestations of BrS are complex. Further studies are needed in order to elucidate this important issue since there is no curative treatment available for BrS to date.

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## Conflicts of interest

The authors declare no conflicts of interest.

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