

Fever-Induced Brugada Phenocopy: A Case Report

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Abstract

Brugada phenocopy (BrP) refers to reversible electrocardiographic (ECG) patterns that mimic Brugada syndrome (BrS) in the absence of an underlying congenital channelopathy. Fever is a recognized trigger that can unmask Brugada-like ECG changes, particularly in older adults. An 83-year-old man presented to the emergency department with fever and respiratory symptoms. Initial ECG demonstrated a type 1 Brugada-like pattern. Laboratory evaluation revealed inflammatory markers consistent with infection. Following antipyretic treatment and normalization of body temperature, repeat ECG showed complete resolution of the Brugada-like changes. The patient was monitored in the cardiology unit without arrhythmic events. This case highlights fever as a reversible cause of Brugada phenocopy in an elderly patient. Prompt recognition and treatment of the underlying trigger are essential to avoid unnecessary invasive interventions such as implantable cardioverter-defibrillator implantation.

Keywords: Brugada phenocopy, elderly, electrocardiography, emergency department, fever

Introduction

Brugada phenocopy (BrP) is characterized by an electrocardiographic (ECG) pattern resembling Brugada type 1 or 2 without the congenital channelopathy found in Brugada Syndrome (BrS). In patients with BrS, three distinct ECG patterns can be observed in the right precordial leads (V1-V3): coved-type ST-segment elevation >2 mm with T-wave inversion (type 1), saddleback ST-segment elevation >2 mm (type 2), or <2 mm (type 3) (1–3). These ECG abnormalities usually resolve after the underlying cause is addressed. This case highlights fever-induced BrP, emphasizing that in the absence of congenital channelopathy, Brugada-like ECG changes may normalize once the fever subsides.

Case Report

An 83-year-old male patient was admitted to the emergency department with complaints of fever, cough, and fatigue.

His medical history included type 2 diabetes mellitus and hypertension. On physical examination, the patient had pharyngeal hyperemia, rales in the right lung, and pretibial edema. At admission, his blood pressure was 139/67 mmHg, heart rate was 103 bpm, body temperature was 39°C, and oxygen saturation was 98%. The initial ECG showed a Brugada type 1-like pattern (Figure-1). Laboratory findings revealed a white blood cell count of $12.22 \times 10^9/L$, C-reactive protein of 88.7 mg/L, sodium of 133 mmol/L, and blood glucose level of 324 mg/dL, while other parameters were within normal limits. A non-contrast thoracic CT scan showed minimal pleural effusion. Given the fever, cough, right-sided rales, and radiological findings, a lower respiratory tract infection was considered the most likely source of fever and started antibiotic treatment. The patient received symptomatic treatment, including antipyretics and hydration. When his body temperature decreased to 36.5°C, a repeat ECG showed normalization of the previously observed Brugada-like pattern (Figure-2). The patient was admitted to the cardiology department for further monitoring.

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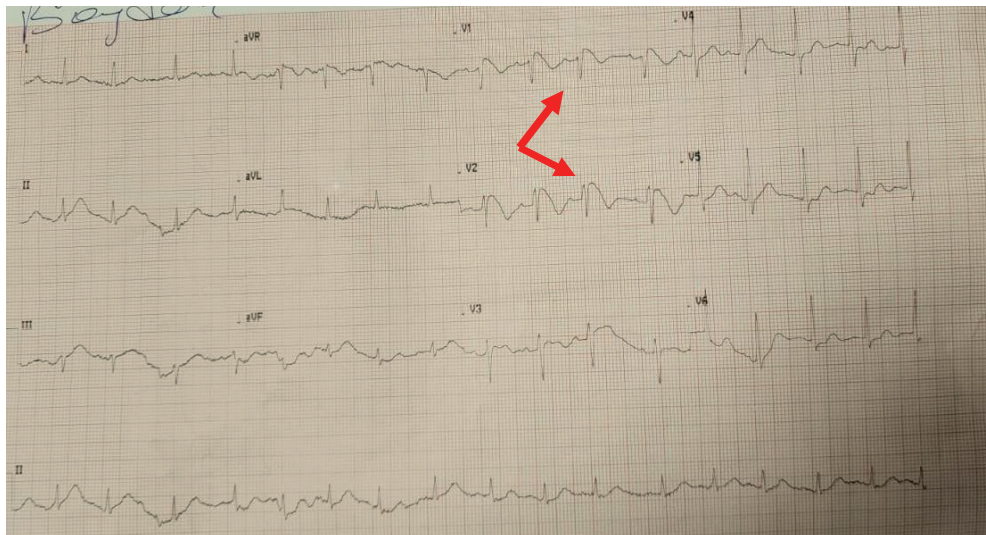


Figure 1. The patient's ECG at Admission: Brugada Phenocopy (red arrow: Coved-type ST-segment elevation with T-wave inversion in leads V1-V2, consistent with a type 1 Brugada-like pattern)

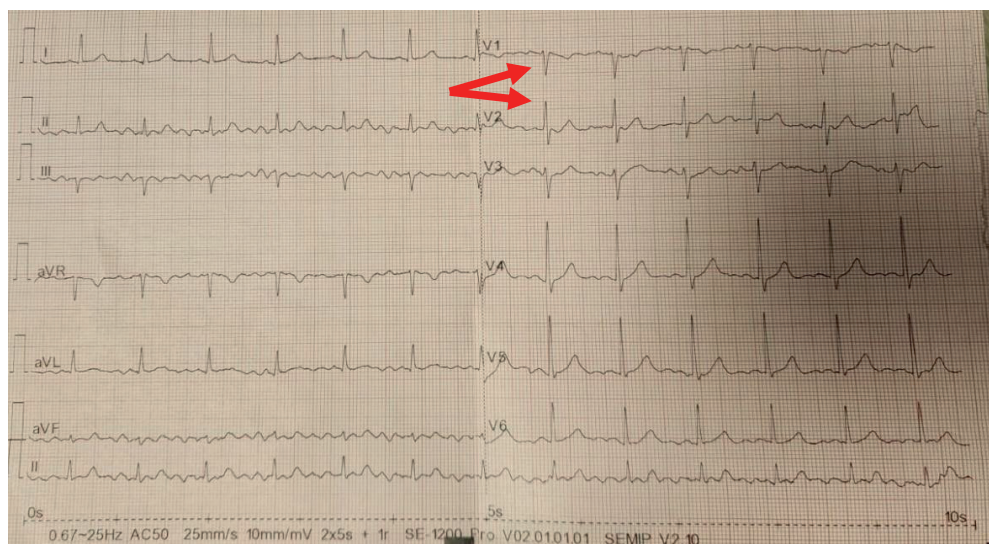


Figure 2. The patient's ECG after antipyretic treatment (red arrow: highlights normalization of the ST segment in leads V1-V2 following defervescence)

Discussion

BrP shares similar ECG patterns with BrS but has different underlying causes. BrS is a genetic disorder known to increase the risk of sudden cardiac death, whereas BrP is a transient ECG pattern triggered by various clinical conditions (4,5). The etiology of BrP commonly includes metabolic disorders (hyperkalemia, hypokalemia, acidosis, hypothermia, hyperthermia, etc.), mechanical compression (mediastinal tumors, etc.), ischemia (myocardial infarction), myocardial and pericardial diseases (myocarditis, pericarditis, etc.), and miscellaneous causes (electric burns, etc.) (6,7). Unlike hypothermia, the precise mechanism by which hyperthermia leads to BrP in previously healthy individuals remains unclear. In our case, fever was identified as the probable trigger based on the clinical presentation.

The diagnosis of BrP rather than true BrS was established according to the criteria proposed by Baranchuk et al. (7), requiring (1) a type 1 or type 2 Brugada ECG pattern, (2) an identifiable underlying condition capable of inducing such changes, (3) resolution of the ECG pattern after the trigger is corrected, and (4) the absence of a personal or family history of syncope, sudden cardiac death, or a previously documented spontaneous type 1 pattern. Our patient fulfilled all four criteria: the type 1 Brugada-like pattern coincided temporally with fever, it fully normalized after antipyretic treatment and defervescence, and there was no personal or family history suggestive of BrS. Genetic testing was therefore not pursued, in keeping with current practice when a clear reversible trigger is present and the ECG normalizes. The ECG pattern normalized after symptomatic treatment and body temperature reduction. Fever-induced BrP is rarely

reported in the literature. The absence of a family history, lack of BrS-associated symptoms, and no medication use are key distinguishing features in these cases. The management of BrP focuses on identifying and treating the underlying cause. Once reversible factors are corrected, ECG abnormalities typically resolve, underscoring the importance of targeted therapy. In contrast, the management of BrS often involves the placement of an implantable cardioverter-defibrillator (ICD) (5).

Conclusion

BrP, unlike BrS, is a transient ECG pattern that requires targeted treatment. Patients with BrP do not require interventions such as ICD implantation. From a clinical perspective, distinguishing Brugada phenocopy from true Brugada syndrome is particularly important in the emergency department, where rapid decision-making is required. Fever is a well-recognized precipitating factor that may transiently alter sodium channel function and induce Brugada-like ECG patterns, even in patients without underlying channelopathies. Misinterpretation of such reversible ECG changes as true Brugada syndrome may lead to unnecessary invasive interventions, including implantable cardioverter-defibrillator implantation, with associated risks and costs. This case underscores the importance of reassessing the ECG after correction of reversible triggers such as hyperthermia. Awareness of fever-induced Brugada phenocopy may help emergency physicians and cardiologists avoid overdiagnosis, reduce unnecessary procedures, and focus on treating the underlying precipitating condition.

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References

1. Sarquella-Brugada G, Campuzano O, Arbelo E, Brugada J, Brugada R. Brugada syndrome: Clinical and genetic findings. *Genetics in Medicine*. 2016. <https://doi.org/10.1038/gim.2015.35>
2. Othieno AA, Isaacs DJ, Vinson DR, Levis JT. ECG Diagnosis: Brugada Syndrome. *Permanente Journal*. 2019. <https://doi.org/10.7812/TPP/19.044>
3. Veltmann C, Schimpf R, Echternach C, Eckardt L, Kuschyk J, Streitner F, et al. A prospective study on spontaneous fluctuations between diagnostic and non-diagnostic ECGs in Brugada syndrome: Implications for correct phenotyping and risk stratification. *Eur Heart J*. 2006;27. <https://doi.org/10.1093/eurheartj/ehl205>
4. Campuzano O, Sarquella-Brugada G, Cesar S, Arbelo E, Brugada J, Brugada R. Update on genetic basis of Brugada syndrome: Monogenic, polygenic or oligogenic? *Int. J. Mol. Sci*. 2020. <https://doi.org/10.3390/ijms21197155>
5. Adytia GJ, Sutanto H. Brugada phenocopy vs. Brugada syndrome: Delineating the differences for optimal diagnosis and management. *Curr Probl Cardiol*. 2024;49:102566. <https://doi.org/10.1016/j.cpcardiol.2024.102566>
6. Eckardt L, Veltmann C. More than 30 years of Brugada syndrome: a critical appraisal of achievements and open issues. *Herzschrittmachertherapie und Elektrophysiologie*. 2024;35. <https://doi.org/10.1007/s00399-023-00983-y>
7. Baranchuk A, Nguyen T, Ryu MH, Femenía F, Zareba W, Wilde AAM, et al. Brugada phenocopy: New terminology and proposed classification. *Annals of Noninvasive Electrocardiology*. 2012. <https://doi.org/10.1111/j.1542-474X.2012.00525.x>