Brugada ECG pattern associated with fever and hypokalemia

Danielle Burghouw, Natasja de Groot, Jaap Deckers

*Medical student, Unit Translational Electrophysiology, Erasmus MC University Medical Center Rotterdam, the Netherlands

†Supervisor, Unit Translational Electrophysiology, Department of Cardiology, Erasmus MC University Medical Center Rotterdam, the Netherlands

‡Department of Cardiology, Erasmus MC University Medical Center Rotterdam, the Netherlands

Correspondence: Danielle Burghouw, e-mail: 328643db@student.eur.nl

Summary

The Brugada syndrome is a channelopathy characterized by typical ST-segment elevations in the precordial leads, also known as the Brugada pattern. This pattern is associated with an increased risk of sudden cardiac death due to ventricular fibrillation. In this case report we describe a 72-year old man who developed a Brugada ECG pattern during an increase of body temperature and hypokalemia. After reduction of body temperature and in the presence of persistent moderate hypokalemia, the Brugada ECG pattern only partly resolved.

A 72-year old Caucasian male was hospitalized for treatment of acute myeloid leukemia and non-Hodgkin lymphoma with chemotherapy. He developed fever episodes of 38-39 °C of unknown origin. He had no history of cardiovascular diseases, except hypertension. The surface electrocardiograms (ECG) of the past two months revealed sinus tachycardia, most likely due to anemia caused by the malignancies, and incomplete right bundle branch block (Figure 1A). His ECG altered when a body temperature of 40.3 °C was reached; ST-segment abnormalities developed in precordial leads V1 and V2, consistent with a Brugada type 1 morphology (Figure 1B). He did not use any medication known for triggering Brugada pattern in susceptible patients. During this fever episode, severe diarrhea caused hypokalemia (3.1 mmol/l). Previous serum potassium concentrations of 3.3 mmol/l were not associated with ECG abnormalities. Antibiotics and potassium supplements were given and the ST-segment elevations (only) partly resolved (Figure 1C).

Figure 1 ECG before, during and after the fever peak

Figure legend

Fig. 1A ECG during hospitalization revealed sinus tachycardia and incomplete right bundle branch block; patient body temperature was 37.3 °C, potassium 3.8 mmol/l.

Fig. 1B ECG recorded 2 days later. Body temperature reached up to 40.3 °C and there was a moderate hypokalemia. Precordial leads V1 and V2 revealed a coved ST-segment, consistent with a Brugada pattern type 1.

Fig. 1C ECG recorded 6 days after the start of potassium supplementation. Fever episodes (< 39.5 °C) and hypokalemia (2.9 mmol/l) were still present. The ECG abnormalities however, only partly resolved.
A moderate hypokalemia between 2.5-3.0 mmol/l persisted for two weeks despite supplementation. Fever episodes also persisted, although the body temperature remained below 39.5 °C. The slight ST-segment abnormalities remained unaltered. After 8 weeks, the patient died of his hematological diseases.

Discussion

We described a 72-year old Caucasian male known with acute myeloid leukemia and non-Hodgkin lymphoma who presented with fever episodes, hypokalemia and a Brugada ECG pattern. The Brugada syndrome (BS) is a channelopathy of the sodium channels characterized by typical ST-segment elevations in the precordial leads; it is estimated to be responsible for at least 4% of all sudden deaths.[1] So far, only the sodium channel SCN5A gene is associated with BS, while this is found in only 18-30 of the Brugada patients.[1] It is most likely there are still other yet unknown mutations in the cardiac sodium channels responsible for BS, explaining why also some SCN5A-negative patients have Brugada-like ECG alterations. It is unknown if there is a risk difference between BS with or without SCN5A mutation. Junttila et al. demonstrated that a Brugada ECG pattern caused by fever, electrolyte imbalances or drug overdose should be considered a risk factor for developing ventricular tachyarrhythmia for both SCN5A-positive and -negative patients.[2] It is therefore essential to identify patients at risk and treat such provoking factors as soon as possible.

Hypokalemia is associated with Brugada ECG patterns and ventricular arrhythmia’s.[3,4] However, the pathogenesis is not yet clear. Araki et al. described a patient presenting with hypokalemia (2.9 mmol/l), covered ST-segment elevation (Brugada type 1) and ventricular tachyarrhythmia’s. After potassium supplementation the ST-segment changed to saddle-back configuration (Brugada type 2) and the ventricular tachyarrhythmia’s disappeared.[3] In a canine model, it was demonstrated that loss of the action potential dome due to tachyarrhythmia’s disappeared.[3] In both cases, the persistence of fever was of similar importance. In conclusion, we described a 72-year old male with a Brugada ECG pattern due to a combination of both elevated body temperature and hypokalemia.

References