EMERGENCY CASEBOOK

Brugada-like electrocardiographic changes induced by fever

M Unlu, F Bengi, B Amasyali, S Kose

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As "time is myocardium" in the settings of acute myocardial infarction, it is important to make the diagnosis as quickly as possible, and a high clinical suspicion is needed to avoid missing the diagnosis, resulting in unwarranted interventions. The electrocardiogram is a crucial tool in the identification of acute chest pain, enabling a detailed analysis of patterns of ST-segment elevation. We describe the case of a 22-year-old man who presented with fever, with dynamic electrocardiographic changes similar to the Brugada syndrome. These electrocardiographic anomalies disappeared when the temperature returned to normal.

The 22-year-old man was admitted to the emergency department with fever, shivering, arthralgia and chest pain that increased with deep breathing, and was referred to cardiology for his electrocardiographic (ECG) changes for suspected acute coronary syndrome. His medical history was unremarkable, and he was not taking any prescribed drugs or cocaine. He had no family history of sudden death, nor had he experienced any episodes of syncope. On physical examination, there was an inflammation of the pharynx. His temperature

was 38.5°C, the blood pressure was 100/60 mm Hg and the pulse was 95/min. Auscultation showed no cardiac murmurs, friction rub or rales. The remainder of the physical examination was normal. The ECG showed incomplete right bundle branch block with ST-segment elevation in leads V1–2, consistent with saddleback-type ECG (fig 1A). Initial laboratory data disclosed a negative troponin T test, an abnormal white cell count of 12 500/mm³, a raised C reactive protein concentration of 14.2 mg/dl and an erythrocyte sedimentation rate of 30 mm/h. A radiograph of the chest showed normal heart size and no consolidation in the lungs. A transthoracic echocardiography showed normal wall motion throughout, with no evidence of pericardial effusion. Several hours after his admission, repeated ECG showed only minimal ST-segment elevation in V2 (fig 1B). The next day, the patient was afebrile and his chest pain resolved with oral ibuprofen gradually. The ECG was completely normal (fig 1C). An electrophysiological study was not performed because neither the patient nor any of his family members had experienced any arrhythmic symptom or sudden cardiac death, and medical follow-up was decided. The patient was

Abbreviation: ECG, electrocardiogram

Figure 1 Twelve-lead electrocardiogram (ECG) of the patient shows incomplete right bundle branch block with (A) and without (B) saddleback-type ST-segment elevation in leads V2, and a completely normal ECG (C).

recommended to come to hospital for urgent treatment of fever when it developed.

DISCUSSION

It is well known that ST-segment elevation can be seen in many conditions. As “time is myocardium” in the settings of acute myocardial infarction, it is important to make the diagnosis as quickly as possible, and a high clinical suspicion is needed to avoid missing the diagnosis, resulting in unwarranted thrombolytic treatment or emergency angiography.¹ In 1992, Brugada and Brugada² described eight patients with a history of cardiac arrest, and ECG findings of right bundle branch block and ST-segment elevation in the right precordial leads in the absence of long QT intervals and any structural heart disease. The ECG in this syndrome may be dynamic over time and may include transient normalisation. Despite all the knowledge on this syndrome acquired over the past decade, the pathophysiology of this syndrome is still under investigation. Insight from cellular electrophysiology suggests that the ST-segment elevation is caused by a shift in the ionic current balance and the creation of a voltage gradient, with predominance of transient outward current in the epicardium over the endocardium.³ The ionic mechanism responsible for the dynamic ECG changes have been shown to be temperature dependent.^{3,4} In the present case, the ECG changes were evident when the fever was present and disappeared once the temperature returned to normal. The ECG appearance was similar to that seen in Brugada syndrome; however, as the patient had no family and personal history for arrhythmia and the drug challenge was negative, the term “Brugada-like” was used. It is possible that an increase in temperature may be the most important factor in

the appearance of these ECG changes. This report emphasises that the emergency doctors should be aware of these transient ECG changes related to fever and should treat fever aggressively.

Authors' affiliations

M Unlu, Department of Cardiology, Diyarbakir Military Hospital, Diyarbakir, Turkey

F Bengi, Department of Emergency Medicine, Diyarbakir Military Hospital, Diyarbakir, Turkey

B Amasyali, S Kose, Department of Cardiology, Gulhane GATA Military Medical School, Ankara, Turkey

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Correspondence to: Dr M Unlu, Diyarbakir Asker Hastanesi, Kardiyoloji Klinigi, 21100 Diyarbakir, Turkey; drmuratunlu@gmail.com

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