

Brugada-like electrocardiographic pattern unmasked by fever in a critical care patient

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Sir,

Brugada syndrome is a channelopathy associated with right bundle branch block and ST segment elevation in the right precordial leads, V1–V3, in the absence of structural heart abnormalities (1). Mutations in a cardiac sodium channel gene (SCN5A) have been reported as the substrate for the syndrome in some patients (2). Past studies suggested that fever could aggravate the dysfunction of the mutant channel, thus raising the possibility that patients with Brugada syndrome may display a more abnormal electrocardiographic pattern or even be at a higher risk of arrhythmias during a febrile state (3).

A 46-year-old previously healthy Caucasian male who was admitted to the intensive care unit because of respiratory failure and sepsis is presented. Chest X-rays revealed normal heart size and lung consolidation. Bronchoalveolar lavage-derived cultures grew *Klebsiella pneumoniae*. The electrocardiogram (ECG) on admission, with the patient being febrile (39.5 °C), showed sinus tachycardia (heart rate: 130 beats/min) and a typical Brugada pattern with right bundle branch block morphology and prominent ST-segment elevation in the right precordial leads (Fig. 1). Transesophageal echocardiography revealed no underlying structural heart disease. Creatinine kinase was mildly elevated with myocardial bound isoenzyme and troponin I levels been normal, whereas serum electrolytes were normal. Two days after admission, during another febrile episode (40 °C), the ECG again

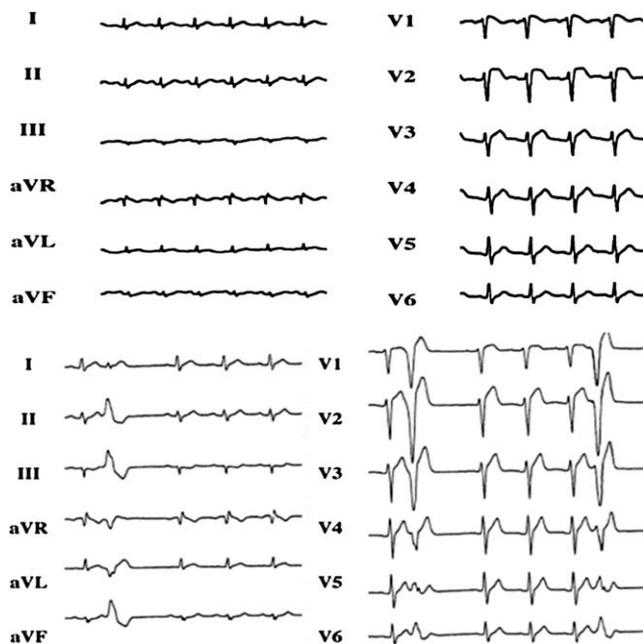


Fig. 1. Twelve-lead electrocardiogram on admission at a body temperature of 39.5 °C; observe the coved-type ST-segment elevation in the right pre-cordial leads (top). Twelve-lead electrocardiogram at a body temperature of 40 °C demonstrating a Brugada-like pattern; observe the ST-segment elevation in the right pre-cordial leads and premature ventricular contractions of left bundle branch block pattern with the inferior axis (bottom).

revealed a Brugada-like pattern (Fig. 1). Complete cardiac workup was negative. There was no family history of sudden death, and the patient had never experienced syncope. The patient's clinical condition gradually improved and the ECG normalized, hence he was finally discharged home. He underwent routine follow-up by a cardiologist. Six months later the patient provided his consent and underwent coronary angiography which was normal.

Since its introduction as a clinical entity in 1992, Brugada syndrome has progressed from being a rare disease to one that is second only to automobile accidents as a cause of sudden death among young adults in some countries. The electrocardiographic manifestations of Brugada syndrome are often dynamic or concealed and may be unmasked or modulated by sodium channel blockers, a febrile state, vagotonic agents, alpha-adrenergic ago-

nists, tricyclic or tetracyclic antidepressants, a combination of glucose and insulin, hypo- and hyperkalemia, hypercalcemia, and alcohol and cocaine toxicity (4). Treatment options for symptomatic patients with Brugada syndrome are limited. The Second Consensus Conference on Brugada syndrome recommended that such patients receive an implantable cardioverter-defibrillator (4).

Misdiagnosing Brugada syndrome as an acute myocardial infarction (MI) is common as the ECG is rather similar. However, MI is a clinical diagnosis with the constellation of chest pain, diffuse ECG findings, and laboratory abnormalities, and these were not seen with this patient. Concluding, the clinical intensivist must be familiar with Brugada syndrome and its management. The effect of fever upon Brugada-like electrocardiographic pattern requires further clarification.

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