Unmasking of Brugada syndrome during a febrile episode

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A 58-year-old Sri Lankan man presented to the hospital with a sore throat, cough and fever of 38.5°C. ST segment elevation was present in leads V1 to V3 of his initial electrocardiogram (ECG) (Figure 1A). Serial cardiac enzymes, chest x-ray, urgent two-dimensional echocardiography and coronary arteriography were normal. The patient's symptoms were attributed to a viral illness. The ST segment elevation resolved with abatement of the fever (Figure 1B). The patient subsequently underwent an electrophysiology study with procainamide (10 mg/kg) drug challenge, which replicated the pattern of ST segment elevation with fever (Figure 1C).

The ECG findings in the present case are consistent with Brugada syndrome, a genetically determined arrhythmogenic disease caused by mutations of the cardiac sodium channel gene

SCN5A. The intermittent nature of ST segment elevation associated with this condition may result in misinterpretation as an acute ST segment elevation myocardial infarction. The pathogenesis of the ECG changes associated with Brugada syndrome is not fully understood but is thought to be due to an alteration in the action potential of the epicardial (but not endocardial) cells. Elevated temperatures may result in a faster rate of sodium channel inactivation and slower recovery from inactivation with subsequent ST segment elevation on the resting ECG. A theoretical increased risk of ventricular arrhythmias exists with fever.

Following the present evaluation, the patient remained free of arrhythmia and awaited evaluation for defibrillator implantation. He was counselled to use acetaminophen whenever a fever develops.

Figure 1 A Electrocardiogram at the time of admission with a fever. ST segment elevation in leads V1 to V3 is seen. B Electrocardiogram at 48 h showing resolution of the ST segment elevation with abatement of the fever. C Replication of the ST segment elevation pattern when the patient was given a procainamide (10 mg/kg) drug challenge

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