CASE REPORT

Electrocardiographic findings compatible with Brugada syndrome in a patient with febrile respiratory infection

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Abstract
Brugada syndrome is an inherited autosomal dominant-type disease characterized by ST-segment abnormalities and increased fatal ventricular tachyarrhythmias. We hereby present a 57-years-old patient with no symptoms or history of cardiovascular disease, diagnosed with febrile respiratory infection (39°C). Electrocardiographic (ECG) findings were typical of Brugada-like type I syndrome that gradually turned to Brugada type II and III, following fever remission, and finally became normal. Other clinical evaluation tests (echocardiographic evaluation, treadmill stress test, Holter ECG, procainamide provocation test) did not relate to Brugada syndrome. Hippokratia 2010; 14 (3): 221-223

Key words: Brugada syndrome, electrocardiography

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Brugada syndrome (Bs) is an inherited disease of autosomal dominant type, with varying pervasiveness, which develops in anatomically normal hearts and is characterized by ST-segment elevation on the right precordial leads (V_1-V_3), a right bundle branch block (RBBB) morphology and an increased incidence of fatal ventricular tachyarrhythmias[1]. We present a case in which appearance of electrocardiographic findings consistent with Bs was associated with high fever. Similar cases have been reported in the recent literature and are related with the thermosensitivity of sodium channels in patients with Bs[2-4].

Description of Case

A 57-years-old man with no history of cardiovascular disease reported to the Emergency Department of "A. Fleming" General Hospital due to pain in the left hemithorax with pleuritic features and a fluctuating 39°C fever over the previous 24 hours. Upon clinical examination, his heart rate was around 100 beats/min and his blood pressure 130/80 mmHg. Pulmonary examination gave findings compatible with left basilar thickening (bluntness, increase in voice tremors and snoring sounds). No other pathological clinical findings were detected. The chest X-ray revealed signs of thickening in the left inferior lobe. Sinus rhythm with RBBB morphology and coved or saddle-shaped ST-segment elevation > 2 mm at the J point in leads V_1-V_3 was recorded on the ECG (Figure 1).

Polymorphonuclear leukocytosis (WBC 22,590/μL; NEUT 90%) was diagnosed. Erythrocyte sedimentation rate (ESR) and CRP levels were elevated (112mm/hr and 54mg/L, respectively). Cardiac enzymes and other biochemical markers were normal.

In order to elucidate the rhythm abnormalities, the patient was admitted to the cardiology department for ECG and cardiac enzymes monitoring and intravenous administration of antimicrobial medication (keftriaxone)

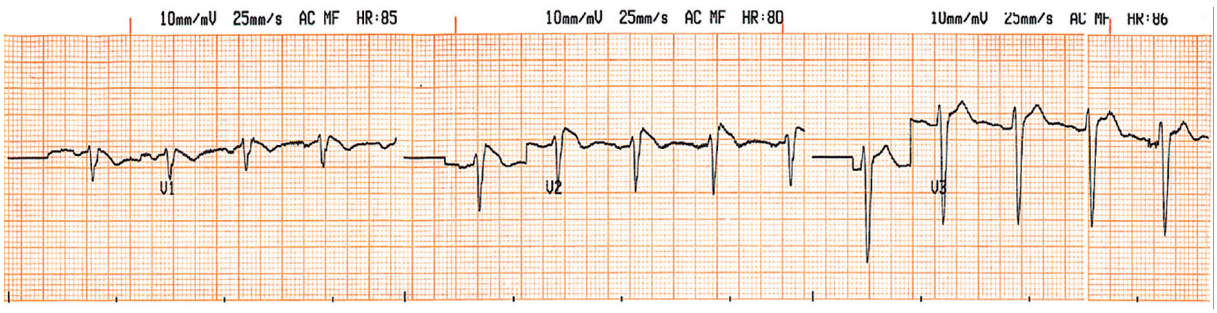

Figure 1: ECG type 1 changes diagnostic of Brugada syndrome occurring during high fever in the patient of the present study.

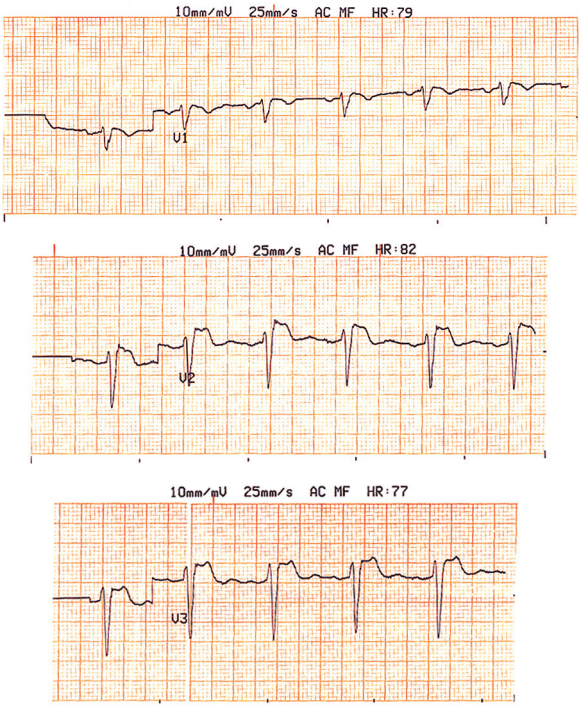

Figure 2: ECG (saddle-like) type 2 changes typical of Brugada syndrome occurring upon initiation of fever remission in the patient of the present study.

was initiated. Two days later the patient started gradually becoming afebrile and no other complications appeared. His cardiac enzymes remained within normal levels. The ECG showed a gradual reduction in the ST-elevation, consistent with fever reduction (Figures 2, 3), which finally returned to normal limits 3 days after admission. The echocardiographic examination was normal and 24h ECG monitoring showed no arrhythmias. A new chest X-ray examination after 5 days verified almost complete elimination of the thickening.

The patient, before discharged from hospital, underwent the procainamide provocation test (intravenous administration of 1g procainamide). No ECG findings of Bs were detected. He was then discharged with instructions to complete the course of medication. A stress test was scheduled for 10 days later and was negative for ischemia or arrhythmias.

Discussion

On a typical ECG of the Bs there is coved (type 1) or saddle-shaped (types 2 & 3, not diagnostic of the syndrome) ST-segment elevation > 2 mm in the right precordial leads (V_1-V_3)[5]. Intravenous administration of class Ia or Ic antiarrhythmic medication may reveal these ECG findings[6]. The specificity and sensitivity of the provocation test for Bs diagnosis differ depending on the sodium channel blocker administered. Procainamide (Ia) compared to ajmaline or flecainide (Ic) produces a less pronounced ST-segment elevation[7]. Even when using flecainide, the sensitivity and specificity of the test, calculated among SCN5A-positive probands and their family members, were 77% and 80%, and its positive and negative predictive values 96% and 36%, respectively[8]. Therefore, a negative Na blocking test does not exclude Bs[9]. The unmasking of the syndrome with high fever has also been demonstrated[10,11], as well as the thermosensitivity of sodium channels in vitro, leading to the predominance of the I_{to} current.

Diagnosis is based on the Brugada ECG changes described (with or without the use of sodium channel blockers) in combination with either confirmed episodes of ventricular fibrillation or tachycardia, positive electrophysiological test for induced ventricular tachycardia, syncope, family history of sudden cardiac death (age <45 years[5]). The risk of sudden cardiac death within 3 years of diagnosis is 30%. There is no effective drug treatment. Implantable cardioverter/defibrillator (ICD) is recommended to prevent sudden cardiac death in symptomatic and type 1 asymptomatic patients and when fatal arrhythmias are induced during an electrophysiological study.

In the case described here a patient, with no symptoms nor any family history, exhibited type 1 ECG findings during fever, which dropped to types 2 and 3 when he became afebrile. Electrophysiological study was rec-

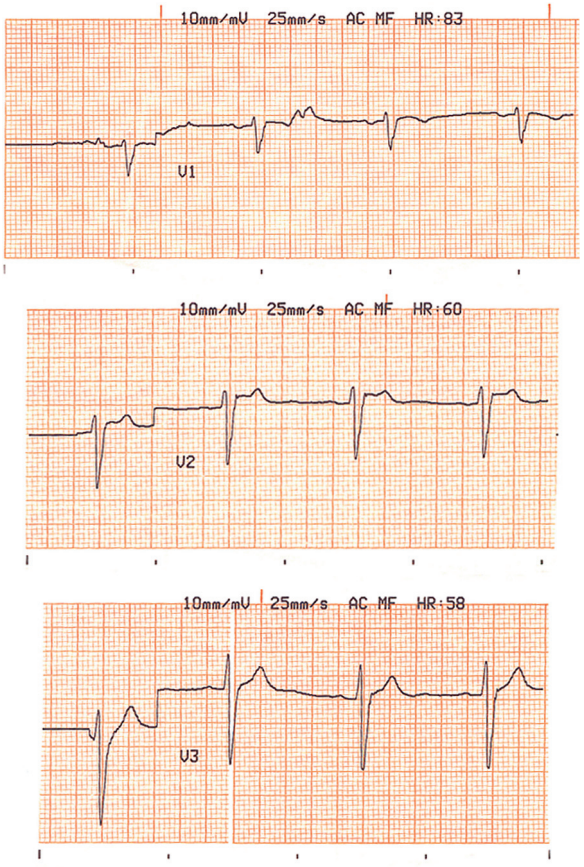

Figure 3: ECG type 3 changes typical of Brugada syndrome occurring during continuation of fever remission in the patient of the present study.

ommended despite the negative procainamide test[5], to which the patient did not consent. Therefore, a regular cardiological follow up was suggested. We support the belief that in the absence of full Bs manifestations and negative sodium channel blocker provocation test, type 1 ECG findings during fever may unmask the syndrome.

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