Transient left ventricular dysfunction during fever-induced Brugada-like electrocardiographic pattern

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After a fall at home that was preceded by lightheadedness, a previously healthy 55-year-old male was admitted to neurosurgery department with a minor head injury. There were no neurological deficits, and head multislice computed tomography revealed no structural abnormalities. On a fourth hospital day, the patient developed a fever (39.1°C) associated with chest pain and dyspnoea. There was a mild leucocytosis, while serial blood and urine cultures were negative. The 12-lead electrocardiogram (ECG) showed the right bundle branch block (RBBB) and ST-segment elevation (STE) in the right precordial leads (a coved-type in V1 and saddleback shaped in V2) consistent with Brugada ECG pattern (Figure A, top panel). The standard echocardiographic examination recorded at hospital admission showed left ventricular ejection fraction (LVEF) of 45% due to hypokinesia of the distal anterior and posterolateral left ventricular (LV) walls (Supplementary material online, Video S1, quad view). Speckle tracking echocardiography showed decreased values of longitudinal peak systolic strain (LPSS) of all LV segments (different shades of red in the bottom panel in Figure A) with global strain of −12.7% (lower limit of normal range −18%). Cardiac troponin I was mildly elevated (1.46 ng/mL; normal range <0.5 ng/mL) while coronary angiography revealed normal coronary arteries. The patient was treated only with antipyretics and 6 days later, the fever waned in parallel with both ECG and echocardiographic changes. There were neither RBBB nor STE noted on discharge ECG (Figure B, top panel).

A follow-up echocardiographic examination demonstrating a complete recovery of LV systolic function (LVEF 65%) is shown in Supplementary material online, Video S2. A complete recovery of LPSS with global strain of −21.8% was also detected by strain echocardiography (a uniform dark shade of the red in the bottom panel in Figure B).

Given a high clinical index of suspicion of an arrhythmic event leading to the head injury, the patient underwent an early post-discharge ajmaline challenge. The test was positive and an implantable cardioverter-defibrillator was inserted.

The Brugada ECG pattern is often dynamic and can be induced by fever due to temperature dependence of ionic mechanisms underlying the Brugada syndrome.1,2 Our report suggests that fever-induced Brugada ECG pattern may also be associated with transient LV systolic dysfunction. Of note, the existence of LV asynergy and ECG changes was strictly associated with persistence of fever itself; however, although less likely, it might also be attributed to direct myocardial injury by possible viral infection. However, a mild cardiac troponin elevation has been previously observed in some cases with fever-induced Brugada-like ECG pattern but normal LV function, suggesting that LV dysfunction in these patients may be subclinical. Further, it has been recently reported that a myocardial performance index of both ventricles may worsen after administration of a sodium channel blocker in patients with Brugada-type ECG pattern,
although visually apparent ventricular dysfunction was not observed. Speckle tracking echocardiography has shown a potential for detecting a subtle changes in LV function in different clinical settings and, as illustrated in this case report, might be valuable for displaying the course of myocardial impairment in patients with fever-induced Brugada-type ECG pattern.

Supplementary material is available at *Europace* online.

**References**

