BRUGADA SYNDROME AND FEVER
CASE REPORT-1

Incessant monomorphic ventricular tachycardia during febrile illness in a patient with Brugada syndrome: fatal electrical storm

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A 55-year-old male with structurally normal heart presented with sustained monomorphic ventricular tachycardia (VT) and was cardioverted into sinus rhythm revealing a right bundle branch block pattern at baseline electrocardiography. Sustained monomorphic and nonsustained polymorphic VT were reproducibly inducible during electrophysiological study. During the diagnostic workup, the patient experienced fever due to hospital based pneumonia, which unmasked typical ST segment changes of Brugada syndrome. In the intensive care unit, fever became intractable leading to incessant monomorphic VT, which was resistant to all medical manoeuvres resulting in the patient’s death. (Europace 2003; 5: 257–261)

Key Words: Brugada syndrome, fever, monomorphic VT, fatal electrical storm.

Introduction

Brugada and Brugada[1] reported a group of patients with recurrent polymorphic ventricular tachycardia (VT) leading to cardiac arrest who had an electrocardiographically (ECG) specific pattern of right bundle branch block (RBBB) and ST segment elevation in leads V₁–V₃ with normal QT interval and no structural heart disease. On the other hand monomorphic sustained VT, which was classically not expected, was also reported but in only a few cases[2–6].

Although the manifestation of Brugada pattern in the ECG during fever has been reported in a few cases[7,8], fatal electrical storm induced by fever has not been published.

We report a case of Brugada syndrome, where fever developed and progressed to fatal monomorphic VT.

Case report

A 55-year-old male with no known cardiac disease was admitted to hospital with monomorphic VT (Fig. 1). Following electrical cardioversion, ECG revealed sinus rhythm with RBBB, left axis deviation, normal QT interval and minimal ST elevation in leads V₁–V₃ (Fig. 2). Physical examination, blood tests including myocardial enzymes and viral antibodies, and chest X-ray were normal. Echocardiography, coronary angiography with the ergonovine test, right and left ventriculography and magnetic resonance imaging of the heart were all within normal limits.

An electrophysiological study, off drugs, revealed normal baseline intracardiac intervals, sinus and atrioventricular node function. Sustained clinical monomorphic VT with RBBB (Fig. 3) and nonsustained polymorphic VT were induced by programmed ventricular stimulation. The patient later developed fever up to
Figure 1  Twelve lead surface ECG of the clinical monomorphic VT.

Figure 2  Baseline ECG.

Europace, Vol. 5, July 2003
39°C due to hospital-acquired pneumonia. All types of ST segment elevation typical of Brugada syndrome such as ‘saddle back’, ‘coved’ and ‘huge’ in precordial leads were demonstrated during fever (Fig. 4). Brugada syndrome was diagnosed based on the clinical and ECG findings. The patient was cardioverted or defibrillated from the same clinical VT several times during the treatment of underlying infection. ECG changes were reproducibly observed during fever exacerbations. Intravenous administration of lidocaine, metoprolol and MgSO4 were unsuccessful in terminating VT storms. Overdrive pacing and dobutamine infusion also failed to stop the arrhythmia. Although amiodarone did not terminate VT attacks, it slightly diminished the number of shocks needed. Nevertheless, the patient required intubation under general anaesthesia because he received 600 external shocks over 4 days. Because of the incessant nature of VT, the patient developed acute multiorgan failure and pulmonary oedema and eventually died.

**Discussion**

Brugada syndrome with its characteristic ECG was originally reported in a group of patients with recurrent polymorphic VT leading to cardiac arrest[3]. Monomorphic VT however, associated with Brugada syndrome was reported in only five patients, exhibiting an RBBB pattern in three patients[3,6] and an LBBB pattern in two[2,4,5]. In our case, programmed ventricular stimulation induced monomorphic VT with RBBB pattern as well as spontaneous attacks all required DC cardioversion. Polymorphic VT accompanying monomorphic VT was reported in two patients similar to our case[5,6].

It is a characteristic that typical ECG changes in Brugada syndrome vary over in time. The administration of class Ia, lc, III drugs as well as fever increases the ST segment elevation[7]. Since increased body temperature unmasked ECG changes of Brugada syndrome in this case, there was no need to use antiarrhythmics to unmask typical ECG changes. Change in function of the mutant sodium channel is observed at physiological temperatures, but not at room temperature[7]. It is interesting that this characteristic of the mutant channel is exaggerated at temperatures above the physiological range, pointing to the possibility that patients with Brugada syndrome may be at more risk during a febrile state[7]. Several Brugada patients displaying fever-induced polymorphic VT have been identified[8,9].

During the hospital stay, simultaneously with the patient’s body temperature rising, classic, intermittent ST segment elevation of Brugada syndrome developed and the patient experienced a fatal VT storm resistant to all medical manoeuvres including pacing. Incessant
Figure 4  All types of ST segment elevation during fever.
ventricular arrhythmias have been reported in Brugada syndrome. The exact mechanisms in triggering and termination of electrical storms remain unclear. Brugada et al. and other investigators stressed the functional electrical change and indicated the importance of increased adrenergic tone. This hypothesis is supported by the observation that sustained VT is induced during isoprenaline infusion and terminated by propranolol. However, it has been reported that an increase in parasympathetic tone causes abnormality in ventricular conduction. In contrast, beta-blockers had no beneficial effect, rather increased the arrhythmic recurrence in our case. In the series reported by Brugada et al., 20% of the patients were chronically treated with beta-blocking agents and no arrhythmic exacerbation was noted. Kasanuki et al. observed deleterious effects of beta-blockers in two patients and suggested that sympathomimetic drugs might be effective in the treatment of ventricular fibrillation attacks. In our patient, dobutamine infusion did not show any beneficial effect.

It has been reported that in patients with right ventricular outflow tract tachycardia and an apparently normal heart, endomyocardial biopsy may be indicative of myocarditis in a significant percentage of cases. Therefore, one could argue that viral myocarditis may be the cause in our case. We cannot definitely exclude such a possibility in our patient, since endomyocardial biopsy was not performed. However, we feel that this is unlikely because: (1) there was no history of a systemic viral illness preceding the onset of palpitations; and (2) normal myocardial enzymes and antibody titres against common viruses did not support the diagnosis of recent myocarditis.

In summary, fever may unmask ECG changes of Brugada syndrome and be responsible for an electrical storm. This can be the first manifestation of the disease and the evolution may be marked by an incessant ventricular malignant arrhythmia resistant to DC cardioversion. According to our knowledge, such an electrical storm of incessant VT, which is resistant to shocks with eventual fatal outcome, may be unique and has not previously been described.

**References**