Brugada syndrome is a clinical and electrocardiographic entity characterized by ST segment elevation in the right precordial ECG leads and sudden death or syncope secondary to malignant ventricular arrhythmia, and has a high recurrence rate. We report a patient with this syndrome who had received an automatic implantable defibrillator, who presented with multiple appropriate discharges because of recurrent episodes of ventricular fibrillation. All episodes were started by a premature ventricular beat of the same morphology and coupling interval. Endovenous procainamide administration, paradoxically, was effective in preventing new episodes. The beneficial antiarrhythmic effect of procainamide in this patient is discussed.

**Key words:** Brugada syndrome. Ventricular fibrillation. Drugs.

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**INTRODUCTION**

Brugada syndrome is a clinical and electrocardiographic entity characterized by a typical ECG pattern and syncope or sudden death. The condition involves malignant ventricular arrhythmia, polymorphic ventricular tachycardia and ventricular fibrillation and has a high recurrence rate. The episodes usually present alone, but occasionally appear as multiple episodes of arrhythmia. At present there is little information on the origin and treatment of this condition, although several potential triggering factors have been suggested recently. We describe a patient with this syndrome who had received an implantable cardioverter defibrillator (ICD) and presented arrhythmia with multiple appropriate discharges secondary to episodes of recurrent ventricular fibrillation.

**CASE STUDY**

A 54-year-old man was seen in April 1996 after aborted sudden death secondary to ventricular fibrillation. The patient had no personal or family history of interest and the physical examination was normal. The electrocardiogram revealed the characteristic pattern of Brugada syndrome (Figure 1). The echocardiogram and cardiac catheterization studies were normal. Electrophysiological study showed an HV interval of 60 ms, and ventricular fibrillation was induced by a drive train followed by two ventricular extrastimuli,..

with coupling of 600/250-180 ms from the apex of the right ventricle. The administration of 20 mg of ajmaline produced marked elevation of the J point and ST segment, which normalized with isoproterenol administration (Figure 1). A single-chamber implantable cardioverter defibrillator was implanted.

On two occasions (August 1998 and November 2001), the patient presented at the emergency room
with sudden, recurrent syncopal episodes occurring at rest with no apparent triggering factor, followed by discharge of the implantable cardioverter defibrillator. Both times, the patient had additional episodes shortly after admission and ECG monitoring indicated ventricular fibrillation. Also on both occasions, the patient was given 1 g of intravenous procainamide in 30 min, followed by maintenance infusion of 20 mg/min, with no further arrhythmias. On the first occasion quinidine was prescribed at a dose of 560 mg/day, which the patient decided to discontinue.

In both incidents, interrogation of the implantable cardioverter defibrillator showed six and seven episodes, respectively, of ventricular fibrillation which were terminated by discharge. All episodes started with a premature ventricular beat of the same morphology and similar coupling as the preceding sinus beat (Figure 2). After his second hospital stay, the patient has continued quinidine therapy at the prescribed dosage with no new recurrences of ventricular fibrillation after one year of follow-up.

REFERENCES