Case Report

Asymptomatic Brugada Patients: to Treat or Not

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A 53 years old man was assisted to emergency department due to palpitations and presyncope. A ventricular tachycardia (VT) was documented and converted with intravenous lidocaine. He had history of sudden death in his two brothers. Type 1 electrocardiographic pattern of the Brugada syndrome was diagnosed (figure 1). Laboratory analysis, coronary angiography and echocardiogram were normal. Programmed electrical stimulation (PES) was induced the same VT (figure 2). A cardiodefibrillator (CD) was implanted and he suffered 8 episodes of ventricular flutter converted with the device in the 48 months follow up (figure 3). His asymptomatic 27 years old son was studied; he had basal type 1 electrocardiogram (figure 4). Electrophysiologic study was performed. HV was measured (63 msec) and sustained ventricular tachyarrhythmias were not induced despite programmed ventricular stimulation with basic cycle lengths at 600, 430 and 330 msec up to three extrastimulus of 200 msec in the right ventricular apex and outflow tract. No treatment was suggested and thirty one months later he died with ventricular fibrillation. Also his asymptomatic 34 years old sister was studied. She had basal type 1 ECG and PES was negative. She refused receiving a CD and quinidine sulphate was started. In the 14 months follow up she is OK.

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Brugada syndrome (BS) was described as a clinical entity in 1992,[1] and is characterized by a coved-type ST-segment elevation in at least two leads, (V1 to V3) of the ECG and a high incidence of sudden cardiac death or syncope secondary to lifethreatening ventricular arrhythmias in structurally normal hearts.[2] The treatment of the asymptomatic patients is controversial.[3-6] Brugada et al. recommended implanting a CD in asymptomatic patients if were induced at PES.[4,5] Others report that PES is not helpful in identifying individuals at higher risk.[3,6] A recent report of meta-analysis showed that history of SCD or syncope, male gender and spontaneous type1 ECG are predictors of ventricular arrhythmic events, but family history of SCD, the presence of an SCN5A gene mutation, or PES are not.[7] Also Paul et al. have questioned the role of PES for risk stratification.[8]

Primary prevention in Brugada patients with CD is accompanied by high rates of complications.[9,10] Infections and inappropriate CD shocks (due to sinus tachycardia, atrial arrhythmias, T wave over-sensing and lead fractures) are frequent in young patients. There are pharmacological options to use. Belhassen was the first to report the effectivity of

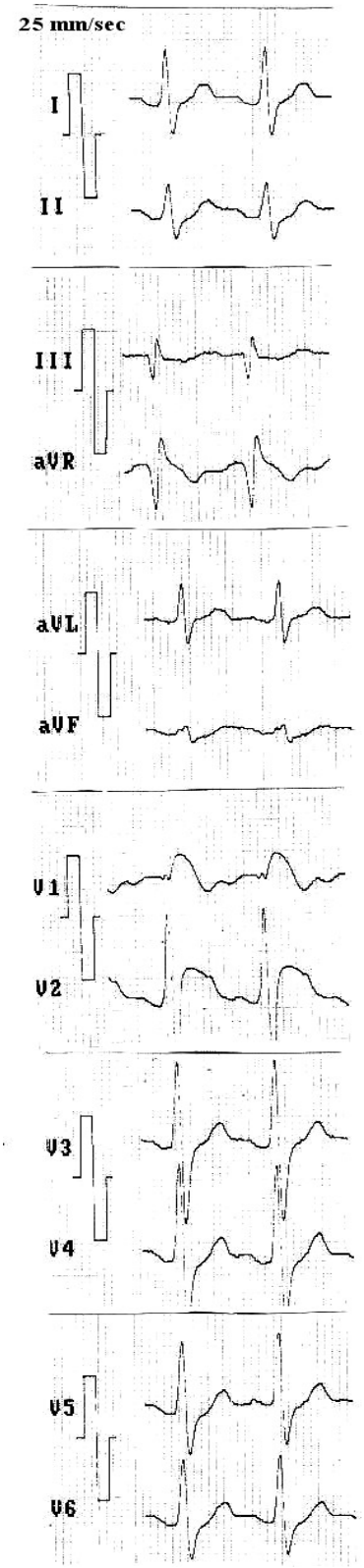

Figure 1: Basal ECG of the 53 years old man. Type 1 electrocardiographic pattern in V1 and V2 leads.

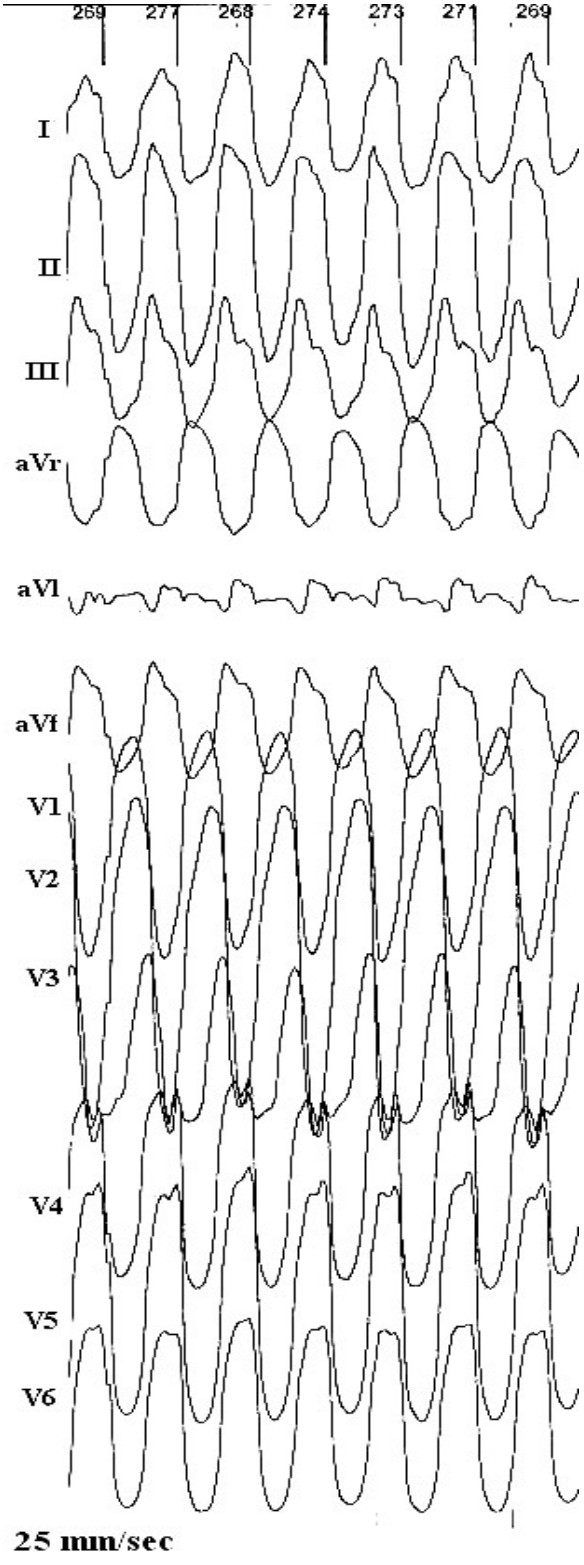

Figure 2: Very fast ventricular tachycardia induced with programmed electrical stimulation.

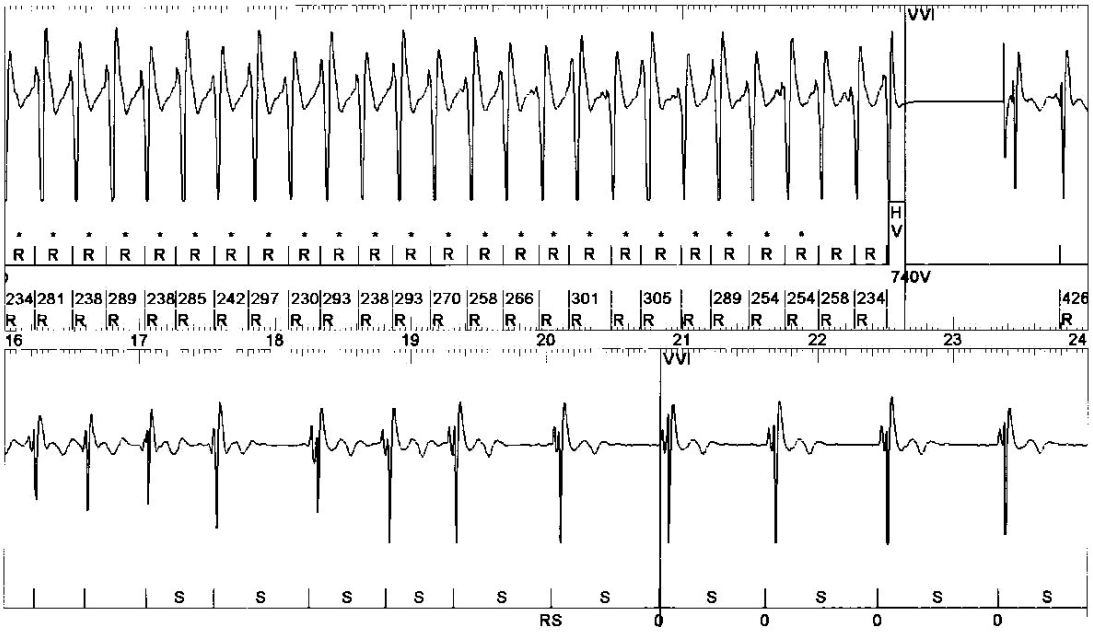

Figure 3: Stored electrogram of the cardiodesfibrillator. Ventricular flutter reverted by the device.

Quinidine to prevent VF induction and spontaneous lifethreatening ventricular arrhythmias in BS patients.[11-13] Others reported the use of this drug to abort electric storm.[14,15]

Should we recommend treatment in some asymptomatic Brugada patients? Our 27 years old patient could be protected with the CD despite of negative PES, due to malignant outcome of his family; his father and two uncles suffered lifethreatening ventricular arrhythmias. What should we do with female relatives? The women have lesser risk, but how much?

Future studies should be done to improve our knowledge about these interesting issues but our suggestion is to individualize the treatment. We must analyse all possible risk factors as gender, family history of sudden death, basal ECG and inducibility at PES in order to achieve the best approach to the real risk.

Conflicts of Interest no declare.

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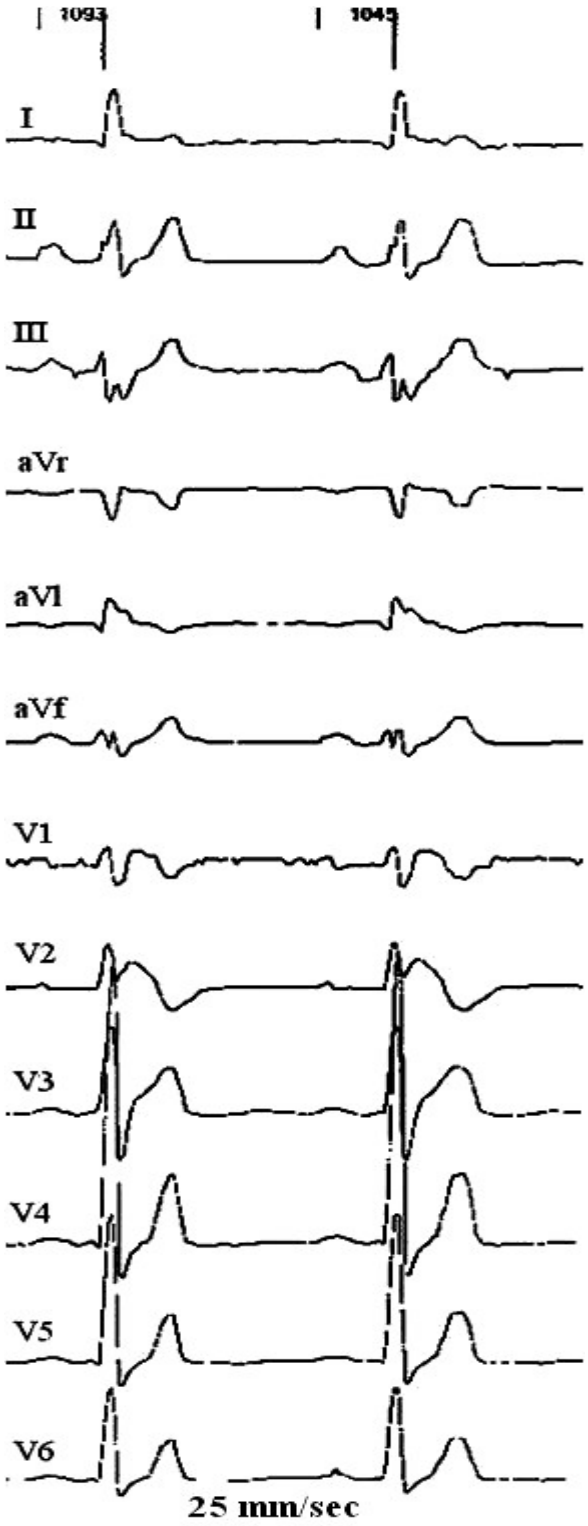

Figure 4: Basal ECG of the 27 years old son.

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