

Successful radiofrequency ablation of fibrillation-inducing ventricular foci in a patient with Brugada syndrome

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Background:

Brugada syndrome is a cardiac conduction disorder characterized by mutations in the SCN5A gene in chromosome 3 encoding for the alpha sub-unit of cardiac sodium channel. This condition is estimated to account for 20% of deaths in patients with an apparently normal heart. In this case, we describe a patient with Brugada syndrome who had a successful radiofrequency ablation of fibrillation-inducing ventricular foci.

Case:

The patient is a 26 year-old male who had a witnessed ventricular fibrillation requiring multiple defibrillations and cardiopulmonary resuscitation. While in the cardiac care unit, he had several episodes of non-sustained polymorphic ventricular tachycardia. After initial recovery, the patient suffered an electrical storm with episodes of ventricular tachycardia deteriorating to ventricular fibrillation. It was observed that ventricular fibrillation was precipitated by premature ventricular depolarizations. Electrocardiogram at this time showed ST-segment elevation in the right precordial leads with inverted T waves characteristic of the Brugada pattern. Electrophysiological study revealed a procainamide-inducible foci on the left ventricular posterior region on the left posterior fascicular system. This foci was successfully ablated using radiofrequency energy. Procainamide re-challenge of up to 1250mg did not induce any more ventricular arrhythmias. He was subsequently implanted with a cardiac defibrillator.

Discussion

The electrophysiologic mechanism for triggering malignant arrhythmias in Brugada syndrome is postulated to be secondary to the heterogenous loss of the epicardial action potential dome resulting in transmural and epicardial dispersion of repolarization. In the majority of the patients, initiation of ventricular fibrillation was preceded by isolated premature ventricular depolarizations

Radiofrequency ablation has been successfully performed in patients with idiopathic ventricular fibrillation, using energy with a target temperature of 55-60 degrees and a maximum power of 50 watts. In a subsequent study, three patients with Brugada syndrome underwent radiofrequency ablation of fibrillation-inducing ventricular foci. The premature depolarizations have a right ventricular outflow tract morphology in two patients and a left bundle-branch block-superior axis in one patient. In a mean follow-up of up to 13 months, all three patients had no recurrence of ventricular arrhythmias, syncope or sudden death. In the patient described in this report, premature depolarizations were of the right bundle-branch block-superior axis in morphology. Nine months after radiofrequency ablation, this patient had no recurrence of ventricular arrhythmias or syncope.

Conclusion:

While the efficacy of this procedure must be confirmed with more patients and with longer period of observations, radiofrequency ablation represents a potentially curative treatment for patients with Brugada syndrome.

Category: Clinical vignette

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