Idiopathic Left Ventricular Tachycardia: What Is New in Pathophysiology? When and How to Perform RF Ablation?

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In 1979, Zipes et al. [1] reported about a peculiar category of ventricular tachycardia characterized by a QRS 0.12 to 0.14 s long, a right bundle branch block configuration and a left deviation of the electrical axis. Further reports have confirmed that this arrhythmia occurs in individuals who are otherwise healthy (idiopathic, I) and originates from the postero-inferior left ventricular septum (left ventricular tachycardia, LVT), as postulated by Zipes et al. [1]; in addition, the modality of onset during programmed electrical stimulation as well as the response to pharmacologic agents have suggested that the mechanism that is most probably responsible for ILVT is reentry [2].

Treatment is indicated if the patient is symptomatic because of the arrhythmia and especially when the arrhythmia has resulted in a tachycardiomyopathy. In asymptomatic patients, treatment with antiarrhythmic therapy is questionable. In fact, although the prognosis in these patients is essentially regarded as a benign one, sudden death has been occasionally reported [3]; in addition, one may not exclude that in a minority of patients who die from a primary ventricular arrhythmia, the initial event consists of an idiopathic ventricular tachycardia degenerating into ventricular fibrillation.

In 1987, Fontaine et al. [4] reported cure of idiopathic VT by means of high-energy direct-current (DC) application. Recently, the advent of radiofrequency current (RFC) catheter ablation has proven to be highly efficacious and safe for the curative treatment of these arrhythmias [5] and is presently being used as the first-line invasive strategy in most centers.

In the present paper, we report our experience in radiofrequency current catheter ablation in patients with ILVT.

Methods

Patients

Five patients (4 males, 1 female; age 32.8 ± 13 years) with recurrent episodes of sustained monomorphic tachycardia exhibiting on the 12-lead ECG a right bund-
le branch block morphology and left superior axis deviation were referred to our institution for curative treatment. Symptoms were palpitations in four and presyncope in one. In all patients, an average of 2 antiarrhythmic drugs (range, 1 to 3) had proven to be ineffective to control symptoms. The idiopathic nature of the underlying arrhythmia was established in all patients as defined by normal cardiac examination, baseline surface ECG, chest X-ray, echocardiogram, left and right ventriculogram and coronary angiogram.

After written informed consent had been obtained, a bicycle-exercise test and an electrophysiologic study were performed to assess the inducibility of clinical tachycardia. If it was inducible at least 3 times, left ventricular endocardial mapping and subsequent RFC ablation were performed. Recording of the earliest activation in the ventricle provided evidence for the ventricular origin of the tachycardia. The success of the ablation procedure was verified by an electrophysiologic study and/or by an additional bicycle-exercise test performed 5 days after the therapeutic procedure. Follow-up was performed on an outpatient basis every three months. An ECG at rest and during exercise and a 24-hour Holter-ECG were performed in each patient.

**Stress test**

In all patients, a bicycle-exercise testing was performed in a supine position: after a warm-up phase set at the work threshold of 50 watts for 3 minutes, stress was increased in a stepwise fashion by 25 watts every 3 minutes until maximal calculated heart rate was achieved. Criteria for discontinuation included ST-segment depression > 0.15 mV in any of the surface ECG leads, fatigue, abnormal blood pressure response, angina pectoris, dyspnea, and sustained ventricular arrhythmias.

**Electrophysiologic study**

Two 6-F quadripolar catheters (5 mm interelectrode distance) were advanced from the right femoral vein into the right ventricle and positioned at the apex and at the septal aspect of the outflow tract. After positioning of the catheters, a bolus of 100 U/kg heparin was given intravenously, followed by an additional bolus of 5000 U heparin intravenously every four hours. Five surface ECG leads (II, III, aVL, V1, V6) and 2 intracardiac electrograms were recorded at a paper speed of 100 mm/s on a Siemens Mingograf recorder (Siemens-Elema, Solna, Sweden). Programmed electrical stimulation was performed with stimuli of 0.5 ms pulse width and an amplitude of twice the diastolic threshold (UHS 20, Biotronic GmbH). All endocardial recordings were filtered at 15 to 500 Hz. Stimulation protocols included two different basic cycle lengths (510, 440 ms) with up to three extrastimuli at two right ventricular sites (apex, outflow tract) and a burst stimulation of the right ventricular apex at cycle lengths of 350-270 ms. If the clinical ventricular tachycardia could not be induced, stimulation was repeated during isoproterenol infusion. The endpoint of the protocol was either a