Case Report

Ablation of Idiopathic Ventricular Tachycardia with Left Bundle-Branch Block Morphology Located in the Pulmonary Trunk

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We report the case of a 26-year-old female patient with palpitations and presyncopes due to nonsustained ventricular tachycardia, who had no structural heart disease. The patient underwent electrophysiological study in an attempt to ablate the arrhythmogenic focus, whose location was determined by using mapping criteria. Because mapping of the right ventricular outflow tract was not successful, the catheter was placed inside the pulmonary artery with satisfactory mapping of the arrhythmogenic focus, and tachycardia was eliminated as soon as radiofrequency was initiated. The patient has remained asymptomatic for 14 months, with no treatment with antiarrhythmic drugs, and no arrhythmias on serial 24-hour Holter.

More than 30 years ago, Zipes and Knope [1] reported the presence of cardiac fibers with the capacity to generate an electrical impulse within thoracic veins. It has only been recently, however, that special attention has been given to the clinical meaning of this finding. The pulmonary veins, and, less often, the superior vena cava and the Marshall vein (embryonic remnant of the left superior vena cava) have been identified as arrhythmogenic foci that induce atrial fibrillation [2-6]. In addition, ablation of those foci has been shown to cure the arrhythmia [2-6].

Like the thoracic veins, the pulmonary artery and aorta also have a myocardial extension with electrical activity. Asirvatham et al [7], in an anatomic study with 230 human hearts, showed the presence of cardiac fibers within the pulmonary artery, and reported that 13% of the hearts studied had cardiac muscle, on average, 3 mm above the pulmonary valve. In addition, application of radiofrequency in the coronary leaflet has been reported to be able to eliminate certain types of extrasystoles [8,9]. However, the ablation of arrhythmogenic foci in the pulmonary artery has rarely been explored, and, so far, only one group has reported the origin of that arrhythmia above the pulmonary valve plane [10,11]. In our study, we report the case of a patient with idiopathic ventricular tachycardia, whose arrhythmogenic focus was eliminated with application of radiofrequency in the pulmonary trunk.

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Case Report

The patient is a 26-year-old female who has had palpitations and presyncopes for 4 years although using propranolol, propafenone, and sotalol. The clinical examination showed irregular cardiac rhythm due to the presence of arrhythmia. The 12-lead electrocardiogram showed sinus rhythm intercalated with nonsustained ventricular tachycardia with left bundle-branch block morphology, lower deviation of the axis in the frontal plane, and S-R transition in V4 (fig.1). The presence of structural heart disease was ruled out through a chest X-ray, echocardiography, and magnetic resonance imaging. The latter did not show replacement of muscle tissue by adipose tissue in the right ventricle. The signal-averaged electrocardiogram was negative for the presence of late potentials. The 24-hour Holter showed 52% ventricular beats, 1672 of which were in the form of nonsustained ventricular tachycardia of up to 15 beats. The diagnosis of idiopathic ventricular tachycardia originating in the right ventricular outflow tract was suspected, and the patient was referred for electrophysiological study for ablation of the arrhythmogenic focus.

The procedure was performed with the patient under sedation and with local anesthesia. The bipolar electrograms were filtered at 30-500 Hz and recorded with 32-channel polygraph. Through punctures of the right femoral vein, 2 catheters were introduced under radioscopic vision and positioned in the right ventricle (apex and outflow tract). During the study, the presence of frequent episodes of spontaneous nonsustained ventricular tachycardia with the same morphology as described were observed. Mapping and ablation were performed under controlled temperature with a 7-Fr catheter and 5-mm tip (EP Technologies, Inc., Sunnyvale, CA, USA). The site for radiofrequency application was determined by comparing the morphology of the stimulated QRS with the QRS of the spontaneous ventricular tachycardia (pace mapping) and by the precocity of the ventricular electrogram in regard to the beginning of the earliest QRS of ventricular tachycardia. Mapping of the right ventricular outflow tract was performed with that technique. Although 12/12 criteria were obtained with stimulation, no precocity was observed and 2 applications of radiofrequency did not eliminate the arrhythmia. Positioning of the ablation catheter in the pulmonary trunk region showed the presence of a ventricular electrogram with 30-ms precocity in relation to the beginning of the earliest QRS and 12/12 criteria with stimulation. The threshold of stimulation was 6 mA with pulse width of 2 ms (fig. 2 and 3). In the first seconds of radiofrequency application at

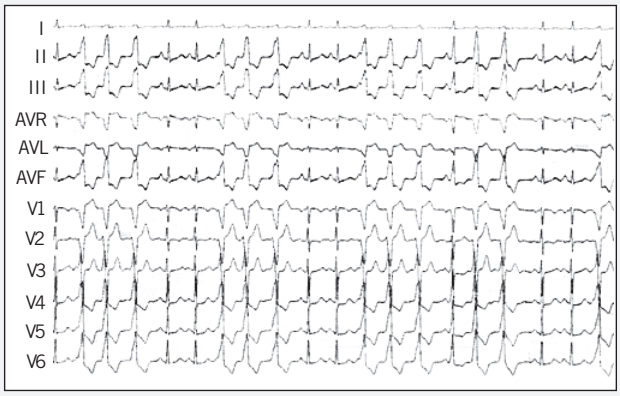

Fig. 1 - Twelve-lead electrocardiogram showing sinus rhythm and frequent episodes of nonsustained ventricular tachycardia with left bundle-branch block morphology in V1 and lower axis deviation in the frontal plane (positive D1, D2, D3, and AvF). Note that QRS complex is predominantly negative until the V3 lead, therefore showing late transition (R>S in V4).

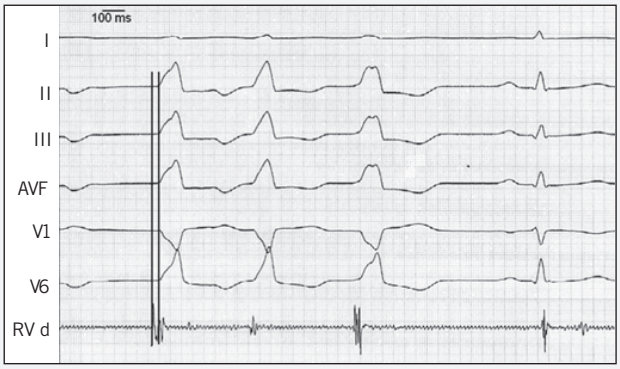

Fig. 2 - Peripheral electrocardiogram in the D1, D2, D3, AvF, V1, and V6 leads and intracavitary electrogram (RVd) recorded at 100-mm/s and 10 mm/mV velocities, respectively. Note that the intracavitary electrogram (distal RV) is the potential recorded by the distal poles of the ablation catheter positioned in the pulmonary trunk, showing 30-ms precocity in regard to the earliest QRS during ventricular tachycardia.

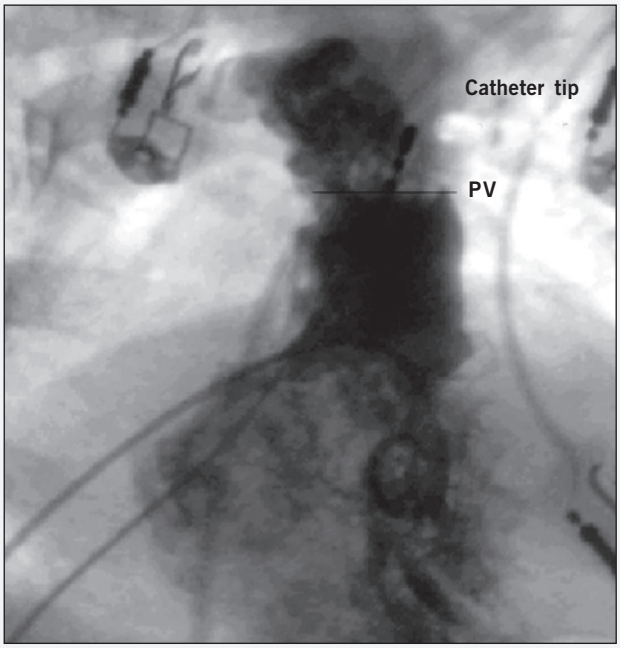

Fig. 3 - Right ventricular angiography showing the site of radiofrequency application (catheter tip) that resulted in the disappearance of ventricular tachycardia. Note that the catheter tip is located above the straight line at the pulmonary valve plane level.

that site, the ventricular tachycardia and extrasystoles were eliminated, without recurrence even after isoproterenol infusion. The patient has remained asymptomatic for 14 months, with no treatment with antiarrhythmic drugs, and no arrhythmias on serial 24-hour Holter monitoring.

Discussion

Although the presence of electrical activity in myocardial fibers within thoracic vessels may be an arrhythmogenic focus, it was only recently that catheter ablation of those triggering foci was shown to cure arrhythmia [2-6,8-10]. In the case of arrhythmias with wide QRS, specific extrasystoles with right bundle-branch block morphology and axis deviation to the inferior quarter of the frontal plane may be eliminated in the coronary leaflets, demonstrating that the origin of the arrhythmia is out of the cardiac chambers[9]. Less frequently, the extrasystoles of the left ventricular outflow tract may have the left bundle-branch block morphology and be confounded with those originating from the right ventricular outflow tract. Differentiation by using electrocardiography has been performed with the R/S ratio in the V3 lead, correlating an early transition with the left ventricular outflow tract, and a late transition (R/S < 1.0 in V3) with the right ventricular outflow tract [8]. Based on that criterion, the morphological pattern of ventricular tachycardia here presented suggested a right ventricular outflow tract origin. However, the application of radiofrequency that cured the arrhythmia was performed in the pulmonary artery. Recently, Timmermans et al [10,11] have reported the cure of ventricular tachycardia with morphology similar to that here presented by using ablation in the pulmonary trunk.

The pulmonary artery origin of the arrhythmia may still be corroborated by the fact that the arrhythmogenic mechanisms of idiopathic ventricular tachycardia of the outflow tract are the automatism or triggering activity, or both, characteristics that make it focal. Therefore, it is acceptable that the myocardial fibers located in the great vessels may be foci of arrhythmias, and, due to their focal nature, the stimulation of the site has the same morphology of the QRS of the spontaneous arrhythmia. Thus, it is also understandable that the punctual application of radiofrequency at the site of arrhythmia origin definitely eliminates it. Our patient, after 14 months of clinical follow-up with serial Holter, remains asymptomatic with no ventricular arrhythmia.

The difficulty in achieving success in ablating that type of arrhythmia has been attributed to supposed epicardial foci. Epicardial mapping has proved to be an option for successful treatment. However, it is worth noting that, as in our case and in that of Timmermans et al [10,11], the arrhythmia focus may originate in the pulmonary trunk, and it may be advantageous to map that site before attempting other forms of mapping or other access routes.

Therefore, ventricular tachycardia with the pattern of left bundle-branch block and axis deviation to the lower quarter in the frontal plane, a morphology that suggests the existence of a focus in the right ventricular outflow tract, may also have its origin in the pulmonary trunk. Radiofrequency application in that site may definitively cure the arrhythmia, and in cases in which ablation in the right ventricular outflow tract failed, mapping of the pulmonary trunk may be a useful alternative before other methods are used.

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