Ablation Therapy of Tachycardia-Related Cardiomyopathy

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Untreated prolonged supraventricular tachycardia is a well-known predisposing condition for dilated cardiomyopathy in children [1]. Elimination of the tachycardia by medication or radiofrequency ablation usually results in reversal of left ventricular dysfunction. Cardiomyopathies secondary to prolonged sustained or non-sustained ventricular tachyarrhythmias have been reported [2–4]. We describe the case of a 14 year old girl in whom frequent premature ventricular contractions were the apparent cause of cardiomyopathy. Radiofrequency ablation of a right ventricular outflow tract focus led to complete recovery of LV function.

Patient Description

A 14 year old girl was referred for evaluation of a cardiac murmur. She had a 4 month history of fatigue and chest discomfort. The family history was negative for cardiac disease. Physical examination revealed a soft 2/6 regurgitant murmur maximal at the apex and an abnormal rhythm. Pulses in the upper extremities and lower extremities were normal and equal and were not delayed. A 12-lead electrocardiogram showed normal sinus rhythm with multiple unifocal PVCs, left bundle branch block and right axis morphology [Figure], suggesting that the problem was related to right ventricular outflow. A 24 hour Holter monitor recording demonstrated sinus rhythm with a mean rate of about 80 beats per minute, 26,198 isolated monomorphic ventricular premature beats, 4,855 ventricular couplets, and 45 runs of 3–4 beats of ventricular tachycardia with a rate of 160 beats per minute. An echocardiogram revealed an end-systolic volume of 23.2 ml, an end-diastolic volume of 66 mm (ejection fraction 49%), and mild to moderate mitral regurgitation. No evidence of right ventricular dysplasia, congenital heart disease or abnormal RV function was seen. LV and RV wall thickness was normal without focal abnormalities. On exercise testing, 84% of maximal heart rate was achieved after 11 minutes without symptoms. Premature beats were present at rest but disappeared at peak effort and reappeared during recovery. The patient was referred for radiofrequency ablation.

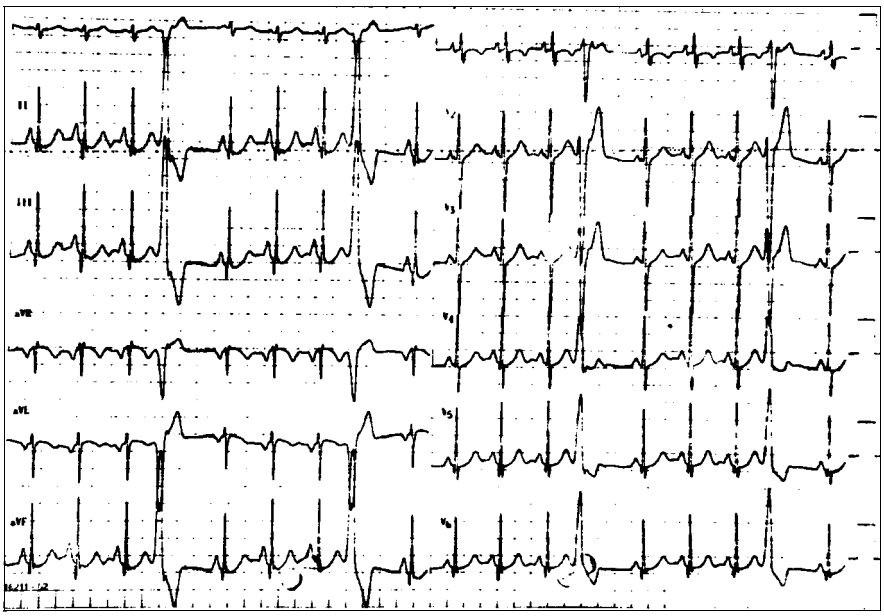
An electrophysiologic study and radiofrequency ablation were performed under conscious sedation with midazolam and diprivan. Five-French quadripolar electrophysiologic catheters were inserted through the right femoral vein and positioned in the right atrium, the right ventricular apex and the His bundle. Ablation was performed with a 6F quadripolar deflectable catheter (Cordis-Webster, USA) with a 4 mm distal tip. Surface ECG leads were recorded along with intracardiac electrograms using a computerized electrophysiologic recording system (Biotronik, Berlin, Germany). Programmed electrical stimulation with up to three extra stimuli at two drive-cycle lengths and rapid burst pacing were performed from the right ventricular apex and outflow tract at twice diastolic threshold at baseline and after

RV = right ventricular

EGG = electrocardiogram

LV = left ventricular

PVC = premature ventricular contraction



Twelve-lead ECG showing normal sinus rhythm with multiple unifocal PVCs, left bundle branch block and right axis morphology.

isoproterenol infusion. No sustained ventricular arrhythmia was induced, but the patient remained in a constant ventricular bigeminy rhythm. Ablation sites in the radiofrequency outflow tract were selected using bipolar pace-mapping in comparison to the native PVC morphology and by detection of sites of early QRS activation during the PVCs. Using a 50 watt radiofrequency generator (Biotronik) under temperature control (70°C), seven radiofrequency applications were delivered in the posteroseptal aspect of the RV outflow tract. During the fifth application, after 5 seconds of energy delivery at a temperature of 50°C, all ectopic activity terminated and did not reappear. Two additional "booster" applications were then delivered to the same site. No ventricular ectopic activity was observed during a 30 minute observation period with programmed electric stimulation and isoproterenol infusion. The patient remained in the hospital for an additional 24 hours without evidence of ectopy.

Four weeks later, a Holter monitor recording showed normal sinus rhythm without ectopy and the echocardiogram was normal, with disappearance of mitral valve regurgitation. The girl has been followed for 2 years during which she has been asymptomatic and echocardiograms and Holter monitor recordings continue to be normal.

Comment

An adolescent girl with multiple premature ventricular contractions and short episodes of non-sustained ventricular tachycardia developed LV dysfunction. Radiofrequency ablation of the ventricular focus abolished the arrhythmia and resulted in complete reversal of the LV dysfunction.

Since there was no other known reason for cardiomyopathy to have developed in this patient, we suggest that the frequent PVCs and recurrent bigeminy created a tachycardia-induced cardiomyopathy that was resolved by ablation of the RV outflow tract focus. Whether the frequent PVCs *per se*, or the resulting irregular heart rhythm caused the tachycardia-induced cardiomyopathy is unclear. The patient did not exhibit significant resting tachycardia and there was no evidence of prolonged runs of ventricular tachycardia by history or Holter recordings. Arrhythmogenic RV dysplasia is the most important entity that needs to be differentiated from idiopathic RV outflow tract arrhythmias, and its pathology involves replacement of myocardium by adipose tissue. Our patient had no evidence of RV dysplasia or other organic heart disease.

While RV outflow tract tachycardia is usually considered benign and is treated symptomatically, both animal models and human studies have shown that incessant or chronic tachycardia can lead to ventricular dysfunction that is reversible with rate or rhythm control [5]. The present case raises the possibility that RV outflow tract, at least in the pediatric age group, may be a cause of clinically significant, reversible cardiomyopathy. Such cases should be treated preferably with ablation. Furthermore, patients with frequent and seemingly benign PVCs should undergo echocardiography to exclude cardiomyopathy since the PVCs may be the cause, and not merely the result, of the cardiomyopathy.

References

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