Short communication

Tachycardiomyopathy: A case report and review of literature

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ABSTRACT

Tachycardiomyopathy is well-recognized reversible form of myocardial dysfunction caused by chronic arrhythmias. Treatment of arrhythmia leads to recovery of myocardial function. Hence, it is of utmost importance to look for and identify arrhythmias in every patient presenting with myocardial dysfunction without any apparent etiology. We hereby present a case of persistent junctional reciprocating tachycardia with cardiomyopathy whose left ventricular function reverted to normal in three months following successful radiofrequency ablation of tachycardia.

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Introduction

Tachycardiomyopathy is an uncommon, however well-recognized entity caused by long standing tachycardia which is usually treatable, reversible and hence have excellent prognosis.¹ Tachycardiomyopathy is reported and seen with all type of tachyarrhythmias. Supraventricular arrhythmias such as atrial fibrillation, atrial flutter, atrial tachycardias and reentrant supraventricular tachycardia (SVT) can lead to tachycardiomyopathy. Ventricular arrhythmias rarely lead to tachycardiomyopathy since this arrhythmia is usually associated with some form of underlying structural heart disease. We hereby report a case of tachycardiomyopathy due to paroxysmal junctional reciprocating tachycardia (PJRT) which was initially missed due to its paroxysmal nature and was later on cured with successful ablation of tachycardia.

Case report

A fourteen year old girl presented first in 1999 with history of shortness of breath. There was a history of fever 2 months back along with easy fatigability. She had heart rate of 110/min with blood pressure of 100/60 mm Hg. Electrocardiogram was interpreted as sinus tachycardia. Echocardiogram revealed a severely depressed left ventricular ejection fraction of approximately 25–30% with global hypokinesia.

She was given a diagnosis of dilated cardiomyopathy probably post-viral myocarditis and underwent endomyocardial biopsy which showed non-specific hypertrophy, nuclear enlargement without any evidence of myocarditis. She had recurrent palpitations. Hence keeping a possibility of tachycardiomyopathy patient underwent electrophysiology study (EPS). During EPS, tachycardia could not be induced this time. Patient was started on oral amiodarone, beta blocker and digitalis along with decongestive therapy and followed. Her LV function improved (LVEF – 55%) over next few months and subsequently she was lost to follow up.

Subsequently the patient presented again in 2006 with worsening dyspnea, palpitations, fever and cough. Echocardiography showed LVEF of 20–25%. Her heart rate was high 160/min. It was noted, however, that she had evidence of an incessant narrow complex supraventricular tachycardia (SVT) on her electrocardiogram (ECG). There was a long RP narrow complex SVT with inverted P waves in leads II, III, and aVF with upright p in V1 (Fig. 1). She was treated with antibiotics and after 10 days underwent electrophysiology study. During the study patient was having incessant tachycardia. It was a narrow complex tachycardia with heart rate of 160/min. It was similar to the baseline ECG. It was a long RP tachycardia with earliest retrograde A at CS 9–10 with a long local VA interval of 170 ms (Fig. 2). Tachycardia was entrained by ventricular pacing at right ventricle apex which showed V-A-V sequence at termination of V-pacing ruling out atrial tachycardia. Post-pacing interval minus tachycardia cycle length of 44 ms was suggestive of pathway. His refractory ventricular premature complex (VPC) reset the tachycardia. Earliest A was mapped at inferior rim of CS os. On basis of these findings diagnosis of orthodromic reciprocating tachycardia utilizing a slowly conducting posteroseptal accessory pathway i.e. PJRT was made. Tachycardia terminated within 5 s of

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Fig. 1. Twelve lead electrocardiogram in electrophysiology lab showing narrow complex tachycardia with HR 160/min with inverted P in II, III, aVF and upright P in V1.

Fig. 2. Tracing showing long RP tachycardia with earliest retrograde A at CS 9–10 with long local VA interval of 170 ms.

Fig. 3. Earliest A was mapped at inferior rim of CS os. Termination of tachycardia occurred within 5 s of radiofrequency burn.

Fig. 4. Post-ablation twelve lead electrocardiogram which shows normal sinus rhythm.

radiofrequency ablation (temperature of 60° C, power of 45 W) at site of earliest retro A (Fig. 3). The lesion was continued for 40 s.

Patient was discharged four days later in normal sinus rhythm (Fig. 4). She came to follow up after three months. Her Left ventricular function has improved to 50% and she was NYHA Class I.

Discussion

PJRT and atrial tachycardia accounts for the most of the causes of tachycardiomyopathy in children. Atrial fibrillation and atrial flutter also leads to tachycardiomyopathy if ventricular rates are not controlled. Ventricular arrhythmias which cause tachycardiomyopathy are usually idiopathic in nature. Sustained monomorphic VT rarely causes tachycardiomyopathy as it is more often associated with pre-existing structural heart disease. Tachycardiomyopathy is seen with very frequent premature ventricular contractions, ventricular tachycardia of outflow tract of right ventricle and fascicular tachycardia.^{2,3} PJRT is usually characterized by incessant form of supraventricular tachycardia and leads to tachycardiomyopathy. However it can be paroxysmal and can be missed as it happened in our patient.⁴ PJRT is a narrow QRS tachycardia with long RP interval which is also seen in atrial tachycardia and atypical AVNRT, hence requires electrophysiological study for precise diagnosis. It is more common in children than in adults. Recognition of PJRT as a cause of non-ischemic cardiomyopathy is important as its ablation leads to dramatic improvement in left ventricular function. Medical therapy is usually ineffective in controlling heart rate. Posteroseptal accessory pathways commonly seen in PJRT as seen in our case these are situated near the os of coronary sinus are high risk area of ablation due to proximity to left circumflex artery.⁵ Cryoablation is a suitable alternative to radiofrequency ablation with minimal risk to damage to coronary artery.⁶

There is usually a rapid clinical improvement after successful ablation of tachycardia. By three months there is usually recovery of left ventricular function as also seen in our patient.⁷ In a recent study majority

of echocardiographic parameters including ejection fraction improved significantly after treatment. However, left ventricular dimensions and volumes remained significantly elevated when compared with control subjects, indicating persistence of negative LV remodeling, even after appropriate treatment and normalization of EF at a mean follow-up of 14 months.⁸ Hence patients should continue to be monitored and treated with anti-reverse remodeling drugs like beta-blockers and ACE inhibitors.

Hence, we have described a case of PJRT related tachycardiomyopathy which was missed initially due to its paroxysmal nature. Diagnosis of Tachycardiomyopathy should be considered in the presence of persistent supraventricular or ventricular tachyarrhythmia with heart failure.

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