

Radiofrequency ablation for fascicular ventricular tachycardia causing tachycardiomyopathy and brief literature review

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ABSTRACT

A 10-years-old boy presented with a history of effort intolerance and palpitations for 4 months. His electrocardiogram showed wide complex tachycardia suggestive of fascicular ventricular tachycardia (VT). The echocardiogram showed moderate-to-severe left ventricular systolic dysfunction without any structural lesion. The tachycardia was unresponsive to adenosine and direct current cardioversion. It responded to oral verapamil. The electrophysiology study confirmed the tachycardia as left posterior fascicular VT. The tachycardia was successfully ablated guided by Purkinje potential on three-dimensional mappings. He showed improvement in ventricular functions before discharge. He is doing well on short-term follow-up.

Keywords: Fascicular ventricular tachycardia, radiofrequency ablation, tachycardiomyopathy

INTRODUCTION

Incessant tachyarrhythmia can cause impairment of ventricular function leading to tachycardiomyopathy. The atrial arrhythmias such as ectopic atrial tachycardia (EAT) is a relatively common cause of incessant tachycardia in the pediatric age group. Idiopathic fascicular ventricular tachyarrhythmia is a very rare tachycardia in the pediatric age group. We report an interesting case of a child who presented with fascicular tachycardia with tachycardiomyopathy along with a brief review of the literature.

CASE REPORT

A 10-year-old boy was transferred from another hospital due to symptomatic wide complex tachycardia. He had decreased exercise tolerance (New York Heart Association

functional Class III) and recurrent palpitations for the past 4 months. On clinical examination, he had a pulse rate of 160/min, a saturation of 98%, and blood pressure of 100/70 mm Hg. The cardiac evaluation revealed that normal heart sounds without any appreciable murmurs. The electrocardiogram (ECG) exhibited regular wide complex tachycardia with QRS width of 140 ms, right bundle branch block (RBBB) morphology, and left axis deviation suggestive of left posterior fascicular tachycardia [Figure 1]. Screening echocardiogram in the emergency room showed moderate-to-severe left ventricular systolic dysfunction, ejection fraction (EF) by M-mode 32% without any structural defects. Initially, intravenous adenosine was administered but the tachycardia did not respond. Then, direct current (DC) cardioversion was delivered twice (100 joules each

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ventricular echo beats were seen even after aggressive stimulation (with and without isoprenaline) from both atria and ventricle. The child was shifted to the pediatric intensive care unit for observational care. His ECG showed normal sinus rhythm, deep q in lead III, and a rightward shift of axis compared to baseline [Figure 7].

His repeat echocardiogram on day 5 of the procedure showed improved left ventricular EF to 50% and clinical improvement. He was discharged on maintenance therapy of diuretics and ACE inhibitors along with antiplatelet therapy. He is doing well on short-term follow-up.

DISCUSSION

Tachycardiomyopathy has been defined as myocardial dysfunction which is completely or partially reversible after the control of the etiological tachyarrhythmia.^[1,2]

Two variants of tachycardiomyopathy are described. The arrhythmia induced and arrhythmia mediated. In arrhythmia-induced tachycardiomyopathy the arrhythmia is the sole cause for myocardial dysfunction. In

arrhythmia mediated tachycardiomyopathy, arrhythmia exacerbates ventricular dysfunction in a patient with concurrent heart disease.

The incessant or chronic tachyarrhythmia leads to ventricular dysfunction. Sustained rapid heart rate, irregular heart rhythm, or AV dyssynchrony can contribute to tachycardiomyopathy.^[3]

The common pediatric arrhythmia causing tachycardiomyopathy in the pediatric age group are EAT 59%, permanent form of junctional reentrant tachycardia (PJ RT). Twenty-three percent, ventricular arrhythmia, a rare cause, found in 7% of the cases.^[2,4]

Fascicular ventricular tachycardias (VTs) are just 10%–15% of total pediatric ventricular arrhythmias making it a rare cause for tachycardiomyopathy in children.^[5,6] There are three variants of fascicular tachycardia: Left posterior fascicular VT with a RBBB and left axis deviation on ECG (common); left anterior fascicular VT with a RBBB and right axis deviation (uncommon); and high septal fascicular VT with relatively narrow QRS complex and normal axis (rare). The underlying mechanism is due to a reentrant circuit which is constituted by an antidromic limb consisting of a zone of slow, decremental conduction in the interventricular left septum proceeding from the base to the apex (verapamil sensitive, giving rise to P1). The lower turnaround point is toward the apex, and the retrograde limb is formed by the Purkinje network (giving P2).

In animal models, it has been shown that persistent tachycardia reduces left ventricular dp/dt max and myocardial blood flow. It also increases the left ventricular wall strain, end-diastolic pressure, and volume. This leads to left ventricular systolic dysfunction and the appearance of heart failure symptoms.^[7] The time frame from the onset of arrhythmia to the development of tachycardiomyopathy varies from 3 days to 120 days.^[2] Ulus *et al.* described the chronology of the development of tachycardiomyopathy in three stages. In the initial compensatory stage which lasts

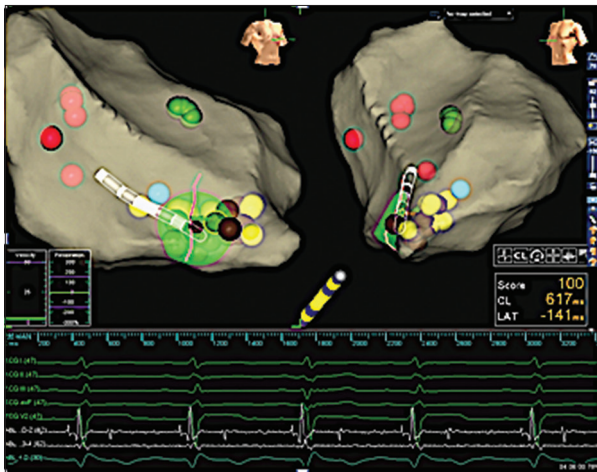


Figure 4: Three dimensional map showing violet as ablation lesions in the lateral and right anterior oblique views



Figure 5: Electrogram showing (a) sustained ventricular tachycardia due to catheter manipulation in the septal area and (b) catheter-induced ectopics very similar to clinical tachycardia

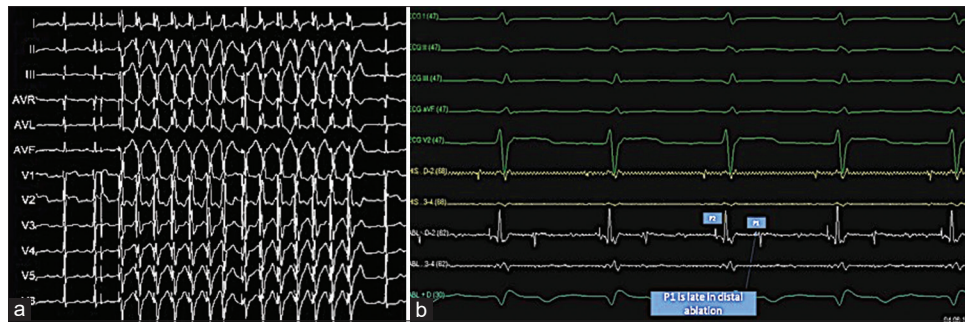


Figure 6: Pace-map showing (a) good match of clinical tachycardia, (b) P2 (Purkinje potential) precedes the QRS, P1 (prePurkinje potential) follows QRS on ablation catheter and is delayed in distal ablation (1-2) compared to proximal (3-4)

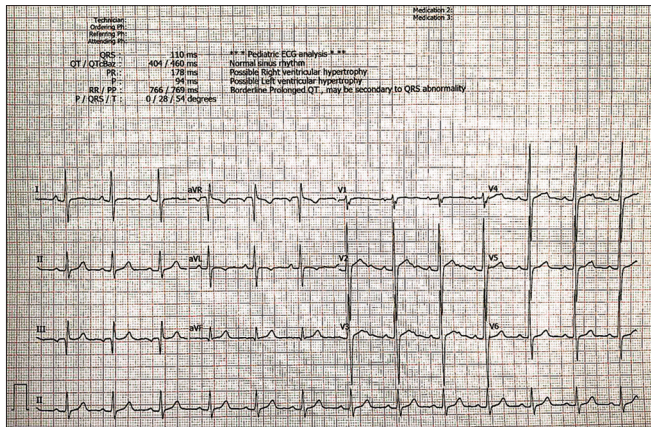


Figure 7: Postradiofrequency ablation electrocardiogram showing deep q in lead III and rightward axis than baseline

approximately 1 week the sympathetic system gets activated, but ventricular pumping function remains normal. In the second phase left ventricular pumping dysfunction and myocardial contractile dysfunction develops. This phase lasts for one to 3 weeks and is accompanied by left ventricular dilatation along with neurohormonal activation.

In the third phase of heart failure developing over 3 weeks the left ventricular severe pumping dysfunction develops. There is significant neurohormonal activation and systemic hemodynamic compromise and pulmonary edema accompanies this phase.^[8]

The clinical features of tachycardiomyopathy in the pediatric age group varies from asymptomatic patient to end-stage heart failure.^[9] The children often present late as they fail to recognize palpitation or express the heart failure symptoms. Hence, it is extremely important to recognize correctly hourly so that the devastating effect of cardiomyopathy can be reversed.

Dilated cardiomyopathy is the most common cause of cardiac transplant in children. Up to 40% of the cases of dilated cardiomyopathy require a cardiac transplant or die within 2 years of diagnosis. Hence, the diagnosis of tachycardiomyopathy is extremely important in the pediatric age group as it is potentially reversible.^[3]

Most important component in the treatment of tachycardiomyopathy is early recognition and appropriate treatment of offending arrhythmia. Normalization of heart rate or rhythm control is the cornerstone of treatment. The ideal treatment to achieve arrhythmia control depends on the nature of tachycardia. The options are antiarrhythmic drug therapy, external DC cardioversion, radiofrequency catheter ablation, or insertion of an implantable cardioverter-defibrillator.^[10]

In spite of verapamil, our case had a recurrence of VT with LV systolic dysfunction. As CMRI was normal it was a definite TCMP and RFA was class 1 indication. Catheter ablation can be performed either during VT or sinus rhythm. In our case tachycardia could not be induced and so linear ablation based on anatomical landmarks defined by EAM was performed. The appearance of P1 after QRS during RFA suggested injury to the slow limb with orthodromic activation during sinus rhythm. After completing ablation we aggressively attempted induction from both RAA and ventricle but could not demonstrate any tachycardia or ventricular echo beat of the same morphology suggesting that we had achieved bidirectional block in that slow conducting zone.

A schematic diagram [Figure 8] adapted from Ramprakash et al.^[11] can better explain the reentrant circuit in fascicular VT. As shown in the figure, there is a slow conducting zone (calcium-sensitive) which gives rise to prePurkinje potential (PP) (P1). This area is captured antidromically during tachycardia, thereby the P1 precedes the PP, P2 during tachycardia. Whereas it is captured orthodromically in sinus rhythm, hence the P1 follows the ventricular complex.

EAM is magnetic-based or impedance-based technology for catheter localization, with some system using both modalities. This mapping was very useful in our case. It helped us in delivering precise lesions and facilitated return to tagged areas easily. Fascicular VT is a rare form of arrhythmia in children with a success rate of ablation exceeding >85%. The risk

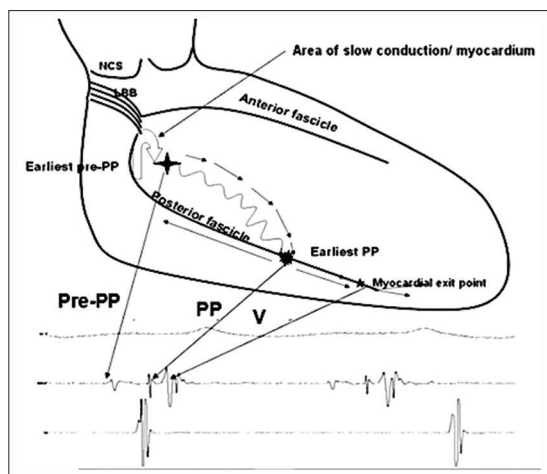


Figure 8: Tachycardia circuit in fascicular ventricular tachycardia. The antegrade limb of the circuit proceeds through the verapamil sensitive zone (curved line) from the basal to apical left ventricular septum giving rise to the prePurkinje potential (P1) as seen in the accompanying electrogram. The lower turn around site of the reentrant circuit occurs in the lower third of the septum with the capture of the fast conduction Purkinje fibers producing Purkinje potential (P2) along the posterior fascicle. From here, antegrade activation occurs down the septum to break through septal myocardium below, and retrograde activation occurs over the posterior fascicle from apical to basal septum forming the retrograde limb of the tachycardia. The reentrant circuit is completed by a zone of slow conduction at the upper turn around point of the circuit located close to the main trunk of the left bundle branch^[11]

of complications in older children and adolescents is quite low.^[12] Although in literature there are very few cases of young adolescents undergoing catheter ablation, it provides a definitive therapy in properly selected cases.

In addition to specific therapy for tachycardia, optimal supportive medical treatment is essential. Treatment with diuretics, beta-blocker, angiotensin-converting enzyme inhibitor, angiotensin receptor blocker, and aldosterone blocker help in reverse remodeling.^[7]

The recovery of tachycardiomyopathy is independent of the appropriate treatment strategy, either medical therapy or RFA. In the animal experimental model tachycardiomyopathy induced by rapid pacing starts showing signs of reversal within 48 h of discontinuation of pacing and slowly improves over the next 2 weeks. The median time of left ventricular systolic function recovery is reported approximately 7 weeks to 10 weeks which is the timeframe for reverse remodeling.^[2,13]

CONCLUSION

Tachycardiomyopathy in the pediatric age group needs strong clinical suspicion along with early appropriate diagnosis and treatment to reverse the devastating effect of arrhythmia causing end-stage heart failure.

The supraventricular arrhythmia is the common cause of tachycardiomyopathy. Fascicular VT is a very rare arrhythmia causing tachycardiomyopathy. Any patient with incessant tachycardia and heart failure deserve detailed analysis of the case and analysis of the ECG for the diagnosis of tachycardiomyopathy. Medical therapy and catheter ablation for cases like fascicular VT in children have good outcome. Long-term follow-up is required to monitor the patient for ventricular function.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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