

Two Case Reports on Incessant Left Ventricular Tachycardia: Curative Therapy with Radiofrequency Ablation

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Abstract

Introduction: Incessant ventricular tachycardia is a rare arrhythmia which can be life threatening. Treatment with anti-arrhythmic agents may occasionally fail. **Clinical Picture:** We report 2 cases of incessant ventricular tachycardia. The first case was a young man with idiopathic left ventricular tachycardia who was in incessant ventricular tachycardia despite treatment with multiple anti-arrhythmic drugs and developed dilated cardiomyopathy. The second case was an asymptomatic girl with the incidental finding of an incessant ventricular tachycardia which originated from the left ventricular outflow tract. **Treatment and Outcome:** Both patients underwent electrophysiologic study and radiofrequency ablation with complete termination of the tachycardia. **Conclusion:** Radiofrequency catheter ablation in experienced centres should be the first-line therapy for incessant ventricular tachycardia.

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Introduction

Left ventricular tachycardia is a recognisable condition in patients with otherwise structurally normal heart. It may arise from various foci in the left ventricle. While it is paroxysmal in nature at most times, occasionally it may be incessant. Generally it follows a benign course¹ and may be treated with anti-arrhythmic agents.^{2,3} However, several case reports have suggested that it may result in tachycardia-induced cardiomyopathy^{2,4-6} or sudden death. The cardiomyopathy may be reversed if the tachycardia can be controlled either by anti-arrhythmic agents^{2,3} or catheter ablation.⁷⁻¹² We report 2 cases of drug-resistant incessant ventricular tachycardia with different presentations and their outcome after radiofrequency ablation.

Case Reports

Case 1

The first patient, a 22-year-old bartender of Chinese origin, had history of intermittent palpitations for 5 years. He was admitted in September 1984 with the complaint of increasing palpitations both in frequency and severity. His baseline effort tolerance was otherwise good. Apart from

a heart rate of 120 beats per minute, the other clinical findings were unremarkable. Chest radiograph was normal.

The electrocardiogram (ECG) (Fig. 1) showed monomorphic, sustained broad complex tachycardia with atrioventricular dissociation, left axis deviation and right bundle branch block (RBBB) morphology. This ECG feature, coupled with the presentation, suggested that the patient had ventricular tachycardia originating from the left posterior fascicle. The tachycardia was terminated with intravenous verapamil. Two-dimensional echocardiogram showed that the left ventricular size was normal but the left ventricular ejection fraction was 40%. Electrophysiologic study later confirmed that the tachycardia originated in the left ventricle and could be aborted with intravenous verapamil. The patient was subsequently treated with oral verapamil. The ventricular tachycardia recurred 8 years later. This time it was resistant to drugs such as verapamil, propranolol and amiodarone. Two-dimensional echocardiogram revealed a dilated left ventricle with global hypokinesia and left ventricular ejection fraction of 15%.

The patient underwent another electrophysiologic study in February 1993 during which sustained monomorphic

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ventricular tachycardia with a cycle length of 550 ms was induced. The earliest activation site during left ventricular mapping was localised to the inferoapical septum near the left posterior fascicle. Radiofrequency ablation at this site terminated the incessant ventricular tachycardia. Coronary angiogram done just prior to electrophysiological study showed normal coronary arteries.

Two more episodes of incessant ventricular tachycardia occurred between 1993 and 1995. Radiofrequency ablations to the inferoapical septum were carried out twice. After the last radiofrequency ablation, the patient remained asymptomatic since. Two-dimensional echocardiogram done 5 months later showed a normal left ventricular size and function with an ejection fraction of 58%.

Case 2

The second patient was a 13-year-old schoolgirl with scoliosis. She was otherwise asymptomatic. Other than the presence of frequent ectopic beats, clinical findings were unremarkable. 24-hour Holter monitoring showed frequent sustained monomorphic ventricular tachycardia. Treadmill exercise stress test revealed frequent ventricular ectopics at rest, which disappeared after stage 1 exercise. No arrhythmia or significant ST changes were observed. Target heart rate was achieved during the test.

Two-dimensional echocardiogram showed a structurally normal heart with normal left ventricular function. She was started on oral verapamil and remained asymptomatic throughout. A repeat echocardiogram in February 1999 showed similar findings, with no evidence of dilated cardiomyopathy.

Although she remained asymptomatic, 24-hour Holter monitoring in May 1999 showed a high density of ventricular tachycardia with rates of 180 beats per minute. At the time of referral to our institution, she was having incessant ventricular tachycardia. The ECG (Fig. 2) showed narrow complex tachycardia with a QRS duration of 0.09 seconds and normal axis. The ventricular tachycardia terminated spontaneously. This was followed by a sinus beat after which the ventricular tachycardia was resumed. Although the QRS complexes were narrow, they were still wider than the QRS complex of the sinus beat. There was a P wave occurring after every second QRS complex, indicating a 2:1 QRS/P relationship. Of particular interest was the presence of an Rs wave pattern in lead V3 and positive QRS complex in lead aVL. These electrocardiographic features suggested that the origin of ventricular tachycardia was at the left ventricular outflow tract.¹³

An electrophysiologic study was performed, during which both left and right ventricles were extensively mapped using an electroanatomical mapping system (CARTO™, Biosense, Tirat Ha Carmel, Israel). The earliest activation

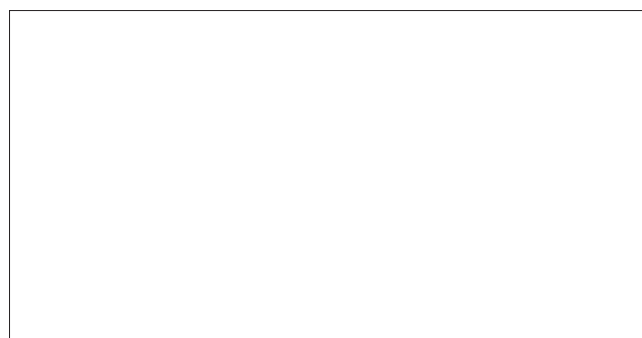


Fig. 1. Sustained monomorphic broad complex tachycardia with atrioventricular dissociation, left axis deviation and right bundle branch block morphology. Such electrocardiogram suggests that the ventricular tachycardia focus most commonly originates from the inferoapical septum of the left ventricle.



Fig. 2. Narrow complex tachycardia, with QRS duration of 0.09 seconds and normal axis. There was a P wave occurring after every second QRS complex indicating a 2:1 QRS/P relationship. The P waves were highlighted by the smaller upward pointing arrows. The ventricular tachycardia was spontaneously terminated. This was followed by a sinus beat (downward pointing arrow) after which the ventricular tachycardia was resumed. The QRS complex during the tachycardia was wider than the QRS of the sinus beat.

site was located at the anterolateral region of the left ventricular outflow tract. Radiofrequency ablation at this site terminated the incessant ventricular tachycardia. The patient has remained in sinus rhythm since then.

Discussion

Although ventricular tachycardias, especially those associated with coronary artery disease, are generally life threatening, certain forms of ventricular tachycardia are classified as “benign”.¹⁴ Among them is the unique idiopathic left ventricular tachycardia, which is characterised by ventricular tachycardia of RBBB morphology and left axis deviation occurring in structurally normal hearts. Most of the time the tachycardia is paroxysmal. This type of ventricular tachycardia shows a male predominance and interestingly is terminated by intravenous verapamil^{15,16} and diltiazem.¹⁶ As seen in the first case, the origin of ventricular tachycardia is usually in the inferoapical portion of the left ventricle.¹⁷

Oral verapamil may prevent the recurrence or slow the ventricular rate. The pathogenesis of this type of ventricular tachycardia is thought to be due to reentry and this reentry

circuit is likely to be of considerable size. Wen et al¹⁸ were able to demonstrate that ablation at a site away from the tachycardia exit site, where the earliest Purkinje potential could be recorded, resulted in termination of the tachycardia. The mean distance between the ablation site and the tachycardia exit site was 3.1 cm. The reentry circuit was then thought to encompass the middle, inferior and lower aspects of the left interventricular septum if the exit site was at the classical inferoapical septum region.

Incessant ventricular tachycardia is a rare, yet recognisable form of idiopathic ventricular tachycardia. One of the complications of incessant ventricular tachycardia is tachycardia-induced cardiomyopathy as shown in the first case. There have been several case reports of tachycardia-induced cardiomyopathy^{4-6,19,20} and control of ventricular tachycardia with drugs such as verapamil² or amiodarone³ have been reported to reverse the cardiomyopathy. However in recent years, radiofrequency catheter ablation has been proven to be the method of choice in the treatment of incessant ventricular tachycardia.²¹⁻²⁵ In idiopathic left ventricular tachycardia, radiofrequency ablation has been associated with high success rates of 90% to 95%.²⁶ Following successful ablation, the tachycardia-induced cardiomyopathy may be “reversed”.⁹⁻¹²

Case 1 was a typical drug-resistant incessant idiopathic left ventricular tachycardia resulting in cardiomyopathy. Significant deterioration of left ventricular function was noted over the years. The electrocardiographic findings of the RBBB and left axis deviation suggested that the ventricular tachycardia exit site was at the inferoapical septum of the left ventricle. The cardiomyopathy was fully reversible once the ventricular tachycardia was cured using radiofrequency ablation.

Case 2, on the other hand, illustrates the early presentation of incessant ventricular tachycardia arising from different focus of the left ventricle. It was interesting to find that the QRS complexes in the ECG were narrow. The shorter QRS duration was caused by excitation from the outflow tract, which spread more rapidly through the septal Purkinje fibres than excitation of the free wall. Kamakura et al²⁶ studied the electrocardiograms of patients with ventricular tachycardia from the left or right ventricular outflow tract. The R/S amplitude ratio of more than 1 in lead V3 localised the origin of the ventricular tachycardia to be from left ventricular outflow tract. Using the above criteria, the probability of the left ventricular outflow tract being the origin of the ventricular tachycardia was 80%.²⁶ While it was not strongly evident that tachycardia-induced cardiomyopathy might eventually occur, nonetheless the tachycardia was incessant even with oral verapamil. With its low complication and high success rate, radiofrequency ablation could have reduced the long-term medical costs

and potentially prevented the complication of tachycardia-induced cardiomyopathy in this patient. Indeed, in many centres, radiofrequency ablation has become the first-line treatment in patients with left ventricular tachycardia.

Conclusion

Although left ventricular tachycardia in structurally normal heart is often termed a benign form of ventricular tachycardia, it may rarely become incessant and causes dilated cardiomyopathy. Significant left ventricular dysfunction may occur as a result of the tachycardia itself. Inspection of the 12-lead ECG alone can usually identify the origin of the ventricular tachycardia with considerable accuracy. Left ventricular dysfunction as a result of incessant ventricular tachycardia may be reversed by radiofrequency catheter ablation. Termination of ventricular tachycardia by radiofrequency catheter ablation not only improves cardiac function, but also reduces long-term medical cost. Thus in experienced centres, radiofrequency catheter ablation should be the first-line therapy for incessant ventricular tachycardia and tachycardia-induced cardiomyopathy.

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