

## Spontaneous resolution of ventricular tachycardia with right bundle branch block morphology: A case report

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Ventricular tachycardia is rare in children. In the absence of structural heart disease, ventricular tachycardia is known as idiopathic ventricular tachycardia and carries a good prognosis.

We report a 14-month-old male child with right bundle branch block incessant ventricular tachycardia without structural heart disease. In this patient ventricular tachycardia was controlled by amiodarone and disappeared during follow-up. We want to stress the benign nature of this tachycardia if the previous treatment protocol had been appropriate.

*Key words:* ventricular tachycardia, children.

The mechanism of ventricular tachycardia (VT) that occurs in the absence of structural heart disease may vary. Triggered activity or reentry through calcium channel-mediated conduction pathways are the postulated mechanisms<sup>1</sup>. Two clinical forms have often been observed: (1) verapamil-sensitive VT characterized by right bundle branch block (RBBB) morphology with superior frontal plane axis, and (2) catecholamine-sensitive VT characterized by left bundle branch block (LBBB) morphology with an inferior frontal axis<sup>2</sup>. Incessant idiopathic VT is a disease of infancy. The most common QRS morphology is that of RBBB with left axis deviation, but all the different morphologies of bundle branch block have been seen in individual patients<sup>3</sup>. There is also another type as described by Garson<sup>4</sup>. In this tachycardia Purkinje cell tumor generates tachycardia. In this study, we report a 14-month-old male child with RBBB ventricular tachycardia without structural heart disease who had resolution of VT during follow-up.

### Case Report

A 14-month-old male child was referred from another hospital because of tachycardia and

congestive heart failure without any benefit from several antiarrhythmic drugs. Physical examination revealed a temperature of 36°C, pulse of 248 bpm, respiration of 32 per minute, and a blood pressure of 100/60 mmHg. He had Gallop rhythm and congestive heart failure. A grade 3/6 systolic murmur was audible at the 4<sup>th</sup>-5<sup>th</sup> left intercostals space and the liver edge was palpable 5 cm below the right costal margin. Chest X-ray revealed cardiomegaly. The electrocardiogram showed a wide QRS tachycardia with a RBBB morphology with superior axis (Fig. 1). Two-dimensional and M-mode echocardiography demonstrated a depressed left ventricle function secondary to incessant VT. There was no structural heart disease in the patient. Initially, adenosine was administered via intravenous injection at a dose of 200 µg/kg, but tachycardia persisted except for supra VT with bundle branch block. Afterwards, the patient was brought to the intensive care unit and amiodarone was given intravenously at a loading dose of 5 mg/kg/hour, followed by an infusion dose of 5 µg/kg/min. Ventricular tachycardia did not recover, so the amiodarone doses were increased to 7.5-10-12.5 µg/kg/min and 15 µg/kg/min,

**Fig. 1.** Surface electrocardiogram recorded during ventricular tachycardia. The typical right bundle branch block-superior axis morphology suggests that ventricular tachycardia originates from left ventricle.

respectively. VT terminated with an infusion dose of 15  $\mu\text{g}/\text{kg}/\text{min}$  of amiodarone and the heart rate reduced to 140 bpm. Afterwards, amiodarone was given orally at a maintenance dose of 5  $\mu\text{g}/\text{kg}/\text{day}$ . Moreover, digoxin and captopril were used in this patient because of congestive heart failure. The patient was hospitalized for 10 days. In this period, 24-hour Holter monitoring and echocardiography were performed in order to demonstrate the efficacy of amiodarone therapy. Holter monitoring revealed normal sinus rhythm and short burst of nonsustained VT attacks. The patient was discharged on amiodarone and digoxin therapy. In the follow-up, twelve-lead electrocardiography (ECG), 24-hour Holter monitoring, echocardiography, and ophthalmologic examination were performed and serum levels of thyroid hormones ( $T_3$ ,  $T_4$ , TSH) and liver enzymes (ALT, AST) were obtained one, three, six and 12 months after initiation

of therapy and were repeated every six months thereafter. Since VT resolved spontaneously, no attempt was made for cardiac catheterization, angiocardiology, electrophysiologic study or endomyocardial biopsy.

During follow-up, short burst of VT and intermittent sinus rhythm were determined on 24-hour Holter monitoring. Verapamil was administered intravenously at a dose of 0.15 mg/kg to exclude the verapamil-sensitive VT, but VT persisted. Amiodarone treatment was then continued. On follow-up he had normal cardiac function assessed by echocardiographic study. There were no effects due to amiodarone therapy. After four years of initiation therapy, amiodarone treatment was stopped because resolution of VT was observed in the patient. All Holter evaluations during the past two years have been normal (Fig. 2). He was followed without antiarrhythmic medication for 18 months. Eighteen months

**Fig. 2.** 24-hour Holter monitor strips demonstrate normal sinus rhythm after discontinuation of the amiodarone.

after discontinuation of the amiodarone, his Holter demonstrated no ventricular ectopy or VT and a normal treadmill study.

### Discussion

Ventricular tachycardia occurring in patients with no apparent structural heart disease is termed idiopathic VT, which could be either right ventricular or left ventricular in origin. Idiopathic left VT has the morphology of a RBBB with a leftward axis. The origin of the VT is usually at the inferior left ventricular midseptum or apex in the region of the posterior fascicle. Although the mechanism of this tachycardia may not always be uniform, investigators have found that idiopathic left ventricular tachycardia can be reentrant<sup>1,2,5</sup>. Moreover, it has been observed that small intramyocardial tumors were the source of permanent VT in infants despite a normal echocardiogram and angiogram. The most common type of such tumor is the Purkinje cell tumor<sup>3,4</sup>. Patients with idiopathic left ventricular tachycardia are generally young and usually symptomatic with sustained VT<sup>5</sup>. In a multicenter study reported by Pfammatter et al.<sup>6</sup>, clinical symptoms or echocardiographic signs of left ventricular dysfunction were observed initially in 36% of patients. VT with LBBB pattern differed significantly from VT with RBBB pattern in the clinical profile. Only 25% of patients with right VT had symptoms compared with 67% of patients with left VT. Our patient also had ventricular tachycardia with RBBB, as well as symptoms and signs of congestive heart failure. Echocardiographic examination revealed impaired left ventricle function due to incessant VT.

Idiopathic left VT is unresponsive to beta-blockers or adenosine but frequently responds to verapamil<sup>1,5</sup>. VT attack was not suppressed by intravenous adenosine administration in our patient. During follow-up, administration of verapamil also failed to control VT. In our patient VT attack was stopped by intravenous amiodarone. In several studies the use of amiodarone and verapamil was found to be more effective in suppression of VT than treatment with beta-blockers in a considerable number of patients<sup>6-8</sup>.

Ventricular tachycardia in children with a normal heart carries a good prognosis<sup>5,6,9,10</sup>.

Pfammatter et al.<sup>6</sup> demonstrated a higher proportion of resolution of VT in infants than in children of older age (>1 year) at initial presentation. Moreover, they demonstrated that outcome was better when VT originated in the right ventricle. Spontaneous resolution of VT occurred in our patient four years after initiation of VT attack.

But during this period the patient needed antiarrhythmic drug therapy to treat left ventricular dysfunction due to incessant VT.

Radiofrequency catheter ablation (RFA) in children has also been successfully performed as an option in treatment of idiopathic VT. But RFA is not suggested as the first-line therapy because of potential risks and complications due to manipulation of catheter. However, it should be considered especially in children with impaired ventricular function who are not responsive to medical therapy<sup>6,11,12</sup>.

In conclusion some types of VT in children have a good long-term prognosis. In most of the affected patients VT may resolve. Therefore, in these patients continued monitoring for resolution of VT is warranted after VT is controlled by antiarrhythmic medication.

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