Case Reports

A case of sustained ventricular tachycardia associated with severe ventricular dysfunction (tachycardiomyopathy)

K Sivakumar[1], **Priyanthi Molligoda**[2]

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Introduction

Belhassen ventricular tachycardia (VT) is an idiopathic tachycardia occurring in structurally normal hearts. The mechanism of this tachycardia is a micro re-entrant focus in the left posterior fascicle of the left bundle, producing a broad QRS tachycardia of right bundle branch block (RBBB) morphology and left axis deviation (LAD). Sustained tachycardia, either of ventricular or supraventricular origin, lasting for hours to days with very fast rates, can result in dilatation of the ventricles and progressive reduction of their systolic contractility. This manifests clinically with cardiomegaly and echocardiographically as dilated cardiomyopathy. This clinical entity called tachycardio-myopathy differs from the usual types of dilated cardiomyopathy in being totally reversible on termination of the tachycardia. We describe a 4 year old child with sustained VT over 3 days resulting in severe ventricular dysfunction.

Case report

A 4 year old male child, who was previously well, had a history of cough, rapid breathing, and not doing well for three days prior to admission. The child was detected to have a fast heart rate (230/min) by the paediatrician and admitted to Durdans hospital, Colombo on 14th November 2002. Echocardiographic evaluation in hospital showed dilatation and severely impaired contractility of both ventricles. He was diagnosed as having dilated cardiomyopathy and referred to Apollo hospital the next day, three days after the onset of his symptoms.

On clinical examination, he was normally grown for his age with a heart rate of 230/min, low pulse volume and a blood pressure of 80/60 mm Hg. There was mild cardiomegaly. Echocardiogram (ECHO) showed significantly impaired ventricular function with an ejection fraction (EF) of 20%. His electrocardiogram (EGG) (Figure 1) showed a ventricular rate of 214/min, broad QRS tachycardia of RBBB morphology, QRS axis of 75° and LAD. Careful analysis of P and QRS waves showed V-A dissociation; the QRS rate was 214/min and the P rate 102/min; P waves are shown in arrows (Figure 2). Based on the EGG features, a diagnosis of left posterior fascicular VT was made

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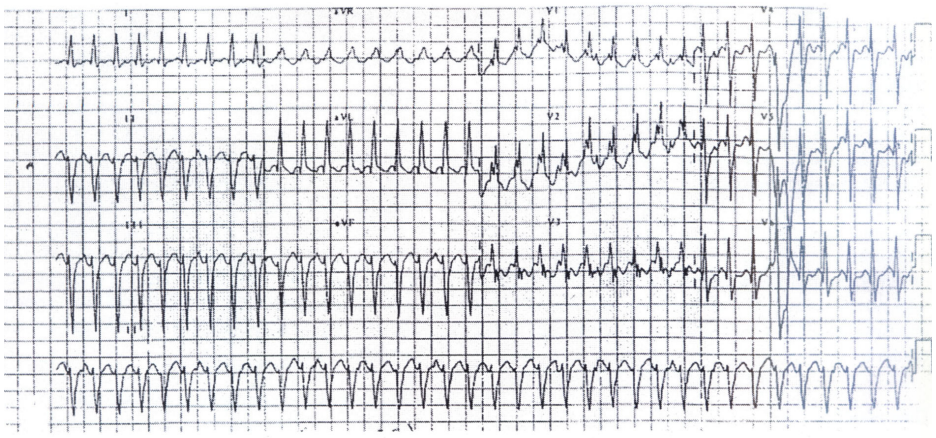

Figure 1

[1]*Consutant Paediatric Cardiologist,* [2]*Consultant Paediatrician, Apollo Hospitals, Colombo.*

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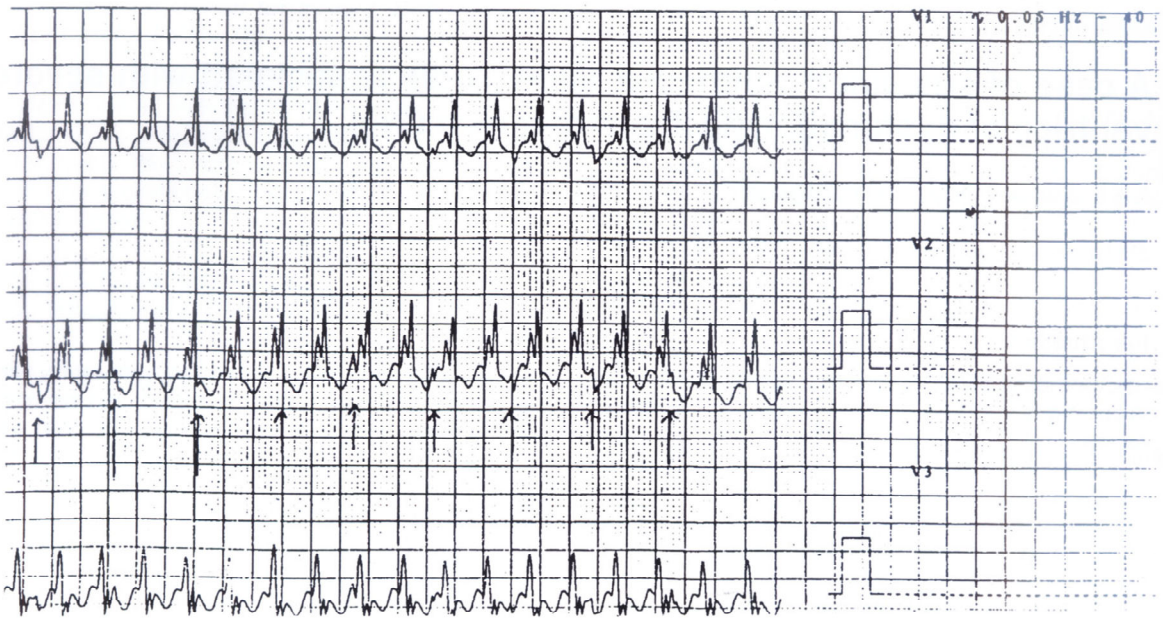

Figure 2

Initial trials to control the tachycardia with intravenous (IV) adenosine (150mg/kg rapid bolus) and metoprolol (two doses of 0.1 mg/kg) failed. Even though this VT is well known to be verapamil sensitive, the ECHO finding of severe ventricular dysfunction precluded use of IV verapamil. The tachycardia was terminated using amiodarone (5mg/kg bolus over 30 minutes followed by infusion of 0.6 mg/kg/hour) within one hour. Subsequently the patient was started on oral verapamil 1 mg/kg once in 6 hours. After 72 hours of termination of the tachycardia, the ECHO showed total normalization of ventricular function and the EF improved to 55%. There was no recurrence of tachycardia till the last follow-up.

Discussion

Sustained tachycardia with very fast rates, either of ventricular or supraventricular origin, lasting for hours to days, can result in dilatation of the ventricles and progressive reduction of their systolic contractility. This clinical entity, called tachcardiomyopathy, differs from the usual types of dilated cardiomyopathy in being totally reversible on termination of the tachycardia[1]. Identification of this entity is clinically important since termination of tachycardia needs usage of drugs like calcium channel blockers and betablockers which reduce inotropicity of the heart further. On the contrary, drugs to improve systolic contractility of failing ventricles, like catecholamine infusions which are administered in dilated cardiomyopathy, are contraindicated since they perpetuate tachycardia and further worsen systolic contractility of ventricles.

In any child presenting with tachyarrhythmia, a 12 lead EGG has to be recorded to differentiate supraventricular tachycardia (SVT) since the treatment strategies and prognoses differ. In this patient, EGG features of broad QRS tachycardia, superiorly directed QRS axis and V-A dissociation established the diagnosis of VT. Three additional features viz. RBBB morphology, LAD and absence of structural abnormality of the heart on ECHO narrowed the diagnosis to IDIOPATHIC LEFT POSTERIOR FASCICULAR VT (Belhassen VT).

The three common types of VT which occur in structurally normal heart (idiopathic VT)[1] are (i) Left fascicular VT (ii) Left outflow tract VT and (iii) Right ventricular outflow tract VT. Fascicular VT are further classified as (i) Left posterior fascicular VT, the commonest form with RBBB morphology and leftward QRS axis, (ii) Left anterior fascicular VT with RBBB morphology and right axis and (iii) Upper septal fascicular VT, the rarest with normal QRS and normal axis[1]. The left posterior fascicular tachycardia (Belhassen VT), is also known as verapamil sensitive VT and originates from a micro reentrant focus near the posterior fascicle of the left bundle[2]. This produces a broad QRS tachycardia of RBBB morphology and LAD, the QRS axis lying between 45° and 90°. The tachyarrhythmia responds well to verapamil but may not respond to many other

anti-arrhythmic drugs making recognition of this arrhythmia very crucial for management[2].

Cardiomyopathy, defined as primary myocardial dysfunction, denotes a dismal prognosis short of heart transplantation[3]. Some causes of "treatable cardiomyopathy" include ventricular outflow tract obstructions, coronary anomalies, sustained tachyarrhythmia and certain metabolic abnormalities. Sustained SVT and VT result in impairment of ventricular function and this entity called tachycardiomyopathy has been recognized since the early 90s[4,5,6]. The termination of tachycardia results in very early normalization of left ventricular function[7,8,9]. Failure to recognize this entity may lead to continuation of the tachycardia for long periods and leads to worsening of the ventricular function and sudden cardiac deaths[10]. Electrical and molecular abnormalities have been identified in the role of fast ventricular rates in development of tachycardiomyopathy and remodelling and dilatation of the heart[11].

Acknowledgments

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