

Bidirectional Ventricular Tachycardia of Unknown Etiology

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ABSTRACT

A 45 years old male presented to the emergency department with palpitations, headache and apprehension. His electrocardiogram revealed bidirectional ventricular tachycardia. He remained vitally stable and responded to intravenous beta-blocker. Initially digitalis toxicity was suspected but history was negative for digitalis intake. The cause remained unidentified in patient despite detailed investigations. During a short follow-up (of 6 months) he remained asymptomatic and no cause was further identified during this period. Some other unseen causes of bidirectional ventricular tachycardia need to be explored.

Key words: *Bidirectional ventricular tachycardia. Cause. Unknown.*

INTRODUCTION

Ventricular tachycardia (VT) is a common arrhythmia that is encountered in cardiology in almost every day practice. Bidirectional ventricular tachycardia is a rare occurrence especially in the absence of digitalis toxicity. Catechoaminergic polymorphic VT (CPVT) is a well-known example of bidirectional VT. The distinguishing characteristic of CPVT is an alternating QRS axis morphology with rotation of 180° on a beat-to-beat basis. It usually occurs with digitalis toxicity, but is also seen in hypokalemia, familial CPVT, myocarditis and ischaemic heart disease. The diagnosis in CPVT cases is based on reproducibly elicited ventricular arrhythmias during exercise stress testing.¹

Experimental evidence indicates that the mechanism of VT in these cases is ectopic discharges by triggered activity mainly in the epicardium. Bidirectional and polymorphic VT are preceded by multiple ectopic beats. It can be difficult to differentiate between bidirectional VT and ventricular bigeminy.² CPVT was first described in a case report in 1975.³ Bidirectional VT has also been described in herbal aconite poisoning, long QT Syndrome and metastatic cardiac tumour. Alternating left and right bundle branch block has also been described as a cause mimicking bidirectional VT.⁴

A case of bidirectional VT was also described in association with non-ischaemic dilated cardiomyopathy and was successfully ablated. The authors concluded that the tachycardia mechanism was two ventricular foci firing in an alternate fashion.⁵ Association with Anderson-Tawil syndrome (long QT type 7) and

hypokalemic periodic paralysis has also been described in literature.⁶

This report describes a case of biventricular tachycardia the cause of which could not be determined.

CASE REPORT

A 45 years old male was brought to Emergency (ER) with complaints of palpitation, headache and apprehension for 2 hours. There was no associated dyspnea or chest discomfort. There was no history of such complaints in the past and family history was negative for such complaints. Risk factors for coronary artery disease were negative and there was no past history of coronary artery disease or family history positive for cardiac disease. He was emotionally disturbed due to death in the family a few days ago.

He was hemodynamically stable with a blood pressure of 130 systolic and 90 diastolic and a pulse of 225 per minute. Examination of the rest of the system was unremarkable. Electrocardiogram (Figure 1) revealed a wide complex tachycardia with QRS width of 136 milliseconds and rate of 233 per minute. The QRS axis showed variability on beat-to-beat basis, rotating almost 180 degree from every other beat. The morphology was more clearly apparent in leads II, III, aVf, V5 and V5 (Figures 2 and 3). Lead V1 revealed right bundle branch pattern. The tachycardia was regularly irregular with changing short and long R-R cycles from beat-to-beat. A diagnosis of bidirectional VT was made and patient responded to beta blockers with return of normal sinus rhythm (Figure 4), although initially treated with xylocaine and amiodarone with no response. Complete blood count, serum electrolytes, urea, creatinine, troponin T and CK-MB were within normal limits in the emergency department. Drug history (for digitalis and other proarrhythmic drugs) was negative and echocardiography in ER and later on in the ward did not reveal any cardiac abnormality. Cardiac catheterization showed completely normal epicardial arteries with normal

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cardiac function. After responding to treatment, his electrocardiogram returned to normal, sinus rhythm with almost normal findings and his corrected QT interval

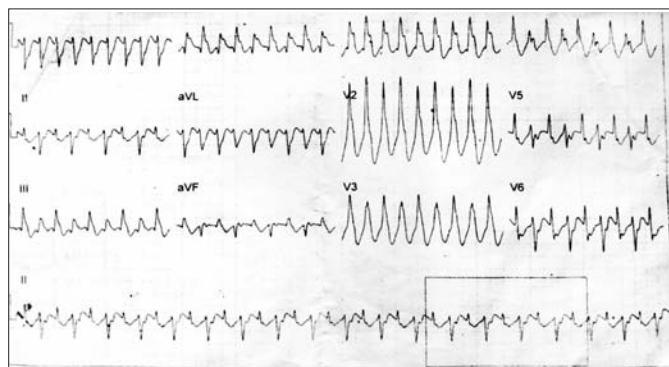


Figure 1: Electrocardiogram showing wide complex bidirectional tachycardia.

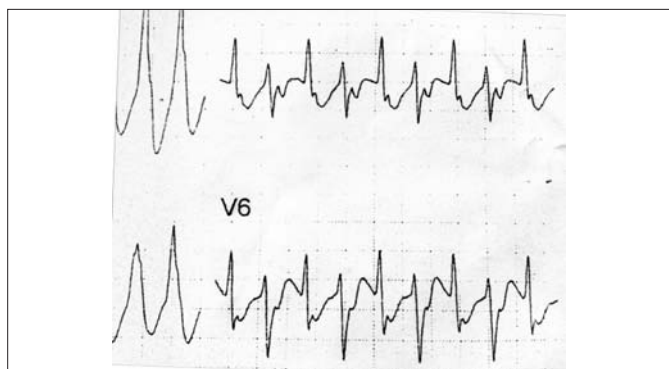


Figure 2: Bidirectional wide complex VT clearly shown in chest leads V5 and V6 in our case.

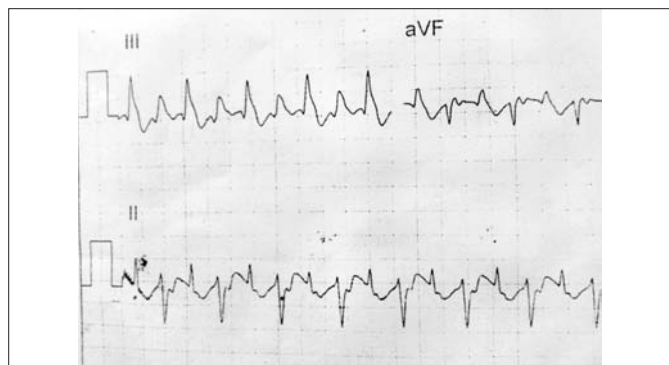


Figure 3: Bidirectional wide complex VT clearly shown in Limb leads II, III and aVf.

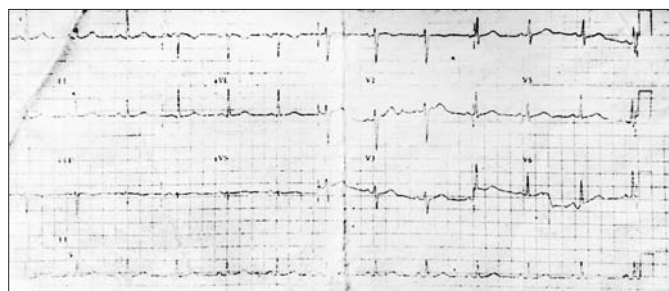


Figure 4: Electrocardiogram showing normal sinus rhythm with normal axis after treatment with intravenous metoprolol.

was within normal limits. Finally, a diagnosis of catecholaminergic polymorphic VT was considered and an exercise tolerance test was arranged to induce VT by exercise but there was no induction of arrhythmia and patient remained asymptomatic. As the patient did not develop any life-threatening clinical features (syncope, pre-syncope or cardiac arrest) and remained hemodynamically stable, we did not perform electrophysiological study.

The cause of bidirectional VT remained obscure in this case. He was discharged on a beta-blocker and advised to stay calm. He was also advised to report immediately to ER if he develops any symptoms related to arrhythmia. He did not develop similar problem during a short period of follow-up and follow-up was continued to reveal the etiology which could not be discovered.

DISCUSSION

Bidirectional VT is defined electrocardiographically as a VT with two QRS morphologies with alternating QRS complexes with changing QRS axis of 180 degrees. The tachycardia is regularly irregular with changing short and long R-R cycles. The mechanism for this changing QRS morphology in bidirectional VT has not been clearly identified. Existing data indicate that the bidirectional change in QRS axis results from ectopic foci firing close to the common left bundle and alternating conduction from the anterior and posterior fascicles.⁶

A variety of clinical conditions have been described in the generation of bidirectional VT.⁴ Although digitalis toxicity is a quite often implicated in its causation, there was no history of digitalis use in this patient. Other etiologies described in literature are myocarditis, myocardial infarction, hypokalemic periodic paralysis, catecholaminergic polymorphic VT metastatic cardiac tumour, Anderson-Tawil syndrome (long QT type 7) and herbal aconite poisoning.⁶ There was no history of aconite ingestion in this patient. Echocardiography did not reveal any evidence of myocarditis or metastatic cardiac tumour and history was also not suggestive of the same. Coronary artery disease was excluded on the basis of normal epicardial coronary arteries on left heart catheterization. The corrected QT interval was within normal limits and there were no clinical features to suggest Anderson-Tawil syndrome. Although CPVT was suspected but the arrhythmia was not induced on the exercise test. History, physical examination and normal serum potassium ruled out hypokalemic periodic paralysis. Although alternating right and left bundle branch is described in literature mimicking as bidirectional VT, baseline ECG would show at least block to consider this as a cause. Despite extensive search, the cause remained obscured. We think that there might still be some unidentified factors that led to the development of bidirectional VT in this patient. Some unidentified drugs or herbal medicines might be

responsible for this but in this case it remained a mystery. Follow-up was advised to reveal the hidden etiology.

Ventricular tachycardia is a common occurrence in cardiac coronary care units, bidirectional VT is seen only rarely. There are various causes implicated but some cases still remained unidentified.

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