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Case Report

Bidirectional ventricular tachycardia with myocardial infarction: A case report with insight on mechanism and treatment

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ABSTRACT

Bidirectional ventricular tachycardia (BVT) is a rare variety of tachycardia with morphologically distinct presentation: The QRS axis and/or morphology is alternating in the frontal plane leads. Since its original description in association with digitalis,[1] numerous cases of this fascinating tachycardia with disparate etiologies and mechanisms have been postulated. We report a patient with BVT in association with non-ST elevation myocardial infarction and severe cardiomyopathy in the absence of digoxin toxicity.

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1. Case report

A 61-year-old African American male with history of myocardial infarction in the remote past came to the emergency department with chest pressure associated with nausea, vomiting, palpitation and diaphoresis for two days, which became progressively worse. On a 12-lead EKG he had sustained monomorphic ventricular tachycardia at a rate of 153 beats per minute at the time of presentation to the ED (Figs. 1 and 2). After initial doses of amiodarone and lidocaine failed to restore sinus rhythm, he was cardioverted by transthoracic approach with 100 J synchronized, biphasic direct current shock. Initially intravenous abciximab drip started

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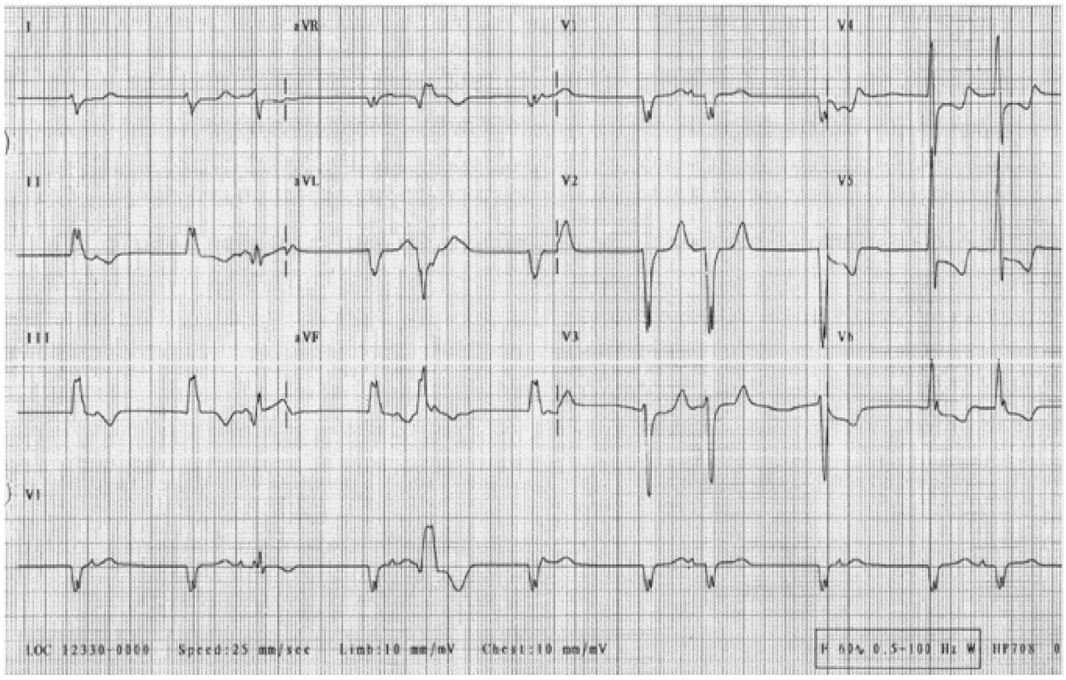

Fig. 1 – Accelerated idio-ventricular rhythm, 3rd complex is sinus with aberrant conduction, 5th beat is a PVC and subsequent blocked PAC.

based on high suspicion of STEMI, which was later discontinued and patient was continued on heparin and amiodarone drips.

He had a history of ischemic cardiomyopathy, hypertension, COPD, hyperlipidemia and chronic renal insufficiency. His left ventricular ejection fraction was 20%. He was taking Fenofibrate 48 mg per day, Altace 10 mg per day, Hydrochlorthiazide 25 mg per day and Furosemide 40 mg twice a day. He was not on any beta-blockers.

Patient was alert and oriented to time place and person during presentation followed by loss of consciousness. His blood pressure was 84/62, heart rate 153, respiratory rate 18. He had S3 gallop and diminished breath sounds with minimal rales bilaterally.

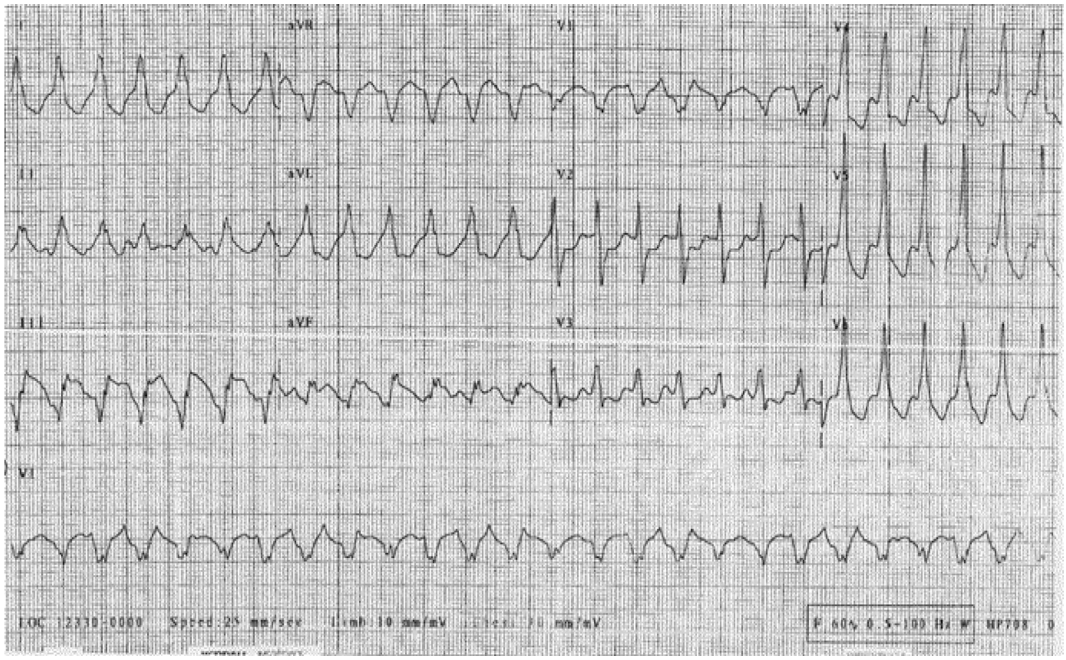

Fig. 2 – Sustained monomorphic ventricular tachycardia.

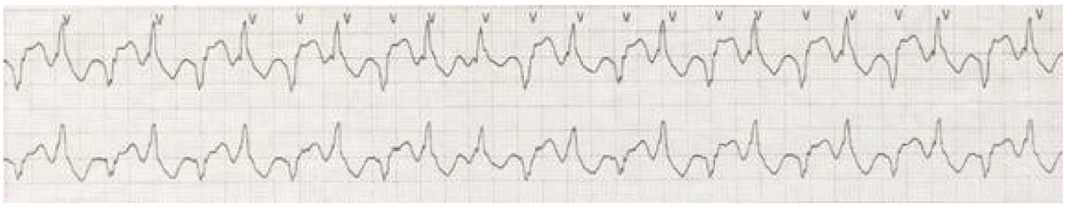

Fig. 3 – Bidirectional ventricular tachycardia.

Abdomen was distended and he had 2+ pitting edema. His initial laboratory data revealed: blood urea nitrogen 41 mg/dl, creatinine 3.5 mg/dl, CPK 110 units/l, CKMB 5.8 ng/ml, Troponin-I 1.6 ng/ml, BNP 748 pg/ml. Electrolytes were normal. A 2-D Echocardiogram revealed dilated cardiomyopathy, mild septal hypertrophy in the outflow tract, LVEF 10%, moderate to severe mitral regurgitation and tricuspid regurgitation.

An urgent coronary angiography showed completely occluded right coronary artery disease with mild diffuse disease in other coronary system. Right atrial pressure of 26 and left ventricular end-diastolic pressure of 48 mm of Hg.

On the 3rd hospital day, patient developed bidirectional ventricular tachycardia (Fig. 3). He was given additional bolus of amiodarone with resolution of VT (Fig. 4).

2. Discussion

Causes of bidirectional ventricular tachycardia:

- Acute ischemia
- Aconitine poisoning
- Catecholaminergic Polymorphic VT
- Digoxin Toxicity
- Familial Hypokalemic Periodic Paralysis
- Fatty replacement in RV
- Tumor of the Ventricle
- Myocarditis

2.1. *Ischemic heart disease*

Mechanism of ventricular tachycardia is complex in the setting of acute coronary syndrome and it is mediated due to interaction of ionic imbalances with neurohumoral changes together with increase in electrical resistance between cardiac myocytes. During ischemia, there is net loss of K^+ in the extracellular space which is compensated by net intracellular Na^+ gain through various major Na influx pathways. Intracellular Na^+ results in increase in Ca^+ influx via Ca^+/Na^+ exchanger. The net increase in Ca^+ may increase delayed after-depolarization (DAD) related arrhythmia including BVT.

BVT is rare in the setting of acute myocardial infarction and very few case reports are described in the literature. A case of BVT was reported in association with acute myocardial infarction and aortic stenosis.[2] Another case report is in a patient with myocardial infarction.[3] Our case is the fourth case report to our knowledge.

In a canine model of ischemia,[4] where LAD was ligated to produce ischemic zone, reentrant bidirectional PVC's were inducible on pacing. If these bidirectional PVC's continued to perpetuate, they would appear as sustained BVT which was occasionally observed during these experiments.[4]

2.2. *Catecholaminergic polymorphic ventricular tachycardia (CPVT)*

Majority of patients with CPVT may present as BVT.[5] It is associated with channelopathy[6] and has been linked to a variant of long QT syndrome. Typically it is a familial[7] form of catecholamine mediated, exercise induced, bidirectional tachycardia with an autosomal dominant pattern of inheritance. An early age of onset and mortality as high as 30% by the age of 30 years has recently been described.[8]

In a normal cardiac cell, depolarization of the membrane initiates calcium influx via voltage dependent L type Ca^{2+} channel. This calcium in turn causes calcium-induced calcium release from ryanodine receptor 2 (RyR2), which in turn is responsible for activation of actin-myosin molecules leading to contraction of the heart muscles. During diastole, calcium enters back into the SR, via the RyR2 receptor.

In patients with CPVT due to a defect in RyR2[8,9] the channel remains open for longer duration time, extending into diastole, increasing the calcium release from SR into the cell. Some of this Ca^{2+} is exchanged via the Na^+/Ca^{2+} exchanger

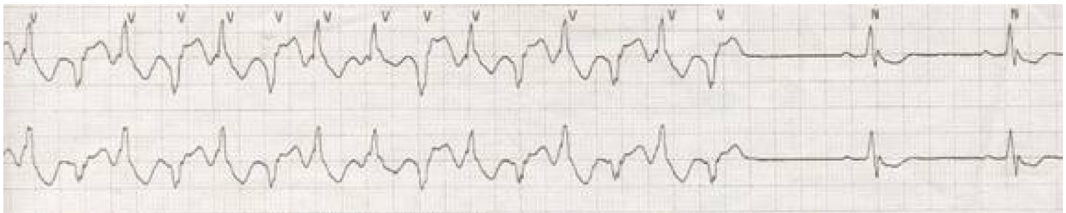

Fig. 4 – Abrupt termination of bidirectional tachycardia.

(NCX). The net increase in transient inward current leads to delayed after-depolarization (DAD) depolarization in Phase IV of monophasic action potential triggering polymorphic or BVT.

2.3. *Digoxin toxicity*

In normal heart Na/K ATP-ase pump is responsible for extrusion of three molecules of sodium in exchange for 2 molecules of potassium. Digoxin binds the Na/K ATP-ase pump resulting in net increase in Na_i. The excess Na_i in the cytoplasm is exchanged with Ca_o^{2+} via Na^+-Ca^{2+} exchanger in 1:3 proportion. Only a small amount of Ca_i is a trigger for a sudden and large flow of calcium from the sarcoplasmic reticulum into the cytoplasm. Actin-myosin chains interact, resulting in enhanced contractility in patients with heart failure.

Digoxin also facilitates opening state of the RyR2 receptor increasing the rate and flow of calcium into the cytoplasm. This sudden release of calcium current is responsible for the positive inotropic effects of digoxin on cardiomyocyte and is also responsible for phase 4 DAD. If the amplitude of this DAD is sufficiently large, it triggers a polymorphic or BVT. Digoxin associated bidirectional ventricular tachycardia was first reported in 1922.[1]

2.4. *Carvedilol and its effect on ryanidine receptor*

Recent data suggest that Carvedilol has a specific RYR receptor blocking effect, hence the drug prevents sudden and large surges of Ca release from the sarcoplasma reticulum. Carvedilol's superiority to metoprolol among HF patients as demonstrated in MERIT-HF trial could have been due to the RYR receptor blocking effect. Based on this assumption, Carvedilol may be a superior agent in the treatment of CPVT where the surge of Ca release is due to genetic defect, which accelerates the release of calcium mediated calcium release from SR via RYR receptor.

3. Conclusion

1 BVT is a rare variety of VT, which is generally seen with digoxin toxicity or catecholaminergic PMVT both of which are associated with one common denominator-intracellular calcium overload. Rarely patients may have BVT due to anatomic causes such as due to fatty replacement in RV (not to be confused with arrhythmogenic RV dysplasia) or tumor of the heart. Rarely, it has been reported in association with acute myocardial infarction. We described a case of BVT in association with acute MI which was managed medically. EP study was not done. Treatment of ischemia was given priority in this patient as ablation therapy has limited role in this disorder. Treatment has to be individualized and therapy with beta-blocker the cornerstone of treatment. However, surgical approach may be needed in the anatomic variety of BVT.

Conflicts of interest

All authors have none to declare.

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