CASE REPORT

Bidirectional Ventricular Tachycardia Due to Coronary Allograft Vasculopathy a Unique Presentation

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Bidirectional ventricular tachycardia (BVT) is an uncommon type of polymorphic ventricular tachycardia (PVT) with alternating polarity of the QRS complex most commonly described in digitalis toxicity. Recent data has demonstrated the possible molecular basis of this electrocardiographic phenomenon. To our knowledge this is the first reported case of BVT in a patient with orthotopic cardiac transplantation and coronary allograft vasculopathy.

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CASE PRESENTATION

A 60-year-old Caucasian male with a history of diabetes, stage 2 chronic kidney disease, severe ischemic cardiomyopathy resulting in an orthotopic cardiac transplantation in 1997 was transferred to our institution for further evaluation and treatment of substernal chest discomfort and acute inferior wall ST-elevation myocardial infarction. A recent elective cardiac biopsy showed evidence of International Society for Heart and Lung Transplantation grade I rejection (ISHLT Grade 1). Emergent angiography demonstrated total occlusion of the distal RCA beyond the PDA. Percutaneous coronary intervention was performed with placement of a drug eluting stent to the infarct related artery. In addition, angiography demonstrated diffuse nonocclusive coronary artery disease. A postprocedure transthoracic echocardiography revealed akinesis of the inferior basilar wall, severe mitral regurgitation, and an ejection fraction of 40%. The patient had a prolonged intensive care stay complicated by pulmonary edema, renal insufficiency, and polymorphic ventricular tachycardia (PVT).

On postoperative day (POD) 2, he developed ventricular bigeminy. The ectopic beats had left bundle branch morphology, inferior axis, and a coupling interval of 320–340 ms. On POD 3, he developed frequent ventricular ectopy with runs of a right bundle branch superior axis nonsustained VT and a cycle length of 260 ms (Fig. 1). He remained hemodynamically stable and asymptomatic.

On POD 5, an ECG was performed that demonstrated ectopic QRS complexes with salvos of nonsustained VT with an alternating left and right bundle branch morphology (Fig. 2). A diagnosis of bidirectional VT (BVT) was made. On POD 6, he experienced sustained VT episodes with right bundle branch block (RBBB) superior axis morphology at a cycle length of 260 ms requiring multiple electrical cardioversions. Antiarrhythmic treatment with amiodarone and lidocaine was effective in suppression of further episodes of VT during this hospitalization. He was referred for electrophysiologic testing and ICD implantation. Sustained VT could not be induced with programmed ventricular stimulation in the basal state, or with isoproterenol infusion. A cardiac defibrillator was implanted for

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Figure 1. Note the variable coupling intervals (*) (CI) between sinus and left bundle/ inferior axis beats (400, 320, 350, 360 ms.) while the CI between the left and right bundle morphology beats (*) is constant (440 ms).

Figure 2. Note the variable CI (*) between sinus and left bundle beats (360, 400 ms); and the constant CI between the alternating left and right bundle beats during VT (440/400 ms).

secondary prevention. The amiodarone and lidocaine were discontinued.

At 1 year of follow up, he has been maintained on immune suppression with mycophenolate mofetil and tacrolimus and has required two hospitalizations for decompensated heart failure. ICD interrogation revealed three episodes of asymptomatic nonsustained VT at rates of 155 bpm not requiring ICD therapy. The patient has not experienced any further episodes of bidirectional VT and has not required antiarrhythmic therapy.

Commentary

BVT is an uncommon type of PVT with alternating polarity of the QRS complex most commonly described during digitalis toxicity. Recent data has demonstrated the possible molecular basis of this electrocardiographic phenomenon. To our knowledge, this is the first reported case of BVT in a patient with orthotopic cardiac transplantation and myocardial infarction related to chronic coronary allograft vasculopathy (CAV) in the absence of cellular or humoral rejection.

The incidence of ventricular arrhythmias (VT, ventricular fibrillation) after orthotopic transplantation (OHT) is largely unknown, although the risk of sudden death in long-term survivors is believed to be high.¹ Case reports have proposed that supraventricular tachycardias, atrial fibrillation, and atrial flutter are common arrhythmias seen in adult patients with OHT in the absence of rejection.² Investigational reports have determined CAV to have prognostic significance in the development of cardiac dysrhythmias.³ Conduction system abnormalities have been associated with CAV in adult OHT recipients, in the presence or absence of rejection and manifests as intranodal and infranodal disease with atrioventricular block and RBBB respectively.³ The various clinical entities and genetic etiologies of BVT are listed in Table 1.

In this case, the presentation in adulthood and noninducibility with isoproterenol make the diagnosis due to CPVT and hereditary channelopathies unlikely. The absence of electrolyte abnormalities and digoxin use exclude metabolic and drug induced etiologies and a normal right ventricular size and function make the diagnosis of arrhythmogenic right ventricular dysplasia very unlikely. The presence of coronary heart disease from coronary angiography favors ischemic heart disease due to CAV as the etiology of BVT in this patient.

Table 1. Causes of Bidirectional VT

Clinical etiologies

Catecholaminergic PVT (CPVT)⁴
Arrhythmogenic right ventricular dysplasia⁵
Myocarditis
Hypokalemia
Familial hypokalemic periodic paralysis
Familial polymorphic VT syndrome
Cardiac metastasis
Digoxin toxicity⁶
Myocardial ischemia
Long QT7

Genetic determinants

16 Megabase mutation on chromosome 1p13–21⁷
Single nucleotide substitution and missense mutation of the cardiac ryanodine (hRyR2) receptor⁸
Missense mutation of the calsequestrin 2 (CASQ2) gene⁹
Mutation of the HERG allele (R1047L)¹⁰

Electrophysiology mechanisms

BVT is defined in terms of the electrocardiographic features and is divided into two principle configurations. (1) Fixed bundle branch block and usually RBBB and alternating frontal plane axis due to delayed after-depolarization induced by triggered beats as in digoxin toxicity and CPVT^{11,12} and (2) alternating bundle branch block related to bifocal automaticity inscribed in opposite directions;¹³ or reentry around a circuit with two alternating exit sites.¹⁴ Although a precise mechanism could not be documented in this case, we propose a nonreentrant mechanism for the frontal plane axis change based on electrocardiographic evidence of variable coupling intervals between premature and normal beats (Fig. 2) are in favor of a nonreentrant mechanism and the second type of principle configuration as listed above. Further circumstantial evidence for nonreentry lies in the constant coupling intervals between alternating (left BBB [LBBB] and RBBB) consecutive premature beats which implies an interdependency of the two ectopic forms (Fig. 2). The phenomenon of BVT in this case may be explained by two potential mechanisms.

- (1) Two parasystolic foci competing with each other—one near the proximal portion of the RB (LBBB/Inferior axis), and the other in a location nearer the inferior aspect of the LB (RBBB/Superior axis),

(2) or by the possibility of a single focus with two different and alternating exit sites under the following conditions

- The conduction time (CT) from the focus (*) to the right bundle (A) < CT to from the focus (*) to the LB (B);
- The CT from the focus (*) to the LB (B) > CT to the RB (A) + transseptal activation time to the left bundle (C), that is, (B > A + C);
- Conduction block of the RB occurs at a higher frequency (slower rate) than the ectopic focus—in order to permit the expression of the alternating site closer to the Left Bundle (Right Bundle/superior axis QRS with a slightly longer coupling interval of 440 ms);
- Right bundle effective refractory period > Left bundle effective refractory period

The complexity of this latter mechanism, the absence of a fixed coupling interval between the sinus and ectopic beats (Fig. 1); and the regularity of the cycle lengths between the alternating left and right bundle ventricular complexes (Fig. 2; 400/440 ms) favor a multiple protected (parasystolic) foci mechanism.

A proposed sequence of arrhythmia evolution includes premature beats that increase progressively to bigeminy, bidirectional tachycardia and irregular sustained VT.

Conclusion

This is the first report demonstrating BVT in a cardiac transplantation recipient related to coronary artery disease and CAV. Although a precise mechanism of arrhythmia cannot be determined we provide an explanation for two possible electrophysiologic mechanisms.

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