Introduction: Bidirectional ventricular tachycardia (VT) is a rare type of VT that appears in digoxin toxicity and young patients with catecholaminergic polymorphic VT (CPVT). The occurrence of bidirectional VT originating from the papillary muscle has not been reported in the literature.

Case Presentation: A 38-year-old female presented with long standing palpitation lasting several months. Palpitation was begun after pregnancy. The patient’s symptom was intensified with exercise and emotional stress. A 12-lead electrocardiography shows bidirectional VT.

Conclusions: Bidirectional VT is a life-threatening VT characterized by right bundle branch morphology (common type) in V1 and beat to beat variation in QRS axis in the limb leads. This arrhythmia usually develops in a patient with digitalis toxicity or in patients with CPVT. Mechanism of the arrhythmia is not well-defined. Alternation of arrhythmia from bidirectional morphology to bigeminy premature ventricular contraction, successful ablation of the VT in territory of the posteromedial papillary muscle, and start-stop pattern show that the arrhythmia has focal origin.

Keywords: Papillary Muscle VT, Bidirectional VT

1. Introduction

Bidirectional ventricular tachycardia (VT) is a rare type of VT that appears in digoxin toxicity and young patients with catecholaminergic polymorphic VT (CPVT). The occurrence of bidirectional VT originating from papillary muscle has not been reported in the literature.

2. Case Presentation

A 38-year-old female presented with palpitation and dizziness from 11 months ago. She could not tolerate any physical activity or emotional stress. Palpitation had a start-stop pattern in rest state and intolerable incessant pattern in physical activity. Past medical history was negative for any medical problems. She did not have family history of sudden cardiac death. She had not received digoxin. Hemodynamic was stable during VT. Baseline ECG demonstrated wide QRS tachycardia with the right bundle branch block (RBBB) pattern in V1, alternating axis deviation in limb leads compatible with bidirectional VT (Figure 1A). Serum K was within normal limit. Baseline QTc was normal. Transthoracic echocardiography demonstrated mild LV dysfunction (left ventricular ejection fraction: 40%-45%). She did not accept any procedure without general sedation. An electrophysiologic study (EPS) was planned for the patient that showed bigeminy premature ventricular contraction (PVC) after sedation with propofol, with morphology similar to one of the QRS complex during VT (1B). Intracardiac echocardiography (ICE) was not available in our electrophysiology center. Left ventriculography was performed in the left anterior oblique (LAO) projection (Figure 2A - B). Geometry was created by the NAVX 3D mapping system. Voltage mapping and local activation time were performed (Figure 2C - D). The earliest ventricular signal was recorded in an upper part of the posteromedial papillary muscle (Figure 2A).

The patient underwent electroanatomic mapping using the NavX EnSite Velocity system (St. Jude Medical, Milwaukee WI). Voltage and activation mapping were performed for VT localization (Figure 2C - D). The optimal ablation site was determined by both local activation time mapping and pace-mapping. Radiofrequency energy was delivered with an irrigated-tip catheter at a power ranging between 20 to 40 W and a maximal temperature of 45°C.

The earliest activation time during tachycardia was recorded in an upper part of the posteromedial papillary muscle.
Premature ventricular contraction was eliminated with radiofrequency (RF) catheter ablation in the aforementioned area. Then, programmed ventricular stimulation was performed with and without isoproterenol infusion and we could not induce any PVC or VT. Trans-thoracic echocardiography demonstrated normal left ventricular function one week after ablation. A 12-lead ECG and exercise stress test were normal one month after ablation.

3. Discussion

Bidirectional VT is a life-threatening VT characterized by right bundle branch morphology (common type) in V1 and beat to beat variation in QRS axis in the limb leads. The arrhythmia cycles can be variable (1). The mechanism of the arrhythmia is not well-defined. Available data indicate that focal areas close to the left bundle and alternating conduction from the left anterior and posterior fascicles are the cause of changing QRS axis (1). Triggered activity and abnormal automaticity are the most common suggested mechanism of the arrhythmia (2). This specific ventricular arrhythmia develops in a few clinical situations. The most common clinical cases associated with bidirectional VT are digitalis toxicity and CPVT. Periodic hypokalemic paralysis, myotonic dystrophy, herbal acetone poisoning, myocarditis, Anderson-Tawil syndrome (long QT type 7), and myocardial infarction are rarely associated with this arrhythmia (1, 3). Normal physical examination, normal serum K, normal cardiac enzyme, and normal QT interval indicate that the patient did not have aforementioned etiologic causes. Patient’s arrhythmia profile was variable from bigeminy PVC and nonsustained VT in rest state to sustained VT during physical activity and emotional state. A right bundle branch block with superior axis morphology is common in patients with idiopathic VTs originating from LV posterior fascicle (LPF) and LV posterior papillary muscle (PM). The PMs are known sites of origin for idiopathic ventricular arrhythmias and ventricular arrhythmias post-infarction. However, no cases of bidirectional VT originated from the PMs have been reported.

Mapping at the site of left anterior and posterior fascicles demonstrated that the aforementioned fascicles are not involved. Early and late diastolic potentials were not observed either in sinus rhythm or during VT or PVCs. Activation mapping revealed that the arrhythmia was originated from the top portion of the posteromedial papillary muscle. Absence of a high frequency potential at the site of origin ruled out the role of the Purkinje system. Suppression of arrhythmia after propranolol prescription, successful RF ablation at the territory of posteromedial papillary muscle, absence of criteria for entrainment, and start-stop pattern suggest that the site of origin was within the PM itself. Focal mechanism of VT might be described with two exit sites. The other explanation is the alternating firing of two separate foci in the PM.
Successful ablation of bidirectional VT originating from bundle branch has already been reported in literature (4, 5).

In this case report, we presented an unusual origin of bidirectional VT. To the best of our knowledge, this is the first report of bidirectional VT originating from papillary muscle.

References