Repetitive Narrow QRS Tachycardia With a Group Beating Pattern

Amir Farjam Fazelifar,1 and Mohammad Rafie Khorgami1,*

1Cardiac Electrophysiology Research Center, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran
*Corresponding author: Mohammad Rafie Khorgami, Cardiac Electrophysiology Research Center, Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran. E-mail: rafikhorgami@gmail.com

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Abstract

Introduction: Dual atrioventricular (AV) node physiology is the substrate for atrioventricular nodal re-entrant tachycardia but electrophysiological manifestation may be different in this group of patients.

Case Presentation: A 44-year-old male known case of coronary artery disease from one year ago presented with frequent palpitation that was refractory to anti-arrhythmic drugs. On surface electrocardiogram (ECG), there was one P wave preceding two to four consecutive QRS complexes. An electrophysiological study was performed and dual AV node physiology in combination with concealed AV node conduction was confirmed. There was unsuccessful radiofrequency ablation (RFA) application in the right posteroseptal area but in second try, successful RFA application was performed in the left posteroseptal area.

Conclusions: Dual ventricular response in dual atrioventricular nodal (AVN) physiology is not the usual presentation and may be misdiagnosed with other rhythm disorders. Considering the typical finding on surface ECG and the exact electrophysiological study, the true diagnosis and management could be done in suspected cases.

Keywords: Dual AV Node Physiology, Dual Ventricular Response, Supraventricular Tachycardia

1. Introduction

Atroventricular nodal re-entrant tachycardia (AVNRT) is the most common cause of paroxysmal supraventricular tachycardia in adult patients (1). The pathophysiology of this arrhythmia is well-defined and present as slow and fast pathway conduction in AV node with re-entry mechanism.

In typical AVNRT, the characteristic of the slow pathway that has a shorter effective refractory period causes this pathway acts as antegrade limb and the fast pathway with longer refractory period roles as retrograde limb of re-entry circuit. During sinus rhythm, the antegrade conduction to distal conduction system usually occurred from fast pathway and (1:1) conduction was seen. However, different characteristics of these pathways in the refractory period and conduction velocity may create a different manifestation of dual AV node physiology (2, 3).

2. Case Presentation

A 44-year-old male presented with frequent episodes of palpitation that begun from two months ago. He mentioned that his palpitation was repetitive and paroxysmal in nature, accompanied with dyspnea and light-headedness. Palpitation was irregular without any correlation with daily activity. Palpitation was waxing and waning in nature at first month, but in the last episode palpitation continued and he referred to hospital. He had a history of the coronary artery disease and coronary artery bypass graft was performed one year ago. Transthoracic echocardiography demonstrated severe LV dysfunction with LVEF about 30% - 35%. The surface electrocardiogram (ECG) was obtained (Figure 1). Surface 12-lead ECG findings can be explained with premature junctional complexes (PJC) or dual AV nodal physiology. Electrophysiological (EPS) study was performed to define the definite arrhythmia mechanism (Figure 2A-2B). Electrophysiological characteristic was compatible with dual AV node physiology in some group beatings (single arrow head) and combination of dual AV node physiology and prolonged conduction of the third sinus beat in others (double arrow heads). The radiofrequency catheter ablation with a standard 4-mm catheter tip was done in the conventional site of this type of arrhythmia in the right posteroseptal region but despite the procedure was done appropriately the tachyarrhythmia induced with the EPS stimulation protocol. The site of ablation changed to the left posteroseptal region and catheter ablation was done successfully without any tachyarrhythmia recurrence.
After one P wave, two consecutive QRS complexes and irregular feature of QRS complex were seen.

3. Discussion

The 12-lead ECG findings in an aforementioned patient can be explained in different ways as follows: Dual AV node physiology with double ventricular response, and PJC or premature atrial beats superimposed on T-wave in triplet pattern (4).
In the presence of irregular paroxysmal supraventricular tachycardia with intervening sinus beats, conduction through the both slow and fast pathways should be considered. Some mechanisms are supposed for this phenomenon but more acceptable mechanisms are as follows: the conduction via fast and slow pathways was done but the retrograde slow pathway blocked after antegrade conduction from fast pathway. In this situation if the distal His-bundle refractory period is shorter than previous upper part of conduction system, conduction was occurred from distal common pathway. Also, it has been considered that in (1:2) phenomenon the very slow conduction from slow pathway makes full recovery time of His bundle and activates from the fast pathway (5-8).

In EP study, after one atrial electrogram two His and two ventricular potential were seen and HV interval was fixed. In junctional extrasystoles in bigeminy, coupling interval is less predictable (4).

In atrial bigeminy, the P wave may be concealed with T-wave, but during the EP study PAC is the earliest atrial electrogram. The 5th beat in intracardiac tracing can be explained as sinus beat with prolonged AV node conduction due to concealed conduction. Finally, one of the best predictors for 1:2 phenomenon is response to slow pathway modification with catheter ablation that causes discontinuation of arrhythmia. Of course in our case the successful ablation achieved in the left posteroseptal region.

In conclusion, the dual AV node physiology with double ventricular response alone or in combination with concealed conduction is a rare and unusual cause of arrhythmia that may be misdiagnosed with other types of supraventricular tachycardia (SVT) and for correct diagnosis and management the ECG finding and electrophysiological study should be exactly evaluated.

References