Coexistence of permanent junctional reciprocating tachycardia with rheumatic valvular disease; a case successfully treated with radiofrequency ablation

Radyo frekans ablasyon ile başarılı bir şekilde tedavi edilen romantizm kapak hastalığının eşlik ettiği permanent junctional reentrant taşıkardili olgu

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Abstract
A seventeen years old female had presented with palpitation. On the electrocardiography (ECG), long RP, narrow QRS tachycardia, inverted P waves in leads D2, D3 and aVF were noticed. On transthoracic echocardiography, rheumatic mitral valvular disease (RMVD) with normal ventricular systolic function was observed. Eccentric mitral regurgitation was also present. Electrophysiology study (EPS) was performed at our institution. According to EPS and surface ECG findings our diagnosis was permanent junctional reciprocating tachycardia (PJRT). Atrial activation appeared earliest and nearly ostium of the coronary sinus. The accessory pathway was detected near the coronary sinus ostium. RF ablation was performed, successfully. Taşıkardinin üçüncü saniyede sonlandığı görüldü. Permanent junctional reciprocating tachycardia can rarely coexist with RMVD. Here, we are reporting a case of PJRT and RMVD coexistence that underwent successful radiofrequency (RF) catheter ablation. This case report confirms that RF catheter ablation should be considered as the treatment of choice in adult patients with PJRT for the following reasons: catheter ablation is highly successful, the complication rate is low, and PJRT may lead to taşıkardia induced cardiomyopathy, which is reversible.

Keywords: Rheumatic mitral valvular disease, permanent junctional reciprocating tachycardia, radiofrequency catheter ablation

Introduction
Rheumatic mitral valvular disease (RMVD) causes the supraventricular arrhythmias which are atrial fibrillation, atrial flutter and atrial tachycardia. Permanent junctional reciprocating tachycardia (PJRT) can rarely coexist with rheumatic mitral valvular disease. Permanent junctional reciprocating tachycardia is a rare form of nearly incessant supraventricular tachycardia occurring predominantly in infants and children and characterized by a long RP interval and, in the typical form, by negative P-waves in leads II, III, and aVF on the surface electrocardiography (ECG). During sinus rhythm, the surface ECG is normal, without manifest pre-excitation (1,2). Here, we are reporting a case of PJRT and RMVD coexistence, who underwent successful radiofrequency (RF) catheter ablation.

Case report
A seventeen years old female had presented with palpitation and had been hospitalized at another medical center where the ECG revealed long RP, narrow QRS tachycardia. She had been on medication for supraventricular tachycardia including diltiazem. She was referred to our clinic with the preliminary diagnosis of incessant atrial flutter or atrial tachycardia. She had short episodes of palpitations since the last decade. She presented to our clinic with a palpitation episode for three days. Her blood pressure was 100/60 and she had a tachycardia with
regular pulserhythm (130 beats/min). Lung auscultation was normal. A mild pansystolic murmur was heard in the left fourth intercostal space.

On the ECG, long RP, narrow QRS tachycardia, inverted P waves in leads D2, D3 and aVF were noticed (Figure 1).

Figure 1. Electrocardiography with supraventricular tachycardia

Figure 2. Fluoroscopic image is obtained in the left anterior oblique orientation. Star: Location of accessory pathway and ablation.
Atrial flutter-waves, manifest as a “sawtooth” pattern, weren’t found in the any leads. On transathoracic echocardiography, RMVD with normal ventricular systolic function was observed. Eccentric mitral regurgitation with a velocity of 4.0 m/sec (2nd degree) was also present. She refused electrical cardioversion. Metoprolol succinate and amiodarone medication were intravenously administered to the patient, but the therapy was not successful. Electrophysiology study (EPS) was performed at our institution. Quadripolar electrode catheters were inserted percutaneously under local anesthesia after premedication, through the right femoral vein and were positioned at high right atrium and the
coronary sinus. Intracardiac electrograms and 12-lead surface ECG were continuously monitored. Standard baseline AH, HV, tachycardia cycle length (TCL), VA (recorded on surface ECG and the high right atrium catheter) intervals were obtained. During baseline EPS testing, the patient had incessant spontaneous narrow tachycardia. Measured TCL, VA and AH intervals were 462, 330 and 39 milliseconds, respectively. We excluded the atrial tachycardia by demonstrating that single ventricular extra stimuli introduced during tachycardia can reproducibly terminate the tachycardia without activating the atria; and atypical form of AV nodal re-entrant tachycardia (fast-slow or slow-slow) by demonstrating the ability to pre-excite the atria with single ventricular extra stimuli applied during tachycardia at a time when the His bundle is refractory. Atrial activation appeared earliest and nearly ostium of the coronary sinus. According to EPS and surface ECG findings our diagnosis was PJRT. Location of the AP was assessed by intracardiac mapping and successful ablation on the basis of the shortest VA interval during tachycardia. The accessory pathway (AP) was detected near the coronary sinus ostium (Figure 2). RF ablation was performed by applying RF pulses of 50 watt output and 55-60°C. Tachycardia was terminated in three seconds (Figure 3, Figure 4). RF ablation was performed for a total of 90 seconds. The scope time was 22 minutes. During RF application, catheter position was continuously monitored with fluoroscopy. PR, QRS, AH and HV intervals were 140, 95, 75 and 40 respectively. The patient was observed for 30 minutes after successful ablation, and programmed electrical stimulation was repeated to confirm that the AP was interrupted before there was conduction through the catheters. Atropine was intravenously administered to reveal AP conduction recurrence. While ventricular-atrial conduction wasn't present, no tachycardia was induced. No complication developed during the procedure and aspirin was given for the next four weeks. She was examined a month later in outpatients clinic and she did not experience any palpitation for this period.

Discussion

The main causes of mitral regurgitation are classified as degenerative (with valve prolapse) and ischemic (i.e., due to consequences of coronary disease) in developed countries, or rheumatic (in developing countries) (3). Electrical remodeling might or might not be associated with anatomic remodeling (i.e., enlargement or stretching of the atria) (4). In patients with RMVD, a state of chronic inflammation in the atrial endocardium and myocardium leads to fibrosis of the atrial muscle and contributes to atrial anatomic remodeling (5). This may be the cause of atrial arrhythmias including atrial fibrillation, atrial flutter and atrial tachycardia. Rarely, PJRT can coexist with RHD.

Classically, the permanent form of PJRT occurs in children or in young adults and is characterized by an incessant (sometimes permanent) supraventricular tachycardia. However, PJRT may be diagnosed at any age and can sometimes express itself as a paroxysmal form of supraventricular tachycardia with a long RP interval (6). Persistent tachycardia is also associated with the development of cardiac dysfunction (7). Palpitation is the most common symptom. Our patient had rheumatic mitral regurgitation but she had no cardiac dysfunction which may be due to the short episodes of tachycardia.

PJRT is an orthodromic atrioventricular (AV) re-entry tachycardia mediated by a concealed, retrogradely conducting AV AP that has slow and decremental conduction properties. Anterograde conduction of this AP is absent, so atrial fibrillation/flutter can't be caused to ventricular fibrillation. PJRT is caused by an AV re-entry using the AV node as the anterograde limb and a slowly conducting AP as the retrograde limb (1,2). The location of the AP is commonly right posteroseptal with an atrial insertion close to the ostium of the coronary sinus, but other locations have been reported (8,9). Chronic uncontrolled tachycardia has been reported to cause tachycardia-induced cardiomyopathy, which usually recovers with adequate ventricular rate control (10). As PJRT is most of the time refractory to drug therapy, RF catheter ablation of the AP has become the treatment of choice (11,12). In the vast majority of PJRT cases, the retrograde slowly conducting, decremental AP is located in the posteroseptal region, and therefore, catheter ablation is usually performed using a right-sided approach (2,6,13). Also our patient had a posteroseptal AP.

Radiofrequency catheter ablation proved to be safe and highly effective for obtaining a definitive cure in patients with PJRT whatever the location of the AP (6,14,15). Recurrences are not rare (from 13 to 23% in the literature; 8% in the present series), but long-term success is usually obtained after a second ablation procedure and ranged from 92 to 100%. The higher recurrence rate in PJRT compared with what is observed in non-decremental APs may be explained by the long, tortuous course of the AP along the AV sulcus (2). The complication rate is low, but cautious application of RF current is mandatory when the AP is located above the ostium of the coronary sinus or in the midseptal region, because the risk of second- or third-degree AV block is in the range of 5–7%. Permanent junctional reciprocating tachycardia in adults may have various clinical presentations and is often paroxysmal (53%), and the retrograde slowly conducting, decremental AP is not infrequently in a non-posteroseptal location.

Conclusion

This case report confirms that RF catheter ablation should be considered as the treatment of choice in adult patients with PJRT for the following reasons: catheter ablation is highly successful, the
complication rate is low, and PJRT may lead to tachycardia induced cardiomyopathy, which is reversible.

References

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