Atrioventricular Nodal Reentrant Tachycardia in Transplanted Heart

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Abstract

Patients after heart transplantation may develop variety of supraventricular or ventricular arrhythmias; however atrioventricular nodal reentrant tachycardia (AVNRT) is very rare. We report a case of a young patient, who underwent orthotopic heart transplantation (OHT) due to nonischemic dilated cardiomyopathy. 10 years after transplantation he began to perceive palpitations and on outpatient visit an AVNRT has been recorded on standard 12-lead ECG. Patient underwent electrophysiology study (EPS) where AVNRT was induced and successful radiofrequency catheter ablation (RFCA) has been performed. Currently, patient is symptoms free at 3 years follow up.

Keywords: Atrioventricular nodal reentrant tachycardia; Heart transplantation; Ablation

Introduction

Supraventricular arrhythmias in transplanted hearts are not rare. Most common is atrial flutter, followed by atrial tachycardia [1]. Until now, there are only few cases demonstrating atrioventricular nodal reentrant tachycardia (AVNRT) in patients who underwent heart transplantation and subsequently radiofrequency catheter ablation (RFCA) of arrhythmia [2-5]. We present a case of a patient, who underwent successful ablation of AVNRT after orthotopic heart transplantation (OHT).

Case Report

18-year-old male patient with history of non-ischemic dilated cardiomyopathy with severe systolic dysfunction, after acute decompensation of heart failure with need of cardiopulmonary resuscitation underwent standard OHT with bilateral anastomoses. He was treated with combination of immunosuppressant’s which comprised of cyclosporine, mycophenolate mofetil and prednisone. Levels of cyclosporine were within therapeutic ranges, endomyocardial biopsies were without any signs of rejection. Invasive coronary angiography and multidetector computed tomography (MDCT) of coronary arteries did not reveal any obstructive lesions. Regular echocardiography measurements showed normal left ventricular ejection fraction, without left ventricular hypertrophy or dilation of cardiac chambers.

10 years after heart transplantation, patient began to perceive occasional palpitations without syncope. During regular outpatient visit at his cardiologist, patient presented with palpitations. The 12-lead ECG where supraventricular arrhythmia of narrow QRS complexes with frequency of 170 bpm with visible retrograde P waves was recorded (Figure 1). During right carotid sinus massage, arrhythmia was promptly terminated to sinus rhythm.

Patient underwent invasive electrophysiological study (EPS) (Figure 2). The donor heart was in sinus rhythm with frequency of 80 bpm, His-ventricular interval was 65 ms. During programmed atrial stimulation with progressively increased prematurity, at 600/280 ms there was a borderline, Atrio-His jump of 50 ms, with an atrial echo, confirming dual nodal physiology. During atrial stimulation train of 600/260 ms, an atrioventricular conduction block occurred at cycle length of 360 ms (167 bpm). After infusion of isoprenaline an AVNRT with cycle length of 315 ms (190 bpm) was induced and ablation of...
slow pathway was performed with accelerated junctional rhythm response. There was no inducible AVNRT after ablation.

Figure 2: Electroanatomical map from En-Site system with region of interest for RFCA ablation of AVNRT. RFCA: Radiofrequency catheter ablation; AVNRT: Atrioventricular nodal reentrant tachycardia.

After 3 years of follow up patient is a symptom free, without palpitations. Last MDCT coronary angiography in 2016 (Figure 3) documented calcium score of 11 without any significant coronary artery stenoses.

Figure 3: MDCT coronary angiogram showing no obstructive lesions. MDCT: Multidetector computed tomography.

Discussion

To our knowledge, there are only four published cases of patients with OHT who underwent successful RFCA of AVNRT, without recurrence in the follow up period [2-5]. There have been reports of increased incidence of arrhythmias in transplanted hearts related to rejection [1]. However in our patient there were no signs of rejection at all.

In transplanted hearts, factors such as altered anatomy, altered autonomic function, allograft rejection and transplant coronary artery disease contribute to development of arrhythmias [6]. Some other major pathophysiological aspects of supraventricular arrhythmias have been recently described. Xie et al. suggested the role of atrial type 2 ryanodine receptor in promotion of atrial fibrillation, mainly through its oxidation by mitochondrial derived reactive oxygen species, which results in subsequent intracellular Ca²⁺ leak [7]. Another work from D´Ascia et al. showed significantly higher risk of development of atrial fibrillation in non-responders of cardiac resynchronization therapy. Authors suggest that in responders, reverse remodeling of left ventricle improves its systolic function, decreases degree of mitral regurgitation and reduces structural atrial remodeling, which contributes to development of atrial arrhythmias [8].

After transplantation, the heart is completely denervated. Our patient during visit where AVNRT was recorded on ECG, converted back to sinus rhythm after carotid sinus massage, which might suggest partial cardiac reinnervation. There has been study which suggests parasympathetic reinnervation of efferent nerve fibers in transplanted hearts based on presence of vagal reflexes in such patients [9].

There was no knowledge of arrhythmia or history of palpitation occurrence in the donor and interesting is that AVNRT was documented after 10 years which is relatively long period with its subsequent interruption by non-pharmacological approach – carotid sinus massage. Our case report confirms what previous cases have proved [1-5,10], that RFCA in transplanted heart can be safely performed, without additional risk in comparison with general population.

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