

A Rare Origin of Left Atrial Ectopic Tachycardia: A Case Report

Ata KIRILMAZ, M.D., Kürşad ERİNÇ, M.D., Ersoy IŞIK, M.D., Ertan DEMİRTAŞ, M.D.
Military Medical Academy of Gülhane, Department of Cardiology, Ankara - Turkey

NÂDİR LOKALİZASYONLU BİR SOL ATRİYAL EKTOPIK TAŞIKARDİ OLGUSU: OLGU SUNUMU

ÖZET

Mitral anulüsünden kaynaklanan sol atriyal taşikardi, kardiyomiyopatiye neden olabilen ender bir taşiaritmidir. Sol ventrikül sistolik fonksiyonları azalmış 20 yaşındaki hastada mitral annulus üzerinde başarılı bir şekilde ablata edilen sol atriyal taşikardi olgusu sunulmuştur.

Anahtar kelimeler: Ablasyon, sol atriyal taşikardi, kardiyomiyopati

Atrial ectopic tachycardia is an uncommon cause of incessant supraventricular tachycardia, which may result in cardiomyopathy (1,2). The cellular mechanisms can be abnormal automaticity, triggered activity or reentry, but clinical behavior seems to be most consistent with increased automaticity from a single atrial focus (3). Although it may resolve spontaneously in a small number of patients (4), the majority of incessant atrial tachycardia will eventually require therapy. Crista terminalis, right and left atrial appendices, orifices of pulmonary veins, coronary sinus ostium and interatrial septum are among the most common sites of origin. Radiofrequency (RF) catheter ablation of these foci is associated with a high success rate and few complications (5-8). This report describes a successful ablation of a left atrial ectopic tachycardia originating from the mitral annulus.

CASE REPORT

A 20 year-old male has presented to our clinic with symptoms of palpitation and dyspnea on exertion since the last 6 months. Physical examination was significant for a mild systolic murmur over the mitral area. His 12 lead ECG (Figure-1A) revealed a supraventricular tachycardia with 2:1 atrioventricular (AV) block with a positive P wave configuration in inferior leads (II, III, aVF) and a negative

configuration in aVL and I. V1 revealed a sharp positive P waves. Although the patient did not have any palpitation during the tachycardia with 2:1 AV block, he felt palpitation following 1:1 ventricular response (Figure 1B) secondary to improved AV conduction with minimal effort. A transthoracic Doppler echocardiography revealed a mild mitral and tricuspid regurgitation, reduced left ventricular EF (44%) with a slightly increased left ventricular diameters (65/45 mm in diastole/systole) and mild pulmonary hypertension (pulmonary arterial systolic pressure: 35 mmHg).

He was considered eligible for RF ablation since he refused to be on any medication. Positive P wave in lead V1 and negative P wave configuration in leads aVL and DI supported a left atrial localization of the tachycardia (9). The procedure was performed in a postabsorptive state with mild sedation with fentanyl and midazolam after obtaining informed consent. A crescent halo catheter with 32 unipolar electrodes (16 bipolar electrograms) was placed posterior to the tricuspid annulus along the crista terminalis and interatrial septum (Figure 2A and 2B). A decapolar diagnostic and a 8 mm tipped roving catheter was positioned to His and the tricuspid annulus-inferior vena cava (TA-IVC) isthmus, respectively to start with. AV block was produced by giving verapamil 5 mg intravenously in order to delineate P wave configuration excluding ventricles from being a part of the tachycardia. Postpacing interval following the entrainment of the tachycardia from TA-IVC isthmus was 570 ms, excluding typical atrial flutter. The first right atrial activity was recorded from the electrode pairs numbered 21-22, which was located next to the mid interatrial septum and was following the surface P wave during the tachycardia (First 3 beats in figure-3). Tachycardia cycle length changed between 332-280 ms during the study with a stable relationship between intracardiac electrograms and surface P waves. Roving the ablation catheter in the right atrium did not disclose an atrial activation preceding the P wave. The left atrial mapping performed with the ablation catheter through the patent foramen ovale revealed the earliest atrial activation anterolateral on the mitral annulus (Figures 2A, 2B and 3). Vigorous attempts of overdrive pacing were unsuccessful in terminating the tachycardia. The catheter manipulation on this area terminated the tachycardia transiently and the application of the RF energy was resulted in elimination of the atrial tachycardia and in restoration of sinus rhythm (Figure 3). The ratio of A and V electrogram amplitudes (1/1) recorded from the ablation catheter on successful site during the tachycardia and the sinus rhythm following the termination during radiofrequency application also confirms the annular localization of the focus (Figure-3). Programmed atrial stimulation up to 2 extrastimuli and burst pacing did not induce any tachycardia.

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Corresponding author: Dr. Ata Kirilmaz, Gülhane Military Medical Academy, Department of Cardiology 06018 Etlik-Ankara, Turkey
Tlf: +90 (312) 304 4252 - 330 7496 Fax: +90 (312) 210 1110

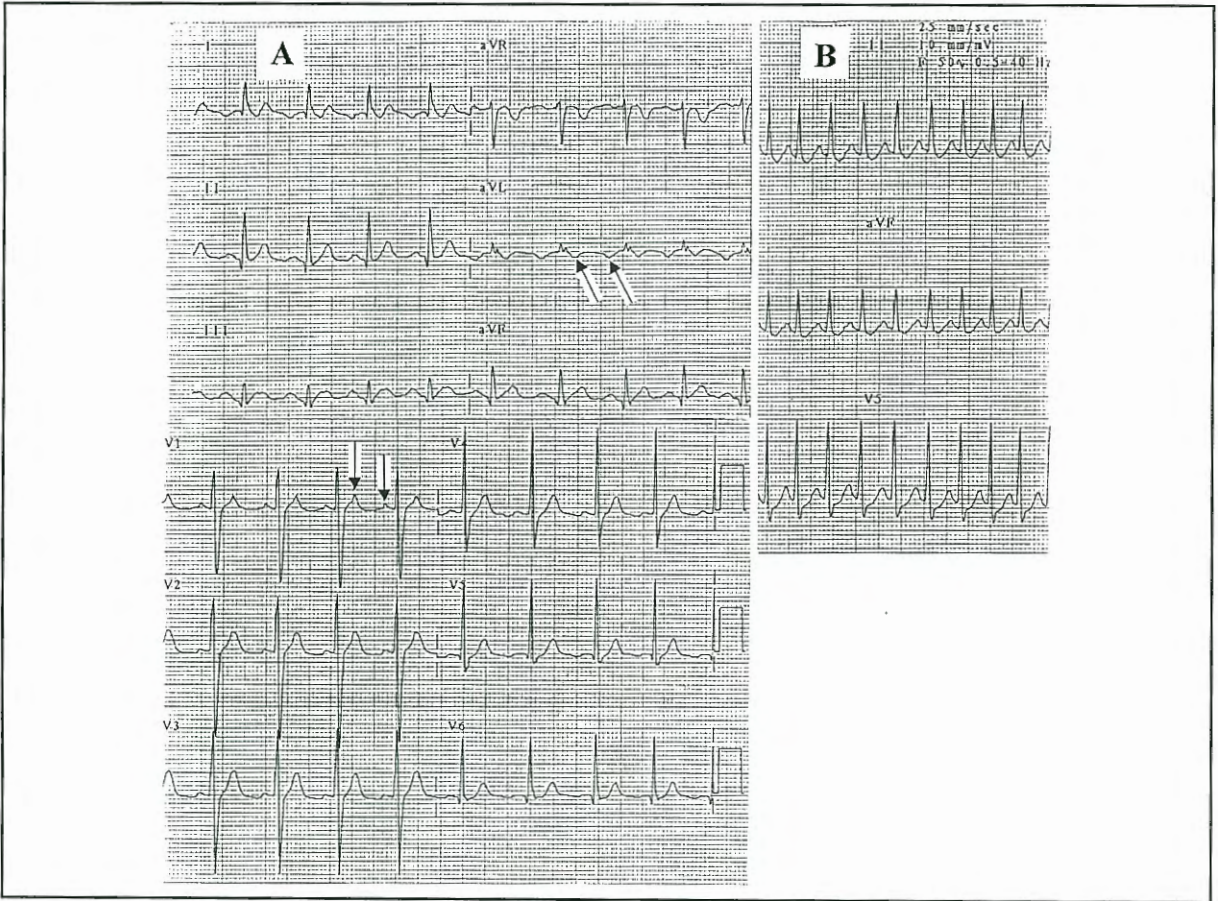


Figure 1. A. The presenting 12 lead ECG of atrial tachycardia. The arrows indicate the P waves with a pp interval of 280 msec. B. 1:1 AV conduction following minimal effort

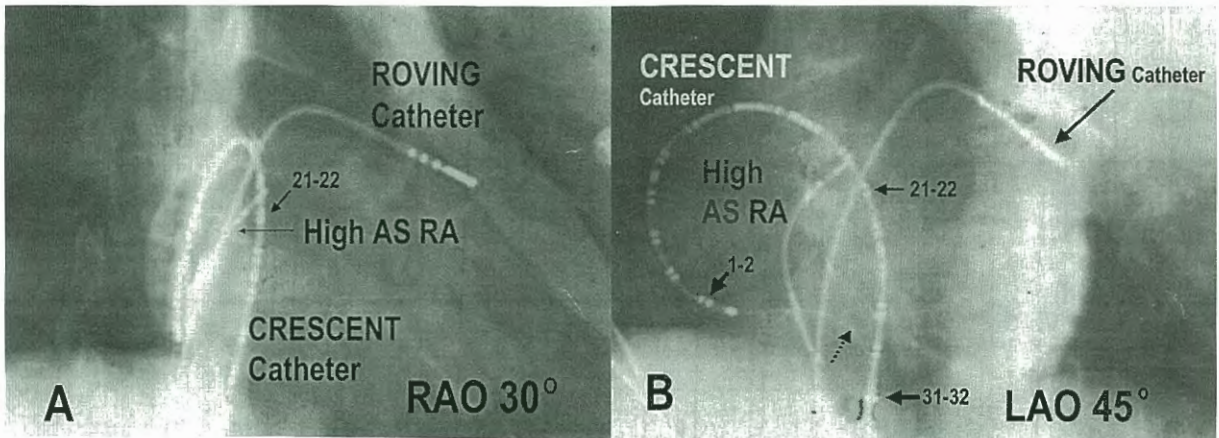


Figure 2. Radiograms at the site of successful ablation. A. RAO: Right Anterior Oblique projection, high AS RA: High Anteroseptal Right Atrium. B. LAO: Left Anterior Oblique projection; Dashed arrow indicates the string of the crescent catheter which gives a halo shape upon pulling (some parts of the pictures were sharpened and radiopaque ECG cables were extracted digitally).

DISCUSSION

Atrial tachycardia originating from the mitral annulus was first published by Mallavarapu et al. (10) in two cases and the mechanism responsible for the tachycardias was best explained by reentry. Nogami

A. et al. (11) reported the third case in which the tachycardia was located on the mitral annulus. The characteristics of their case have resembled an accessory AV node not electrically connected to the ventricle and reentry as the possible mechanism.



Figure 3. Termination of the tachycardia during RF energy application. The electrogram recorded from the distal electrode pairs of the ablation catheter precedes the surface P wave by 37 ms. A and V ratio is approximately equal during the tachycardia and the sinus rhythm which indicates that the ablation catheter is on the mitral annulus. V1 was amplified and clipped to visualize the P wave. C: Crescent catheter A: Atrial electrogram, V: Ventricular electrogram, ABL ds: Distal electrode pair of the ablation catheter.

We present this case as the fourth one in the literature in English. In our case, vigorous attempts of entrainment and of overdrive suppression the tachycardia without termination, and immediate initiation of the tachycardia without preceding any atrial ectopy following catheter-induced transient termination all favor for increased automaticity or triggered activity as the cellular mechanism. The patient was in sinus rhythm on follow-up and the left ventricular diameters were slightly improved in 3 months.

This case represents a rare origin of left atrial tachycardia possibly with a cellular mechanism other than reentry.

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