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Address for Correspondence/Yazışma Adresi: Vincenzo Russo, MD
Second University of Naples, Chair of Cardiology, Naples-Italy
Phone: 0039 0817062355
E-mail: v.p.russo@libero.it
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Successful catheter ablation of symptomatic premature ventricular contractions originating from mitral annulus

Mitral anülüsten kaynaklanan semptomatik ventriküler erken atımların başarılı kateter ablasyonu

Introduction

Premature ventricular contractions (PVC)/ventricular tachycardia's (VT) rarely originate from mitral annulus (1). In these cases, radiofrequency catheter ablation (RFCA) is an important treatment option.

Herein, we present a patient with PVC refractory to medical therapy, who was successfully treated with RFCA.

Case Report

A 20-year-old male patient was admitted to our department with the complaint of palpitation. He had been having palpitations for 4 years. Medical treatment with calcium channel blocker and beta-blocker was unsuccessful. Physical examination of cardiovascular and other systems was normal. Resting electrocardiogram (ECG) showed PVCs with a right bundle branch block morphology and inferior axis (Fig.1). QRS notching was observed in the inferior leads of the PVCs. Exercise ECG and transthoracic echocardiography were within the normal range. The monomorphic PVCs (8000 beats/day) were detected in Holter ECG. Electrophysiological study was performed. Programmed ventricular stimulation did not induce ventricular tachycardia. Electrophysiological mapping was performed during PVCs. During PVC, the earliest ventricular activation was seen in the distal electrode of the coronary sinus. After placing the steerable 4-mm-tip ablation catheter (Mariner; Medtronic, Minneapolis, MN, USA) to the left ventricle with retrograde aortic approach, mapping of the aortic cusps and left ventricle outflow tract was performed. Early activation site was not detected at the aortic cusp and left ventricle outflow tract. With left ventricular mapping, earliest ventricular activity during PVCs was recorded in the anterolateral of the mitral annulus. In this site during the PVC, local ventricular

activation preceded the QRS onset by 28 ms (Fig. 2). Radiofrequency ablation applied to this site and PVCs disappeared (Fig. 3). PVCs were not observed during follow-up examinations at second month.

Discussion

Idiopathic PVCs mostly originate from ventricular outflow tracts. RFCA is successfully performed to these sites. Premature ventricular

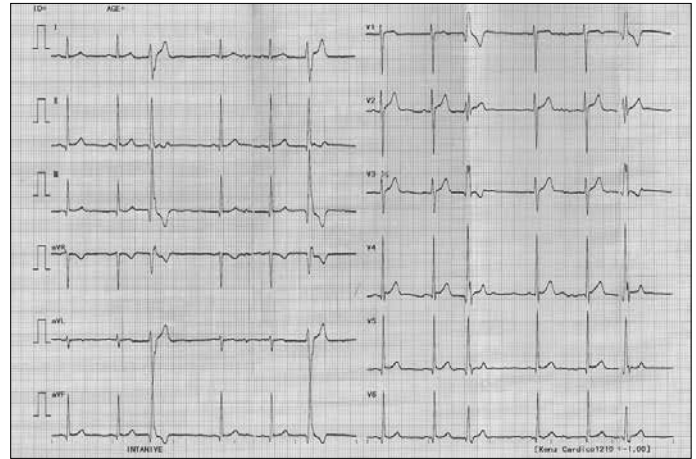


Figure 1. Surface ECG of the patient with premature ventricular contractions

ECG - electrocardiogram

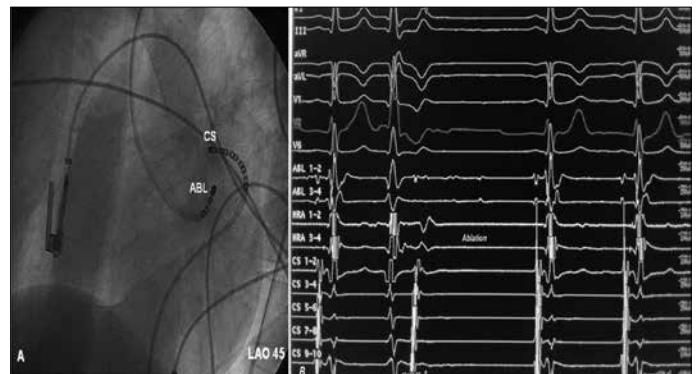


Figure 2. A) Left anterior oblique position, current catheter positions are shown while premature ventricular contractions disappeared. B) Surface and intracardiac electrograms recording at the site of successful ablation of the premature ventricular contractions

ABL-ablation, CS- coronary sinus



Figure 3. Premature ventricular contractions are not observed on the ECG after the procedure

ECG - electrocardiogram

contraction/ventricular tachycardia's originating from mitral annulus are rarely reported (1).

PVCs arising from the mitral annulus frequently originate from anterolateral, posteroseptal and posterior sites (2). It has been reported that 2/3 of the PVCs arising from the mitral annulus originate from anterolateral site (2). Furthermore, small part of these arrhythmias originates from the anteroseptal site of the mitral annulus. Ablation of this site may be technically very challenging. Cases have been reported that successful catheter ablation of the premature ventricular contraction origin from the anteroseptal site of the mitral annulus can be performed either by a transeptal or transaortic approach in literature (3, 4). Anterolateral site of the mitral annulus is in close proximity to anterior of the right ventricle outflow tract, left ventricular epicardium near to the left sinus Valsalva and subvalvular region of the left ventricular outflow tract. Idiopathic PVC/VTs frequently originates from these sites that support this theory (5). In our case, early activation sites are not detected at the aortic root region and left ventricle outflow tract. By mapping of the left ventricle, at the time of PVCs, earliest ventricular activity is recorded in the anterolateral of the mitral annulus. In this site during the PVC, local ventricular activation preceded the QRS onset by 28 ms, when radiofrequency ablation applied to this site, PVCs immediately disappeared.

Adequate analysis of characteristics of ECG helps to determine the origin of mitral annulus sourced PVC/VT and may shorten the duration of the electrophysiological study. While the PVCs originating from anterolateral of mitral annulus has inferior axis, those originating from posterior annulus has superior axis. While QRS polarity in DI and aVL leads of PVCs originating from anterior annulus is negative, those originating from posterior annulus have positive QRS polarity in DI and aVL leads. Additionally it is shown that all the patients ECGs with mitral annulus originated PVC/VTs have s waves in lead V6 (2). In our case, ventricular premature contractions showed right bundle branch block pattern. Derivasyon lead (DI) showed rS pattern, V6 lead had an s wave and inferior axis. QRS notching in the inferior leads supported anterolateral origin. All these ECG findings showed that premature contractions were originating from anterolateral site of mitral annulus.

Conclusion

Premature ventricular contraction with right bundle branch block pattern can originates from mitral annulus. Medical therapy is the treatment of choice in these patients. Radiofrequency catheter ablation should be considered in patients' refractory to medical therapy.

Ömer Uz, Fethi Kılıçaslan, Mehmet Tezcan
Department of Cardiology, Gülhane Military
Medical Academy, Haydarpaşa, İstanbul-Turkey

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Address for Correspondence/Yazışma Adresi: Dr. Ömer Uz
Gülhane Askeri Tıp Akademisi Haydarpaşa, Kardiyoloji Kliniği, İstanbul-Türkiye
Phone: +90 216 542 34 65 Fax: +90 216 348 78 80
E-mail: homeruz@yahoo.com

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Prolonged asystole during hypobaric chamber training

Açık basınç ortamında oluşan hipokside uzamış asistoli

Introduction

An asystole, defined as the absence of myocardial electrical activity (1), is a state, which may occur due to acute hypobaric hypoxia. It can be seen even in completely healthy individuals (2) and may cause hazardous results compromising flight safety. Asystole is usually associated with an organic heart disease; coronary heart disease, myocardial infarction, myocarditis, congenital heart diseases, hypoxia, acidosis, hypo-hyperkalemia.

Case Report

A 36-year-old, male helicopter pilot was taken to hypobaric chamber training. His electrocardiography, chest X-Ray and biochemical parameters revealed to be completely normal. He had no history of syncope or presyncope. He was exposed to hypobaric environment for about one hour including 5 minutes staying at a simulated altitude of 30.000 feet. On the 47th minute from the training onset, the pilot had nausea, vomiting, excessive sweating, and loss of positional awareness symptoms and finally lost his consciousness on the 46th second after the mask off. The training was stopped and the pilot was assessed for emergency treatment by the internal observer. He was taken the oxygen mask on and regained his consciousness while being placed in Trendelenburg position. After the training the subject was re-examined by cardiologist and his vital values, electrocardiography, echocardiography and head-up tilt tests were normal. During this event an ambulatory blood pressure monitoring (ABP) and 12-lead rhythm monitoring were being performed for a planned study, although it was not a routine assessment. In his Holter recordings there were no signs of arrhythmia, however asystole lasting 16 second followed by a sinus bradycardia lasting 10 second were seen on the monitor (Fig. 1) (Video 1. See corresponding video/movie images at www.anakarder.com). The heart rate, ABP and heart rate variability (HRV) parameters of the subject were recorded (Table 1, 2). Due to the absence of any complaint of performing the daily activities further researches including electrophysiology study were not conducted. Two months after discharge,