Case Report

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Internal Medicine Section

Mapping and Ablation of Premature Atrial Contractions Originating from the Posterior Mitral Annulus: A Case Report

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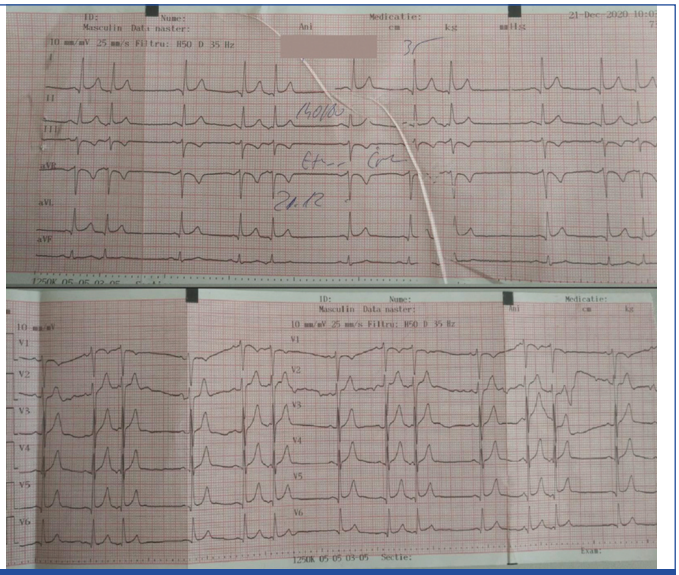
ABSTRACT

Premature Atrial Contractions (PAC) are frequent arrhythmias. Previously, regarded as a benign electrocardiographic finding, they are now linked to adverse outcomes such as, stroke and all-cause mortality. Furthermore, a high burden of PACs >1.500/24 hours has a predicted probability of atrial fibrillation occurrence. Herein, the authors presented a case of a 35-year-old male patient, with high burden PACs, originating in the posterior mitral annulus. Treatment with class IC (flecainide, propafenone) antiarrhythmic drugs and beta blockers (bisoprolol) was ineffective in controlling the tachycardia, therefore, catheter ablation was performed. The procedure was performed using the Three-Dimensional (3D) Biosense Webster CARTO 3 electroanatomical mapping system. The area of the earliest atrial signal was located at the posterior mitral annulus. Successful elimination of the ectopy was obtained with Radiofrequency (RF) application on the posterior mitral ring. A single RF application of 30 W for 60 seconds abolished PACs, with no further recurrence. Holter Electrocardiogram (ECG) showed, no PACs at one, six and 12 months follow-up. Catheter ablation remains an effective approach to cure the arrhythmia, when medical treatment with antiarrhythmic drugs is ineffective or undesirable in patients with high burden PACs.

Keywords: Atrial premature contractions, Cardiac mapping, Electrophysiological study, Mitral valve, Radiofrequency ablation

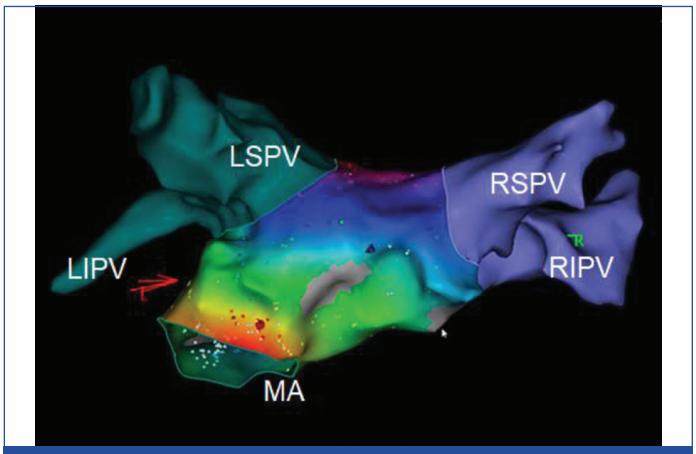
CASE REPORT

A 35-year-old male patient, with frequent PACs manifested by palpitations that started three years before, was hospitalised for Electrophysiology (EP) study and catheter ablation. One year before his presentation, was diagnosed with high burden PACs 28000/24 hours with an average cycle length of sinus rhythm of 750 ms. Despite treatment with oral flecainide 300 mg/day (100 mg every eight hours) and beta blockers (bisoprolol 2,5 mg), symptoms persisted, with 17.000 PACs/24 hours. Therefore, catheter ablation was proposed. At presentation, the patient's heart rate was 85 Beats Per Minute (bpm) with a Blood Pressure (BP) of 130/70 mmHg, without any signs of heart failure. The ECG showed PACs with a positive P wave in lead V1, suggestive of left atrial origin. The morphology of the P wave was also negative in the inferior leads suggestive of an origin in the inferior part of the left atrium [Table/Fig-1].

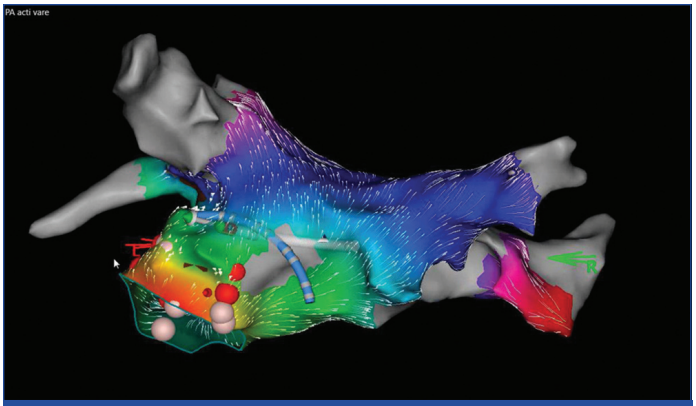


[Table/Fig-1]: 12-lead ECG shows the morphology of P waves in PACs: negative in inferior leads: II, III and AVf, biphasic and low amplitude in V1.
AVf: Augmented vector foot

After the patient gave informed written consent, EP study was performed using standard right and left heart catheterisation techniques. One quadripolar electrode catheter was placed at the level of the bundle of His, a decapolar electrode catheter placed inside the coronary sinus and the ablation catheter inside the right atrium, followed by mitral ring mapping through a trans-septal approach. Using the Claris Saint Jude EP system, tracings from surface ECG leads (I, III, {augmented Vector Left (aVL)}, and V2) and bipolar intracardiac electrograms {His, Coronary Sinus (CS) and Ablation (Abl)} filtered at a bandpass of 50-300 Hz were displayed on the screen for exact measurements using callipers [Table/Fig-2]. A 3D mapping system (CARTO 3 Biosense Webster) was used for exact localisation of the atrial focus. During atrial ectopy, the onset of the earliest endocardial signal was mapped. Activation mapping was started inside the right atrium, then trans-septal puncture mapping was performed. The area of the earliest atrial signal was located at the posterior mitral annulus [Table/Fig-2,3].

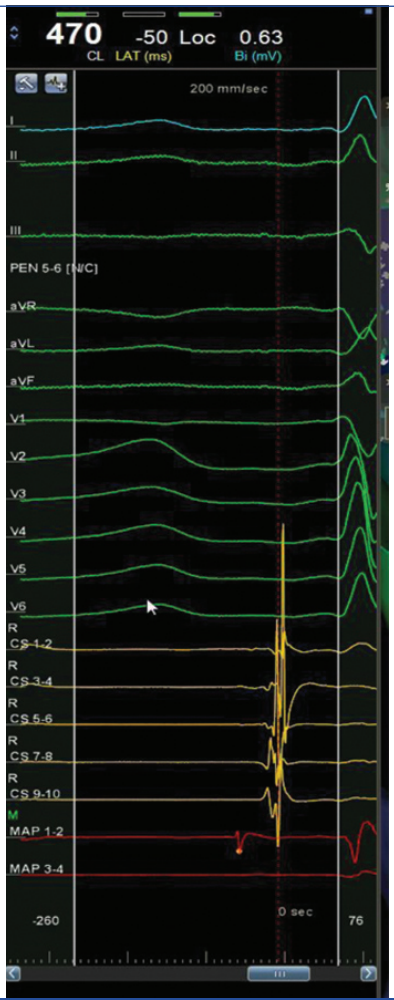


[Table/Fig-2]: Activation map using the 3D CARTO 3 mapping system, displays radial activation of the left atrium beginning at the posterior mitral annulus and extending to the posterior atrial wall and pulmonary veins. Red indicates the earliest activated zone, while purple indicates the most latest.
LSPV: Left superior pulmonary vein; RSPV: Right superior pulmonary vein; LIPV: Left inferior pulmonary vein; RIPV: Right inferior pulmonary vein; MA: Mitral annulus



[Table/Fig-3]: Vector map using the 3D CARTO 3 mapping system: The conduction velocity across the posterior wall of the left atrium is precisely depicted on the vector map; In addition, radial propagation from the posterior mitral annulus to the pulmonary veins.

The catheter tip placed on the posterior mitral ring demonstrated the characteristic annular motion, when viewed from right and left anterior oblique fluoroscopic views, with the catheter motion synchronised to the motion of the CS catheter in both fluoroscopic projections. Furthermore, atrial to ventricular ratio was <1 with a ventricular electrogram >0.5 mV at the site [Table/Fig-4]. Successful elimination of the ectopy was obtained with RF application at this site. RF ablation was carried out at the point of earliest endocardial activity in the power control mode, with a maximum temperature of 60°C and a target power of 30 W. The catheter utilised was an irrigated tip SmartTouch deflectable catheter, with a 4 mm distal electrode used to deliver RF current from the distal electrode to a large skin electrode, positioned on the posterior thorax of the patient. A single RF application 30 W, 60 seconds at the posterior mitral annulus abolished PACs with no recurrence after atropine injection. To confirm the success of the procedure, a 30 minute waiting period was used, ectopy being absent during this period of time. The patient was free of symptoms and the 12-lead ECG showed, absence of PACs. Furthermore, Holter ECG showed no PACs at one, six, and 12 months follow-up.



[Table/Fig-4]: Intracardiac electrograms illustrate the local potential at the ablation location. Please note the signal on the ablation catheter (Map) that is 15 ms prior the onset of the surface P wave. Map 1-2 ablation catheter, distal electrodes. Map 3-4 ablation catheter, proximal electrodes. CS 1-2 to CS 9-10 coronary sinus catheter-distal to proximal electrodes.

DISCUSSION

The PACs are arrhythmias, characterised by abnormal electrical impulses that originate in the right or left atrium. The mechanism of PACs may be re-entrant or focal. Focal PACs are characterised by radial spread in all atrial directions from a single site of initial atrial activation. The most common site of origin of left PACs or atrial tachycardia are the pulmonary veins, left atrial appendage and mitral annulus [1]. PACs are frequent findings in patients with and without structural heart disease and may be symptomatic and refractory to antiarrhythmics [2,3]. Previously regarded as a benign ECG finding, they are now linked to adverse outcomes such as, stroke and all-cause mortality [4]. In the Event Monitoring Belt for Recording Atrial Fibrillation After a Cerebral Ischaemic Event (EMBRACE) study, a high burden of PACs >1,500/24 hours was associated with a 40% predicted risk of atrial fibrillation [5]. Without proper treatment, frequent PACs may induce tachycardia-induced cardiomyopathy [6-8]. To date, no guidelines or expert consensus provide recommendations for catheter ablation of PACs. In the systematic review and meta-analysis by Meng L et al., high burden was defined by a lower threshold of >30 PACs per hour and/or runs of 20 PACs or >218 PACs per 24 hours [9]. However, increased PAC burden without documented Atrial Fibrillation (AF) is not an indication for oral anticoagulation. The index patient had 28.000 PACs/24 hours, and despite class IC antiarrhythmic drugs and beta-blockers, the patient remained symptomatic with 17.000 PACs/24 hours. A high burden PACs may be associated with PAC-induced cardiomyopathy, dilation of the left ventricle and decrease in the ejection fraction [6-8]. Electroanatomical mapping can determine the precise location of PAC-inducing focus [10]. Although, a recent study has indicated a distribution of Atrial Tachycardia (AT) foci around the tricuspid annulus, only isolated occurrences of AT originating from the mitral annulus have been documented [11].

The surface ECG P wave morphology is able to provide a general indication of PAC origin. Prior research determined that, a positive P wave in lead V1 and a negative P wave in lead aVL were very specific for a focus in the left atrium [12]. Furthermore, in a recent research of the paced P wave morphology from the four PVs in 30 patients, lead V1 invariably displayed an upright shape [13]. Moreover, in the study of Kistler PM et al., with mitral annulus tachycardia, the P wave was biphasic (negative followed by positive) in the precordial leads and had low amplitude in the limb leads [14]. The index patient had a biphasic low amplitude P wave in lead V1 with superior axis (negative P wave in the inferior leads). PACs can originate from various structures of the right or left atrium. The most common origins of the left atrium are the pulmonary veins, the left atrial appendage, and the mitral annulus. Mitral annulus is a fibrous structure which is surrounded by atrial myocardium that is specialised and distinct from left and right atrial myocytes as suggested by McGuire MA et al., [15]. Cells around the tricuspid annulus have “nodal-like” properties, lack connexin 43 expression and respond to adenosine administration [16]. Wit AL et al., noticed that, the anterior leaflet of the mitral valve includes muscle fibres in direct continuity with the left atrial myocardium above the fibrous annulus [17]. These muscle fibres displayed “nodal-like” action potentials, initiating impulses that might propagate to the Left Atrium (LA). Nonetheless, the junctional region of the mitral annulus is a common site of conduction block, so potentially providing the substrate for reentry initiation.

Electroanatomical systems are used for PAC mapping and ablation. A mitral annular origin should be considered for PAC, when the earliest right atrial activity is detected in the para-Hisian area (about 0 to 20 ms before P wave onset) and if the P wave morphology exhibits the aforementioned characteristics [14]. Local potentials at the target sites region show evidence of fractionated or sharp potentials, as reported by Kay GN et al., [18]. However, fractionated potentials at the target sites were only found by Liuba I and Walfridsson H in 4 of 35 focal ATs (11%) [19]. The research of Wang Y et al., revealed that, 70% of the areas, where focal tachycardias occurred, had fractionated or complex potential [20]. In the index patient, the author's have discovered a sharp potential that occurred 15 milliseconds, before the beginning of the P wave. Catheter ablation at this location eradicated PACs, preventing other recurrences.

CONCLUSION(S)

In the present case, successful elimination of the PACs was obtained with RF application on the posterior mitral ring. When medical treatment with antiarrhythmic drugs is ineffective or undesirable in patients with high burden PACs, catheter ablation remains an effective technique to cure the arrhythmia upon detailed mapping and identification of the arrhythmia's source.

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