Catheter ablation of atrial tachycardia after interatrial defect repair with patch apposition

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Abstract. – A 54-year-old woman with history of septal atrial mixoma surgically treated and drug-refractory supraventricular tachyarrhythmia underwent catheter ablation of macro-reentry areas near the pericardial patch placed to repair an interatrial defect. The use of ablative therapy has been successful to cure this arrhythmia.

Key Words:
Catheter ablation, Electro-anatomic mapping, Supraventricular tachyarrhythmia, Cavo-tricuspid isthmus, Pericardial patch.

Introduction

The frequency of premature or delayed supraventricular tachyarrhythmias in patients who underwent cardiac surgery for congenital or acquired cardiovascular diseases is greater than the general population indexed for age and gender. This increase is associated with greater morbidity and mortality. It is generally assumed that the electrophysiological mechanism involved in delayed is a macro-reentry around a scar. This type of supraventricular arrhythmias is usually paroxysmal but sometimes becomes recurrent and refractory to usual pharmacological therapies. In such cases the current approach is the ablative therapy. The article describes the case of a 54-year-old woman affected by recurring episodes of “incisional” atrial tachycardia unresponsive to usual mono- and multidrug pharmacological therapy.

Case Report

A 54-year-old woman, weighting 78 kg, with family history for coronary artery disease, dyslipidemia and hypothyroidism treated with levothyroxine 75 mg/die has been admitted for recurrent episodes of palpitations. A transesophageal echocardiogram (TEE), performed after two thrombo-embolic events at age 36, demonstrated an atrial irregularly bordered mass through the foramen ovale, sizing 2.6 × 1.8 cm in the right atrium and 4.7 × 1.3 cm in the left atrium. The mass was moving during the cardiac cycle and its caudal fragment in the left atrium wedged in the atrio-ventricular inflow canal developing a functional stenosis and a little left-to-right shunt at the fossa ovalis level.

She underwent surgery in order to remove the mass and the residual interatrial defect was repaired with a pericardial patch. Histology diagnosed the mass to be a hourglass mixoma.

Since age 42, the patient had recurrent episodes of palpitations, with symptoms of low cardiac output, diagnosed to be high ventricular rate (mean >170 bpm) paroxysmal atrial fibrillation, initially treated with pharmacological therapy and then with external cardioversion. She started an anti-arrhythmic drug therapy with amiodarone, propafenone, sotalol and flecainide initially in monotherapy, then combined (propafenone plus sotalol, flecainide plus sotalol). However, the patient remained symptomatic for palpitations, dizziness, algid perspiration and pollachiuria with many hospitalizations.

Instrumental Exams

Twelve-lead ECG at admission showed a narrow QRS tachycardia with a mean ventricular rate of 150 bpm; a pharmacological treatment with propafenone 750 mg daily, sotalol 80 mg daily and enoxaparin sodium 4000 IU every twelve hours was started.

A TEE with 3D reconstruction of both atria and atrio-ventricular junctions showed a normal contractile global and segmental left ventricular function (EF 60%) and, normal dimensions of the ex-
plored cardiac chambers; the inter-atrial septal study proved the absence of a shunt with color-doppler examination even after the infusion of saline solution and Valsalva manoeuvre. The patch (2 × 1.8 cm) in fossa ovalis over the treated defect was present (Figure 1).

ECG Holter monitoring showed a continuous supraventricular tachycardia, with variable atrio-ventricular conduction and a mean heart rate of 100 bpm (min. 71 bpm, max. 174 bpm).

After interrupting for 3 days all anti-arrhythmic drugs, she underwent an electrophysiological study. The following catheters were positioned through the femoral veins: decapolar Livewire in the right atrium, quadripolar Josephson in the right ventricular apex, quadripolar Courand at the His bundle, decapolar CSL in the coronary sinus (St. Jude Medical, St. Paul, MN, USA).

At baseline, a narrow QRS tachycardia with mean ventricular rate of 145-160 bpm was recorded. During catheter positioning near the inter-atrial septum, recovery of sinus rhythm with heart rate of 65 bpm was observed; baseline intervals were AH 110 ms, and HV 46 ms.

A right atrium burst at 220 ms induced (without AH jump) the same tachycardia with cycle length of 376 ms, atrio-ventricular conduction 1:1 and ventriculo-atrial conduction time of 194 ms.

Entrainment mapping of the tachycardia at 350 ms in the right atrium at the posterior-lateral-inferior wall near the pericardial patch showed a post-pacing interval <30 ms. The 3D electro-anatomic mapping (EnSite, St. Jude Medical, St. Paul, MN, USA) confirmed the location of a critical macro-reentry isthmus. RF ablation was performed from the anterior-septal aspect of the pericardial patch to the inferior vena cava with the interruption of the tachycardia during RF application.

Finally, atrial burst at 220 ms induced a cavo-tricuspid isthmus dependent atrial flutter, that was promptly ablated showing a transition from right to left atrial flutter with 323 ms cycle length, mean ventricular rate of 92 bpm, and 2:1 atrio-ventricular conduction.

In the following days, there was no evidence of supraventricular arrhythmias on ECGs and the patient was discharged with a prophylactic anti-arrhythmic therapy for the next three months: Flecainide 100 mg daily, Sotalol 80 mg daily; Acenocumarol (INR range 2-3), ω3 fatty acids ethyl esters 3 gr daily and Atorvastatin 80 mg daily were administered for 30 days.
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Figure 2. Illustration of right intra-atrial 3D electro-anatomic mapping (EnSite).

At 1, 3, 12 months follow-up after the procedure the patient remained asymptomatic for palpitations, dizziness and dyspnoea with 72-hour EKG Holter monitoring showing sinus rhythm, rare single and coupled premature atrial beats, and no tachycardia.

Discussion

Subjects affected by congenital cardiovascular disease have a higher risk to develop arrhythmias than normal people\(^1\). This greater frequency is probably determined by three pivotal factors: the simultaneous presence of primary anomaly of the specialized conduction system and abnormal conduction pathways (e.g. in 25% of cases there is an association between accessory atrio-ventricular conduction pathways and Ebstein anomaly\(^2\)\(^3\), the morphological and functional changes induced by congenital malformation on cardiac structures that act as a pro-arrhythogenic substrate and, finally, the corrective cardiac surgery. The use of patches and suture lines, and surgical scars represent, in fact, a crucial substrate for arrhythmia genesis\(^4\)\(^6\) with increased mortality and morbidity\(^7\).

The combination of some cardiac defect and surgical procedures has been associated with a higher prevalence of arrhythmias, for example the procedures on atria such as inter-atrial defects repairs\(^6\)\(^8\), the Mustard and Sennings procedure, the Fontan anastomosis, the tetralogy of Fallot and the partial or fair anomalous pulmonary veins connection repair. One patient over four that undergo a Fontan and Mustard procedure develop an atrial arrhythmia\(^9\).

The reentry phenomenon represents the electrophysiological mechanism for these arrhythmias which develops around a central zone thwarting the conduction and represented by normal or extraneous/anomalous cardiac structures\(^10\).

In case of incisional atrial tachycardias, which appear during the reparative process after atrial surgical lesions, the macro-reentry develops always around a central obstacle that, in this case, is represented by a scar\(^11\), a prosthetic patch, a suture line or a blockage due to ablative procedures\(^12\)\(^15\).

RFCA (Radio Frequency Catheter Ablation) has become the treatment of choice for this kind of arrhythmias\(^16\)\(^20\).

The macro-reentrant atrial tachycardias analysis is focused on mapping the re-entrant circuit activation sequence with recognition of slow conduction areas. Successful ablation therapy depends on the correct identification of both the re-entrant circuit and its critical isthmuses. Entrainment stimulation allows to locate critical isthmuses.

In our case, TEE examination with 3D reconstruction of the interatrial septum showed a wide scar bulge along the patch where the electrophysiological mapping highlighted macro-reentrant phenomena.

The use of RFCA at the level of the anatomic structures involved, the cavo-tricuspid isthmus and the area near the patch’s edges, was able to cure the arrhythmias.

The association between the entrainment technique with the 3D electro-anatomical mapping allowed to identify two critical isthmuses among the electrical silent regions, so called low voltage areas due to atrial myopathy, and responsible for a double loop interatrial tachycardia.

The ablation of these different isthmuses broke the tachycardia; the ablation lines were, in fact, signed between low voltage areas and anatomical boundaries.

Recently, precise 3D electro-anatomical maps of post-pacing intervals (within 30 ms of tachycardia cycle length) around the activation loops demonstrated to characterize the whole activation circuit. The colour coded 3D entrainment mapping establishes and visualizes accurately the location of the reentrant circuit, indeed allows to plan linear RF lesions strategy for the macro-reentry atrial tachycardia treatment\(^19\).

Conclusions

In this patient, we observed how the coexistence of several factors such as the anatomic and structural ones, for example the cavo-tricuspid
isthmus region and a previous surgical suture can be responsible of symptomatic tachyarrhythmias often resistant to usual drug therapies.

In addition, this particular case shows how conventional pacing combined with 3D electroanatomic mapping is an effective method for the localization of critical isthmuses for re-entrant atrial circuits, suggesting tailored focal and linear ablation strategies.  

In such circumstances the ablative therapy represents the first therapeutic choice because it is characterized by a low appearance of complications and recurrences.

References


