

Ablation of a single pulmonary vein arrhythmic focus triggering several supraventricular arrhythmias

Luigi Sciarra, Leonardo Corò, Manuela Bocchino, Nadir Sitta, Elena Marras, Pietro Delise

Division of Cardiology, Hospital of Conegliano Veneto, Conegliano Veneto (TV), Italy

Key words:

Atrial fibrillation;
Atrial tachycardia;
Transcatheter ablation.

Pulmonary veins are a well-recognized source of focal ectopies that may trigger atrial fibrillation. Many ablative strategies, in particular ablation of the four pulmonary vein ostia, have been developed in order to cure atrial fibrillation. In some patients, the predominant arrhythmia may be an ectopic atrial tachycardia arising from a pulmonary vein and atrial fibrillation may be only a consequence of rapid atrial activation. There is a paucity of data regarding the electrocardiographic and electrophysiological characteristics of pulmonary vein tachycardia and the ablation strategy of this arrhythmia. In the present paper, we describe a case of a young woman with an arrhythmic focus localized in the right superior pulmonary vein with episodes of atrial tachycardia, paroxysmal atrial fibrillation and atrial flutter, who was successfully treated with transcatheter ablation.

(Ital Heart J 2004; 5 (12): 946-950)

© 2004 CEPI Srl

Received May 14, 2004;
revision received
September 28, 2004;
accepted September 29,
2004.

Address:

Dr. Luigi Sciarra

Via Monchera, 15/1
31010 Farra di Soligo (TV)
E-mail: bs.ls@iscali.it

Introduction

Left atrial focal tachycardia is a relatively uncommon form of atrial arrhythmia. In the left atrium, foci tend to cluster around the pulmonary veins (PVs)¹ and, more recently, have also been described around the mitral annulus². It is well known that focal activity arising from the PVs may trigger atrial fibrillation (AF)³. In patients with AF, foci are often multiple, involve multiple veins⁴, and usually are located deep within the trunk of the vein⁵. In this respect, many ablative strategies, in particular ablation of the four PV ostia, have been developed in order to cure AF^{4,6,7}. Nevertheless, there is a paucity of data on the electrocardiographic and electrophysiological characteristics of PV tachycardia and the ablation strategy of this arrhythmia. We report a case of a young patient with a single arrhythmogenic focus triggering different forms of supraventricular arrhythmias, such as atrial tachycardia, AF and atrial flutter.

Case report

A 26-year-old woman was referred to our hospital in 2002 for frequent palpitations. She was symptomatic for daily episodes of sudden-onset palpitations since 15 years, and during the last few months

her symptoms had increased in duration and intensity. Echocardiography, performed in 2001, showed mild mitral valve prolapse without significant mitral regurgitation and a normal biatrial size, biventricular structure and function. At 12-lead ECG frequent premature supraventricular beats (PSB) with the "P on T" phenomenon were observed (Fig. 1); the P morphology was: positive in the inferior leads and in lead D1, flat in aVL, negative in aVR, and positive in all the precordial leads. The prematurity of the ectopic P waves was variable and sometimes they were not conducted to the ventricles. Holter monitoring (2002) showed sinus rhythm at a normal rate with frequent PSB with the "P on T" phenomenon, sometimes in sequences of non-sustained atrial tachycardia, and some episodes of sustained paroxysmal AF triggered by the same beats, with episodes of a very high ventricular rate (220 b/min). Before planning an antiarrhythmic therapy, the patient experienced an episode of common atrial flutter with spontaneous transformation into AF, which was successfully cardioverted into sinus rhythm by means of DC-shock. An antiarrhythmic therapy was started: flecainide at a dosage of 50 mg bid and atenolol 25 mg daily. Nevertheless, symptoms remained almost unchanged and repeat Holter monitoring showed the persistence of the atrial ectopy with episodes



Figure 1. Twelve-lead ECG showing sinus rhythm with premature supraventricular beats in a bigeminal sequence. The ECG was recorded during therapy with diltiazem. See text for the description of the ectopic P-wave morphology.

of sustained common atrial flutter. In order to eliminate the episodes of atrial flutter and to improve symptoms a successful ablation of the cavo-tricuspid isthmus was performed. The class IC antiarrhythmic drug was continued. Unfortunately, no clinical benefit was observed during follow-up. A mild improvement was achieved with oral diltiazem. The patient was readmitted several times for recurrence of atrial tachycardia and AF. The recorded onsets of AF showed that the arrhythmia was always triggered by the same PSB with the same previously described 12-lead morphology of the P wave. In view of the patient's age, we decided not to place her on amiodarone. In November 2003 an electrophysiological study (EPS) and subsequent new ablation procedure was performed. Before ablation, the patient was submitted to spiral computed tomography in order to access the anatomy of the PVs: a slightly dilated os of the right superior PV was observed (Fig. 2). A decapolar Lasso catheter (Biosense Webster, Irwindale, CA, USA) for PV mapping and a 4 mm tip irrigated ablation catheter were inserted into the left atrium through a transseptal puncture. A quadripolar catheter was placed in the coronary sinus as well. Frequent atrial ectopic beats were recorded during EPS. With conventional mapping (according to prematurity criteria in bipolar recordings obtained from the Lasso catheter and from the mapping catheter moved in the four different vein ostia, when compared to the beginning of the ectopic P wave at the surface ECG) the focal activity was identified in the right superior PV. A segmental disconnection of this vein, guided by the Lasso catheter, was successfully performed. The disconnection of the vein was confirmed by the absence of potentials in the vein both in sinus rhythm and in coronary sinus pacing, and by the lack of atrial capture during pacing from the vein. Nevertheless, the ectopic activity still persisted. At this

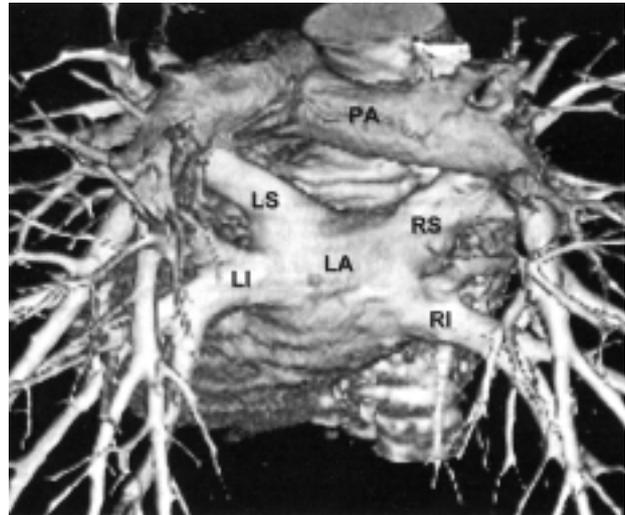


Figure 2. Three-dimensional reconstruction of the heart with spiral computed tomography. In the postero-anterior view, the posterior wall of the left atrium (LA) and the four pulmonary vein ostia are observable. The right superior pulmonary vein (RS) os is slightly dilated. LI = left inferior pulmonary vein; LS = left superior pulmonary vein; PA = pulmonary artery; RI = right inferior pulmonary vein.

point we hypothesized that we had probably performed a disconnection of the vein just distal to the vein os and decided to perform a further mapping of the ostial and antral region. The Lasso catheter was hence slightly withdrawn toward the ostium. In this position a pre-potential was recorded just on the roof of the os (Fig. 3) and we therefore proceeded to ablate this site with periprocedural success. Unfortunately, a few hours after ablation the focal activity was found to be still present and the patient's symptoms remained unchanged. For this reason, another ablation procedure was planned and performed in January 2004. A Biosense Webster irrigated (4 mm tip) ablation catheter was in-

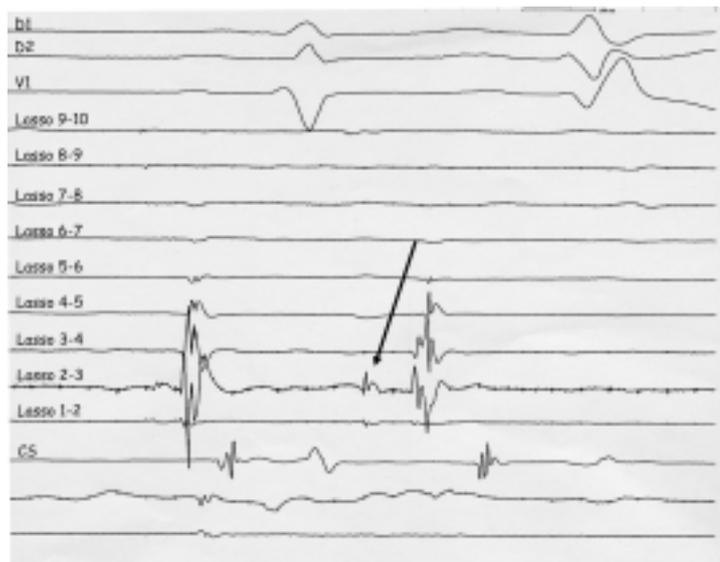


Figure 3. Intracavitary recording during the second ablative procedure. The intracavitary recording was performed after successful electrical disconnection of the right superior pulmonary vein, probably performed in a position just distal to the ostium. The Lasso catheter may be seen at the os of the vein. An ectopic atrial beat is recorded; a pre-potential is clearly evident in the 2-3 bipole of the Lasso catheter that corresponds to the roof of the vein. CS = coronary sinus.

serted in the left atrium at the site of the previous interatrial septal puncture. Unfortunately, no firing was present during the procedure and we therefore decided to try to obtain an electroanatomic map of the left atrium using the CARTO system. A circumferential ablation of the left atrium around the right superior PV os, at a distance of 0.5-1 cm from the os, was performed (Fig. 4). At 4 months of follow-up, the patient was completely asymptomatic and on no medication. Holter monitoring confirmed the absence of any atrial ectopic activity.

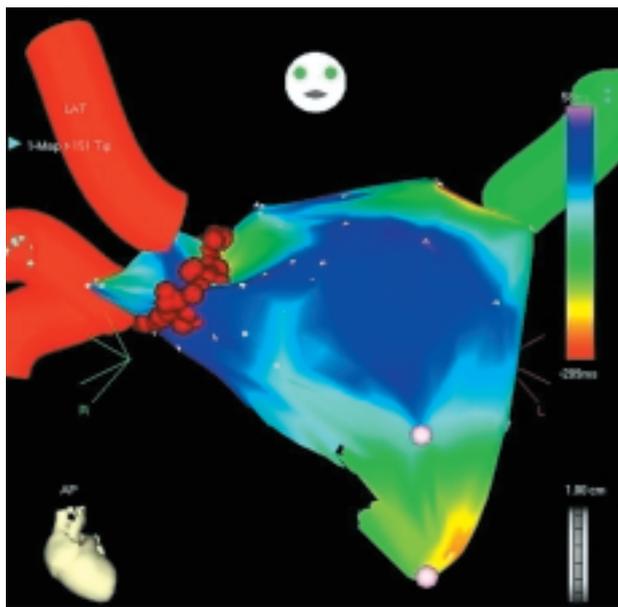


Figure 4. The CARTO image in the antero-posterior view showing the ablation line encircling the right superior pulmonary vein os (red points).

Discussion

To date, the electrophysiological and electrocardiographic characteristics of focal atrial tachycardia arising from the PVs have not been analyzed in detail. Tang et al.⁸ described 14 cases of left atrial tachycardia: in 11 cases it was a PV tachycardia (10 from the upper veins); unfortunately, the precise location of the foci and the long-term outcome of ablation were not reported in this study. Other studies⁹⁻¹¹ included only a small number of patients and no definite conclusions may be drawn. In a recent paper, Kistler et al.¹² described for the first time a consistent group of patients with PV tachycardia, and analyzed the long-term success rate of transcatheter ablation. The study included 27 patients with PV tachycardia; a focal ablative approach was associated with a very high success rate: a long-term success rate of 96%; 4 patients with a recurrence of the arrhythmia (only one from the same site) that was successfully re-ablated in 3¹². No patient presented with subsequent development of AF or atrial tachycardia from a different site¹². Moreover, a particular propensity of the foci to be located in the superior veins was also observed¹². The foci were located more frequently at the os rather than deep within the vein.

The electrophysiological characteristics of the arrhythmia in our patient were similar to those described by Kistler et al.¹²: the focus was located in a superior vein and it was ostial (on the roof of the os). Such an arrhythmia seems to be somewhat different from the common PV focal activity that triggers AF. In the last case it has been observed that foci frequently originate deep within the trunk and are often located in multiple veins⁵ even though the same group of Bordeaux could

identify and successfully ablate the focus in a single vein in a significant number of patients with AF⁵. However, in such a population, recurrences of foci in other sites were not infrequent and a second or third procedure was performed in a significant number of patients. In our opinion, patients with PV tachycardia seem to have different characteristics to those of patients with PV AF. It could be important to identify patients with PV tachycardia and to distinguish them from the general population with PV AF. In fact, for such patients an ablative approach targeted on a single vein may be effective, simpler and with a lower risk of complications. Obviously, we cannot tell whether in future another focus arising from another vein will start to fire in our patient, and our follow-up is too short to provide a definitive answer. Nevertheless, the only consistent reference in the literature seems to confirm a positive long-term outcome of a focal radiofrequency ablation in such patients¹².

The site of origin of the arrhythmia could be predicted by analyzing the P-wave morphology on the 12-lead ECG. In fact, all the available 12-lead ECGs suggested that the morphology of the ectopic P wave was the same. The positivity of the P wave in the inferior leads was consistent with a high location of the foci. Moreover, the P wave was positive in D1, flat in aVL, and negative in aVR: such characteristics (combined with the morphology in the precordial leads) suggested an origin from the right veins. On the basis of these considerations, the focus was suspected to be located in the right superior PV.

Other considerations about the variety of clinical arrhythmias in our patient may be drawn. At least three different arrhythmias were present in the same patient: atrial tachycardia, AF and atrial flutter. In this particular respect, our patient was different from the subjects studied by Kistler et al.¹²: in this study, the only clinical arrhythmia was PV tachycardia. Nevertheless, it is important to stress that the association between atrial tachycardia and AF or atrial flutter is not a surprising finding. It is well known that some reentrant tachycardias, such as atrioventricular nodal reentrant tachycardia, atrioventricular reentrant tachycardia and atrial flutter, may induce AF. This is the so-called "tachycardia-induced AF", as previously described by the group of Prystowsky¹³ and by our group¹⁴. In our patient, we considered AF as a consequence of the principal arrhythmia that was PV tachycardia, and we thought that by ablating the main arrhythmia, we would have also resolved the secondary one. This hypothesis was confirmed by the fact that after the effective focal ablation procedure no episode of AF was recorded. Furthermore, cavo-tricuspid isthmus ablation determined no clinical benefit. Thus, in our patient it is reasonable to consider even atrial flutter as a secondary arrhythmia.

Moreover, it is interesting to point out that the arrhythmogenic vein in our patient (the right superior

PV) was slightly dilated with respect to the other veins. This is not a surprising finding. In fact, it has been previously reported that in patients with paroxysmal AF the arrhythmogenic veins have significantly larger diameters than the non-arrhythmogenic ones¹⁵. At present, the correlations between vein dilation and the sites of origin of PV tachycardia are not known.

As far as the focal ablative procedure is concerned, some considerations need to be made. In the second ablative procedure we tried to perform a focal ablation. The focus was localized in the right superior PV. After the electrophysiological disconnection of this vessel, that was probably performed in a position just distal to the ostium, the focus was still present. A prepotential was recorded on the roof on the PV os. On the basis of these observations, we hypothesized that the focus was likely to be located just on the roof of the ostium. We performed ablation at this site with a periprocedural success. Unfortunately, an early recurrence of the arrhythmia occurred. Probably, we had achieved only a partial modification of the focus that was still active a few hours after ablation. In consideration of the clinical conditions of our patient, we decided to perform a third ablation. Before the procedure, electrocardiographic monitoring showed the presence of the usual ectopic activity with the same characteristics. Nevertheless, no firing was observed during the procedure or under isoproterenol infusion. However, we knew where the focus was localized on the basis of the previous ablation procedure and of the stable morphology of the ectopic P wave at the surface ECG. So an electroanatomical encircling of the right superior PV was performed using the CARTO system. If the firing is not present and it is hence not possible to localize it during ablation, electroanatomic ablation offers a good alternative to perform an "empirical" ablation. Radiofrequency can be delivered at a certain distance from the os of the vein in order to encircle the vein, or probably to eliminate the focus. However, this may be a possible alternative only if one is absolutely sure that the ectopic activity arises from a given vein.

References

1. Chen SA, Tai CT, Chiang CE, Ding YA, Chang MS. Focal atrial tachycardia: reanalysis of the clinical and electrophysiological characteristics and prediction of successful radiofrequency ablation. *J Cardiovasc Electrophysiol* 1998; 9: 355-65.
2. Kistler PM, Sanders P, Hussin A, et al. Focal atrial tachycardia arising from the mitral annulus: electrocardiographic and electrophysiologic characterization. *J Am Coll Cardiol* 2003; 41: 2212-9.
3. Jais P, Haissaguerre M, Shah DC, et al. A focal source of atrial fibrillation treated by discrete radiofrequency ablation. *Circulation* 1997; 95: 572-6.
4. Haissaguerre M, Jais P, Shah DC, et al. Electrophysiological end point for catheter ablation of atrial fibrillation initi-

- ated from multiple pulmonary venous foci. *Circulation* 2000; 101: 1409-17.
5. Haissaguerre M, Jais P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med* 1998; 339: 659-66.
 6. Pappone C, Rosanio S, Oreto G, et al. Circumferential radiofrequency ablation of pulmonary vein ostia. A new anatomic approach for curing atrial fibrillation. *Circulation* 2000; 102: 2619-28.
 7. Marrouche NF, Martin DO, Wazni O, et al. Phased-array intracardiac echocardiography monitoring during pulmonary vein isolation in patients with atrial fibrillation: impact on outcome and complications. *Circulation* 2003; 107: 2710-6.
 8. Tang CW, Scheinman MM, Van Hare GF, et al. Use of P wave configuration during atrial tachycardia to predict site of origin. *J Am Coll Cardiol* 1995; 26: 1315-24.
 9. Pappone C, Stabile G, De Simone A, et al. Role of catheter-induced mechanical trauma in localization of target sites of radiofrequency ablation in automatic atrial tachycardia. *J Am Coll Cardiol* 1996; 27: 1090-7.
 10. Anguera I, Brugada J, Roba M, et al. Outcomes after radiofrequency catheter ablation of atrial tachycardia. *Am J Cardiol* 2001; 87: 886-90.
 11. Lesh MD, Van Hare GF, Epstein LM, et al. Radiofrequency catheter ablation of atrial arrhythmias: results and mechanisms. *Circulation* 1994; 89: 1074-89.
 12. Kistler PM, Sanders P, Fynn SP, et al. Electrophysiological and electrocardiographic characteristics of focal atrial tachycardia originating from the pulmonary veins: acute and long-term outcomes of radiofrequency ablation. *Circulation* 2003; 108: 1968-75.
 13. Prystowsky EN. Tachycardia-induced tachycardia: a mechanism of initiation of atrial fibrillation. In: Di Marco JP, Prystowsky EN, eds. *Atrial arrhythmias: state of the art*. Armonk, NY: Futura Publishing Company, 1995: 81-95.
 14. Delise P, Gianfranchi L, Paparella N, et al. Clinical usefulness of slow pathway ablation in patients with both paroxysmal atrioventricular nodal reentrant tachycardia and atrial fibrillation. *Am J Cardiol* 1997; 79: 1421-3.
 15. Yamane T, Shah DC, Jais P, et al. Dilatation as a marker of pulmonary veins initiating atrial fibrillation. *J Interv Card Electrophysiol* 2002; 6: 245-9.