Case Report

Small Loop Re-entry Tachycardia Between Left Atrium and Left Superior Pulmonary Vein: A Late Arrhythmogenic Complication After Pulmonary Vein Ablation Isolation. How Useful Is P-Wave Morphology?

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We present a 72-year-old woman who exhibited a highly-symptomatic narrow QRS tachycardia (cycle length 300 ms) seven months after a pulmonary vein ablation for persistent atrial fibrillation. The electrophysiological study revealed a focal atrial re-entry tachycardia across two gaps in the previous ablation line close to the ostium of the left superior pulmonary vein. Radiofrequency ablation of the first gap terminated the tachycardia, while ablation of the second gap led to re-isolation of the left superior pulmonary vein. The patient remains asymptomatic after a follow up of 6 months.

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Atrial tachycardia after pulmonary vein isolation may be caused by a variety of mechanisms. Here we describe a case of focal atrial re-entry tachycardia across two gaps in the previous ablation lesions.

Case presentation

A 72-year-old woman was referred to our cardiology department because of a persistent narrow-QRS complex tachycardia with a frequency of 100 bpm, accompanied by palpitations and dyspnoea on exertion. The patient had been suffering for 6 years from paroxysmal and in the last two years from persistent atrial fibrillation, presumably secondary to a pre-existing hypertensive heart disease. Despite three electrical cardioversions and various antiarrhythmic drugs (including sotalol and amiodarone), restoration of sinus rhythm had been feasible for only a short period of time (less than four weeks). For that reason, 12 months earlier she had undergone at our institution a successful pulmonary vein isolation by means of radiofrequency ablation, after coronary angiography had excluded coronary artery disease. During this procedure a circumferential ablation line was created around both septal and lateral pulmonary veins, resulting in the disappearance of pulmonary vein potentials in a decapolar circular catheter (Lasso, Biosense Webster) that was placed close to the pulmonary vein ostium (Figure 1). Additionally, pacing at all poles of the Lasso catheter had led to local capture but no conduction to the left atrium, thereby proving the presence of bi-directional block.

Seven months after pulmonary vein isolation, with the patient off antiarrhythmic drugs, sudden palpitations occurred. On the ECG atypical flutter with 2:1 atrioventricular conduction was documented.

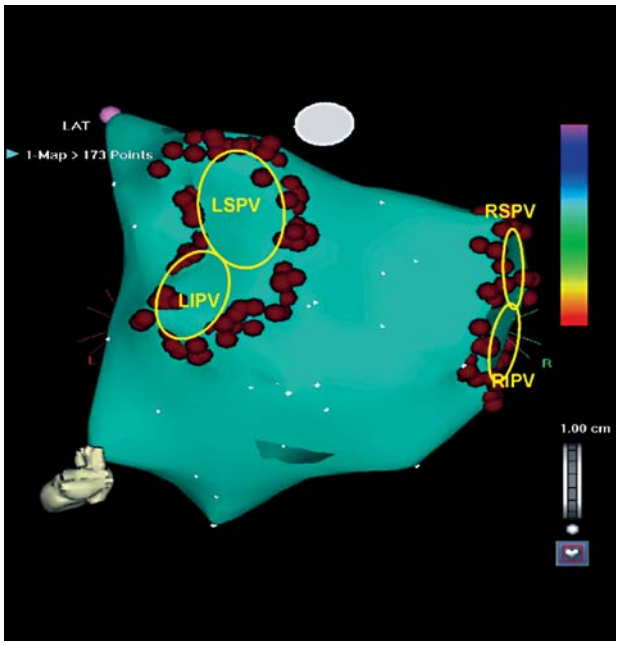

Figure 1. Anatomical reconstruction of the left atrium showing the ablation points (red spots) around left and right pulmonary veins during the pulmonary vein isolation procedure. LSPV – left superior pulmonary vein; LIPV – left inferior pulmonary vein; RSPV – right superior pulmonary vein; RIPV – right inferior pulmonary vein.

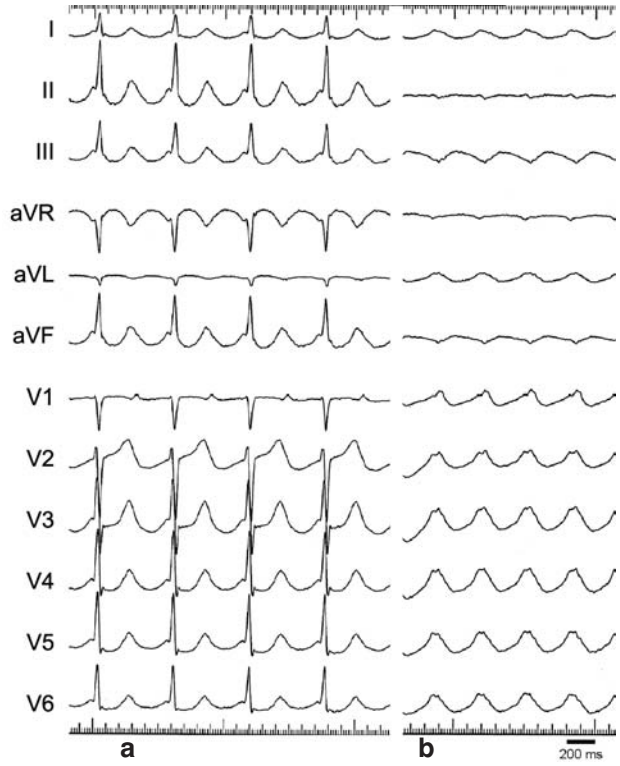

Figure 2. Twelve-lead ECG of the clinical tachycardia (a) and after adenosine-induced-complete atrioventricular block (b).

One week after restoration of sinus rhythm by external electrical cardioversion the same arrhythmia recurred. The patient was started on amiodarone and electrical cardioversion was performed. In the following three months the arrhythmia recurred four times, necessitating electrical cardioversion. After the fifth recurrence the patient was referred to our institution for electrophysiological study and ablative treatment. Transthoracic echocardiography revealed a slightly enlarged left atrium (45 mm) and slight mitral and tricuspid valve regurgitation. There was no left ventricular hypertrophy and the ejection fraction was 70%. Transoesophageal echocardiography excluded atrial thrombi.

The 12-lead ECG showed atypical flutter (cycle length 300 ms) with 2:1 atrioventricular conduction and a frequency of 100 beats per minute (Figure 2a). Administration of 12 mg adenosine i.v. led to intermittent AV block and exposed the flutter waves: these were positive in I, aVL, in all precordial leads and negative in III and aVF. Isoelectric lines between the flutter waves were present in only two leads, II and aVR (Figure 2b).

During an electrophysiological study, activation of the right atrium occurred in a counter-clockwise direction and coronary sinus activation occurred from proximally to distally. Rapid atrial pacing at different sites of the right atrium, as well at the left side of the interatrial septum, and at a pacing interval that was only slightly shorter than the tachycardia cycle length (280 ms), was followed by a long post-pacing interval (PPI) – evidence that neither region was part of the re-entry circuit.

After transseptal puncture two long sheaths (SL1, St. Jude Medical, St. Paul, MN, USA) were introduced into the left atrium (one for a decapolar Lasso catheter and one for the ablation catheter). Both left superior and right inferior pulmonary vein showed an electrical reconnection to the left atrium, while left inferior and right superior pulmonary veins were still isolated. The earliest activation of the left superior pulmonary vein was at the anterior-inferior ostial segment. Detailed electroanatomical mapping was performed and identified the earliest atrial activation in the superior segment of the ostium of the left superior pulmonary vein, with clockwise activation of the posterior left atrial wall (Figure 3). Rapid atrial pacing at this site accelerated the tachycardia without any

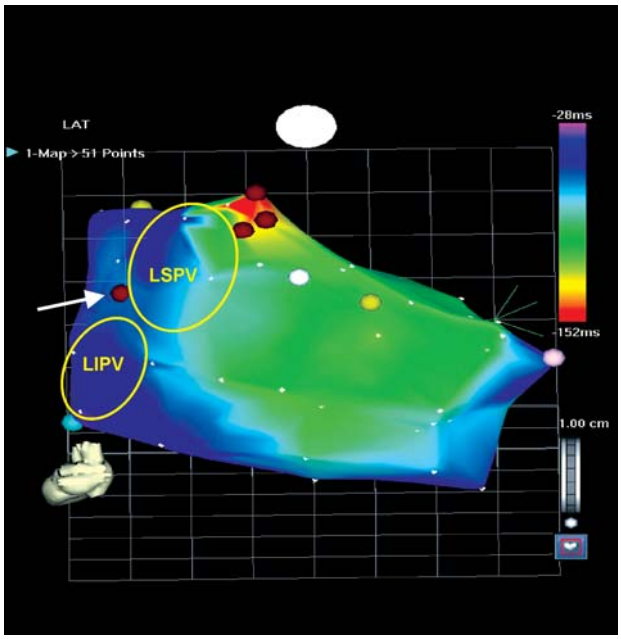

Figure 3. Activation mapping of the left atrium with an electroanatomical mapping system (CARTO) showing the earliest activation at the superior portion of the LSPV ostium (red area). After termination of the tachycardia, one additional ablation at the inferior-anterior segment of the LSPV ostium (white arrow) led to complete re-isolation of this vein. LSPV – left superior pulmonary vein; LIPV – left inferior pulmonary vein.

change in the activation sequence and was followed by a PPI that was identical to the tachycardia cycle length (Figure 4a). Entrainment mapping at several left atrial sites other than the ostium of the left superior pulmonary vein resulted in significantly longer PPIs than the tachycardia cycle length. Therefore, a macro re-entrant circuit could be excluded. Radiofrequency (RF) energy ablation was performed at the superior segment of the ostium of the left superior pulmonary vein. During the third RF application (30 W, upper temperature limit 43°C, irrigation with 17 ml/min saline solution) at this site the tachycardia terminated (Figure 4b). Conduction from the left atrium to the left superior pulmonary vein was still present, but with a significant time delay (196 ms). The earliest pulmonary vein potential on the Lasso catheter was registered at the inferior-anterior segment of the pulmonary vein ostium. After one additional RF application at this site the left superior pulmonary vein was completely isolated (Figure 5). Finally, the right inferior pulmonary vein was re-isolated.

Amiodarone was stopped 4 weeks after successful ablation. During 6 months' follow up the patient has remained free of symptoms and of any arrhythmia.

Discussion

The pulmonary veins are the main source of triggers initiating atrial fibrillation. Isolation of these triggers, in order to prevent the initiation of arrhythmias,[1-3] has evolved from focal ablation at the site of the earliest activity (segmental) to wider ablation around the ostia of each of the ipsilateral pulmonary veins (circumferential pulmonary vein ablation, CPVA). This provides an arrhythmogenic substrate for left atrial tachycardia, predisposing to re-entry around anatomical or ablation-created conduction barriers or through gaps within the linear lesions (gap-related re-entry).[4-6] The incidence of these tachycardias varies between 4%[4] and 31%[7] in different electrophysiology centres.

Mechanisms of left atrial tachycardias after atrial fibrillation ablation

The most common atrial tachycardia after pulmonary vein isolation is macro re-entrant tachycardia around the isolated pulmonary veins or the mitral annulus,[8] accounting for 33%[7] or up to 82%[9] of atrial tachycardias. There are, though, other re-entry tachycardias with a small-loop mechanism (up to 36%), or other unknown mechanisms (maybe triggered activity or abnormal automaticity, up to 31%), which are difficult to determine, because extensive pacing or pharmacological manoeuvres can cause degeneration into atrial fibrillation.

The two main groups of re-entry tachycardias are differentiated according to the mapped activation pattern of the left atrium and the size of the re-entrant circuit. A macro re-entry circuit is located in a large part of the left atrium (minimum diameter >3 cm), whereas a small-loop re-entrant circuit shows a diameter <3 cm and coverage of the whole cycle length and centrifugal activation of the remainder of the left atrium.[10] Small-loop re-entrant tachycardias are further divided into gap-related tachycardia and extremely-slow-conduction–related tachycardias. In the gap-related tachycardia, the re-entrant circuit crosses the previously deployed CPVA lesions at least twice (in two gaps or one large gap), whereas in extremely-slow-conduction–related tachycardia, the re-entrant circuit is not located across but adjacent or tangential to the previously deployed CPVA lesions. The characteristic feature of the latter re-entrant tachycardias is a small, distinctive area of markedly slow conduction maintaining the re-entry. Finally there are unstable tachycardias that cannot be specified due to spontaneous

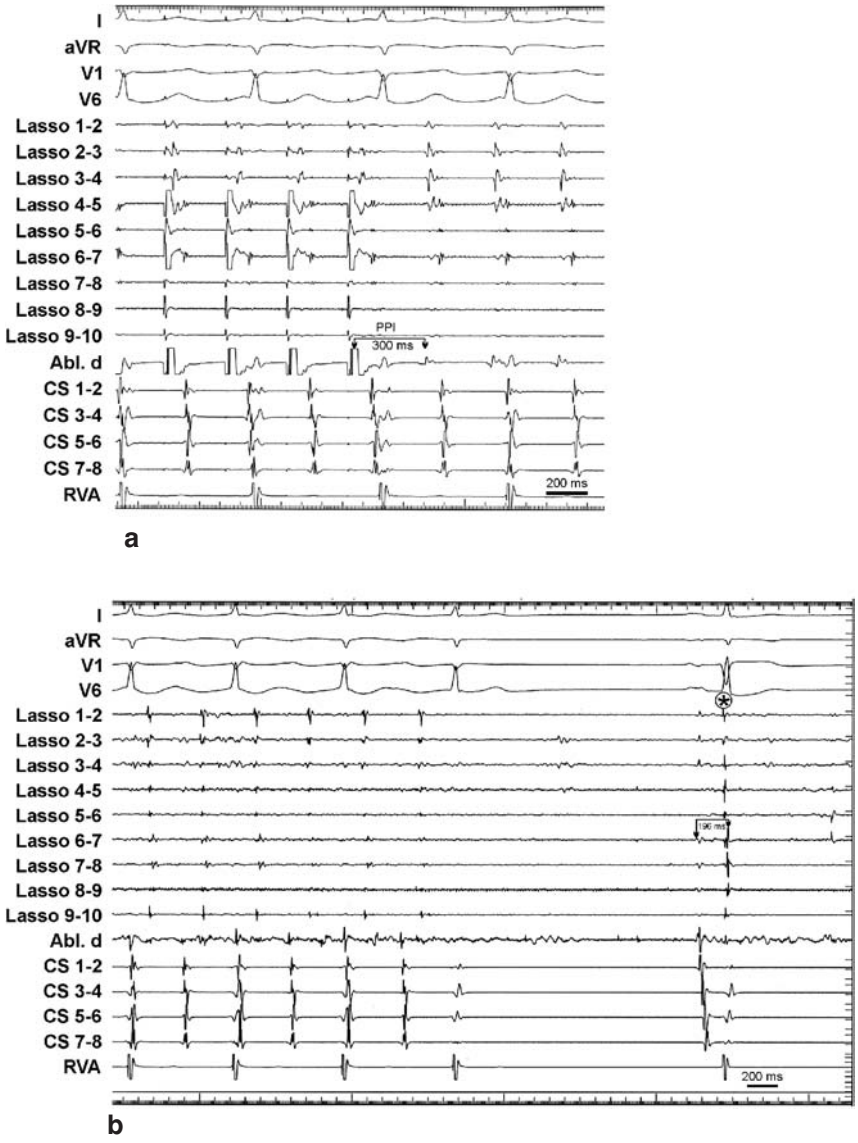

Figure 4. a. Entrainment of the tachycardia at the superior segment of the left superior pulmonary vein ostium with a postpacing interval of 300 ms (identical to tachycardia cycle length). Activation sequence during pacing is the same as during tachycardia.
b. During the third radiofrequency application at this site there was termination of the tachycardia. Delayed conduction in the left superior pulmonary vein is still present (star).

or entrainment-induced change to another tachycardia or atrial fibrillation.[7]

Satomi et al[11] reported 6 cases of such gap-related focal re-entry atrial tachycardia after pulmonary vein isolation and proposed criteria for diagnosis: 1) recovered pulmonary vein conduction with one-to-one left atrium-pulmonary vein or pulmonary vein-left atrium conduction during tachycardia; 2) identification of sites with perfect entrainment within the pulmonary veins and the left atrium near the involved pulmonary veins; 3) entrainment within the pulmonary veins with identical P-wave morphology to that during tachycardia; 4) identification of two conduction gaps in the previous circular lesions, one conduction gap presented as the entrance with earliest pulmonary vein activation and one presented as the exit with earliest atrial activation; 5) termination of the tachycardia by RF delivery at either the entrance or the exit site.

In the case described here all the abovementioned criteria were met and the patient could be successfully treated without any recurrence of this or any other tachycardia.

Interestingly, although the positive P waves in lead I and aVL implied an origin from the right pulmonary veins,[12,13] the tachycardia origin in our case proved to be at the ostium of the left superior pulmonary vein. The left atrial roof and interatrial septum were activated early after the exit of propagation from the left superior pulmonary vein and were followed by activation of the coronary sinus from proximal to distal. The septal to lateral left atrial activation explain the

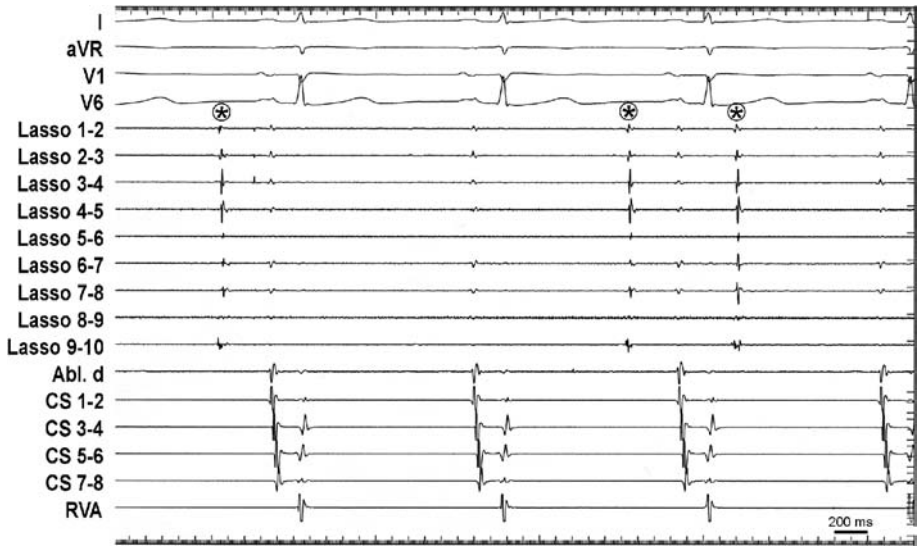

Figure 5. After complete isolation of the left superior pulmonary vein, spontaneous electrical activity is present but it is completely dissociated from the left atrium.

unexpected morphology of the P waves. A plausible explanation for that is that propagation along the left lateral wall occurred relatively late because of slow-conduction across the ablation lines created during the first ablation procedure.

Takahashi et al[14] reported 9 cases of atrial tachycardias after catheter ablation of atrial fibrillation, where entrainment mapping excluded macro re-entries and facilitated the classification of atrial tachycardias as focal. In these cases the P-wave morphology in V_1 was associated with the location of the atrial tachycardia, being biphasic positive/negative for atrial tachycardias arising from septal regions and monophasic positive in atrial tachycardias from lateral regions. The same ECG manifestation was previously observed in articles about patients with left atrial flutter.[15] However, in contrast to both of these conclusions, the P-wave morphology in our case was biphasic positive/negative, despite originating from the lateral left superior pulmonary vein. This proves that previous ablation lines[16,17] can lead to unexpected activation of the left atrium and thus unexpected P-wave forms. Therefore, P-wave morphology in patients who have already had an ablation procedure can indeed be misleading and the 12-lead ECG should not be used as a single diagnostic tool.

Shah et al[13] have shown that micro re-entrant atrial tachycardias after ablation of atrial fibrillation are characterised by an isoelectric line between the P waves in all 12 ECG leads. On the other hand, in macro re-entrant tachycardias continuous atrial activity was reflected by the lack of an isoelectric line between P waves. In the case presented, although the tachycardia mechanism was a small-loop re-entry, continuous electrical activity was registered in all ECG-leads apart from II and aVR. This implies the absence of a zone of very slow conduction, which is usually present in these types of arrhythmias and causes the isoelectric line between the P waves.

Conclusion

Focal re-entry atrial tachycardia related to a double gap along a previous ablation line is a well-characterised iatrogenic tachycardia that can appear weeks or months after radiofrequency ablation of the pulmonary veins and can be highly symptomatic. P-wave morphology may be misleading in the assessment of the origin and/or the mechanism of tachycardia, as it can be significantly influenced by the changes in the electroanatomical properties of the left atrium after the primary ablation procedure.

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