Case Report

Left Atrial Tachycardia After Pulmonary Vein Isolation for Atrial Fibrillation

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Left atrial tachycardia (AT) has been reported to occur after pulmonary vein isolation (PVI) for the treatment of atrial fibrillation (AF). We treated 3 patients who developed AT of different mechanisms following PVI. In case 1, focal AT originating at the ostium of the left superior PV was demonstrated and focal radiofrequency ablation was performed at the breakthrough point at the ostium of the left superior PV terminated the AT. In case 2, AT was shown to be counterclockwise macroreentrant AT around the left inferior PV through the conduction gap of the left sided posterior wall for which linear ablation was performed between left superior and inferior PVs. Focal ablation at the conduction gap terminated the AT. In case 3, a macroreentrant AT propagating around the mitral annulus was demonstrated and linear ablation between left inferior pulmonary vein and mitral annulus (mitral isthmus) terminated the AT.

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Introduction

Circumferential pulmonary vein (PV) isolation (I) has been shown to be effective treatment for atrial fibrillation (AF). Circumferential PVI has also been reported to decrease mortality and morbidity and improve quality of life compared to medical therapy. However, development of atrial tachycardia (AT) or atrial flutter (AFL) was reported to occur acutely in approximately 10% of patients after PVI with sites of origin at left atrial roof, left atrial appendage, left superior PV antrum, right superior PV and mitral isthmus. Mesas et al. also described AT after PVI as a possible midterm complication, mostly related to reentry around the incomplete lesions or involving the mitral isthmus. This case report describes 3 patients who developed 3 different types of left AT after PVI.

Case Reports

Case 1

A 54-year-old man underwent segmental PVI to 4 PVs at Nihon University Hospital in December 2003. He began to experience repeated episodes of palpitation 3 months after PVI and resting ECG showed AT (Figure 1). He was readmitted for repeat
electrophysiological study, and radiofrequency catheter ablation (RFCA) of the AT was performed in March 2004. Electroanatomical mapping (CARTO®, Biosense-Webster, Diamond Bar, California, USA) of the AT (Cycle length 340 ms) showed that the earliest activation site was located at the ostium of the left superior pulmonary vein (LSPV) (Figure 2). A circumferential duo-decapolar catheter (Lasso®, Biosense-Webster, Diamond Bar, California, USA) was placed at the LSPV ostium and a focal AT originating from the LSPV ostium was demonstrated with the AT conducting to LSPV and LA (Figure 3). The earliest activation point was located at electrodes 17–18 and 19–20 of the Lasso catheter and RFCA to that point resulted in termination of the AT in 10 sec. This patient is free of symptoms for 1 year without antiarrhythmic drugs.

**Case 2**

A 53-year-old man underwent tricuspid valve-inferior vena cava isthmus ablation for common atrial flutter in November, 2003. After that procedure, he suffered from paroxysmal AF and underwent extended ipsilateral PVI in January, 2004. However, he experienced repeated episodes of palpitation for 6 months after PVI, and 12-lead ECG showed AT (Figure 4). He was admitted to Nihon University Hospital for repeat electrophysiological study and RFCA for the AT (cycle length 410 ms) in June, 2004. Electroanatomical mapping of the left atrium during the AT was performed and the activation map showed the conduction gap between scar and scar in the left posterior wall of the LA. Propagation map showed slow conduction across the gap and a single loop reentry around the left inferior PV (Figure 5). Activation time of the propagation map (385 ms) was identical to the cycle length of the AT (390 ms). RFCA was performed at the gap and the AT was terminated 14.3 sec after the energy delivery. This patient is free of symptom for 9 months without antiarrhythmic drugs.

**Case 3**

A 53-year-old man underwent RFCA for WPW
syndrome and common atrial flutter in 1997. After that, he suffered from paroxysmal AF and underwent extended ipsilateral PVI in August 2004. However, AT recurred within 2 weeks after PVI and proved refractory to antiarrhythmic drugs, persisting for 2 months. He was admitted to Nihon University Hospital for repeat electrophysiological study and RFCA for the AT (Cycle length 600 ms) in August 2004. Because CS was activated from distal to proximal direction during the AT (Figure 6), a duo-decapolar halo catheter was placed around the mitral annulus. The AT was shown to activate the mitral annulus from anterolateral to posterior direction. Left atrial pacing between the left inferior PV and mitral annulus showed that post-pacing interval was identical to the AT cycle length and concealed

Figure 3 Intracardiac recordings of the tachycardia in case 1.
Note that the earliest atrial activation was located at the PV 11–12 of the Lasso® catheter (small and dull potential) and then, the atrium activation was directed to PV 17–18 (black solid arrow) and from which left superior pulmonary vein was activated and conducted to PV 5–6 (dashed arrow). Premature contraction was observed within the PV (PV 13–14) (asterisk), but it did not conduct to the LA, so that contraction did not reset the tachycardia. PV: pulmonary vein, CS: coronary sinus, ABL: ablation catheter LA: left atrium.

Figure 4 12-lead ECG of the atrial tachycardia in case 2.
Note that 2:1 atrioventricular conduction as shown in the arrow in a VF lead.
Figure 5  Propagation map of the tachycardia in case 2.
Note that the tachycardia showed counterclockwise activation around the left inferior pulmonary vein. Pink color: left superior pulmonary vein, left side gray color: left inferior pulmonary vein, right side gray color: right superior pulmonary vein, yellow color: right inferior pulmonary vein.

Figure 6  Intracardiac recordings of the tachycardia in case 3.
Note that coronary sinus was activated from distal to proximal direction and the activation around the mitral annulus recorded by Halo® catheter was also directed from distal to proximal (MV 1–2 to MV 9–10). However, the activation sequence of the Halo® catheter of the proximal electrodes (MV 11–12 to 17–18) was from proximal to distal and no potential was recorded from MV 19–20. The reason for proximal to distal conduction might be explained by the location of the proximal portion of the Halo® electrodes. If the proximal portion of the Halo® catheter was located at the septal side of the right side posterior line and the right side posterior line connected right inferior pulmonary vein and mitral annulus, activation around the mitral isthmus was blocked between right inferior pulmonary vein and the mitral annulus, proximal portion of the Halo® catheter was activated from left atrial roof, thus activation sequence of the septal side of the right posterior line was directed from proximal to distal direction. Post-pacing interval (191 ms) was identical to tachycardia cycle length (198 ms). His: His bundle electrogram recording position, MV: mitral valve annulus, CS: coronary sinus, ABL: ablation catheter placed at the mitral isthmus, STIM: stimulation artifact.
entainment phenomenon was observed. However, we were not able to demonstrate the entire tachycardia circuit because we did not use CARTO® system in this case. Left atrial linear ablation was performed between left inferior PV and mitral annulus (Figure 7), the AT was terminated and was not inducible with atrial extrastimuli and burst pacing. This patient was free of symptoms for 9 months.

**Discussion**

Left AT has been reported to occur after segmental and circumferential PVI with frequencies ranging from 2.9–29%. We described 3 different types of AT after PVI. Left AT occurred in 3% of cases after PVI in our institution. Case 1 demonstrated focal tachycardia pattern originating from ostium of the left superior PV. However, it might also be possible that the mechanism of this AT consisted of reentry between the ostium of the left superior PV and the inside on the left superior PV through more than 2 conduction gaps between left superior PV ostium and left superior PV. Mesas et al. reported 14 left ATs, 3 of which were characterized as focal AT and 11 as macroreentrant AT utilizing the mitral isthmus, the posterior wall, or gaps on previous encircling lines. Thus, detailed activation mapping and entrainment pacing around the ostium of the left superior PV might reveal the mechanism of the AT. Case 2 demonstrated macro-reentrant AT across the mitral isthmus (left inferior PV–mitral annulus). Because, we could not demonstrate the entire tachycardia circuit, it was not clear that the AT conducted around the mitral annulus similar to common type right atrial flutter or between mitral isthmus and the left pulmonary veins. The AT was terminated by linear ablation of the mitral isthmus as previously reported. Our experience with 3 patients is consistent with previous reports that left atrial tachycardia after segmental or circumferential pulmonary vein ablation for AF can be due to macro-reentrant or focal mechanism. Reentry occurs most commonly across the mitral isthmus, the posterior wall, or gaps on previous ablation lines and such gaps and foci occur most commonly at left superior PV and at the septal aspect of the right PVs. These arrhythmias can be successfully ablated with an electroanatomical mapping system.

**References**

Cardiovasc Electrophysiol 2003, 26: 417–421