Atrial tachycardia from an unusual site—Left atrial appendage tachycardia: Challenges in ablation

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ABSTRACT

Left atrial appendage (LAA) tachycardia are rarely encountered in clinical practice (2.1% of focal atrial tachycardia). Out of these, the ones arising from the distal part of LAA are difficult to ablate due to higher risk of LAA perforation and thromboembolism. We hereby present a patient with LAA tachycardia mapped to the tip of LAA with the help of the CARTO system and ablated. This case highlights the inherent challenges faced in such a scenario.

Introduction

Focal atrial tachycardias (FAT) are generally seen originating from specific structures like crista terminalis, tricuspid or mitral annulus, triangle of Koch, or pulmonary vein ostia.[1] If activation times at these sites are not early, then uncommon sites like left atrial appendage (LAA) may be looked at. AT originating from the LAA form around 2.1% of the focal ATs.[2] Their electrocardiographic[3] and electrophysiological[3,4] characteristics have been described before. Ablation in and around the LAA has been associated with thromboembolism or perforation of LAA.[3] We present one such FAT arising from LAA which was mapped to the tip of LAA and ablated. Our patient did not require epicardial access. We have highlighted the difficulties/challenges of ablation at this site.

Case History

A young 24-year-old woman was referred to us with tachycardia, which was detected on a routine physical check-up. She tended increased heart rates, which was long-standing and did not complain of any syncope or palpitations.

The basal electrogram [Figure 1] revealed that she had an ongoing tachycardia with a cycle length of approximately 400 ms. The P wave was inverted in leads I and aVL which suggested an origin from the left lateral part of the LA: left superior pulmonary vein or the LAA. A 2D-echocardiography showed normal chamber dimensions with no regional wall motion abnormalities with a normal left ventricular ejection fraction. In order of the incessant nature of the tachycardia, she was taken up for electrophysiology study.

A decapolar catheter was placed in the coronary sinus and quadripolar catheters were placed in the His bundle region and in the right ventricle apex. The intra-cardiac tracings [Figure 2] confirmed a long RP tachycardia with a cycle length of 425 ms with eccentric retrograde conduction, which ruled out atypical
AV nodal reentrant tachycardia. Ventriculo-atrial dissociation was seen during ventricular pacing during the tachycardia, which also ruled out pathway-mediated reentrant tachycardia. Injection of metoprolol slowed the tachycardia and isoprenaline accelerated the tachycardia.

Trans-septal puncture was performed with the Brokenborough needle and a Preface 7F sheath (Bio sense Webster) was introduced in the left atrium. Intravenous heparin as per body weight was injected intravenously. A 7F, 3.5-mm tip open irrigated Coolflow Thermocouple catheter (Bio sense Webster) was used for mapping and subsequent ablation. The activation map of the tachycardia was carried out in both the right and left atrium with the atrial electrogram in CS5,6 taken as the reference electrode. The timing intervals during activation ranged from 61 ms to -98 ms as Figure 3, which was less than 50% of the tachycardia cycle length suggestive of a focal tachycardia mechanism. The earliest activation of the tachycardia was at the tip of the LAA where the local activation was -96 ms [Figure 3] compared to the atrial electrogram in coronary sinus (CS5,6) electrodes. This amounted to a local activation time of -29 ms compared to the onset of the P wave during the tachycardia. The M1 signal [Figure 3] is the unipolar signal showing the qs pattern at the site of earliest activation.

Radiofrequency ablation was carried out with 30 W power at that site which accelerated the tachycardia with subsequent termination of the tachycardia to sinus rhythm. Figure 4 shows the site of ablation at the tip of LAA.

She has been now off medications since last 2 years. She was treated with a 6-week course of aspirin post-procedure.

**Discussion**

FAT arising from the LAA are rare (2.1% of FAT\(^5\)) and are not frequently encountered in clinical practice. The majority of them arise due to increased automaticity and are likely to be induced by isoprenaline infusion during the EP study. Our patient’s tachycardia slowed with beta-blocker and accelerated with isoprenaline injection. This is mostly seen with focal automatic tachycardias as they are strongly influenced by the sympathetic tone. Meanwhile, reentry tachycardias (both macro and micro reentry) are not responsive to beta-blockers for practical purposes, though there might be exceptions.

The hallmark of the P wave originating from the LAA on the ECG is the deep negative P waves in leads I and aVL, with positivity in inferior leads. The P wave is biphasic in V1 and positive in the other precordial leads.\(^6\)

Wang et al. and Yamada et al.\(^3,7\) reported that 3% (7 out of 246 ATs) and 26% (13 of the 50 patients) of the AT were arising from the LAA, respectively. They were all associated with eccentric retrograde conduction and long RP intervals. The mean cycle length reported was 381 ± 34 ms. Yamada reported that they originated from the medial LAA in 11 patients and lateral LAA in 2 patients out of the 13 patients studied.\(^7\) Wang found that the tachycardia originated from the base, mid-part, and the apex in 2, 3, and 2 patients, respectively, out of the 7 patients.\(^3\) All the AT arising were successfully ablated. However, 2 patients had recurrence when they were followed...
up by Wang et al.\textsuperscript{[3]} for 2 years, while none had recurrence in the series by Yamada et al.\textsuperscript{[7]} at 8 years of follow-up. Our patient has had a follow-up of 2 years during which she has been asymptomatic.

Approach to the ablation in the LAA can both be endocardial or epicardial. The focus can be ablated endocardially in the majority of the cases as mentioned in the above series. This region is approached by doing a transseptal puncture and mapping the focus. Performing a transseptal puncture requires expertise and skill to avoid complications like cardiac tamponade and bleeding. Epicardial mapping will need pericardial access and adequate precautions concerning injury to adjoining structures while ablating.

Radiofrequency ablation in the LAA needs to be cautious due to the high risk of perforation due to manipulation of the catheter in the LAA. The risk of loss of LAA contractility due to ablation may predispose to the formation of clots. No change in the LAA flow velocities was noted before and after ablation in the series reported by Yamada et al.\textsuperscript{[7]} Similarly, no thromboembolic complications were reported by Wang et al.\textsuperscript{[3]} and by Qian et al.,\textsuperscript{[5]} with 6 months of aspirin treatment. Our patient was treated with a 6-week course of aspirin therapy.

Given the increased risks of ablation in the LAA, epicardial ablation of these tachycardias has been suggested to be safer and was successfully demonstrated in 2 cases by Phillips et al.\textsuperscript{[8]} There has been a recent report of the ablation of proximal LAA tachycardia with cryoablation with the smaller CB2 balloon.\textsuperscript{[9]}

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Conflicts of interest
There are no conflicts of interest.

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