Adenosine sensitive focal atrial tachycardia originating from the non-coronary aortic cusp

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We report on the characteristics and the ablation procedure of a focal atrial tachycardia originating from the non-coronary aortic cusp. The electrophysiological features of the tachycardia included: (i) incessant pattern; (ii) easy induction and termination by atrial stimulation; (iii) earliest right atrial activation at the para-hisian area; and (iv) termination by adenosine. Left-sided mapping revealed the earliest atrial activation during the tachycardia at the non-coronary aortic cusp. Radiofrequency energy application at this site successfully terminated the tachycardia. Mapping of the non-coronary aortic cusp should always be considered when the earliest right atrial activation is recorded at the para-hisian area in order to avoid the risk of atrioventricular block by inappropriate ablation near the His-bundle region.

Introduction

Focal atrial tachycardias (ATs) are commonly located in the crista terminalis, near the tricuspid and mitral annulus, within the pulmonary veins, at the ostia of the coronary sinus (CS), and at the para-hisian region. 1 Atrial tachycardias arising from the non-coronary aortic cusp have rarely been described. 2–4 In the present study, we report on the characteristics and the ablation procedure of a focal AT originating from the non-coronary aortic cusp.

Electrophysiological study and radiofrequency ablation

A 60-year-old woman with daily episodes of palpitations was referred to our centre for electrophysiological study and ablation. All anti-arrhythmic drugs were discontinued at least five half-lives prior to the study. Three electrode catheters were inserted via the right femoral vein. One 6 Fr quadripolar electrode catheter (2-5-2 mm Josephson, Bard Electrophysiology) was positioned at the His-bundle (HB) region. A 6 Fr 10-polar catheter (2-5-2 mm Livewire, St Jude Medical Inc.) was used to map the right atrium (RA) and the CS. A 7 Fr deflectable catheter with a 4 mm tip electrode and 2-5-2 mm spacing between electrodes (temperature-guided; Cordis Webster Inc.) was used for mapping and RF current application. The surface electrocardiogram (ECG) and intra-cardiac electrograms were recorded utilizing a computerized multichannel system (BARD Laboratory System).

Baseline ECG recordings demonstrated a slow (cycle length 544 ms) long RP tachycardia. The P-wave morphology was biphasic in inferior leads (II, III, and aVF) and in lead V1 and positive in leads I and aVL (Figure 1A). The diagnosis of AT was established using commonly accepted diagnostic criteria. 1 The tachycardia was sensitive to adenosine (12 mg) and was easily induced and terminated by programmed atrial stimulation. Conventional RA mapping during the tachycardia demonstrated the earliest local atrial activation (preceded the onset of the P-wave by 18 ms) at the superior aspect of the HB region (Figure 1B). Due to the high risk of atrioventricular (AV) block and based on the algorithm proposed by Das et al., 2 we considered reasonable to map retrogradely the non-coronary aortic cusp and attempt one RF application at the site of earliest activation. In the case of an unsuccessful attempt (no termination within 10 s), we would proceed to left atrial (LA) septal mapping through a trans-septal puncture. Aortic root mapping was performed via the right femoral artery under systemic anticoagulation with intravenous administration of heparin (60 IU/kg) following aortography and identified the earliest local activation during the AT (preceded the onset of the P-wave by 51 ms) within the non-coronary aortic cusp (Figure 2A). A single RF application (25 W, 50 °C) completely eliminated the AT within 2.5 s (total application time 120 s) (Figure 2B). Fluoroscopically, the successful site of ablation (the area of the non-coronary aortic cusp) was located slightly supero-posterior to the site of HB recording in the RA (Figure 3). After ablation, the AT was not inducible with extensive programmed stimulation with isoproterenol.

Discussion

The present report describes the case of a successfully ablated focal AT originating within the non-coronary aortic cusp. The main electrophysiological characteristics of the tachycardia included: (i) incessant pattern; (ii) easy induction and termination by atrial stimulation; (iii) earliest RA activation at the para-hisian area; and (iv) termination by adenosine. Mapping of the aortic root identified the earliest atrial activation within the non-coronary aortic cusp, and a single RF application at this site successfully terminated the AT.
Enhanced automaticity, triggered activity, and micro-re-entry are considered as the three major mechanisms of focal ATs.\textsuperscript{1,5} Adenosine suppresses enhanced automaticity by hyperpolarizing membrane potential and terminates cAMP-dependent triggered activity and calcium-dependent micro-re-entry.\textsuperscript{5} On the contrary, macro-re-entrant ATs are adenosine insensitive.\textsuperscript{5} Adenosine has been shown to terminate most focal ATs arising from either the crista terminalis or septal locations including non-coronary aortic cusp.\textsuperscript{3,5} However, septal ATs are highly sensitive to adenosine and display different electrophysiological characteristics from free-wall ATs.\textsuperscript{6} In animal studies, atrial cells along the AV junction, including the slow pathway region, exhibit nodal-like action potentials and are highly sensitive to adenosine.\textsuperscript{7} In the present case, the tachycardia was reproducibly induced and terminated by atrial stimulation and terminated following administration of adenosine. Based on these findings, the tachycardia mechanism was due to either triggered activity or micro-re-entry.

Limited data regarding ATs arising from the non-coronary aortic cusp exist in the current literature.\textsuperscript{2–4} The surface ECG is of limited value in the discrimination of a non-coronary aortic cusp or a para-hisian origin of the AT.\textsuperscript{2–4} Das et al.\textsuperscript{2} have recently reported that 7 out of 10 patients with AT exhibiting the earliest RA activation at the peri-AV nodal region were finally successfully ablated in the non-coronary aortic cusp. Ouyang et al.\textsuperscript{3} have demonstrated that the mean difference of the earliest atrial activation in the non-coronary aortic cusp and the RA was only 12.2 $\pm$ 6.9 ms in nine patients with an AT originating from the non-coronary aortic cusp. In this study, five patients had been initially ablated at the para-hisian area without success.\textsuperscript{3}

The anatomy of the interatrial septum is complex, leading to potential difficulties in identifying the site of the AT origin in this region. The non-coronary aortic cusp is located more posterior in relation to other cusps and forms the superior margin of the interatrial septum.\textsuperscript{3,4,8} Because of the fairly thick and immediately adjacent atrial myocardium of the atrial septum, large atrial electrograms
are usually recorded when mapping the non-coronary aortic cusp. Yamada et al. suggested that the non-coronary aortic cusp exhibits direct musculature connection with the HB region and the mid-interatrial septum in the LA. In animal studies, RF catheter ablation within the non-coronary aortic cusp has been shown to create lesions at the LA septum located between the floor of the fossa ovalis.

Figure 2 (A) Tracings of the surface leads I, II, III, aVF, V1, and V6 and intra-cardiac recordings during left atrial mapping demonstrated the earliest local atrial activation (preceded the onset of the P-wave by 51 ms) within the non-coronary aortic cusp (ABL 1/2). (B) Ablation at this site terminated the tachycardia within 2.5 s.

Figure 3 (A) Aortic root angiography (left anterior oblique view) showing the close proximity of the non-coronary cusp (NCC) with the His-bundle region (HB). (B) Fluoroscopic left anterior oblique and (C) right anterior oblique projections showing the ablation catheter (ABL) within the non-coronary aortic cusp. RA, right atrium; RV, right ventricle.
and the mitral annulus, an area that is difficult to map using either a retrograde or a trans-septal puncture approach. These findings are strongly indicate the close anatomical proximity of the non-coronary aortic cusp with the interatrial septum.

Pre-ablation aortography and continuous visualization of the ablation catheter during RF delivery should always be used in order to minimize the risk of damaging the coronary arteries. Understanding the exact anatomy of the coronary cusps is also of paramount importance before RF ablation. High-output pace mapping may be helpful to distinguish non-coronary from right coronary cusp. In the former results in atrial capture, whereas in the later in ventricular capture. Ablation within the non-coronary aortic cusp as long as the catheter is positioned and directed in a straight posterior manner or a leftward manner away from the commissure with the right coronary cusp will not damage the conduction system. In previous studies, there were no complications including embolic events following non-coronary cusp RF ablation. However, based on the clinical experience during ablation of other left-sided arrhythmias, a more prudent strategy is to use cooled RF ablation or cryoablation within the non-coronary cusp in order to minimize the risk of thrombus formation. In conclusion, the diagnosis and RF ablation of focal ATs arising from the non-coronary aortic cusp are very challenging for the physicians. Mapping of the non-coronary cusp should always be considered when the earliest right atrial activation is recorded at the para-Hisian area in order to diminish the risk of AV block by inappropriate RF deliveries near the HB region.

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**References**