Journal of Arrhythmia 30 (2014) 197–200

Contents lists available at ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

Case Report

Adenosine-sensitive atrial tachycardia originating from the mitral annulus–aorta junction

Naoto Kino, MD, Toshiya Kurotobi, MD, PhD*, Daisuke Tonomura, MD, Kentaro Yano, MD, Keiichi Furubayashi, MD, PhD, Takao Tsuchida, MD, PhD, Yoshihisa Shimada, MD, PhD, Hitoshi Fukumoto, MD, PhD

Cardiovascular Division, Shiroyama Hospital, 2-8-1, Habikino, Habikino City, Osaka 583-0872, Japan

ARTICLE INFO

Article history:
Received 15 April 2013
Received in revised form
7 July 2013
Accepted 26 September 2013
Available online 16 November 2013

Keywords:
Atrial tachycardia
Left aortic coronary cusp
Ablation
Adenosine
His bundle

ABSTRACT

Here we report a case of adenosine-sensitive focal atrial tachycardia that was successfully ablated from the left atrium at a site located adjacent to the posterior site of the left coronary cusp in spite of failed ablations from the superior-septal right atrium and non-coronary aortic cusp. In the case of an unsuccessful ablation from the right superoseptum and non-coronary aortic sinus, the left atrial septum represents a potential optional site for ablation.

1. Introduction

Many adenosine-sensitive focal atrial tachycardias (ATs) originating from the vicinity of the His bundle can be delineated from the earliest activation site in the right atrium around the His bundle. Recent reports have demonstrated that in some cases, these types of AT can be successfully ablated from the non-coronary cusp (NCC) [1,2].

Here we report a case of adenosine-sensitive focal AT that was successfully ablated from the left atrium at a site located adjacent to the posterior site of the left coronary cusp (LCC) in spite of failed ablations from the superior-septal right atrium and NCC.

2. Case report

A 61-year-old woman, without any significant past history of illness, who was suffering from frequent palpitations since the age of 20 was referred to our institution. Findings from a chest X-ray and 12 lead electrocardiogram (ECG), laboratory data, and echocardiographic data were normal.

After obtaining informed consent, an electrophysiological study and catheter ablation procedure were performed. All antiarrhythmic drugs were discontinued 1 week prior to the procedure. Four electrode catheters were inserted via the right femoral and cervical veins, and positioned in the coronary sinus (6Fr 20 pole catheter), high right atrium (5Fr quadripolar catheter), right ventricular apex (5Fr quadripolar catheter), and His bundle region (5Fr 10 pole catheter). The intracardiac electrograms and surface ECGs were continuously monitored and recorded using EP Workmate (St. Jude Medical, Minneapolis, Minnesota, USA).

No retrograde conduction was observed during ventricular pacing. After administering isoproterenol (ISP) infusion (0.5 μg/min), we detected retrograde AV nodal conduction with the earliest atrial activation recorded at the His bundle region. Under the ISP infusion, an AT (cycle length: 385 ms) (Fig. 1) was easily induced by atrial extrastimulus and/or burst pacing from the right atrium without a jump-up in the atrial-His interval. The AT episodes were inducible during stimuli of basic cycle length, and spontaneous self-termination was observed without atrial premature beats. No negative correlation was observed between the coupling interval of the atrial extrastimulus that induced the tachycardia and the post-extrastimulation return cycle. The AT persisted irrespective of spontaneous AV block. An intravenous bolus of 2.5-mg adenosine-5′-triphosphate (ATP) administered during tachycardia reproducibly terminated the AT without AV block. AV dissociation was observed during ventricular overdrive pacing in AT. Tachycardia was also induced by a ventricular extrastimulus with a V-A-A-V pattern. The activation sequence during tachycardia differed from that observed during sinus rhythm and retrograde AV nodal conduction. In the right atrium, the earliest activation site was recorded at the proximal His site that preceded the onset of the P-wave on the 12 lead ECG by

* Corresponding author. Tel./ fax: +81 6578 5557.
E-mail address: drk21cent@gmail.com (T. Kurotobi).

1880-4276/$ - see front matter © 2013 Japanese Heart Rhythm Society. Published by Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.joa.2013.09.006

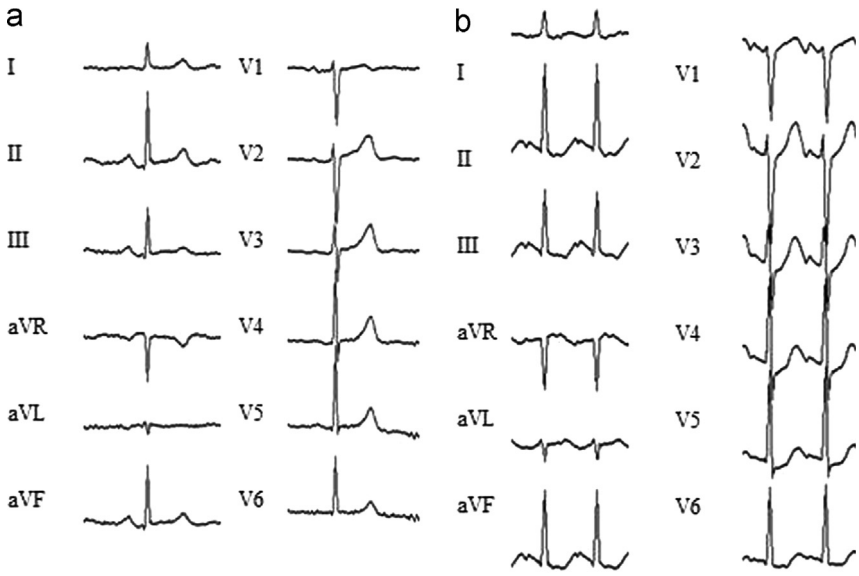

Fig. 1. Surface ECG during sinus rhythm (a) and atrial tachycardia (AT) (b). The P-wave polarity during the AT was positive in leads II, III, aVF, and V1, and negative in leads I and aVL. The AT cycle length was 385 ms.

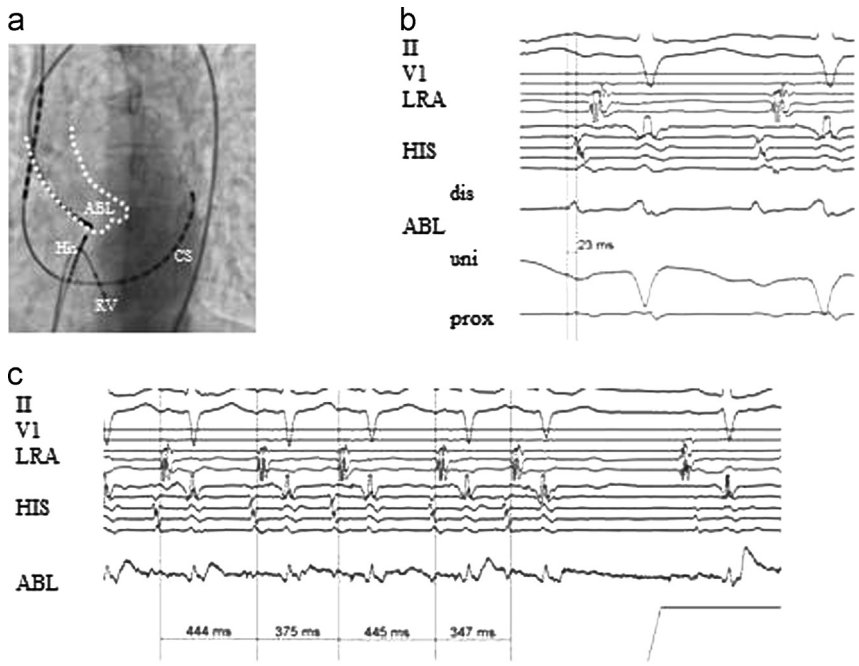

Fig. 2. Findings from the ablation at the non-coronary cusp (NCC). (a) The ablation catheter is located in the NCC adjacent to the His catheter in the left anterior oblique fluoroscopic view. (b) The body-surface and intracardiac ECGs of the AT. The earliest local atrial potential preceded the onset of the P-wave by 23 ms. (c) Ablation from that site could temporarily terminate the AT following an unstable AT cycle length. LRA, lateral right atrium; His, His bundle; ABL, ablation site; dis, distal electrode; uni, unipolar potential; prox, proximal electrode; CS, coronary sinus and RV, right ventricle.

22 ms. RF energy delivered with a 4-mm tip catheter (30 W, maximal temperature of 55 °C) was applied to the earliest activation site in the right atrium, but it could not successfully terminate the AT. Therefore, we mapped the aortic NCC using the retrograde approach (Fig. 2). RF energy applications at that site transiently terminated the AT following an unstable AT cycle length, but pacing recurrently induced the AT, which then transiently terminated. The subsequent AT activation sequence and cycle length did not change significantly. Despite carefully searching the LCC, we could not find an earlier activation site. Finally, we mapped the left atrium using the transseptal approach. The earliest activation site in the left atrium was recorded in the area just behind the LCC (Fig. 3). RF energy application at this site successfully terminated the AT, and the AT could no longer be induced.

3. Discussion

A previous report demonstrated an adenosine-sensitive reentrant AT not involving an AV nodal pathway in the vicinity of the apex of Koch's triangle, which could be ablated at the earliest atrial activation site in the right supero-septal region [3]. Otomo et al. reported a “left-variant” form of an adenosine-sensitive AT due to focal reentry involving the leftward AV nodal transitional area