Journal of Arrhythmia 31 (2015) 318–322

Contents lists available at ScienceDirect

Journal of Arrhythmia

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

Case Report

A case of paroxysmal atrial fibrillation with a non-pulmonary vein trigger identified by intravenous adenosine triphosphate infusion

Masahiro Esato, MD, PhD[a,*], Naoto Nishina, MD[a], Yoshitomi Kida, MD[a], YeongHwa Chun, MD[b]

[a] Department of Arrhythmia, Ijinkai Takeda General Hospital, 28-1, Minamimachi, Ishidamori, Fushimiku, Kyoto City, Kyoto 601-1495, Japan
[b] Arrhythmia Care Center, Takeda Hospital, Kyoto, Japan

ARTICLE INFO

Article history:
Received 1 May 2014
Received in revised form
16 January 2015
Accepted 13 March 2015
Available online 22 April 2015

Keywords:
Atrial fibrillation
Adenosine triphosphate
Nonpulmonary vein trigger
Circumferential pulmonary vein isolation

ABSTRACT

A 54-year-old woman was referred to our institution with frequent chest discomfort and was diagnosed with drug-refractory paroxysmal atrial fibrillation. Radiofrequency catheter ablation (RFCA) was performed using a three-dimensional electroanatomic mapping system. After completion of left and right circumferential pulmonary vein isolation (CPVI), an intravenous bolus of adenosine triphosphate (ATP, 20 mg) was administered to evaluate the electric reconduction between the pulmonary vein (PV) and left atrium (LA). Although no PV–LA reconduction was observed, atrial fibrillation (AF) was reproducibly induced. As the duration of AF was very short (< 20 s), no further RFCA to the LA was performed. One month later, the patient presented with frequent atrial tachyarrhythmias (ATs), and RFCA was repeated. Although no electric reconduction was observed in the left- or right-sided PVs, incessant ATs and AF were induced after an intravenous bolus administration of ATP. The earliest atrial activation site initiating ATs was consistently identified from electrodes positioned in the superior vena cava (SVC), and both ATs and AF were no longer inducible after electric isolation of the SVC. ATP-induced PV/non-PV ectopy may be a marker of increased susceptibility to autonomic triggers of AF and could potentially predict recurrent AF after CPVI.

1. Introduction

Although the pulmonary veins (PVs) represent the predominant source of atrial fibrillation (AF), non-PV triggers play an important role in initiating and maintaining AF in approximately 20% of cases [1–3]. Episodes of atrial tachyarrhythmias (ATs) and AF originating from non-PV triggers are often unpredictable and difficult to identify, owing to their transient duration and diverse locations. Furthermore, the precise location may remain unknown, even with the use of a three-dimensional electroanatomic mapping system (3DEAM).

Several studies have indicated that an intravenous injection of adenosine, in the form of adenosine triphosphate (ATP), can induce the transient reconnection of isolated PVs after electric isolation, consistent with unmasking dormant conduction between the PVs and the left atrium (LA) [4,5]. Furthermore, ATP, when given as an intravenous bolus, can induce AF [6]. In addition, several studies have recently described the usefulness of an ATP injection for inducing and identifying PV and/or non-PV triggers after circumferential pulmonary vein ablation (CPVI) [7,8]. Here, we describe a case of paroxysmal AF originating from a non-PV trigger, which was precisely identified using ATP infusion and successfully treated using radiofrequency catheter ablation (RFCA).

2. Case report

A 54-year-old woman was referred to our institution because of frequent episodes of palpitation and chest discomfort. Although she was taking several antiarrhythmic drugs, a symptomatic 12-lead electrocardiogram (ECG) revealed AF, and RFCA was indicated. After obtaining informed consent from the patient, RFCA was performed under deep sedation/analgesia using propofol and dexmedetomidine. Two circular mapping catheters (EPstar Libero, Japan Lifeline Inc., Tokyo, Japan) were placed in the superior and inferior PVs, respectively, via a transseptal puncture site, and the left and right-sided ipsilateral PVs were circumferentially and extensively ablated, using 3DEAM (CARTO, Biosense Webster, Inc., Diamond Bar, CA, USA) under electrophysiological guidance. After the initial electric isolation of all 4 PVs, a 20-mg ATP bolus was injected to provoke dormant PV conduction during coronary sinus pacing. Although no electric reconduction was observed in the left or right PVs, AF was reproducibly induced (Fig. 1). The duration of AF was very short (< 20 s), and no further AF episodes were observed, even during an

* Corresponding author. Tel.: +81 755726331; fax: +81 755718877.
E-mail address: m-esato@takedahp.or.jp (M. Esato).

http://dx.doi.org/10.1016/j.joa.2015.03.004
1880-4276/

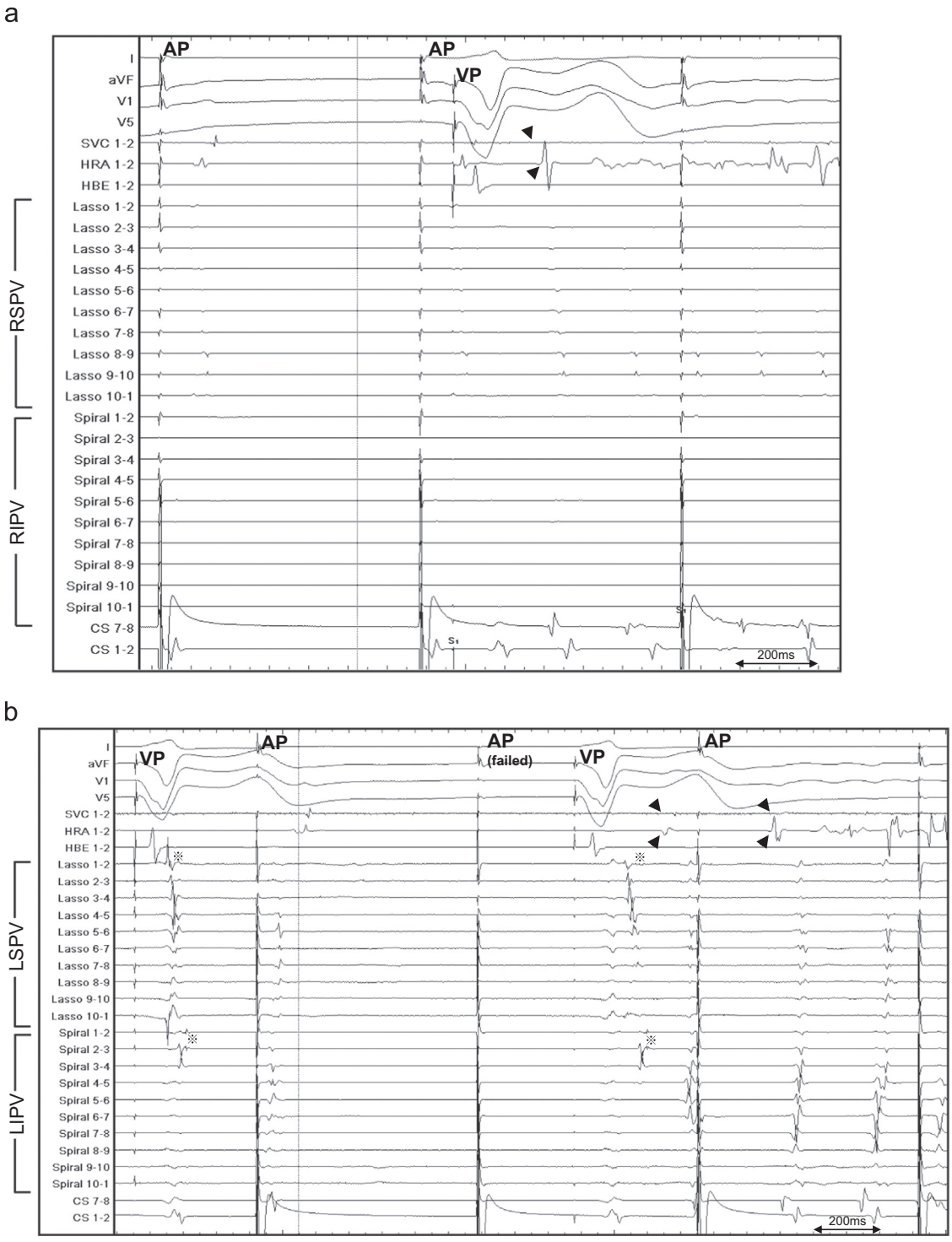

Fig. 1. Induction of AF via ATP infusion. Two circular catheters were positioned at both the superior and inferior right (a) and left (b) PVs. The asterisk indicates electrically dissociated PV potentials. The earliest atrial activation site initiating AF was obtained from the electrodes (black arrow heads) positioned between the HRA and SVC. RSPV, right superior pulmonary vein; RIPV, right inferior pulmonary vein; LSPV, left superior pulmonary vein; LIPV, left inferior pulmonary vein; AP, atrial pacing; VP, ventricular pacing; AF, atrial fibrillation; ATP, adenosine triphosphate; PV, pulmonary vein; HRA, upper right atrium; and SVC, superior vena cava.

intravenous drip infusion of isoproterenol (ISP: dosage up to 5 μg/min), so no further mapping or RFCA was performed, with the exception of linear ablation at the cavotricuspid isthmus.

However, the patient was later re-admitted with frequent palpitation symptoms similar to those exhibited prior to the initial RFCA. Despite oral class I antiarrhythmic drug and beta-blocker treatment, ECG revealed incessant ATs and symptomatic AF (Fig. 2), so repeat RFCA was performed. Intravenous bolus infusion of ATP did not produce dormant electrical reconduction in either the left or right PVs, but incessant AF/ATs were induced. The earliest atrial activation site initiating incessant AF/ATs was consistently observed from the electrodes placed between the superior vena cava (SVC) and the upper right atrium, which was the location identical to that observed in the initial procedure. To identify the precise ectopic origin, a circular multielectrode-mapping catheter was positioned superior to the atriocaval junction within the SVC based on venography. During