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Successful ablation of frequent atrial premature beats from non-coronary aortic cusp with remote magnetic navigation

Ahmed Taha Hussein Abdelwahed [a,b,1], *Heikki Mäkynen* [a,1], *M.J. Pekka Raatikainen* [c,*,1]

[a] *Heart Center Co, Tampere University Hospital, Tampere, Finland*
[b] *Zagazig University Hospital, Zagazig, Egypt*
[c] *Department of Medicine, Division of Cardiology, Central Finland Health Care District and University of Eastern Finland, Keskussairaalantie 19, FI-40620 Jyväskylä, Finland*

ARTICLE INFO

Article history:
Available online 21 October 2015

Keywords:
Atrial premature beats
Non-coronary cusp
Magnetic navigation
Catheter ablation

ABSTRACT

A 59-year-old female with structurally normal heart was admitted to our hospital for treatment of highly symptomatic, drug refractory atrial premature beats (APB). ECG revealed atrial parasystolic trigeminy. The arrhythmogenic focus was mapped and ablated using magnetic remote navigation and 3D electroanatomical mapping system. To our knowledge, this is the first report on successful ablation of frequent APBs in the non-coronary aortic cusp.

Introduction

Atrial premature beats (APB) are commonly encountered in clinical practice. They have traditionally been considered benign, but recent evidence indicates that frequent APBs are a strong predictor of atrial fibrillation development and may be associated with increased risk of cardiovascular death in general population [1]. In addition, frequent APBs may cause cardiomyopathy [2]. We present a 59 year-old female with frequent highly symptomatic APBs. Her heart was structurally normal heart and there was no evidence of any sustained tachyarrhythmias. To our knowledge, this is the first report on successful ablation of frequent APBs in the non-coronary aortic cusp.

Case report

A 59-year-old female with a long history of highly symptomatic frequent APBs presented to our hospital for catheter ablation. She had been treated ineffectively with several beta blockers and calcium antagonists. Flecainide was stopped because of intolerable adverse effects. Twelve-lead ECG showed normal sinus rhythm with frequent APBs. P wave was negative in inferior leads II, III, avF, positive in avR and V1–V3,

* Corresponding author. Tel.: +358 50 3169001.
E-mail address: pekka.raatikainen@ksshp.fi (M.J.P. Raatikainen).
Peer review under responsibility of Indian Heart Rhythm Society.
[1] All authors have read the manuscript and contributed to the work.
http://dx.doi.org/10.1016/j.ipej.2015.10.004

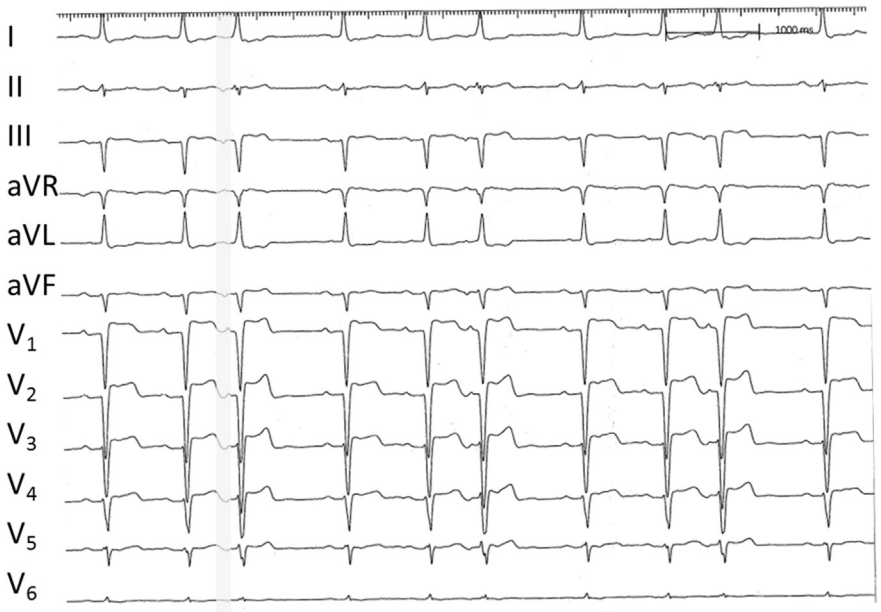

Fig. 1 – Twelve lead ECG showing frequent atrial premature beats. P wave was negative in inferior leads II, III, avF, positive in avR and V_1–V_3, indifferent in lead I, avL and V_4–V_6 (shaded area).

indifferent in lead I, avL and V4–V6 (Fig. 1). APB burden in repeated Holter recordings was 20–40% (29000 APBs in the last Holter recording). Neither atrial fibrillation nor any other sustained supraventricular tachyarrhythmia was detected. Clinical examination and thyroid function were normal. Transthoracic echocardiography revealed no structural abnormalities. Left ventricular ejection fraction (LVEF) was 76%.

Antiarrhythmic medication was discontinued two days before the electrophysiological examination. Diagnostic catheters were placed in the coronary sinus and right ventricular apex vie right femoral vein. Mapping and ablation was performed retrogradely via right femoral artery with a 3.5 mm tip open irrigated ablation catheter (Biosense Webster Navistar RMT, Diamond Bar, CA, USA) using remote magnetic navigation (Epoch, Stereotaxis Inc., St Louis, MO, USA) and 3D electroanatomical mapping system (Carto3, Biosense Webster). Mapping was started from the right atrium because of the P wave morphology and coronary sinus activation sequence. Fast anatomical activation mapping demonstrated that the earliest activation in the right atrium was in the vicinity of the His bundle (Fig. 2). No ablation attempt was made although local activation at this site preceded the P wave

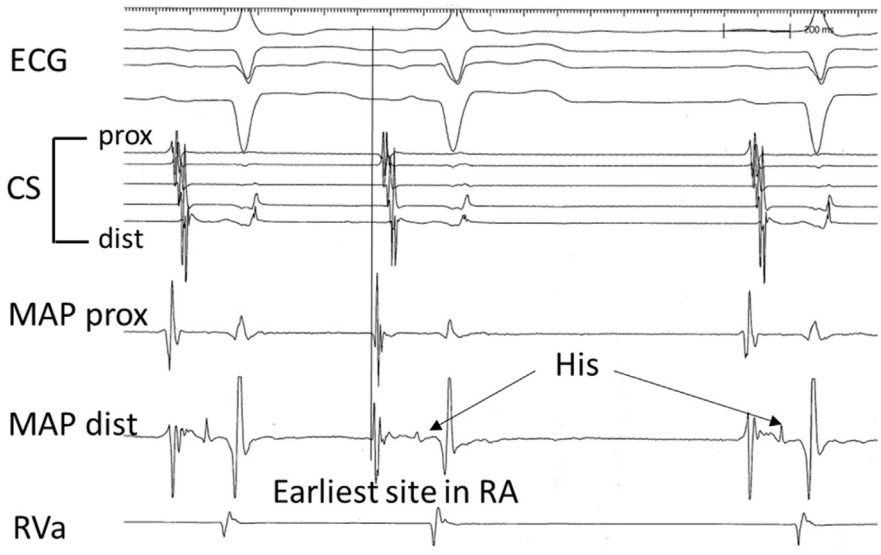

Fig. 2 – During the atrial premature beat coronary sinus activated from proximal to distal. The earliest local atrial activation in the right atrium was detected close to the His bundle. At this site no ablation was performed as the His deflection was clearly visible. Shown are surface ECG leads I, III, avF and V_1. CS = coronary sinus, His = His deflection, MAP = mapping catheter, RVa = right ventricular apex.