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Focal atrial tachycardias related to non-incisional low voltage areas resulting from an undiscovered atrial septal defect

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Abstract

A 50-year-old woman was admitted for frequent palpitation with a past medical history of surgical secundum Atrial Septal Defect (ASD) repair. The Holter showed ectopic atrial burden as high as 70.3%, and cardiac tomography discovered an inferior sinus venosus ASD. The electrophysiological study confirmed two focal origins of ATs located at the low posterolateral Right Atrium (RA) and high crista terminalis, and both were associated with non-incisional low voltage areas. Ablation succeeded in reducing the ectopic atrial burden to 0.44%. Another surgical repair of ASD was strongly recommended because of persisting left to right shunt.

Keywords

Electrophysiology; Atrial tachycardia; Atrial septal defect; Low voltage areas; Ablation.

Introduction

Atrial tachycardias (ATs) are common in patients after surgical repair of atrial septal defects, the mechanism of which is most often macro-reentry [1,2], occasionally focal origin related to atriotomy scars or previous ablation lines [2,3], while the rest was scarcely documented. We report a case of dual focal post-incisional atrial tachycardias related to non-incisional scars.

Case Report

A 50-year-old female presented with frequent palpitation for the past three months. 24-hour-Holter revealed 84,214 premature atrial complexes (PACs) /119,717 total beats (70.3%) and frequent paroxysmal atrial tachycardias, which were irresponsive to metoprolol. She had surgically repaired a secundum atrial

septal defect (ASD) 45 years ago. She was admitted, and the physical examination and laboratory tests were within normal range. Echocardiography displayed atria enlargement (left atrial anteroposterior diameter 42 mm, right atrial diameters 51 × 60 mm), Right Ventricular (RV) enlargement (RV outflow tract diameter 37 mm), severe tricuspid regurgitation and mild pulmonary hypertension (estimated systolic pulmonary artery pressure (sPAP) 48 mmHg), with a normal Left Ventricular (LV) diameter and Ejection Fraction (EF) (LV end-diastolic diameter 38 mm and LVEF 60%). Cardiac cinematic tomography revealed an inferior sinus venosus ASD (17 × 12 mm) with no anomalous pulmonary venous drainage.

Her Holter and Electrocardiograms (ECGs) exposed mainly two ectopic atrial origins. The P' wave of the first origin was negative in inferior leads, positive-negative in lead V1, negative through V2-6, the width of which was nearly the same as the sinus P wave, indicating a low Crista Terminalis (CT) origin. The P' wave of the second origin exhibited the same vector of the sinus P wave but was narrower, suggesting a high and septal origin near the CT (Figure 1). A thorough discussion together with cardiac surgeons and the patient concluded an Electrophysiology Study (EPS) as the first step to treatment.

At the beginning of EPS, she was in an intermittent AT with a Cycle Length (CL) of 524 ms or frequent PACs (coupling interval 466 ms) in accordance with the first P' wave's shape from ECGs. Repetitive spontaneous onset, cool down before spontaneous termination, and variability in cycle length all argue for automaticity. An area of low bipolar voltages (<0.10 mV) was mapped lateral to the mid-inferior crista terminalis where the inferior vena cava joined the RA (Figure 2), using a PentaRay catheter (Biosense Webster, Inc., Diamond Bar, CA, USA). The earliest atrial activation (EAA) was also within the low voltage area (LVA) at the low posterolateral RA. The AT disappeared, but PACs with higher amplitude of P' waves in the inferior leads persisted after the first ablation with a Thermo Cool® Smart Touch catheter (Biosense Webster, Inc., Diamond Bar, CA, USA) (Figure 2).

EAA was remapped to be higher than the first target, still in the LVA. Ablation was continued within the LVA until PACs finally vanished. The following isoprenaline test provoked another faster AT, with a CL of 398-498 ms and rapid 1:1 conduction to the ventricle. Although difficult to discern, the P' wave's shape resembled the second AT in the Holter. Its mechanism was also arguably automaticity. The EAA was pinpointed at the high CT adjacent to an LVA (Figure 2), with double potentials detected nearby. Focal ablation eliminated the second AT quickly (Figure 2), and the sinus rhythm withstood the 45-minute observation, including the isoprenaline test and repetitive atrial and ventricular electric stimulations. Surgeons recommended selective ASD repair, only to be refused by the patient.

Her palpitation ameliorated substantially afterward, and another Holter three months later displayed only 401 PACs/91,358 total beats (0.44%). One year later, she presented at the outpatient for increasing shortness of breath. Echocardiography showed neither decrease of the atria's diameters nor lowering of sPAP. Surgery repair was proposed again, while she still hesitated at the last follow-up.

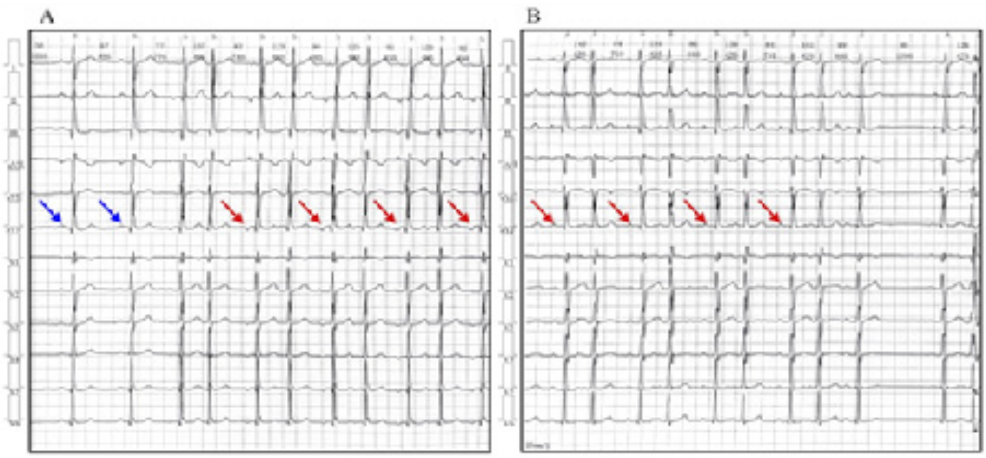

Figure 1: Two focal atrial ectopies from the patient's Holter. (A) The P' wave shape of the first premature atrial beat: blue arrow indicated sinus beat, and red arrows pointed at the first atrial ectopies. (B) The P' wave shape of the second premature atrial beat as indicated by red arrows. See text for detailed discussion.

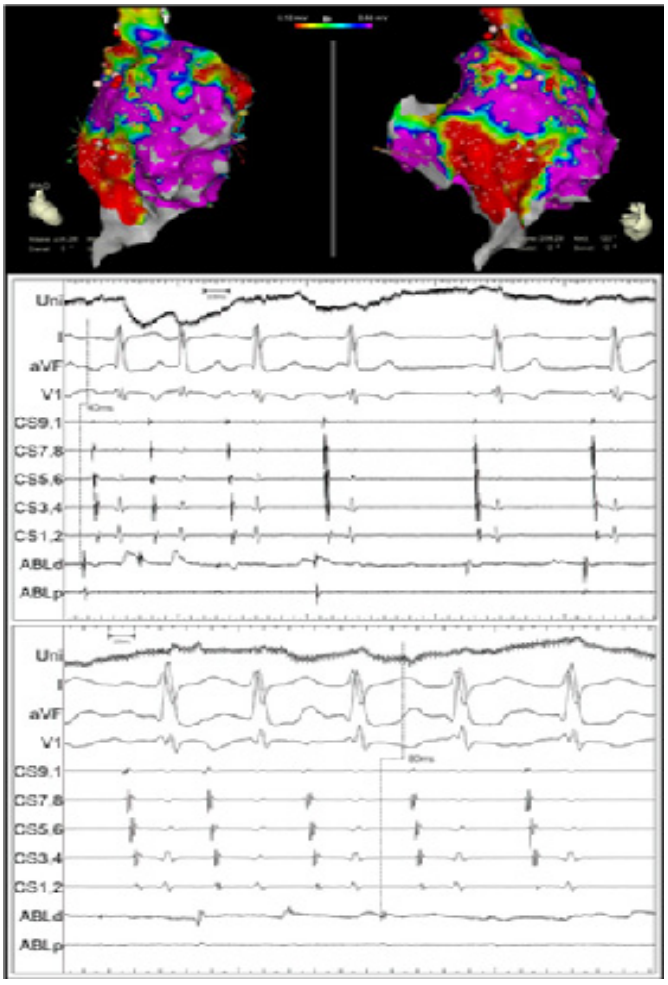

Figure 2: Ablation targets of the two atrial tachycardias (ATs). Upper panel - Targets shown in right anterior oblique (RAO) views (left in RAO 45°, right in RAO 122°). The targets of the first AT clustered within the low posterolateral right atrium Low Voltage Area (LVA) and those of the second AT were at high crista terminalis near another LVA. Middle panel – Intracardiac electrograms of the first target of AT 1. The earliest atrial activation (EAA) was 40ms ahead of the P' wave. Lower panel – Intracardiac electrograms of the target of AT 2. The EAA was 80ms preceding the P' wave. The P' wave looked different from the electrocardiogram in figure 1, which was arguably caused by the fusion with T wave.

Discussion

Most atrial tachycardias after surgical ASD repair were macro-reentries, dominated by cavotricuspid isthmus-related and incisional scar-related circuits [1,2]. Focal atrial tachycardias, however, have been seldomly reported, many of which were related to a slow conduction zone from the previous incision or ablation lines [2,3]. The highlight of this case was that both focal ectopies pertained to non-incisional LVAs.

The patient underwent the secundum ASD repair at five years old, leaving noticeable incisional scars 45 years later neither on cinematic tomography nor on electroanatomic mapping of the right atrial free wall. LVAs in this case were considered the manifestation of remodeling from right atrial volume overload. Recent research on epicardium mapping in patients with right atrial volume overload exposed the RA was predisposed to electrical remodeling, especially within the intercaval region. The explanation was speculated that pectinate muscles clustered in the intercaval region and acted as RA volume reserve during preload increasing [4]. The LVA establishing the first AT was at the junction of the intercaval region and right atrial free wall and was close to the inferior sinus venosus ASD. Moreover, the left to right shunt of flow might constantly hit that area, gradually prompting electric remodeling (Figure 3). The first AT witnessed that its EAA changed within the LVA during ablation, and complete elimination necessitated multiple ablations, both of which implied the close relationship existed between the first AT and the LVA and the exact origin might locate beyond the endocardium. The second AT was at high CT, just at the side of another LVA. CT was both the most prevalent origin of focal right atrial tachycardia [5] in patients with normal hearts and the most probable endocardial site predisposed to electrical remodeling from right atrial stretch [6]. Likewise, we inferred that the second AT was attributed to non-incisional LVA. Focal ablation eradicated this AT, implying an endocardial origin. What should be emphasized here was that LVA did not necessarily mean scar tissue [7]. Conduction delay or conduction block within or near the LVA initiated the tachycardia. It was a pity that intracardiac echocardiography was not used to explore the exact structures around both targets. Some postoperative atrial tachycardias in ASD patients recurred late with novel origins [8] fortunately, her sinus rhythm thrived at least to the last follow-up at one year. Although the burden of atrial ectopies decreased principally, the patient suffered shortness of breath late after the ablation with no reduction in her atrial diameters due to the patent ASD. The inferior sinus venosus ASD was challenging to detect in transthoracic echocardiography, while transesophageal echocardiography or contrast-enhanced cinematic tomography would be more capable [9]. We did not know why the inferior sinus venosus ASD was left untreated in her first surgery; however, she evidently required another surgery as soon as possible.

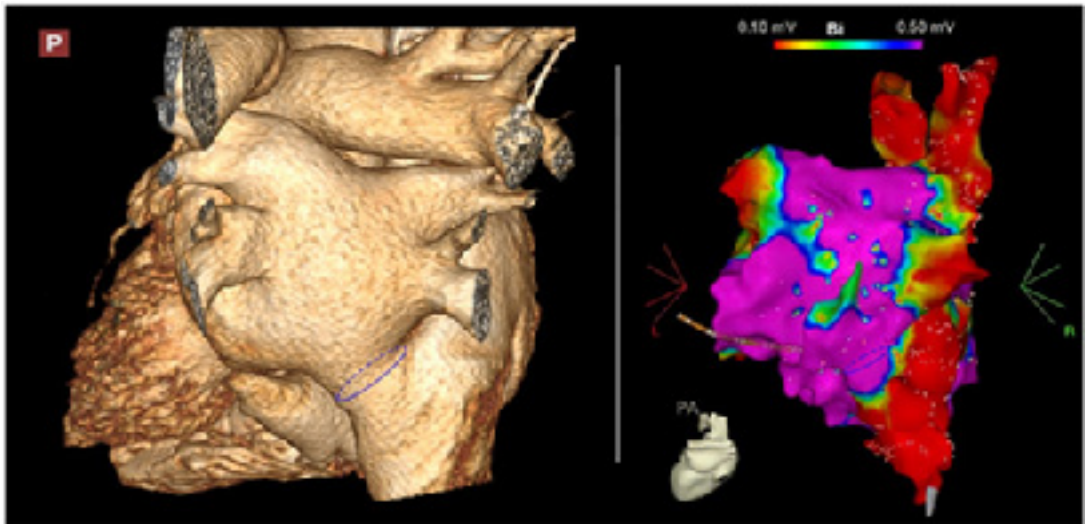

Figure 3: Locational adjacency between the inferior sinus venosus atrial septal defect and the low posterolateral low voltage area. Left and right pics showed the reconstruction from cardiac cinematic tomography and bipolar voltage electroanatomic mapping both in the posteroanterior views. The blue circles illustrated the inferior sinus venosus atrial septal defect.

Conclusion

Although rare, focal atrial tachycardias after ASD repair can be related to non-incisional low voltage areas. Other etiologies should be considered if atrial diameters could not diminish with the decrease of atrial ectopic burden after ablation.

Declarations

Ethics statement: Written informed consent was provided by this patient for the publication of images or data in this article.

Author contributions: Shu Zhang and Qi Sun conducted the EPS and collected all the clinical data. Fengyuan Yu wrote the manuscript. All authors contributed to the article and approved the submitted version.

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