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A Difficult Case of Atrial Tachycardia

Meruka Hazari MD, Deepak Saluja MD

Rutgers- Robert Wood Johnson Medical School.

Abstract

A 60-year-old male with a history of prior ablation of typical cavotricuspid isthmus-dependent atrial flutter presented to us with recurrent palpitations. The surface 12-lead EKG was consistent with atrial tachycardia, for which an ablation was planned. The patient was found to have multiple marcroreentrant left atrial tachycardias utilizing areas of scar as substrate. Isthmuses of tissue with fractionated electrograms associated with scar were targeted for ablation.

Introduction

While well known in patients with prior ablation or cardiac surgery, left atrial tachycardias in patients without previous surgery or ablation are much less common. The available literature suggests that spontaneous scarring in characteristic areas of the left atrium produces the substrate for marcroreentry, which commonly involves the mitral valve and right-sided pulmonary veins. These rhythms can be successfully ablated by targeting isthmuses of slow conduction created either between adjacent areas of scar or between an area of scar and an anatomical barrier. We describe the electrophysiological findings and results of ablation in a patient with scar-related macroreentrant left atrial tachycardias without prior surgery or ablation.

Case Presentation

A 64 year-old man with a history of hyperlipidemia and coronary artery disease with a previous stent to the right coronary artery for angina, and a prior ablation for typical cavotricuspid isthmus-dependent atrial flutter presented with intermittent palpitations. An echocardiogram showed an ejection fraction of 60% with normal valvular function, a normally sized left atrium (LA), and no wall motion abnormalities. A 12-lead EKG of the presenting rhythm (Fig. 1) showed an atrial tachycardia (AT) with an atrial cycle length of approximately 220ms.

The patient presented to the electrophysiology laboratory in sinus rhythm. An AT with a cycle length and morphology similar to that shown in Fig 1 was induced. The earliest activation on the decapolar coronary sinus (CS) catheter was seen in CS 3-4 (Fig 2A). Ablation and circular mapping catheters were delivered to the LA via double transseptal punctures. Examination of the endocardial LA revealed large areas of decreased voltage (<0.5mV) on the anterior, septal, and posterior aspects of the LA, as well as on the roof (Fig 3A, B, and C).

An activation map of the initial tachycardia (AT1) suggested a double-loop pattern of reentry with an isthmus of tissue between two areas of dense scar near the posterior right-sided pulmonary vein carina acting as the common pathway (see Fig 4A and Propagation video #1). Ablation lesions were applied connecting areas of scar surrounding the circuit (Fig 3C, arrows), first resulting in variable exit block (Fig 5B), then complete isolation of the circuit with resumption of sinus rhythm in the body of the atrium (Fig 5C). Ablation of fractionated EGMs in the common isthmus (Fig 3C, asterisk) resulted in termination of the isolated tachycardia (Fig 5C).

Burst pacing from the CS initiated a second tachycardia (AT2) with left to right CS activation (Fig 2B). Entrainment demonstrated that two separate CS bipoles were included in the circuit (Fig 6A and B), and an electroanatomic activation map confirmed the diagnosis of perimitral flutter (Fig 4B and Propagation Video #2). An ablation line was created from pre-existing scar in the anterior LA to the mitral valve (Fig 3A), resulting in termination of AT2.

Burst pacing from the CS initiated a third tachycardia (AT3). The CS activation sequence of AT3 was reminiscent of perimitral flutter, and may have been mistaken for a recurrence of AT2. However, placement of the ablation catheter distal to the distal CS electrode indicated nonlinear activation of the CS, which is inconsistent with perimitral activation (Fig 2C). Furthermore, entrainment of the proximal CS was inconsistent with its participation in the circuit (Fig 6C). An activation map was created (Fig 4C and Propagation Video #3), showing AT3 to be a reentrant circuit rotating around the anterior scar and breaking through the previously created mitral line. Additional ablation in the region of the anterior mitral line resulted in termination of the tachycardia.

No other tachycardias were inducible, and the patient has remained

Disclosures:
None.

Corresponding Author:
Deepak Saluja, M.D.
Department of Cardiology
Rutgers- Robert Wood Johnson Medical School
One Robert Wood Johnson Place
MEB 583
New Brunswick, NJ 08903

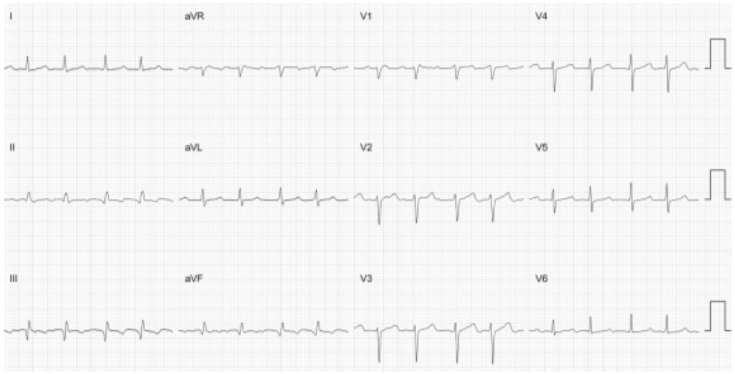

Figure 1: Twelve-lead EKG of clinical tachycardia

free of palpitations in two years of follow-up.

Discussion

We describe a case of multiple left atrial reentrant circuits associated with endocardial scarring in a patient with no prior ablation or surgery and an apparently structurally normal heart. Literature on the study of atrial tachyarrhythmia is hampered by heterogeneity in terminology. Traditionally, organized non-sinus atrial tachyarrhythmias have been classified as either atrial flutter or atrial tachycardia (AT) based upon the surface EKG characteristics of an upper atrial rate cutoff of 240-250/min and an isoelectric baseline between atrial deflections as defining AT.[1] However, this classification is insensitive to mechanism, which has become more precisely understood as mapping technology has advanced. In practice, many electrophysiologists utilize the term atrial flutter to describe a macroreentrant circuit, and atrial tachycardia to describe a focal mechanism. This classification is insensitive to rate, and presupposes a knowledge of the tachycardia mechanism, which may be apparent only after invasive study. We will use the term atrial tachycardia to generally describe an organized atrial tachyarrhythmia of non-sinus origin, and atrial macroreentrant tachycardia (AMRT) to describe an atrial rhythm due to a reentry circuit of measurable size with fixed and/or functional barriers.

Left atrial tachycardia (LAT) is commonly seen after atrial fibrillation ablation, occurring with a frequency of 3-30%.[2] Areas of incomplete prior ablation frequently provide regions of slow conduction that may used as substrates for reentry.[2-4] However, LAT occurring in a naïve and structurally normal heart is less common. Focal LAT has been described as occurring in between 9 to 37% of

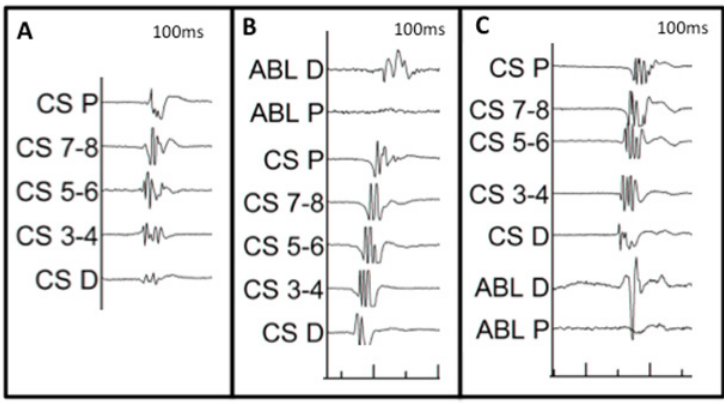

Figure 2: The CS activation sequences of AT1 (A), AT2 (B) and AT3 (C) are shown. In 2B, the ablation catheter (ABL) is located anterior to the CS os. In 2C, ABL D is located just distal to the distal CS electrode

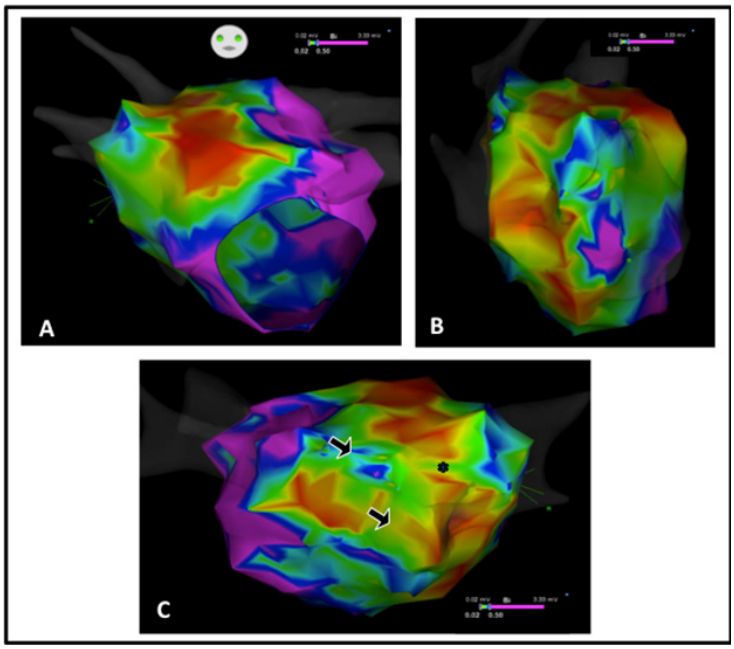

Figure 3: Voltage maps of the LA shown in modified AP (A), right lateral (B), and PA (C) views. AT1 was a double-loop reentry centered around the isthmus of tissue in between two scars near the posterior carina of the two right sided pulmonary veins (asterisk). Ablation (performed at the site of the black arrows) connecting adjacent regions of scar resulted in isolation of tachycardia with resumption of sinus rhythm in the main body of the atrium. EGMs at the site of the asterisk were fractionated, and ablation there resulted in termination of tachycardia. See text

all focal tachycardias,[5] but left AMRT may be less common than that, occurring in 2.6% of all organized atypical reentrant tachycardias described in one series,[6] and comprising only 10% of all patients referred for any type of LAT ablation in another.[7]

The available literature suggests that in these patients, macroreentry is associated with regions of atrial scarring, particularly in the anterior, superior, and posterior aspects of the LA near the right-sided pulmonary veins, which can encompass a significant portion

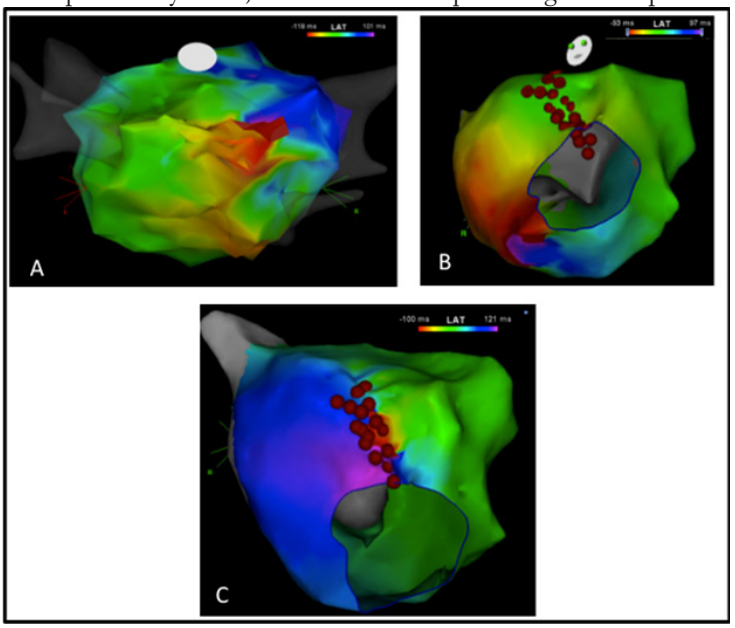

Figure 4: Activation maps of AT1 (A), AT2 (B), and AT3 (C)

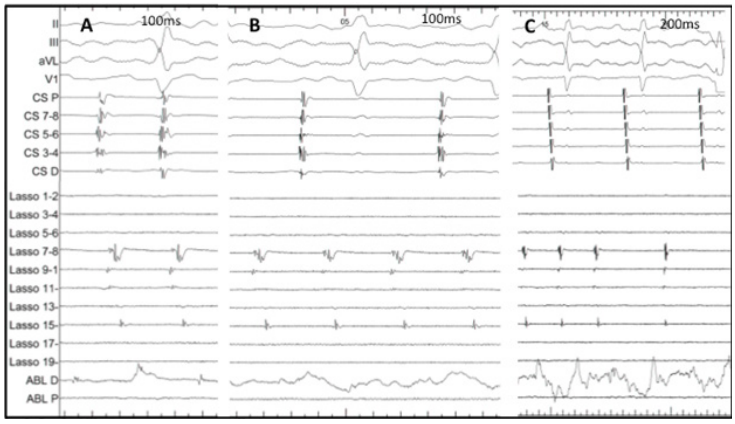

Figure 5: **A) AT1 is shown, with 1:1 conduction out of the circuit to the rest of the atrium. Ablation connecting areas of scar around the circuit resulted in variable block (B), then isolation of the circuit from the remainder of the atrium, which converted to sinus rhythm (C). Ablation of fractionated signal tissue in the common isthmus of the circuit resulted in termination of the isolated tachycardia (C). The circular mapping catheter (Lasso 1-20) is positioned in the right upper pulmonary vein**

of the atrial mass (on average 25%[8]). Narrow isthmuses of tissue between adjacent areas of scar or between an area of scar and an anatomic barrier (such as the mitral annulus) frequently demonstrate fractionated, long-duration electrograms, which create the substrate for reentry, and can be successfully targeted by ablation.[7,8] Acute success rates for ablation are as high as 82%,[6] although recurrence rates may be as high as 20-45% in medium-term follow-up.[6-8] The arrhythmias seen commonly involve either the right-sided pulmonary veins and/or the mitral valve, with double-loop reentry implicated in a substantial number of cases.[6,7]

The diagnosis of left AMRT may not be suspected prior to invasive study, since patients do not have common risk factors for left-sided arrhythmias. A left-sided origin may be suggested on the surface EKG by the presence of an upright or isoelectric p-wave in V1,[7] but focal mechanism is frequently suspected, since isoelectric intervals between p-waves corresponding to long-duration fractionated EGMs are commonly encountered.[7,8]

The case we describe parallels many of the features typically found in these patients. We induced three tachycardias associated with scars in the anterior, superior, and posterior aspects of the LA. The ability to map the entire the cycle length, the demonstration of critical isthmuses of conduction, and the response to entrainment suggest these rhythms were reentrant.

Ablation between regions of adjacent scar was used first to isolate, then to terminate, AT1, which was a double-loop reentry circuit near the posterior right-sided venous carina. AT2 was a perimitral flutter passing through an area of fractionation between the anterior scar and the mitral annulus. Ablation through this area, connecting the scar with the valve annulus, terminated tachycardia. AT3 rotated around an anterior scar and passed through the same isthmus of tissue utilized in AT2. Although these features were present in the atrium at baseline, AT3 was induced after ablation was performed in this region, and as such we cannot rule out an iatrogenic etiology.

Rapidly distinguishing between distinct arrhythmias that may appear similar in the limited number of fixed bipoles available in a typical study is key for the electrophysiologist ablating multiple ATs. Failing to recognize arrhythmia transformation can result in

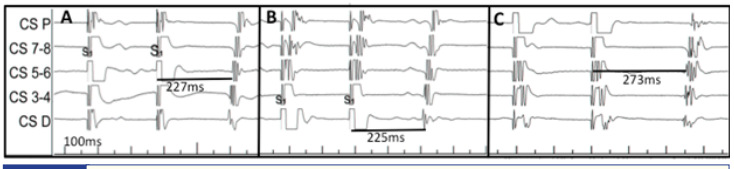

Figure 6: **Entrainment of AT2 from CS 5-6 (A) and distal CS (B) at an S1 of 210ms demonstrates post-pacing intervals of 227ms and 225ms, respectively. The tachycardia cycle length of AT2 was 220ms. C) Entrainment of AT3 from proximal CS at an S1 of 220ms demonstrates a post-pacing interval of 273ms. The cycle length of AT3 was 232ms**

inaccurate activation maps and prolonged procedure times. Strategic sampling of activation and entrainment points can, in some cases, be used to rapidly guide AT mapping.

Conclusion

In our patient, the CS activation and cycle lengths of AT2 and AT3 were similar (Fig 2B and 2C). In the case of AT2, performing entrainment from two different CS locations (Fig 6A and B) rapidly suggested a perimitral circuit, which could then be confirmed with activation mapping. Similarly, in the case of AT3, placing the mapping catheter distal to the distal CS electrode rapidly demonstrated nonlinear CS activation, and entrainment from the proximal CS (Fig 6C) confirmed that a recurrence of perimitral flutter could be ruled-out, allowing the focus to shift to mapping a new arrhythmia. This case also demonstrates the utility of fully assessing scar burden, when present, in patients with multiple tachycardias, as scars may be implicated in either the genesis of the arrhythmia and/or be targeted by ablation.

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