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Case report | Peer reviewed article

Origin not always in the right atrium

Re-entrant atrial tachycardia in a patient with arrhythmogenic right ventricular cardiomyopathy

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Summary

Although typical atrial flutter and atrial fibrillation are not uncommon in patients with arrhythmogenic right ventricular cardiomyopathy (ARVC), there is limited information on atrial tachycardia in this population. A 59-year-old man with a diagnosis of definite ARVC according to the 2010 Task Force Criteria without overt left ventricular involvement presented with recurrent palpitations. The surface 12-lead electrocardiogram raised the suspicion of atrial tachycardia. The patient had no history of cardiac surgery or cardiac ablation. During tachycardia passive activation of the right atrium was visible through invasive electroanatomical mapping indicating a left atrial origin of the tachycardia. High-density mapping of the left atrium confirmed a re-entrant tachycardia with a figure-of-eight activation pattern originating from a small scar in the anterior left atrial wall. Radiofrequency catheter ablation targeting the area of slow conduction in this area terminated the tachycardia and rendered it non-inducible.

Case description

A 59-year-old male physically active patient was referred to our centre for catheter ablation of recurrent drug-resistant sustained atrial tachycardia. He had been diagnosed with a definite diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) according to the 2010 Task Force Criteria, without overt left ventricular involvement 3 years before [1]. No additional cardiovascular risk factors were present. The 12-lead surface electrocardiogram (ECG) indicated a sustained atrial tachycardia with variable atrioventricular conduction with a positive P wave in lead V1 and the inferior leads, and negative in lead aVL, indicating a left atrial origin (fig. 1A).

During an electrophysiological study, a standard decapolar catheter was placed in the coronary sinus as the reference electrode. The endocardial recording showed an atrial tachycardia with a tachycardia cycle length of 278 ms, with a concentric coronary sinus activation sequence (fig. 1B).

To better understand the substrate and arrhythmia mechanism we performed invasive electroanatomical mapping starting from the right atrium and then the left atrium through a fluoroscopy-guided single transseptal puncture using a Brockenbrough (BRK XS) transseptal needle via a long steerable sheath (Agilis medium curve, Abbott, Chicago, IL, US). Three-dimensional activation mapping was performed during atrial tachycardia using the CARTO3 (Biosense Webster, Diamond Bar, CA, US) system. A multipolar high-resolution mapping catheter (PentaRay, Biosense Webster) and later a 3.5-mm tip SmartTouch ThermoCool (Biosense Webster) ablation catheter were subsequently placed in both atria for mapping. The CS electrogram was used as the reference during mapping. A total of 2990 points were collected to reconstruct the geometry of the atrial chambers. A bipolar endocardial voltage of ≤0.05 mV was defined as scar. The right atrial activation map showed the earliest activation located at the lower septum of the atrium indicating passive activation of the right from the left atrium (fig. 2A). Accordingly, the total activation time of the right atrium did not span the whole atrial tachycardia cycle length as revealed by the histogram (fig. 2B). When the atrial tachycardia was mapped in the LA, the bipolar endocardial map confirmed an area of low voltage in the left anterior atrial wall corresponding to a zone of slow conduction with fragmented potentials. The local activation time map manifested as a figure-of-eight re-entrant circuit connected by the slow conduction zone covering the entire cycle length of the tachycardia (fig. 2 D–F, video). The activation propagated caudally through the area of low voltage serving as the critical isthmus with long fractionated signals due to slow conduction.

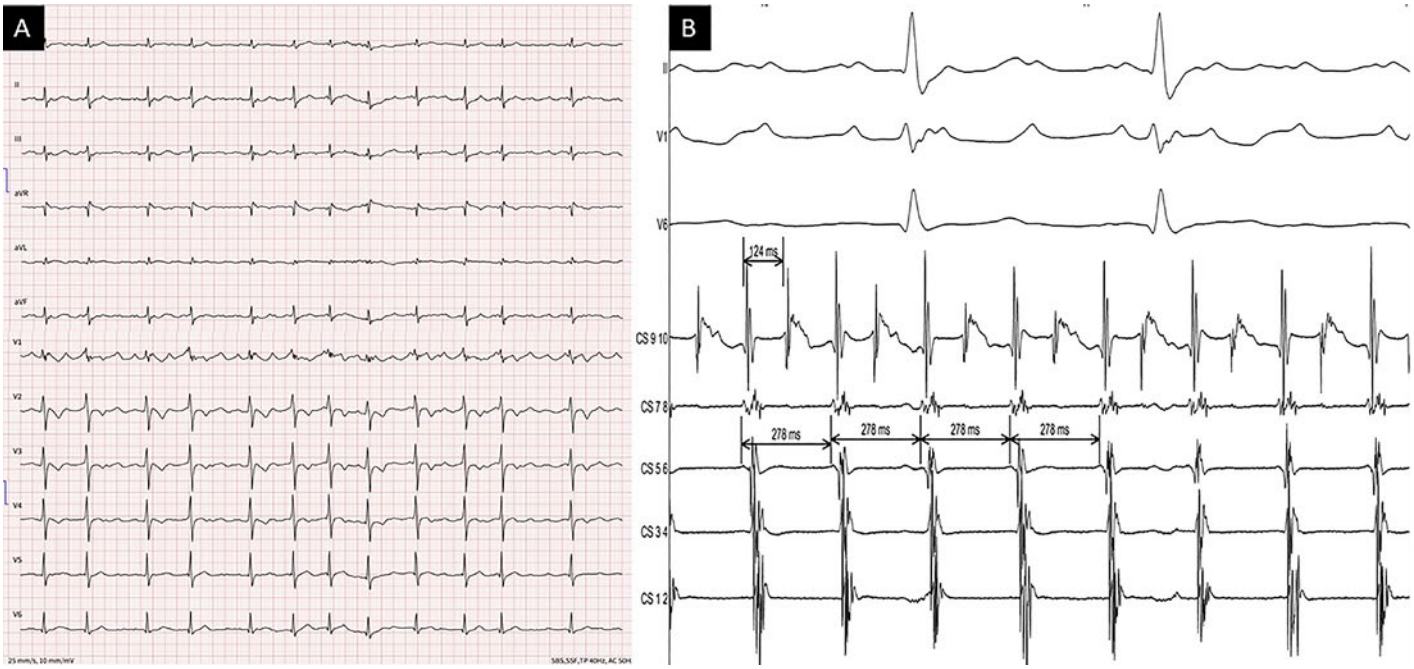

Figure 1: A) Surface 12-lead ECG of the atrial tachycardia (paper speed 25 mm/s). **B**) Surface ECG leads II, V1 and V6 and endocardial recordings during atrial tachycardia. CS: decapolar catheter (CS 1–10) in the coronary sinus showing an atrial tachycardia cycle length of 278 ms and double potentials at the proximal CS 9/10 (124 ms).

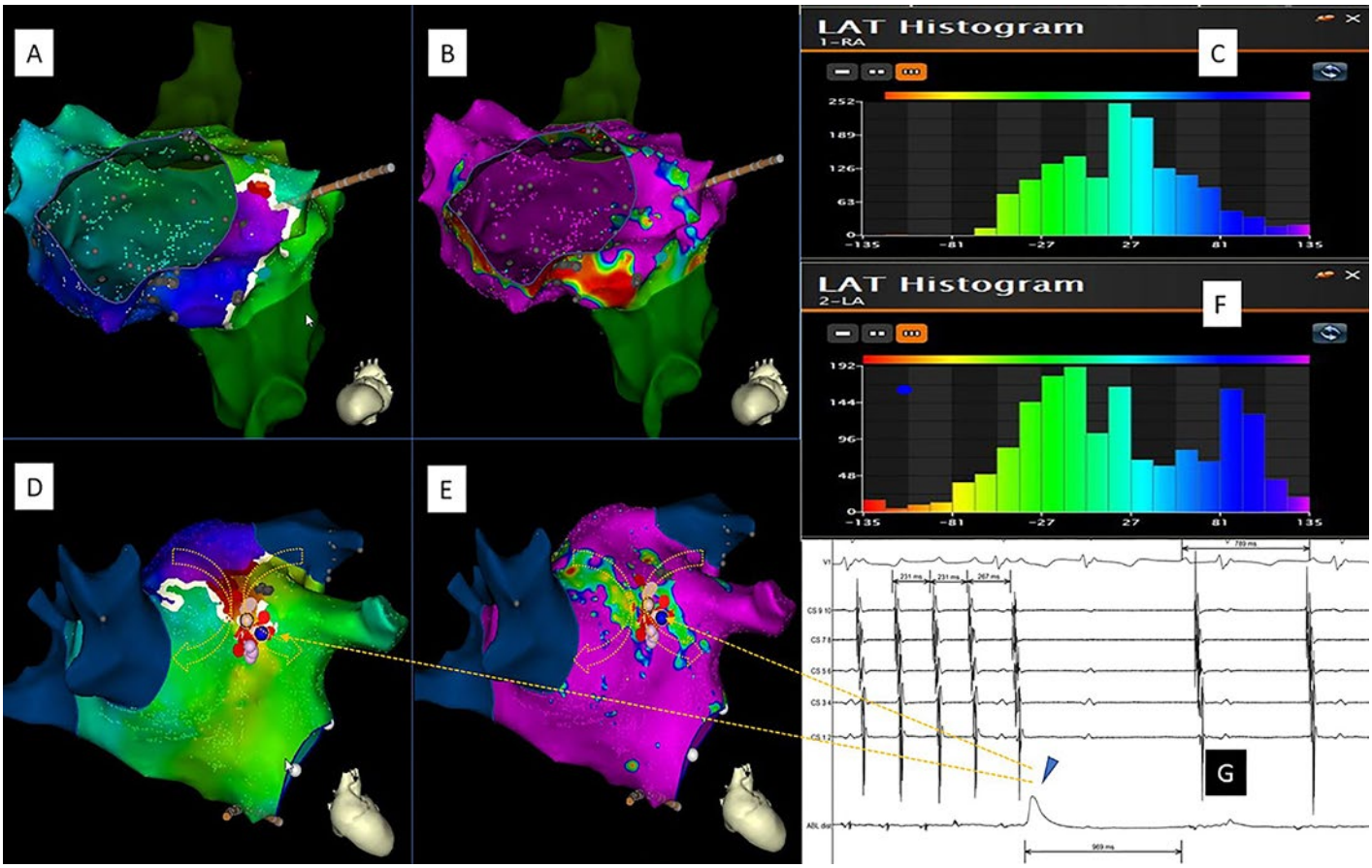

Figure 2: A) Local activation time (LAT) map of the right atrium during atrial tachycardia, with a pseudo early-meets-late re-entry surrounding the cavotricuspid isthmus with delayed conduction. **B**) Voltage map of the right atrium during atrial tachycardia with an area of low voltage in the area corresponding to the LATmap. **C**) A blank in the LAT histogram of global activation due to the delayed conduction on the LATmap is visible. **D**) LATmap of the left atrium during atrial tachycardia showing a figure-of-eight re-entry, connected via a zone of slow conduction in the anterior wall of the left atrium. The schematic circuit is indicated by the yellow arrows. **E**) Endocardial bipolar voltage map of the left atrium during tachycardia with an area of low voltage on the anterior wall, surrounded by channels of myocardial tissue. The tachycardia terminated at this area by tactile manipulation with the ablation catheter (blue point). **F**) Complete distribution (red, orange, blue and purple bars) in the LAT histogram of the global activation of tachycardia, corresponding to the full tachycardia circuit.

Mechanical termination of the atrial tachycardia into sinus rhythm occurred during mapping via the ablation catheter at the area of the critical isthmus on the anterior left atrial wall (fig. 2G). A continuous 60 seconds of radiofrequency delivery with a power of 30–35 W, temperature limit of 43°C and irrigation rate of 17 ml/min at the site of tactile termination was applied. Another 10 minutes of ablation at the low voltage area at the anterior left atrial wall was performed. No more atrial tachycardia was inducible during aggressive atrial stimulation (programmed stimulation and burst pacing) at the end of the procedure.

Discussion

In the present case, activation mapping demonstrated a figure-of-eight re-entrant atrial tachycardia originating from the anterior wall of the left atrium in a patient with definite ARVC without overt left ventricular involvement [2].

As an inherited cardiomyopathy, ARVC is characterised by a progressive loss of myocytes with fibrofatty replacement [1]. LA re-entrant tachycardia is mostly described in patients with prior cardiac surgery or after left-sided catheter ablation, but is rather uncommon in ARVC patients. It is known that surgical incisions, mitral valve disease and ablation gaps can serve as the substrate for focal or macro-re-entrant left atrial tachycardia [3]. Meanwhile, there is evidence suggesting that both atrial chambers can be affected in patients with ARVC [4]. The tachycardia circuit was revealed by high density activation mapping corresponding to the low voltage substrate in the anterior left atrial wall in our patient.

Conclusions

In accordance with previous literature we showed that the left atrium can also exhibit an arrhythmogenic substrate and can be involved in the pathophysiological changes in ARVC, even in the absence of overt left ventricular involvement and other traditional risk factors.

Disclosure statement

No financial support and no other potential conflict of interest relevant to this article was reported.

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