Atrial tachycardia arising from the tricuspid annulus – Insight into electrocardiographic and electrophysiologic features

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A 52-year-old male was referred to our hospital for invasive management due to recurrent symptomatic episodes of sustained narrow QRS tachycardia refractory to antiarrhythmic therapy. He had no history of structural heart disease and the transthoracic echocardiogram was normal.

Following a signed inform consent, the patient underwent an electrophysiology study (EP). Two 6F diagnostic quadripolar catheters were positioned at the His and the right ventricular (RV) apex, while a steerable decapolar catheter was positioned in the coronary sinus (CS). Twelve lead electrocardiogram (ECG) and intracardiac electrograms were recorded and analyzed. The basic intracardiac intervals were within normal limits. Atrial pacing with one extrastimulus delivered at different coupling intervals induced reproducibly a narrow QRS, long RP tachycardia at a cycle length of 450 msec with consistent 1:1 AV relationship and a proximal to distal CS activation pattern. Interestingly, shortening of the coupling interval of the atrial extrastimulus triggering tachycardia initiation was associated with postpacing (last paced stimulus to first tachycardia beat) interval prolongation (Fig. 1a). Ventricular overdrive pacing from the RV apex demonstrated ventriculoatrial dissociation, thus excluding atrioventricular reentry tachycardia. Multiple attempts of atrial overdrive pacing from the proximal CS bipole failed to show ventriculoatrial linking which was highly suggestive of an atrial tachycardia (AT).

The 12-lead ECG showed a characteristic bifid, predominantly negative p wave in the inferior leads with a "w" pattern, negative in leads V1 to V6 and positive in lead aVL (Fig. 1b). The atrial activation sequence and the P-wave morphology were highly indicative of a right AT arising from the inferolateral part of the tricuspid annulus (TA).

Subsequently, a detailed high resolution activation mapping of the tachycardia was performed using a mini-basket array catheter (Intellamap Orion Mapping Catheter, *Boston* Scientific - Rhythmia HDx 3D mapping system, Boston Scientific). The earliest endocardial activation was recorded 35 msec before the P wave onset at the inferolateral TA (7 o'clock in LAO projection) (Fig. 2). It is worth noting that during activation mapping, a second, delayed, low-frequency atrial electrogram was recorded reproducibly at the earliest activation site (Fig. 2c).

Following confirmation of the earliest site of tachycardia activation, the basket catheter was withdrawn and an irrigated ablation catheter

was advanced (IntellaNav MiFi Open Irrigated Catheter, standard curve, Boston Scientific). Radiofrequency ablation lesions were deployed at the earliest site with a power of 30 W and a maximum temperature of 43 °C, resulting in termination of the AT within few seconds. During sinus rhythm, low voltage fragmented electrograms were recorded at the site of tachycardia termination. Following catheter ablation, the tachycardia could not be reinduced by atrial pacing even during isopreterenol infusion. The patient was discharged and was arrhythmia free during a three months' follow-up.

Discussion

We present a case of an atrial tachycardia arising from the inferolateral TA. Tricuspid annulus is a relatively uncommon source of ATs, representing the site of origin of only 13% of focal atrial tachycardias originating from the right atrium [1]. Analysis of the P wave morphology can provide guidance regarding the site of origin of the tachycardia. Tachycardias originating from the inferoanterior aspect of the right atrium display negative P wave in the inferior leads, negative polarity in leads V1 and V2 with late precordial transition, and positive P wave in lead aVL [1]. In our case, the tachycardia P wave displayed similar ECG characteristics, denoting an inferior-anterior to superior-posterior wavefront propagation. However, a well-acknowledged caveat of the P wave morphology for identification of the atrial tachycardia origin is its suboptimal precision due to limited spatial resolution, reported to be approximately 17 mm [2]. Furthermore, in case of atrial tachycardias originating from the anterior-inferior segment of the right atrium, the direction of depolarization of the interatrial septum and the left atrium (which mainly defines P wave morphology) varies depending on the relative conduction velocity of the centrifugal tachycardia wavefront via two "competitive" routes. The latter include the cavotricuspid isthmus, posteriorly bounded by the eustachian ridge (resulting in low to high activation of the interatrial septum and left atrium) and the superior segment of the tricuspid annulus (resulting in high to low activation of the interatrial septum and left atrium). The abovementioned limiting factors should be taken into account in the evaluation of the P wave morphology in patients with atrial tachycardias.

Contrary to the well-established pathophysiology of automaticity, the underlying mechanism of the presented atrial tachycardia is consistent with intra-atrial reentry. This distinctive feature is supported by (a) the reproducible tachycardia onset with programmed stimulation

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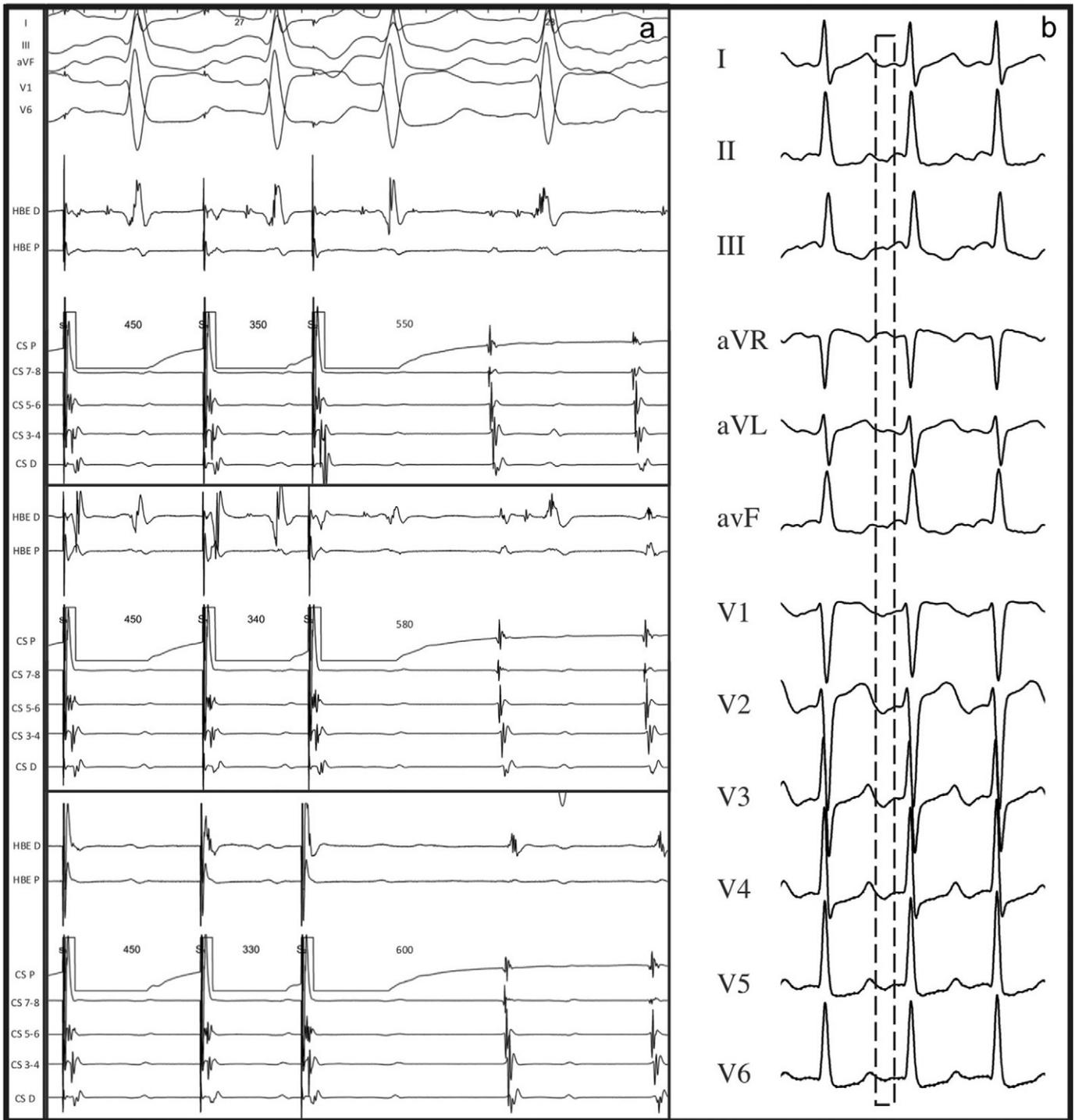*E-mail address:* dimasvestas@gmail.com (D. Asvestas).

Fig. 1. Panel a: Reproducible initiation of the tachycardia with an atrial drive train followed by an atrial extrastimulus delivered at progressively shorter cycle length. Please note the inverse relationship between the coupling interval of the atrial extrastimulus triggering tachycardia initiation (350 ms –>340 ms –>330 ms) and the postpacing interval measured from the last paced stimulus to the first tachycardia beat (550 ms –>580 ms –>600 ms). Panel b: Twelve-lead electrocardiogram during atrial tachycardia originating from the inferolateral tricuspid annulus. Note the P-wave morphology with a negative polarity in leads V1 to V5, bifid, negative in leads II, III, aVF and positive in lead aVL.

and (b) the inverse relationship between the coupling interval of the premature beat triggering tachycardia induction and the postpacing (last paced stimulus to first tachycardia beat) interval. The latter finding is indicative of the presence of conduction delay during tachycardia onset which is a prerequisite for induction of reentrant arrhythmias [3]. However, we could not confirm the mechanism of the tachycardia with entrainment as atrial pacing at the site of earliest activation terminated the tachycardia.

It should also be noted that in the site of earliest local atrial activation, a second delayed atrial potential was recorded reproducibly. This finding may be indicative of specific electrophysiological properties of atrial tissue in the TA area presenting nodal-like features. Similar type of cells has been proposed to serve as electrophysiological substrate, triggering atrial tachycardias. Nogami et al. reported two patients with reentrant AT originating from the TA. They showed the presence of spiky diastolic potentials during the tachycardia hypothesizing the
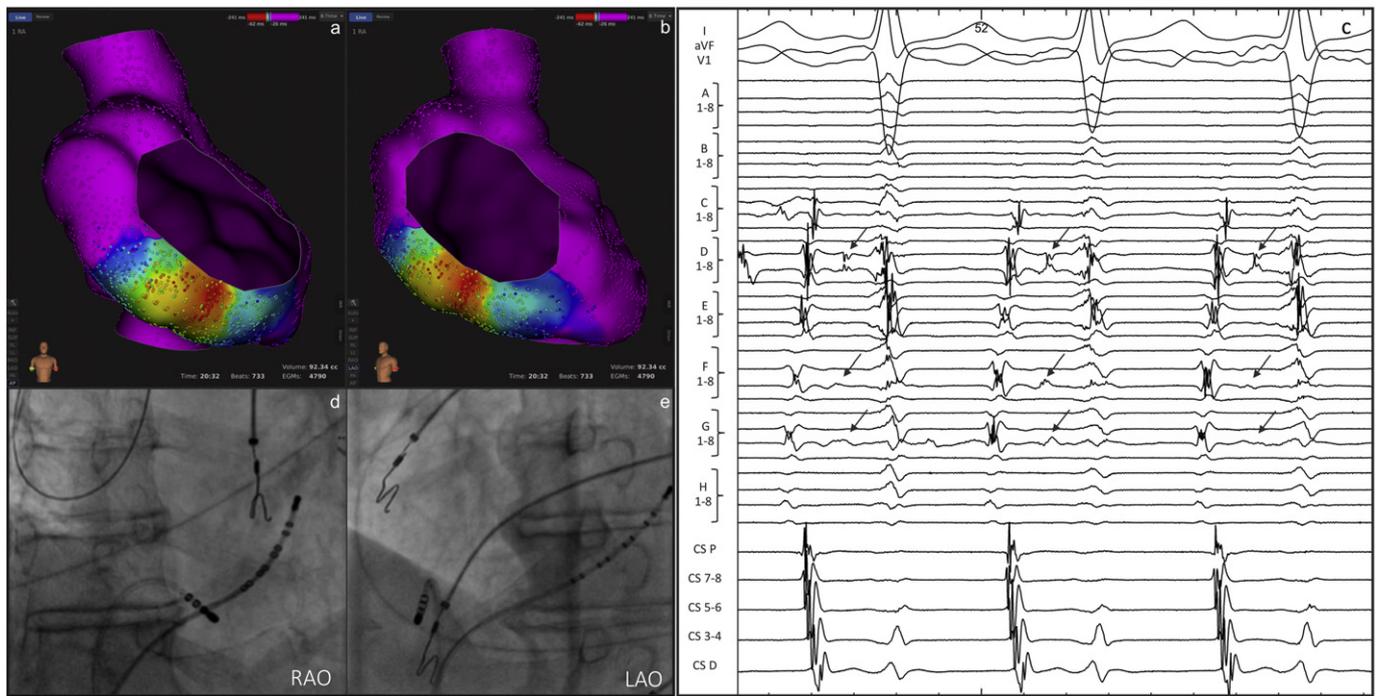
Fig. 2. A three-dimensional electroanatomic map during the atrial tachycardia identified the site of the earliest activation (35 msec before the P wave onset) at the inferolateral tricuspid annulus; anteroposterior (a) and left anterior oblique view (b). A second delayed atrial electrogram (arrows) was recorded at the site of the earliest activation (c). Right anterior oblique (d) and left anterior oblique (e) fluoroscopic views of the catheter at the site of successful ablation.

presence of accessory atrioventricular node without ventricular connection [4]. McGuire et al. have also reported that the atrial tissue around the TA may present interesting electrophysiological features due to the presence of cells with nodal-type characteristics which are not part of the compact AV node but may be the substrate of the slow AV nodal pathway [5]. However, in our case, we failed to demonstrate nodal characteristics and decremental properties of these potentials.

In conclusion, tricuspid annulus is a rare site of origin of atrial tachycardias. The recognition of specific P wave morphology is helpful but should be interpreted in light of pertinent shortcomings. The underlying mechanism varies and although usually due to focal automaticity, it may be rarely related to microreentry.

References

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