Unusual cause of incessant atrial tachycardia

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Introduction

Atrial tachycardia (AT) is a common cause of supraventricular tachycardia (SVT). It most often originates from previously well-characterized sites within the left and right atrium such as the tricuspid annulus, crista terminalis, pulmonary veins, ostium of the coronary sinus, and mitral annulus.[1–6] The aortic sinuses of Valsalva and ascending aorta are in close anatomical proximity to the left atrium (LA). The contact area between the two is variable and corresponds to a low-voltage area on electroanatomical mapping in several studies.[7,8] We report a case highlighting the importance of this anatomical relationship and its role in arrhythmogenesis.

KEY TEACHING POINTS

- The left atrium anterior wall and ascending aorta are in close anatomical proximity.
- The ascending aorta left atrial contact area may play an important role in atrial fibrosis and arrhythmogenesis of atrial arrhythmia and atrial fibrillation.
- The aortic root should be evaluated by imaging in patients with arrhythmias arising from this area.

Case report

An 82-year-old woman with history of hypertension and hyperlipidemia presented to the emergency room after onset of severe exertional fatigue and near syncope. She was found to be in an incessant long RP SVT (Figure 1). This was refractory to diltiazem, sotalol, and amiodarone. Transthoracic echocardiogram demonstrated moderate-to-severe aortic stenosis with preserved left ventricular ejection fraction. Decision was then made to proceed with an electrophysiology study and ablation

The patient presented to the electrophysiology (EP) laboratory in a long RP tachycardia with tachycardia cycle length of 420 ms and 1:1 atrioventricular (AV) conduction. P waves were positive in the inferior leads, as seen in Figure 1. With ventricular overdrive pacing, the first fully paced beat did not advance the atrium to pacing cycle length, thereby suggesting ventriculoatrial dissociation and ruling out pathway-mediated tachycardia. Postpacing response could not be assessed owing to termination of tachycardia. EP study during sinus rhythm did not demonstrate any evidence of an accessory pathway or dual AV nodal physiology. Tachycardia was reinduced with burst pacing and right atrial (RA) activation mapping was performed using the Carto 3D mapping system (Biosense Webster, Irvine, CA) and a multielectrode mapping catheter. This demonstrated earliest RA activation in the posterior interatrial septum. Combination of positive P waves in inferior leads and location of earliest RA activation site away from the fast and slow pathway regions made AV nodal reentrant tachycardia very unlikely and a diagnosis of AT was made. The electrogram at the earliest RA activation site was after the onset of the P wave. We then proceeded with LA activation mapping. Transseptal access was obtained in standard fashion using intracardiac echocardiography (ICE) and fluoroscopy. Activation mapping of the LA was then performed and earliest activation was seen at the anterior interatrial septum. At this site, a low-amplitude fractionated electrogram was seen to precede the P wave by 20 ms (Figure 2). ICE showed this site to be apposed to the aortic root, at the site of a focal aneurysmal dilation (Figure 3B). Ablation at 20 watts was then performed at this site with a bidirectional 3.5 mm force-sensing irrigated catheter (Biosense Webster), which resulted in immediate termination of tachycardia without acceleration. A few lesions were then given around this point for consolidation and subsequently this tachycardia was no longer inducible. Contrast-enhanced computed tomography post ablation confirmed compression of the LA anterior wall by focal aneurysmal dilation of the aortic

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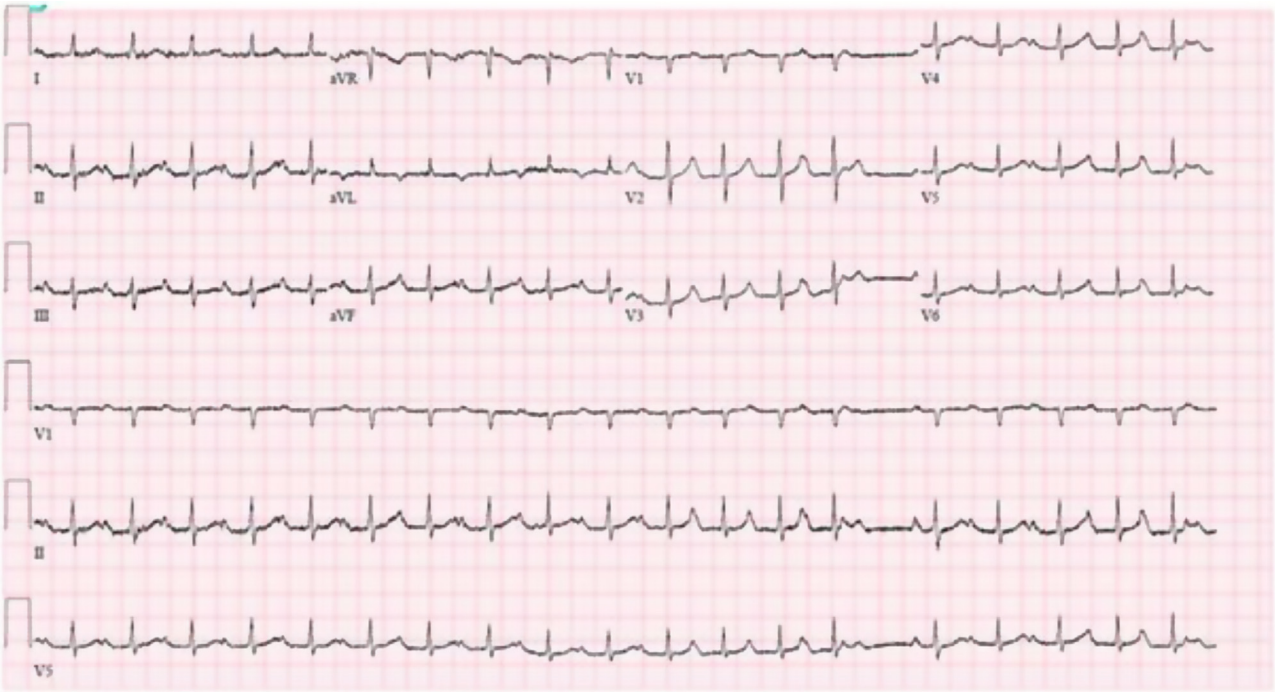

Figure 1 Twelve-lead electrocardiogram showing narrow complex tachycardia.

root (Figure 3A). She maintained sinus rhythm for the remainder of an uneventful hospital course. Amiodarone was discontinued 24 hours after ablation. The patient was seen in EP clinic for 1 month follow-up, at which point she remained asymptomatic without recurrence of SVT.

Discussion

The LA anterior wall is in close proximity to the aortic root and ascending aorta. Local fibrosis at this site has been implicated in the mechanism of persistent atrial fibrillation (AF). In a study by Nakahara and colleagues,[8] of 40 patients with

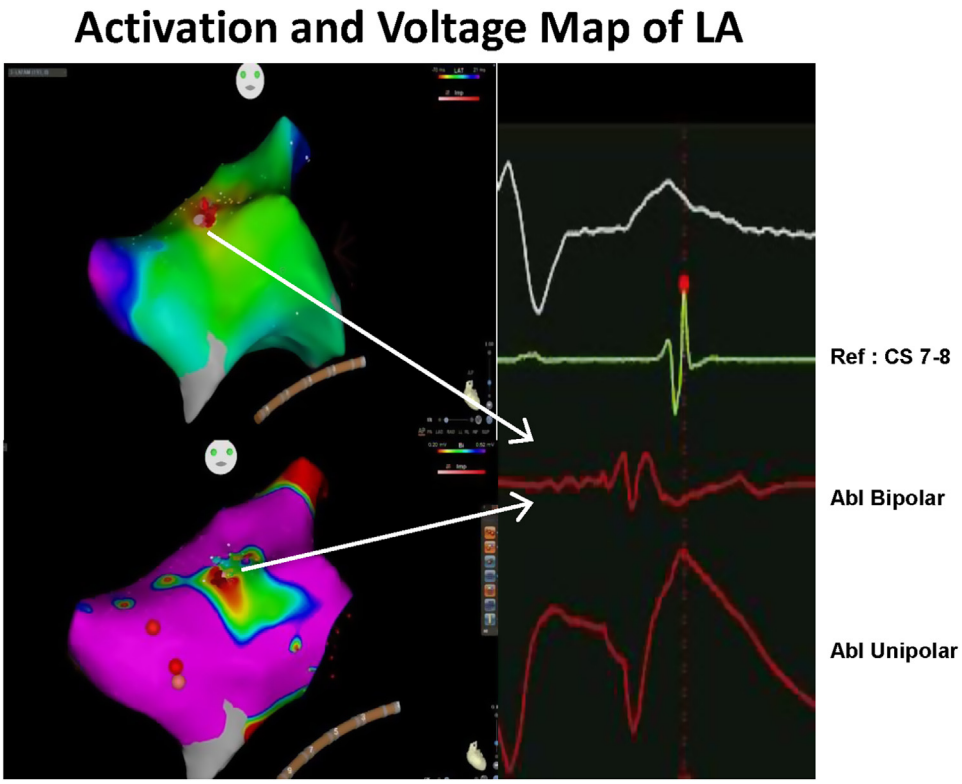

Figure 2 Activation (upper left) and voltage (bottom left) map of the left atrium with the local unipolar and bipolar electrogram at the successful ablation site.

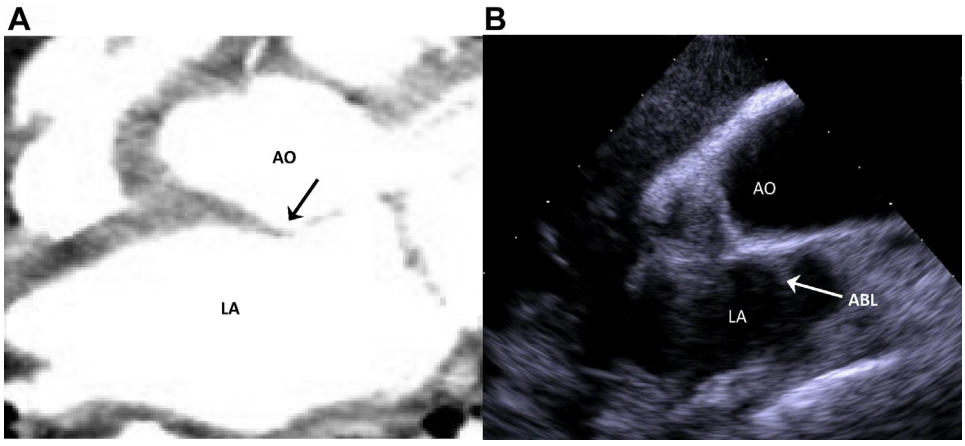

Figure 3 **A:** Computed tomography (CT) demonstrating localized compression of left atrium (LA) by focal aortic (AO) root aneurysm (*black arrow*). **B:** Intracardiac echocardiography image of ablation catheter at the successful ablation site (ABL) corresponding to the CT image.

persistent AF, 98% had contact between the anterior LA anterior and the sinus of Valsalva/aorta. Targeted ablation of the contact area regions with low-voltage zones resulted in AF termination in 28% of patients.[8] Our case report provides further evidence that this contact area can serve as arrhythmogenic substrate to drive atrial arrhythmias. Our patient had focal aneurysmal dilation of the aortic root as visualized on computed tomography scan and ICE, even though the overall dimension of the ascending aorta was within normal limits. We hypothesize that repeated mechanical compression of the LA anterior wall by this focal dilation can result in fibrosis, as evidenced by abnormal voltage in this area, seen in our patient. The low-voltage area <0.2 mV containing the successful ablation site was measured to be 0.93 cm^2 and the area with voltage <0.5 mV was around 5 cm^2.The extent of fibrosis may be dependent on the contiguous aorta-LA area. In our patient the area of contact was focal, thereby limiting the extent of fibrosis and presenting as focal/micro-reentrant tachycardia. In patients with larger contact area the resulting fibrosis would be more extensive and thus result in multiple AT/AF.[9,10]

In conclusion, we report a rare case of incessant AT arising from the arrhythmogenic effect of LA anterior wall compression by the aortic root.

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