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4:2:1 conduction of an AF initiating trigger

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ABSTRACT

A 44 year old male with idiopathic dilated cardiomyopathy was undergoing persistent atrial fibrillation (AF) ablation. Following antral ablation, AF terminated into a regular narrow complex rhythm. Earliest activation was mapped to a focus in the superior vena cava (SVC) which was conducted in a 2:1 ratio to the atria which in turn was conducted with 2:1 ratio to the ventricles, resulting in an unusual 4:2:1 conduction of the SVC tachycardia. 1:1 conduction of the SVC tachycardia to the atrium preceded initiation of AF. During AF, SVC tachycardia continued unperturbed. Sinus rhythm was restored following catheter ablation of the focus.

Case presentation

A 44 year old Chinese gentleman with symptomatic persistent atrial fibrillation (AF) and idiopathic dilated cardiomyopathy (ejection fraction of 40%) was undergoing AF ablation. After ipsilateral pairs of pulmonary veins were isolated, AF terminated and the following 12 lead ECG was recorded (Fig. 1, 50 mm/s speed).

The 12 lead ECG demonstrates a regular narrow complex rhythm at 75 beats per minute (bpm). Closer inspection of the ECG reveals the presence of non-conducted P waves (most visible in V1, superimposed on T waves), establishing the diagnosis of an atrial tachycardia with 2:1 AV conduction. The P-wave morphology is consistent with a high right atrial focus and therefore the circular mapping catheter (CMC) was positioned at the superior vena cava (SVC) – right atrium (RA) junction. The intracardiac electrograms in Fig. 2 indicate the presence of a rapid SVC tachycardia (300 bpm) with 2:1 conduction to the atrium (150 bpm) which in turn conducted in a 2:1 ratio to the ventricle, resulting in a 4:2:1 conduction pattern.

Occasionally, SVC-RA conduction would intermittently improve, allowing for 1:1 SVC-RA conduction (Fig. 3 upper panel). Acceleration of the atrial rate precedes initiation of AF. During AF, electrical activation within the SVC remains regular, suggestive of "retrograde" RA to SVC conduction block, preventing overdrive termination of SVC tachycardia (Fig. 3 lower panel).

Ablation at the earliest site of activation within the SVC resulted in abrupt slowing of the tachycardia before eventual termination to sinus rhythm. Atrial pacing at this point, confirmed unidirectional block into the SVC. Further segmental ablation was performed at the SVC-RA junction to isolate the SVC and establish bidirectional block between the SVC and RA.

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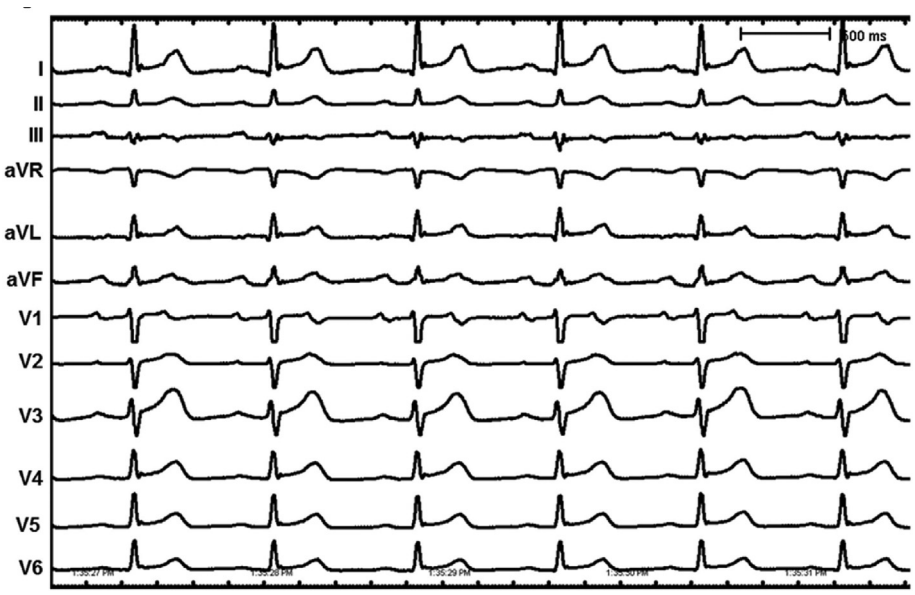

Fig. 1 – 12 Lead ECG after termination of AF.

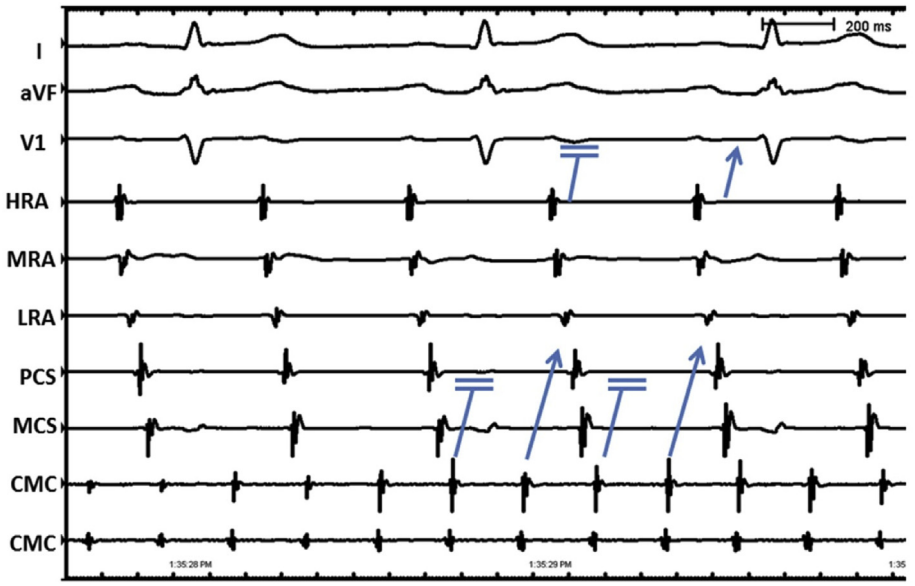

Fig. 2 – Intracardiac electrograms from high right atrium (HRA), mid right atrium (MRA), low right atrium (LRA), proximal coronary sinus (PCS), middle coronary sinus (MCS) and circular mapping catheter (CMC).

Discussion

The arrhythmogenic capabilities of thoracic veins, in particular the pulmonary veins, to initiate and perpetuate AF are well recognized. In Asian cohorts, as many as 6% of patients have AF-initiating foci originating from the SVC [1]. Unusually in this case, conduction across SVC-RA junction modulated the arrhythmogenicity of the SVC foci and protected the foci from being interrupted by ensuing AF.