Atrial flutter with atypical presentation

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1. Case report

1.1. Brief clinical history

Fifty-five year old lady presented with complaints of recurrent palpitations for last 5 years. She had already undergone 3 previous attempts at ablation at another institution; the details of the procedures were not available. Previous cardiac disease or cardiac surgery was absent. Presenting ECG was consistent with counter-clock wise (CCW) atrial flutter (Negative flutter waves in II, III, aVF and positive flutter wave in V1) (Fig. 1A) [1].

Echo showed mild enlargement of the right atrium (RA) and normal pressure Tricuspid Insufficiency.

Fig. 1. A) ECG at the time of presentation. There is no clear Isoelectric line in III. Note the negative 'f' waves in inferior leads and isoelectric/biphasic 'p' in V1, consistent with CCW flutter. B) Electrogram recordings from the coronary sinus and the duodecapolar catheters in RA (bipolar 7-8 to 13-14 have been hidden, See text for details). Inset: A static fluoroscopic frame recorded simultaneously shows duodecapolar catheter in the RA, decapolar catheter in CS and ablation catheter. Note that the duodecapolar catheter is in good contact with the RA wall.

Abbreviations: AT, Atrial tachycardia; CCW, Counter-clock wise; CS, Coronary sinus; CTI, Cavo-tricuspid isthmus; ECG, Electrocardiogram; EGM, Electrogram; EPS, Electrophysiological study; RA, Right atrium.

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1.2. Electrophysiological study

The patient was subjected to electrophysiological study (EPS). Fig. 1B shows the electrograms (EGMs) recorded from the coronary sinus (CS) catheter (6F, steerable decapolar catheter, Bard Electrophysiology, Boston, MA, USA) and duo-decapolar catheter (7F, steerable, Livewire™, St. Jude Medical, St. Paul, Minn., USA) placed in RA. Atrial activation during flutter was from proximal to distal in the CS and Cranio- Caudal in the duodecapolar catheter suggestive of CCW activation in RA (Fig. 1B).

Mapping with duo-decapolar and ablation catheter showed almost entire lateral wall to be scarred (Fig. 1B, bipoles DD7-8 to DD13-14 hardly record any electrical activity despite good catheter contact and therefore have been hidden in the figure).

Tachycardia was entrained from the cavo-tricuspid isthmus (CTI) with concealed fusion and post pacing interval within 10 msec of tachycardia cycle length confirming that this site was part of the tachycardia circuit.

1.3. Radiofrequency ablation

In view of the ECG findings, CCW activation pattern in the duodecapolar recordings and entrainment results, ablation at the CTI was started (4mm, irrigated, 50 W, 40 c, 20 cc/min) [2]. Just as the CTI line was being completed, there was a switch in the atrial activation pattern, evident on the surface P waves, without any change in tachycardia cycle length or the activation sequence on the recording catheters (Fig. 2A and B).

Entrainment was tried again from both sides of the isthmus and other regions in RA including multiple points at crista, but entrainment with concealed fusion was not obtained anywhere (Fig. 2C). The tachycardia now behaved more like a focal tachycardia than re-entry, as it was non-entrainable and had a clear isoelectric interval between the P waves.

On the basis of earliest atrial activity during mapping, the origin of the tachycardia was mapped to the upper-mid Crista region and was easily ablated by point ablation at this site (Fig. 2D). Post restoration of sinus rhythm, CTI isthmus block was confirmed by differential pacing maneuvers.

No sustained tachycardia could be induced on atrial pacing maneuvers at basal state and on isoprenaline.

2. Discussion

The present case is an example highlighting the challenges faced during ablation of atrial tachycardia. A tachycardia which was initially typical CCW atrial flutter switched to a tachycardia which appeared to be focal in origin arising in the crista terminalis region. Probable mechanisms of the unusual features seen in this complex tachycardia and the points in favor/against of each explanation are given below.

The change in P wave morphology is explained by the pre-dominant atrial activation vector in both the possibilities given below:

Possibility 1: Dual loop tachycardia.

Dual loop reentry tachycardia, is one definite possibility. The initial tachycardia was CCW flutter. Following CTI block the tachycardia continued in the second loop. (Fig. 3) [3].

Points which are difficult to explain on the basis of this possibility:

a) Both tachycardias had exactly the same cycle length
b) Inability to entrain the second tachycardia favors non-reentry rather than a “loop”

Possibility 2: Focal tachycardia driving the Flutter

A focal atrial tachycardia (AT) on the free wall of the right atrium
that has induced typical cavotricuspid isthmus dependent flutter and the automatic tachycardia continuously entrains and maintains the flutter. Ectopic atrial tachycardia got unmasked after the CTI was blocked (Fig. 4).

This is a more plausible explanation, as a) The two tachycardias had the same cycle length, b) The ECG features after CTI ablation was that of a focal AT c) the second tachycardia could not be entrained and d) It was abolished by a single point ablation.

Three dimensional electro-anatomic mapping may have given better insight into the mechanism of the two tachycardias seen in this case. Electro-anatomic mapping is not routinely used for CCW atrial flutter at our center. Whether the complex tachycardia and area of electrical inactivity in the RA could have been due to earlier ablation procedure or an atrial myopathy could be argued. Atrial cardiac MRI was not available at the time of the study.

**Conflict of interest disclosures**

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