Case Report

Organised Atrial Fibrillation Simulating Atrial Flutter

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Electrophysiological study, cavotricuspid isthmus ablation, circumferential pulmonary vein ablation, electroanatomical mapping.

We present a case of drug-refractory paroxysmal atrial fibrillation in which episodes of AF spontaneously alternated with a regular arrhythmia resembling atrial flutter. Isthmus-dependent atrial flutter was initially diagnosed and the patient was treated with cavotricuspid isthmus ablation. Two months later the patient returned with recurrent arrhythmias. On the 12-lead ECG there was again evidence of atrial flutter with both typical and atypical characteristics. On electrophysiological study, careful mapping revealed that the regular rhythm actually represented organised slow atrial fibrillation. Circumferential pulmonary vein ablation resulted in the abolition of both arrhythmias.

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Although atrial fibrillation (AF), typical and atypical atrial flutter appear to represent separate clinical entities, recent electrophysiological data indicate an important interrelationship between these arrhythmias.[1-3] Approximately 30% of patients undergoing successful ablation of atrial flutter may develop AF at follow-up,[4,5] whereas a similar proportion of patients subjected to pulmonary vein ablation for AF may experience isthmus-dependent atrial flutter following the procedure.[6] We and others have previously reported on patients with simultaneous episodes of AF alternating with atrial flutter.[2,7] This patient group, which on electrophysiological study is identified by the recording of flutter waves from the right atrium and AF from the left atrium, may benefit from ablation of the cavotricuspid isthmus.[7]

Here we present a case of drug-refractory paroxysmal AF alternating with a regular arrhythmia that resembled atrial flutter but actually represented organised slow AF.

Case description

A 59-year-old lady presented with episodes of drug-refractory disabling paroxysmal arrhythmias. On the basis of 12-lead ECGs obtained during symptomatic episodes, atrial flutter was suspected (Figure 1). Ambulatory electrocardiographic monitoring revealed episodes of atrial fibrillation alternating with a narrow QRS complex tachycardia suggestive of atrial flutter. The patient had an unremarkable physical examination and no evidence of ischaemia or structural cardiac disease.

On electrophysiological study, AF was easily induced by high rate right atrial pacing. However, continuous recording from both the right and left atrium revealed episodes of typical AF and presumed atrial flutter alternating between the left and right atria (Figure 2). The 12-lead ECG displayed characteristics of AF, as well as typical and atypical atrial flutter. Since the episodes of presumed flutter were not sustained in order to allow detailed mapping, and in view of the reported response of organised fibrillation to cavotricuspid isthmus ablation,[8] an ablation procedure was undertaken with accomplishment of bidirectional cavotricuspid isthmus block.

Two months later the patient was read-

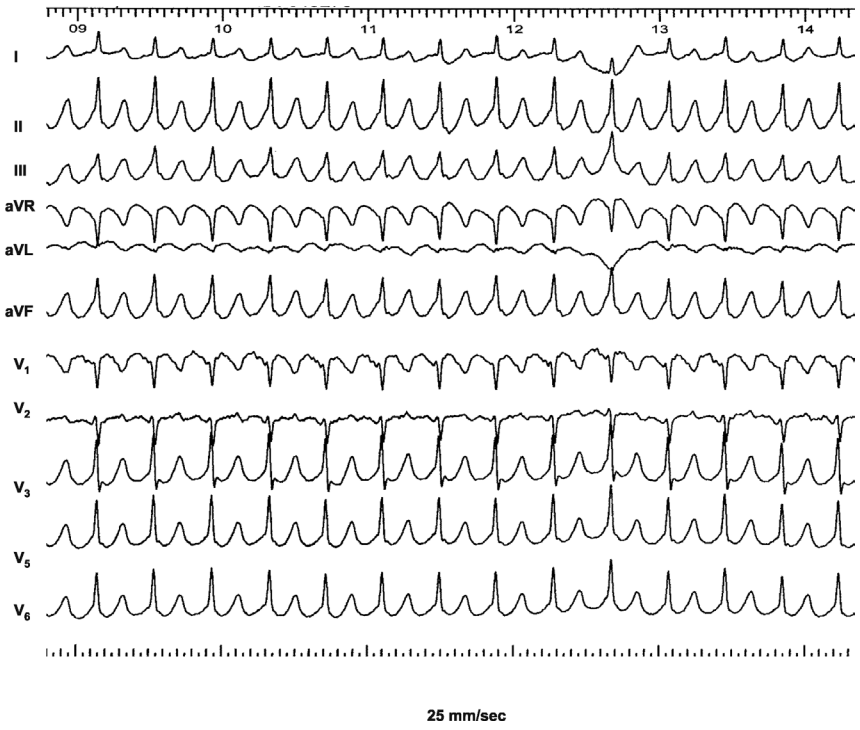

Figure 1. Surface ECG at presentation. There is suggestion of flutter waves with 1:1 conduction.

mitted due to recurrent arrhythmia episodes of the same nature. Isoprenaline infusion was commenced and a detailed electrophysiological study was undertaken. Episodes of typical AF alternating with a regular rhythm resembling atrial flutter were again recorded. Changes between the arrhythmia forms were spontaneous without pacing or pharmacologic intervention. Detailed mapping revealed continuing episodes of a right atrial flutter-like rhythm co-existing with left atrial AF (Figure 3). These episodes alternated with a left atrial flutter-like rhythm co-existing with right atrial AF (Figure 4). Although this was a case reminiscent of previous reports of flutter-fibrillation episodes,[2,7] careful inspection of the electrograms revealed that there was a lack of consistent clockwise or counterclockwise activation sequence (Figures 3 and 4). Second, bidirectional block along the cavotricuspid isthmus was still present and demonstrated. Entrainment mapping from the

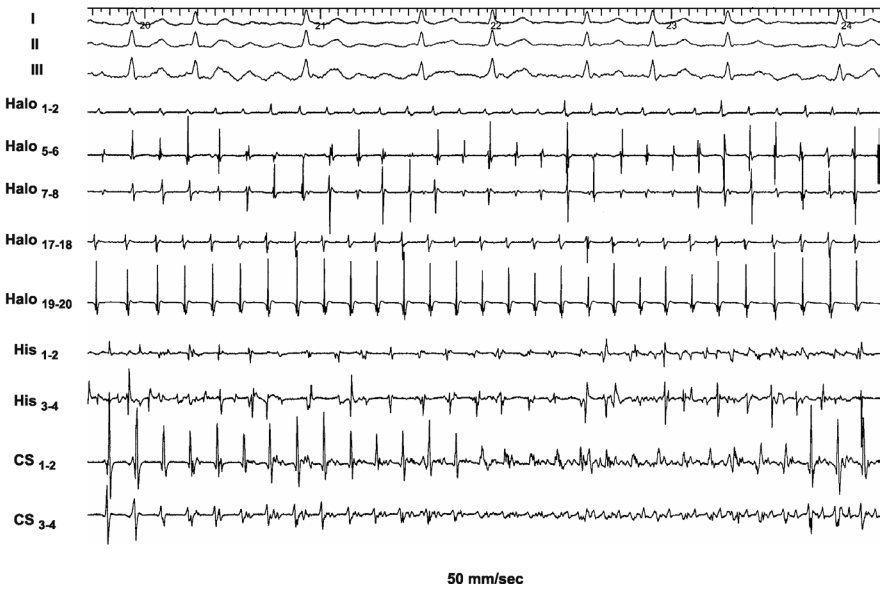

Figure 2. Episodes of atrial flutter spontaneously alternating with atrial fibrillation, mainly at the left atrium. I, II, III: ECG leads, Halo: Halo multipolar catheter, His: His bundle, CS: coronary sinus.

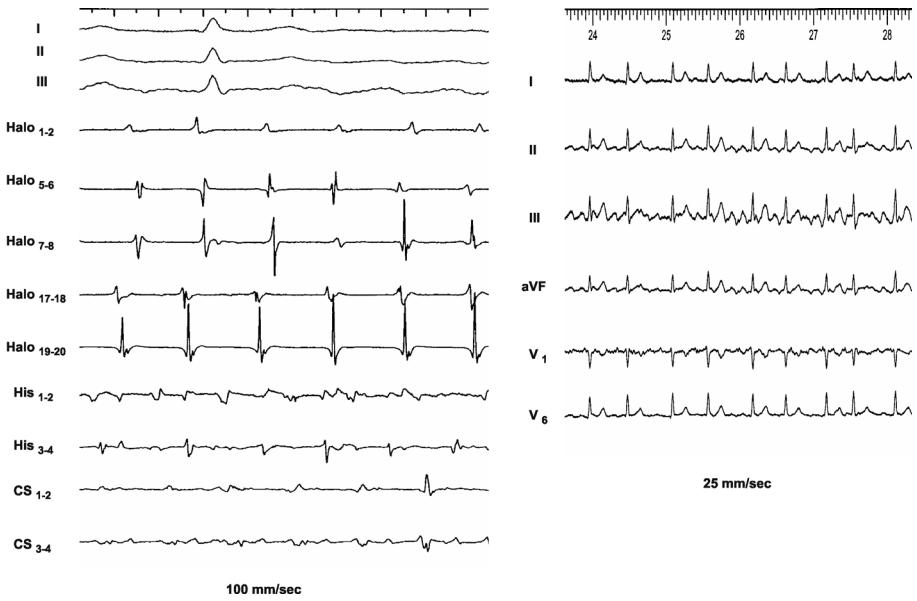

Figure 3. Presumed atrial flutter in most of the right atrium and atrial fibrillation in the septum (as evidenced by the His bundle recordings) and the left atrium. Careful inspection reveals that there is no consistent activation pattern on the Halo pairs. Abbreviations as in Figure 2.

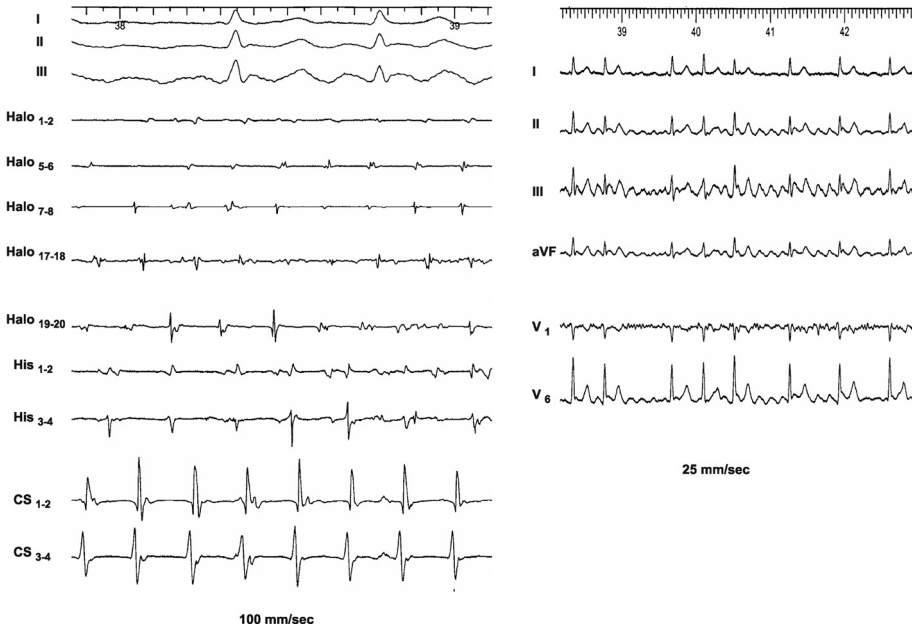

Figure 4. Presumed atrial flutter in the left atrium and atrial fibrillation in the right atrium. Again the distal CS atrial electrogram precedes that of the proximal, thus arguing against typical atrial flutter. Abbreviations as in Figure 2.

cavotricuspid isthmus area initially gave the impression of dependence of the arrhythmia on the cavotricuspid isthmus, as was the case at the first ablation procedure (Figure 5). However, on more careful inspection, there was capture of the atrial electrograms on the pairs 3-4 and 5-6 of the Halo catheter, but not on the remaining pairs. Following the two initial post-pacing beats the initial activation sequence resumed, with the atrial electrogram recorded on pairs 3-4 and 5-6 clearly behind the electrograms recorded on pairs 17-18 and 19-20 (Figure 5). It was therefore thought that the recorded regular arrhythmia actually represented a form of localised, organised AF or atrial tachycardia or flutter, which was giving the impression of isthmus-dependent flutter. Circumferential pulmonary vein ablation with the aid of electroanatomical mapping (CARTO) was undertaken and no arrhythmia was inducible following the procedure. Three months later, the patient remains free of arrhythmias on no antiarrhythmic medication.

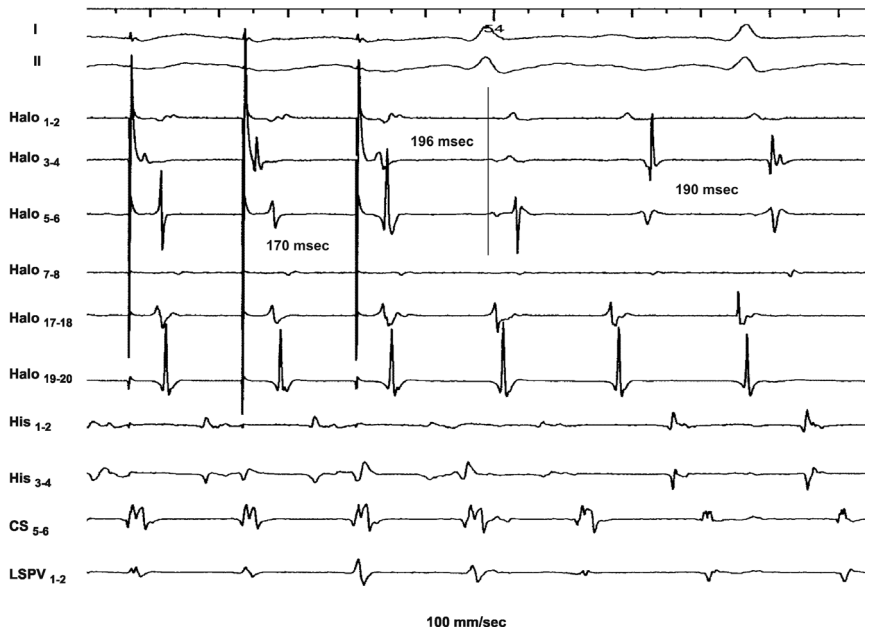

Figure 5. Entrainment mapping from the area of the cavotricuspid isthmus. The flutter cycle length is 190 ms and the apparent post-pacing interval 196 ms. However, the flutter cycle length accelerates only on pairs 3-4 and 5-6, without consistent advancement of the other atrial electrograms. This suggests localised capture without resetting of the arrhythmia circuit. Abbreviations as in Figure 2.

Discussion

The interrelationship between AF and atrial flutter is now well established. It appears that atrial flutter carries a fibrillatory role in the pathogenesis of AF,[3] whereas atrial fibrillation may create the conditions for the development of a functional line of block between the venae cavae and thus promote induction of isthmus-dependent atrial flutter.[2] Interestingly, isthmus-dependent atrial flutter may present with atypical ECG characteristics,[9] particularly when it occurs following pulmonary vein isolation for AF.[10] Both typical and atypical forms may respond to cavotricuspid isthmus ablation.[11,12] Kumagai et al[8] have also reported on isthmus ablation that resulted in a 75% success rate of preventing paroxysmal AF with an organised pattern around the tricuspid annulus.

In view of this information, and since the initial arrhythmia episodes did not allow detailed mapping at the first ablation attempt, the diagnosis of isthmus-dependent flutter probably degenerating into AF was made. However, cavotricuspid isthmus block had no effect on arrhythmia recurrence. Both the regular rhythm and typical AF were subsequently recorded as before ablation. Circumferential ablation around the ostia of the pulmonary veins, which rendered the AF non-inducible, also resulted in abolition of the regular atrial rhythm. Thus, the recorded regular rhythm represented either a case of organised AF (as suggested by the change of activation sequence in Figures 3 and 4) or a left atrial tachycardia or flutter that was related to paroxysmal AF.

In conclusion, our report emphasises the need for a detailed electrophysiology study in all patients with an apparent clinical diagnosis of atrial arrhythmias such as flutter or fibrillation subjected to catheter ablation. Careful analysis of activation patterns in both atria may establish the correct diagnosis and avoid unnecessary ablation procedures.

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