Spontaneous Conversion of Long-Standing Atrial Fibrillation/Flutter

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There have been no recent descriptions of the spontaneous conversion of long-standing atrial fibrillation (AF) or flutter (AFl) to sinus rhythm which, in the past, has been associated with rheumatic mitral valve disease and treatment with digoxin. We present 3 contemporary cases, all of whom progressed from AF to slow AFl and then spontaneously converted to slow sinus or junctional rhythm. None of these patients had rheumatic heart disease or were treated with digoxin. In conclusion, we believe that they provide support for the broader view that this uncommon phenomenon is associated with a severe atrial myopathy due to scar and inflammation. **(Am J Cardiol 2020;131:122−124)**

The spontaneous conversion of long-standing atrial fibrillation (AF) or flutter (AFl) to sinus rhythm, often with associated sinus node dysfunction, is an uncommon, but well described phenomenon originally reported by Burch in 1939.[1] Its occurrence has been related to the presence of both rheumatic mitral valve disease and treatment with digoxin.[2-4] Pathologic studies of such patients[5] and their echo findings of enlarged, poorly contractile atria,[4] have suggested key roles for fibrosis and, potentially, inflammation in the etiology of this event. There have been no descriptions of this phenomenon in almost 40 years and we feel that the growth of knowledge during this period merits its review and further discussion of its mechanisms.

We therefore present 3 contemporary patients, none of whom had rheumatic heart disease or were treated with digoxin, who presented unexpectedly with sinus or junctional rhythm after being in AF or AFl for over a year as well as supportive evidence that this phenomenon is related to a profound atrial myopathy, making the ability to sustain AF or AFl tenuous and often including sinus node dysfunction. All of our patients had preserved left ventricular function, symptomatic sinus bradycardia or junctional rhythm on presentation, all required pacing, and all ultimately reverted back to low amplitude AFl.

Our 3 patients were identified over a 3-year period. Their clinical characteristics are summarized in Table 1. In each case a rate control, rather than a rhythm control, strategy was being pursued, and multiple clinical encounters and electrocardiograms (ECG)s confirmed the persistence of atrial dysrhythmia over a period of more than 1 year during which the patients were asymptomatic. All of our patients had evidence of severe atrial enlargement without associated significant valvular disease or ventricular dysfunction. Each patient had factors potentially contributing to atrial fibrosis and inflammation, including the long-standing atrial dysrhythmia itself, chronic infection, advanced old age, and a history of pericarditis/chronic graft versus host disease in the setting of a bone marrow transplant for the treatment of chronic lymphocytic leukemia.

They all presented with weakness or syncope due to concomitant sinus node dysfunction manifest as either junctional rhythm, prolonged pauses or slow sinus rhythm. As shown in Figure 1, Patient 1 presented with junctional rhythm and a slow isorhythmic atrial rhythm, which was confirmed by our ability to capture the atrium during atrial pacing, Figure 1.

Although all of our patients initially had AF, this subsequently evolved to slow, low amplitude, AFl before their spontaneous conversion to sinus or junctional rhythm as illustrated in Figure 2. Patient 3 ultimately developed heart failure with a preserved ejection fraction, and as he did not want to take antiarrhythmic drugs, he underwent tricuspid isthmus ablation and pulmonary vein isolation in an effort to restore sinus rhythm. Left atrial mapping at the time of that procedure confirmed the presence of extensive right and left atrial scar, Figure 3.

It has been postulated that severe scarring of the atria could result in "autoablation" and the inability to sustain AF because of the lack of sufficiently large contiguous areas of electrically functional atrial myocardium, and perhaps incomplete lines of such scarring could transform AF into AFl. We feel our findings support this view. In addition, the chronic infections of Patient 1, the extreme old age of Patient 2, and the pericarditis/chronic graft versus host disease in Patient 3, are consistent with the additional role of inflammation in this process.

We believe that the phenomenon of spontaneous conversion of long-standing AF or AFl, including the progression from AF to low amplitude slow AFl, and then to sinus rhythm, associated with profound sinus node dysfunction, while uncommon, does occasionally occur and that clinicians should be aware of this possibility. Its previous association with both rheumatic heart disease and digoxin merits reconsideration, as although it is most likely the result of a severe atrial myopathy due to scar and inflammation,[6-8] its cause may be variable.

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Table 1
Pertinent findings in 3 patients with spontaneous conversion of long-standing atrial fibrillation/flutter

Variable	Patient 1	Patient 2	Patient 3
Age (years)	70	100	74
Gender (M/F)	M	M	M
Age at onset of AF (years)	67	98	68
Coronary artery disease	0	+	0
Hypertension	+	+	0
Chronic kidney disease	+	0	0
Left atrial volume index (ml/m^2)	85	46	54
Left ventricular function	Normal	Normal	Normal
Heart failure	0	0	+
Inflammation/fibrosis factors	Chronic infection	Advanced age	Pericarditis graft vs host disease
Pacemaker required	+	+	+

AF = atrial fibrillation; F = female; M = male.

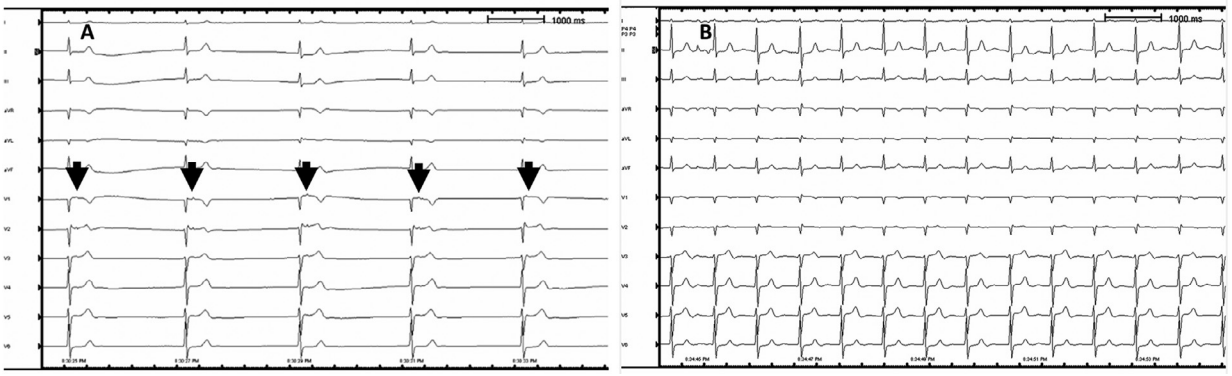

Figure 1. *Panel A*: Spontaneous junctional rhythm with evidence of organized isorhythmic atrial rhythm (arrows). *Panel B*: Paced atrial rhythm demonstrating atrial capture and intact AV conduction.

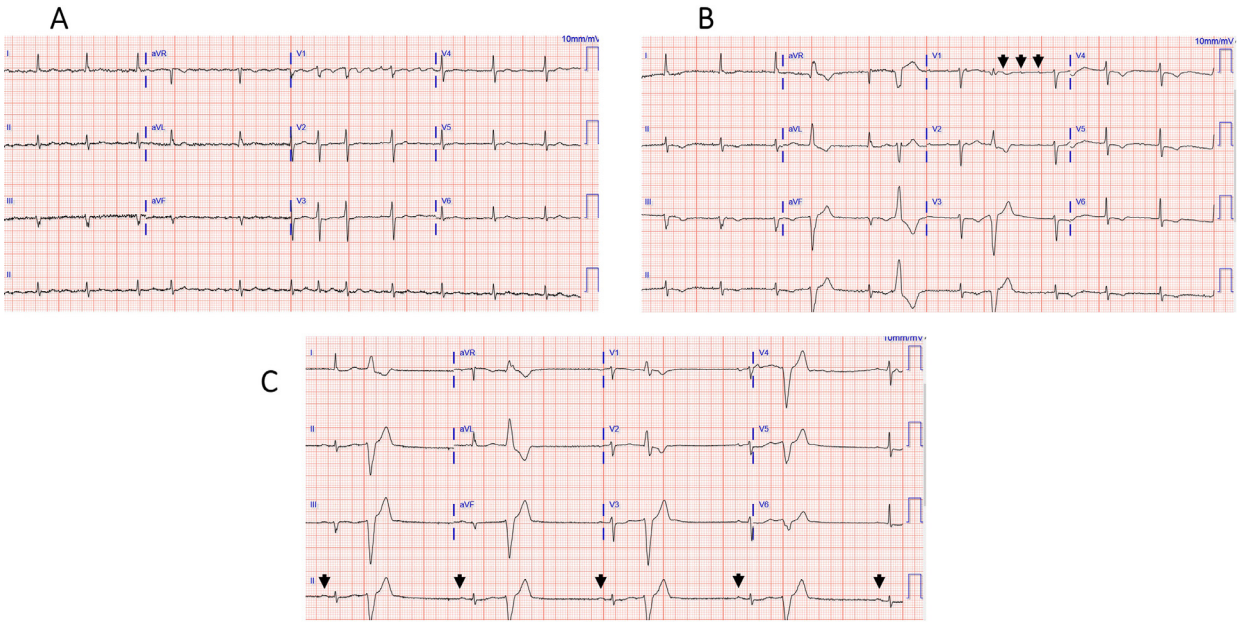

Figure 2. Representative ECGs, Patient 3. *Panel A*: Atrial fibrillation. *Panel B*: Slow, low amplitude atrial flutter (arrows: flutter waves). *Panel C*: Spontaneous sinus rhythm with ventricular bigeminy (*arrows is italicized here, but not elsewhere*: sinus P waves).

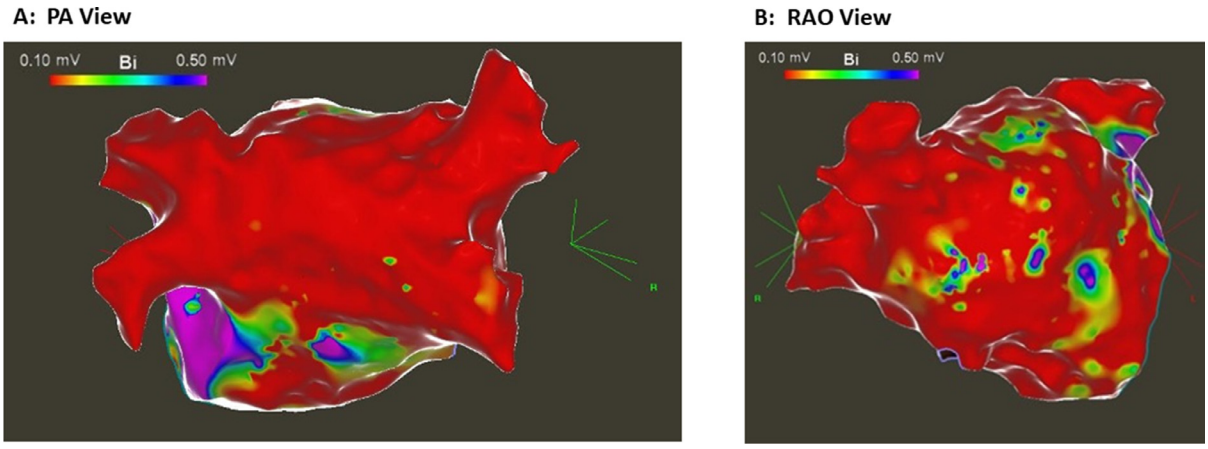

Figure 3. Posteroanterior (*Panel A*) and right anterior oblique (*Panel B*) views of left atrial voltage map, Patient 3. Red areas indicate scarring (~0.10 mV). mV = millivolt; PA = posterior anterior; RAO = right anterior oblique. (Color version of figure is available online.)

Disclosures

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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