Peri–coronary sinus atrial flutter associated with prior slow pathway ablation

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Introduction

A human anatomic study has shown that a cuff of striated muscle consistently surrounds the venous wall of the coronary sinus (CS) and is connected to the left atrium (LA) by muscular bundles in a variable fashion.[1] The presence of electrical connections between the CS musculature and LA myocardium provides a potential anatomic substrate for reentry. In fact, atypical atrial flutter (AFL) involving the CS musculature in its reentrant circuits has been reported,[2,3] and some studies have suggested that reentry via the CS–LA electrical connections could contribute to the initiation and maintenance of atrial fibrillation.[4,5]

Here, we describe a patient with a unique form of atypical AFL with a reentrant circuit utilizing the CS musculature and myocardium adjacent to the CS that occurred as an iatrogenic tachyarrhythmia following prior ablation of atrioventricular nodal reentrant tachycardia (AVNRT).

Case report

A 66-year-old man presented to our hospital, complaining of a 2-month history of palpitations and easy fatigability. At the age of 62, he had undergone slow pathway ablation for the slow-fast form of AVNRT in our department. In the previous session, radiofrequency (RF) energy was applied 3 times at the proximal CS up to 1 cm inside from the CS ostium, because RF ablation at the right inferoseptum had been ineffective. However, those ablation attempts in the CS failed and a successful ablation finally was accomplished at the superior aspect of Koch's triangle. On a 12-lead surface electrocardiogram, he now presented with AFL with sawtooth-like flutter

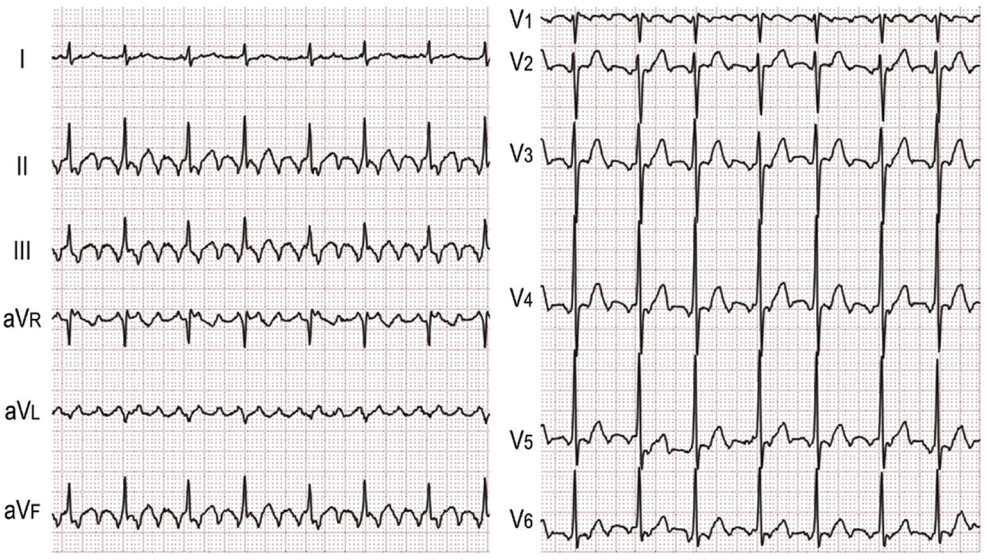

Figure 1 Surface 12-lead electrocardiogram showing atrial flutter (AFL) with 3:1 atrioventricular conduction. Sawtooth-like negative flutter waves were noted in the inferior leads. The negative flutter waves in all precordial leads were not compatible with typical AFL.

KEYWORDS Ablation; Atrial flutter; Coronary sinus; Reentry
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KEY TEACHING POINTS

- Peri–coronary sinus (CS) atrial flutter utilizing the CS musculature, adjacent left atrial (LA) myocardium, and CS–LA bridging muscular bundles can occur secondary to radiofrequency ablation within the CS.
- A disparate CS activation pattern may indicate the peri-CS atrial flutter.
- A critical slow conduction site in the CS associated with the prior ablation should be an ablation target for the peri-CS atrial flutter.

waves at a cycle length of 200 ms (Figure 1). The flutter waves were negative in the inferior leads and all precordial leads. A 24-hour Holter monitoring revealed that the AFL persisted throughout the day of monitoring. The transthoracic echocardiography was normal except for a slightly enlarged LA.

In an electrophysiological study, the AFL incidentally terminated when we attempted to insert a multielectrode catheter (6-mm interelectrode spacing for the CS; Japan Lifeline, Tokyo, Japan) into the CS. Atrial burst pacing during isoproterenol infusion induced nonclinical AFL (AFL #1). AFL #1 had the same cycle length and different morphologies when compared to the clinical AFL. During AFL #1, double potentials with decreasing interpotential intervals from the proximal to distal CS were noted (Figure 2A, upper). The early components had a high amplitude and frequency that seemed to represent local potentials from the CS musculature, whereas the late components had a low amplitude and frequency, indicating far-field LA potentials. Although the difference between the postpacing interval and tachycardia cycle length (PPI-TCL) at the cavotricuspid isthmus (CTI) was <20 ms, creation of a CTI conduction block did not affect AFL #1. We constructed biatrial activation maps during AFL #1 with an electroanatomic mapping system (EnSite

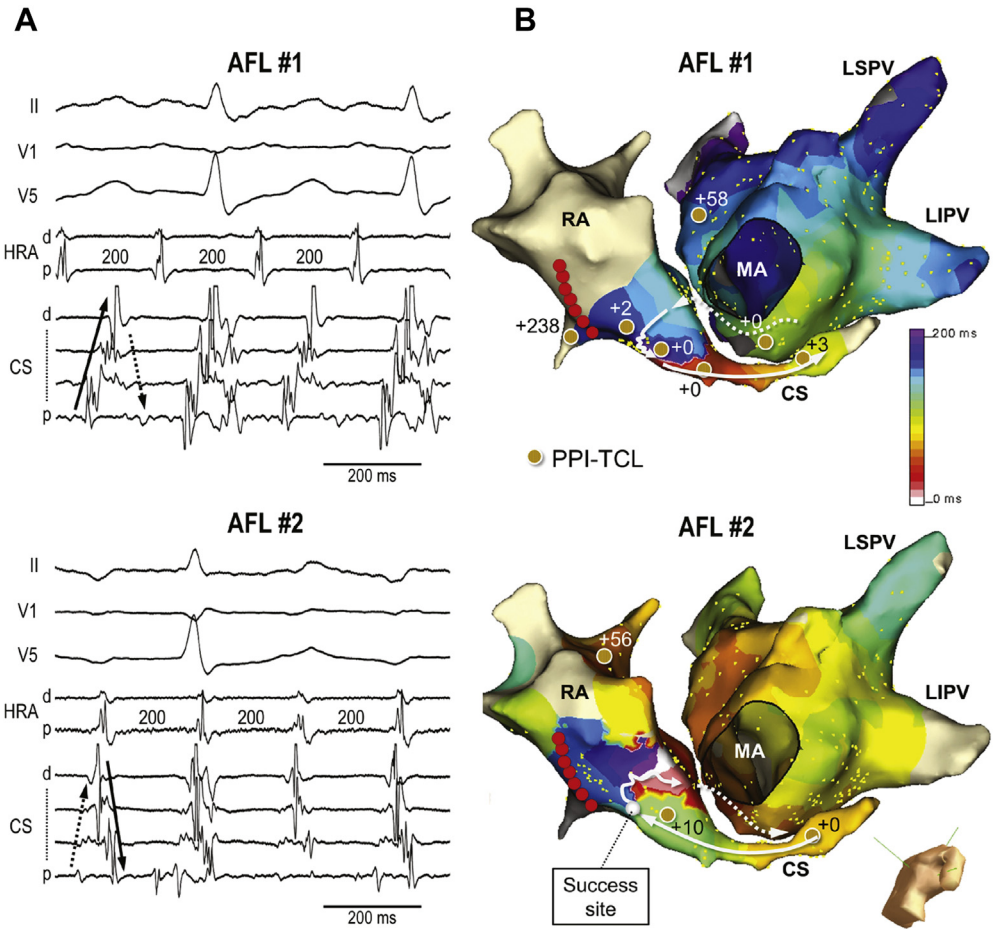

Figure 2 **A:** Surface electrocardiogram and intracardiac electrograms from the high right atrium (HRA) and coronary sinus (CS) during induced (AFL #1) and clinical (AFL #2) atrial flutter (AFL). The CS disparate activation pattern was observed both in AFL #1 and AFL #2, but the direction of the CS musculature (*arrows*) and left atrium (LA; *dotted arrows*) activations was the opposite between AFL #1 and AFL #2. **B:** Activation maps of the right atrium (RA) and LA during AFL #1 and AFL #2. Note the counterclockwise and clockwise activations around the CS area in AFL #1 and AFL #2, respectively. A conduction block line across the cavotricuspid isthmus is shown by the red tags. A partial conduction block with slow conduction was noted in the proximal CS. LSPV = left superior pulmonary vein; LIPV = left inferior pulmonary vein; MA = mitral annulus; PPI–TCL = difference between the postpacing interval and tachycardia cycle length.

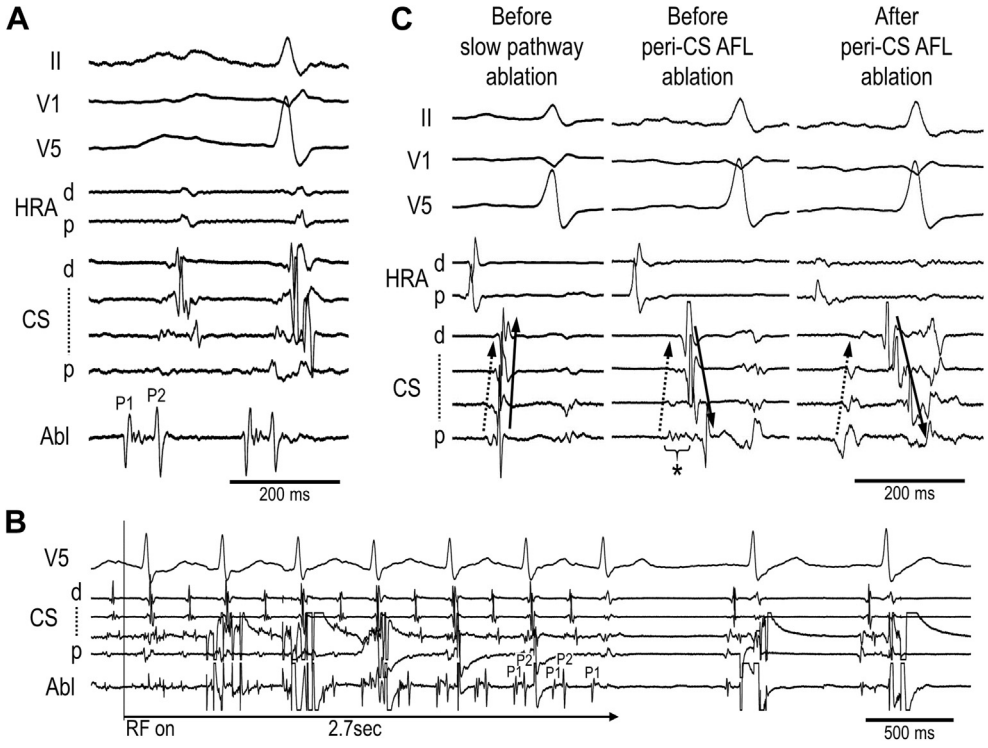

Figure 3 **A:** Intracardiac electrograms at the successful ablation site (see Figure 2B) for AFL #2. Note the double potentials (P1 and P2) with intervening fragmented activities. Abl = ablation catheter; other abbreviations as in Figure 2. **B:** The AFL terminated 2.7 seconds after the initiation of the radiofrequency (RF) application with conduction block between P1 and P2. **C:** Alterations in the coronary sinus (CS) electrograms during sinus rhythm before and after the ablation of the slow pathway and AFL. Before the slow pathway ablation in the previous session, the left atrium and CS electrograms were fused and activated in the same direction (left panel). A disparate activation pattern was already noted before the AFL ablation. Note the fragmented delayed potentials in the proximal CS (*asterisk*), implying an incomplete conduction block in the proximal CS (middle panel). There was a complete conduction block in the proximal CS after the AFL ablation, although isolation of the CS musculature was not achieved (right panel).

NavX, St. Jude Medical, St. Paul, MN), demonstrating macroreentry utilizing the CS and adjacent atrial myocardium in a counterclockwise direction (Figure 2B, upper). The LA mapping confirmed that the LA activations were responsible for the late components of the CS electrograms. Entrainment mapping showed that the PPI-TCLs were <20 ms only in the peri-CS area. Because AFL #1 terminated during the entrainment pacing, we attempted to reinduce AFL #1, and at that time the clinical AFL was induced (AFL #2, Figure 2A, lower). A similar CS activation pattern was observed during AFL #2, but the relationship of each component was reversed. Biatrial activation and entrainment mapping during AFL #2 revealed peri-CS macroreentry in a clockwise direction (Figure 2B, lower). In both AFL #1 and AFL #2, a critical slow conduction site was present in the proximal CS. An RF application at that site, where the double potentials with intervening fragmented potentials were recorded, terminated AFL #2 (Figure 3A and B). AFL #1 was considered to share the critical slow conduction site with AFL #2, because AFL #1 was no longer inducible after the successful ablation of AFL #2. We tried to eliminate the electrical connection between the distal CS and LA, since it also provided a critical pathway for both types of AFLs. Multiple RF applications delayed the conduction from the LA to the CS, but failed to block the LA–CS connection (Figure 3C). During a 17-month follow-up period, without any antiarrhythmic drugs, no tachycardia recurred.

Discussion

The electrical connection between the distal CS and LA links the inferior right atrium to the LA myocardium via the CS musculature, and forms an important pathway for interatrial propagation in human hearts.[1] The present case demonstrated that inferior interatrial pathways can provide anatomic substrates for macroreentry when slow pathway ablation for AVNRT impairs the conduction in the proximal CS. The peri-CS AFL was a unique atrial tachyarrhythmia with a reentrant circuit confined to the area around the CS. Because the septal part of the CTI was close to the peri-CS AFL circuit, the PPI-TCL at the CTI was <20 ms, which usually indicates CTI-dependent AFL. Thus, a short PPI-TCL at the CTI should be interpreted with caution unless a complete mapping of the AFL circuit is made, since creating a block line in the CTI is ineffective in peri-CS AFLs, as shown in the present case.

Olgin and colleagues[2] reported a case with atypical AFL involving the CS musculature exhibiting double potentials in the CS electrograms with disparate activation sequences, similar to those in our case. In their case, activation and entrainment mapping showed that the anterior and lateral

LA along the mitral annulus were parts of the reentrant circuit, but the inferior and posterior LA close to the CS were not. Hence, the LA components of the double potentials in the CS did not represent activations in the critical pathway of the AFL. It was reported that a CS disparate activation pattern was observed in 13% of AFLs with a history of persistent atrial fibrillation ablation, regardless of the tachycardia mechanism.[6] In contrast, both the double potentials in the CS were critical for the AFL in this patient, indicating that the disparate CS activation pattern was mandatory for the peri-CS AFL.

To the best of our knowledge, this is the first report demonstrating peri-CS AFL. The prior slow pathway ablation for AVNRT seemed to be responsible for this unique form of atypical AFL, because the impaired conduction in the proximal CS was not present before slow pathway ablation in the previous session (Figure 3C). Thus, special attention should be paid to RF application in the CS, as peri-CS AFL could occur following RF applications within or around the CS for any type of arrhythmias. It might be useful to consider the possibility of peri-CS AFL if a disparate CS activation pattern is observed in patients with atypical AFL.

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