Sustained Fibrillation Within the Left Atrial Appendage During Catheter Ablation for Recurrent Atrial Tachyarrhythmia

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Abstract

The left atrial appendage (LAA) has recently been recognized as a potential source of arrhythmia in patients undergoing repeat ablation procedures for atrial fibrillation (AF). In this case report we describe sustained fibrillation contained entirely within the LAA, that continued even after electrical isolation of the LAA was performed. This case supports the concept that in selected patients with AF, catheter ablation strategies may need to incorporate LAA isolation to minimize recurrence.

Introduction

The left atrial appendage (LAA) has been recognized as a potential source of arrhythmia in patients undergoing repeat ablation procedures for atrial fibrillation (AF).1

Case

A 58 year-old man with a history of symptomatic persistent atrial fibrillation (AF) unresponsive to cardioversion and dronedarone therapy presented for his third catheter ablation procedure for atrial tachyarrhythmia. At his first procedure, performed 14 months prior to the current one, he presented in AF and underwent circumferential pulmonary vein isolation with wide circles around the left- and right-sided veins in pairs. He remained in AF after pulmonary vein isolation was achieved, and further lesions were delivered in the form of a roof line and a posterior mitral isthmus line from the mitral annulus to the line that circled the left inferior pulmonary vein. During ibutilide infusion, AF transformed into an organized atrial tachyarrhythmia and eventually converted to sinus rhythm. Further ablation was performed to achieve conduction block across both lines. A repeat procedure was performed 3 months later for atypical atrial flutter, during which recurrent conduction was noted across the mitral isthmus line. Repeat ablation along the prior posterior line resulted in conversion of flutter to sinus rhythm. However, during further ablation to achieve mitral isthmus block, inadvertent electrical isolation of the left atrial appendage (LAA) was noted. Cavo-tricuspid isthmus dependent atrial flutter was induced at that time and was also ablated. The patient was maintained on warfarin and remained clinically stable for 6 months, but then developed frequent symptomatic atrial premature complexes that were minimally responsive to beta blocker and to flecainide therapy. The
week prior to this procedure, he developed acute palpitations and was found to be in atrial tachycardia/atrial flutter with variable AV conduction and ventricular rate 140 beats per minute. The 12-lead electrocardiogram (ECG) after rate control was achieved revealed negative p waves in leads I/L and bifid p waves in leads III and aVF (Figure 1). Burst pacing from the high right atrium and from the coronary sinus down to cycle length 180 milliseconds resulted in no inducible arrhythmia, before and during isoproterenol infusion. However, during rapid pacing from a 20-pole Lasso catheter in the LAA, re-entrant tachycardia within the appendage was induced, with cycle length 230 milliseconds and with variable conduction to the remainder of the left atrium. Ablation at an area of fractionated electrograms at the superior ridge between the LAA and the left superior pulmonary vein resulted in conversion to sinus rhythm.

Repeat burst pacing from the LAA resulted in sustained fibrillatory activity within the LAA, with variable conduction to the remainder of the atrium that manifested on the surface ECG as an irregular atrial rhythm with varying p wave morphology (Figure 3a).

Two ablation lesions within the atrial appendage at areas of fractionated electrograms failed to terminate fibrillation. Finally we performed further ablation at the base of the appendage in order to isolate the LAA, resulting in sustained sinus rhythm without atrial ectopy on the surface ECG (Figure 3b).

Fluoroscopic images of the site of LAA isolation are shown in Figure 4.

Cardioversion was performed to terminate the LAA rhythm. The patient has remained arrhythmia free for 7 months while on low-dose beta blocker, and has been anticoagulated with therapeutic warfarin.

Discussion

To our knowledge this is the first description of sustained fibrillation contained entirely within the LAA. Recently, Di Biase and colleagues observed that the LAA may be a source of atrial arrhythmia in as many as 27 percent of patients presenting for repeat ablation procedures for AF. This case illustrates the potential importance of the LAA not only in triggering, but in the longer-term maintenance of AF. Our patient’s LAA by preoperative CT scan measured 28 by 22 by 44 mm, and we speculate that a critical mass of LAA tissue was the primary contributor to the possibility of sustained fibrillatory activity.

Of note, at the outset of the current procedure, pulmonary vein isolation was present, and block was intact across each of the prior ablation lines (roof, mitral isthmus, cavo-tricuspid isthmus). Sustained arrhythmia was inducible only with rapid pacing from the anterior left atrium at the LAA, in the setting of prolonged conduction time between the posterior and anterior left atrium. The anterior left atrium may be a useful site at which to test for inducibility of arrhythmia after roof and mitral isthmus lines are intact, particularly when stimulation at other sites such as the right atrium and coronary sinus does not result in arrhythmia.

Figure 1: Twelve-lead ECG during recurrent atrial tachycardia/atrial flutter. P waves are marked with arrows.

Figure 2: Prolonged conduction time from p wave onset to a circular mapping catheter (Lasso) in the left atrial appendage.
Inadvertent isolation of the LAA during catheter ablation has been described during ablation at sites other than at the LAA, such as at the Bachmann bundle or during creation of mitral isthmus lines, as occurred in our patient during his previous procedure. During the current procedure, after sustained fibrillation was induced in the LAA we weighed the risks and benefits of intentional LAA isolation. We decided to pursue LAA isolation due to the clinical circumstance and in light of the option for subsequent LAA closure by minimally invasive methods. It is important to note that we cannot prove that electrical isolation of the LAA cured our patient’s clinical arrhythmia, since the ablation was performed in response to an induced tachycardia. However, his clinical stability during the 7 months since his last procedure argues in favor of LAA isolation having been necessary to achieve sustained benefit from catheter ablation.

Although the thromboembolic risk associated with LAA isolation in general has not been established, clearly there is potential for sustained fibrillation to occur within the LAA of our patient, without ability to detect it by surface ECG. This case offers proof of concept that anticoagulation may need to be continued in certain patients after intentional isolation even if mechanical function of the LAA is initially intact. LAA closure may be particularly useful for patients who require electrical LAA isolation.

References

