A 39-year-old man developed highly symptomatic, paroxysmal atrial fibrillation. Treatment with antiarrhythmic drugs failed, and he underwent an ablation procedure at an outside hospital at age 41 years. This procedure involved isolation of the pulmonary veins and creation of a left atrial roof line, a mitral isthmus line, and a cavotricuspid isthmus line. The patient had a structurally normal heart with a calculated CHADS2 risk of zero, so he was treated with warfarin for several months after the procedure and was subsequently switched to aspirin. His atrial fibrillation recurred 2 years after ablation, requiring multiple cardioversions. Of note, the patient presented with cardioembolic events, including a transient ischemic attack and a splenic infarct, in the context of subtherapeutic anticoagulation levels following cardioversion. Owing to the severity of his atrial fibrillation-related symptoms and vocation-related concerns regarding his candidacy for long-term warfarin therapy, the patient was then referred for a surgical ablation procedure. He underwent minimally invasive robotic surgical cryomaze and left atrial appendage (LAA) suture ligation. Cryothermal lesions were placed in a “box” around all pulmonary veins, and this lesion set was then connected with further cryolesions to the mitral annulus and to the base of the LAA. It was thought that ligation of the LAA could facilitate safe discontinuation of coumadin anticoagulation even if his atrial fibrillation were to recur. Several months after surgical ablation, the patient presented to our arrhythmia service with incessant tachycardia resistant to cardioversion despite amiodarone therapy and dyspnea on light exertion. He was found to be in atypical atrial flutter with 2:1 atrioventricular conduction (Figure 1). A trans-thoracic echocardiogram revealed a dilated left ventricle with an ejection fraction of 40%. Based on failure of medical therapy and presence of tachycardia-mediated cardiomyopathy, the decision was made to proceed with repeat catheter ablation.

A preprocedural computed tomography angiogram (CTA) revealed the presence of contrast in the LAA, consistent with incomplete LAA ligation, with a small neck connecting the LAA to the left atrial (LA) body (Figure 2a). A transesophageal echocardiogram (TEE) with Doppler on the day of the procedure revealed no evidence of left atrial thrombus, but blood flow was present in the narrow communication between the body of the LA and the LAA (Figure 3a). Initial mapping revealed that the left pulmonary veins (PVs) were not
Figure 1: Electrocardiogram performed on the patient when he was referred to our service revealed an atypical atrial flutter with 2:1 atrioventricular conduction (a). Electroanatomical mapping during the final ablation procedure confirmed the presence of an atypical atrial flutter involving the left atrial (LA) roof (b, earliest activation shown as red). Ablation points in the LA roof line as well as some of the ablation points associated with isolation of the left pulmonary veins (PVs) are visible on this map. After successful ablation of the LA roof, the patient was noted to be in an atrial tachycardia, which emanated from the septal aspect of the left atrium, adjacent to the LA roof line (c, earliest activation shown as red). Ablation points delivered in the area of the right PVs in order to ablate the new atrial tachycardia and to complete isolation of the posterior wall are visible in this map.
Figure 2: A computed tomography angiogram (CTA) performed after surgical ligation of the left atrial appendage (a) reveals a narrow communication between the body of the left atrium and the appendage (red arrow). A repeat CTA, performed in conjunction with planned percutaneous closure of the left atrial appendage (LAA), revealed no evidence of contrast in the LAA (red arrow).
Figure 3: (a) A transesophageal echocardiogram (TEE) with Doppler on the day of the final ablation procedure revealed blood flow within the area of communication between the left atrium and the left atrial appendage (LAA). There was no thrombus present in the LAA so the ablation procedure was performed. Repeat TEE with Doppler performed in advance of planned percutaneous closure of the LAA (b) revealed that the appendage had sealed spontaneously. A thrombus was visible within the isolated LAA.
isolated. Ablations were delivered at the ostia of the left PVs and electrical isolation was achieved. Further electroanatomic mapping confirmed that the patient had an atypical atrial flutter, which was successfully ablated at the LA roof at a site of fractionated electrograms (Figure 1b). Further ablation was performed to completely isolate the posterior wall. Block across the mitral isthmus and cavitricuspid isthmus lines was confirmed.

Rapid atrial pacing then induced a new atrial tachycardia after ablation of the presenting arrhythmia on the LA roof. An activation map was created, and revealed an atrial tachycardia emanating from an area adjoining the previously created RSPV ablation line (Figure 1c). An attempt at entrainment at this site (with fractionated potentials noted on the mapping/ablation catheter) with atrial pacing led to termination of the tachycardia. The tachycardia could not be reinduced with the catheter at this site, so ablation was performed at the site of the micro-reentrant circuit (Figure 1c). Rapid atrial pacing down to a cycle length of 200 ms could not induce any further tachycardia after the ablation. The patient remained in normal sinus rhythm after the procedure, and his cardiomyopathy and symptoms completely resolved.

Incomplete surgical ligation of the LAA can be an important and under-recognized source of thromboembolic events.2,3 Coumadin was continued in this case due to risk of further thromboembolic events related to stagnation of blood in the LAA. Vocation-related risk of anticoagulation therapy was an ongoing concern (employment as a corrections officer prevented him from working while taking warfarin, and he is also a recreational motorcyclist). In order to facilitate safe termination of coumadin, the patient then returned 6 months after catheter ablation for percutaneous closure of the communication between the LAA and the LA body.1 A preprocedural TEE with Doppler now revealed no communication between the body of the LA and the LAA. Thrombus was visible within the enclosed LAA (Figure 3b). Occlusion of the LAA was confirmed with a repeat CTA (Figure 2b), so percutaneous closure was not indicated. Warfarin was stopped and aspirin was resumed at that point, since the LAA was completely occluded and there was no evidence of valvular disease. The patient has remained free of arrhythmia and embolic events for 3 years after the final ablation procedure, and has returned to his vocation as a corrections officer and hobby as a motorcyclist.

Discussion

Incessant atrial tachycardia after either catheter or surgical ablation of atrial fibrillation can lead to severe symptoms and tachycardia-mediated cardiomyopathy, and is a frequent cause for repeat ablation procedures. Surgical ablation procedures allow concomitant LAA ligation or amputation, but incomplete ligation or reopening of a ligated LAA exposes the patient to the risk of thromboembolism. As demonstrated in this case, it is possible for an open connection between a surgically ligated LAA and the left atrium to seal spontaneously over time.

References