Initial experience with post Lariat left atrial appendage leak closure with Amplatzer septal occluder device and repeat Lariat application

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BACKGROUND Left atrial appendage (LAA) ligation with the Lariat device is a therapeutic option to prevent thromboembolic stoke in patients with nonvalvular atrial fibrillation (AF) at high risk for systemic thromboembolization and bleeding related to use of anticoagulation. In rare cases, this procedure could leave the LAA incompletely ligated with continued risk of stroke.

OBJECTIVE The purpose of this study was to investigate the incidence and characteristics of LAA leak following ligation using the Lariat device and the feasibility of leak closure with the Amplatzer septal occluder device or a repeat Lariat application.

METHODS Seventy-one consecutive patients who underwent LAA ligation by the Lariat device were followed-up with transesophageal echocardiography to evaluate for the presence of appendage leaks, characterization of the leaks, and the presence of any thrombus. Patients with LAA leaks underwent definite closure of the leak.

RESULTS Six patients had LAA leaks with a mean leak size of 4.3 ± 0.6 mm. All leaks were concentric in nature. None of the patients had LAA thrombus. Leaks in 5 of these patients were successfully closed using an Amplatzer septal occluder device (St. Jude Medical); the leak in the sixth patient was closed using a repeat Lariat procedure.

CONCLUSION LAA leaks from incomplete ligation of the LAA following the Lariat procedure are not uncommon and could be successfully closed with an Amplatzer septal occluder device or a repeat Lariat procedure.

KEYWORDS Left atrial appendage; Atrial fibrillation; Lariat procedure; Amplatzer septal occluder; Thromboembolic stroke

ABBREVIATIONS AF = atrial fibrillation; **ASD** = atrial septal defect; **ASO** = Amplatzer septal occluder; **ISLL** = incompletely surgically ligated left atrial appendage; **LA** = left atrium; **LAA** = left atrial appendage; **TEE** = transesophageal echocardiography

Introduction

The left atrial appendage (LAA) has been shown to be the most common source for thrombus formation leading to systemic thromboembolic events in the majority of patients with atrial fibrillation (AF).[1] More than 90% of left atrial (LA) thrombi in these patients have been demonstrated to be in the LAA.[2] This makes the LAA a high-yield target for surgical or percutaneous exclusion in patients with intolerance or contraindication to oral anticoagulants. particularly in those at high risk for bleeding and in patients with cardioembolic stroke despite therapeutic anticoagulation. This strategy is also adopted in certain patients as an alternative to lifelong anticoagulation as a choice even in the absence of contraindication to anticoagulation. There has also been an increasing body of evidence on the role of the LAA as a nonpulmonary vein source of trigger for AF.[3] LAA exclusion procedures, which can also electrically isolate the appendage, may have a role in AF burden reduction as well.

LAA exclusion can be achieved with different strategies[4–7]: (1) surgical ligation or exclusion; (2) percutaneous endovascular occlusion using the Watchman LAA system (Atritech Inc, Plymouth, MN) or the Amplatzer cardiac plug (AGA Medical, Plymouth, MN); or (3) pericardial suture ligation of the LAA base with the Lariat snare device (SentreHEART, Redwood, CA) using a combined endocardial and epicardial approach. Incomplete occlusion of the LAA by any of these methods could lead to continued risk of thromboembolism with the need for continued anticoagulation or a definitive closure strategy in patients at prohibitively high risk for anticoagulation.

We describe 5 cases of incomplete LAA ligation using the Lariat device that were successfully closed by endocardial occlusion using an Amplatzer septal occluder (ASO) device

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(St. Jude Medical, Minneapolis, MN) and 1 case in which a repeat Lariat ligation was performed.

Methods and results

Case series

Seventy-one patients underwent the Lariat procedure at a single center, and all patients were followed-up by transesophageal echocardiography (TEE) 3 months postprocedure. Six patients (8.5%, 4 males, mean age 69 years; Table 1) had LAA leaks with a mean leak size of 4.3 ± 0.6 mm. All leaks were concentric in nature. None of the patients had LAA thrombus.

Case 1

A 74-year-old woman with permanent AF status post multiple left atrial antral ablation who suffered an embolic occipital cerebrovascular accident despite taking Coumadin ($CHADS_2 = 4$, CHADSVASC = 5, HAS-BLED = 3) with a risk of fall prohibitively high for taking Coumadin underwent percutaneous ligation of the LAA with the Lariat device. The patient had a large LAA with an os measuring 40 mm. Considerable challenge was encountered encircling the whole appendage with the Lariat device, requiring multiple attempts. A 1-mm central leak at the end of the procedure was confirmed by TEE and angiography. Three months postprocedure, TEE showed a persistent central leak of 4 mm with full opacification of the LAA with contrast study (Figure 1).

Case 2

A 61-year-old man with recurrent AF status post multiple left atrial antral ablation had a transient ischemic attack while taking therapeutic Coumadin ($CHADS_2 = 4$, CHADSVASC = 4, HAS-BLED = 4) and underwent percutaneous ligation of the LAA with the Lariat device. The procedure was unremarkable without any postprocedural leaks. Three months postprocedure, TEE showed a new central leak of 5 mm with an appendage emptying velocity of 0.4 m/s.

Case 3

A 65-year-old man with nonischemic cardiomyopathy with cardiac resynchronization therapy with defibrillator (CRT-D), hypertension, and persistent AF ($CHADS_2 = 2$, CHADSVASC = 2, HAS-BLED = 2) underwent percutaneous ligation of the LAA with the Lariat device along with pulmonary vein isolation for AF. Two months later, a 5-mm recanalization of the LAA was seen on TEE without any evidence of thrombus (Figures 2 through 4).

Case 4

A 70-year-old man with paroxysmal AF, coronary artery disease, pacemaker for sick sinus syndrome and multiple embolic cerebrovascular accidents despite anticoagulation with rivaroxaban (CHADS2 = 3, CHADSVASC = 5, HAS-BLED = 3) underwent percutaneous ligation of the LAA with the Lariat device and AF ablation. The procedure was unremarkable without any postprocedural leaks. Three months postprocedure, TEE showed a new central leak of 4 mm.

Case 5

A 79-year-old woman with longstanding persistent AF despite 2 antiarrhythmic drugs ($CHADS_2 = 2$, CHADSVASC = 5, HAS-BLED = 2) underwent percutaneous

Table 1 Baseline patient characteristics and details of appendage leak in the 6 patients

Patient	1	2	3	4	5	6
Age (years)	74	61	64	72	78	67
Sex	F	M	M	M	F	M
Past medical history	Permanent AF	Persistent AF	Persistent AF	Paroxysmal AF	Persistent AF	Persistent AF
	NICM	Bicuspid aortic valve	NICM	CAD	Hypertension	CAD
	AVN ablation PPM		CRT-D	SSS, PPM	Carotid disease	SSS, PPM
	Embolic CVA	TIA	HTN	Embolic CVA		Embolic CVA
	EF 40%	EF normal	EF normalized	EF normal	EF normal	EF normal
$CHADS_2$	4	4	2	3	2	3
CHADSVASC	5	4	2	5	5	4
HAS-BLED	3	4	2	3	2	3
LA size/volume	4.8 cm/151 cc	4.6 cm/138 cc	5 cm/185 cc	5.3 cm/165 cc	4.6 cm/122 cc	5.2 cm/178 cc
Indication for Lariat	CVA despite therapeutic Coumadin	CVA despite therapeutic Coumadin	LAA AF triggers	CVA despite therapeutic OAC	AF burden reduction	TIA despite therapeutic warfarin
TEE post 3 months	4-mm leak	5-mm leak	5-mm leak	4-mm leak	3.5-mm leak	4.5-mm leak
ASO device	5 mm	5 mm	5 mm	5 mm	4 mm	None (repeat Lariat ligation)

AF = atrial fibrillation; AVN = atrioventricular node; ASO = atrial septal occluder; CAD = coronary artery disease; CRT-D = cardiac resynchronization therapy with defibrillator; CVA = cerebrovascular accident; EF = ejection fraction; HTN = hypertension; LA = left atrium; LAA = left atrial appendage; NICM = nonischemic cardiomyopathy; OAC = oral anticoagulation; PPM = permanent pacemaker; SSS = sick sinus syndrome; TEE = transesophageal echocardiography; TIA = transient ischemic attack.