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Surgical Left Atrial Appendage Ligation Is Frequently Incomplete: A Transesophageal Echocardiographic Study

Edward S. Katz, MD, FACC, Theofanis Tsiamtsiouris, MD, Robert M. Applebaum, MD, FACC, Arthur Schwartzbard, MD, FACC, Paul A. Tunick, MD, FACC, Itzhak Kronzon, MD, FACC

New York, New York

OBJECTIVES This study sought to determine the incidence of incomplete ligation of the left atrial appendage (LAA) during mitral valve surgery.

BACKGROUND Ligation of the LAA to prevent future thromboembolic events is commonly performed during mitral surgery. However, success in completely excluding the appendage from the circulation has never been systematically assessed.

METHODS Using transesophageal Doppler echocardiography, we studied 50 patients who underwent mitral valve surgery and ligation of the LAA. Thirty patients were studied immediately postoperative, and 20 patients were studied 6 days to 13 years after surgery. Incomplete ligation was detected by demonstrating a color jet traversing the separation between the left atrial body and appendage.

RESULTS Transesophageal echocardiography detected incomplete LAA ligation in 18 of 50 (36%) patients. The incidence of incomplete ligation was not significantly different between patients studied immediately postoperative and patients studied at various times after surgery. Type of mitral surgery (repair vs. replacement), operative approach (sternotomy vs. port access), left atrial size or degree of mitral regurgitation did not significantly correlate with the incidence of incomplete appendage ligation. However, the power to detect a significant difference in left atrial size was only 64%. Spontaneous echo contrast or thrombus was identified within appendages in 9 of 18 (50%) patients with incomplete ligation, while 4 of these 18 (22%) patients had thromboembolic events.

CONCLUSIONS Surgical LAA ligation is frequently incomplete. The similar incidence of incomplete ligation detected immediately postoperative and at various times thereafter suggest that this results from an intraoperative phenomenon rather than from gradual dehiscence of sutures over years. The incidence of incomplete left atrial ligation was unrelated to type of surgery, surgical approach, left atrial size or degree of mitral regurgitation. Residual communication between the incompletely ligated appendage and the left atrial body may produce a milieu of stagnant blood flow within the appendage and be a potential mechanism for embolic events. (J Am Coll Cardiol 2000;36:468–71) © 2000 by the American College of Cardiology

Ligation of the left atrial appendage (LAA) is commonly performed during mitral valve (MV) surgery (1–3). Because the LAA is a frequent site of clot formation in patients with MV disease, especially in those with atrial fibrillation, it is thought that complete obliteration of the communication between the LAA and the body of the left atrium (LA) eliminates the potential for stagnant blood flow within the appendage and for embolization of a thrombus formed within. Complete obliteration of the appendage cavity is, therefore, the surgical aim, but this has been difficult to verify by objective means. Using transesophageal echocardiography, we and others (4–7) have previously reported the incidental finding of patent flow between the LA and its appendage (or incomplete ligation) after attempts at complete surgical ligation. Success, however, in completely excluding the LAA from the circulation has never been systematically assessed. In addition, reasons for incomplete ligation (that is, its relationship to left atrial size, pressure or surgical technique, for example) have not been evaluated. In this study, we assessed the incidence of incomplete ligation of the LAA and explored various mechanisms that may play a role.

METHODS

We studied 50 patients (ages 25–83, mean 63 years; 35 women, 15 men) who underwent MV surgery and ligation of the LAA. The operations were performed by six experienced cardiovascular surgeons. In each case, ligation of the LAA was accomplished using a double row of 4–0 prolene running sutures, sewn from within the LA. Thirty-five (70%) patients underwent MV replacement, while 15 (30%) had MV repair and annuloplasty. Eight of 50 patients (16%) had cardiac surgery via the minimally invasive/port access approach, and the remainder underwent surgery via the more traditional median sternotomy. Thirty patients (60%) were studied in the operating room immediately after cardiopulmonary bypass, and 20 patients (40%) were studied from six days to 13 years (mean 64 months) after surgery.

From the Department of Medicine, Division of Cardiology, New York University School of Medicine, New York, New York.

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Abbreviations and Acronyms

LA = left atrium
LAA = left atrial appendage
MV = mitral valve

Multiplanar transesophageal echocardiography was performed on all patients, exploring the LAA in various planes from 0 to 120°. Incomplete LAA ligation was diagnosed by color flow Doppler, which demonstrated a to-and-fro jet traversing the separation between the LAA and the left atrial body (Fig. 1A). The LAA was explored for thrombus and for the presence of spontaneous echo contrast. Left atrial size was assessed by transthoracic echocardiography, performed within 48 h of the surgery or at the time of transesophageal echocardiography. Mitral insufficiency was assessed at the time of transesophageal echocardiography. The severity of mitral insufficiency was determined using color flow Doppler to measure the area of the maximal regurgitant jet (8).

Statistics. Categorical variables were compared using the chi-square method. A p value <0.05 was considered significant.

RESULTS (TABLE 1)

Incomplete LAA ligation was detected in 18 of the 50 (36%) patients. The incidence of incomplete ligation was as frequent in those studied intraoperatively as it was in those studied at various times after surgery (10/30, 33% vs. 8/20, 40%, p = 0.63).

Left atrial size was available in 49 patients. Table 2 shows the distribution of left atrial size among patients with completely and incompletely ligated LAAs. A severely dilated LA did not significantly correlate with the presence of incomplete left atrial ligation (Table 1). Although the incidence of incomplete ligation was higher in patients with a severely dilated LA (≥6.0 cm) than it was in patients with a smaller LA (6/12, 50% vs. 12/37, 32%), this difference did not reach statistical significance (p = 0.27).

Table 3 shows the distribution of degrees of mitral insufficiency among patients with completely and incompletely ligated LAAs. The degree of mitral insufficiency did not correlate with the surgical outcome of an incompletely ligated appendage (Table 1). Comparing patients with greater than mild mitral insufficiency to those with mild or no mitral insufficiency, there was an equal proportion of incompletely ligated LAAs (5 of 14, 36% vs. 13 of 36, 36%, p = 0.97).

Neither surgical approach nor surgical procedure predisposed to an incompletely ligated LAA (Table 1). Three of eight patients (38%) with a port access minimally invasive approach versus 15 of 42 patients (36%) MV resulted in incompletely ligated appendages (p = 0.92). In patients who underwent MV replacement, 13 of 35 patients (37%) had incompletely ligated appendages versus 5 of 15 (33%) who underwent MV repair (p = 0.80).

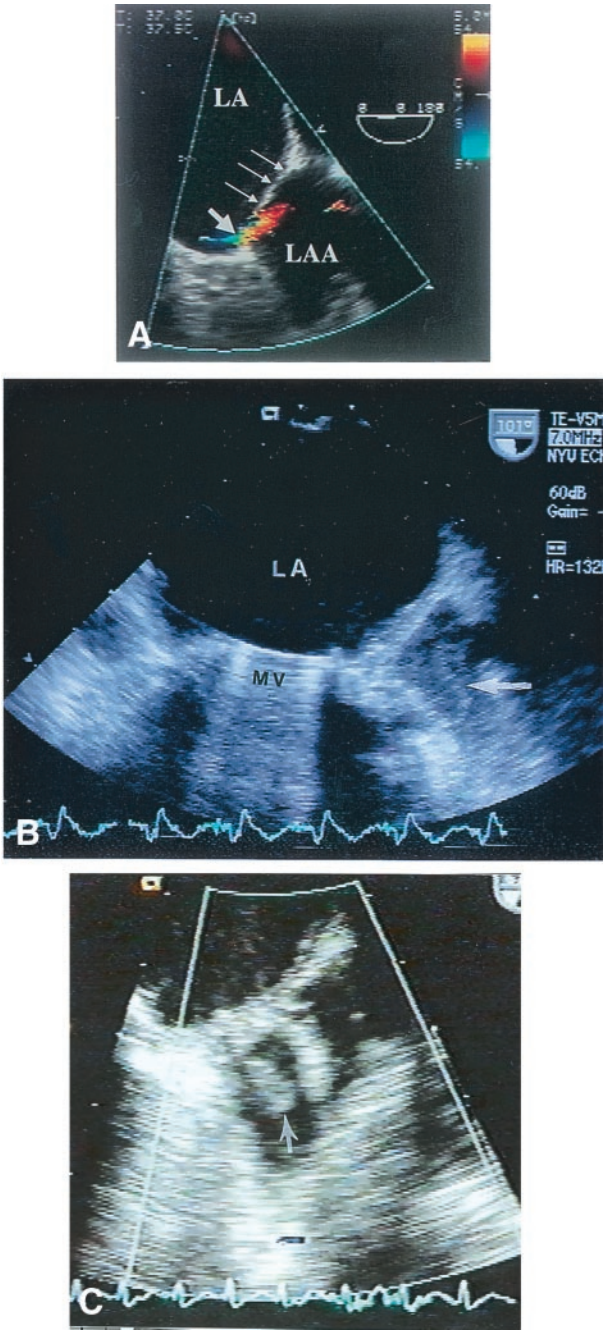

Figure 1. (A) Transesophageal echocardiogram, transverse plane, of an incompletely ligated LAA. Color flow traverses the separation between the LAA and the left atrial body **(bold arrow)**. The ligation suture line is shown with **thin arrows**. **(B)** Vertical plane transesophageal echocardiogram of a patient with a mitral mechanical prosthesis and an incompletely ligated LAA. Note the presence of spontaneous echo contrast **(arrow)** in the LAA compared with the relative absence of this finding in the left atrial body. **(C)** Vertical plane transesophageal echocardiogram of a patient with a mitral bioprosthesis and an incompletely ligated LAA. Thrombus is seen within the appendage **(arrow)**. LA = left atrium; LAA = left atrial appendage; MV = mitral valve.

Spontaneous echo contrast or thrombus was seen within the incompletely ligated LAA in 9 of 18 (50%) patients

Table 1. Comparison of Categorical Variables in Patients With Ligated and Incompletely Ligated Left Atrial Appendages

	Ligated Left Atrial Appendage (n = 32) n (%)	Incompletely Ligated Left Atrial Appendage (n = 18) n (%)	
Transesophageal Echocardiogram			
Intraoperative	20 (67%)	10 (33%)	p = 0.63
After surgery (6 days–13 years)	12 (60%)	8 (40%)	
Left Atrial Size			
<6.0 cm	25 (68%)	12 (32%)	p = 0.27
≥6.0 cm	6 (50%)	6 (50%)	
Mitral Valve Regurgitation			
≤Mild	23 (64%)	13 (36%)	p = 0.97
Moderate–severe	9 (64%)	5 (36%)	
Surgical Procedure			
Mitral valve replacement	22 (63%)	13 (37%)	p = 0.80
Mitral valve repair	10 (67%)	5 (33%)	
Surgical approach			
Median sternotomy	27 (64%)	15 (36%)	p = 0.92
Minimally invasive	5 (62%)	3 (38%)	

(Fig. 1, B and C). In six of these nine patients (67%) with spontaneous echo contrast, the contrast was actually more dense within the appendage than within the LA body.

Four patients with the finding of an incompletely ligated LAA had thromboembolic phenomena (one stroke, one transient ischemic attack, two mesenteric emboli). Review of the referral reasons for the 20 patients studied with transesophageal echocardiography in our laboratory at various times after surgery revealed that five were referred to exclude a cardiac source of embolization. Four of these patients were from the subgroup of eight with incompletely ligated appendages (50%) versus one patient (17%) from the group of 12 with completely excluded appendages.

DISCUSSION

Ligation of the LAA is frequently performed during MV surgery to eliminate a potential source of emboli. However, the success of completely excluding the LAA from the circulation has never been systematically addressed. Transesophageal echocardiography offers unique visualization of the appendage in the beating heart (9,10) and can evaluate the integrity of the surgical ligation. Usually, when the LAA is ligated, its cavity is obliterated with clot (since no flow enters the cavity) and cannot be seen during echocardiography. This appearance was the same whether the patient was studied in the operating room or months after the surgery. When the appendage is incompletely ligated, not only can the appendage cavity be visualized but flow can be seen within the appendage as well as through an opening in the ligation site.

Factors contributing to incomplete ligation. We discovered that 36% of the time the LAA was found to be incompletely ligated after attempts at excluding it from the left atrial body. Factors, such as an enlarged LA or significant mitral regurgitation, which may be thought to increase left atrial tension and pressure (perhaps predisposing to incomplete ligation or dehiscence of sutures), did not appear to correlate with this finding. We also did not observe a correlation between appendage size and the incidence of incomplete ligation. In addition, the surgical procedure (mitral repair or replacement) and operative approach (traditional sternotomy or minimally invasive approach) did not change the incidence of incomplete ligation. It is possible, however, that the sample size in this report may have been too small to exclude a significant effect of these variables on the development of incomplete left atrial ligation. The power of this study to detect a significant difference in left atrial size (the variable closest to achieving a significant effect) between the group with incomplete ligation and the group with complete ligation is only 64% (assuming a clinically meaningful difference in left atrial size of 1.0 cm,

Table 2. Left Atrial Size Distribution in Patients With Ligated and Incompletely Ligated LAA

Left Atrial Size (cm)	Ligated LAA (n = 31)	Incompletely Ligated LAA (n = 18)
≤4.0	2	2
4.1–4.9	12	5
5.0–5.9	11	5
≥6.0	6	6

LAA = left atrial appendage.

Table 3. Distribution of Mitral Regurgitation in Patients With Ligated and Incompletely Ligated LAA

Mitral Regurgitation	Ligated LAA (n = 32)	Incompletely Ligated LAA (n = 18)
≤Mild	23	13
Moderate	7	5
Severe	2	0

LAA = left atrial appendage.

a significance level of 0.05 and using a two-tailed test). To achieve a power of 80%, a sample size of n = 74 would be needed.

Incomplete left atrial appendage ligation was as commonly seen in the operating room, evaluating the patient by transesophageal echocardiography immediately after terminating cardiopulmonary bypass, as it was seen in our laboratory evaluating patients referred for transesophageal echocardiography at various times after the surgery. This suggests that incomplete LAA ligation is not a degenerative process with suture dehiscence over time, but rather is present immediately after the initial surgery. Incomplete ligation may be secondary to several surgical factors. First, the running sutures used may not start and end exactly at the most distal edges of the atrial appendage, which may not be recognized with the appendage empty and unstretched while on cardiopulmonary bypass during surgery. In addition, caution must be taken during appendage ligation to avoid deep suture bites, which may involve the left circumflex coronary artery or its branches that may course in the area. This meticulous care may lead to shallower suture bites that may dehisce when the LA is once again filled and stretched after cardiopulmonary bypass. Both of these mechanisms may play a role, as in many cases flow was detected both at the edge of the appendage orifice (apparently around the end of the suturing line) and through an area at the midpoint of the appendage orifice (through the suture line). Lynch, et al. (7) reported six cases of incomplete LAA ligation when a purse string suture was used to accomplish the ligation, a technique different from that used by our surgeons. The actual incidence, however, of incomplete LAA ligation using their technique was not addressed.

Clinical implications. The clinical significance of an incompletely ligated LAA has never been studied. Theoretically, creating a small communication between the LA and LAA may produce stagnation of low velocity blood flow within the atrial appendage. The appendage would then be a model for thrombus formation and continue to serve as a potential source of embolization since a port of entry into the systemic circulation still exists. Although the numbers in our study are small, several observations support this theory. First, spontaneous echo contrast (a marker for stagnant blood flow and a precursor of thrombus formation) was seen within the appendage in half of the patients with incomplete ligation. Second, and perhaps more importantly, in two-thirds of patients with spontaneous echo contrast within the incompletely ligated appendage, the contrast was actually more dense within the appendage than within the left atrial body, suggesting a more stagnant and thrombogenic milieu. In two patients, frank thrombus was seen within the incompletely ligated appendage.

The ultimate question, however, is whether patients with incompletely ligated LAAs will have a higher incidence of thromboembolic events. In our study, four patients with incompletely ligated appendages had such events (one patient with Starr-Edwards prosthesis, two with St. Jude prosthesis and one patient status after mitral repair). This is quite a high number considering that only eight patients with incomplete ligation had any potential for long term follow-up (the other 10 patients with incomplete ligation were discovered in the operating room). However, one cannot exclude other etiologies for embolization (as mechanical prostheses or atrial fibrillation) and referral bias still clouds this issue. Certainly, a prospective long-term follow-up study is needed to definitely answer this question, controlling for other risk factors for thromboembolization and for the use of anticoagulation.

If the incidence of thromboembolic phenomena is, in fact, higher in patients with incompletely ligated appendages, several issues will need to be addressed. First, should LAA ligation be an obsolete procedure? Second, can any intraoperative measures be taken to assure complete exclusion of the appendage from the circulation? Third, are certain alternative techniques better than others in achieving complete ligation or, perhaps, would amputation and actual elimination of the appendage be a better procedure?

Reprint requests and correspondence: Dr. Itzhak Kronzon, 560 First Avenue—HW 228, New York, New York 10016. E-mail: Itzhak.Kronzon@med.nyu.edu.

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