Characteristics and Implications of Left Atrial Calcium on Cardiac Computed Tomography in Patients With Earlier Mitral Valve Operation

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Left atrial calcium (LAC) is often observed in patients who have undergone mitral valve (MV) surgery, but little is known about its characteristics and clinical implications. Therefore, we sought to investigate the structural and hemodynamic significance of LAC and its association with clinical outcomes. We investigated 327 patients with repaired or prosthetic MV who underwent cardiac CT from 2010 to 2017. The degree of LAC was analyzed and classified into three groups: group 1 (no LAC), group 2 (mild-to-moderate LAC), and group 3 (severe LAC). Clinical and echocardiographic characteristics and clinical outcomes were compared in three groups. LAC was seen in 79 (24.2%) patients. Groups 2 and 3 showed more prevalent atrial fibrillation, a rheumatic etiology, a higher number of previous surgeries, a larger LA volume index, and higher pulmonary artery systolic pressure than group 1. Paravalvular leakage of the MV increased progressively according to severity of LAC (15.4% in group 1, 39.3% in group 2, and 66.7% in group 3, p <0.001). Event-free survival rate for major adverse cardiovascular adverse events (log rank p = 0.033) and all-cause mortality (log rank p <0.001) were significantly different according to LAC group. In Cox regression analyses, presence of severe LAC was an independent predictor of all-cause mortality (hazard ratio: 4.44, 95% confidence interval: 1.71 to 11.58, p = 0.002). LAC on cardiac CT is not uncommon and reflects more advanced LA remodeling and a stiff LA. The presence and severity of LAC are associated with a worse clinical outcome after MV surgery. **(Am J Cardiol 2020;128:60−66)**

Left atrial (LA) remodeling and compliance affect clinical presentation and predict cardiovascular outcome, especially in patients with mitral valve (MV) disease.[1−3] Numerous studies have demonstrated a significant association between enlarged LA volume or decreased LA compliance and development of atrial fibrillation, heart failure, stroke, and death.[2−4] Although there are various echocardiographic methods available to evaluate LA size and function, limitations exist, especially in patients who have undergone MV operation due to acoustic shadowing caused by a prosthetic valve and lack of information about LA tissue characteristics.[2,5] Cardiac computed tomography (CT) has high spatial resolution and may offer beneficial information in evaluation of LA. A few case reports have demonstrated massive LA calcium (LAC) that resembles a "porcelain or coconut atrium," which contributes to a decrease in LA compliance and may lead to elevated LA pressure and heart failure.[6,7] In this study, we aimed to investigate (1) the incidence and degree of LAC as determined by cardiac CT in patients who had undergone MV surgery; (2) structural and hemodynamic differences according to degree of LAC; (3) any factors associated with presence of LAC; and (4) clinical significance of LAC in this population.

Methods

We retrospectively searched the database for cardiac CT examinations performed between March 2010 and December 2017. We included 327 patients with MV surgery, MV repair or replacement, as the study population. Patients who underwent surgical ablation or radiofrequency catheter ablation for atrial fibrillation were not included. Demographic data and information on the surgical findings were collected from patient electronic medical records. Cardiac CT was performed by clinician's discretion when prosthetic valvular dysfunction was suspected or coronary evaluation was needed. The Institutional Review Board at Yonsei University College of Medicine approved this retrospective study, and informed consent was waived.

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All CT scans were performed with a dual-source CT scanner (SOMATOM Definition Flash; Siemens Healthcare, Forchheim, Germany) or a 64-slice multidetector CT (Somatom Sensation 64; Siemens Medical Solution, Erlangen, Germany) in electrocardiographically gated data acquisition mode. The CT scans were performed with the triple-phase injection method (70 mL of iopamidol followed by 30 mL of 30% iopamidol blended with saline solution and 20 mL of saline solution at a rate of 5 mL/s). Images were generated from the raw datasets using filtered back projection. Image reconstruction was performed with a medium kernel (I36f or b36f), and the reconstruction slice thickness was 0.75 mm, with 0.5 mm increments between slices. For all patients, 10 transverse data sets were reconstructed for every 10% of the cardiac cycle (0% to 90%), and the reconstructed images were transferred to an image server and analyzed using dedicated 3D software (Aquarius iNtuition, version 4.4.11; TeraRecon, Inc, San Mateo, CA). The software identified calcification with density >130 HU. We classified the degree of LAC into three groups according to extent of calcification in the LA; no LAC (group 1), mild-to-moderate LAC (group 2, <50% of LA circumference), and severe LAC (group 3, >50% of LA circumference; Figure 1). Assessment of a prosthetic MV via CT consisted of an evaluation for presence of paravalvular leakage (PVL) and other abnormalities, such as prosthetic valve obstruction (vegetation, pannus, or thrombus) or limitation of motion of the prosthetic MV. A short-axis view of the MV was used to assess the presence and location of PVL. CT analyses were independently performed by two radiologists blinded to the clinical information, transthoracic echocardiogram (TTE), and transesophageal echocardiogram (TEE) results as well as the CT analysis results of the other reader. When disagreement in CT analysis was observed between the CT readers, the final decision was made through a consensus reading.

All participants underwent a standard 2-dimensional (2D) TTE within 90 days of the CT examination. Conventional 2D and Doppler measurements were obtained in accordance with the recommendations of the American Society of Echocardiography guidelines using commercially available equipment.[8] Comprehensive evaluations were performed to evaluate the prosthetic valve function using both routine and modified views. The calculated pulmonary artery systolic pressure was defined as 4 x (maximum velocity of the tricuspid regurgitant jet)^2 + right atrial pressure. Right atrial pressure was estimated by measuring the diameter of the inferior vena cava and its response to inspiration.[9] Valvular function was evaluated using Doppler measurements, such as peak or mean diastolic pressure gradient for repaired MV or prosthetic MV. A high-velocity, eccentric turbulent jet with its origin beyond the edge of the sewing ring was considered to indicate PVL. A laminar, low-velocity regurgitant jet with its origin within the orifice of the sewing ring was considered to indicate transvalvular leakage. TEE was performed at the clinician's discretion using a Philips iE33 ultrasound system and an X7-2t transesophageal transducer (Philips Medical Systems, Andover, MA).

Patients were followed up after initial echocardiographic evaluation across a median of 40 months for major adverse cardiovascular events (MACE) that included all-cause death, hospitalization for heart failure, and incidence of stroke. The occurrence of any of the aforementioned clinical events was ascertained by review of hospital records and by telephone interview as necessary.

Continuous variables are presented as mean ± SD, and categorical variables are expressed as percentage. The patient groups were compared using Student's *t* test for continuous variables and $\chi 2$ statistics for categorical variables. To identify any potential independent associations between clinical factors and LAC, linear relationships were assessed using univariable linear regression. Variables that displayed statistical significance upon univariable analysis as well as age were entered into a multivariable linear regression model. Kaplan-Meier survival curves were used to plot all clinical events according to time to the first event. Moreover, Cox proportional hazards regression models reporting hazard ratio (HR) and 95% confidence interval (CI) was performed to determine the impact of presence of LAC or severe LAC on the clinical outcome. For multivariable analysis, variables with statistical significance in univariable analysis were included. A value of $p < 0.05$ was considered significant. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS), v. 22.0 (SPSS, Inc., Chicago, IL).

Results

The study population consisted of 327 patients. The mean age was 61 years, and 216 patients (66.1%) were women. The overall incidence of LAC was 24.5% (80 cases). Mild-to-moderate LAC was present in 56 cases (17.1%), whereas severe LAC was seen in 24 cases (7.3%). The baseline clinical and echocardiographic characteristics were compared among the groups (Table 1). There was no significant difference in age, gender, or underlying disease, such as hypertension, diabetes mellitus, and chronic renal failure. The incidence of atrial fibrillation, rheumatic etiology, previous LA appendage ligation, number of previous MV operation, and time interval after initial MV operation were higher in groups 2 and 3 than in group 1. There were no differences in LV chamber size or left ventricular ejection fraction among the three groups. Notably, LA volume index, mean diastolic pressure gradient across the MV, and pulmonary artery systolic pressure were higher in the advanced LAC group. Moreover, incidence of PVL was significantly higher as degree of LAC advanced (Table 1 and Supplement Figure 1). Intriguingly, pulmonary artery systolic pressure significantly increased in groups 2 and 3 compared with group 1 regardless of presence of PVL (Supplement Figure 2).

The factors associated with degree of LAC in patients who underwent MV surgery are demonstrated in Table 2. Rheumatic etiology of MV dysfunction, time interval after initial MV operation, previous LA appendage ligation, number of previous MV operations, and a larger LA volume index were all independently associated with presence of LAC. Atrial fibrillation was associated with presence of LAC in univariate analysis, but its significance was attenuated in multivariate analysis.

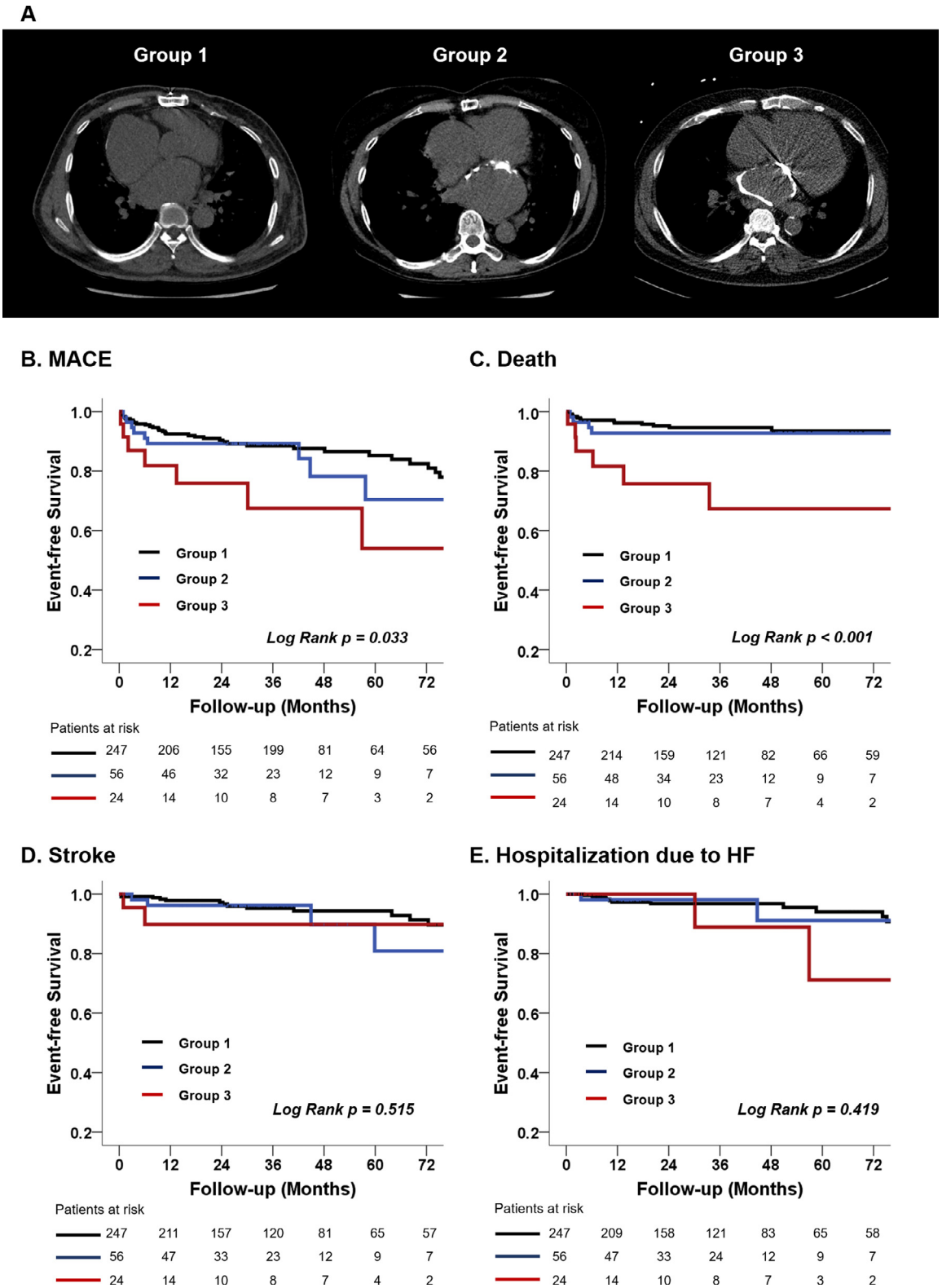

Figure 1. (A) Classification of left atrial calcium. Group 1, no LAC; group 2, mild-to-moderate LAC; and group 3, severe LAC. Event-free survival of major cardiovascular events (B), all-cause mortality (C), stroke (D), and hospitalization due to heart failure (E).

Over the course of the study period, 52 patients (15.9%) experienced the composite study endpoint. Specifically, there were 24 (7.3%) deaths from all causes, 20 (6.1%) cases of stroke, and 16 (4.9%) hospitalizations for heart failure. Figure 1 demonstrates event-free survival rates according to LAC group. Notably, for both MACE and all causes of mortality, patients in the severe LAC group demonstrated a worse event-free survival rate than the other

Table 1
Baseline clinical and echocardiographic characteristics

Variable	Degree of LAC			p Value
	No (n = 247)	Mild to moderate (n = 56)	Severe (n = 24)	
Age, (years)	60.7 ± 12.5	61.4 ± 9.6	65.0 ± 7.3	0.223
Men	87 (35.2%)	15 (27.3%)	8 (33.3%)	0.529
Hypertension	147 (59.5%)	40 (71.4%)	14 (58.3%)	0.241
Diabetes mellitus	55 (22.3%)	12 (21.4%)	7 (29.2%)	0.722
Dyslipidemia	84 (34.0%)	19 (33.9%)	4 (16.7%)	0.219
Chronic renal failure	36 (14.6%)	9 (16.1%)	3 (12.5%)	0.914
Atrial fibrillation	173 (70.0%)	52 (92.9%)*	22 (91.7%)*	<0.001
Warfarin use	240 (97.2%)	56 (100.0%)	24 (100.0%)	0.314
Statin use	110 (44.5%)	24 (42.9%)	6 (25.0%)	0.182
Previous op. type				
MV repair	39 (15.8%)	1 (1.8%)*	0 (0.0%)*	0.003
MV replacement	208 (84.2%)	55 (98.2%)*	24 (100.0%)*	0.003
Bioprosthetic	23 (11.1%)	4 (7.3%)	2 (8.3%)	0.455
Prosthetic	185 (88.9%)	51 (92.7%)	22 (91.7%)	0.455
Reason for MV operation				
Etiology				
Rheumatic	153 (61.9%)	48 (85.7%)*	21 (87.5%)*	<0.001
Degenerative	49 (19.8%)	4 (7.1%)*	0 (0.0%)*	0.005
Infective endocarditis	15 (6.1%)	1 (1.8%)	0 (0.0%)	0.209
Unknown	29 (11.7%)	3 (5.4%)	3 (12.5%)	0.362
MV dysfunction				
Mitral stenosis	126 (51.0%)	33 (58.9%)	14 (58.3%)	0.483
Pure mitral regurgitation	92 (37.2%)	18 (32.1%)	3 (12.5%)*	0.047
Unknown	29 (11.7%)	5 (8.9%)	7 (29.2%)*,†	0.032
Number of MV operation	1.1 ± 0.3	1.4 ± 0.6*	1.4 ± 0.9	<0.001
Time from initial MV operation to cardiac CT (years)	14.2 ± 9.3	21.8 ± 7.8	23.0 ± 8.0	<0.001
Concomitant surgery				
AV replacement	72 (29.3%)	15 (27.8%)	6 (25.0%)	0.895
TV surgery	87 (35.4%)	32 (59.3%)*	11 (45.8%)	0.004
CABG	6 (2.4%)	1 (1.8%)	0 (0.0%)	0.720
LAA ligation	11 (4.5%)	9 (16.1%)	5 (20.8%)	0.001
Concomitant CT finding				
Presence of CAC	80 (35.9%)	16 (32.7%)	10 (41.7%)	0.752
Degree of CAC by Agaston score	130 ± 458	122 ± 370	208 ± 504	0.763
LAA thrombus	21 (8.5%)	2 (3.6%)	3 (12.5%)	0.927
Chamber & hemodynamic parameters				
LVEDD (mm)	50.4 ± 7.3	51.0 ± 6.1	50.1 ± 7.4	0.84
LVESD (mm)	34.7 ± 7.2	34.9 ± 6.5	34.2 ± 6.7	0.911
LVEF (%)	61.5 ± 9.8	61.6 ± 9.6	62.0 ± 12.3	0.843
LA volume index (ml/m^2)	95.0 ± 69.9	131.3 ± 103.6*	172.6 ± 108.2*	<0.001
PASP (mm Hg)	39.8 ± 15.8	49.1 ± 16.4*	61.8 ± 18.2*,†	<0.001
Mitral valve function				
MDPG (mm Hg)	4.8 ± 2.9	5.3 ± 2.3	6.6 ± 3.3*,†	0.01
Paravalvular leak	38 (15.4%)	22 (39.3%)*	16 (66.7%)*,†	<0.001

AV = aortic valve; CABG = coronary artery bypass graft; CAC = coronary artery calcium; LA = left atrium; LAC = left atrial calcium; LVEDD = left ventricular end-diastolic dimension; LVEF = left ventricular ejection fraction; LVESD = left ventricular end-systolic dimension; MDPG = mean diastolic pressure gradient; MV = mitral valve; PASP = pulmonary artery systolic pressure; TV = tricuspid valve.

p <0.05.*Compared with no LAC group.†Compared with mild-to-moderate LAC group.

groups (p = 0.033 and p <0.001, respectively; Figure 1). Univariate Cox regression analysis was performed to identify potential prognostic factors for all causes of mortality (Table 3). Patients with severe LAC had a higher risk of both MACE and all-cause mortality. Other potential risk factors for MACE or all-cause mortality were advanced age, male gender, and chronic renal failure. Presence of atrial fibrillation, larger LA volume index, and any degree of LAC also tended to be correlated with MACE or all-cause mortality. Finally, in multivariate analysis revealed that presence of severe LAC was an independent prognostic factor (HR: 4.44, 95% CI: 1.71 to 11.58, p = 0.002) for all-cause mortality after adjusting for age, male gender, chronic renal failure, atrial fibrillation, and LA volume index. In terms of occurrence of MACE, presence of severe LAC showed marginal significance (HR: 2.16, 95% CI: 0.96 to

Table 2
Factors associated with the presence of LAC

	Univariable		Multivariable	
	HR (95% CI)	p	HR (95% CI)	p
Age	1.02 (0.99-1.04)	0.157		
Male gender	0.84 (0.50 -1.44)	0.530		
Hypertension	1.49 (0.88-2.51)	0.137		
Diabetes mellitus	1.17 (0.66-2.09)	0.595		
Dyslipidemia	0.75 (0.44-1.30)	0.306		
Chronic renal failure	1.21 (0.61-2.38)	0.588		
Atrial fibrillation	3.51 (1.67-7.38)	0.001	1.62 (0.68-3.90)	0.280
Warfarin use	2.14 (0.25-18.00)	0.485		
Statin use	0.80 (0.48-1.33)	0.388		
Rheumatic etiology	3.06 (1.63-5.74)	0.001	2.68 (1.26-5.70)	0.011
Mitral stenosis	1.30 (0.79-2.13)	0.309		
Prosthetic MV	1.08 (0.456-2.54)	0.867		
Time from initial MV operation to cardiac CT (year)	1.08 (1.04-1.12)	<0.001	1.08 (1.04-1.12)	<0.001
LA appendage ligation	4.97 (2.14-11.56)	<0.001	7.39 (2.62-20.85)	<0.001
Numbers of operation	3.08 (1.79-5.29)	<0.001	2.07 (1.04-4.10)	0.037
LA volume index	1.10 (1.07-1.14)	<0.001	1.01 (1.00-1.01)	0.030

CT = computed tomography; LA = left atrium; LAC = left atrial calcium; MV = mitral valve.

4.84, p = 0.062), although age, chronic renal failure, and LA volume index had prognostic significance. Any degree of LAC did not show statistical significance on MACE or all-cause mortality after adjusting age, male gender, chronic renal failure, atrial fibrillation, and LA volume index.

Discussion

The principal findings of the present study were that (1) LAC is not a rare finding in patients who have undergone MV surgery; (2) LAC is associated with rheumatic heart disease, large LA volume index, longer time interval after

Table 3
Predictors for clinical outcomes

	Univariable		Multivariable (Model 1)		Multivariable (Model 2)	
	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p
MACE						
Age	1.06 (1.03-1.09)	<0.001	1.05 (1.02-1.08)	0.001	1.36 (1.02-1.08)	0.001
Male gender	1.76 (1.02-3.05)	0.044	1.58 (1.00-2.78)	0.112	1.61 (0.92-2.83)	0.098
Hypertension	1.50 (0.84-2.67)	0.175				
Diabetes mellitus	0.65 (0.30-1.37)	0.255				
Dyslipidemia	0.74 (0.40-1.36)	0.331				
Chronic renal failure	2.86 (1.59-5.17)	<0.001	2.23 (1.22-4.08)	0.009	2.27 (1.24-4.15)	0.008
Atrial fibrillation	2.04 (0.92-4.52)	0.079	1.35 (0.59-3.09)	0.472	1.38 (0.60-3.14)	0.448
Rheumatic etiology	1.12 (0.61-2.08)	0.712				
Number of operations	1.32 (0.78-2.24)	0.295				
LA volume index	1.00 (1.00-1.01)	<0.001	1.00 (1.00-1.01)	0.037	1.00 (1.00-1.01)	0.033
Presence of LAC	1.62 (0.91-2.90)	0.103	1.36 (0.75-2.48)	0.313	■	■
Presence of severe LAC	2.71 (1.22-6.02)	0.015	■	■	2.16 (0.96-4.84)	0.062
All-cause mortality						
Age	1.07 (1.02-1.12)	0.003	1.07 (1.02-1.12)	0.008	1.06 (1.01-1.12)	0.013
Male gender	1.02 (0.43-2.38)	0.970	1.03 (0.44-2.45)	0.942	1.03 (0.44-2.45)	0.939
Hypertension	0.79 (0.35-1.76)	0.557				
Diabetes mellitus	0.31 (0.07-1.30)	0.110				
Dyslipidemia	0.51 (0.19-1.37)	0.181				
Chronic renal failure	2.40 (0.99-5.78)	0.052	1.78 (0.73-4.39)	0.208	1.77 (0.71-4.43)	0.225
Atrial fibrillation	3.48 (0.82-14.80)	0.091	2.38 (0.54-10.50)	0.253	2.54 (0.57-11.26)	0.221
Rheumatic etiology	0.92 (0.39-2.15)	0.844				
Number of operations	1.52 (0.84-2.76)	0.166				
LA volume index	1.00 (1.00-1.01)	0.096	1.00 (1.00-1.01)	0.866	1.00 (1.00-1.01)	0.943
Presence of LAC	2.21 (0.98-4.98)	0.056	2.02 (0.88-4.65)	0.099	■	■
Presence of severe LAC	5.29 (2.09-13.36)	<0.001	■	■	4.44 (1.71-11.58)	0.002

MACE = major adverse cardiovascular events; LA = left atrium; LAC = left atrial calcium.

initial MV operation, previous LA appendage ligation and the higher number of previous MV surgeries; (3) presence and severity of LAC were associated with elevated pulmonary artery systolic pressure and significantly higher incidence of PVL; and (4) presence of severe LAC was associated with worse clinical outcome.

Since LAC was first described in 1898, several case reports have demonstrated massive LAC. However, no investigation has determined the incidence of LAC in actual clinical populations. A previous study performed during the 1990s reported a rate of 2.2% for extensive LAC in patients who underwent valvular operation.[10] In the study, the patients underwent x-ray for the diagnosis of LAC and the sensitivity of x-ray for detection of LAC could be low. At our institution, we routinely perform cardiac CT in patients with suspected prosthetic valve dysfunction, which can provide detailed visualization of LAC at high resolution. Presence of LAC was revealed in 24.5% of patients who underwent MV surgery, which is higher than previously speculated.

Several theories for the pathophysiology of LAC have been suggested. A previous study reviewed 16 cases of LAC and found that LAC was predominant in female patients and was usually associated with rheumatic heart disease.[11] Rheumatic heart disease along with chronic strain, stress, and inflammation could be a potential cause of LAC.[11] Another study reported eight cases of massive LAC. In all these cases, the patients had rheumatic heart disease and atrial fibrillation and had undergone previous valve operation.[10] In some cases, LAC progressed after the MV surgery despite no LAC present at the time of the surgery.[6] Some investigators have suggested that previous ulceration of the LA wall could be an origin of calcification. Beyond rheumatic ulceration of the LA wall, we suggest that left atriotomy and LA appendage ligation performed during the MV surgery could be another precipitating factor that influences the development of LAC. In our study, there was a clear difference between the three LAC groups in incidence of rheumatic etiology of MV disease and number of previous MV surgeries; we identified statistically significant association between these variables and LAC, which was consistent with the findings of previous studies.[6,10] LAC has rarely been reported in chronic renal disease or dialysis patients in previous studies[12,13]; however, there was no statistically significant association between presence of LAC and chronic renal failure in our study. Further studies will be needed to confirm our findings.

There was significant difference in LA volume index and incidence of atrial fibrillation among our three LAC groups, which may reflect more advanced LA remodeling and irreversible changes in the advanced LAC group. However, there was no significant difference in LA volume index between groups 3 and 2. LA dilatation is thought to be aggravated by MV disease progression. However, when calcium is deposited within the LA wall, it may prevent further dilatation of the LA, decrease its compliance, and increase the LA pressure.[14] Elevated LA pressure can influence the pulmonary veins and right side of the heart and eventually lead to elevation of pulmonary arterial pressure. Such decreased LA compliance may produce a worse clinical outcome when patients are faced with hemodynamic deterioration, such as volume or pressure overload.

Moreover, the incidence of PVL was significantly higher in the advanced LAC group. No previous study has explored the association between LAC and PVL. One study reported that mitral annular calcification, focal calcification of the LA, could be a potential risk factor for PVL.[15] Presence of calcification can inhibit the routine placement of annular valve sutures, prevent proper placement of a prosthetic valve, and contribute to development of PVL, which often leads to reoperation.[16] In our study, patients with advanced LAC had a significantly higher number of previous MV surgeries and a higher incidence of PVL. We can therefore speculate that LAC and decreased LA compliance may form a structural substrate that is susceptible to cracks due to mechanical stresses between the prosthesis and MV annulus. Intriguingly, in patients with PVL, the hemodynamic compromise represented by elevated pulmonary artery systolic pressure was exaggerated, which suggests clinical fragility to volume overload in patients with advanced LAC.

The severe LAC group showed significantly worse clinical outcome that was mainly driven by all-cause mortality. Several other studies have reported that LA compliance modulates hemodynamic burden and subsequently affects clinical outcome.[1,4,17] Although there was statistically no significant difference in stroke or hospitalization rate due to heart failure between the three groups, the cumulative incidence of hospitalization due to heart failure was higher when comparing the severe LAC group with the no or mild-to-moderate LAC group. We suggest that severe LAC patients with decreased LA compliance are easily affected by various hemodynamic deteriorated conditions, such as heart failure caused by volume overload, PVL, infection, or bleeding, which may subsequently lead to increased mortality.

This study has several limitations. First, this study was retrospective in design, which causes inherent potential limitations. Second, there are limited data regarding patients with chronic renal failure since our study only included patients who underwent cardiac CT. Therefore, the effect of chronic renal failure on LAC may have been underestimated. Further studies using noncontrast cardiac CT would be warranted in patients with chronic renal failure. Third, in our study population, rheumatic valve disease was the main etiology of MV dysfunction and showed a significant association with LAC. As the causes of MV disease shows heterogeneity according to race and regions around the world,[18–20] our results may not be generalized to patients in other populations. Fourth, although we described main etiology of MV disease, data regarding the preoperative MV dysfunction, stenosis or pure regurgitation, which would be helpful for understanding the pathophysiology of LAC, could not be defined in 41 patients (12.5%). Fifth, in multivariable analysis, the number of cardiovascular events was low to fully describe the prognostic values of variables, further study with larger population would be needed for the final conclusion. Nevertheless, because there is growing interest in the structural changes of the LA due to recent advances in interventional MV procedures and inflammation associated with atrial fibrillation or radiofrequency catheter ablation, our study may be meaningful for understanding the structural and hemodynamic characteristics related to LAC and the clinical implications of LAC.

In conclusion, the presence of LAC is not uncommon in patients who have undergone MV surgery and may reflect more advanced LA remodeling and the presence of irreversible changes as well as provide valuable prognostic information. Evaluation and quantification of LAC and a solid understanding of its structural, hemodynamic, and clinical implications are important, especially in the era of transcatheter MV interventions.

Author Contribution

Jah Yeon Choi: Investigation, Formal analysis, Writing - Original draft preparation, Resources, **Young Joo Suh:** Resources, Validation, **Young Jin Kim**: Resources, Validation, **Seung-Hyun Lee:** Resources, Investigation, **Sak Lee:** Resources, Investigation, **Geu-Ru Hong**: Investigation, Resources, Conceptualization, **Jong-Won Ha**: Investigation, Resources, **Chi Young Shim**: Conceptualization, Supervision, Reviewing and Editing, Formal analysis, Resources, Methodology

Disclosures

The authors have no conflict of interest to report.

Supplementary materials

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