



# Left Atrial Minimum Volume Index at Cardiac MRI Predicts Adverse Outcomes after Acute Myocardial Infarction

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See also the editorial by Weir-McCall and Hua in this issue.

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**Background:** Left atrial (LA) structural and functional parameters are associated with prognosis after acute myocardial infarction (AMI).

**Purpose:** To explore the prognostic value of LA minimum volume index (LAVI<sub>min</sub>) as measured at cardiac MRI and its incremental predictive value beyond LA functional parameters for predicting major adverse cardiovascular events (MACE) after AMI in a large population.

**Materials and Methods:** This prospective study enrolled patients with AMI who underwent percutaneous coronary intervention and subsequent cardiac MRI between February 2014 and January 2024. MACE included all-cause death, reinfarction, unplanned revascularization, and heart failure hospitalization. Univariable and multivariable Cox regression analyses were used to evaluate the association between LAVI<sub>min</sub> and MACE. Receiver operating characteristic analysis and Kaplan-Meier analysis were used to evaluate the prognostic value of LAVI<sub>min</sub> in participants with AMI.

**Results:** A total of 1191 participants (mean age, 58 years ± 11 [SD]; 1007 male participants) were included. Among them, 183 individuals experienced MACE over a median follow-up of 38 months (IQR, 20–57 months). After adjusting for clinical risk factors and cardiac MRI parameters, a larger LAVI<sub>min</sub> was independently associated with MACE (hazard ratio, 1.06 [95% CI: 1.05, 1.08];  $P < .001$ ). Receiver operating characteristic analysis revealed that LAVI<sub>min</sub> (area under the receiver operating characteristic curve [AUC], 0.74) had better discriminative ability for MACE than LA maximum volume index (LAVI<sub>max</sub>) (AUC, 0.65;  $P < .001$ ) and LA conduit strain (AUC, 0.64;  $P < .001$ ). Traditional risk predictors plus LAVI<sub>min</sub> had greater prognostic value for MACE (C index, 0.75) than traditional risk factors alone (C index, 0.69;  $P < .001$ ) or traditional risk predictors plus LAVI<sub>max</sub> (C index, 0.72;  $P = .03$ ).

**Conclusion:** LAVI<sub>min</sub> was an independent predictor of MACE after AMI, with incremental prognostic value and improved discriminative ability over traditional risk factors including cardiac MRI parameters.

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Left atrial (LA) volume serves as a prognostic marker of acute myocardial infarction (AMI), with an enlarged LA indicating poor prognosis (1,2). The underlying pathophysiologic mechanism may involve left ventricular (LV) diastolic dysfunction after AMI, leading to increased LV filling pressure (3) and subsequent LA pressure overload and increased LA volume through the opening of the mitral valve.

The LA volume index is a critical variable in the diastolic evaluation algorithm outlined in current guidelines (4); this algorithm places particular emphasis on the LA maximum volume, a related variable. However, larger changes may be observed in the LA minimum volume than in the LA maximum volume (5), as the latter is a combined measure of the LA minimum volume and reservoir volume, and the LA reservoir volume may decrease due to progressive LV dysfunction, offsetting the increase in LA minimum volume (6,7). In addition, LA minimum volume but not LA maximum volume is associated with acute LV function following ST elevation myocardial infarction, indicating that the former is a more sensitive marker of sudden changes in LV filling (8). This finding aligns with a study demonstrating that LA minimum volume index (LAVI<sub>min</sub>) at three-dimensional echocardiography is superior to the corresponding LA maximum volume index (LAVI<sub>max</sub>) for predicting cardiac events (9).

Although echocardiography has been widely used to assess LA structure and function (10,11), the excellent spatial resolution,

accuracy, and reproducibility of cardiac MRI make it more appropriate for the detailed evaluation of LA volume as well as LA strain (12).

Although LA volume index has been extensively studied in cardiovascular prognostic studies (11,13), less is known about the prognostic value of LAVI<sub>min</sub> derived from cardiac MRI in patients with AMI and its incremental prognostic value beyond established markers such as LA strain parameters. Therefore, the aim of this study was to assess the overall prognostic value of LAVI<sub>min</sub> in participants with AMI and its incremental predictive value over LA functional parameters from cardiac MRI for predicting major adverse cardiovascular events (MACE) in a large AMI cohort.

## Materials and Methods

### Study Participants

This prospective study included consecutive patients with AMI from two medical centers who underwent percutaneous coronary intervention within 12 hours and cardiac MRI examination within 7 days from symptom onset. Participants were included between February 2014 and January 2024. Part of the study population was included in two previous studies (14,15) exploring the prognostic value of ventricular strain parameters and strain rate in predicting adverse outcomes after AMI. Detailed inclusion and exclusion criteria are provided in Appendix S1. The

## Abbreviations

AMI = acute myocardial infarction, AUC = area under the receiver operating characteristic curve, HR = hazard ratio, LA = left atrium, LAEF = LA ejection fraction,  $LAVI_{max}$  = LA maximum volume index,  $LAVI_{min}$  = LA minimum volume index, LGE = late gadolinium enhancement, LV = left ventricle, LVEF = LV ejection fraction, MACE = major adverse cardiovascular events

## Summary

Left atrial minimum volume index at cardiac MRI had incremental prognostic value beyond traditional risk factors in predicting major adverse cardiovascular events among participants with acute myocardial infarction.

## Key Results

- In this prospective study of 1191 participants with acute myocardial infarction (AMI) and a median follow-up of 38 months, left atrial minimum volume index ( $LAVI_{min}$ ) at cardiac MRI was independently associated with major adverse cardiovascular events (MACE) after AMI (hazard ratio, 1.06;  $P < .001$ ).
- $LAVI_{min}$  had incremental prognostic value over traditional risk factors in predicting MACE (C index, 0.75 vs 0.69;  $P < .001$ ) while being obtainable during routine cardiac MRI examination.

institutional review boards of both centers approved the study protocol, which complied with the principles of the Declaration of Helsinki. All participants provided written informed consent before the cardiac MRI examination, after the percutaneous coronary intervention procedure.

## Cardiac MRI Acquisition

All cardiac MRI examinations were performed within 7 days of the index event using one of two 3.0-T scanners (Magnetom Verio, Siemens Healthineers; Ingenia, Philips) and following standard scanning protocols. Detailed cardiac MRI acquisition and sequence parameters are available in Appendix S1 and Table S1.

## Cardiac MRI Parameter Assessment

Cardiac MRI scans were postprocessed using dedicated software (cvi42; Circle Cardiovascular Imaging). LA minimum and maximum volumes were measured using the biplane area-length method; the mitral annulus and LA roof were delineated on two- and four-chamber views at LV end diastole and end systole, after which the endocardial border of the LA was semiautomatically contoured and manually corrected (Fig 1).  $LAVI_{max}$  and  $LAVI_{min}$  are LA volumes indexed to body surface area. The LA appendage and pulmonary veins were not included in the LA volume. Late gadolinium enhancement (LGE), LA strain, and LV, right atrial, and right ventricular volumes and function were also quantified. Details of postprocessing methods are presented in Appendix S1.

## Clinical Outcomes and Follow-up

Data on clinical follow-up were collected through medical record reviews, clinical assessments, or telephone interviews performed by a physician who was blinded to the cardiac MRI data. Specifically, clinical outcomes were assessed via telephone interviews every 6 months when medical records and clinical assessments were unavailable. The primary end point was MACE, which included all-cause death, reinfarction, unplanned revascularization, and hospitalization for heart failure. For participants with multiple events, only the first event was considered in the primary end point analysis.

## Statistical Analyses

Continuous variables are presented as means  $\pm$  SDs or medians and IQRs and were analyzed using the Student *t* test or the Mann-Whitney *U* test, as appropriate. Categorical variables are reported as frequencies and percentages and were compared using the  $\chi^2$  test or Fisher exact test. The normality of the data was assessed with the Kolmogorov-Smirnov test.

Time-dependent receiver operating characteristic (ROC) analysis was used to quantify the ability of the variables to discriminate outcomes at each time point, and comparisons were performed with the Uno C statistic. The risk of MACE was depicted using Kaplan-Meier curves stratified by  $LAVI_{min}$  and  $LAVI_{max}$  tertiles. Kaplan-Meier survival analyses were also performed with  $LAVI_{min}$  and  $LAVI_{max}$  as dichotomous variables: The optimal cutoff values for  $LAVI_{min}$  and  $LAVI_{max}$  were determined using simple ROC curve analysis and the Youden index. The area under the ROC curve (AUC) values for  $LAVI_{min}$ ,  $LAVI_{max}$ , and LA ejection fraction (LAEF) were calculated to evaluate the prognostic accuracy of these measures in predicting outcomes, and the simple ROC curves were compared using the DeLong test in the total AMI population.

Univariable and multivariable Cox regression analyses were performed to evaluate the prognostic value of  $LAVI_{min}$  for predicting MACE in the AMI cohort. Variables with  $P < .10$  in the univariable analysis were included in the multivariable Cox regression model. Standardized independent variables (using *Z* score standardization) were used in additional univariable and multivariable Cox regression analyses to ensure that the variables were compared on the same scale and to eliminate any confounding arising from their original units. Model 1 included age, diabetes mellitus, and Killip class. Model 2 included model 1 variables plus LV end-diastolic volume index, LV end-systolic volume index, LV ejection fraction (LVEF), LGE, microvascular obstruction, intramyocardial hemorrhage, LV aneurysm, and LV thrombus. Model 3 included model 2 variables plus  $LAVI_{max}$ . Model 4 included model 2 variables plus  $LAVI_{min}$ . Model 5 included model 2 variables plus  $LAVI_{max}$  and  $LAVI_{min}$ . The comparisons of C indexes between models were adjusted using Bonferroni correction. The incremental prognostic value of  $LAVI_{min}$ ,  $LAVI_{max}$ , and other parameters was assessed by calculating changes in the global  $\chi^2$  value of the likelihood ratio test with nested regression models and by calculating the C index. Inter- and intraobserver variability in the measurement of LA structural and functional parameters was assessed using the intraclass correlation coefficient.

Statistical analyses were performed by two authors (J.W. and J.X.) using SPSS (version 26.0; IBM), R (version 4.3.2; R Foundation for Statistical Computing), and GraphPad Prism (version 9.0; GraphPad). Two-tailed  $P < .05$  was considered to indicate a statistically significant difference or correlation.

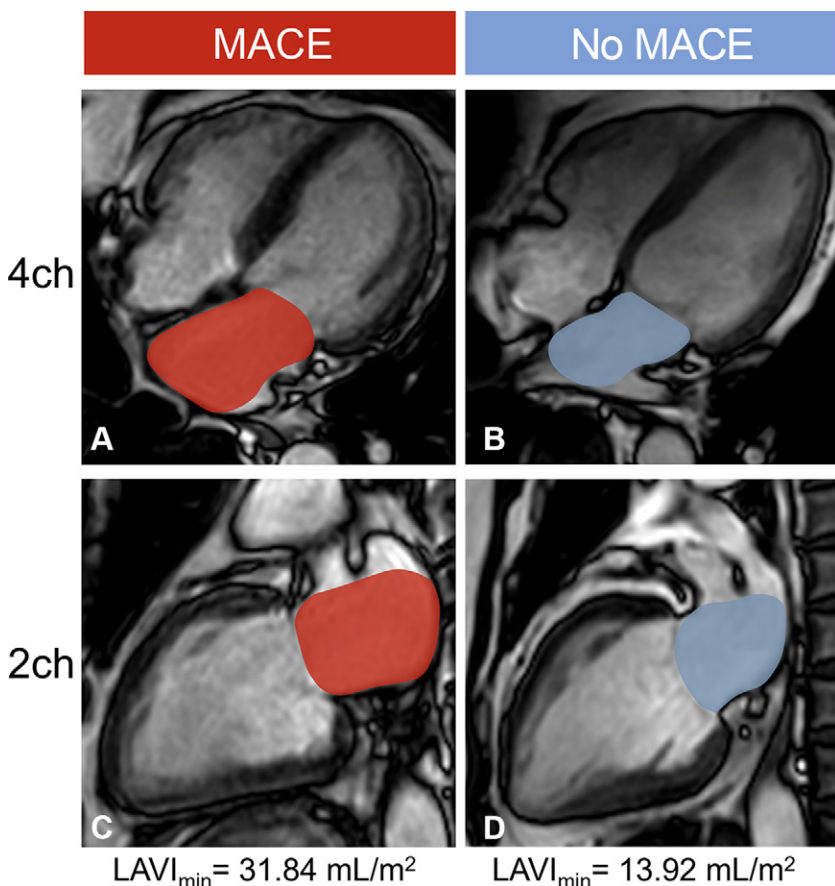
## Results

### Study Population

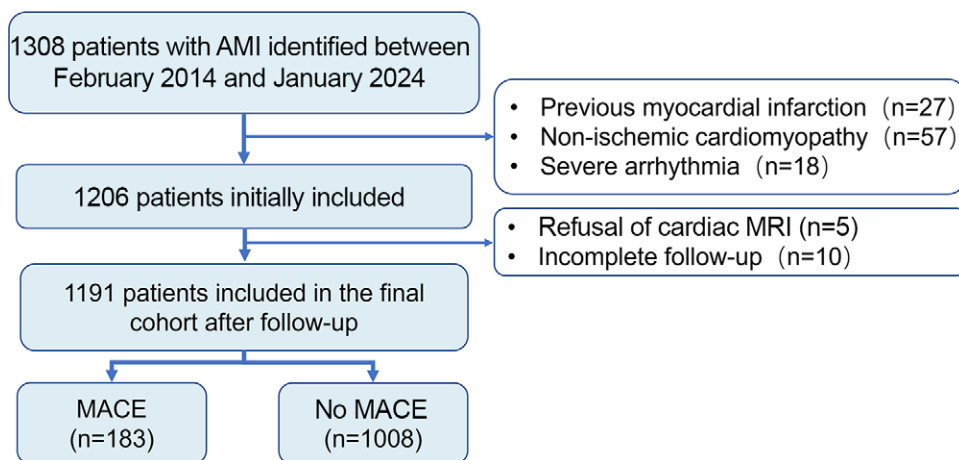
Among the 1308 identified patients, the following were excluded from the study: patients with previous myocardial infarction ( $n = 27$ ), patients with nonischemic cardiomyopathy ( $n = 57$ ), patients with severe arrhythmia ( $n = 18$ ), patients who refused cardiac MRI ( $n = 5$ ), and patients with incomplete follow-up ( $n = 10$ ) (Fig 2). Thus, 1191 participants (mean age, 58 years  $\pm$  11 [SD]; 1007 [84.6%] men) who underwent cardiac MRI after percutaneous

coronary intervention (median time from intervention to imaging, 5 days [IQR, 3–6 days]) were included (Table 1). During a median follow-up of 38 months (IQR, 20–57 months), 183 of the 1191 participants (15.4%) experienced MACE (median time

to MACE, 14 months [IQR, 10–23 months]), and 1008 (84.6%) participants did not experience MACE (median follow-up time, 42 months [IQR, 20–57 months]). The observed MACE included 67 reinfarctions, 43 unplanned revascularizations, 33 hospitalizations for heart failure, and 40 all-cause deaths. Compared with participants without MACE, those with MACE were generally older (mean age, 60 years vs 58 years;  $P = .007$ ); had a greater prevalence of diabetes mellitus, dyslipidemia, and smoking; had higher N-terminal pro-B-type natriuretic peptide level; had a higher likelihood of Killip class greater than 1 at admission; and were more likely to be taking an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker or a  $\beta$ -blocker (Table 1).



**Figure 1:** Cardiac MRI cine images in the (A, B) four-chamber (4ch) and (C, D) two-chamber (2ch) views illustrate contouring (red and blue areas) for measurement of left atrial (LA) minimum volume index ( $LAVI_{min}$ ) at end diastole in two representative participants with acute myocardial infarction (AMI). The LA appendage and pulmonary veins were not considered part of the LA volume. (A, C) Images in a 76-year-old man with a history of coronary artery disease who underwent contrast-enhanced cardiac MRI 4 days after AMI and experienced a major adverse cardiovascular event (MACE), in this case death, during follow-up.  $LAVI_{min}$  was 31.84 mL/m<sup>2</sup> in this participant. (B, D) Images in a 38-year-old man with a history of angina who underwent contrast-enhanced cardiac MRI 4 days after AMI and did not experience MACE during follow-up.  $LAVI_{min}$  was 13.92 mL/m<sup>2</sup> in this participant.



**Figure 2:** Study flowchart. The median follow-up time for major adverse cardiovascular events (MACE) was 38 months. AMI = acute myocardial infarction.

### Cardiac MRI Parameters Related to MACE

Among the included participants, those with MACE had greater  $LAVI_{min}$  (median, 23.33 mL/m<sup>2</sup> [IQR, 20.18–27.24 mL/m<sup>2</sup>] vs 16.94 mL/m<sup>2</sup> [IQR, 12.60–22.71 mL/m<sup>2</sup>];  $P < .001$ ) and  $LAVI_{max}$  (median, 37.18 mL/m<sup>2</sup> [IQR, 32.08–46.52 mL/m<sup>2</sup>] vs 32.73 mL/m<sup>2</sup> [IQR, 25.51–41.04 mL/m<sup>2</sup>];  $P < .001$ ) than those without MACE (Table 2). Participants with MACE presented with reduced LA reservoir strain (median, 20.80% vs 23.00%;  $P < .001$ ), booster strain (median, 11.30% vs 12.20%;  $P = .001$ ), and conduit strain (median, 8.40% vs 10.80%;  $P < .001$ ) compared with participants without MACE. Additionally, participants with MACE had lower LVEF (median, 44.00% vs 48.00%;  $P < .001$ ), greater extent of LGE (median, 32% vs 24%;  $P < .001$ ), greater right ventricular end-systolic volume index, lower right ventricular ejection fraction, and higher LV global longitudinal strain ( $P = .01$ ) than participants without MACE (Table 2). Patients with MACE were more likely to have microvascular obstruction (59.6% vs 45.6%;  $P = .001$ ), intramyocardial hemorrhage (51.4% vs 36.4%;  $P < .001$ ), LV aneurysm (28.4% vs 16.8%;  $P < .001$ ), and LV thrombosis (16.9% vs 8.6%;  $P = .001$ ) than participants without MACE.

### Association of LA Volume Index with Cardiac MRI Parameters

$LAVI_{min}$  was strongly correlated with  $LAVI_{max}$  ( $r = 0.78$ ;  $P < .001$ ). There was also a weak to moderate association between  $LAVI_{min}$  and LA strain parameters and LVEF, with  $r$  values ranging from  $-0.41$  to  $-0.27$  (all  $P < .001$ ) (Table S6).  $LAVI_{max}$  also showed a weak to moderate association

**Table 1: Characteristics of Included Participants**

Characteristic	All Participants (N = 1191)	Participants with MACE (n = 183)	Participants without MACE (n = 1008)	P Value
Mean age (y)	58.19 ± 11.28	60.25 ± 10.51	57.82 ± 11.38	.007
Median age (y)	59 (51–67)	61 (54–67)	59 (50–66)	.007
Sex				.47
Male	1007 (84.6)	158 (86.3)	849 (84.2)	
Female	184 (15.4)	25 (13.7)	159 (15.8)	
Body mass index*	25.11 (23.03–24.46)	24.96 (22.90–27.30)	25.20 (23.05–27.50)	.62
Risk factors				
Diastolic blood pressure (mm Hg)	77.09 ± 11.88	75.28 ± 11.02	77.42 ± 12.01	.03
Systolic blood pressure (mm Hg)	125.40 ± 18.55	125.37 ± 19.32	125.41 ± 18.42	.98
Diabetes mellitus	468 (39.3)	94 (51.4)	372 (36.9)	<.001
Dyslipidemia	516 (43.3)	83 (45.4)	433 (43.0)	<.001
Smoking	506 (42.5)	94 (51.4)	412 (40.9)	.008
Killip class > 1	287 (24.1)	66 (36.1)	221 (21.9)	<.001
Laboratory indexes				
Peak troponin I (ng/mL)	15.90 (7.09–26.32)	16.00 (10.02–26.68)	15.77 (6.27–26.22)	.14
NT-proBNP (ng/mL)	1.08 (0.70–1.56)	1.10 (0.69–1.70)	1.08 (0.70–1.54)	.33
Peak CRP (mg/L)	6.30 (1.29–27.77)	7.98 (1.19–36.21)	5.99 (1.31–26.55)	.28
Total cholesterol (mg/dL)	4.73 ± 1.94	4.89 ± 1.20	4.71 ± 2.04	.24
Triglycerides (mg/dL)	1.54 (1.04–2.31)	1.56 (1.04–2.32)	1.53 (1.05–2.29)	.93
Low-density lipoprotein (mmol/L)	2.85 ± 0.99	2.72 ± 0.90	2.87 ± 1.00	.05
High-density lipoprotein (mmol/L)	1.01 (0.85–1.21)	0.98 (0.84–1.24)	1.01 (0.85–1.20)	.86
Serum creatinine (μmol/L)	76 (67–86)	76 (66–86)	76 (69–87)	.12
eGFR (mL/min/1.73 m <sup>2</sup> )	90 (76–101)	90 (76–102)	90 (76–100)	.90
Angiographic variables				
Culprit vessel				.30
Left anterior descending	698 (58.6)	110 (60.1)	588 (58.3)	
Left circumflex	127 (10.7)	24 (13.1)	103 (10.2)	
Right coronary artery	366 (30.7)	49 (26.8)	317 (31.4)	
No. of diseased vessels > 1	511 (42.9)	74 (40.4)	437 (43.4)	.46
Pre-PCI TIMI flow grade 0	852 (71.5)	135 (73.8)	717 (71.1)	.47
Post-PCI TIMI flow grade 3	1140 (95.7)	172 (94.0)	968 (96.0)	.21
Time from symptoms to balloon (min)	163.00 ± 74.63	166.27 ± 78.91	162.41 ± 73.86	.52
Door-to-balloon time (min)	23 (13–46)	22 (13–52)	23 (13–46)	.40
Time from PCI to CMR (d)	5 (3–6)	5 (4–6)	5 (3–6)	.03
Time from AMI to CMR (d)	5 (4–6)	5 (4–6)	5 (4–6)	.03
Medications				
Aspirin	832 (69.9)	130 (71.0)	702 (69.6)	.71
Statin	834 (70.0)	133 (72.7)	701 (69.5)	.40
ACE-I or ARB	611 (51.3)	116 (63.4)	495 (49.1)	<.001
β-blocker	738 (62.0)	130 (71.0)	608 (60.3)	.006

Note.—Continuous data are reported as means ± SDs or medians with IQRs in parentheses and were compared using the Student *t* test or Mann-Whitney *U* test. Categorical data are reported as numbers of participants with percentages in parentheses and were compared using the  $\chi^2$  test. *P* values were calculated for comparison of participants with and without MACE. ACE-I = angiotensin-converting enzyme inhibitor, AMI = acute myocardial infarction, ARB = angiotensin receptor blocker, CMR = cardiac MRI, CRP = C-reactive protein, eGFR = estimated glomerular filtration rate, MACE = major adverse cardiovascular event, NT-proBNP = N-terminal pro-B-type natriuretic peptide, PCI = percutaneous coronary intervention, TIMI = Thrombolysis in Myocardial Infarction.

\* Calculated as weight in kilograms divided by height in meters squared.

with LA strain parameters, with *r* values ranging from  $-0.26$  to  $-0.13$  (all *P* < .001).  $LAVI_{\min}$  and  $LAVI_{\max}$  were negatively correlated with LAEF (*r* =  $-0.63$  and  $-0.40$ , respectively; both *P* < .001) and positively associated with LGE, LV end-diastolic volume index, and LV end-systolic volume index, with *r* values ranging from 0.09 to 0.40 (all *P* < .001) (Table S6).

In addition,  $LAVI_{\min}$  and  $LAVI_{\max}$  increased as the time from AMI to cardiac MRI increased (Fig S2). However, the correlations

between  $LAVI_{\min}$  and  $LAVI_{\max}$  and the time from AMI to cardiac MRI were weak (*r* = 0.11 and 0.07, respectively; *P* < .001 and *P* = .02, respectively).

#### The Prognostic Value of $LAVI_{\min}$ for Predicting MACE

In univariable Cox regression analysis,  $LAVI_{\min}$  was associated with MACE (hazard ratio [HR], 1.06 [95% CI: 1.05, 1.07]; *P* < .001) (Table 3). After adjusting for significant factors

**Table 2: Cardiac MRI Parameters in Included Participants**

Parameter	All Participants (N = 1191)	Participants with MACE (n = 183)	Participants without MACE (n = 1008)	P Value
<b>Left atrium</b>				
LAVI <sub>min</sub> (mL/m <sup>2</sup> )	18.10 (13.37–24.45)	23.33 (20.18–27.24)	16.94 (12.60–22.71)	<.001
LAVI <sub>max</sub> (mL/m <sup>2</sup> )	33.70 (26.52–41.89)	37.18 (32.08–46.52)	32.73 (25.51–41.04)	<.001
LA reservoir strain (%)	22.60 (17.00–28.80)	20.80 (14.10–24.40)	23.00 (17.40–29.30)	<.001
LA booster strain (%)	12.10 (9.00–15.20)	11.30 (8.20–13.70)	12.20 (9.10–15.50)	.001
LA conduit strain (%)	10.50 (6.70–14.50)	8.40 (5.20–11.30)	10.80 (7.10–14.80)	<.001
LAEF (%)	53.64 ± 11.63	51.30 ± 12.72	54.06 ± 11.37	.003
<b>Left ventricle</b>				
LV EDVI (mL/m <sup>2</sup> )	75.50 (63.30–88.90)	79.90 (67.03–97.87)	74.50 (62.48–87.70)	<.001
LV ESVI (mL/m <sup>2</sup> )	37.88 (28.34–51.00)	41.80 (31.80–58.04)	37.09 (28.06–49.44)	<.001
LVEF (%)	47.16 (38.00–55.31)	44.00 (33.73–52.00)	48.00 (39.00–55.51)	<.001
LV GLS (%)	−7.84 (−10.60 to −5.45)	−7.25 (−9.58 to −4.39)	−8.01 (−10.70 to −5.58)	.01
LV SVI (mL/m <sup>2</sup> )	35.39 ± 10.06	34.64 ± 10.60	35.53 ± 9.96	.27
LV mass index (g/m <sup>2</sup> )	60.80 (52.80–70.60)	65.94 (54.90–74.91)	60.20 (52.30–68.88)	<.001
LGE (% of LV)	26 (15–36)	32 (22–40)	24 (15–36)	<.001
IMH	461 (38.7)	94 (51.4)	367 (36.4)	<.001
MVO	569 (47.8)	109 (59.6)	460 (45.6)	.001
LV aneurysm	221 (18.6)	52 (28.4)	169 (16.8)	<.001
LV thrombosis	118 (9.9)	31 (16.9)	87 (8.6)	.001
<b>Right ventricle</b>				
RV EDVI (mL/m <sup>2</sup> )	71.30 (60.53–84.24)	74.20 (60.90–90.27)	70.99 (60.37–83.34)	.11
RV ESVI (mL/m <sup>2</sup> )	32.47 (25.50–43.26)	34.93 (26.40–49.68)	32.14 (25.45–42.37)	.01
RVEF (%)	53.19 (44.97–59.44)	51.55 (40.19–58.07)	53.61 (45.91–59.59)	.001
RV SVI (mL/m <sup>2</sup> )	37.02 ± 9.76	36.18 ± 10.76	37.17 ± 9.57	.21
<b>Right atrium</b>				
RA EDVI (mL/m <sup>2</sup> )	14.03 (9.79–18.63)	14.40 (10.15–19.30)	13.99 (9.61–18.45)	.27
RA ESVI (mL/m <sup>2</sup> )	24.71 (18.38–31.03)	25.74 (18.89–30.65)	24.37 (18.17–31.13)	.25
RAEF (%)	53.00 (44.76–61.73)	52.00 (43.76–60.00)	53.00 (44.81–63.00)	.20

Note.—Continuous data are reported as means ± SDs or medians with IQRs in parentheses. Categorical data are reported as numbers of participants with percentages in parentheses. EDVI = end-diastolic volume index, ESVI = end-systolic volume index, GLS = global longitudinal strain, IMH = intramyocardial hemorrhage, LA = left atrium, LAEF = LA ejection fraction, LAVI<sub>max</sub> = LA maximum volume index, LAVI<sub>min</sub> = LA minimum volume index, LGE = late gadolinium enhancement, LV = left ventricle, LVEF = LV ejection fraction, MACE = major adverse cardiovascular event, MVO = microvascular obstruction, RA = right atrium, RAEF = RA ejection fraction, RV = right ventricle, RVEF = RV ejection fraction, SVI = stroke volume index.

identified with the univariable Cox analysis, multivariable analysis revealed that LAVI<sub>min</sub> remained an independent predictor of MACE (adjusted HR per 1-mL/m<sup>2</sup> increase, 1.06 [95% CI: 1.05, 1.08], *P* < .001; adjusted HR per 1-SD increase, 1.79 [95% CI: 1.56, 2.06], *P* < .001) (Tables 4, S2). LAVI<sub>max</sub> was also found to be independently associated with MACE in the multivariable analysis (adjusted HR per 1-mL/m<sup>2</sup> increase, 1.03 [95% CI: 1.02, 1.04], *P* < .001; adjusted HR per 1-SD increase, 1.48 [95% CI: 1.29, 1.70], *P* < .001) (Tables 4, S2).

Kaplan-Meier survival analysis demonstrated that participants in the highest LAVI<sub>min</sub> tertile (>21.47 mL/m<sup>2</sup>) and the middle tertile had an increased risk of MACE compared with those in the lowest tertile (≤14.87 mL/m<sup>2</sup>) (log-rank test, *P* < .001 for both) (Fig 3A). Likewise, participants in the highest LAVI<sub>max</sub> tertile (>38.53 mL/m<sup>2</sup>) and the middle tertile had an increased risk of MACE compared with those in the lowest tertile (≤29.00 mL/m<sup>2</sup>) (log-rank test, *P* < .001 for both) (Fig 3B). Participants with AMI could also be effectively stratified using the optimal LAVI<sub>min</sub> cutoff of 18.74 mL/m<sup>2</sup> (HR, 7.00

[95% CI: 4.70, 10.41]; *P* < .001) (Fig 3C) or the optimal LAVI<sub>max</sub> cutoff of 33.72 mL/m<sup>2</sup> (HR, 2.61 [95% CI: 1.90, 3.61]; *P* < .001) (Fig 3D).

In terms of each specific MACE outcome, LAVI<sub>min</sub> independently predicted reinfarction (adjusted HR, 1.04 [95% CI: 1.02, 1.06]; *P* < .001), heart failure (adjusted HR, 1.04 [95% CI: 1.02, 1.07]; *P* < .001), unplanned revascularization (adjusted HR, 1.04 [95% CI: 1.01, 1.06]; *P* = .002), and all-cause death (adjusted HR, 1.05 [95% CI: 1.03, 1.07]; *P* < .001) after adjustment for age, diabetes mellitus, Killip class, LVEF, and LGE (Table S4). Participants with LAVI<sub>min</sub> greater than 18.74 mL/m<sup>2</sup> showed an increased risk of each MACE outcome compared with those with LAVI<sub>min</sub> of 18.74 mL/m<sup>2</sup> or lower (Fig 4).

#### Improved MACE Risk Prediction with LAVI<sub>min</sub>

The results of the time-dependent receiver operating characteristic analysis demonstrated that LAVI<sub>min</sub> exhibited a greater AUC (0.77) for predicting MACE than did LAVI<sub>max</sub> or the other LA functional parameters (Fig 5). Additionally,

LAVI<sub>min</sub> demonstrated higher C index (0.73) than LAVI<sub>max</sub>, LAEF, and LA strain parameters (all *P* < .001) (Table S3). When LAVI<sub>min</sub> was added to model 2 (age, diabetes mellitus,

Killip class, LV end-diastolic volume index, LV end-systolic volume index, LVEF, LGE, microvascular obstruction, intramyocardial hemorrhage, LV aneurysm, and LV thrombus),

the resulting model (model 4) demonstrated better prognostic value than model 2 variables plus LAVI<sub>max</sub> (model 3) (C index, 0.75 vs 0.72; *P* = .03) or model 2 plus LA strain or LAEF parameters (Table 5). Among the nested models, model 4, which included LAVI<sub>min</sub>, provided incremental prognostic value for predicting MACE beyond traditional risk factors (model 2) (C index, 0.75 vs 0.69;  $\chi^2$  value of likelihood ratio test, 92.05 vs 69.93; *P* < .001). Adding both LAVI<sub>min</sub> and LAVI<sub>max</sub> to model 2 (model 5) significantly improved the prognostic value compared with model 2 plus LAVI<sub>max</sub> (model 3) (C index, 0.75 vs 0.72;  $\chi^2$  value of likelihood ratio test, 92.24 vs 90.44; *P* = .003) but not compared with model 2 plus LAVI<sub>min</sub> (model 4) (C index, 0.75 vs 0.75;  $\chi^2$  value of likelihood ratio test, 92.24 vs 92.05; *P* = .21) (Fig 6).

LAVI<sub>min</sub> had higher discriminative ability for MACE (AUC, 0.74) than LAVI<sub>max</sub>, LA strain parameters, or LAEF (all *P* < .001) (Fig S3). In terms of individual MACE outcomes, LAVI<sub>min</sub> had discriminative ability for predicting reinfarction (AUC, 0.57; *P* = .04) and heart failure (AUC, 0.69; *P* < .001) (Table S5).

**Table 3: Univariable Cox Regression Analysis for Prediction of Major Adverse Cardiovascular Events in Participants with AMI**

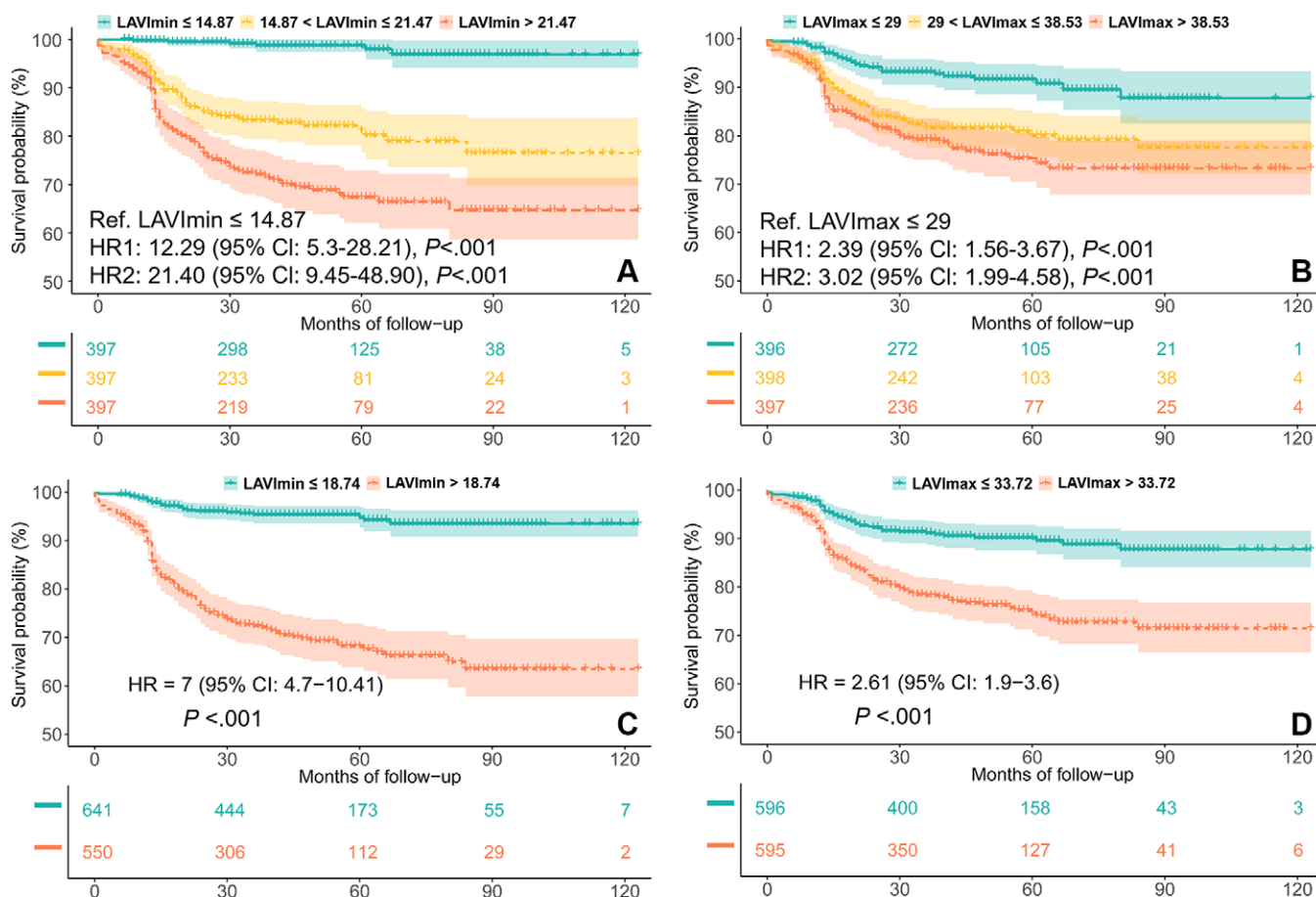
Variable	Coefficient	Hazard Ratio	<i>P</i> Value
Age	0.02 (0.004, 0.03)	1.02 (1.01, 1.03)	.008
Diabetes mellitus	0.58 (0.29, 0.87)	1.79 (1.34, 2.40)	<.001
Killip class	0.46 (0.24, 0.67)	1.59 (1.28, 1.97)	<.001
Dyslipidemia	0.12 (-0.17, 0.41)	1.13 (0.84, 1.51)	.43
Smoking	0.37 (0.08, 0.66)	1.44 (1.08, 1.93)	.01
Time from AMI to CMR	0.11 (0.03, 0.20)	1.12 (1.03, 1.23)	.01
RV ESVI	0.01 (0.004, 0.02)	1.01 (1.005, 1.02)	<.001
RVEF	-0.02 (-0.04, -0.01)	0.98 (0.97, 0.99)	<.001
LA reservoir strain	-0.06 (-0.09, -0.04)	0.95 (0.93, 0.96)	<.001
LA booster strain	-0.06 (-0.09, -0.03)	0.94 (0.91, 0.97)	<.001
LA conduit strain	-0.09 (-0.12, -0.06)	0.91 (0.89, 0.94)	<.001
LAEF	-0.02 (-0.03, -0.009)	0.98 (0.97, 0.99)	.001
LV EDVI	0.01 (0.006, 0.01)	1.01 (1.006, 1.01)	<.001
LV ESVI	0.01 (0.006, 0.01)	1.01 (1.005, 1.01)	<.001
LV mass index	0.02 (0.007, 0.02)	1.02 (1.01, 1.02)	<.001
LVEF	-0.02 (-0.03, -0.01)	0.98 (0.97, 0.99)	<.001
LGE	0.01 (0.005, 0.02)	1.01 (1.006, 1.02)	.003
MVO	0.53 (0.23, 0.82)	1.69 (1.26, 2.28)	<.001
IMH	0.53 (0.24, 0.82)	1.70 (1.28, 2.28)	<.001
LV thrombus	0.75 (0.37, 1.14)	2.12 (1.44, 3.12)	<.001
LV aneurysm	0.69 (0.36, 1.01)	1.98 (1.44, 2.74)	<.001
LAVI <sub>min</sub>	0.06 (0.05, 0.06)	1.06 (1.05, 1.07)	<.001
LAVI <sub>max</sub>	0.03 (0.02, 0.04)	1.02 (1.01, 1.03)	<.001

Note.—Data in parentheses are 95% CIs. AMI = acute myocardial infarction, CMR = cardiac MRI, EDVI = end-diastolic volume index, ESVI = end-systolic volume index, IMH = intramyocardial hemorrhage, LA = left atrium, LAEF = LA ejection fraction, LAVI<sub>max</sub> = LA maximum volume index, LAVI<sub>min</sub> = LA minimum volume index, LGE = late gadolinium enhancement, LV = left ventricle, LVEF = LV ejection fraction, MVO = microvascular obstruction, RV = right ventricle, RVEF = RV ejection fraction.

**Table 4: Multivariable Cox Regression Analysis for Prediction of Major Adverse Cardiovascular Events in Participants with Acute Myocardial Infarction**

Variable	Multivariable Analysis with LAVI <sub>min</sub>			Multivariable Analysis with LAVI <sub>max</sub>		
	Coefficient	Hazard Ratio	<i>P</i> Value	Coefficient	Hazard Ratio	<i>P</i> Value
Age						
Diabetes mellitus	0.47 (0.17, 0.76)	1.61 (1.19, 2.16)	.002	0.49 (0.19, 0.79)	1.63 (1.21, 2.19)	.001
Killip class	0.33 (0.09, 0.57)	1.40 (1.11, 1.75)	.006	0.32 (0.09, 0.55)	1.38 (1.10, 1.73)	.008
LA reservoir strain	-0.07 (-0.10, -0.04)	0.93 (0.90, 0.97)	<.001	-0.07 (-0.10, -0.04)	0.93 (0.90, 0.96)	<.001
LA booster strain	0.07 (0.02, 0.12)	1.08 (1.02, 1.13)	.007	0.07 (0.03, 0.11)	1.08 (1.02, 1.14)	.007
LA conduit strain	-0.07 (-0.10, -0.04)	0.93 (0.90, 0.97)	<.001	-0.07 (-0.10, -0.04)	0.93 (0.90, 0.96)	<.001
LAEF	0.03 (0.01, 0.05)	1.04 (1.02, 1.05)	<.001	0.02 (0.004, 0.04)	1.02 (1.01, 1.04)	.004
LV EDVI	0.02 (0.002, 0.04)	1.02 (1.01, 1.03)	.01	0.02 (0.0004, 0.04)	1.02 (1.01, 1.04)	.014
LV ESVI	-0.03 (-0.05, -0.006)	0.97 (0.95, 0.99)	.02	-0.03 (-0.06, -0.005)	0.97 (0.95, 1.00)	.04
LVEF	...	...	...	-0.02 (-0.04, -0.002)	0.98 (0.96, 1.00)	.049
IMH	0.37 (0.07, 0.67)	1.45 (1.07, 1.95)	.02	0.44 (0.14, 0.74)	1.54 (1.15, 2.08)	.004
LV thrombus	0.40 (-0.01, 0.81)	1.49 (0.99, 2.26)	.04	0.49 (0.33, 0.65)	1.63 (1.08, 2.44)	.02
LAVI <sub>min</sub>	0.06 (0.05, 0.07)	1.06 (1.05, 1.08)	<.001	...	...	...
LAVI <sub>max</sub>	...	...	...	0.03 (0.02, 0.04)	1.03 (1.02, 1.04)	<.001

Note.—Data in parentheses are 95% CIs. EDVI = end-diastolic volume index, ESVI = end-systolic volume index, IMH = intramyocardial hemorrhage, LA = left atrium, LAEF = LA ejection fraction, LAVI<sub>max</sub> = LA maximum volume index, LAVI<sub>min</sub> = LA minimum volume index, LV = left ventricle, LVEF = LV ejection fraction.



**Figure 3:** Kaplan-Meier survival curves illustrating the incidence of major adverse cardiovascular events (MACE) stratified by tertiles and cutoff values of (A, C) left atrial minimum volume index (LAVI<sub>min</sub>) and (B, D) LA maximum volume index (LAVI<sub>max</sub>). (A) Participants in the highest tertile (>21.47 mL/m<sup>2</sup>) and middle tertile of LAVI<sub>min</sub> exhibited a lower MACE-free survival probability than those in the lowest tertile. (B) Participants in the highest tertile (>38.53 mL/m<sup>2</sup>) and middle tertile of LAVI<sub>max</sub> exhibited a lower MACE-free survival probability than those in the lowest tertile. Hazard ratio (HR) 1 is for the comparison of the lowest and middle tertiles; HR2 is for the comparison of the lowest and highest tertiles. (C, D) The risk of MACE was significantly higher in individuals with higher (C) LAVI<sub>min</sub> (>18.74 mL/m<sup>2</sup>) and (D) LAVI<sub>max</sub> (>33.72 mL/m<sup>2</sup>) using the optimal cutoff based on the Youden index.

**Discussion**

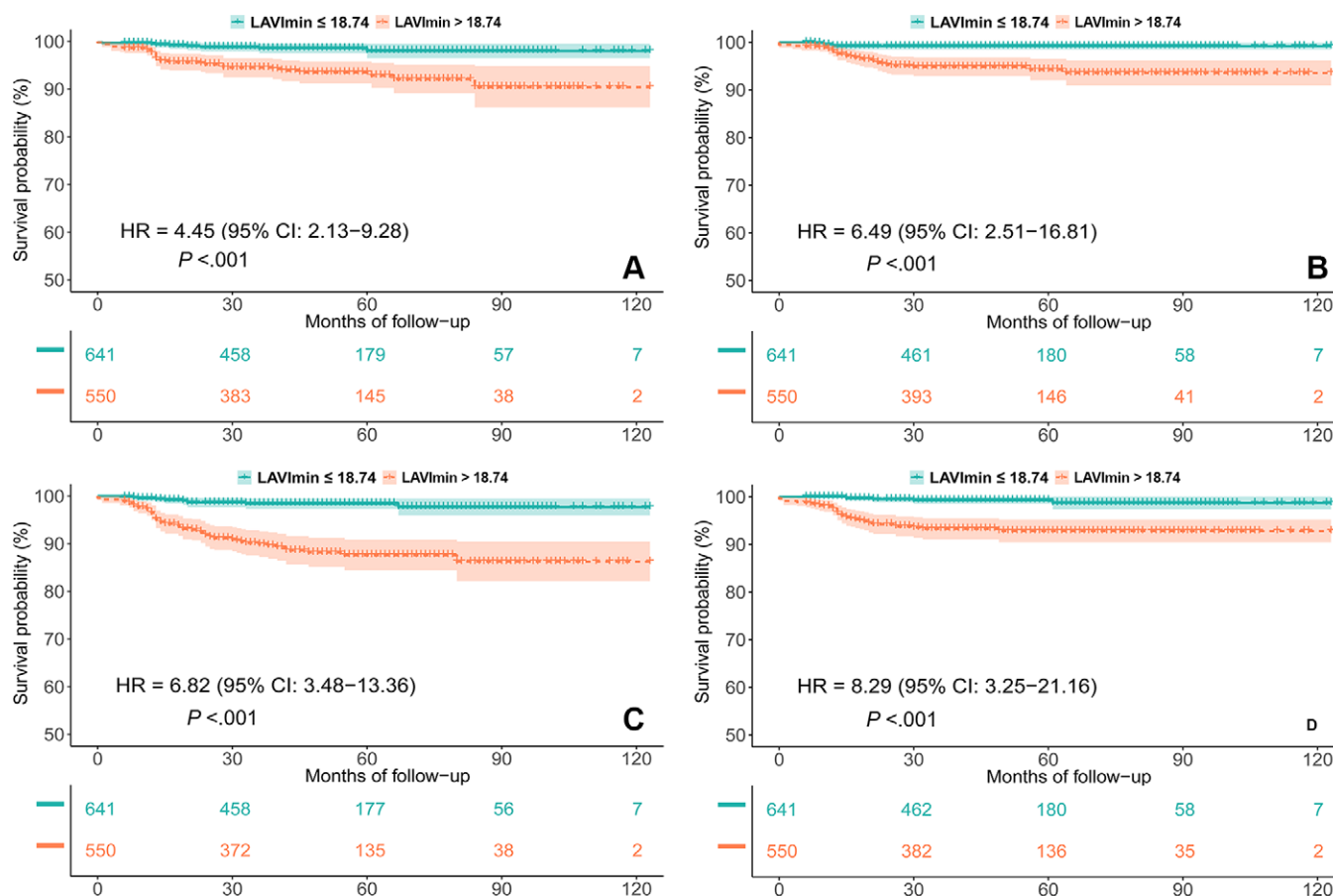
In this study, the prognostic value of the left atrial (LA) minimum volume index (LAVI<sub>min</sub>) for predicting major adverse cardiovascular events (MACE) was studied in a large cohort of participants with acute myocardial infarction. Individuals with LAVI<sub>min</sub> greater than 18.74 mL/m<sup>2</sup> or LA maximum volume index (LAVI<sub>max</sub>) greater than 33.72 mL/m<sup>2</sup> had a greater risk of MACE than participants with LAVI<sub>min</sub> or LAVI<sub>max</sub> below those cutoffs, respectively. LAVI<sub>min</sub> and LAVI<sub>max</sub> were found to be independent predictors of MACE after adjusting for established clinical and cardiac MRI risk factors, LA strain parameters, and LA ejection fraction. Adding LAVI<sub>min</sub> to traditional predictors had a greater incremental prognostic value than adding LAVI<sub>max</sub>.

The clinical significance of LAVI<sub>min</sub> has been increasingly emphasized in recent studies (10,13). The LA is directly affected by the LV cavity pressure during diastole. As LV diastolic function progressively deteriorates and LV end-diastolic pressure increases, LA volume increases, LA passive emptying decreases, and conduit function is impaired, with a compensatory increase in active LA emptying in the early stages of LV diastolic dysfunction (16,17). Measured at LV end diastole, when the LA is exposed to the LV, LA minimum volume serves as an early marker of diastolic dysfunction

and elevated filling pressure (18–20). LA minimum volume shows good temporal sensitivity to such changes and strongly correlates with LV diastolic function (8,18), an independent predictor of cardiovascular outcomes (21).

LA structure and function are gaining recognition for their prognostic value in AMI. For example, Leng et al (22) and Schuster et al (23) concluded that LA strain was independently associated with MACE; however, LAVI<sub>min</sub> was not included in their multivariable analysis. Zhang et al (24) showed that LA conduit strain had prognostic significance in 276 patients with AMI. Nayyar et al (25) reported that LA reservoir strain and LAVI<sub>min</sub> were independently associated with MACE in a cohort of 202 patients with AMI. The limited sample size of the latter studies may restrict the generalizability of their results. However, these studies highlight the compensatory role of the LA in mitigating LV failure after AMI. The prognostic significance of LAVI<sub>min</sub> and LA strain, in addition to traditional clinical and cardiac MRI risk factors, has been less explored.

Our study further expanded on previous research regarding the importance of LA volume by directly comparing LAVI<sub>min</sub> and LA functional parameters and revealed that, among LA functional parameters, LAVI<sub>min</sub> was an independent prognostic factor for MACE, which is supported



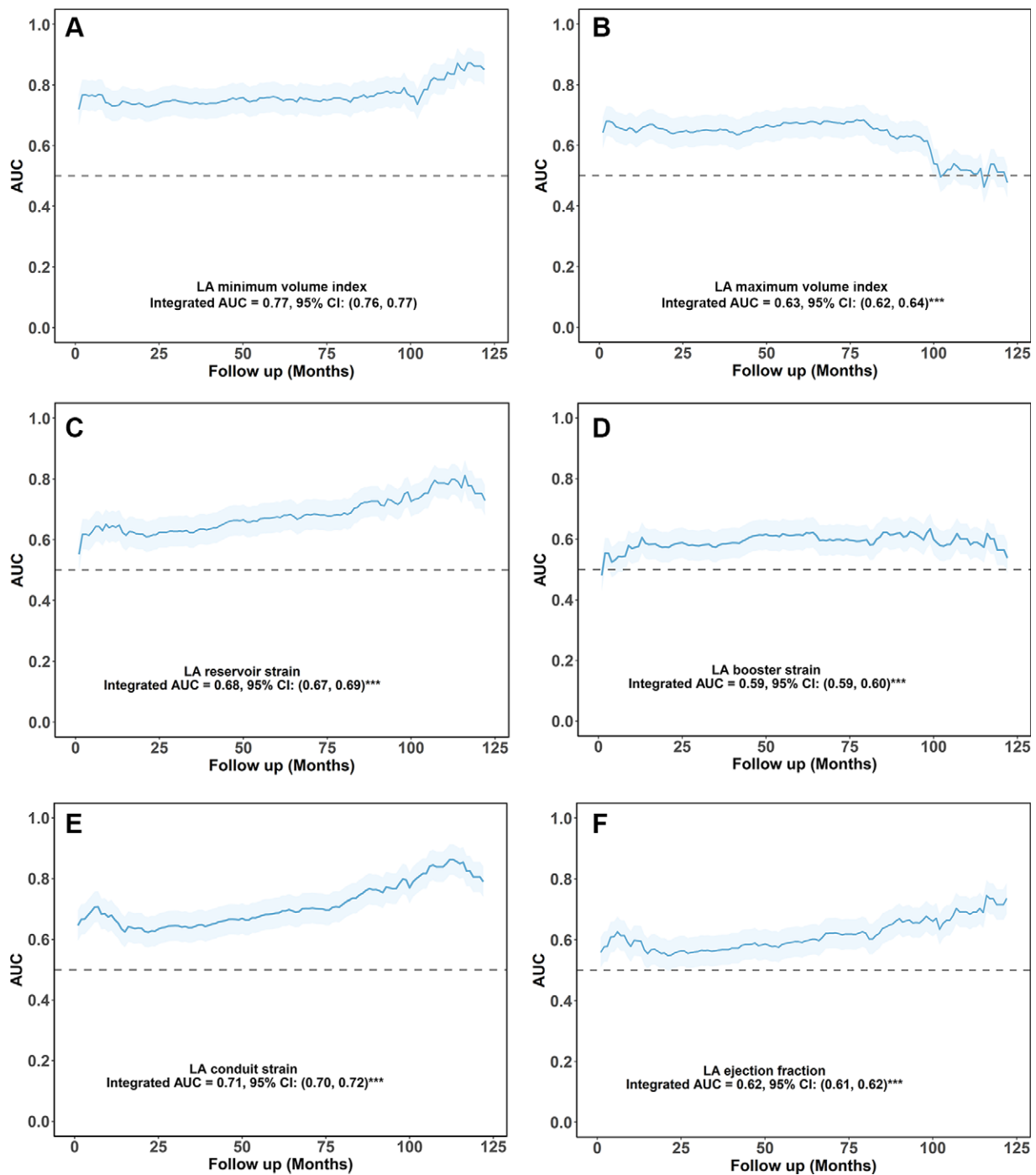
**Figure 4:** Kaplan-Meier survival curves illustrating the incidence of (A) unplanned revascularization, (B) heart failure, (C) reinfarction, and (D) all-cause death stratified by left atrial minimum volume index (LAVI<sub>min</sub>) based on the optimal cutoff. Participants with higher LAVI<sub>min</sub> values (>18.74 mL/m<sup>2</sup>) presented significantly lower event-free survival probabilities than those with lower LAVI<sub>min</sub> values (≤18.74 mL/m<sup>2</sup>) for each of the outcomes (all  $P < .001$ ).

by the findings of Nayyar et al (25). Moreover, LAVI<sub>min</sub> showed superior prognostic capability compared with LAVI<sub>max</sub>, LAEF, and LA strain. This finding can be explained as follows: First, the increase in LA minimum volume is more pronounced than the increase in LA maximum volume as diastolic function worsens, with this difference more evident in the early stages of diastolic dysfunction (18). Second, in the early stages of impaired LV relaxation, enhanced LA contractile function contributes to a preserved LAEF (26). Third, LA reservoir strain and LA conduit strain are influenced by atrioventricular junction motion and closely correlate with LV global longitudinal strain (27), which may confound their ability to reflect diastolic function. Although LA booster strain is directly influenced by LA function (27), LA contractility decreases above a threshold of severe LA enlargement, consistent with the Frank-Starling mechanism (28). Therefore, LAVI<sub>min</sub> offers valuable prognostic information that is available before decompensatory LA enlargement significantly impairs LA function.

Although both LAVI<sub>min</sub> and LAVI<sub>max</sub> provide incremental prognostic value, the model adding both LAVI<sub>min</sub> and LAVI<sub>max</sub> to traditional predictors failed to offer incremental value compared with the model adding only LAVI<sub>min</sub>, likely due to the overlapping prognostic roles of these two parameters, as evidenced by their strong correlation ( $r = 0.78$ ). Given the well-established prognostic significance of LA

strain, combining LAVI<sub>min</sub> with measures of LA strain could improve the assessment of LV filling pressure (29), providing comprehensive information by integrating both LA structure and function assessment in patients after AMI.

Although LA assessment is not routinely performed in clinical practice, mounting evidence highlights its importance for evaluating cardiac pathogenesis and disease progression after AMI (22,23,25). Our research, involving a large cohort of participants with AMI, provides compelling evidence that supports the integration of LAVI<sub>min</sub> into clinical evaluation. Importantly, doing so does not require additional scanning time, as LAVI<sub>min</sub> can be calculated using cine images that are already acquired in standard cardiac MRI protocols. Thus, LAVI<sub>min</sub> can provide complementary prognostic information beyond traditional LV-centric risk factors after AMI, ultimately enhancing risk stratification with minimum workflow disruption. The excellent reproducibility and straightforward measurement technique make LAVI<sub>min</sub> accessible for clinical adoption, and LAVI<sub>min</sub> measurement could help guide follow-up frequency and therapeutic optimization and allow early detection of adverse cardiac remodeling (30). As medicine becomes more personalized, incorporating LAVI<sub>min</sub> into post-AMI assessments could support more targeted interventions, thereby improving outcomes, especially in patients with established risk factors such as diabetes, elevated Killip class, or reduced LVEF.



**Figure 5:** Time-dependent receiver operating characteristic analysis was conducted for (A) left atrial (LA) minimum volume index ( $LAVI_{min}$ ), (B) LA maximum volume index, (C) LA reservoir strain, (D) LA booster strain, (E) LA conduit strain, and (F) LA ejection fraction.  $LAVI_{min}$  demonstrated the highest integrated area under the receiver operating characteristic curve (AUC). The shaded area represents the 95% CI around the AUC estimates. \*\*\* =  $P < .001$  for the comparison with  $LAVI_{min}$  using the Uno C statistic.

Our study has several limitations. Because data were acquired on two different 3.0-T scanner models, there is the possibility of systematic differences in measurements between the systems, although standardized protocols were used to minimize this effect (31,32). Additionally, LA volumes were derived from standard two- and four-chamber cine images, not dedicated LA-focused views. This approach was chosen to enable assessment of parameters available in routine clinical practice, but we acknowledge that specialized views may offer greater measurement accuracy (33). Furthermore, the

timing of cardiac MRI within the first week after AMI varied, which likely influenced LA volume. However, our primary prognostic findings remained robust after adjusting for this factor in statistical analysis. The optimal timing for post-AMI risk stratification with cardiac MRI remains an area for future investigation. Finally, further research with extended follow-up duration is needed to better assess the long-term prognostic value of  $LAVI_{min}$  in patients with AMI.

In conclusion, left atrial minimum volume index ( $LAVI_{min}$ ) derived from cardiac MRI in participants with acute

myocardial infarction was associated with a greater risk of major adverse cardiovascular events during follow-up and provided additional prognostic information compared with traditional risk factors and LA function parameters. Our findings highlight the potential utility of  $LAVI_{min}$ , which could be integrated into routine clinical practice as a quick and reproducible measure obtainable in a single cardiac MRI acquisition.

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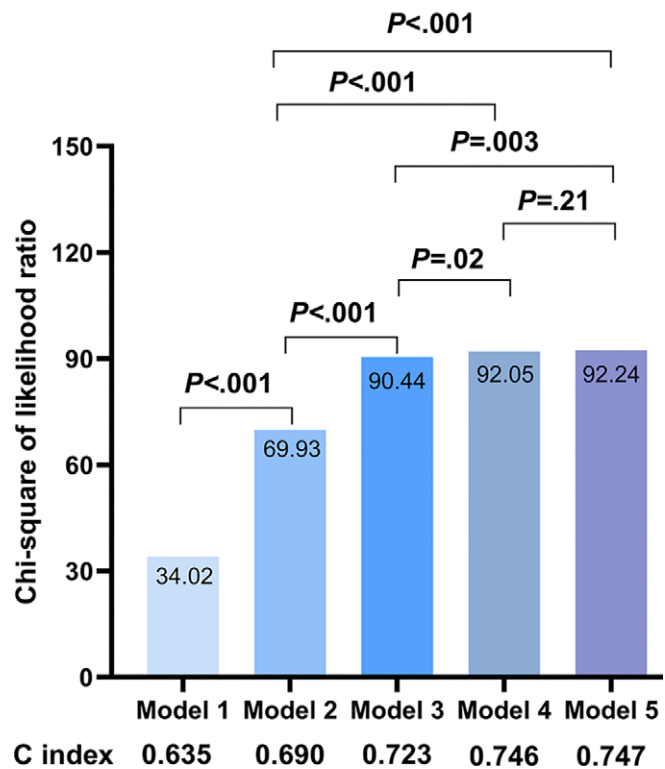
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**Table 5: Incremental Prognostic Value of Adding LA Parameters to a Model with Established Clinical and Cardiac MRI Risk Factors for the Prediction of Major Adverse Cardiovascular Events**

Model	C Index	$\chi^2$ Value of Likelihood Ratio Test	P Value	Bonferroni-corrected P Value
Model 2	0.69 (0.65, 0.73)	69.93	<.001	<.001
Model 2 + LAEF	0.69 (0.65, 0.73)	69.95	<.001	<.001
Model 2 + LA booster strain	0.70 (0.66, 0.74)	73.64	.002	.01
Model 2 + LA reservoir strain	0.70 (0.66, 0.75)	83.80	<.001	.002
Model 2 + LA conduit strain	0.70 (0.66, 0.75)	88.08	.002	.01
Model 2 + $LAVI_{max}$ (model 3)	0.72 (0.68, 0.76)	90.44	.005	.03
Model 2 + $LAVI_{min}$ (model 4)	0.75 (0.71, 0.78)	92.05	Ref.	Ref.

Note.—Data in parentheses are 95% CIs. Model 2 included age, diabetes mellitus, Killip class, left ventricular end-diastolic volume, left ventricular end-systolic volume index, left ventricular ejection fraction, late gadolinium enhancement, microvascular obstruction, intramyocardial hemorrhage, left ventricular aneurysm, and left ventricular thrombus. P values derive from comparison of the C index between each model and model 4. LA = left atrium, LAEF = LA ejection fraction,  $LAVI_{max}$  = LA maximum volume index,  $LAVI_{min}$  = LA minimum volume index, Ref. = reference.



**Figure 6:** Incremental prognostic value of left atrial (LA) minimum volume index ( $LAVI_{min}$ ) and LA maximum volume index ( $LAVI_{max}$ ) over clinical information and traditional cardiac MRI parameters. Model 1 included age, diabetes mellitus, and Killip class. Model 2 included model 1 variables plus left ventricular (LV) end-diastolic volume index, LV end-systolic volume index, LV ejection fraction, late gadolinium enhancement, microvascular obstruction, intramyocardial hemorrhage, LV aneurysm, and LV thrombus. Model 3 included model 2 variables plus  $LAVI_{max}$ . Model 4 included model 2 variables plus  $LAVI_{min}$ . Model 5 included model 2 variables plus  $LAVI_{max}$  and  $LAVI_{min}$ . Models were compared using the  $\chi^2$  value of the likelihood ratio test.

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