

Original Research Article

BASELINE INDEXED LEFT ATRIAL VOLUME AND 30-DAY CARDIOVASCULAR OUTCOMES IN PATIENTS WITH ACUTE CORONARY SYNDROME UNDERGOING REVASCULARIZATION: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Left atrial volume index (LAVI) reflects cumulative left ventricular filling-pressure and atrial remodeling burden. Its short-term prognostic value in Indian patients with acute coronary syndrome (ACS) requires further evaluation. The objective: To determine the association between baseline LAVI and 30-day major adverse cardiovascular events (MACE) in patients with ACS undergoing coronary angiography and planned revascularization.

Materials and Methods: This single-centre prospective observational study included 190 adults with ACS. Transthoracic echocardiography was performed within 48 hours of admission. Participants were classified as normal LAVI (≤ 34 mL/m²; n=83) or increased LAVI (>34 mL/m²; n=107) and followed for 30 days. The composite MACE endpoint comprised unstable angina/non-fatal myocardial infarction, ischemic stroke, heart-failure hospitalization, new-onset atrial fibrillation, or cardiovascular death.

Results: Thirty-day MACE occurred in 41/107 (38.3%) patients with increased LAVI and 5/83 (6.0%) with normal LAVI ($p<0.001$; crude OR 9.69). Increased LAVI was associated with diastolic dysfunction, moderate mitral regurgitation, and triple-vessel disease (all $p<0.001$), while LVEF was similar between groups ($p=0.365$). LAVI predicted MACE with an AUC of 0.728 (95% CI 0.645-0.805); a 33.3 mL/m² cut-off provided 93.5% sensitivity and 96.2% negative predictive value. LAVI >34 mL/m² remained independently associated with MACE (adjusted OR 3.41, 95% CI 1.05-11.11; $p=0.042$).

Conclusion: Increased baseline LAVI identifies a high-risk ACS phenotype and provides early prognostic information complementary to LVEF and coronary anatomy.

Keywords: Acute coronary syndrome, left atrial volume index, echocardiography, major adverse cardiovascular events, diastolic dysfunction, revascularization.

INTRODUCTION

Acute coronary syndrome (ACS) remains a major cause of cardiovascular morbidity and mortality, with a substantial burden in India. Contemporary management emphasizes timely invasive evaluation and revascularization, but early outcome is influenced not only by the culprit coronary lesion but also by the underlying structural and functional state

of the myocardium.^[1] Left ventricular ejection fraction (LVEF) is routinely used for risk assessment; however, it may not fully capture diastolic impairment, chronic filling-pressure burden, or atrial remodeling.

Left atrial volume index (LAVI) is the left atrial volume normalized to body surface area and is the recommended volumetric measure of left atrial size.^[2] Unlike many Doppler indices that vary with

acute loading conditions, left atrial enlargement reflects the cumulative effect of chronically elevated left ventricular filling pressure, impaired relaxation, mitral regurgitation, and atrial pressure-volume loading.^[3,4] LAVI may therefore function as an integrated marker of cardiovascular stress.

Previous studies have shown that increased left atrial volume predicts mortality, heart-failure hospitalization, and other cardiovascular events after myocardial infarction and in coronary heart disease.^[5-9] A systematic review and meta-analysis by Ahmeti et al. found higher rates of MACE, mortality, and hospitalization among ACS patients with increased LAVI.^[10] Subsequent studies, including the HIJ-PROPER analysis and an Indian cohort reported by Saklecha et al., similarly demonstrated adverse outcomes with elevated LAVI.^[11,12] Evidence from Indian revascularized ACS populations remains limited, and the relationship of LAVI with diastolic dysfunction, mitral regurgitation, coronary disease extent, and early MACE merits further study. We therefore evaluated the association between baseline LAVI and 30-day cardiovascular outcomes in patients with ACS undergoing coronary angiography and planned revascularization. Secondary objectives were to estimate the prevalence of increased LAVI and compare diastolic dysfunction, mitral regurgitation, and triple-vessel disease across LAVI categories.

Aim

To determine the association between baseline LAVI and 30-day major adverse cardiovascular events (MACE) in patients with ACS undergoing coronary angiography and planned revascularization.

MATERIALS AND METHODS

Study design and sample size: This was a single-centre prospective observational study conducted in the Department of Cardiology, S.M.S. Medical College and Hospital, Jaipur. The study included 190 adults (>18 years) presenting with STEMI, NSTEMI, or unstable angina who were planned for coronary angiography and subsequent management. Patients with hypertension, moderate-to-severe valvular heart disease, pre-existing atrial fibrillation, restrictive or hypertrophic cardiomyopathy, constrictive pericarditis, chronic renal, pulmonary, or hepatic disease, malignancy, or an inadequate echocardiographic window were excluded. Institutional Ethics Committee approval and written informed consent were obtained.

Data collection and echocardiographic examination

Demographic data, cardiovascular risk factors, presenting symptoms, examination findings, laboratory values, electrocardiographic findings, and ACS subtype were recorded using a predefined

proforma. Comprehensive transthoracic echocardiography was performed within 48 hours of admission using a Philips EPIQ C7 system. Standard chamber dimensions, LVEF, regional wall-motion abnormalities, valvular regurgitation, transmitral flow, tissue-Doppler e' velocities, E/e', deceleration time, and tricuspid-regurgitation jet velocity were assessed in accordance with accepted echocardiographic recommendations.^[2,3]

Left atrial volume was measured at end systole from apical four- and two-chamber views, excluding the pulmonary veins and left atrial appendage, using the modified Simpson method and/or biplane area-length method. LAVI was calculated as left atrial volume divided by body surface area. Participants were categorized as normal LAVI (≤ 34 mL/m²) or increased LAVI (> 34 mL/m²). Coronary angiography documented the extent of coronary artery disease as mild/non-obstructive disease, single-vessel disease (SVD), double-vessel disease (DVD), triple-vessel disease (TVD), or left-main disease. Subsequent PCI, CABG, or medical management was determined by routine clinical indications. All participants were followed for 30 days. Composite MACE was defined as the occurrence of at least one of the following: unstable angina/non-fatal myocardial infarction, ischemic stroke, heart-failure hospitalization, new-onset atrial fibrillation, or cardiovascular death. The primary analysis compared MACE incidence between LAVI categories.

Statistical analysis

Statistical analysis was performed using SPSS version 28.0. Categorical variables are presented as frequency and percentage; continuous variables as mean $\pm$ standard deviation and/or median with interquartile range. Normality was assessed using the Kolmogorov-Smirnov test. Independent-samples t-test or Mann-Whitney U test was used for continuous variables, and chi-square or Fisher's exact test for categorical variables. Crude odds ratios (ORs) with 95% confidence intervals (CI) quantified associations. Receiver-operating-characteristic (ROC) analysis assessed LAVI discrimination for 30-day MACE. Multivariable logistic regression evaluated independent predictors. A two-sided $p < 0.05$ was considered statistically significant.

RESULTS

The study included 190 patients; 130 (68.4%) were male, and 169 (88.9%) were aged ≥ 50 years. NSTEMI was the most common ACS diagnosis (42.6%), followed by unstable angina (40.5%) and STEMI (16.8%). Increased LAVI was present in 107 patients (56.3%). PCI was performed in 167 (87.9%), CABG in 16 (8.4%), and optimal medical therapy was used in 7 (3.7%).

Table 1: Baseline clinical profile of study participants (N=190)

Characteristic	n (%)
Age < 50 years	21 (11.0)
Age 50-59 years	56 (29.5)

Age 60-69 years	71 (37.4)
Age ≥70 years	42 (22.1)
Male sex	130 (68.4)
Urban residence	99 (52.1)
Diabetes mellitus	84 (44.2)
Dyslipidemia	108 (56.8)
Current smoking	108 (56.8)
Unstable angina	77 (40.5)
NSTEMI	81 (42.6)
STEMI	32 (16.8)
Normal LAVI (≤34 mL/m ²)	83 (43.7)
Increased LAVI (>34 mL/m ²)	107 (56.3)

Values are number (percentage).

Age, anthropometric measurements, vital signs, routine hematological values, renal function, electrolytes, and LVEF were comparable between LAVI groups. Total cholesterol and troponin I were

higher with increased LAVI. Left atrial dimensions and volume were greater, while tissue-Doppler e' velocities were lower and deceleration time and TR jet velocity were higher in the increased-LAVI group.

Table 2: Selected clinical and echocardiographic variables according to LAVI category

Variable	Normal LAVI (n=83)	Increased LAVI (n=107)	p-value
Age (years)	62.55 ± 9.90	60.73 ± 10.83	0.228
BMI (kg/m ²)	18.50 ± 1.16	18.78 ± 1.14	0.100
Total cholesterol (mg/dL)	194.35 ± 35.81	258.65 ± 78.17	<0.001
Troponin I, median	0.13	0.32	0.034
LA AP diameter (mm)	33.22 ± 2.87	34.35 ± 2.49	0.005
LVEF (%)	51.22 ± 7.05	50.75 ± 6.95	0.365
Septal e'	7.40 ± 1.47	6.20 ± 1.18	<0.001
Lateral e'	9.37 ± 1.87	7.85 ± 1.49	<0.001
Mean e'	8.39 ± 1.63	7.02 ± 1.26	<0.001
Deceleration time (ms)	180.42 ± 51.79	208.04 ± 41.98	<0.001
TR jet velocity (m/s)	2.11 ± 0.43	2.54 ± 0.43	<0.001
LA volume (mL)	46.79 ± 5.31	55.92 ± 5.32	<0.001
LAVI (mL/m ²)	31.13 ± 2.10	36.73 ± 1.35	<0.001

Values are mean ± SD unless otherwise stated. Independent t-test or Mann-Whitney U test was used as appropriate.

Diastolic dysfunction was present in 41.0% of the normal-LAVI group and 92.5% of the increased-LAVI group. Moderate mitral regurgitation, Grade II/III diastolic dysfunction, and left-main disease

occurred only among patients with increased LAVI. TVD was present in 56.1% of patients with increased LAVI compared with 16.9% with normal LAVI (p<0.001).

Table 3: Diastolic function, mitral regurgitation, and coronary anatomy by LAVI category

Characteristic	Normal LAVI (n=83)	Increased LAVI (n=107)	p-value
Diastolic function: normal	49 (59.0)	8 (7.5)	<0.001
Grade I dysfunction	34 (41.0)	59 (55.1)	
Grade II dysfunction	0 (0.0)	34 (31.8)	
Grade III dysfunction	0 (0.0)	6 (5.6)	
Mitral regurgitation: none	26 (31.3)	14 (13.1)	<0.001
Trivial MR	12 (14.5)	17 (15.9)	
Mild MR	45 (54.2)	61 (57.0)	
Moderate MR	0 (0.0)	15 (14.0)	
Mild/non-obstructive CAD	7 (8.4)	1 (0.9)	<0.001
Single-vessel disease	40 (48.2)	19 (17.8)	
Double-vessel disease	22 (26.5)	18 (16.8)	
Triple-vessel disease	14 (16.9)	60 (56.1)	
Left-main disease	0 (0.0)	9 (8.4)	

Values are number (percentage). Reported p-values refer to the overall distribution within each variable block; chi-square test.

Composite 30-day MACE occurred in 46 patients (24.2%). Event incidence was 38.3% in the increased-LAVI group and 6.0% in the normal-LAVI group (p<0.001), corresponding to a crude OR of 9.69 (95% CI 3.62-25.94) and relative risk of 6.36.

Recurrent unstable angina/non-fatal MI, ischemic stroke, and heart-failure hospitalization were significantly more frequent with increased LAVI. New-onset atrial fibrillation showed a strong but non-significant trend.

Table 4: Thirty-day MACE according to baseline LAVI category

Outcome	Normal LAVI (n=83)	Increased LAVI (n=107)	p-value
Composite 30-day MACE	5 (6.0)	41 (38.3)	<0.001
Unstable angina/non-fatal MI	3 (3.6)	16 (15.0)	0.010

Ischemic stroke	0 (0.0)	6 (5.6)	0.036
Heart-failure hospitalization	0 (0.0)	6 (5.6)	0.036
New-onset atrial fibrillation	2 (2.4)	10 (9.3)	0.051
Cardiovascular death	0 (0.0)	3 (2.8)	0.258

Values are number (percentage). Chi-square or Fisher's exact test was used as appropriate.

Table 5: ROC performance of LAVI for predicting 30-day MACE

ROC metric	Value
Area under the curve	0.728
95% confidence interval	0.645-0.805
Optimal cut-off	33.3 mL/m ²
Sensitivity	93.5%
Specificity	52.8%
Positive predictive value	38.7%
Negative predictive value	96.2%
Accuracy	62.6%

ROC, receiver-operating characteristic; MACE, major adverse cardiovascular events.

In the multivariable model, LAVI >34 mL/m² and TVD remained independently associated with 30-day

MACE. LVEF was not independently associated with the endpoint.

Table 6: Multivariable logistic regression for 30-day MACE

Predictor	Adjusted OR	95% CI lower	95% CI upper	p-value
LAVI >34 mL/m ²	3.41	1.05	11.11	0.042
Triple-vessel disease	6.29	2.72	14.57	<0.001
LVEF, per 1% increase	1.01	0.95	1.07	0.807

OR, odds ratio; CI, confidence interval; LVEF, left ventricular ejection fraction.

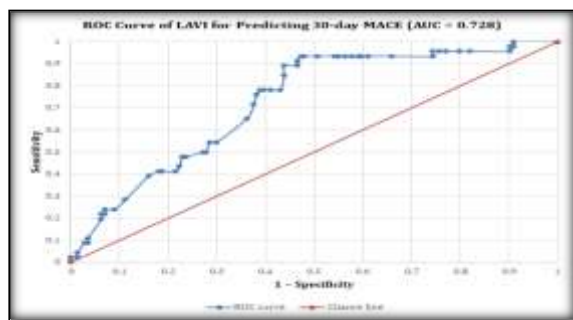


Figure 1: ROC analysis of LAVI for predicting 30-day MACE

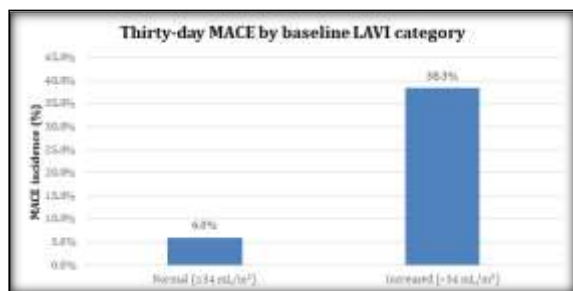


Figure 2. Incidence of composite 30-day MACE according to baseline LAVI category

Patients who developed MACE had higher mean LAVI than those without MACE (36.23 ± 2.28 vs 33.66 ± 3.30 mL/m²; $p < 0.001$), whereas LVEF was similar ($50.28 \pm 7.74\%$ vs $51.17 \pm 6.73\%$; $p = 0.498$). ROC analysis showed moderate discrimination. The optimal 33.3 mL/m² threshold was close to the standard upper-normal value and had high sensitivity and negative predictive value.

DISCUSSION

The principal finding of this prospective study was the marked association between increased baseline LAVI and early adverse cardiovascular events after ACS. More than half of the cohort had LAVI >34 mL/m², and 30-day MACE was more than six times as frequent in this group as among patients with normal LAVI. Elevated LAVI remained independently associated with MACE after adjustment, while LVEF did not distinguish either LAVI groups or MACE status. LAVI therefore appears to capture prognostic information that is not represented by systolic function alone.

The present results closely parallel those of Saklecha et al., who studied 190 Indian patients with ACS undergoing revascularization. They reported 30-day MACE in 20.7% of patients with higher LAVI versus 6.3% with lower LAVI, and identified LAVI and TVD as independent predictors while LVEF was not.^[12] Their ROC-derived LAVI cut-off of 33.35 mL/m² (AUC 0.775) was nearly identical to the 33.3 mL/m² threshold in the present study. The higher event rate in our increased-LAVI group may reflect differences in coronary-disease burden, endpoint composition, or clinical severity.

Broader evidence supports the same direction of association. In the meta-analysis by Ahmeti et al., which included 2,705 ACS patients from 11 cohorts, MACE occurred in 33.7% of patients with increased LAVI compared with 15.9% with lower LAVI; mortality and hospitalization were also more frequent. 10 Ri et al. used a threshold of 34 mL/m² in 226 ACS patients without previous atrial fibrillation and observed higher long-term cardiovascular event rates among those with increased LAVI. 11 Cordeiro

et al. reported progressively worse outcomes across increasing categories of left atrial enlargement after STEMI treated with primary PCI.^[13]

A strong relationship between LAVI and diastolic dysfunction was evident. Grade II and III dysfunction occurred exclusively among patients with increased LAVI, tissue-Doppler e' velocities were lower, and deceleration time and TR jet velocity were higher. This is physiologically plausible because chronic elevation of left ventricular filling pressure exposes the left atrium to persistent afterload and gradual remodeling.^[3,4] The graded rise in MACE from 5.3% with normal diastolic function to 66.7% with Grade III dysfunction further supports the hemodynamic basis of LAVI. The close association with mitral regurgitation may reflect both ischemic ventricular remodeling and cumulative atrial volume loading.

Coronary anatomy was another important correlate. TVD was present in 56.1% of the increased-LAVI group but only 16.9% of the normal-LAVI group; left-main disease occurred only with increased LAVI. Saklecha et al. similarly found more severe coronary disease with higher LAVI.^[12] Liu et al., in 7,818 patients with established three-vessel disease, found that progressively higher LAVI identified increasing long-term risks of adverse cardiac and cerebrovascular events and mortality.^[14] These findings suggest that coronary anatomy and LAVI are complementary: one describes epicardial disease burden, whereas the other reflects the cumulative myocardial and hemodynamic response.

The association of higher troponin I with increased LAVI may indicate coexistence of greater acute myocardial injury and a more vulnerable structural phenotype. Montero-Cabezas et al. demonstrated that impaired flow in the dominant coronary atrial branch after STEMI was associated with subsequent atrial remodeling, supporting a possible contribution from direct atrial ischemia.^[15] Nevertheless, the present observational data cannot separate pre-existing atrial enlargement from acute loading or ischemic effects. Contemporary work increasingly extends atrial assessment beyond size. Tang et al. found the left atrial function index independently predicted outcomes after PCI for acute myocardial infarction,^[16] and Schuster et al. reported incremental prognostic information from left atrial reservoir function beyond LVEF and other advanced imaging markers.^[17] Tangen et al. nevertheless confirmed in a contemporary AMI cohort that simple LAVI remained an independent predictor of mortality and MACE after adjustment for LVEF, strain, filling pressure, and mitral regurgitation.^[18] This supports the practical relevance of routine LAVI where advanced atrial strain analysis is not universally available.

The ROC result has a clinically useful pattern: the 33.3 mL/m² threshold had high sensitivity (93.5%) and negative predictive value (96.2%) but modest specificity (52.8%). A normal LAVI may therefore help identify patients at relatively low short-term risk, whereas increased LAVI should not be used

alone to predict an event. It is better interpreted alongside clinical features, coronary anatomy, diastolic function, and established risk scores.

Strengths and Limitations

Strengths include prospective data collection, standardized echocardiographic assessment within 48 hours, indexing of left atrial volume to body surface area, detailed evaluation of diastolic and angiographic variables, adjudication of clinically meaningful 30-day events, ROC analysis, and multivariable modeling. Limitations include the single-centre design, modest sample and event counts, 30-day follow-up, lack of serial LAVI measurements, absence of left atrial strain, and incomplete comparison with established GRACE, TIMI, or SYNTAX scores. Baseline measurements during acute illness may also be influenced by transient loading conditions. Finally, the observational design establishes association rather than causality.

CONCLUSION

In patients with ACS undergoing coronary angiography and planned revascularization, increased baseline LAVI was common and identified a high-risk phenotype characterized by advanced diastolic dysfunction, greater mitral regurgitation, more extensive coronary artery disease, and substantially higher 30-day MACE. LAVI >34 mL/m² and TVD were independent predictors, whereas LVEF was not. Routine LAVI measurement may complement LVEF and coronary anatomy in early ACS risk stratification, but multicentre studies with longer follow-up and incremental modeling against validated risk scores are required.

Ethics approval and consent to participate: The study was approved by the Institutional Ethics Committee of S.M.S. Medical College and Attached Hospitals, Jaipur (Ref No.- 531/MC/EC/2026). Written informed consent was obtained from all participants.

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