

Clinical implication of LAVI over A' ratio in patients with acute coronary syndrome

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ABSTRACT

Purpose The ratio of the left atrial volume index (LAVI) and late diastolic mitral annular velocity (A') is a useful echocardiographic index for identifying advanced left ventricular (LV) diastolic dysfunction in patients with dyspnoea. We investigated the clinical implications and prognostic value of the aforementioned ratio (LAVI/A') in patients with ST elevation (STE) or non-STE (NSTEMI) acute coronary syndrome (ACS).

Methods We studied 212 patients with ACS.

All patients underwent electrocardiography, echocardiography and measurement of plasma B-type natriuretic peptide (BNP) level on admission. The study endpoints were hospitalisation and mortality because of heart failure (HF).

Results There was a significant, moderate positive correlation between LAVI/A' and natural logarithm (Ln) BNP level among the participants ($r=0.48$, $p<0.0001$). During a mean follow-up of 17 months, eight patients died and nine patients were hospitalised because of HF. The receiver operating characteristics curve indicated that $LAVI/A' \geq 3.0$ predicted these events (log-rank, $p=0.0021$). A significant and moderate positive correlation existed between LAVI/A' and Ln BNP level in the NSTEMI-ACS group ($n=128$; $r=0.58$, $p<0.0001$). However, the correlation between LAVI/A' and Ln BNP level was weaker in the STE-ACS group ($n=84$; $r=0.33$, $p=0.0017$).

Conclusion LAVI/A' was related to plasma BNP levels in patients with ACS, particularly in those with NSTEMI-ACS. This index was useful for predicting cardiac events in patients with ACS.

INTRODUCTION

Left ventricular (LV) diastolic dysfunction has been shown to be related to cardiovascular events,^{1,2} with left atrial (LA) size being linked to LV diastolic dysfunction and elevated LV filling pressure.³⁻⁵ The LA volume has been accepted to be a stable indicator that reflects the duration and severity of LV diastolic dysfunction.⁶ The LA volume is useful for monitoring cardiovascular risk and for guiding therapy.⁶ In addition, an increased LA volume index (LAVI) has been shown to be a powerful predictor of mortality after acute myocardial infarction (AMI).^{7,8} In a recent study,⁹ LAVI had similar predictability as LV ejection fraction (EF) for heart failure (HF) hospitalisation and mortality in ambulatory adults with coronary artery disease (CAD).

Late diastolic mitral annular velocity (A'), which is measured by tissue Doppler imaging (TDI), has been shown to be a relatively preload-independent variable for evaluating LV diastolic function.¹⁰

In patients with decreased LV function, A' can be used to assess LA function, particularly LA systolic function.^{11,12} Reduced A' velocity is a predictor of cardiac death, suggesting an important role for compensatory LA booster pump function.¹³ The LA volume progressively increases² and A' velocity decreases^{12,14} as the LV diastolic dysfunction advances. Park *et al*¹⁵ reported that the LAVI/A' ratio was a useful parameter for identifying advanced LV diastolic dysfunction and predicting clinical outcomes in patients with dyspnoea.

On the other hand, both LV systolic and diastolic functions, as evaluated using echocardiography, can effectively predict cardiovascular events in patients with acute coronary syndrome (ACS).^{16,17} Moreover, approximately 70% of patients with ACS are classified under non-ST elevation ACS (NSTEMI-ACS) and long-term outcomes for these patients are worse for up to 10 years after the event. Several factors are related to this, including an increased prevalence of multivessel disease, a greater likelihood of residual ischaemia and the presence of comorbidities such as diabetes and chronic kidney disease.^{18,19}

In this study, we aimed to investigate the clinical implications and prognostic value of the LAVI/A' ratio in patients with ST elevation (STE) ACS and NSTEMI-ACS. We also examined how this echocardiographic index correlated with the B-type natriuretic peptide (BNP) levels and pulmonary artery wedge pressure (PAWP).

METHODS

Participants

We studied 671 consecutive patients diagnosed with ACS who were admitted to the coronary care unit of our hospital between January 2006 and May 2008. All patients underwent electrocardiography (ECG) and echocardiography, including TDI on admission, and we excluded patients with atrial fibrillation, atrial flutter or significant valvular abnormalities. Patients were eligible for inclusion if they had AMI or unstable angina pectoris ($n=212$; mean (SD) age, 65 (12) years; 166 men). Pulmonary artery catheters were indwelled in 46 patients.

All patients were informed of the purpose and methods of the study in detail, and they provided written informed consent.

Echocardiography

At a median of 1 day (range, 0–5 days) after admission, transthoracic echocardiography was performed using commercially available ultrasound equipment (Sonos 5500; S3 probe; Philips Medical Systems, Andover, Massachusetts, USA). The LV



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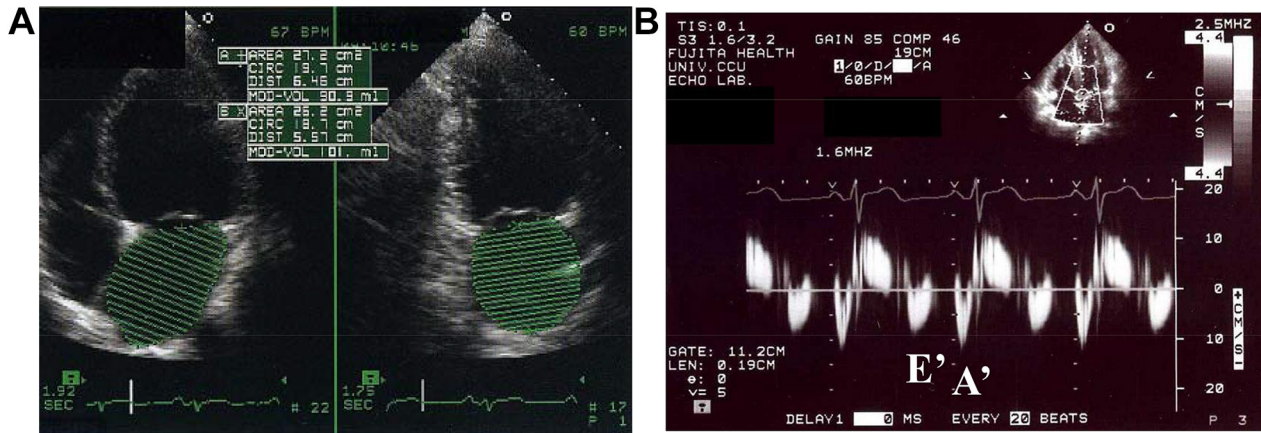


Figure 1 A method of left atrial volume measurement (A) and measurement of mitral annular velocity by Doppler tissue imaging (B).

volume was measured at both end systole and end diastole by the biplane modified Simpson's method with apical 4-chamber and 2-chamber views, and the LV ejection fraction (LVEF) was calculated. Subsequently, the LV end-diastolic dimensions, LV end-systolic dimensions, interventricular septal dimensions and LV posterior wall dimensions were measured from a parasternal long-axis view using conventional two-dimensional measurements.

The peak velocities of early (E) and late (A) diastolic filling, E/A ratio and E-wave deceleration time were derived from pulsed wave Doppler recordings of mitral inflow. The LA volume was assessed using the biplane Simpson's method from apical 4-chamber and 2-chamber views in end systole. LAVI was obtained by correcting for body surface area. Peak early (E') and late (A') diastolic mitral annular velocities were acquired at the septal site of the mitral annulus using TDI from an apical 4-chamber view (figure 1). The E/E' and LAVI/A' ratios were calculated.

Electrocardiogram

All participants underwent 12-lead ECG on admission (FDX-4520LA, Fukuda Denshi, Tokyo, Japan). Patients with STE at the J point in two or more consecutive leads (with the cut-off point >0.2 mV in lead V1, V2 or V3 and >0.1 mV in the other leads) were defined as having STE-ACS. Patients with ST-segment depression, T-wave inversion or no ECG abnormalities were defined as having NSTEMI-ACS.

Plasma BNP and serum creatine kinase

After blood was drawn from the antecubital veins or femoral arteries, it was added to tubes filled with ethylenediaminetetraacetic acid for measuring the BNP levels and in plain tubes for measuring the creatine kinase (CK) levels. The tubes were then centrifuged, and the separated plasma or serum was frozen at (-80°C) until analysis. The BNP level was measured by chemiluminescence enzyme immunoassay, and the CK level was measured by an ultraviolet test.²⁰

Pulmonary artery wedge pressure

After informed consent was obtained from appropriate patients, right heart catheterisation was performed using a 7.5-French gauge, flow-directed Swan-Ganz catheter, which was introduced through a femoral vein.

Clinical assessment and end points

Follow-up clinical information was collected by a review of medical records. The primary study end points were mortality and rehospitalisation because of HF.

Statistical analysis

Continuous variables are presented as mean \pm SD. Differences in the baseline characteristics between groups were determined using the Student's t-test for continuous variables and the χ^2 test for dichotomous variables. Correlations between the LAVI/A' ratio and natural logarithm (Ln) BNP levels as well as PAWP were assessed by linear regression analysis. Multivariate proportional hazard models were used to detect the predictors of the predefined end points. The area under the receiver operating characteristics (ROC) curve (AUC) was used to evaluate optimal cut-offs for selected outcome predictors, and predictive values were calculated. Event-free survival curves were generated using the Kaplan-Meier method, and the differences between the groups were analysed using the log-rank test. Statistical significance was defined as a p value of <0.05 . We used JMP (V.8.0 for Windows, SAS, Cary, North Carolina, USA) for all statistical analyses.

RESULTS

Correlation with clinical outcomes

During a mean follow-up at 17 (range, 0–35) months, eight patients died and nine patients were rehospitalised because of HF. Univariate predictors of the composite end points are presented in table 1. LVEF and peak E' velocity were significantly reduced in patients with an endpoint, whereas age and the LAVI/A' ratio significantly increased in these patients. ROC analysis revealed that an LAVI/A' ratio of ≥ 3.0 was the optimal cut-off for predicting mortality and rehospitalisation because of HF in all participants (AUC=0.61, $p<0.05$; sensitivity and specificity, 52.9% and 80.5%, respectively).

Patients were classified into two groups according to the LAVI/A' ratio: Group A, with a LAVI/A' ratio of ≥ 3.0 (n=48) and Group B with a LAVI/A' ratio of <3.0 (n=164). The clinical characteristics and echocardiographic parameters for each group are shown in tables 2 and 3. The Kaplan-Meier survival curves for Groups A and B are shown in figure 2. The incidence of events was significantly higher in Group A (log-rank, $p=0.0021$). The multivariate proportional hazards analysis showed that the LAVI/A' ratio was an independent and strong

Table 1 Univariate predictors of the composite end point of mortality and HF rehospitalisation

	End point absent (N=195)	End point present (N=17)	P values
	Mean±SD or %	Mean±SD or %	
Age, years	63.7±11.7	67.1±12.9	0.25
Male gender, %	81	47	0.24
Hypertension, %	66	82	0.18
Lipid metabolism abnormality, %	45	47	0.88
Smoking habit, %	57	59	0.88
Diabetes mellitus, %	41	59	0.17
PCI within 24 hours, %	47	41	0.67
Anticoagulant	96	88	0.15
ACE inhibitors	21	58	0.0005
ARB	70	53	0.13
Beta-blockers	61	71	0.44
BNP, pg/mL	210.7±499.9	318.4±321.9	0.41
Max CK, IU/L	1639.4±1875.6	3871.8±4169.5	<0.0001
LV ejection fraction, %	52.3±11.0	44.1±12.9	0.0040
Mitral E velocity, cm/s	73.8±18.5	69.5±21.6	0.38
Mitral A velocity, cm/s	82.0±20.8	89.8±23.3	0.15
Mitral E/A ratio	0.9±0.3	0.8±0.2	0.07
DcT, ms	234.5±59.0	219.7±64.4	0.34
LAVI, mL/m ²	27.9±9.6	30.5±10.1	0.28
Septal annular TDE			
Peak E' velocity, cm/s	9.3±1.9	8.3±1.9	0.04
Peak A' velocity, cm/s	12.6±2.3	11.6±2.7	0.10
E/E' ratio	8.2±2.7	8.3±2.9	0.94
LAVI/A'	2.3±1.0	2.8±1.3	0.04

ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blocker; BNP, B-type natriuretic peptide; CK, creatine kinase; DcT, deceleration time of mitral E wave; HF, heart failure; LAVI, left atrial volume index; LV, left ventricular; PCI, percutaneous coronary intervention; TDE, tissue Doppler echocardiography.

predictor of mortality and rehospitalisation because of HF (HR, 5.32; 95% CI 1.50 to 18.92; $p=0.0104$) (table 4).

Correlations of the LAVI/A' ratio with Ln BNP

There was a significant and moderate positive correlation between Ln BNP and the LAVI/A' ratio among all participants ($r=0.49$, $p<0.0001$) (figure 3A); however, the correlation

Table 2 Clinical characteristics in patients with LAVI/A' ≥ 3.0 and with LAVI/A' < 3.0 in all the subjects

	LAVI/A'		P values
	>3.0 (N=48)	<3.0 (N=164)	
	Group A	Group B	
Age, years	67.5±11.0	62.9±11.9	0.02
Male gender, %	65	82	<0.01
BSA, m ²	1.7±0.2	1.7±0.2	0.50
Systolic BP, mm Hg	137.5±31.5	134.7±24.2	0.52
Diastolic BP, mm Hg	68.8±19.0	72.2±16.1	0.21
Hypertension, %	79	64	0.05
Lipid metabolism abnormality, %	52	43	0.32
Smoking habits, %	47	60	0.10
Diabetes mellitus, %	59	38	0.01
Prior MI, %	17	16	0.89
BNP, pg/mL*	569.8±899.2	120.9±201.7	<0.0001
Max CK, IU/L	1720.3±2467.5	1847.1±2147.6	0.73
PAWP, mm Hg†	13.5±6.5	10.4±4.9	0.09

*Data available in 202 patients.

†Data available in 46 patients.

BNP, B-type natriuretic peptide; BP, blood pressure; BSA, body surface area; CK, creatine kinase; LAVI, left atrial volume index; MI, myocardial infarction; PAWP, pulmonary artery wedge pressure.

Table 3 Echocardiographic parameters in patients with LAVI/A' ≥ 3.0 and with LAVI/A' < 3.0 in all the subjects

	LAVI/A'		P values
	>3.0 (N=48)	<3.0 (N=164)	
	Group A	Group B	
Two-dimensional echocardiography			
LV ejection fraction, %	45.7±13.1	53.4±10.6	<0.0001
LV end-diastolic dimension, mm	48.4±9.4	45.1±5.6	<0.01
LV end-systolic dimension, mm	34.0±8.8	32.6±9.1	0.35
Interventricular septum dimension, mm	12.2±2.9	11.5±2.3	0.06
LV posterior wall dimension, mm	12.3±3.0	11.6±1.9	0.06
LAVI, mL/m ²	41.3±8.0	24.2±6.0	<0.0001
Transmitral flow			
Mitral E velocity, cm/s	77.7±22.3	72.3±17.5	0.08
Mitral A velocity, cm/s	86.0±22.4	81.6±20.6	0.21
E/A ratio	0.9±0.3	0.9±0.3	0.82
DcT, ms	222±50	237±62	0.12
Mitral annular motion			
Peak E', cm/s	8.3±1.9	9.5±1.8	<0.001
Peak A', cm/s	10.8±1.8	13.0±2.3	<0.0001
E/E' ratio	9.6±3.8	7.8±2.1	<0.0001
LAVI/A'	3.9±0.7	1.9±0.5	<0.0001

DcT, deceleration time of mitral E wave; LAVI, left atrial volume index; LV, left ventricular

between the LAVI/A' ratio and Ln BNP was stronger in the NSTEMI-ACS group ($r=0.58$, $p<0.0001$) (figure 3B) than the STE-ACS group ($r=0.33$, $p=0.0017$). There was also a significant positive correlation between the LAVI/A' ratio and the PAWP result ($r=0.30$, $p<0.0001$) (figure 4).

DISCUSSION

Although the LAVI/A' ratio has previously been reported to be useful in detecting advanced LV diastolic dysfunction and predicting the prognosis in patients hospitalised with dyspnoea,¹⁵ this is the first study to show its clinical usefulness in patients with CAD. In particular, the LAVI/A' ratio had a significant positive correlation with Ln BNP, indicating an increase in the LAVI/A' ratio being associated with higher BNP levels.

BNP is released from the cardiac ventricles in response to myocyte stretching, and an elevation of LV diastolic pressure can increase the plasma BNP levels.²¹ After an AMI, elevated BNP concentrations identify patients at risk for adverse LV

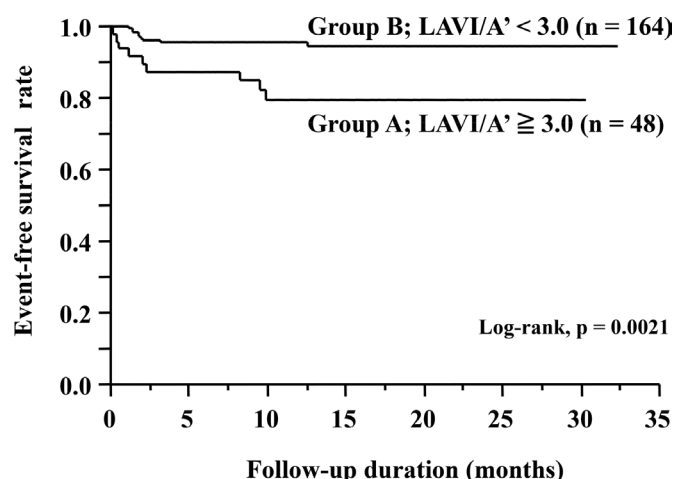
**Figure 2** Kaplan-Meier plot of survival free from mortality and HF rehospitalization.

Table 4 Multivariate proportional hazards analysis predict of mortality and HF rehospitalisation

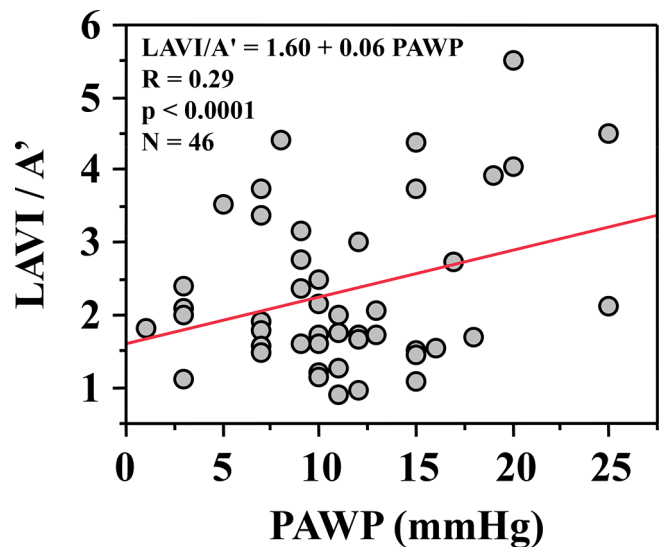
	HR	95% CI	P values
Gender, male	0.17	0.05 to 0.57	0.004
Age, years	1.00	0.93 to 1.08	0.94
LV ejection fraction, %	0.97	0.91 to 1.03	0.31
E/A	0.10	0.00 to 2.54	0.17
DcT, ms	0.99	0.98 to 1.00	0.27
E/E'	0.77	0.55 to 1.05	0.11
LAVI, mL/m ²	0.87	0.76 to 0.98	0.03
LAVI/A'	5.32	1.50 to 18.92	0.01
BNP, pg/mL	1.00	1.00 to 1.00	0.70

BNP, B-type natriuretic peptide; DcT, deceleration time of mitral E wave; HF, heart failure; LAVI, left atrial volume index; LV, left ventricular.

remodelling, LV dysfunction, HF and death, independent of age and history of HF and LVEF. Even in patients with unstable angina and no evidence of myocardial necrosis or HF, increased concentrations of BNP are associated with an increased risk of death.²² Given that BNP had a significant correlation with LV end-diastolic pressure, the LAVI/A' ratio should increase as the LV end-diastolic pressure increases.

In this study, patients in the NSTEMI-ACS group had a stronger correlation with Ln BNP compared with those in the STEMI-ACS group. This may be explained by the fact that NSTEMI-ACS is more likely to occur in the setting of chronic repetitive episodes of arterial occlusion with platelet embolisation. However, STEMI-ACS is a result of more acute myocardial injury; therefore, the LAVI/A' ratio may not necessarily change in parallel with the BNP levels in patients with STEMI-ACS. Consequently, the LAVI/A' ratio was more correlated with Ln BNP in the NSTEMI-ACS group.

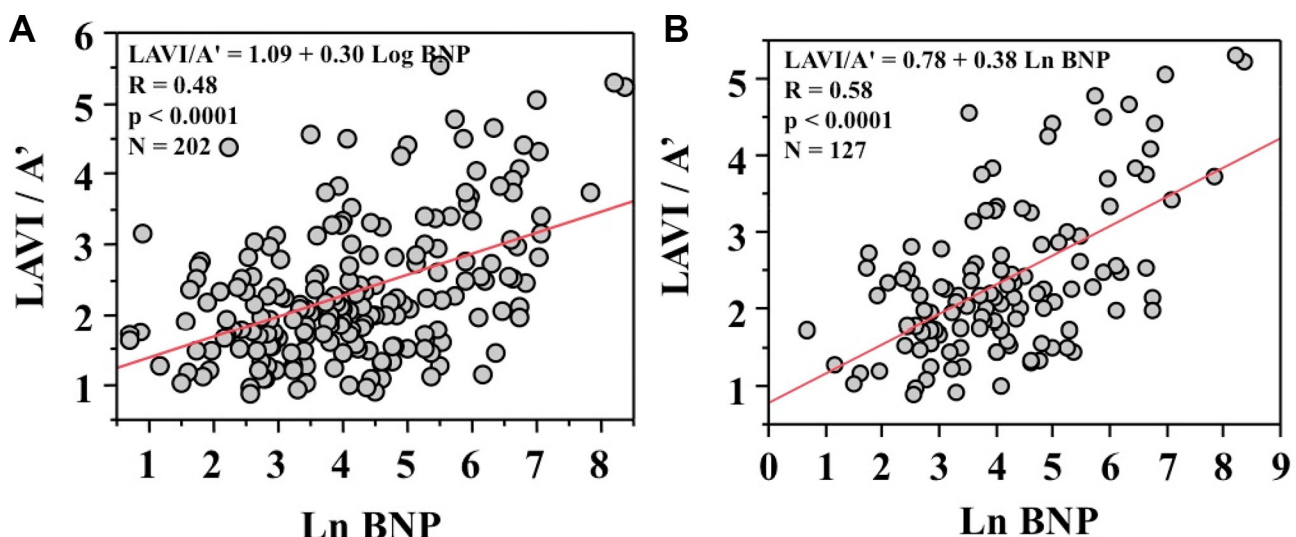
According to Park *et al*, a LAVI/A' ratio of 4.0 was the best cut-off value for identifying advanced diastolic dysfunction.¹⁵ In contrast, we showed a lower optimal cut-off value of 3.0 for predicting cardiac events in patients with CAD. This difference may have resulted from the fact that there was no record of the aetiology of dyspnoea in the study by Park *et al* and that they could have included patients with a wider range of heart diseases. We consider it to be reasonable to have different cut-off values for the LAVI/A' ratio among heart diseases and recommend that the LAVI/A' ratio of 3.0 quoted in this study be taken

**Figure 4** Correlation between LAVI/A' with PAWP.

in the context of our population. Further research will be needed to confirm our assertions in prospective studies.

The correlation between the LAVI/A' ratio and PAWP was weak. This is consistent with the previous observation that the LAVI/A' ratio may be potentially useful in the diagnosis of chronic diastolic dysfunction, whereas PAWP represents a more acute stage of elevated LV filling pressure.¹⁵

LA is exposed to LV filling pressures through the open mitral orifice during diastole; therefore, its size is influenced by the same factors that determine the diastolic filling pressure. When LV diastolic dysfunction becomes evident, the LA pressure increases to maintain adequate filling,²³ and the increased atrial wall tension leads to stretching of the atrial myocardium and chamber dilatation. Therefore, the LA volume reflects the long-term exposure of the left atrium to abnormal LV diastolic function and filling pressure,⁵ and the LAVI itself is a stable indicator of LV diastolic function. However, LAVI is simply a manifestation of structural information, regardless of its function. In this study, LAVI was not shown to be a significant predictor of cardiac events, despite including participants without LA dilatation even among those with cardiac events, who were not considered to

**Figure 3** Correlation between LAVI/A' with Ln BNP.

have severely damaged LV diastolic function. Finally, although LAVI is derived from simple morphological information, A' reflects LA function. Thus, the LAVI/A' ratio offers an effective measure for predicting LV diastolic dysfunction by combining both structural and functional data to detect subtler changes in LV diastolic dysfunction, particularly in patients with relatively preserved diastolic function.

The magnitude of A' has been shown to be altered by age.^{24–26} However, previous studies were performed in healthy participants, and in this study of patients with ACS, there was no correlation between A' and age. This is consistent with the evidence by Abe *et al*,²⁷ who showed that a decreased A' can result from significantly decreased functional reserves in the pulmonary venous system, from LA pump dysfunction or from a significant elevation in the LA pressure.

It is well known that the E/E' ratio can be used to estimate PAWP and that there is a strong relationship between the two variables.^{28–29} The E/E' ratio is also a powerful predictor of survival after AMI, with one report showing that a value of >15 predicts decreased survival.¹⁶ However, we did not find the E/E' ratio to be a significant predictor, possibly because too few patients had an elevated E/E' ratio (E/E', >15) in this study. Although the clinical significance of mild elevations in the E/E' ratio (ie, 8 < E/E' < 15) remain unclear,³⁰ we think that the LAVI/A' ratio provides a useful indicator of outcomes in patients without an elevated E/E' ratio (ie, with an E/E' ratio of <15).

Limitations

This study has several limitations. First, patients with atrial fibrillation, atrial flutter or significant valvular abnormalities were excluded. It is possible that many of these patients had LA dilatation regardless of the LV filling pressure. In addition, because A' is produced by atrial contraction, it is absent in atrial fibrillation. Second, patients in this study were undergoing medical treatment at an intensive care unit where it is sometimes difficult to acquire good image quality because of either the use of respirators or postural limitations. It is not possible to obtain accurate LAVI values from poor quality images. Third, the number of patients may be insufficient to provide an accurate ROC analysis or true predictive values. Finally, it is important to note that this was only an initial study to demonstrate the usefulness of the LAVI/A' ratio for predicting mortality and hospitalisation

because of HF in patients with ACS. Further prospective studies with larger samples will be needed to confirm our findings.

CONCLUSION

The LAVI/A' ratio is available as a new echo index which reflects LV chronic diastolic function in patients with ACS. It can predict mortality and rehospitalisation because of HF particularly in those with NSTEMI-ACS. An increase in the LAVI/A' ratio suggests that the PAWP and BNP levels are elevated.

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Contributors HM and AY designed the study, analysed and interpreted the data and wrote the manuscript; KuS and KeS performed the experiments and collected the data. MI, TI, JI and YO reviewed and provided critical input for the manuscript. All authors contributed to writing the manuscript and gave final approval.

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Competing interests None declared.

Patient consent Obtained.

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Key questions

What is already known about this subject?

- ▶ Left atrial volume index (LAVI)/A' is reported to be a useful echo parameter to identify advanced diastolic dysfunction in patients with dyspnoea.

What does this study add?

- ▶ We showed that LAVI/A' is also a helpful index in patients with acute coronary syndrome (ACS), particularly in those with non-ST elevation ACS (NSTEMI-ACS).

How might this impact on clinical practice?

- ▶ LAVI/A' ratio may help predict mortality and rehospitalisation because of heart failure particularly in patients with NSTEMI-ACS, because we found that an increase in LAVI/A' indicated elevation in pulmonary artery wedge pressure and B-type natriuretic peptide levels, reflecting left ventricular diastolic dysfunction.

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