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Prognostic value of right atrial strain in patients with chronic heart failure

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Abstract

Aims Right ventricular dysfunction is a well-established prognostic marker in patients with heart failure (HF). However, the prognostic significance of right atrial (RA) function remains unclear. Given its sensitivity to systemic congestion, RA function may provide additional insights into HF disease progression and management. This study aimed to investigate whether RA reservoir function serves as an independent prognostic indicator in patients with chronic HF.

Methods A total of 613 patients with chronic HF and a left ventricular (LV) ejection fraction of less than 50% who underwent echocardiographic assessment at Okayama University Hospital between January 2018 and March 2023 were included (median age: 68 (58–76) years; 69% male). RA reservoir function was quantified using two-dimensional speckle-tracking echocardiography. The primary endpoint was cardiovascular death or HF-related hospitalization. Kaplan–Meier survival analysis was performed to examine the association between RA reservoir function and clinical outcomes.

Results During a median follow-up period of 41 months (range: 12–91 months), 119 patients experienced cardiac events. Compared with event-free patients, those with cardiac events exhibited a significantly larger RA maximum volume index (38 mL/m^2 vs. 31 mL/m^2, $P < 0.001$) and a significantly lower RA reservoir longitudinal strain (RASr) (17% vs. 22%, $P < 0.001$). Kaplan–Meier analysis demonstrated that patients with RASr ≤ 20% had significantly poorer event-free survival than those with RASr > 20%, even without RA volume enlargement (log-rank test, $P < 0.001$). Multivariate Cox regression analysis identified RASr as an independent predictor of cardiac events (hazard ratio: 0.95, 95% confidence interval: 0.93 to 0.97, $P < 0.001$).

Conclusions In patients who experienced adverse cardiac events, a reduced RASr and an increased RA maximum volume were observed. Furthermore, a reduced RASr was independently associated with an increased risk of cardiovascular death and HF-related hospitalization in patients with chronic HF and LV dysfunction. These findings indicate that RASr may serve as a valuable prognostic marker for the risk stratification and management of chronic HF.

Keywords Right atrial function, Right atrial strain, Chronic heart failure, Echocardiography

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Introduction

The right atrium is anatomically connected to the inferior and superior vena cave and is directly influenced by venous pressure [1]. Additionally, the right atrial (RA) function is intrinsically dependent on preload and is modulated by right ventricular (RV) diastolic pressure [1–4]. Notably, previous studies have demonstrated that RA wall expansion directly enhances cardiac output by 10%–20%, thereby contributing to an increase in stroke volume [5]. Then, RA function plays a crucial role in modulating venous congestion in heart failure (HF) patients. Despite extensive research on left atrial (LA) function in HF, investigations focusing on RA function and its prognostic implications remain limited. Recent studies have established an association between LA function, assessed by two-dimensional speckle-tracking echocardiography (2D-STE), and adverse clinical outcomes in patients with HF [6–9]. Furthermore, functional assessments of LA using strain analysis have demonstrated superior prognostic value compared with conventional morphological parameters, such as LA diameter and volume [10]. In the current study, we hypothesized that RA reservoir longitudinal strain (RASr) may serve as a comprehensive non-invasive indicator, reflecting both volume overload and RV diastolic pressure, with the potential to provide valuable prognostic insights into disease severity and the prediction of cardiovascular events in patients with chronic HF and left ventricular (LV) dysfunction. Therefore, this study aimed to evaluate RA morphology and reservoir function using 2D-STE in patients with chronic HF and to elucidate the clinical significance of RA reservoir function in this population.

Methods

Study population

This retrospective study analyzed patients with chronic HF and LV ejection fraction of less than 50% who underwent echocardiography as outpatients at Okayama University Hospital between January 2018 and March 2023. Patients with a history of cardiac amyloidosis, pericardial disease, adult congenital heart disease or prior open-heart surgery were excluded. Additionally, individuals experiencing acute decompensated HF were not included. Patients with suboptimal echocardiographic image quality that precluded reliable tracking of the RA walls were also excluded. A total of 613 patients with adequate imaging for analysis were included in the study (Fig. 1). All patients met the clinical diagnostic criteria for HF as defined by the European Society of Cardiology guidelines [11]. This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. All patients provided written informed consent for the examinations, and the study was approved by the ethical committee of our institution.

Echocardiographic parameters

Transthoracic echocardiography (TTE) was performed using commercially available ultrasound systems (Vivid E95; GE Healthcare, Milwaukee, WI, USA; and Aplio i800; Canon Medical Systems, Otawara, Japan). Key echocardiography parameters included LV end-diastolic volume, LV end-systolic volume, LV ejection fraction, LV mass index, LA volume index, early diastolic mitral inflow velocity to mitral annular velocity (E/e') ratio, tricuspid regurgitation (TR) peak velocity, and inferior vena cava diameter. LV ejection fraction was quantified using

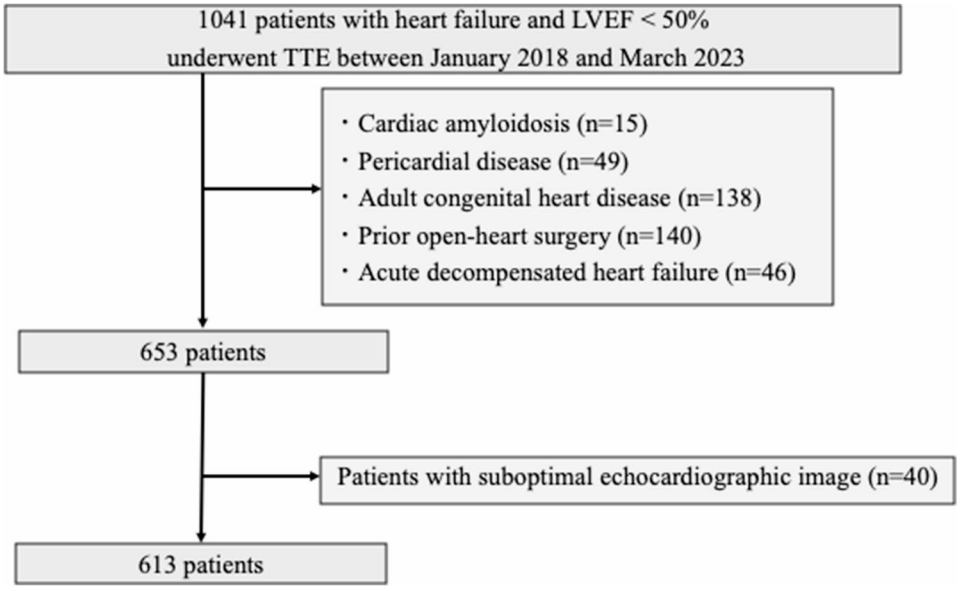

Fig. 1 Inclusion and exclusion criteria for the study subjects. LVEF = left ventricular ejection fraction, TTE = transthoracic echocardiography

the modified disc summation method. Furthermore, RV parameters, including RV end-diastolic area, RV end-systolic area, and RV fractional area change (RVFAC), were obtained from an RV-focused apical four-chamber view. All echocardiographic measurements were performed in accordance with the standardized guidelines of the American Society of Echocardiography [12].

Strain analysis

RA strain and volumetric assessments were performed using TOMTEC-ARENA Cardiac Performance Analysis software. All echocardiographic images were acquired in DICOM format and subsequently analyzed at the core laboratory. The 2D-STE was used for offline measurement of atrial longitudinal strain using a dedicated atrial strain analysis module from apical four-chamber view images, which provided clear visualization of the right atrium (Fig. 2). The software originally developed for LA strain analysis was adapted for RA assessment. For strain measurement, three anatomical reference points were designated at the tricuspid annulus and the superior aspect of the RA roof. The software automatically delineated the RA endocardial border and executed speckle-tracking analysis throughout a single complete cardiac cycle. In cases where the soft-generated endocardial border was inaccurate, manual adjustments were performed to optimize tracking fidelity. The 2D-STE analysis yielded values for RASr, which were derived from the global longitudinal strain calculated based on endocardial tracing. In patients with atrial fibrillation (AF), RASr was measured by averaging three consecutive cardiac cycles with adequate image quality and tracking, in accordance with current echocardiographic recommendations. Additionally, RA maximum volume was quantified. All 2D-STE analyses were performed by a single independent and experienced cardiologist, who was blinded to the patients' clinical date to ensure unbiased information.

Clinical outcomes

The primary endpoint was cardiac death or hospitalization for HF. Patients were monitored from the date of echocardiographic assessment until the occurrence of the first documented cardiac event or the end of follow-up. Follow-up data were systematically retrieved from medical records.

Statistical analysis

Continuous variables are expressed as mean ± standard deviation or median (interquartile range), while categorical variables are presented as numbers and percentages. Comparisons between the two groups were performed using the independent *t*-test and Mann-Whitney *U* test for continuous variables, and the χ^2 test for categorical variables. For survival analyses, patients were stratified into two groups based on the median values of RASr, RA maximum volume index, and RA area. Kaplan–Meier analysis was employed to estimate event-free survival rates, with differences between groups evaluated using the log-rank test. Cox proportional hazards regression analysis was performed to identify independent predictors of adverse cardiac events. The variables for their multivariate analysis were selected based on two primary criteria: statistical significance in univariate analysis and clinical considerations. We considered variables that demonstrated a statistically significant association in the univariate analysis. Beyond this statistical threshold, the clinical relevance and importance of the variables were also factored into their selection process. Then, Model 1 included clinical parameters, including age, male, New York Heart Association (NYHA) functional class III or IV, chronic kidney disease, AF and RASr. Model 2 included variables pertinent HF assessment indications, including LV ejection fraction, LA volume index, E/e' ratio, TR peak velocity, significant TR, RVFAC, RA maximum volume index and RASr. Hazard ratios are reported with corresponding 95% confidence intervals. To evaluate the

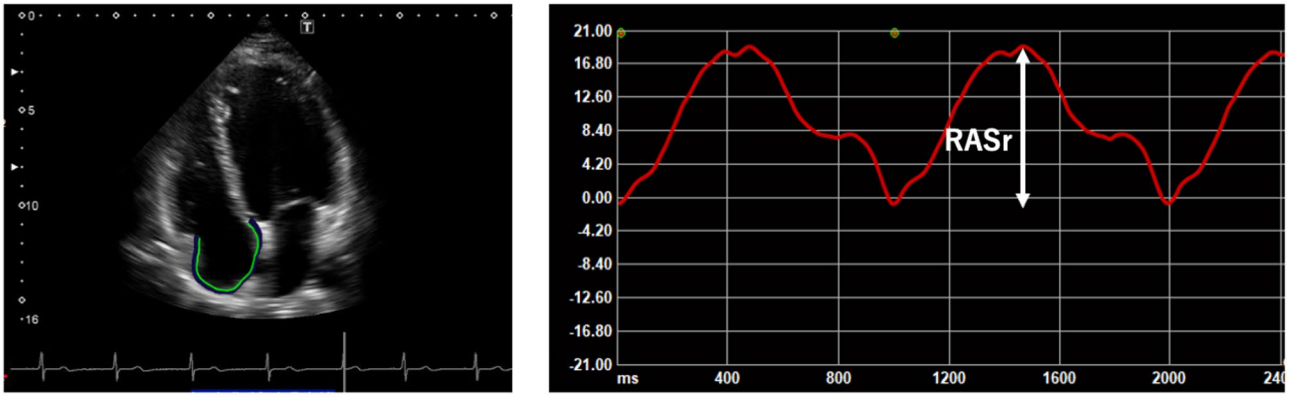

Fig. 2 RA longitudinal strain was measured using 2D-STE. The RA strain value at ventricular end-diastole was set to zero, with the highest value defined as RASr. RA = right atrial, RASr = right atrial reservoir longitudinal strain, STE = speckle-tracking echocardiography

incremental prognostic value of RASr beyond conventional parameters, we performed a likelihood ratio test by comparing nested Cox proportional hazards models. Specifically, we compared a baseline model including LA volume index alone with an extended model incorporating both LA volume index and RASr. Improvement in model fit was assessed based on the chi-square difference between the two models. Statistical analyses were conducted using JMP version 18.0 (SAS Institute Inc., Cary, NC, USA), with statistical significance defined as a p-value < 0.05.

Interobserver- and intra-observer differences were analyzed for 20 randomly selected images. The RA maximum volume and RASr were evaluated by two blinded observers and by a single observer at two different time points. Reliability was calculated using Pearson's correlation coefficient. Variability was calculated as the percentage error of each measurement and derived as the difference between the two measurements divided by the mean value.

Results

Patient characteristics

Patient characteristics of the study population are presented in Table 1. The median age of all patients was 68 (58–76) years, and 424 patients (69%) were male. The etiological distribution of HF was as follows: coronary artery disease in 283 patients (46%), dilated cardiomyopathy in 239 patients (39%), hypertrophic cardiomyopathy in 25 patients (4%) and primary valvular disease in 19 patients (3%). AF was observed in 117 patients (19%). The distribution of HF symptoms, according to NYHA functional class, was observed as 23% for NYHA I, 56% for NYHA II, 20% for NYHA III and 1% for NYHA IV. Most patients were receiving guideline-directed medical therapy, including angiotensin-converting enzyme inhibitors

Table 1 Clinical characteristics

Variables	All (n = 613)	Events		p-value
		Present (n = 119)	Absent (n = 494)	
Age, years	68 (58–76)	71 (62–77)	68 (57–76)	0.009
Male	424 (69)	85 (73)	339 (69)	0.582
Body mass index, kg/m^2	24 ± 5	23 ± 5	24 ± 5	0.556
Comorbidity				
Hypertension, n (%)	241 (39)	39 (33)	202 (49)	0.117
Diabetes, n (%)	174 (28)	45 (38)	129 (26)	0.013
Dyslipidemia, n (%)	273 (45)	54 (45)	219 (44)	0.838
CKD, n (%)	367 (60)	87 (73)	280 (57)	0.001
COPD, n (%)	50 (8)	7 (6)	43 (9)	0.312
Pacemaker/CRT, n (%)	76 (12)	28 (24)	48 (10)	< 0.001
Atrial fibrillation, n (%)	117 (19)	24 (20)	93 (19)	0.738
NYHA functional class, n (%)				
I	139 (23)	9 (8)	130 (26)	< 0.001
II	345 (56)	48 (40)	297 (60)	< 0.001
III	125 (20)	58 (49)	67 (14)	< 0.001
IV	5 (1)	4 (3)	1 (1)	0.006
Cardiac disease				
Coronary artery disease, n (%)	283 (46)	55 (46)	228 (46)	0.657
Dilated cardiomyopathy, n (%)	239 (39)	43 (36)	196 (40)	0.374
Hypertrophic cardiomyopathy, n (%)	25 (4)	9 (8)	16 (3)	0.032
Primary valvular disease, n (%)	19 (3)	4 (3)	15 (3)	0.740
Others, n (%)	47 (8)	8 (7)	39 (8)	0.666
Medical therapy				
ACE-I or ARB or ARNI, n (%)	384 (63)	73 (61)	311 (63)	0.752
Beta-blocker, n (%)	527 (86)	106 (89)	421 (85)	0.307
Aldosterone antagonist, n (%)	357 (58)	84 (71)	273 (55)	0.003
SGLT2-I, n (%)	188 (31)	39 (33)	149 (30)	0.581
Diuretics, (%)	292 (48)	85 (71)	207 (42)	< 0.001
Tolvaptan, n (%)	92 (15)	46 (39)	46 (9)	< 0.001

Data are presented as mean ± standard deviation or median (interquartile range) for continuous variables, and as numbers with percentages for categorical variables

ACE-I Angiotensin-converting enzyme inhibitor, *ARB* Angiotensin II receptor blocker, *ARNI* Angiotensin receptor–neprilysin inhibitor, *CKD* Chronic kidney disease, *COPD* Chronic obstructive pulmonary disease, *CRT* Cardiac Resynchronization Therapy, *NYHA* New York Heart Association, *SGLT2-I* Sodium–glucose cotransporter 2 inhibitor

or angiotensin receptor blockers, beta-blockers, and mineralocorticoid receptor antagonists. Additionally, nearly 48% of patients were prescribed diuretics.

Echocardiographic findings

Echocardiographic parameters are summarized in Table 2. The mean LV end-diastolic volume index was 87 ± 31 mL/m^2, while the mean LV end-systolic volume index was 56 ± 27 mL/m^2. The mean LV ejection fraction was 38 ± 9%. The LA volume index averaged 48 ± 19 mL/m^2, the mean E/e' ratio was 15.5 ± 8.0 and the mean TR peak velocity was 2.4 ± 0.4 m/sec. Regarding RV parameters, the mean RV end-diastolic area index was 12 ± 3 cm^2/m^2, RV end-systolic area index was 7 ± 2 cm^2/m^2, and RVFAC was 42 ± 7%. Severe mitral regurgitation was present in 49 patients, whereas severe TR was identified in 13 patients. The mean RA area was 17 ± 6 cm^2, the RA maximum volume index was 32 ± 18 mL/m^2, and the RASr was 21 ± 10%. Patients who experienced cardiac events demonstrated a reduced LV ejection fraction and RASr, along with increased LA volume index, RA maximum volume index.

Prognostic value of RA strain

During the median follow-up period of 41 months (range: 12–91 months), 119 of the 613 patients experienced cardiac events, including 50 cases of cardiac death and 69 hospitalizations for HF. Patients were stratified into two groups based on the median values of RASr, RA maximum volume index, and RA area. The median values were 20% for RASr, 28 mL/m² for RA maximum volume index, and 16 cm² for RA area. Kaplan–Meier

Table 2 Echocardiography parameters

Variables	All (*n* = 613)	Events		*p*-value
		Present (*n* = 119)	Absent (*n* = 494)	
LV end-diastolic volume, mL	144 ± 54	160 ± 64	139 ± 51	< 0.001
LV end-diastolic volume index, mL/m^2	87 ± 31	98 ± 35	84 ± 29	< 0.001
LV end-systolic volume, mL	93 ± 47	110 ± 56	89 ± 44	< 0.001
LV end-systolic volume index, mL/m^2	56 ± 27	67 ± 32	53 ± 25	< 0.001
LV ejection fraction, %	38 ± 9	34 ± 9	38 ± 9	< 0.001
LV mass index, g/m^2	123 ± 33	133 ± 34	120 ± 33	< 0.001
LA volume index, mL/m^2	48 ± 19	58 ± 25	46 ± 17	< 0.001
E/e' ratio	15.5 ± 8.0	19.6 ± 10.8	14.6 ± 6.8	< 0.001
TR peak velocity, m/sec	2.4 ± 0.4	2.5 ± 0.5	2.3 ± 0.4	< 0.001
Inferior vena cava diameter, mm	12 ± 5	14 ± 6	12 ± 5	0.002
RV end-diastolic area, cm^2	20 ± 5	21 ± 6	20 ± 5	0.084
RV end-diastolic area index, cm^2/m^2	12 ± 3	13 ± 4	12 ± 3	0.002
RV end-systolic area, cm^2	12 ± 4	13 ± 5	12 ± 4	0.017
RV end-systolic area index, cm^2/m^2	7 ± 2	8 ± 3	7 ± 2	0.003
RV fractional area change, %	42 ± 7	41 ± 8	43 ± 6	0.013
RA area, cm^2	17 ± 6	19 ± 8	17 ± 6	< 0.001
RA maximum volume, mL	53 ± 30	61 ± 35	51 ± 28	< 0.001
RA maximum volume index, mL/m^2	32 ± 18	38 ± 21	31 ± 16	< 0.001
RA minimum volume, mL	37 ± 27	46 ± 32	35 ± 25	< 0.001
RA minimum volume index, mL/m^2	23 ± 16	29 ± 19	21 ± 14	< 0.001
RA reservoir strain, %	21 ± 10	17 ± 9	22 ± 10	< 0.001
Mitral regurgitation				
Non/mild/moderate/severe, n (%)	196/206/162/49 (32/34/26/8)	22/40/37/20 (19/34/31/17)	174/166/125/29 (35/34/25/6)	< 0.001
Aortic regurgitation				
Non/mild/moderate/severe, n (%)	312/156/95/50 (51/25/16/8)	49/35/22/13 (41/29/19/11)	263/121/73/37 (53/25/15/7)	0.199
Aortic stenosis				
Non/mild/moderate/severe, n (%)	604/1/3/5 (99/0.1/0.4/0.8)	116/1/2/0 (97/1/2/0)	488/0/1/5 (99/0/0.2/1)	0.071
Tricuspid regurgitation				
Non/mild/moderate/severe, n (%)	359/84/157/13 (58/14/26/2)	58/33/22/6 (49/28/18/5)	301/51/135/7 (61/10/27/1)	< 0.001

Data are presented as mean ± standard deviation or numbers with percentages

E/e' early diastolic mitral inflow velocity to mitral annular velocity ratio, *LA* left atrial, *LV* left ventricular, *RA* right atrial, *RV* right ventricular

survival analysis revealed that a reduced RASr (≤ 20%), and increased RA maximum volume index (≥ 28 mL/m^2) were significantly associated with the occurrence of cardiac events (log-rank test, $p < 0.001$, $p = 0.016$, respectively; Fig. 3A and B). In contrast, no significant difference in event-free survival was observed between groups stratified by RA area (log-rank test, $p = 0.134$; Fig. 3C). Furthermore, Kaplan–Meier survival curves demonstrated that patients with RASr ≤ 20% exhibited significantly worse outcomes, even without RA volume enlargement (log-rank test, $p < 0.001$; Fig. 4).

Univariate analysis identified age, NYHA functional class III or IV, chronic kidney disease, LV end-diastolic volume index, LV ejection fraction, LA volume index, E/e' ratio, TR peak velocity, moderate or severe TR, RVFAC, RA area, RA maximum volume index and RASr as factors associated with cardiac events (Table 3). However, in the multivariable Cox proportional hazards regression model, only age, NYHA functional class III or IV, AF, LV ejection fraction, LA volume index E/e' ratio and RASr remained independently associated with cardiac events, whereas RVFAC and RA maximum volume index were not independently predictive (Table 3).

A likelihood ratio test was performed by comparing a model including LA volume index alone (χ^2 = 31.7) with an extended model incorporating both LA volume index and RASr (χ^2 = 49.4). The chi-square difference was 17.7 with 1 degree of freedom ($p < 0.001$), indicating that the addition of RASr significantly improved the prognostic model compared with LA volume index alone (Fig. 5).

Reproducibility

There was good agreement in the measurements of RA maximum volume between the two blinded observers ($r = 0.95$, $p < 0.01$) and the measurements obtained by the same observer at the different time points ($r = 0.97$, $p < 0.01$). There was also good agreement for measurements of RASr between the two blinded observers ($r = 0.93$, $p < 0.01$) and for measurements obtained by the same observers at the different time points ($r = 0.96$, $p < 0.01$).

Discussion

This study represents the first comprehensive investigation into the relationship between RA reservoir function measured by 2D-STE and prognosis in a large cohort of patients with chronic HF and LV dysfunction. The findings demonstrate that decreased RASr, and increased RA maximum volume were significantly associated with adverse cardiac events in patients with chronic HF. Furthermore, RASr was an independent predictor of these events and the model fit of conventional echocardiographic parameters for cardiac events prediction was systematically evaluated.

Previous studies have been demonstrated that RA reservoir function declines with elevated pulmonary artery pressure, increased RV filling pressure, and RV dysfunction [13, 14]. Additionally, chronic volume overload due to severe TR has been linked to reduced RA reservoir strain [15]. Furthermore, RA reservoir strain has been shown to reflect the severity of RV diastolic dysfunction [16]. Based on these findings, we propose that RA reservoir strain may serve as a novel indicator of HF, reflecting RV diastolic pressure and RA volume load. Although RV dysfunction is widely recognized as a poor prognostic factor in HF, it is often diagnosed at an advanced stage when right-side heart failure has already progressed irreversibly [17]. Therefore, the prognostic significance of RA reservoir function, in addition to RV function, is becoming increasingly evident as an early marker of mortality risk in HF patients [18]. In our study, although there were significant differences in RV enlargement and RVFAC between patients with and without cardiac events, the values remained within the normal range, and no deterioration in RV function was observed. These findings indicate that RASr serves as a reliable and early prognostic marker in patients before the onset of overt RV failure. To further address the potential confounding effect of TR, we performed a sensitivity analysis excluding patients with severe TR ($n = 13$). Although the number of patients with severe TR was small, the results were consistent with the main analysis, and reduced RASr remained a significant predictor of adverse cardiac events (log-rank test, $p < 0.001$).

Univariate analysis demonstrated that RA area and volume enlargement, reduced RASr, as well as increased LA volume index and E/e' ratio, were associated with adverse outcomes. In a multivariate analysis that included established HF indicators such as LV ejection fraction, LA volume index, E/e' ratio, RA volume and RVFAC, RASr remained an independent predictor, supporting its clinical relevance in risk stratification. Furthermore, the likelihood ratio test demonstrated that incorporating RASr into the prognostic model provided significant incremental value over LA volume index alone. These findings highlight the role of RASr as a complementary parameter to established LA indices, reinforcing its utility in comprehensive risk stratification in patients with chronic HF (Fig. 5). To further explore the impact of RA volume on the prognostic value of RASr, patients were stratified by RA maximum volume index into ≥ 28 mL/m^2 and < 28 mL/m^2. Kaplan–Meier analysis within each subgroup demonstrated that RASr ≤ 20% remained significantly associated with adverse cardiac events regardless of RA volume (log-rank test, $p < 0.001$ for both subgroups; Supplementary Figure). These showed that adverse cardiac events were observed in patients with reduced RASr irrespective of RA volume enlargement, indicating the

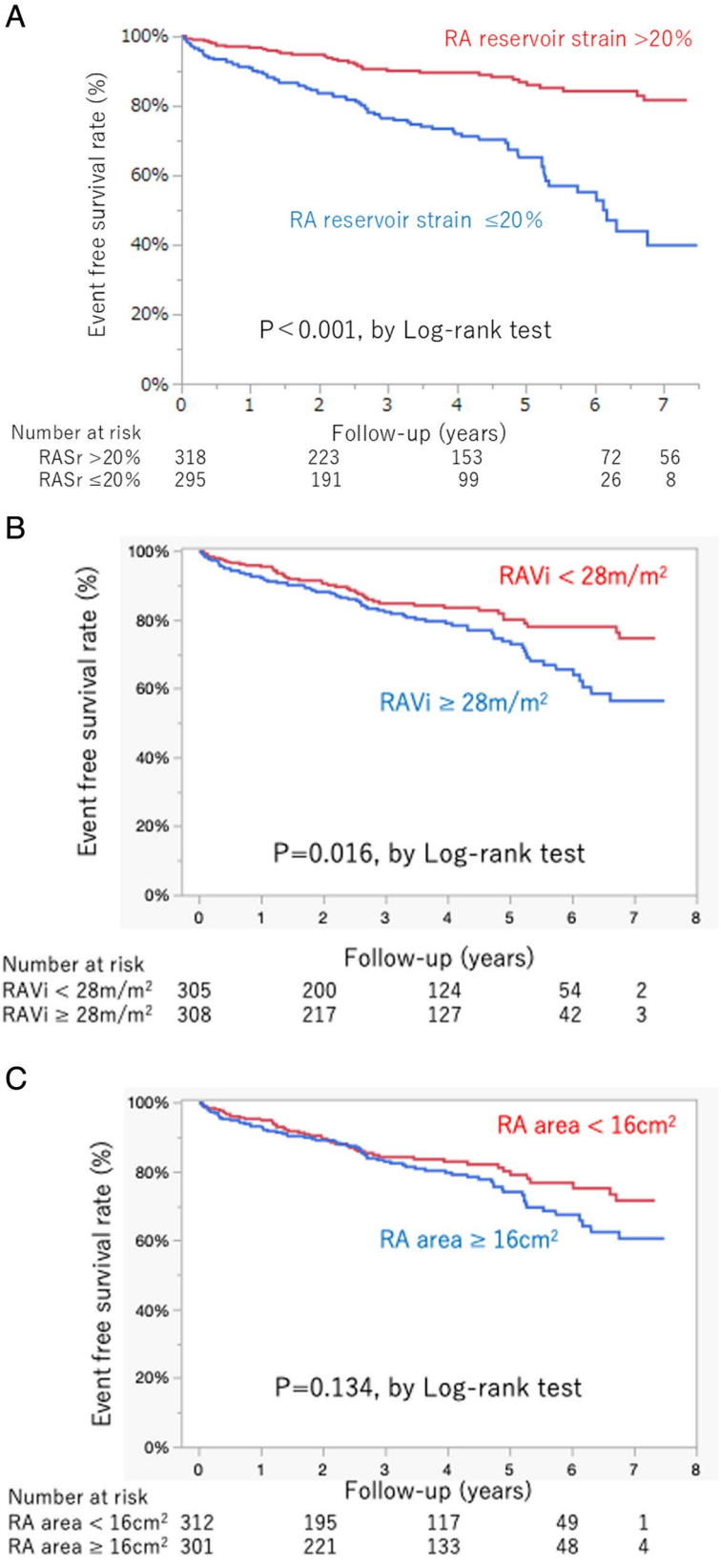

Fig. 3 Kaplan–Meier curve analysis of event-free survival rate based on RA reservoir strain (**A**), RA maximum volume index (**B**), and RA area (**C**). The endpoint was cardiac death or hospitalization for heart failure. RA reservoir strain ≤ 20% and RAVi ≥ 28 mL/m^2 were significantly associated with cardiac events. RA = right atrial, RASr = right atrial reservoir longitudinal strain, RAVi = right atrial maximum volume index

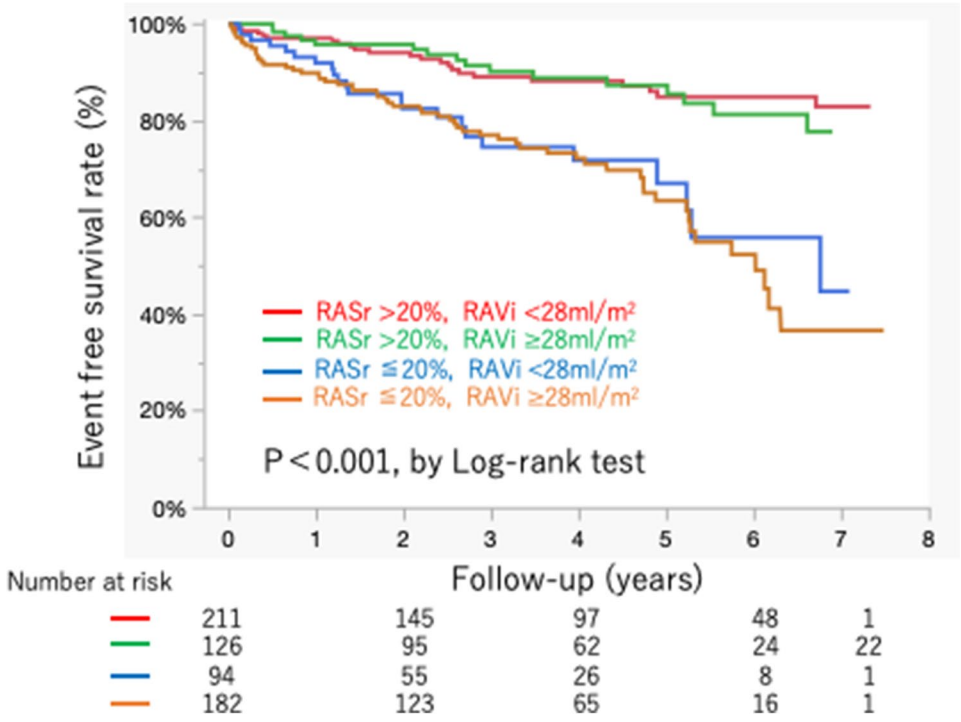

Fig. 4 Kaplan–Meier curve analysis of event-free survival rate based on the combination of RASr and RAVi. RASr = right atrial reservoir longitudinal strain, RAVi = right atrial maximum volume index

Table 3 Univariable and multivariable analysis for prediction of cardiac outcomes

	Univariate			Multivariate 1			Multivariate 2		
	HR	95% CI	*P* value	HR	95% CI	*P* value	HR	95% CI	*P* value
Age	1.03	(1.01, 1.04)	0.002	1.02	(1.03, 1.03)	0.043			
Male sex	1.17	(0.78, 1.74)	0.441	1.10	(0.74, 1.64)	0.644			
NYHA functional class III or IV	5.73	(3.98, 8.25)	< 0.001	3.84	(2.59, 5.69)	< 0.001			
CKD	2.10	(1.40, 3.15)	< 0.001	1.27	(0.83, 1.95)	0.278			
AF	1.10	(0.70, 1.73)	0.674	2.23	(1.34, 3.71)	0.002			
LV end-diastolic volume index, mL/m^2	1.01	(1.00, 1.01)	< 0.001						
LV ejection fraction, %	0.97	(0.96, 0.98)	< 0.001				0.97	(0.95, 0.99)	0.011
LA volume index, ml/m^2	1.02	(1.01, 1.03)	< 0.001				1.01	(1.00 1.02)	0.049
E/e' ratio	1.05	(1.04, 1.07)	< 0.001				1.04	(1.02, 1.07)	< 0.001
TR peak velocity, m/sec	1.04	(1.03, 1.06)	< 0.001				0.89	(0.54, 1.46)	0.645
Moderate or severe TR	2.81	(1.84, 4.31)	< 0.001				1.47	(0.87, 2.49)	0.148
RV end-diastolic area index, cm^2/m^2	1.02	(0.98, 1.05)	0.319						
RV end-systolic area index, cm^2/m^2	1.05	(0.99, 1.08)	0.050						
RV fractional area change, %	0.96	(0.94, 0.99)	0.005				1.00	(0.97, 1.03)	0.833
RA area, cm^2	1.05	(1.02, 1.07)	0.001						
RA maximum volume index, ml/m^2	1.02	(1.01, 1.02)	< 0.001				1.00	(0.98, 1.01)	0.741
RA reservoir strain, %	0.94	(0.92, 0.96)	< 0.001	0.94	(0.92, 0.96)	< 0.001	0.96	(0.94, 0.99)	0.002

Variables for multivariate analysis 1 included Age, Male, NYHA functional class III or IV, CKD, AF, RA reservoir. Variables for multivariate analysis 2 included LV ejection fraction, LA volume index, E/e' ratio, TR peak velocity, significant TR, RV fractional area change, RA maximum volume index, and RA reservoir strain. Per 1 unite increase in variables and per 1unite absolute decrease in LV ejection fraction and RA reservoir strain

AF atrial fibrillation, *CKD* chronic kidney disease, *CI* confidence interval, *E/e'* early diastolic mitral inflow velocity to mitral annular velocity ratio, *HR* hazard ratios, *LA* left atrial, *LV* left ventricular, *NYHA* New York Heart Association, *RA* right atrial, *RV* right ventricular, *TR* tricuspid regurgitation

incremental prognostic value of RA strain assessment beyond conventional RA volume evaluation.

Several studies have explored the association between RA function and prognosis in patients with pulmonary hypertension [19–23]. In our cohort of chronic HF patients with LV dysfunction, the mean RASr was 21 ± 10%, indicating marked RA dysfunction. Direct comparisons with pulmonary hypertension cohorts should

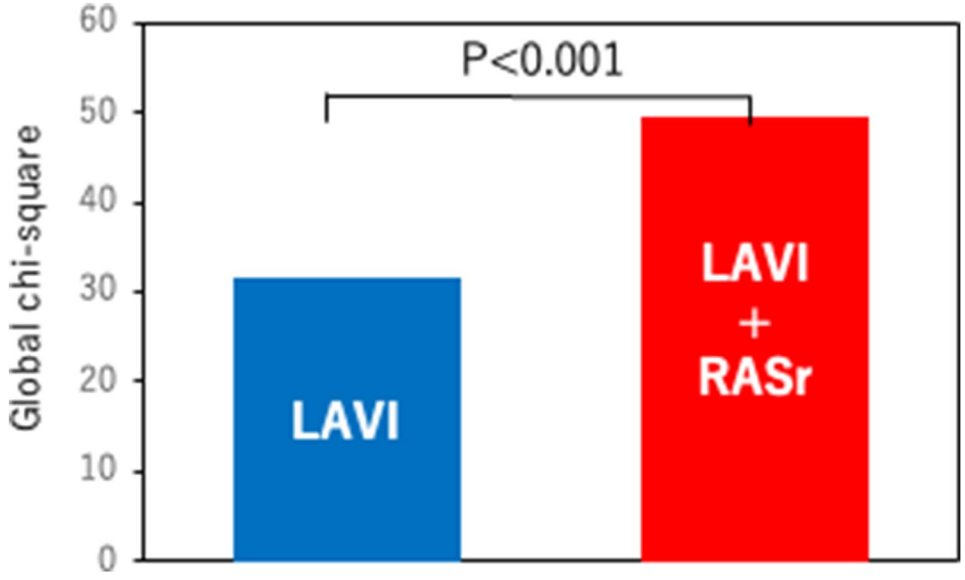

Fig. 5 Incremental prognostic value of RA reservoir strain. The addition of RA reservoir strain provided significant incremental prognostic information beyond LA volume alone. LA = left atrial, LAVI = left atrial volume index, RA = right atrial, RASr = right atrial reservoir longitudinal strain

be interpreted cautiously due to potential differences in patient characteristics and methodology. Furthermore, previous reports have been shown that reduced RA reservoir strain is associated with the new-onset and recurrence of AF, emphasizing the importance of RA reservoir strain as an indicator of atrial remodeling [24, 25]. On the other hand, following catheter ablation for AF, RA strain was associated with recurrence in univariable analysis but was not retained as an independent predictor in the multivariable model, suggesting that RA strain should be interpreted as part of a comprehensive assessment of cardiac function [26]. In our cohort, patients with AF had significantly lower RASr compared to those without AF (11.0 ± 6.5% vs. 23.7 ± 9.2%, $p < 0.001$), highlighting the close relationship between RA dysfunction and the presence of AF. Reduction in RA strain reflects functional impairment of the right atrium, which is closely associated with the degree of structural remodeling, particularly fibrosis and hypertrophy [27]. In chronic HF, sustained pressure and volume overload accelerate fibrosis, inflammation, and hypertrophy of the RA myocardium. Furthermore, the activation of systemic neurohumoral factors secondary to LV failure, such as the renin–angiotensin–aldosterone system and the sympathetic nervous system, is a well-recognized contributor to atrial fibrosis [28]. Taken together, these mechanisms emphasize that RA strain reflects multiple structural and functional determinants. It is also important to recognize that RASr is not a pure measure of atrial function alone but rather a composite parameter that integrates both active and passive contributors to RA physiology.

Clinical implications

The assessment of RA function in HF patients using 2D-STE remains relatively underexplored. Notably, no prior studies have evaluated RA strain in a cohort of chronic HF patients with LV dysfunction as large as that examined, in the present study. While LV function is typically the primary focus in HF, RA function plays a critical role in the pathophysiology of congestion and its management. The findings of the present study provide an incremental prognostic value of assessing RA reservoir function through strain analysis over conventional morphological parameters such as RA volume. Importantly, RASr appears to integrate multiple aspects of cardiac physiology and may allow earlier identification of patients at risk, before overt right-sided failure develops. Given the rising prevalence of HF patients, RA function is likely to garner increasing attention in both future research and clinical practice.

Study limitations

This study has several limitations. First, it was a single-center, retrospective analysis, which may limit the generalizability of the findings. Because of the retrospective design, some parameters considered important prognostic indicators in HF, such as LV global longitudinal strain and tricuspid annular plane systolic excursion, serum brain natriuretic peptide and hemoglobin levels were not available in all patients and could not be included in the analysis. Sodium-glucose cotransporter 2 inhibitors, which are now considered a cornerstone of HF therapy, were not widely used in our cohort because they were first approved for the treatment of HF in Japan in November 2020. Consequently, most patients had not

received sodium-glucose cotransporter 2 inhibitors at the time of enrollment. Additionally, approximately 8% of the study population had comorbid chronic obstructive pulmonary disease, although patients with severe chronic obstructive pulmonary disease enough to cause pulmonary hypertension were not included. Second, software specifically designed for RA strain analysis was not available at the time of the study, therefore, software originally developed for LA strain analysis was adapted for RA assessment. RA volume was assessed using a single-plane method, which should be considered when interpreting RA measurements. We included patients with optimal echocardiographic image quality, leading to the exclusion of those with suboptimal images. This may have introduced a selection bias, thereby limiting the applicability of our findings to the broader population of HF patients. Finally, we did not perform external validation or establish a clinically applicable cut-off value for RASr. Further studies are warranted to confirm the reproducibility and generalizability of our findings.

Conclusions

Our findings demonstrate that patients who experienced adverse cardiac events exhibited reduced RASr and increased RA maximum volume. Furthermore, reduced RASr was independently associated with an increased risk of cardiovascular death and HF-related hospitalization in patients with chronic HF and LV dysfunction. These results indicate that RASr may serve as valuable prognostic marker for this patient population.

Abbreviations

AF	Atrial fibrillation
E/e'	Early diastolic mitral inflow velocity to mitral annular velocity
HF	Heart failure
LA	Left atrial
LV	Left ventricular
NYHA	New York Heart Association
RA	Right atrial
RASr	Right atrial reservoir longitudinal strain
RV	Right ventricular
RVFAC	Right ventricular fractional area change
STE	Speckle-tracking echocardiography
TR	Tricuspid regurgitation
TTE	Transthoracic echocardiography

Supplementary Information

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Supplementary Material 1. Subgroup analysis of the prognostic value of RA reservoir strain according to RA maximum volume index. Kaplan–Meier curves for patients with (A) RAVi ≥ 28 mL/m^2 and (B) RAVi < 28 mL/m^2. In both subgroups, RA reservoir strain ≤ 20% was significantly associated with adverse cardiac events. RA = right atrial, RAVi = right atrial maximum volume index.

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Authors' contributions

All authors contributed to the conception of the study and participated in discussions throughout the research. RN, YT, MN, TN, NT, MT and KN collected the data. RN and MN performed the statistical analysis. RN wrote the main manuscript text and prepared all figures and tables. All authors reviewed the manuscript.

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Data availability

The data presented in this study are available on request from the corresponding author.

Declarations

Ethics approval and consent to participate

This study was approved by ethical committee of Okayama University (The ethical code: 2404-052).

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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