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Relationship Between Left Atrial Coupling Index and Atrial High-Rate Episodes

Ayca Arslan  | Dogan Ilis  | Inanç Artac  | Muammer Karakayali  | Timor Omar  | Yuksel Erata  | Yavuz Karabag  | Ibrahim Rencuzogullari 

Department of Cardiology, Kafkas University School of Medicine, Kars, Turkey

Correspondence: Ayca Arslan (hivda.arslan@hotmail.com)

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ABSTRACT

Objective: This study aimed to explore the role and compare the accuracy of the left atrial coupling index (LACI) with other efficient traditional parameters for predicting atrial high-rate episodes (AHREs) in patients with cardiac implantable electronic devices (CIEDs).

Methods: This retrospective study included 203 patients with CIEDs whose echocardiographic data were recorded at least 6 months ago. The study population was divided into two groups based on whether the patients had developed AHRE. Subsequently, patients in the two groups were compared in terms of echocardiographic measurements, including left atrial functions, LACI, left atrial volume index (LAVI), left ventricle end-diastolic diameter (LVEDD), and lower left ventricle ejection fraction (LVEF).

Results: AHRE was observed in 42.8% ($n = 87$) of the 203 patients. Those with AHRE had a higher mean mitral E/Em, LAVI, LACI, LVEDD, and lower LVEF. LACI (OR: 1.752, 95% CI: 1.356–2.263; $p < 0.001$), LAVI, and age were found to be independent predictors of AHREs. A receiver operating characteristic (ROC) curve comparison demonstrated that the LACI was a better predictor of AHRE than the LAVI.

Conclusion: AHRE often progresses to atrial fibrillation, which has unfavorable consequences; therefore, predicting AHRE becomes vital. LACI, an easily accessible echocardiographic parameter, is a more robust predictor of AHRE than traditional parameters such as LAVI.

1 | Introduction

Subclinical arrhythmic events such as atrial high-rate episodes (AHREs)—detected by pacemakers, implantable cardioverter-defibrillators (ICDs), and cardiac resynchronization therapy devices—are increasingly recognized as markers of cardiovascular risk. Even in the absence of a prior history of atrial fibrillation

(AF), patients with AHREs have an increased risk of developing clinical AF, leading to an increased likelihood of adverse outcomes, such as ischemic stroke and systemic thromboembolism. AHREs reflect underlying atrial remodeling processes, including fibrosis and electrical instability, which predispose the atrium to both arrhythmogenesis and thrombus formation. Therefore, AHREs' detection enables early risk stratification

Abbreviations: AF, atrial fibrillation; AHRE, atrial high-rate episode; CIED, cardiac implantable electronic device; ICD, implantable cardioverter-defibrillator; LACI, left atrial coupling index; LAVI, left atrial volume index; LVEF, left ventricular ejection fraction; RVFAC, Right ventricular fractional area change; TDI, tissue Doppler imaging.

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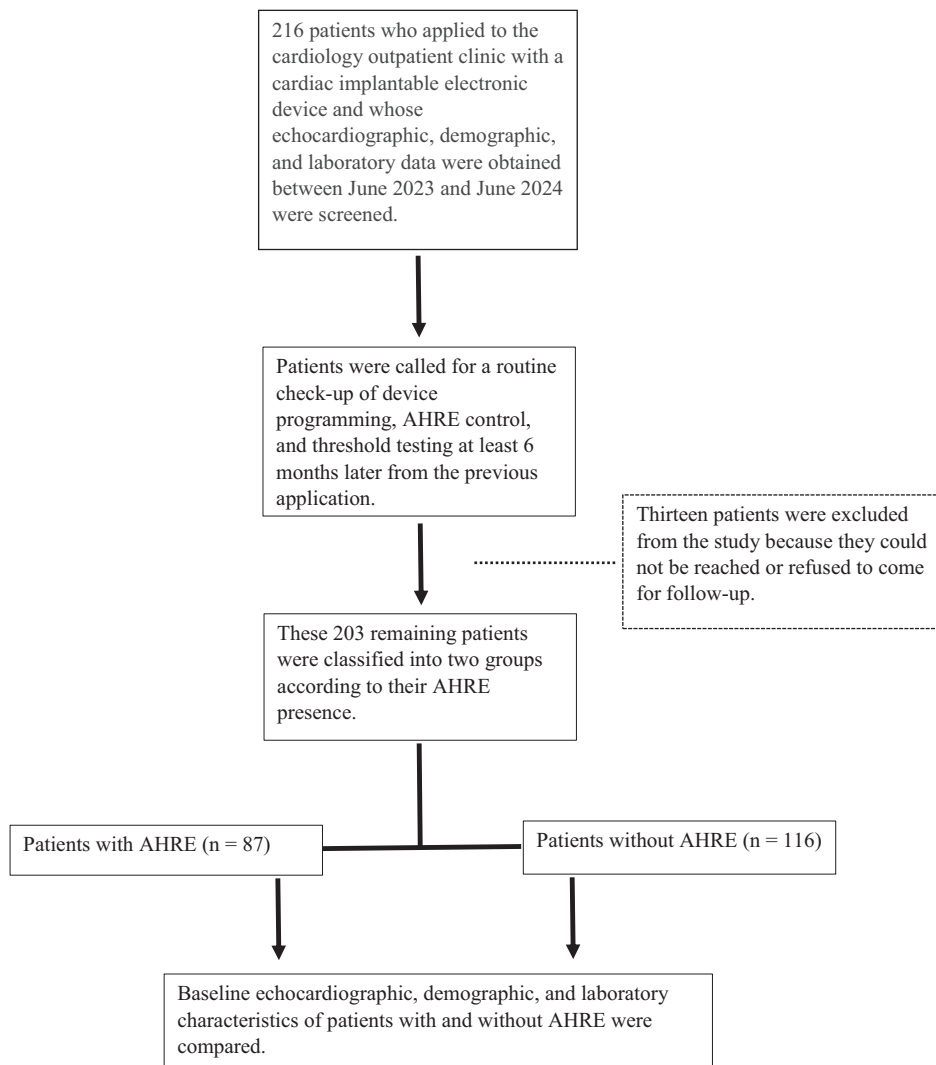


FIGURE 1 | Flowchart illustrating the study design and patient selection process.

and intervention to prevent severe cardiovascular complications [1, 2].

Various echocardiographic measurements, including left atrial dilatation and increased left atrial volume index (LAVI), have been identified as predictors of AHRE development and persistence thus far. However, the left atrial coupling index (LACI) is a novel parameter that integrates both volumetric and mechanical properties of the left atrium. To date, several studies have demonstrated that high LACI values are strongly associated with heart failure, cardiovascular mortality, and poor prognoses in patients with dyspnea or acute coronary syndrome. Moreover, high values of LACI have also strongly predicted mortality in patients with mitral valve prolapse. Given its prognostic value, LACI is increasingly recognized as a critical marker for risk stratification in various cardiovascular conditions [3–5]. However, the association between LACI and AHRE remains underexplored.

Considering AHRE's potential role in atrial arrhythmias and AF, this study aimed to evaluate the association between LACI and AHREs in patients with cardiac implantable electronic devices (CIEDs).

2 | Methods

Patients ($n = 216$) with CIEDs who had been admitted to the cardiology outpatient clinic between June 2023 and June 2024 were screened, and their echocardiographic, demographic, and laboratory data were collected. They were scheduled for a routine follow-up visit at least 6 months after their previous evaluation to assess device programming, AHREs, and threshold testing. Thirteen patients were excluded because either they could not be reached or they refused to attend the follow-up visit. The remaining 203 patients were classified into the following two groups: patients with AHRE ($n = 87$) and those without AHRE ($n = 116$) (Figure 1). The baseline echocardiographic, demographic, and laboratory characteristics of the two groups were then compared. The local ethics committee approved the protocol of this study in accordance with the Declaration of Helsinki.

2.1 | Definition of AHRE

AHREs refer to instances of atrial tachyarrhythmia, which are characterized by atrial rates surpassing 190 beats per min and

lasting for 30 s or more when detected by CIEDs [6]. The AHRE images were analyzed by an experienced electrophysiologist thoroughly, excluding lead noise or artifacts.

2.2 | Imaging Protocol

The Philips EPIQ 7c echocardiography device had been used to evaluate the transthoracic echocardiography (TTE) at the index application. Philips EPIQ 7c's EchoPAC software was used for offline measurements performed according to the recommendations of the American Society of Echocardiography [7, 8].

The left ventricular end-diastolic and end-systolic volumes, measured in apical four- and two-chamber views, were used to calculate the left ventricular ejection fraction (LVEF) using Simpson's rule.

Left atrial volume (LA volume), which presents the largest LA size, was measured in apical four- and two-chamber views at the ventricular end-systole using the biplane method of disks (modified Simpson's rule).

By dividing the left atrial volume by the body surface area, LAVI was determined.

2.2.1 | LACI

This parameter was determined by the ratio of LAVI to the a'-tissue Doppler imaging (TDI), as measured from the atrial side of the medial mitral annulus.

2.2.2 | Velocities of Transmitral Inflow

To evaluate this parameter, peak early (E) and peak late (A) velocities in the apical four-chamber view were measured using pulsed-wave Doppler echocardiography, and the E/A ratio was also computed.

2.2.3 | Mitral Annular Diastolic Velocities

The E/Em ratios for both walls were computed after TDI (Em, Am) at the lateral and septal sites.

2.2.4 | Right Ventricular Fractional Area Change (RVFAC)

Using two-dimensional echocardiography, RVFAC was assessed in the apical four-chamber view targeted at the right ventricle. Right ventricular systolic function was indicated by RVFAC, which is the percentage change in the right ventricular area between diastole and systole. The formula used for measuring it was as follows:

$$\text{RVFAC} = (\text{end-diastolic RV area} - \text{end-systolic RV area}) / \text{end-diastolic RV area} \times 100$$
 was the formula used to compute it.

2.2.5 | Right Ventricular Myocardial Velocities

Using pulsed-wave Doppler echocardiography, myocardial velocities (E' , A' , and S') from the tricuspid annular plane at the level of the right ventricular free wall's basal segment were also measured.

Echocardiographic measurements were performed by a single experienced cardiologist blinded to the clinical data.

2.3 | Statistical Analysis

The statistics were analyzed using SPSS version 22.0 (SPSS Inc., Chicago, IL). Descriptive statistics for continuous variables with a normal distribution were displayed in terms of mean and standard deviation, while those without a normal distribution were displayed as median and interquartile ranges. Categorical variables were represented in terms of percentages. The Kolmogorov-Smirnov test was employed to verify whether the continuous variables were normal. Depending on the data distribution, the Mann-Whitney U test or the two-sample Student's t -test was used to examine differences among the patients in the two groups. The variables identified as significant during the univariate analysis were further evaluated using multivariate analysis. Each independent variable was assigned a hazard ratio (HR) with a 95% confidence interval. The optimal cut-off values for predicting the development of AHRE in CIED patients were determined using receiver operating characteristic (ROC) curves and the corresponding area under the curve (AUC) values. p values less than 0.05 were considered statistically significant.

3 | Results

In this study, the mean age of the 203 patients was 66 ± 13 years, and 41.8% of them were females. Based on the presence of AHREs, patients were divided into the following two groups: AHRE (+) (with AHREs; $n = 87$, 42.8%) and AHRE (−) (without AHREs; $n = 116$, 57.1%). Higher hypertension (HT) rates were observed in the AHRE (+) group. Table 1 displays the patients' baseline characteristics and laboratory values.

The AHRE (+) group had significantly higher left-sided metrics than the AHRE (−) group, including E/Em mean ($9.5 [6.9-12.7]$ vs. $11.7 [9.1-15.5]$; $p < 0.001$), LACI (6.8 ± 3.2 vs. 4.1 ± 1.6 ; $p < 0.001$), and LAVI ($45 \pm 9 \text{ mL/m}^2$ vs. $36 \pm 9 \text{ mL/m}^2$; $p < 0.001$). In addition, Em septal (5.1 ± 1.5 vs. 5.9 ± 1.9 ; $p = 0.004$), Am septal (7.5 ± 2.0 vs. 9.2 ± 2.5 ; $p < 0.001$), Em lateral (6.9 ± 1.9 vs. 8.0 ± 2.9 ; $p = 0.031$), and Am lateral (9.2 ± 3.1 vs. 10.4 ± 3.1 ; $p = 0.021$) were all markedly lower in patients with AHRE (+). Furthermore, for right-sided characteristics, the AHRE (+) group had substantially lower RVFAC (41 ± 12 vs. 44 ± 11 ; $p = 0.021$) and RVS' ($11.0 \pm 2.83 \text{ cm/s}$ vs. $11.8 \pm 2.7 \text{ cm/s}$; $p = 0.044$) values than the AHRE (−) group. Table 2 shows the distribution of the patients' echocardiogram results.

Using variables $p < 0.05$ in univariate analysis (age, HT, E, Em septal, Am septal, Am lateral, Em lateral, E/Em mean mitral, LAVI, LACI, LVEDD, LVEF, RVFAC, and RV S'), logistic regression analyses were performed to find independent predictors of

TABLE 1 | Demographic, clinical, and laboratory characteristics of all patients with AHRE (–) and AHRE (+) group, with *p* value.

	AHRE (–) (<i>n</i> : 116)	AHRE (+) (<i>n</i> : 87)	Total patients (<i>n</i> : 203)	<i>p</i> value
Age (years)	64 ± 12	70 ± 13	66 ± 13	0.001
Female gender, <i>n</i> (%)	44 (37.9)	41 (47.1)	85 (41.8)	0.190
Body surface area (kg/m ²)	1.84 ± 0.14	1.83 ± 0.15	1.83 ± 0.15	0.545
Hypertension, <i>n</i> (%)	62 (53.4)	63 (72.4)	125 (61.5)	0.006
Diabetes mellitus, <i>n</i> (%)	31 (26.7)	32 (36.7)	63 (31.0)	0.126
CAD, <i>n</i> (%)	80 (68.9)	68 (78.1)	148 (72.9)	0.146
Stroke, <i>n</i> (%)	7 (6.0)	10 (11.4)	17 (8.3)	0.166
Smoking, <i>n</i> (%)	22 (18.9)	13 (14.9)	35 (17.2)	0.454
ASA, <i>n</i> (%)	55 (47.4)	39 (44.8)	94 (46.3)	0.715
P2Y12 inh, <i>n</i> (%)	12 (10.3)	11 (12.6)	23 (11.3)	0.610
ACEi, <i>n</i> (%)	62 (53.4)	45 (51.7)	107 (52.7)	0.808
ARB, <i>n</i> (%)	36 (31.0)	30 (34.4)	66 (32.5)	0.605
Antiarrhythmic treatment, <i>n</i> (%)	7 (6.0)	8 (9.1)	15 (7.3)	0.395
Beta-blocker, <i>n</i> (%)				
Bisoprolol, <i>n</i> (%)	8 (6.8)	8 (9.1)	16 (7.8)	0.823
Metoprolol, <i>n</i> (%)	51 (43.9)	42 (48.2)	93 (45.8)	
Nebivolol, <i>n</i> (%)	3 (2.5)	5 (5.7)	8 (3.9)	
Carvedilol, <i>n</i> (%)	22 (18.9)	11 (12.6)	33 (16.2)	
Statin (%)	59 (50.8)	42 (48.2)	102 (50.2)	0.716
SGLT-2 inh, <i>n</i> (%)	22 (18.9)	11 (12.6)	33 (16.2)	0.228
Loop diuretic, <i>n</i> (%)	38 (32.7)	34 (39.0)	72 (35.4)	0.353
MRA (%)	53 (45.6)	38 (43.6)	91 (44.8)	0.776
Device type, <i>n</i> (%)				
PM, <i>n</i> (%)	56 (48.2)	46 (52.8)	102 (50.2)	0.682
ICD, <i>n</i> (%)	33 (28.4)	20 (22.9)	53 (26.1)	
CRT, <i>n</i> (%)	27 (23.2)	21 (24.1)	48 (23.6)	
WBC (10 ³ /μL)	7.7 ± 2.7	8.0 ± 2.1	7.8 ± 2.5	0.156
Neutrophil (10 ³ /μL)	5.2 ± 2.3	5.55 ± 2.07	5.38 ± 2.20	0.154
Hemoglobin (g/dL)	13.1 ± 1.8	13.26 ± 1.70	13.18 ± 1.79	0.654
Glucose (mg/dL)	130 ± 57	138 ± 62	133 ± 59	0.464
Sodium (mmol/L)	140 ± 3	139 ± 4	139 ± 3	0.846
Potassium (mmol/L)	4.4 ± 0.4	4.5 ± 0.5	4.5 ± 0.4	0.168
Calcium (mmol/L)	9.4 ± 0.7	9.4 ± 0.6	9.4 ± 0.7	0.729
Magnesium (mmol/L)	1.9 ± 0.3	2.0 ± 0.3	2.0 ± 0.3	0.370
eGFR (mL/dk/m ²)	66 ± 21	68 ± 20	67 ± 21	0.237
CRP (mg/L)	5.4 (1.9–10.8)	3.9 (1.5–8.8)	5.0 (1.5–10.0)	0.307
Serum albumin (g/L)	40 ± 4	40 ± 4	40 ± 4	0.852
Uric acid (mg/dL)	5.9 ± 2.1	6.5 ± 2.3	6.1 ± 2.2	0.247
TSH (mIU/L)	1.3 (0.9–2.1)	1.2 (0.7–1.8)	1.3 (0.8–2.0)	0.205

Abbreviations: ACEi, angiotensin-converting-enzyme inhibitor; AHRE, atrial high rate episode; ARB, angiotensin receptor blocker; ASA, acetyl salicylic acid; CAD, coronary artery disease; CRP: C-reactive protein; CRT: cardiac resynchronization therapy; eGFR, estimated glomerular filtration rate; ICD, implantable cardioverter-defibrillator; MRA, mineralocorticoid receptor antagonist; PM, pacemaker; SGLT 2 inh, sodium glucose co-transporter 2 inhibitor; TSH: thyroid-stimulating hormone; WBC: white blood cell.

TABLE 2 | Echocardiographic parameters of all patients with AHRE (–) and AHRE (+) group, with *p* value.

	AHRE (–) (<i>n</i> : 116)	AHRE (+) (<i>n</i> : 87)	Total patients (<i>n</i> : 203)	<i>p</i> value
E, mitral (cm/s)	67 ± 23	74 ± 23	70 ± 23	0.028
A, mitral (cm/s)	76 ± 23	77 ± 29	76 ± 26	0.727
E/A, mitral	0.8 (0.6–1.2)	0.8 (0.6–1.4)	0.8 (0.6–1.3)	0.281
Em septal, mitral (cm/s)	5.9 ± 1.9	5.1 ± 1.5	5.5 ± 1.8	0.004
Am septal, mitral (cm/s)	9.2 ± 2.5	7.5 ± 2.0	8.4 ± 2.4	< 0.001
Em lateral, mitral (cm/s)	8.0 ± 2.9	6.9 ± 1.9	7.5 ± 2.6	0.031
Am lateral, mitral (cm/s)	10.4 ± 3.1	9.2 ± 3.1	9.9 ± 3.1	0.021
E/Em mean, mitral	9.5 (6.9–12.7)	11.7 (9.1–15.5)	10.3 (7.8–13.7)	< 0.001
LAVI (mL/m ²)	36 ± 9	45 ± 9	40 ± 10	< 0.001
LACI	4.1 ± 1.6	6.8 ± 3.2	5.3 ± 2.7	< 0.001
LVEDD (mm)	49 ± 8	51 ± 8	50 ± 8	0.019
LVEF, (%)	36 ± 6	34 ± 7	36 ± 7	0.009
RV S', (cm/s)	11.8 ± 2.7	11.0 ± 2.8	11.5 ± 2.8	0.044
RVFAC, (%)	44 ± 11	41 ± 12	43 ± 12	0.021

Abbreviations: A: peak late inflow velocity; AHRE, atrial high rate episode; Am, tissue Doppler myocardial velocity at atrial contraction; E, peak early inflow velocity; Em, early diastolic tissue Doppler velocity; LACI: left atrial coupling index; LAVI, left atrial volume index; LVEDD, left ventricle end-diastolic diameter; LVEF, left ventricle ejection fraction; RV S', right ventricle systolic (S') tissue Doppler velocity; RVFAC, right ventricle fractional area change.

TABLE 3 | Univariate and multivariate logistic regression analysis for predictors of AHRE (+).

Variable	Univariate			Multivariate		
	Odds ratio	95% CI	<i>p</i> value	Odds ratio	95% CI	<i>p</i> value
Age	1.041	1.016–1.066	0.001	1.032	1.004–1.061	0.026
HT	2.286	1.261–4.145	0.006			
E, mitral (cm/s)	1.012	0.999–1.024	0.061			
Em septal, mitral (cm/s)	0.770	0.648–0.914	0.003			
Am septal, mitral (cm/s)	0.716	0.620–0.826	< 0.001			
Em lateral, mitral (cm/s)	0.847	0.751–0.955	0.007			
Am lateral, mitral (cm/s)	0.882	0.803–0.969	0.009			
E/Em mean, mitral	1.066	1.011–1.124	0.019			
LAVI (mL/m ²)	1.111	1.071–1.152	< 0.001	1.051	1.005–1.100	0.031
LACI	1.921	1.566–2.537	< 0.001	1.752	1.356–2.263	< 0.001
LVEDD (mm)	1.028	0.995–1.063	0.101			
LVEF (%)	0.960	0.921–1.000	0.051			
RV S'	0.895	0.808–0.992	0.035			
RVFAC (%)	0.975	0.953–0.999	0.038			

Abbreviations: AHRE, atrial high rate episode; Am, tissue Doppler myocardial velocity at atrial contraction; E, peak early inflow velocity; Em, early diastolic tissue Doppler velocity; HT, hypertension; LACI, left atrial coupling index; LAVI, left atrial volume index; LVEF, left ventricle ejection fraction; RVFAC, right ventricle fractional area change.

AHRE (+). According to the multivariate analysis (Table 3), age (OR = 1.032; 95% CI, 1.004–1.061; *p* = 0.026), LAVI (OR = 1.051; 95% CI, 1.005–1.100; *p* = 0.031), and LACI (OR = 1.752; 95% CI, 1.356–2.263; *p* < 0.001) were independent predictors of AHRE in patients with CIEDs.

LACI had a higher AUC value for AHRE prediction than LAVI (0.829, *p* < 0.0001 vs. 0.772, *p* < 0.0001, respectively) (Figure 2). With a sensitivity of 81.6% and a specificity of 77.6%, the ideal cut-off value for LACI in AHRE prediction was more than 5.0146.

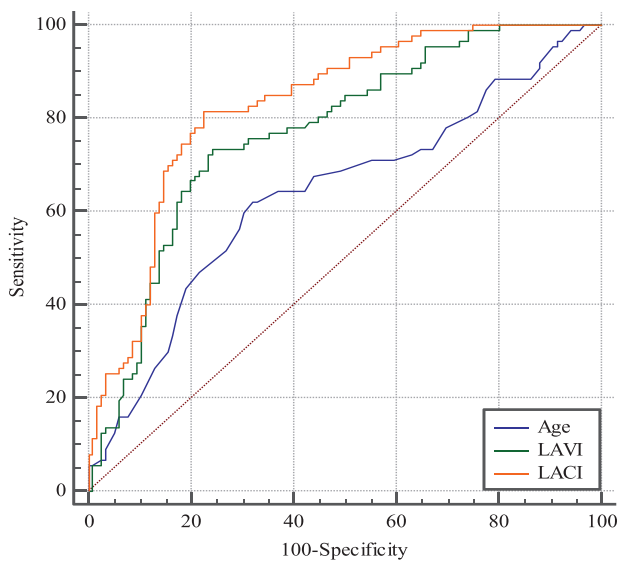


FIGURE 2 | ROC curve analysis for age, LACI, and LAVI for AHRE existence. LACI, left atrium coupling index; LAVI, left atrial volume index; ROC, receiver operating characteristic. [Color figure can be viewed at wileyonlinelibrary.com]

4 | Discussion

In patients with CIED, LACI was not only associated with AHRE but also played the role of its independent predictor. As an echocardiographic parameter, LACI provided a more efficient measurement for accurate AHRE prediction than LAVI.

AHREs detected through CIEDs have been consistently associated with major adverse cardiac events [9]. Notably, AHREs are detected in approximately 30%–70% of patients with CIED [10]. Moreover, AHREs become more frequent and tend to last longer over time, eventually leading to AF [11–13]. Also, it has been established that AHREs are associated with systemic embolism and stroke—especially in patients who have high CHA₂DS₂-VASc scores—and cardiovascular death [14, 15]. Given their frequency, progression to AF, and its relationship with adverse outcomes, early prediction of AHREs has become critically important for clinical risk stratification. Several factors, including advanced age, hypertension, LA enlargement, and congestive heart failure, have been identified as predictors of AHREs in patients with CIEDs [11, 16]. Consistent with previous studies, in our study, advanced age was associated with AHRE development [9, 16]. Advancing age causes increased instances of comorbid conditions, such as hypertension, which leads to an increased pressure load and wall tension in the atrium. This elevated pressure can contribute to atrial enlargement, a key factor in the development of AHREs. This process, along with the increased oxidative stress caused by aging, can cause damage to the atrial myocardium, creating a proarrhythmic environment that is conducive to the development of AHREs. The higher frequency of CIED implantation in older patients may also explain the increase in AHREs in this age group, as observed.

Hypertension is AHREs' well-established risk factor that leads to chronic strain on the heart, particularly in the left atrium [17].

In this study, patients with AHREs were found to have a higher prevalence of hypertension. Elevated blood pressure causes an increased afterload on the left ventricle, which is transmitted to the left atrium, thus resulting in LA dilation [18]. Over time, this dilation can lead to fibrosis and electrical disturbances in the atrium, which increases the likelihood of arrhythmias, such as AHREs. Furthermore, hypertension-induced atrial remodeling, including electrical changes, further predisposes patients to arrhythmia. In addition, chronic activation of the renin-angiotensin-aldosterone system (RAAS) in hypertensive patients may further exacerbate atrial remodeling and fibrosis, further contributing to the proarrhythmic environment. Such a combination of structural and electrical changes in the atrium of hypertensive individuals significantly increases the risk of AHREs. Therefore, effective management of hypertension is crucial in preventing AHREs and associated complications, such as stroke and AF.

Structural and functional changes in the heart are closely related to arrhythmia. One of the best diagnostic tools for evaluating these changes is echocardiography. To date, many AHRE-related echocardiographic measurements and parameters have been established. Consistent with previous studies, the results of this study showed that patients with AHREs had significantly higher LAVI and E/Em mean ratio values. Artaç et al. demonstrated that atrial arrhythmia burden was independently associated with alterations in left atrial (LA) and left ventricular (LV) diastolic functions, with elevated LA volumes linked to the development of AHREs [16]. Similarly, Tarek et al. showed that an LAVI value greater than 34.73 mL/m² was an independent predictor of AHREs in patients with CIEDs [19]. LA enlargement was primarily related to the volume overload of the left atrium, which is mostly associated with LV diastolic dysfunction. An important indicator of ventricular dysfunction was the E/Em mean ratio, which was significantly higher in patients with AHREs. Moreover, patients with AHREs were observed to have significantly lower RV S' and RVFAC values, indicating impaired right ventricular (RV) systolic function. Supporting the results of this study, Artaç et al. [16] investigated the relationship between RV function and AHREs and reported that RV S' was significantly associated with AHREs. AHREs may contribute to RV dysfunction through mechanisms such as increased pulmonary venous and arterial pressures, thus imposing a greater afterload on the RV and leading to systolic impairment.

In this study, LACI was shown to be an independent predictor of AHRE for the first time, even better than LAVI. This finding can be attributed to changes in the left atrium over time. The progress of LA dilation often causes fibrosis. Fibrotic tissue disrupts electrical conduction, promoting the formation of ectopic foci and micro-reentry loops, ultimately resulting in AHREs. In addition, fibrosis in the left atrium impacts its contraction, as evidenced by a low atrial tissue Doppler index (a'-TDI) value that reflects poor late diastolic contraction. Considering that AHREs emerge not only in left atrial anatomical enlargement but also in restricted movement of the left atrium, LACI was speculated to be its better predictor, as it merges both movement and volume changes. The ratio of LAVI to a'-TDI provides LACI's value, and a high LACI value indicates that both the volumetric and mechanical properties of the left atrium are impaired.

The progression of AHRE to AF is a complex process associated with both structural and functional worsening of the left atrium and ventricles. In this study, markers of LA remodeling, LV diastolic dysfunction, and RV systolic impairment were shown to be significantly associated with AHREs, emphasizing the role of a comprehensive cardiac evaluation in these patients. These findings contribute to a growing understanding of the pathophysiological mechanisms underlying AHREs and their potential progression to AF. Given that AHRE progresses to AF with unfavorable consequences, including heart failure and thromboembolic events, using strong predictors of AHRE remains vital. Although further multicenter validation studies are warranted, LACI, as a novel marker, may improve risk stratification and early treatment strategies, particularly in preventing thromboembolic events and optimizing arrhythmia management.

4.1 | Limitations

This study was not without limitations. First, while a multicenter, fully prospective, large-scale study may provide robust information, this was a single-center study involving a relatively small number of patients. Therefore, it may potentially cause a selection bias (e.g., device-specific detection thresholds for AHRE) that possibly affects generalizability. Second, the patients included in the study had different types of devices; therefore, the predictive power of the LACI for AHRE seems to be a general result for all CIEDs, without revealing details about the predictive power for individual device types. Third, patients did not have a clear interval of routine check-ups to determine whether they experienced AHRE. Even though all patients were checked at intervals longer than 6 months, these intervals were not exactly equal.

5 | Conclusion

Considering that AHRE progresses to AF with ominous consequences, its prompt detection remains vital. LACI simultaneously evaluates both the anatomical and functional properties; hence, it may play a pivotal role in predicting AHREs and mitigating its transition to AF and atrial arrhythmia.

Author Contributions

Ayca Arslan and Dogan Ilis have given substantial contributions to the conception or the design of the manuscript. Timor Omar, Muammer Karakayali, and Yuksel Erata have given substantial contributions to the collection of the data. Inanc Artac and Yavuz Karabag have participated mostly in statistical data analysis. Ayca Arslan and Ibrahim Rencuzogullari have participated in the design, analysis, and drafting of the manuscript. All authors have participated in data analysis and drafting the manuscript. All authors read and approved the final version of the manuscript. All authors contributed to the manuscript and read and approved the final version of the manuscript. All of the authors contributed to the planning, conduct, and reporting of the work. All contributors are responsible for the overall content as guarantors.

Ethics Statement

The study protocol was approved by the local ethics committee (Ethics Committee of the Dean of the Faculty of Medicine of Kafkas University—80576354-050-99/613 numbered ethics committee approval).

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The authors have nothing to report.

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