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RESEARCH ARTICLE

Left atrial remodeling in epilepsy: A comparative echocardiographic study of left atrial emptying fraction and left atrial volumetric index in patients with epilepsy

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Abstract

Objective: Patients with epilepsy (PWE) are at increased risk for cardiac abnormalities, including arrhythmias, structural changes, and sudden unexpected death in epilepsy (SUDEP). Although previous studies have examined ventricular dysfunction, left atrial (LA) structural and mechanical changes remain underexplored. This study aimed to evaluate LA remodeling in PWE using echocardiographic markers—LA volume index (LAVI), LA emptying fraction (LAEF), and E/e′ ratio—as well as to examine their associations with SUDEP-7 scores.

Methods: This prospective, cross-sectional study included 58 PWE and 49 healthy controls. Participants underwent transthoracic echocardiography at least 24 h after a seizure. LAEF and LAVI were calculated using the biplane area–length method. PWE were further stratified into two groups: medically controlled epilepsy (MCE) and drug-resistant epilepsy (DRE). The risk of SUDEP was assessed using the SUDEP-7 inventory.

Results: PWE exhibited significantly higher maximum LAVI ($p = .047$), lower total LAEF ($p = .001$), and reduced LA active emptying fraction ($p = .001$), alongside increased LA passive emptying fraction ($p = .028$) when compared to the control group. However, no significant differences were noted in the E/e′ ratio. Furthermore, maximum LAVI demonstrated a positive correlation with SUDEP-7 scores ($r = .265$, $p = .044$). Patients with DRE exhibited significantly higher SUDEP-7 scores and longer disease duration in comparison to MCE.

Significance: This study reveals that reduced LAEF and elevated LAVI may serve as early, underrecognized markers of LA remodeling in chronic epilepsy—changes that are not captured by conventional diastolic indices such as the E/e′ ratio. Given their strong association with SUDEP-7 scores, LAEF and LAVI show promise as novel echocardiographic biomarkers for identifying individuals at high risk for cardiovascular events and SUDEP.

KEYWORDS
echocardiograpy, epilepsy, epileptic heart, left atrial emptying fraction, left atrial volumetric index, sudden unexpected death in epilepsy

1 | INTRODUCTION

Extensive clinical and population-based studies have shown that chronic epileptic seizures adversely affect both electrical and mechanical cardiac function, thereby increasing the risk of cardiovascular events, including myocardial infarction, arrhythmias, and sudden unexpected death in epilepsy (SUDEP).[1–3] The findings presented herein substantiate the concept of the "epileptic heart," characterized by myocardial and vascular damage induced by seizures, mediated through recurrent surges of catecholamines and episodes of hypoxia.[1] Although arrhythmias, cardiovascular diseases, and heart rate variability are frequently highlighted in the literature, it is essential to acknowledge the significant insights gained from studying structural changes in the heart. Advanced echocardiographic investigations have demonstrated that chronic epilepsy is linked to stress-induced cardiomyopathy, which results from recurrent sympathetic activation and catecholamine release. This condition contributes to myocardial remodeling, diastolic dysfunction, and increased risk of heart failure and arrhythmias.[1,2]

The left atrium (LA) plays a pivotal role in modulating left ventricular (LV) filling and the maintenance of optimal cardiac output, operating through reservoir, conduit, and contractile phases. The LA volumetric index (LAVI) is a standardized echocardiographic measurement of LA size, normalized for body surface area (BSA), and serves as a reliable surrogate marker for chronic LV diastolic burden and sustained elevated LA pressure.[4,5] LAVI is commonly elevated in various cardiovascular and systemic conditions, including hypertension, heart failure, atrial fibrillation, mitral valve disease, diastolic dysfunction, chronic kidney disease, and diabetes mellitus. Consequently, LAVI is acknowledged as a significant echocardiographic biomarker, serving not only for diagnostic stratification but also for longitudinal risk assessment in both populations with and populations without overt cardiovascular disease.[6] The LA emptying fraction (LAEF) is a dynamic echocardiographic parameter that evaluates the contractile function of the LA and its role in ventricular filling. This measure is often diminished in pathological conditions such as atrial fibrillation and hypertensive heart disease, frequently occurring prior to the onset of structural alterations, including atrial enlargement. The LAEF provides an assessment of the contractile performance of the LA, particularly during late diastole, thereby serving as a dynamic indicator of atrial mechanical function.[7] LAVI and LAEF function as integrated indicators of LA health, with LAVI representing chronic structural remodeling and LAEF denoting real-time mechanical performance.[5]

Key points

- LAEF is significantly reduced in patients with epilepsy, suggesting early left atrial dysfunction.
- Elevated LAVI levels in epilepsy patients indicate chronic left atrial remodeling.
- Higher LAVI correlates with SUDEP-7 scores, suggesting it may be a risk marker for SUDEP.

In studies utilizing echocardiography in patients with epilepsy (PWE), the findings most frequently indicate an increase in LV stiffness and diastolic dysfunction, a reduction in global myocardial strain, and subtle decreases in LV ejection fraction.[2,3,8,9] However, the structural and functional alterations in the LA have not been thoroughly examined. Dysfunction of the LA, which is characterized by modified volume indices or diminished emptying efficiency, may serve as an indicator of early subclinical cardiovascular remodeling and could potentially affect the clinical course or response to therapeutic interventions. The aim of this study was to compare LAVI and LAEF between PWE and a control group, as well as within the medically controlled epilepsy (MCE) and drug-resistant epilepsy (DRE) subgroups of the PWE.

2 | MATERIALS AND METHODS

In this study, 58 epilepsy patients who were followed up at a neurology outpatient clinic and 49 healthy controls from the hospital staff were included. The participants' ages ranged from 18 to 55 years, and they were enrolled in this cross-sectional prospective study. The classification criteria were established in accordance with the International League Against Epilepsy (ILAE) guidelines. Exclusion criteria included individuals with a documented history of cardiac disease, clinically diagnosed sleep disorders, intellectual disabilities, coexisting neurological or psychiatric disorders, systemic illnesses such as hypertension, diabetes mellitus, cerebrovascular disease, and anemia, as well as those receiving treatment with

antiarrhythmic medications. This research was conducted at the Department of Neurology, Kirsehir Training and Research Hospital, from January 2024 to September 2024, in adherence to the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Kirsehir Ahi Evran University Health Sciences Scientific Research Ethics Committee on June 6, 2023, under approval number 2023–11/70.

Demographic information and clinical characteristics pertinent to seizures were systematically recorded, encompassing the duration of epilepsy, seizure type and frequency, routine electroencephalographic (EEG) findings, and current usage of antiseizure medications (ASMs). Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (BMI = weight [kg]/height^2 [m^2]). BSA was calculated using the formula: BSA = .007184 × height (cm).725 × weight (kg).425. The cohort of individuals with epilepsy was stratified into two primary subgroups: those with DRE and those with MCE. In accordance with the criteria established by the ILAE, DRE is defined as the failure to achieve sustained seizure freedom following adequate trials of two well-tolerated, appropriately selected, and properly administered ASMs, whether utilized individually or in combination.[10] Conversely, patients who had maintained seizure-free status for more than 1 year while undergoing pharmacological treatment were classified as having MCE. Seizures were categorized as focal, generalized, or of unknown origin, based on ILAE diagnostic and classification standards. EEG findings were similarly classified into four categories: normal, nonspecific findings, focal abnormalities, and generalized abnormalities.

The risk of SUDEP was evaluated utilizing the SUDEP-7 inventory, a validated instrument that incorporates both clinical and electroclinical characteristics to estimate the individual risk of SUDEP.[11] The SUDEP-7 score for each participant was determined using the available clinical and seizure-related data. The total score was documented on a scale from 0, indicating the lowest risk, to 12, representing the highest risk.

2.1 | Echocardiography

Transthoracic echocardiography was conducted in both the epilepsy and control groups, adhering to standardized imaging protocols in accordance with the guidelines established by the American Society of Echocardiography.[12] All examinations were conducted with participants positioned in the left lateral decubitus position, utilizing an Artida echocardiography system (Toshiba Medical Systems). Standard apical four-chamber and two-chamber views were obtained and optimized to enhance endocardial definition. To reduce autonomic variability, all echocardiographic assessments were performed during an interictal period of <24 h following the most recent seizure. All echocardiographic evaluations were executed and interpreted by a single board-certified cardiologist who remained blinded to the clinical classification of the epilepsy subgroups.

LAVI was calculated using the biplane area–length method at ventricular end-systole, which corresponds to the maximal size of the LA. The LA volume was obtained by tracing the endocardial borders in both apical views, while excluding the pulmonary veins and LA appendage. This volume was subsequently indexed to BSA and expressed in mL/m^2. The preatrial LAVI was calculated as the LA volume measured immediately prior to mitral valve closure (end-diastole), employing the same biplane area-length method and indexed to BSA (mL/m^2). The minimum LA volume (LAmin) was assessed at end-diastole, directly following mitral valve closure, again using the biplane area–length method from both apical four-chamber and two-chamber views. The LAEF was evaluated as a comprehensive measure of LA contractile function, calculated using the formula: (LAmax–LAmin)/LAmax.[7] The LA passive emptying fraction (LA-PEF) was determined by the ratio of the LA passive emptying volume to Vmax. Conversely, the LA active emptying fraction (LA-AEF) was calculated as the ratio of the LA active emptying volume to volume before atrial contraciton. The E/e′ ratio is defined as the ratio of the early diastolic velocity of mitral inflow (E) to the early diastolic velocity of mitral annular motion (e′).[13] This measurement reflects the reservoir and booster pump phases of LA function. The left ventricular ejection fraction (LVEF) was evaluated utilizing Simpson's biplane method, which is based on standard apical four-chamber and two-chamber views. All studies were interpreted by a board-certified cardiologist who was blinded to the participants' clinical and demographic information.

2.2 | Statistical analyses

Statistical analyses for the study were conducted utilizing SPSS version 29.0 (IBM SPSS Statistics for Windows, version 29.0). The normality assumption for quantitative variables was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Descriptive statistics for the variables are presented as mean ± SD and median (25th–75th percentile). Comparisons between two groups were performed using the independent *t*-test and Mann–Whitney *U*-test. The homogeneity of variances was evaluated using Levene's test for homogeneity. Categorical variables were analyzed with the chi-squared test, considering the number of categories and expected values. The relationships

between variables were examined using Spearman's rho correlation analysis. In all statistical analyses, *p*-values < .05 were interpreted as statistically significant.

3 | RESULTS

The study comprised 58 patients with epilepsy and 49 healthy controls. Age and gender distribution of the patient and control groups were similar ($p > .05$). The mean age of PWE was 34 years (range = 26–45), with females constituting 53.4% of the sample. The median duration of diagnosis was 12 years (range = 6–22). The mean ASM utilization per patient was 2,[1,2] with 28 patients (48.3%) using one ASM, 20 patients (34.5%) using two ASMs, and 10 patients (17.2%) using three or more ASMs. Demographic and clinical data for the epilepsy group are summarized in Table 1.

The descriptive statistics and comparative results regarding demographic and LA functional parameters of PWE and healthy control subjects are presented in Table 2. No statistically significant differences were observed between the two groups concerning gender distribution ($p = .802$), age ($p = .175$), BMI ($p = .928$), and BSA ($p = .894$). Smokers numbered 22 (37.9%) in the epilepsy group and 19 (38.8%) in the control group. There was no alcohol consumption in both groups. However, the difference in total LAEF between the groups was statistically significant ($p = .001$), with PWE exhibiting lower total LAEF values compared to the control group. Conversely, the LA-PEF values were higher in PWE than in the control group, with this difference also being statistically significant ($p = .028$). Additionally, the comparison of LA-AEF values revealed a statistically significant difference ($p = .001$), with PWE demonstrating lower LA-AEF values than the control group. The median maximum LAVI was significantly higher in the epilepsy group (28.95 [range = 25.97–32.12] mL/m^2) compared to the control group (28.4 [range = 22.2–30.8] mL/m^2, $p = .047$), indicating early structural remodeling of the atrium in PWE. No significant differences were noted between the groups regarding preatrial LAVI and LAmin ($p = .863$ and $p = .259$, respectively). No significant difference was found between epilepsy and control groups in terms of E/e' ratio and LVEF ($p = .599$, $p = .219$, respectively). There was no significant correlation observed between the annual number of generalized convulsive seizures and any of the echocardiographic biomarkers ($p > .05$).

TABLE 1 Demographic and clinical data of patients with epilepsy ($n = 58$).

Age (year)	34 (26–45)
Female Gender	31(53.4)
Seizure type	
Focal	40 (69%)
Generalized	8 (13.8%)
Unknown etiology	10 (17.2%)
EEG findings	
Normal	15 (25.9%)
Non specific findings	13 (22.4%)
Focal findings	25 (43.1%)
Generalized findings	5 (8.6%)
Seizure free status	
Medically controlled epilepsy	28 (48.3%)
Drug-resistant epilepsy	30 (51.7%)
Annual number of GCS	0 (0–2)
Medication	
Levetiracetam	50 (86.2%)
Valproic acid	15 (25.9%)
Lamotrigine	12 (20.7%)
Carbamazepine	9 (15.5%)
Lacosamide	7 (12.1%)
Topiramate	5 (8.6%)
SUDEP-7_Score	2 (0–3)

Abbreviations: EEG, electroencephalographic; GCS, generalized convulsive seizures; SUDEP, sudden unexpected death in epilepsy.

The patient population was categorized into two groups: DRE (51.7%) and MCE (48.3%). The comparative analysis of LA function parameters and demographic variables is presented in Table 3. The results indicate that there were no statistically significant differences between the groups regarding gender, age, BMI, and BSA values, with *p*-values of .778, .112, .957, and .914, respectively. Notably, the maximum LAVI values for patients in the DRE group were higher than those in the MCE group; however, this difference did not reach statistical significance ($p = .240$). Additionally, preatrial LAVI, LAmin, and total LAEF values and E/e' ratio did not show significant differences between the two groups ($p > .05$). Conversely, the duration of diagnosis was significantly longer in the DRE group compared to the MCE group, with a statistically significant difference in time to diagnosis ($p = .030$). Furthermore, the SUDEP-7 scores for the DRE group were significantly higher than those for the MCE group, with this difference being statistically significant ($p < .001$).

The correlation between clinical parameters and LA functional parameters is presented in Table 4. The correlation analysis revealed statistically significant positive correlations between age and the SUDEP-7 score ($r = .336$, $p = .010$), the duration of diagnosis ($r = .425$, $p = .001$), maximum LAVI ($r = .474$, $p < .01$), preatrial LAVI ($r = .372$, $p = .004$), and LAmin ($r = .500$, $p < .001$). Additionally, significant positive correlations were identified between the SUDEP-7 score and both the duration of diagnosis

TABLE 2 Comparison of demographic and LA functional characteristics of epilepsy and control groups.

Characteristic	Epilepsy, $n=58$	Control, $n=49$	p
Age, years	34 (26–45)	38 (33.5–44)	.175
Female gender	31 (53.4)	25 (51.0)	.802
BMI	27.82 ± 6.14	27.72 ± 5.26	.928
BSA	1.87 ± .22	1.88 ± .22	.894
Smoking	22 (37.9)	19 (38.8)	.930
Max LAVI	28.95 (25.97–32.12)	28.4 (22.2–30.8)	.047
Preatrial LAVI	26.6 (24.6–28.32)	26.6 (22.65–29.15)	.863
Min LA volume	15.0 (13.6–16.92)	14.8 (13.1–16.65)	.259
Total LAEF	45.38 ± 10.78	51.51 ± 6.95	.001
Passive LAEF	26.12 ± 10.07	22.20 ± 7.63	.028
Active LAEF	25.91 ± 11.08	32.89 ± 10.64	.001
E/e′ ratio	4.6 (4.03–5.8)	4.54 (4.13–5.64)	.599
LVEF	58 (55–60)	60 (55.5–60)	.219

Abbreviations: BMI, body mass index; BSA, body surface area; LA, left atrial; LAEF, LA ejection fraction; LAVI, LA volumetric index; LVEF, left ventricular ejection fraction; Max, maximum; Min, minimum.

TABLE 3 Comparison of demographic and LA functional characteristics of drug-resistant epilepsy and medically controlled epilepsy.

Characteristic	Medically controlled epilepsy, $n=28$	Drug-resistant epilepsy, $n=30$	p
Age, years	31.0 (25.25–42.5)	27.5 (28.75–47.25)	.112
Female gender	16 (57.1)	15 (50.0)	.778
BMI	27.05 (24.32–31.87)	27.20 (23.40–31.35)	.957
BSA	1.87 ± .21	1.88 ± .23	.914
Disease duration	8.0 (3.25–17.0)	13.5 (7.75–23.75)	.030
SUDEP-7 score	0 (0–0)	3 (2–4)	<.001
Max LAVI	28.45 (25.90–30.50)	29.65 (26.22–34.82)	.240
Preatrial LAVI	25.95 (24.02–28.50)	27.0 (24.77–28.32)	.602
Min LA Volume	14.90 (13.45–17.80)	15.05 (13.90–16.92)	.732
Total LAEF	44.43 ± 8.5	46.26 ± 12.63	.524
Passive LAEF	27.98 ± 9.62	24.38 ± 10.34	.176
Active LAEF	23.83 ± 11.19	27.85 ± 10.80	.170
E/e′ ratio	4.28 (4.07–5.25)	4.67 (4.18–5.72)	.330
LVEF	58 (55–60)	58 (54.8–60)	.946

Abbreviations: BMI, body mass index; BSA, body surface area; LA, left atrial; LAEF, LA ejection fraction; LAVI, LA volumetric index; LVEF, left ventricular ejection fraction; Max, maximum; Min, minimum; SUDEP, sudden unexpected death in epilepsy.

($r=.488$, $p<.001$) and maximum LAVI ($r=.265$, $p=.044$). Furthermore, a statistically significant negative correlation was observed between the duration of diagnosis and LA-PEF ($r=-.307$, $p=.020$).

4 | DISCUSSION

To the best of our knowledge, this study is the first to demonstrate a significant reduction in LAEF in individuals with PWE compared to healthy controls. Although no previous studies have directly evaluated LAEF in epilepsy cohorts, our findings are consistent with a growing body of evidence that suggests the presence of subclinical cardiac dysfunction in this population. Several echocardiographic investigations have reported elevated E/e′ ratios, prolonged isovolumetric relaxation times, and increased A-wave velocities in patients with epilepsy,[3,8,14] all of which reflect impaired diastolic function and elevated LV filling pressures—factors known to impair LA mechanical function and reduce atrial contractility.[15] The clinical significance of LAEF has been thoroughly documented in

TABLE 4 Correlation between clinical and LA functional parameters in patients with epilepsy.

		Age	SUDEP-7 score	Disease duration	Max LAVI	Preatrial LAVI	Min LA volume	Total LAEF	Passive LAEF	Active LAEF
Age	*r*	1.000	.336[a]	.425[a]	.474[a]	.372[a]	.500[a]	−.172	−.195	.021
	p		.010	.001	<.001	.004	<.001	.196	.143	.873
SUDEP-7 score	*r*		1.000	.488[a]	.265[b]	.190	.107	.091	−.198	.213
	p			<.001	.044	.153	.426	.497	.137	.108
Disease duration	*r*			1.000	.214	.240	.157	−.057	−.307[b]	.157
	p				.106	.070	.240	.669	.019	.241
Max LAVI	*r*				1.000	.878[a]	.731[a]	−.029	−.300[b]	.195
	p					<.001	<.001	.829	.022	.143
Preatrial LAVI	*r*					1.000	.722[a]	−.117	−.566[a]	.276[b]
	p						<.001	.384	<.001	.036
Min LA volume	*r*						1.000	−.411[a]	−.411[a]	−.117
	p							.001	.001	.384
Total LAEF	*r*							1.000	.421[a]	.659[a]
	p								<.001	<.001
Passive LAEF	*r*								1.000	−.270[b]
	p									.040
Active LAEF	*r*									1.000
	p									

Abbreviations: LA, left atrial; LAEF, LA ejection fraction; LAVI, LA volumetric index; Max, maximum; Min, minimum; SUDEP, sudden unexpected death in epilepsy.

[a]Correlation is significant at the .01 level.

[b]Correlation is significant at the .05 level.

cases of heart failure with preserved ejection fraction and atrial fibrillation. Our findings indicate that analogous mechanisms may play a role in seizure-related cardiac remodeling, potentially heightening the risk of arrhythmias and sudden cardiac death within this population.

The LA volume is regarded as a morphological indicator of LV diastolic dysfunction and is extensively acknowledged as a "barometer" of the chronicity and severity of elevated LV filling pressures.[16] In the study conducted by Fialho et al., it was observed that the LAVI was statistically significantly elevated in patients diagnosed with temporal lobe epilepsy, with a mean duration of diagnosis of 22.5 ± 10.67 years, in comparison to the control group.[9] Conversely, a study by Bilgi et al. involving 30 newly diagnosed and untreated PWE, reported that LAVI was comparable between the epilepsy and control groups.[8] Our current study similarly observed a higher LAVI in PWE. Fialho et al. proposed that this discrepancy may be attributed to the younger age demographic in the cohort studied by Bilgi et al. It is also important to note that the study by Bilgi et al. focused on newly diagnosed patients, whereas both the studies by Fialho et al. and our present investigation included individuals with chronic epilepsy. Although we did not find a relationship between disease duration and LAVI in this study, disease duration, prolonged exposure to epileptic seizures, and side effects of ASMs may have influenced LAVI. Furthermore, a high maximum LAVI was positively correlated with SUDEP-7 scores, indicating that LAVI may serve as a predictive parameter for SUDEP. Devinsky et al. notably reported that postmortem analyses of individuals with epilepsy demonstrated the presence of myocardial fibrosis, despite the absence of gross cardiac abnormalities.[17,18] This observation supports the hypothesis that subclinical structural remodeling, such as elevated LAVI, may serve as a functional correlate of microscopic myocardial alterations. Such insights could elucidate the reasons why certain patients experience sudden death, even when cardiac imaging appears unremarkable.

The E/e′ ratio has been established as a fundamental measure for estimating LV filling pressure.[13] In two prior studies, the E/e′ ratio was observed to be elevated in patients with epilepsy compared to a control group[8,9]; however, our study did not corroborate this finding. Likewise, we did not observe any significant differences in the E/e′ ratio between the DRE and MCE subgroups. The LAVI and LAEF are indicative of long-term structural and functional alterations in the LA, typically

resulting from chronic hemodynamic stress or autonomic dysfunction associated with epilepsy. The E/e′ ratio is a dynamic and load-dependent measure that is sensitive to acute fluctuations in LV filling pressure and can be affected by various factors, including volume status, heart rate, and pharmacological agents. E/e′ ratio may appear normal even if chronic atrial remodeling is present in a patient without recent seizures during echocardiographic evaluation.

In our examination of potential associations between ASMs and LA functional parameters, we identified weak correlations between some ASMs, such as levetiracetam, topiramate, and lamotrigine, and selected echocardiographic markers. However, these findings lacked statistical robustness. The limitations of this analysis were exacerbated by the small subgroup sizes and the heterogeneity of drug use within the cohort. Given the limited strength and interpretability of these associations, and in keeping with the exploratory nature of the study, we chose not to include these results in the primary findings. We recommend that further research involving larger, pharmacologically stratified populations is essential to clarify these potential associations.

This study presents several limitations that must be acknowledged. First, its single-center, cross-sectional design restricts the ability to draw causal inferences or evaluate the progression of LA changes over time. Moreover, despite the established autonomic and electrophysiological effects associated with certain classes of ASM, the influence on cardiac parameters remains unexamined due to the heterogeneous distribution of these drugs. Although LA function was comprehensively assessed and LVEF was included, important left ventricular diastolic parameters such as tricuspid regurgitation maximum flow rate, septal and lateral e' velocities, degree of diastolic dysfunction, LV mass index, and strain imaging were not included. In addition, physical activity that may affect echocardiographic biomarkers was not questioned in our study. Lastly, the absence of longitudinal follow-up precludes the evaluation of the prognostic significance of the echocardiographic changes observed in relation to arrhythmias, SUDEP, or cardiac morbidity. Future prospective studies involving larger and more diverse cohorts, comprehensive pharmacologic profiling, and extended follow-up are necessary to validate and expand upon these findings.

5 | CONCLUSIONS

The present findings underline that LAEF and LAVI may offer enhanced sensitivity for the detection of subclinical cardiac involvement in patients with epilepsy, particularly in chronic cases, with changes in the assessment of LA dysfunction not detected by conventional diastolic markers such as the E/e' ratio. Further multicenter studies on the assessment of LA function are needed.

AUTHOR CONTRIBUTIONS

Selcen Duran: Conceptualization (lead); methodology (equal); formal analysis (lead); data curation (equal); writing—original draft (lead); project administration (lead); investigation (equal); visualization (lead); supervision (lead). **Yalcin Boduroglu:** Methodology (equal); data curation (equal); investigation (equal); writing—review & editing (equal).

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None.

CONFLICT OF INTEREST STATEMENT

Neither of the authors has any conflicts of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

PATIENT CONSENT

This study did not involve identifiable patient data; therefore, patient consent was not required.

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