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Left Ventricular Hypertrophy Findings on Electrocardiogram Predict Impaired Left Atrial Functions

Elektrokardiyogramdaki Sol Ventrikül Hipertrofisi Bulguları Sol Atriyal Fonksiyonlarda Bozulmayı Öngörüyor

ABSTRACT

Objective: Electrocardiographic left ventricular hypertrophy (ECG LVH) holds significant clinical importance in cardiovascular disease. Pathological processes that lead to left ventricular hypertrophy (LVH) also induce remodeling and impair left atrial (LA) function. Atrial function can be assessed using speckle-tracking echocardiography. This study investigates the potential impact of ECG LVH on LA strain.

Methods: A total of 62 individuals diagnosed with LVH, based on the echocardiographic left ventricular mass index, were included. ECG LVH was assessed using established protocols: the Sokolow-Lyon voltage criteria (SV1 + RV5/RV6 > 35 mm), Cornell voltage criteria (RaVL + SV3 > 28 mm for men and > 20 mm for women), and the Cornell product criteria [(SV3 + RaVL + (for women 8 mm)] x QRS duration > 2440 mm x ms). Participants were categorized into two groups based on the presence or absence of ECG LVH. The relationship between LA strain measures and ECG characteristics was explored.

Results: The study population had a median age of 58.3 ± 10.1 years, with 40.3% being female, 91.9% hypertensive, and 35.5% diabetic. Nineteen patients (30.6%) were identified with ECG LVH based on Sokolow-Lyon voltage, Cornell voltage, or Cornell product criteria. These patients exhibited significantly reduced LA reservoir, conduit, and contraction strains ($P < 0.001$). Statistically significant correlations were observed between all three phases of LA strain measures and Sokolow-Lyon voltage (reservoir $r = -0.389$, $P < 0.01$; conduit $r = -0.273$, $P < 0.05$; contraction $r = -0.359$, $P < 0.01$), Cornell voltage (reservoir $r = -0.49$, $P < 0.001$; conduit $r = -0.432$, $P < 0.001$; contraction $r = -0.339$, $P < 0.01$), and Cornell product (reservoir $r = -0.471$, $P < 0.001$; conduit $r = -0.387$, $P < 0.01$; contraction $r = -0.362$, $P < 0.01$).

Conclusion: ECG LVH is associated with impaired LA strain, validating its use as an effective tool for predicting LA dysfunction.

Keywords: Cornell product, Cornell voltage, left atrial strain, left ventricular hypertrophy, Sokolow-Lyon voltage

ÖZET

Amaç: Elektrokardiyografik sol ventrikül hipertrofisi (EKG SVH) kardiyovasküler hastalıklarda prognostik öneme sahiptir. SVH'ye neden olan patolojik süreçler sol atriyumun (LA) yeniden şekillenmesini ve işlev bozukluğunu tetikler. Atriyal fonksiyonlar speckle tracking ekokardiyografi ile değerlendirilebilir. Çalışmamızın amacı EKG SVH'nin LA strain'i üzerindeki potansiyel etkisini araştırmaktır.

Yöntem: Ekokardiyografik sol ventrikül kitle indeksi ile SVH'si olan altmış iki hasta bu çalışmaya dahil edildi. EKG SVH aşağıdakilere göre ölçüldü: Sokolow Lyon voltajı SV1 + RV5/RV6 > 35 mm; Cornell voltajı RaVL + SV3 > 28 mm (erkek cinsiyet) > 20 mm (kadın cinsiyet); veya Cornell ürünü (SV3 + RaVL + (kadın cinsiyet 8 mm)) x QRS süresi) > 2440 mm x ms. Hastalar EKG SVH varlığı veya yokluğuna göre iki gruba ayrıldı. LA strain değerleri ile EKG bulguları arasındaki ilişki araştırıldı.

Bulgular: Hastaların yaş ortalaması 58,3 ± 10,1 yıl, %40,3'ü kadın, %91,9'u hipertansif ve %35,5'i diyabetikti. Sokolow-Lyon voltajı, Cornell voltajı veya Cornell ürününe göre 19 hastada (%30,6) EKG SVH vardı. EKG SVH'si olan hastalarda sol atriyal rezervuar, kondüit ve kasılma strainleri anlamlı derecede düşüktü ($P < 0,001$). Sol atriyal strain değerlerinin her üç fazı ile Sokolow-Lyon voltajı (rezervuar $r = -0,389$, $P < 0,01$; konduit $r = -0,273$, $P < 0,05$; kontraksiyon $r = -0,359$, $P < 0,01$), Cornell voltajı (rezervuar $r = -0,49$, $P < 0,001$; konduit $r = -0,432$, $P < 0,001$; kasılma $r = -0,339$, $P < 0,01$) ve Cornell ürünü (rezervuar $r = -0,471$, $P < 0,001$; konduit

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r = -0,387, P < 0,01; kasılma r = -0,362, P < 0,01) arasında da istatistiksel olarak anlamlı bir korelasyon vardı.

Sonuç: EKG SVH, bozulmuş LA strain ile ilişkilidir ve LA disfonksiyonunu öngörebilmek için yararlı bir araçtır.

Anahtar Kelimeler: Cornell ürünü, Cornell voltaj, sol atriyal strain, sol ventrikül hipertrofisi, Sokolow-Lyon voltaj

The adaptive response to an increased ventricular workload commonly results in left ventricular hypertrophy (LVH). LVH frequently develops secondary to hypertension, the most common cause, as well as valvular heart disease and cardiomyopathies.[1] The Sokolow-Lyon voltage, Cornell voltage, and Cornell product criteria are extensively used to diagnose LVH on an electrocardiogram (ECG).[2] Although the ECG has a low sensitivity for detecting LVH, it is still linked to impaired left ventricular (LV) functions and serves as a significant marker for cardiovascular morbidity and mortality.[3]

As LVH progresses, it also leads to left atrial (LA) remodeling and dysfunction.[4] Although the increased left atrial volume index (LAVI) remains the main indicator for LA remodeling, it has some limitations. LAVI is based on static volumetric evaluation and does not capture the dynamic characteristics of the LA.[5] However, it is noteworthy that LA dysfunction can be detected by speckle-tracking echocardiography (STE) even in the absence of obvious structural deformations of the LA.[6] LA strain has been explored in various physiological and pathological conditions, including hypertension, heart failure, diabetes mellitus, coronary artery disease, atrial fibrillation, and valvular heart pathologies.[7] Recently, the LA strain has been recommended as a standard measure for predicting LV loading pressures.[8]

The aim of this study was to analyze LA functional characteristics using STE in individuals diagnosed with LVH by transthoracic echocardiography (TTE) and to investigate the relationship between ECG LVH and LA strain values.

Materials and Methods

A total of 62 patients diagnosed with LVH by TTE in a cardiology outpatient clinic and presenting with normal LV ejection fraction were enrolled in the study. Exclusion criteria included patients with complete right or left bundle branch block, documented previous coronary artery disease, atrial fibrillation or significant arrhythmia, valvular disease more severe than mild, uncontrolled diabetes or hypertension, or poor echocardiographic images. Patients suspected of having cardiomyopathy (e.g., hypertrophic cardiomyopathy, amyloidosis) underwent a thorough clinical evaluation, pedigree analysis, ECG, Holter monitoring, laboratory testing, and multimodal imaging to ensure an accurate diagnosis. Additionally, patients with suspected coronary artery disease, particularly those exhibiting T-wave inversion on ECG, were excluded following noninvasive stress imaging. Patients with uncontrolled hypertension were also excluded due to significantly lower LA strain parameters.[9]

ABBREVIATIONS

ECG LVH	Electrocardiographic left ventricular hypertrophy
GLS	Global longitudinal strain
LA	Left atrium
LAVI	Left atrial volume index
LV	Left ventricle
LVH	Left ventricular hypertrophy
LVM	Left ventricular mass
LVMI	Left ventricular mass index
STE	Speckle tracking echocardiography
TTE	Transthoracic echocardiography

The 12-lead ECGs were obtained using a Mindray Beneheart R12 monitor with default calibration settings (25 mm/sec and 10 mm/mv). The QRS duration was automatically calculated by the monitor, and manual measurements were taken for S in leads V1 and V3, and R in leads V5, V6, and aVL. Electrocardiographic left ventricular hypertrophy (ECG LVH) was assessed using the established protocols: Sokolow-Lyon voltage criteria (SV1 + RV5/RV6 > 35 mm), Cornell voltage criteria (RaVL + SV3 > 28 mm for men and > 20 mm for women), or the Cornell product criteria ((SV3 + RAVL + (8 mm for women)) x QRS duration > 2440 mm x ms).[10,11]

The echocardiographic studies were conducted by an experienced echocardiographer using a VIVID E95 (GE, USA) with a 3.5 MHz transducer. With the patient in a partly left lateral decubitus position, LV end-diastolic and end-systolic dimensions were recorded. The Devereux formula was used to quantify left ventricular mass (LVM): LVM (g) = 0.80 x {1.04 x [(septal thickness + internal diameter + posterior wall thickness)^3 - (internal diameter)^3]} + 0.6 g. The body surface area was then indexed to LVM. Echocardiographic LVH was determined based on the American Society of Echocardiography recommendations, defined as a left ventricular mass index (LVMI) exceeding 115 g/m^2 in men and 95 g/m^2 in women.[12]

To measure LA strain values, LA scans were obtained from apical four-chamber images over three heartbeats and analyzed using Echopac software. The endocardium was traced both manually and automatically, with the software identifying the region of interest along the LA border. An LA strain curve pattern was generated using R-R gating, starting from the initiation of the QRS complex. The zero base for the longitudinal strain curve was set at the ventricular end-diastole. The initial peak of the upright longitudinal strain curve indicates atrial reservoir function, strain at early diastole represents atrial conduit function, and strain at late diastole demonstrates atrial contraction function.[13]

The echocardiographic recordings were stored, and images were evaluated. Both the examiner and reviewer are authors of the study. The reviewer conducted a repeat strain analysis on all recordings. Intraobserver and interobserver variability were assessed using the coefficient of variation [CV = 100 (s/x) (%)], demonstrating a high level of agreement with coefficients considered low (< 10%).

Data were presented as frequencies or mean values with standard deviation. The Shapiro-Wilk test was conducted to confirm the normal distribution of the data. Group differences for continuous

variables were assessed using either an independent two-sample t-test or the Mann-Whitney U test. For categorical variables, Pearson's chi-squared test, Fisher's exact test, and Yates correction were applied as appropriate. Pearson correlation analysis was conducted to examine associations among continuous numerical parameters. A Pearson correlation coefficient (r) value less than 0.2 was interpreted as very weak, 0.2-0.4 as weak, 0.4-0.6 as moderate, 0.6-0.8 as good, and greater than 0.8 as a strong association. All tests were two-tailed, and a p-value of less than 0.05 was considered statistically significant. Data processing was performed using SPSS (Statistical Package for the Social Sciences) for Windows, Version 25.0 (SPSS Inc., Chicago, IL, USA).

No artificial intelligence-based technologies, including large language models (LLMs), chat-bots, or image generators, were utilized in the production of this submitted work. The content presented in this manuscript is the result of traditional human-authored processes.

The study adhered to the principles set forth in the Declaration of Helsinki, ensuring strict compliance with ethical standards and participants' rights throughout the research process.

Ethical Consideration

All procedures were performed with informed consent of the patients. Approval for the study was granted by the İzmir Katip Çelebi University Ethics Committee (Approval date 18.03.2021, number 164).

Results

The study population consisted of 62 individuals with a median age of 58.3 ± 10.1 years, 40.3% of whom were female. Ninety-two percent of the patients had hypertension, and 35.5% had diabetes. Table 1 displays the demographic, clinical, and echocardiographic features of the patients. ECG LVH was detected in 19 patients (30.6%) using the Sokolow-Lyon voltage, Cornell voltage, or Cornell product criteria.

Table 2 illustrates the basal echocardiographic findings in patients with and without ECG LVH. Patients with ECG LVH exhibited a higher LVMI (138.8 ± 22.9 g/m^2 vs. 117.4 ± 11.5 g/m^2, *P* = 0.001), an elevated E/E' ratio (10.7 ± 2.4 vs. 9.1 ± 2.2, *P* = 0.015), and a reduced absolute LV Global Longitudinal Strain (LV GLS) (19.2% ± 2.4 vs. 21.1% ± 1.7, *P* < 0.001) compared to those without ECG LVH.

Table 1. Characteristics of the patients

Age, years	58.3±10.1
Women, n	25 (40.3)
Hypertension, n	57 (91.9)
Diabetes, n	22 (35.5)
Renal failure, n	6 (9.7)
Chronic obstructive respiratory disease, n	11 (17.7)
Hyperlipidemia, n	24 (38.7)
Peripheral arterial disease, n	2 (3.2)
Smoking status (never smoker), n	33 (53.2)
EF %	64.8±8.9
LV mass index (g/m^2)	124.0±18.6
LV mass index (g/m^2)-males	127.3±17.2
LV mass index (g/m^2)-females	119.1±19.9
Relative wall thickness	0.59±0.09
LA volume index (mL/m^2)	28.2±6.7
E wave velocity (cm/s)	70.8±15.0
A wave velocity (cm/s)	92.7±14.9
E/A	0.79±0.2
E/E'	9.6±2.3
LV GLS %	20.6±2.2
LA- strain reservoir %	35.1±7.3
LA- strain conduit %	15.5±5.1
LA- strain contraction %	19.7±4
Sokolow-Lyon voltage (mm)	24.5±8.3
Cornell voltage (mm)	16.2±4.5
Cornell product (mm x ms)	1826.2±625.9
ECGLVH (+)	19 (30.6)
Drug use	
Beta blocker	23 (37.1)
ACEI or ARB	46 (74.2)
Ca channel blocker	28 (45.2)
Diuretic	29 (46.8)
Oral antidiabetic	17 (27.4)
Insulin	8 (12.9)

ACEI, Angiotensin converting enzyme inhibitors; ARB, Angiotensin receptor blockers; EF, Ejection fraction; LV, Left ventricle; LA, Left atrium; GLS, Global longitudinal strain; ECG LVH, Electrocardiographic left ventricular hypertrophy; LV, Left ventricle. Values are mean standard ± deviation

Table 2. Echocardiographic findings of the patients with or without ECG LVH

	ECG LVH (-) (n=43)	ECG LVH (+) (n=19)	*P*
	Mean ± SD		
EF %	64.8±10.3	64.8±4.3	0.971
LV mass index (g/m^2)	117.4±11.5	138.8±22.9	**0.001**
LA volume index (mL/m^2)	27.1±5.7	30.6±8.1	0.052
Relative wall thickness	0.58±0.08	0.62±0.09	0.106
E/E'	9.1±2.2	10.7±2.4	**0.015**
E/A	0.79±0.18	0.76±0.18	0.542
LV GLS %	21.1±1.7	19.2±2.4	**<0.001**
LA- strain reservoir %	38.3±4.3	27.7±7.3	**<0.001**
LA- strain conduit %	17.3±3.7	11.2±5.3	**<0.001**
LA- strain contraction %	21.0±3.5	16.5±3.4	**<0.001**

EF, Ejection fraction; LV, Left ventricle; LA, Left atrium; GLS, Global longitudinal strain.

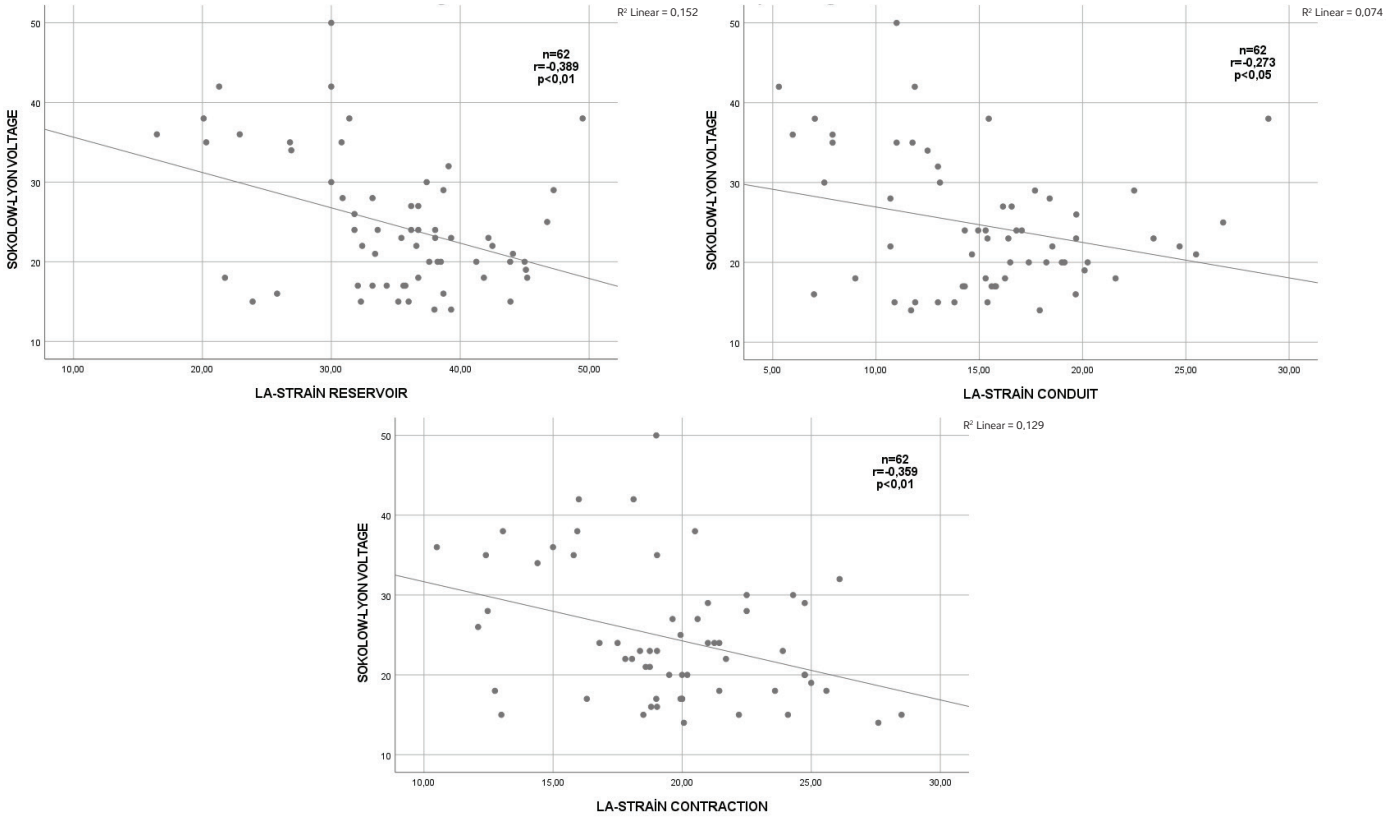


Figure 1. The correlation between Sokolow-Lyon voltage and LA strain values

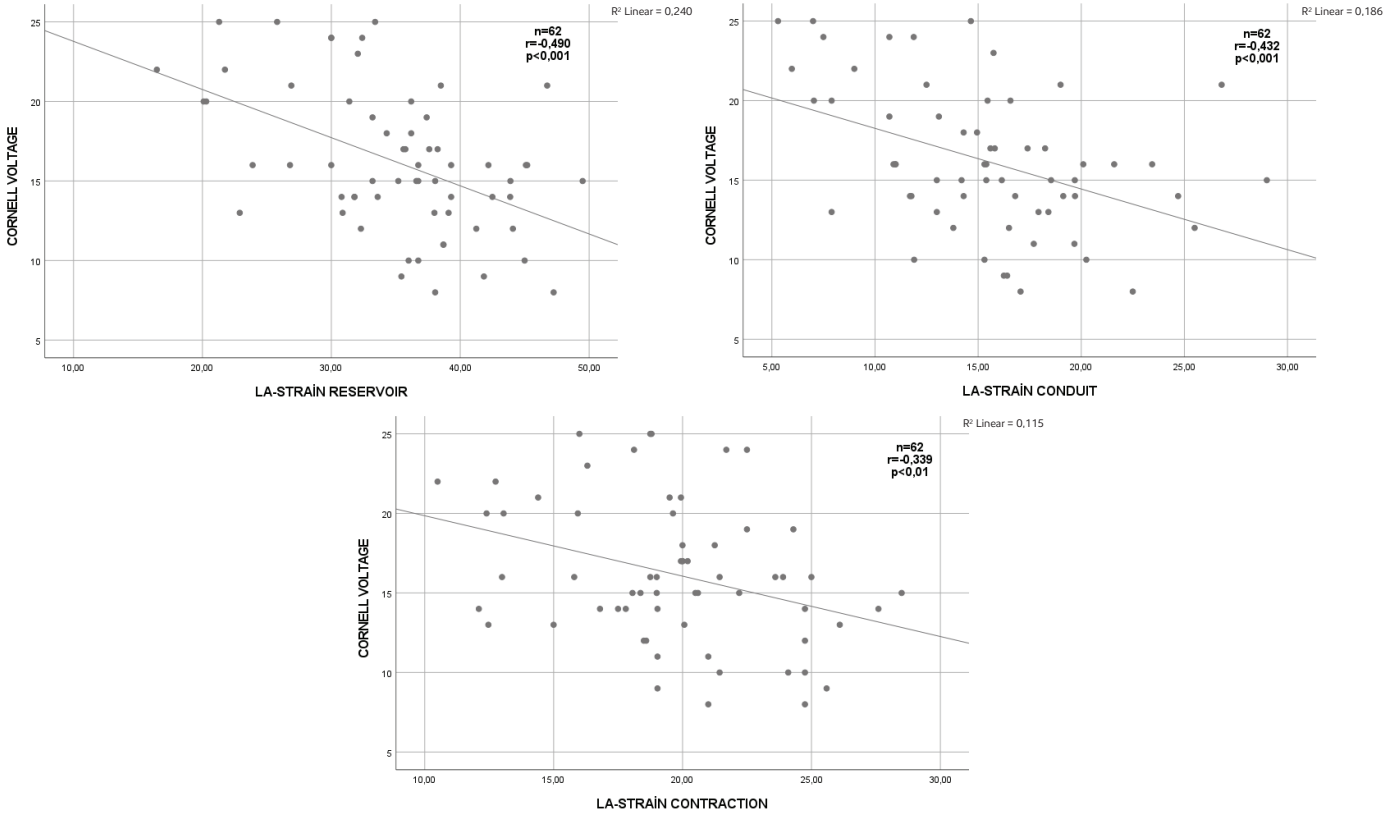


Figure 2. The correlation between Cornell voltage and LA strain values.

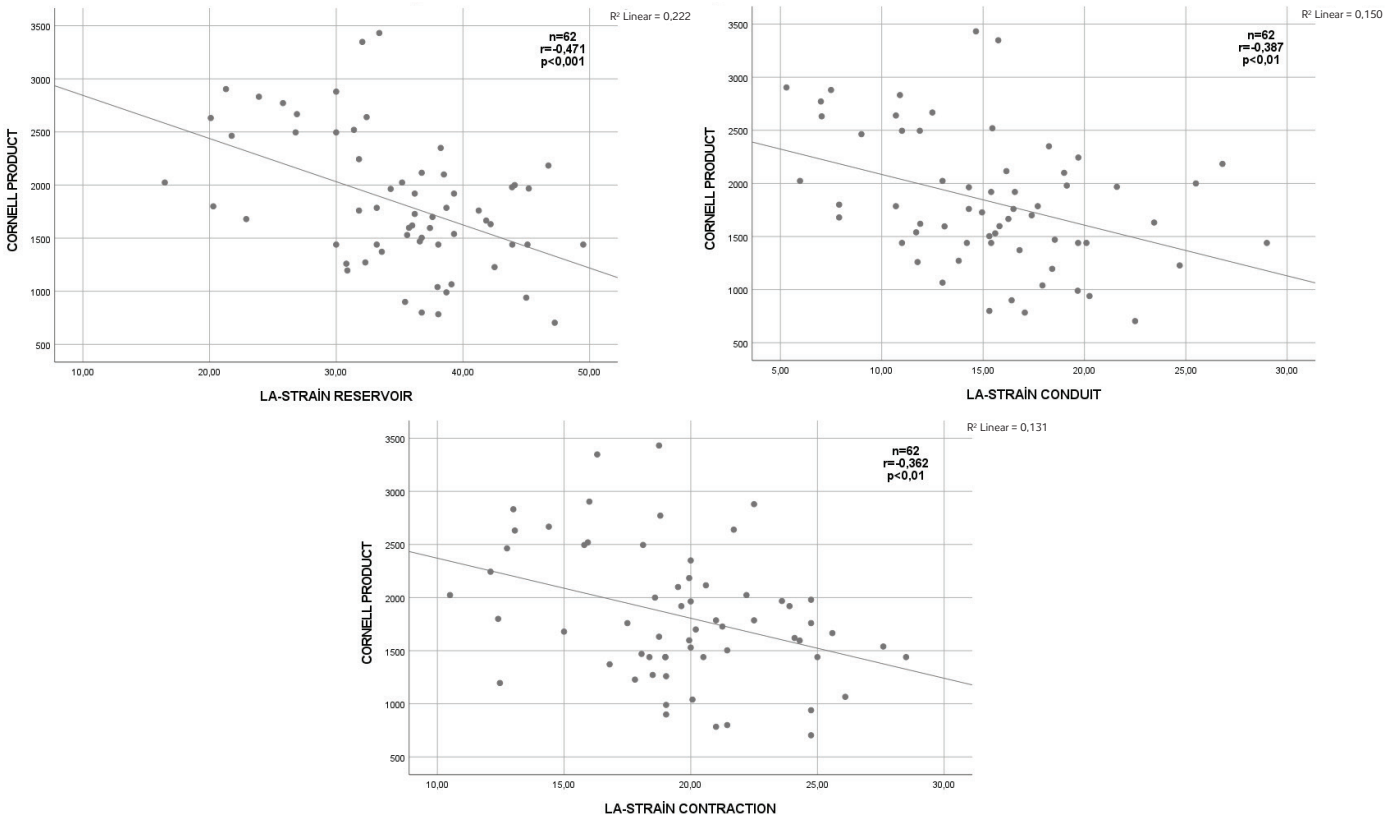
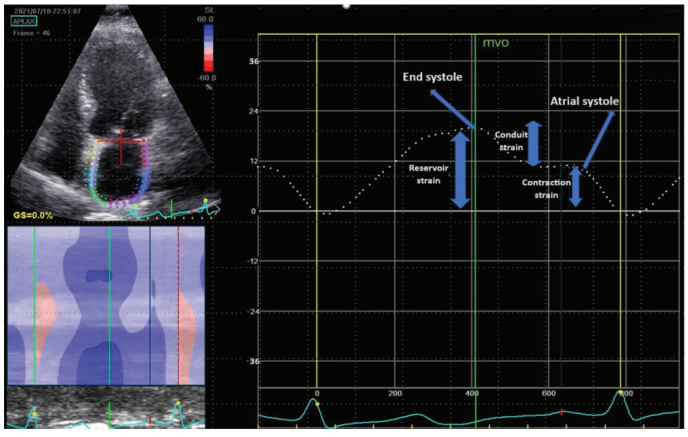


Figure 3. The correlation between Cornell product and LA strain values.



Reservoir strain: 21,3 Conduit strain: 11,2 Contraction strain:10,1

Figure 4. Decreased LA strain parameters.

In patients with ECG LVH, LA reservoir, LA conduit, and LA contraction strain values were significantly lower than those without ECG LVH (27.7% ± 7.3 vs. 38.3% ± 4.3, $P < 0.001$; 11.2% ± 5.3 vs. 17.3% ± 3.7, $P < 0.001$; 16.5% ± 3.4 vs. 21.0% ± 3.5, $P < 0.001$, respectively). Additionally, statistically significant correlations were observed across all three phases of LA strain measures and Sokolow-Lyon voltage (reservoir $r = -0.389$, $P < 0.01$; conduit $r = -0.273$, $P < 0.05$; contraction $r = -0.359$, $P < 0.01$), Cornell voltage (reservoir $r = -0.49$, $P < 0.001$; conduit $r = -0.432$, $P < 0.001$; contraction $r = -0.339$, $P < 0.01$), and Cornell product (reservoir $r = -0.471$, $P < 0.001$; conduit $r = -0.387$, $P < 0.01$; contraction $r = -0.362$, $P < 0.01$). These correlations are depicted in Figures 1, 2, and 3. A representative figure of the decreased LA strain parameters is shown in Figure 4.

LA strain values were found to be correlated with T wave abnormalities on ECG. Patients with T wave inversion exhibited

Table 3.LA strain values according to T wave abnormality

	LA reservoir strain %		LA conduit strain %		LA contraction strain %	
	Median (min-max)	***P***	**Median (min-max)**	***P***	**Median (min-max)**	***P***
Patients with T wave inversion (n=12)	30.4 (20.1-45.1)	**0.001**	11,4 (5.3-20.1)	**0.007**	16.2 (12.8-25)	**0.022**
Patients without T wave inversion (n=50)	36.8 (16.5-49.5)		16.2 (6.0-29)		20 (10.5-28.5)	

Table 4. Correlation between echocardiographic findings and LA strain values

	LA reservoir strain	LA conduit strain	LA contraction strain
LV mass index (g/m²)	-0.495***	-0.433***	-0.347**
LV GLS %	0,436**	0,328**	0,374**
Relative wall thickness	-0.132	-0.251*	0.079
LA volume index (mL/m²)	-0.192	-0.101	-0.220*
E/e'	-0.270*	-0.390**	0.006

*P < 0.05, **P < 0.01, ***P < 0.001

Table 5. LA strain values according to LA volume index

	LA volume index <34 mL/m² (n=51)	LA volume index >34 mL/m² (n=11)	*P*
	Mean ± SD		
LA reservoir strain %	35.4±6.7	33.1±9.2	0.338
LA conduit strain %	15.5±4.9	14.8±6.0	0.687
LA contraction strain %	19.9±3.8	18.2±4.5	0.221

significantly lower LA reservoir strain (30.4% vs. 36.8%, P = 0.001), LA conduit strain (11.4% vs. 16.2%, P = 0.007), and LA contraction strain (16.2% vs. 20%, P = 0.022) compared to those without T wave inversion (Table 3).

This study also examined the correlation between LA strain values and other echocardiographic parameters (Table 4). Significant correlations were found between LVMI and LV GLS with the reservoir (LVMI: r = -0.495, P < 0.001, LV LGS: r = 0.436, P < 0.01), conduit (LVMI: r = -0.433, P < 0.001, LV GLS: r = 0.328, P < 0.01), and contraction (LVMI: r = -0.347, P < 0.01, LV GLS: r = 0.374, P < 0.01) strains. Additionally, a significant correlation was identified between LAVI and LA contraction strain (r = -0.220, P < 0.05). However, no correlations were found between LAVI and LA reservoir or conduit strain parameters (Table 5).

Discussion

The study results indicate that patients with LVH findings on ECG exhibited significantly lower LA reservoir, conduit, and contraction strain values compared to those without ECG LVH. Furthermore, a significant correlation was identified between ECG LVH criteria and LA strain values, suggesting that the presence of LVH on ECG may be a useful tool for predicting abnormal LA function.

LVH can be identified by surface ECG. Although the sensitivity of ECG in detecting LVH is limited, the presence of LVH features on surface ECG serves as an indicator of LV mass and function and correlates with a higher cardiovascular risk. In a study reported by Ishikawa et al.,[14] the Cornell product and Sokolow-Lyon voltage were associated with an increased LV mass index, and the Cornell product was linked to reduced LV GLS. Another study investigating the relationship between QRS voltage and LV function found a significant association between Sokolow-Lyon voltage and LV mass, LV EF, GLS, and E/e' ratios.[15] Consistent with previous research, our study demonstrates that LVH findings on ECG are associated with increased LVMI, higher E/e', and reduced LV GLS.

Repolarization anomalies are present in a significant fraction of patients with ECG LVH. In patients with ECG LVH and preserved LV systolic function, T-wave inversion is linked to a greater likelihood of diastolic dysfunction. Patients with T-wave inversion exhibit greater values for septal and lateral E/e', as well as LAVI, when comparisons are made between different echocardiographic estimates of LA pressure.[16] The results of our study indicated that patients with T-wave inversion exhibit significantly lower LA reservoir, conduit, and contraction voltage values compared to patients without T-wave inversion, supporting the presence of LA dysfunction in this patient group.

LVH is associated with functional impairment of both the LV and LA. It has been suggested that the mechanism behind LA impairment in hypertensive patients with LVH is increased LV filling pressures due to LV diastolic dysfunction.[17] Several studies have investigated the link between hypertension and impaired LA function using various measurement techniques, including STE parameters, tissue doppler imaging, indexes of mitral and pulmonary vein flows, and phasic/volumetric measurements (18-20). The measurement of LA strain has become a valuable tool for evaluating diastolic function of the LV and estimating the pressures involved in LV filling.[21] Numerous publications have explored the relationship between LA strain and LVH etiologies such as hypertension, hypertrophic cardiomyopathy, and cardiac amyloidosis.[22,23] Research on LA STE deformation parameters has shown that hypertensive patients with LVH on echocardiography have markedly impaired LA reservoir, conduit, and contraction function.[24-26] Consistent with these studies, we discovered a significant correlation between the LVMI and LA reservoir, conduit, and contraction strain values. Additionally, we demonstrated that patients with LVH on echocardiography who also had LVH findings on ECG exhibited worse LA function by STE compared to those without LVH findings on ECG. To our knowledge, this is the first investigation of the connection between the occurrence of LVH findings on ECG and STE deformation parameters for LA function. Furthermore, we discovered a significant correlation

between Sokolow-Lyon voltage, Cornell voltage, and Cornell product and LA deformation parameters. These findings indicate that the presence of LVH findings on ECG not only predicts worse LV function but also worse LA function.

Increased LA dimensions are recognized as a surrogate sign of LA dysfunction and LV diastolic dysfunction.[27,28] However, enlargement of the LA alone is an insufficient marker of the early stages of LV diastolic dysfunction, as LA remodeling may require time to occur. Therefore, LA dysfunction can be evident despite a normal LA size. STE may reveal LA dysfunction prior to the development of LA conformational changes and dilatation.[28] Hypertension causes functional deterioration in the LA even before the onset of LV hypertrophy, and it leads to structural changes in the LA characterized by an increase in LA volume.[29,30] Mondillo et al.[25] and Sahebjam et al.[21] demonstrated that diabetic and/or hypertensive individuals with normal LA dimensions exhibited decreased LA deformation dynamics and considerably reduced strain measures in each of the LA segments, respectively. In our study population, the mean LAVI was 28.2 ± 6.7 mL/m^2, which falls within the normal range.[12] Thus, our study population comprises patients with echocardiography-diagnosed LVH and normal or mildly increased LA size. In this particular patient group, we have not observed any correlation between LAVI and LA reservoir or conduit strain values, consistent with previous studies suggesting that LA size is an insensitive indicator of LA dysfunction in the initial stages of the disease, and LA deformation dynamics by STE is a more sensitive instrument to identify LA dysfunction before the development of LA structural deterioration. Moreover, our findings indicate that the presence of LVH on an ECG may serve as an indicator of LA dysfunction, providing a more reliable sign of LA dysfunction than LA size, especially if LA dimensions are within the normal or slightly increased range.

Limitations

This study was conducted in a single center and had a relatively small sample size, which may limit its statistical power and generalizability. Additionally, LVMI was calculated using 2D echocardiography, although evidence suggests that cardiac magnetic resonance imaging is more accurate. However, it is worth noting that echocardiography boasts high repeatability in diagnosing LVH and remains the most widely accepted modality in clinical practice.

Conclusion

ECG LVH is associated with impaired LA strain, highlighting its utility as a predictive tool for LA dysfunction.

Ethics Committee Approval: Approval for the study was granted by the İzmir Katip Çelebi University Ethics Committee (Approval Number: 164, Date: 18.03.2021).

Informed Consent: All procedures were performed with informed consent of the patients.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Design – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Supervision – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Resource – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Materials – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Data Collection and/or Processing – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Analysis and/or Interpretation – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Literature Review – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Writing – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.; Critical Review – U.K., N.K.E.; E.Ö., S.V.E., M.O.G., M.T.

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