Correlation between Left Atrial Echocardiographic Deformation Parameters and Invasive Left Ventricular End-Diastolic Pressure Measurements

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Received 25 February 2024; Accepted 21 April 2024

Abstract

Background: *The noninvasive estimation of elevated left ventricular end-diastolic pressure (LVEDP) is a critical step in assessing left ventricular diastolic dysfunction (LVDD). Nonetheless, most echocardiographic parameters currently used for this purpose have significant limitations. Recent studies have highlighted the utility of left atrial (LA) strain as a noninvasive method for estimating LVEDP. This study aimed to explore the correlations between LA deformation parameters, measured using speckle-tracking echocardiography (STE), and invasively obtained LVEDP.*

Methods*: This prospective study involved 82 patients in sinus rhythm who underwent left heart catheterization at our center. All participants underwent comprehensive transthoracic echocardiography and peak atrial longitudinal strain (PALS) assessment via STE within 12 hours before catheterization.*

Results*: LVEDP was elevated in 45 patients (54.9%) and normal in 37 (45.1%). PALS, LA ejection fraction, and septal E' showed moderate inverse correlations with LVEDP (r= −0.590, P=0.001; r= −0.463, P=0.001; and r= −0.449, P=0.001, respectively). The E/E' ratio also exhibited a moderate correlation with LVEDP (r=0.567, P=0.001). Lateral E' and the E/A ratio demonstrated weaker inverse correlations with LVEDP (r= −0.231, P=0.037 and r= −0.229, P=0.038, respectively). In multivariate logistic regression analysis, age (OR, 1.14, 95% CI, 1.02 to 1.27), PALS (OR, 0.77, 95% CI, 0.65 to 0.91), and the E/E' ratio (OR, 1.36, 95% CI, 1.11 to 1.89) were identified as independent predictors of an LVEDP≥12 mm Hg. PALS demonstrated the highest diagnostic accuracy for predicting an LVEDP≥12 mm Hg, with an AUC of 0.849 (95% CI, 0.764 to 0.935; P<0.001). A PALS cutoff value of 35% yielded a sensitivity of 81.1% and a specificity of 81.4% for predicting elevated LVEDP.*

Conclusion: *PALS emerged as a reliable noninvasive parameter for predicting elevated LVEDP. Its application may facilitate the earlier identification of LVDD.*

J Teh Univ Heart Ctr 2024;19(S1):24-31

This paper should be cited as: Noroozi M, Khalilipur E, Baay M, Khesali H, Bakhshandeh H, Cheraghi S, et al. Correlation between Left Atrial Echocardiographic Deformation Parameters and Invasive Left Ventricular End-Diastolic Pressure Measurements. J Teh Univ Heart Ctr 2024;19(S1):24-31. DOI: 10.18502/jthc.v19is1.18476

Keywords: Left atrium; Longitudinal strain; Left ventricular end-diastolic pressure; Echocardiography; Cardiac catheterization; Speckle tracking; Diastolic dysfunction

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DOI: 10.18502/jthc.v19is1.18476

Introduction

Left ventricular diastolic dysfunction (LVDD) is a key predictor of mortality and adverse outcomes in the general population. Furthermore, the presence of LVDD is a diagnostic criterion for heart failure with preserved ejection fraction,[1, 2] underscoring the growing interest in developing more accurate and reliable methods for detecting LVDD in routine clinical practice. Elevated left ventricular end-diastolic pressure (LVEDP) represents the primary physiological manifestation of LVDD.[3]

Left cardiac catheterization remains the gold standard for assessing LVEDP and LVDD. Nevertheless, it is an invasive procedure associated with potential complications, leading to the widespread use of noninvasive methods, particularly echocardiography, for evaluating LV filling pressure. Several echocardiographic parameters, such as the E/E' ratio, have demonstrated strong correlations with LVEDP.[4] Still, the reliability of these measures remains a subject of debate, as not all studies agree on their diagnostic value.[5]

The interaction between the left atrium (LA) and the LV throughout the cardiac cycle plays a critical role in determining the diastolic function of the heart. LVDD, along with the subsequent increase in LVEDP, can lead to structural and functional changes in the LA over time. LA function is often indirectly evaluated through measurements of LA size or volume. Speckle-tracking echocardiography (STE) offers a valuable method for assessing LA deformation, as it is relatively independent of load conditions and geometric assumptions and is less influenced by changes in loading.[6, 7]

Several studies have indicated that peak atrial longitudinal strain (PALS), measured using either Doppler or STE, correlates well with LVEDP and pulmonary capillary wedge pressure (PCWP).[8–11]

Based on these findings, the present study aimed to investigate the correlations between LA ejection fraction, systolic deformation parameters assessed by STE, and invasively measured LVEDP.

Methods

Setting and design

We prospectively enrolled 82 patients in sinus rhythm who consecutively underwent left heart catheterization at our major tertiary care facility between January 2022 and December 2022. All participants underwent 2D echocardiography and PALS assessment using STE within 12 hours before catheterization. Patients were excluded from the study if they met any of the following criteria: incomplete clinical data, non-sinus rhythm, recent acute coronary syndromes (<72 h), prosthetic heart valves, moderate or severe mitral regurgitation, any degree of mitral or aortic stenosis, or poor imaging quality of the LA endocardial border.

Invasive LVEDP measurement

A 6 Fr pigtail catheter was calibrated to atmospheric pressure before being percutaneously inserted into the LV via the radial or femoral artery. LVEDP was measured at the onset of the QRS complex on electrocardiography. In line with previous studies, an LVEDP≥12 mm Hg was defined as elevated LV filling pressure.[11–13] LVEDP measurements were performed by 2 investigators who were blinded to the echocardiographic data.

Echocardiography

All echocardiographic examinations were conducted by a board-certified echocardiographer who was blinded to the patients' LVEDP measurements. Each patient underwent comprehensive transthoracic echocardiography (TTE) in accordance with the latest TTE guidelines.[14] The examinations were performed using a Philips EPIQ machine equipped with a 2.5 MHz transducer. The LA dimension was measured in the parasternal long-axis view at the end of the systole. Transmitral flow velocities (E and A waves) were obtained using pulsed-wave Doppler in the apical 4-chamber view (Figure 1-A). The mitral early diastolic peak velocity (E wave), late diastolic peak velocity (A wave), and the E/A ratio were measured. Left ventricular ejection fraction (LVEF) was calculated using Simpson's biplane method. The septal mitral annulus early diastolic velocity (E′) was assessed using tissue Doppler imaging (Figure 1-B & C), and the E/E′ ratio was calculated. A cutoff value exceeding 14 was used to indicate elevated LV filling pressure. Diastolic function was graded according to the latest recommendations for evaluating LV diastolic function.[15] In the apical 2- and 4-chamber views, left atrial volume (LAV) was measured using Simpson's biplane method. Measurements were taken in the end-systolic frame, just before mitral valve (MV) opening, to determine the maximal left atrial volume (LAVmax), and in the end-diastolic frame, coinciding with the R-wave on the ECG, to determine the minimal left atrial volume (LAVmin). LAV was then indexed to body surface area. The left atrial ejection fraction (LAEF) was calculated using the following formula:

$$(\text{LAVImax} - \text{LAVImin}) / \text{LAVImax}$$

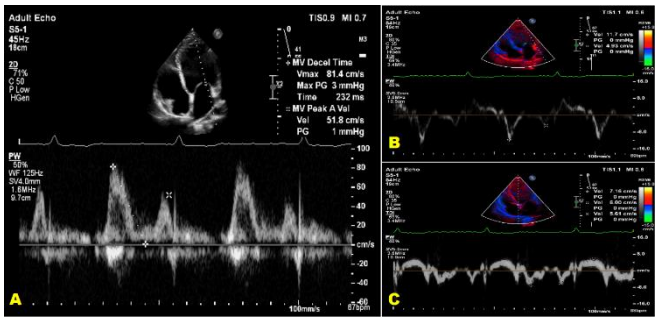


Figure 1. The images showcase (A) pulsed wave Doppler tracing of mitral inflow velocities, (B) tissue Doppler tracing of lateral mitral annular velocity, and (C) tissue Doppler tracing of septal mitral annular velocity.

STE

For STE, apical 4- and 2-chamber view images were acquired to measure LA strain. The LA endocardial border was manually traced to define the complete myocardial region of interest, which comprised 6 segments. If segmental tracking met the quality criteria for analysis, the software processed the data, while poorly tracked segments were automatically excluded. Longitudinal strain curves were

generated using TomTec software for each view (Figure 2). PALS was measured at the end of the atrial reservoir phase. Apical 4- and 2-chamber view images were captured over 3 consecutive heart cycles, with simultaneous electrocardiographic recording. The frame rate for image acquisition was set between 60 and 80 frames per second.

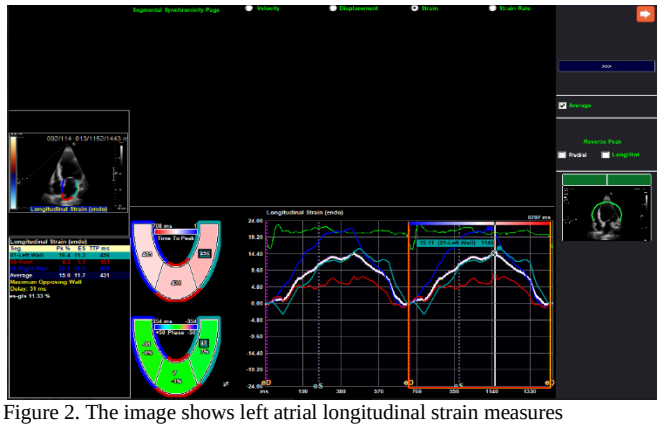


Figure 2. The image shows left atrial longitudinal strain measures calculated using TomTec software in the 4-chamber view.

Data collection

Clinical baseline characteristics, echocardiographic parameters, and catheterization data were collected. Key baseline characteristics, such as age, sex, hypertension, diabetes mellitus, hyperlipidemia, and smoking status, were obtained from the patients' medical records or self-reported by the patients (referred information). Echocardiographic parameters included MV E velocity, MV A velocity, septal E', lateral E', the E/A ratio, the E/E' ratio, pulmonary artery pressure, grade of diastolic dysfunction, LA dimension, LA end-diastolic and end-systolic volume indices, LA longitudinal strain, LAEF, and LVEF. Catheterization data, including the number of culprit vessels and LVEDP, were also recorded.

Statistical Analysis

Data were analyzed using descriptive statistics, including mean ± standard deviation (SD), medians, frequencies, and percentages, as appropriate. Differences between subgroups were evaluated using the independent t-test for continuous, normally distributed variables and the χ^2 test (or Fisher's exact test) for categorical variables.

Correlations between variables were assessed using Pearson's correlation test. Factors associated with elevated LVEDP were examined using univariate and multivariate logistic regression analyses. A receiver operating characteristic (ROC) curve analysis was performed to determine sensitivity, specificity, and the optimal cutoff point for LA strain.

All variables with a *P*-value <0.1 in the univariate analysis were included in the multivariate models. In addition to demographic covariates, all other predefined echocardiographic indices were included as quantitative covariates in the model. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. A *P*-value <0.05 was considered statistically significant. All analyses were conducted using Stata software (version 14.1; Stata Corp, College Station, TX, USA).

Ethics

The study protocol was approved by the institutional research ethics committee in November 2021. Written informed consent was obtained from all participants, and individual personal information was maintained with strict confidentiality.

Results

Table 1 shows the patients' clinical characteristics, echocardiographic measurements, and cardiac catheterization data. LVEDP was elevated in 45 patients (54.9%) and normal in 37 (45.1%). Significant differences between the 2 groups were observed in systolic and diastolic blood pressure in the catheterization laboratory, septal E', lateral E', the E/A ratio, the E/E` ratio, PALS, and LAEF. The mean values of systolic blood pressure (160.88±30.58 vs 140.27±17.07; *P*=0.001), diastolic blood pressure (91.77±15.85 vs 80.0±11.30; *P*=0.031), and the E/E` ratio (10.54±3.67 vs 7.92±2.34; *P*=0.001) were significantly higher in patients with an LVEDP≥12 mm Hg than in those with an LVEDP<12 mm Hg. The mean values of septal E' (6.15±1.43 vs 8.17±1.57; *P*=0.0001), lateral E' (8.64±2.20 vs 10.58±2.58; *P*=0.001), the E/A ratio (0.83±0.25 vs 0.98±0.37; *P*=0.032), PALS (27.49±7.62 vs 37.48±5.72; *P*=0.001), and LAEF (39.80±4.10 vs 47.91±15.11; *P*=0.001) were significantly lower in patients with an LVEDP≥12 mm Hg than in those with an LVEDP<12 mm Hg.

The correlations between LVEDP and echocardiographic variables are presented in Table 2. PALS, LAEF, and septal E' had a moderate inverse correlation with LVEDP (*r*= −0.590, *P*=0.001; *r*= −0.463, *P*=0.001; and *r*= −0.449, *P*=0.001, respectively); The E/E` ratio also had a moderate correlation with LVEDP (*r*= 0.567, *P*=0.001). However, lateral E' and the E/A ratio showed a weak inverse correlation with LVEDP (*r*= −0.231, *P*=0.037 and *r*= −0.229, *P*=0.038, respectively). A stronger correlation was demonstrated between LVEDP and PALS (*r*= −0.738), LAEF (*r*= −0.547), septal E' (*r*= −0.606), and the E/E` ratio (*r*=0.717) among patients with a decreased EF (Table 2).

The univariate multiple regression analysis demonstrated that age (OR, 1.06; 95% CI, 1.01 to 1.12), LAEF (OR, 0.92; 95% CI, 0.87 to 0.97), PALS (OR, 0.80; 95% CI, 0.72 to 0.88), the E/A ratio (OR, 0.20; 95% CI, 0.04 to 0.92), septal E' (OR, 0.42; 95% CI, 0.29 to 0.62), lateral E' (OR, 0.71; 95% CI, 0.58 to 0.87), and the E/E` ratio (OR, 1.42; 95% CI, 1.16 to 2.06) were independent predictors of an increased LVEDP among the covariates examined. The predictive values of age, PALS, LAEF, the E/A ratio, septal E', lateral E', and the E/E` ratio were evaluated in multivariate logistic regression analyses. Age (OR, 1.14; 95% CI, 1.02 to 1.27), PALS (OR, 0.77; 95% CI, 0.65 to 0.91), and the E/E` ratio (OR, 1.36; 95% CI, 1.11 to 1.89) were independent predictors of an LVEDP≥12 mm Hg in their respective model (Table 3).

To further evaluate the utility of echocardiographic indices in predicting elevated LVEDP, an ROC curve

analysis was performed. LA longitudinal strain demonstrated the highest diagnostic accuracy, with a cutoff value of 35% yielding a sensitivity of 81.1% and a specificity of 81.4%. The area under the ROC curve (AUC) for LA longitudinal strain was 0.849 (95% CI, 0.764 to 0.935; P<0.001) in predicting an LVEDP ≥12 mm Hg (Figure 3). In contrast, the E/E' ratio, a classic method, showed weaker diagnostic accuracy, with a lower AUC of 0.758 (95% CI, 0.648 to 0.868; P<0.001) (Figure 4).

Table 1. Baseline clinical, cardiac catheterization, and echocardiographic data

***P*-value**	**LVEDP≥12 mm Hg (N=45)**	**LVEDP<12 mm Hg (N=37)**	**Variables**
Age (y)	58.43±10.02	63.62±8.38	0.206**
Sex (male %)	26 (70.3)	27 (60.0)	0.231*
BMI	25.33±3.65	25.84±3.48	0.730**
HTN	27 (73.0)	38 (84.4)	0.231*
DM	9 (24.3)	16 (35.6)	0.196*
HLP	17 (45.9)	20 (44.4)	0.534*
Smoking	8 (21.6)	10 (22.2)	0.582*
SBP in the cath lab	140.27±17.07	160.88±30.58	0.001**
DBP in the cath lab	80.0±11.30	91.77±15.85	0.031**
Number of culprit vessels			
Single-vessel disease	13 (35.1)	16 (35.6)	
Two-vessel disease	2 (5.4)	5 (11.1)	0.802*
Three-vessel disease	8 (21.6)	10 (22.2)	
MV E velocity	0.71±0.15	0.69±0.19	0.199**
MV A velocity	0.75±0.19	0.82±0.18	0.099**
Septal E'	8.17±1.57	6.15±1.43	0.001**
Lateral E'	10.58±2.58	8.64±2.20	0.001**
E/A ratio	0.98±0.37	0.83±0.25	0.032**
E/E` ratio	7.92±2.34	10.54±3.67	0.001**
PH	1 (2.7)	7 (20)	0.119*
MR			
Mild	23 (62.2)	24 (53.3)	0.281*
Mild to moderate	14 (37.8)	21 (46.7)	
TR			
Mild	33 (89.2)	43 (95.6)	0.249*
Moderate	4 (10.8)	2 (4.4)	
LAD	3.71±0.48	3.67±0.33	0.672**
LAEDVI	16.05±4.26	17.88±5.89	0.119**
LAESVI	28.43±8.55	30.20±7.79	0.331**
PALS	37.48±5.72	27.49±7.62	0.001**
LAEF	47.91±15.11	39.80±4.10	0.001**
LVEF	48.10±13.91	46.88±10.99	0.659**

LVEDP, Left ventricular end-diastolic pressure; BMI, Body mass index; HTN, Hypertension; HLP, Hyperlipidemia; DM, Diabetes mellitus; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PAP, Pulmonary artery pressure; LVDD, Left ventricular diastolic dysfunction; LVEF, Left ventricular ejection fraction; MR, Mitral regurgitation; TR, Tricuspid regurgitation; LAEF, Left atrial ejection fraction; PALS, Peak atrial longitudinal strain; LAD, Left atrial dimension; LAEDVI, Left atrial end-diastolic volume index; LAESVI, Left atrial end-systolic volume index

Table 2. Correlations (*r*) between echocardiographic variables and LVEDP

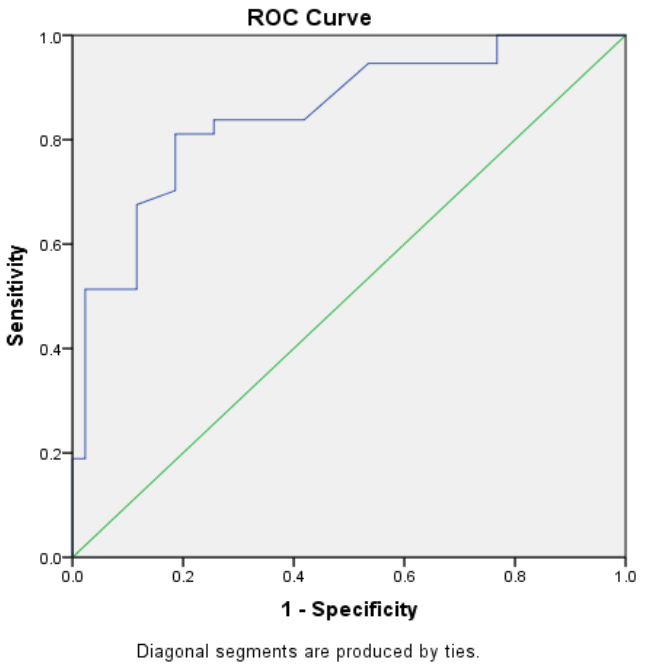
Variables	All Patients (N=82)	*P*-value	EF>50% (N=41)	*P*-value	EF<50% (N=41)	*P*-value
LAD	-0.083	0.459	-0.115	0.476	-0.018	0.910
LAEDVI	0.141	0.207	0.048	0.764	0.287	0.068
LAESVI	0.038	0.733	0.033	0.839	0.004	0.982
PALS	-0.590	0.001	-0.530	0.001	-0.738	0.001
LAEF	-0.463	0.001	-0.413	0.007	-0.547	0.001
MV E Velocity	-0.032	0.776	-0.117	0.468	0.107	0.507
MV A Velocity	0.235	0.034	0.129	0.421	0.390	0.012
Septal E’	-0.449	0.001	-0.353	0.023	-0.606	0.001
Lateral E’	-0.292	0.037	-0.231	0.064	-0.104	0.519
E/A ratio	-0.229	0.038	-0.327	0.037	-0.026	0.872
LVEF	-0.075	0.505	-0.029	0.858	-0.054	0.738
E/E` ratio	0.567	0.001	0.462	0.001	0.717	0.001

LVEDP, Left ventricular end-diastolic pressure; LVEF, Left ventricular ejection fraction; LAEF, Left atrial ejection fraction; PALS, Peak atrial longitudinal strain; LAD, Left atrial dimension; LAEDVI, Left atrial end-diastolic volume index; LAESVI, Left atrial end-systolic volume index
* Pearson’s correlation test

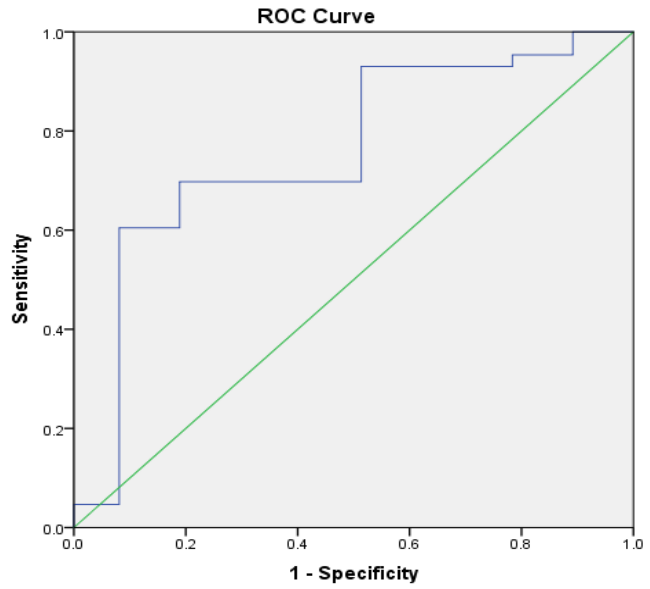
Table 3. Multivariate regression analysis to identify predictors of elevated LVEDP

Variables	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Age	1.06 (1.01-1.12)	0.016	1.14(1.02-1.27)	0.017
Female	1.57(0.62-3.96)	0.334	-	
Smoking	1.03 (0.36-2.96)	0.948	-	
DM	1.71 (0.65-4.51)	0.274	-	
HLP	0.94 (0.39-2.25)	0.892	-	
HTN	2.01(0.67-5.94)	0.207	-	
LAEF	0.92(0.87-0.97)	0.003	0.95(0.89-1.01)	0.092
PALS	0.80 (0.72-0.88)	0.001	0.77 (0.65-0.91)	0.003
LAESVI	1.02(0.98-1.08)	0.327	-	
LAEDVI	1.07(0.98-1.17)	0.123	-	
LAD	0.77(0.26-2.32)	0.668	-	
LVEF	0.93(0.75-1.16)	0.555	-	
E/A ratio	0.20(0.04-0.92)	0.039	1.95(0.8-46.09)	0.679
MV E velocity	0.42(0.03-5.22)	0.505	-	
MV A velocity	7.49(0.67-82.81)	0.105	-	
Septal E’	0.42(0.29-0.62)	0.001	0.83(0.39-1.73)	0.622
Lateral E’	0.71(0.58-0.87)	0.001	0.85(0.48-1.51)	0.601
E/E` ratio	1.42(1.16-2.06)	0.001	1.36(1.11-1.89)	0.006

LVEDP, Left ventricular end-diastolic pressure; HTN, Hypertension; HLP, Hyperlipidemia; DM, Diabetes mellitus; LVEF, Left ventricular ejection fraction; LAEF, Left atrial ejection fraction; PALS, Peak atrial longitudinal strain; LAD, Left atrial dimension; LAEDVI, Left atrial end-diastolic volume index; LAESVI, Left atrial end-systolic volume index
*logistic regression



AUC=0.849
Figure 3. The image illustrates the ROC curves for PALS in predicting an LVEDP≥12 mm Hg.
ROC, Receiver operating characteristics; PALS, Peak atrial longitudinal strain; LVEDP, Left ventricular end-diastolic pressure



AUC=0.758
Figure 4. The image displays the ROC curves for the E/E' ratio in predicting an LVEDP≥12 mm Hg.
ROC, Receiver operating characteristics; LVEDP, Left ventricular end-diastolic pressure

Discussion

In this study, we measured PALS alongside other diastolic parameters of the left heart and assessed their relationship with invasively measured LVEDP. Elevated LVEDP was observed in 45 patients (54.9% of the study population). PALS demonstrated a moderate inverse correlation with LVEDP (r= −0.590, P=0.001), which was slightly stronger than that of the E/E' ratio. PALS achieved a diagnostic accuracy of 0.849 (95% CI, 0.764 to 0.935; P<0.001) for predicting elevated LVEDP. In multivariate analysis, age, PALS, and the E/E' ratio were identified as independent predictors of increased LVEDP.

The noninvasive estimation of LVEDP is essential for evaluating diastolic function. Nonetheless, many widely used echocardiographic parameters for this purpose have limitations. LA strain provides a practical and reproducible method for assessing LA function and has recently been recommended as a valuable parameter in the evaluation of LVDD.[3, 16] Therefore, in this study, we aimed to explore the association between LA deformation parameters, measured using STE, and invasively obtained LVEDP. Our findings demonstrated an inverse correlation between PALS and LVEDP. Consistent with our results, Fan et al[10] also reported significantly impaired LA strain in patients with elevated LVEDP and LVEF≥50%. Similarly, Singh et al[17] concluded that left atrial reservoir strain (LASr) decreased as diastolic function deteriorated. Cameli et al[11] also identified a strong correlation between global PALS and LVEDP. Ohara et al[18] and Yeh et al[19] reported comparable findings in studies involving patients with coronary artery disease and pediatric heart transplant recipients, respectively.

In the early stages of LVDD, ventricular compliance decreases, and relaxation is impaired. As a result, passive early trans-mitral diastolic flow is reduced, and the atrial pump function increases to compensate. With further declines in LV distensibility, atrial pressure rises to maintain cardiac output, potentially reducing LA compliance. Consequently, LA function is impaired due to elevated LVEDP and diminished LA compliance.

PCWP typically increases following LA decompensation and a significant rise in LVEDP. A previous study found that PCWP often underestimated LVEDP and had only moderate predictive value for diagnosing patients with normal or elevated LVEDP.[20] In this study, we defined an LVEDP≥12 mm Hg as elevated to facilitate earlier diagnosis of diastolic dysfunction.

We observed a stronger correlation between LVEDP and PALS (r= −0.738) in patients with reduced LVEF compared with those with preserved EF. Similarly, Cameli et al[21] suggested that PALS was a more reliable measure of LV filling pressure in patients with advanced systolic heart failure. Nagueh et al[22] also demonstrated that LASr accurately estimated LV filling pressure in patients with reduced LVEF. However, Singh et al[9] argued that LASr was less accurate in estimating LV filling pressure in patients with LV systolic dysfunction compared with those with normal systolic function. They proposed a cutoff of 20% for identifying elevated LVEDP in their study population.

According to our results, a PALS cutoff value of 35% predicted an LVEDP≥12 mm Hg with 81.1% sensitivity and 81.4% specificity. Furthermore, PALS demonstrated superior diagnostic accuracy for predicting LVEDP compared with the E/E' ratio, suggesting that LA strain may detect LVDD at an earlier stage than the E/E' ratio. These findings align with previous studies. Morris et al[23] reported that a PALS value <23% had 73% sensitivity and 76% specificity for identifying LVDD. Fan et al[10] found that LASr at a cutoff of 26.7% could predict an LVEDP>16 mm Hg with 90% sensitivity and 82.9% specificity. Similarly, Ohara et al[18] observed that LA

contractile strain at a cutoff value below 11.6% predicted an LVEDP>15 mm Hg with an AUC of 0.84. Lin et al[24] demonstrated that LASr (AUC=0.75) and septal E/E' (AUC=0.76) were more effective in predicting LVEDP and identifying LVDD than other conventional parameters. Cameli et al[11] concluded that global PALS with a cutoff of 18% achieved the highest overall diagnostic accuracy (AUC=0.87) for predicting elevated filling pressure, with a sensitivity of 96% and specificity of 92%. Similarly, Inoue et al[25] found that an LASr<18% and an LA pump strain value <8% predicted elevated LV filling pressure more accurately than conventional Doppler indices, with accuracy rates of 75% for LASr and 72% for pump strain. A potential explanation for the lower accuracy of the E/E' ratio in assessing LVEDP is that both mitral E and E' velocities occur during early diastole and are influenced by multiple factors, including ventricular recoil, suction, MV function, and intraventricular pressure gradients. Additionally, technical limitations should be considered. The accuracy of the E/E' ratio may be compromised by the angle dependency of Doppler and tissue Doppler imaging. Moreover, mitral annular tissue Doppler imaging velocities are influenced by numerous factors, such as translational motion of the heart, tethering effects, regional wall motion abnormalities, and conduction delays. Therefore, based on our findings and those of similar studies,[5, 11] the E/E' ratio should not be used as the sole parameter for determining LVEDP.

Limitations

The results of this study should be interpreted in the context of several limitations. First, the data were obtained from a single center, which may limit the generalizability of our findings to the broader population. Further multicenter studies are needed to validate our results. Second, patients with atrial fibrillation or other arrhythmias were excluded from the study. Since STE measurements depend on the cardiac cycle, it was not feasible to include patients in non-sinus rhythm. Consequently, our study population was restricted to patients in sinus rhythm, and the findings may only apply to this group. Third, the study participants were admitted for cardiac catheterization for various reasons, not solely for heart failure evaluation. Finally, although patients were recruited consecutively, the blood pressure of participants in the catheterization lab was borderline high, and a relatively high percentage had elevated LVEDP. This suggests a higher risk of diastolic dysfunction compared with the general population.

Conclusion

Based on the findings of this study, we recommend integrating PALS into the routine assessment of LVDD, given its excellent sensitivity, high reproducibility, and strong feasibility. Further research is needed to explore the optimal integration of LA strain and other echocardiographic parameters, such as the E/E' ratio, into multiparametric diagnostic models. This would help validate the best cutoff values for these parameters and improve the differentiation between elevated and normal LVEDP levels.

Conflict of Interest

The authors declare that they have no conflicts of interest. Furthermore, they have no financial interests pertaining to any aspect of the study.

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