ORIGINAL ARTICLE

Noninvasive Estimation of Left Ventricular Diastolic Function in Patients with Hypertension and Normal Ejection Fraction Using 2-Dimensional Speckle Tracking Echocardiography

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Abstract

Background: ***Hypertension leads to increased left ventricular (LV) wall stress, afterload, and remodeling, resulting in diastolic dysfunction and hypertensive heart disease. Assessing LV end-diastolic pressure (LVEDP) is crucial for early detection of diastolic impairment.***

Aim: ***To assess diastolic function, with a particular focus on (LVEDP), in hypertensive patients utilizing speckle tracking echocardiography (STE), a non-invasive technique, to compare the non-invasive findings obtained from (STE) with the invasive measurements acquired through cardiac catheterization.***

Methods: ***This study compares LVEDP in 25 hypertensive vs. 25 normotensive patients using invasive catheterization and echocardiography-derived KT index, which states that Estimated LVEDP= 10.8–12.4 [KT index] where KT index equals [log10 (active LAEF/minimum LAV)].***

Results: ***Hypertensive patients showed significantly elevated estimated LVEDP ($p < 0.0011$), measured LVEDP ($p < 0.0011$) than the upper limit of normal LVEDP. Passive and active left atrial ejection fractions (LAEF%) were significantly reduced ($p < 0.0011$), indicating impaired atrial function. Wilcoxon analysis confirmed a strong correlation between estimated and measured LVEDP ($p < 0.001$).***

Conclusion: ***Speckle tracking echocardiography provides a reliable, non-invasive method for detecting diastolic dysfunction, demonstrating a strong association between hypertension and elevated LVEDP.***

Keywords: left atrium; 2D speckle tracking strain; HFpEF; LVEDP; HTN

1. Introduction

Congestive heart failure (CHF), particularly diastolic heart failure, has high morbidity and mortality, yet challenges in defining diastolic dysfunction hinder early detection.[1] Hypertension is a major contributor to diastolic dysfunction and heart failure with preserved ejection fraction (HFpEF), leading to left ventricular (LV) hypertrophy, increased filling pressures, and left atrial (LA) enlargement.[2] Although cardiac catheterization is the gold standard for assessing LV diastolic properties and filling pressures, its invasive nature limits routine application. Due to the complex and multifactorial nature of diastolic function, diagnosis cannot rely on a single echocardiographic parameter. Current guidelines advocate for a comprehensive algorithm integrating multiple measures, such as indexed left atrial volume (LAV), E-wave velocity, E/é ratio, and tricuspid regurgitation velocity. It is important to recognize that Doppler-derived parameters reflect an instantaneous state of LV diastolic function, which can acutely vary with changes in loading conditions, necessitating careful interpretation in clinical assessments.[3] Advances in echocardiography enable volume-based LA assessments, improving the evaluation of LV filling pressures.[4,5]

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Speckle tracking echocardiography further refines LA function analysis by quantifying strain and deformation through time-left atrial volume curves (TLAVCs) enabling the precise assessment of LA function by measuring three key volumes: maximal LA volume (LAVmax) at left ventricular (LV) end-systole, pre-atrial contraction LA volume (LAVpreA) at the ECG P-wave onset, and minimal LA volume (LAVmin) at LV end-diastole. Phasic LA functions are quantified through total LA ejection fraction (LAEF) for reservoir function, passive LAEF for conduit function, and active LAEF for booster pump function. These are calculated as Total LAEF = [(LAVmax − LAVmin)/LAVmax] × 100%, Passive LAEF = [(LAVmax − LAVpreA)/LAVmax] × 100%, and Active LAEF = [(LAVpreA − LAVmin)/LAVpreA] × 100%. These parameters provide a comprehensive evaluation of LA function across the cardiac cycle.[6] This study evaluates diastolic function in hypertensive patients, focusing on LV end-diastolic pressure (LVEDP), using speckle tracking echocardiography to improve diagnostic accuracy and patient management.

2. Patients and methods

This study was conducted in the Echocardiography and Cardiac Catheterization Laboratory of the Cardiology Department at Bab El Sharia University Hospital, Al-Azhar University, from January 2024 to September 2024. Patients who were over 18 years of age presenting with angina or angina-equivalent symptoms and normal left ventricular (LV) systolic function from both genders were included. The participants were divided into two groups: a case group of 25 hypertensive patients and a control group of 25 normotensive individuals. Patients with impaired LV systolic function, poor echocardiographic window, multivessel coronary artery disease, prior CABG surgery, significant primary valvular or pericardial disease, arrhythmias, and those with advanced renal, hepatic, or pulmonary conditions were excluded. The study received Ethics Committee approval from Al-Azhar University Hospitals. All participants provided informed consent.

Patients were examined to obtain clinical information such as weight, body mass index (BMI), and BP measurement in both arms. Labs, which include CBC, electrolytes, hepatic function tests, and serum creatinine, were obtained. LVEDP was invasively measured via left heart catheterization using a Philips Allura Xper FD10/10 and its associated fluid-filled system through Retrograde access to the left ventricle using an angled pigtail-shaped catheter. Echocardiographic examination: was performed with a Philips Infinity CVx ultrasound machine, following ASE guidelines.[7,8] The study recorded various parameters, including left ventricular end-diastolic and end-systolic volumes, measured using a modified Simpson's biplane volumetry technique. The left ventricular ejection fraction was subsequently calculated. Pulsed-wave Doppler was employed to obtain mitral inflow and mitral annulus tissue Doppler measurements through the apical 4-chamber view, recording peak early filling velocity (E), peak atrial velocity (A), E/A ratio, E deceleration time, E', A,' and E/è.' Speckle tracking echocardiography was used to assess left atrial (LA) volumes and function. LA volumes were measured as maximal LA volume (LAVmax) at LV end-systole, pre-atrial contraction LA volume (LAVpreA) at the onset of the P-wave on the ECG, and minimal LA volume (LAVmin) at LV end-diastole. Phasic LA function was evaluated through total LA ejection fraction (LAEF) for reservoir function, passive LAEF for conduit function, and active LAEF for booster pump function, calculated using specific formulas for each phase. Finally, left ventricular end-diastolic pressure (LVEDP) was estimated using the Kawasaki-Tanaka (KT) index, expressed as LVEDP = 10.8–12.4 [log10 (active LAEF/minimum LAV)], which has been previously validated in estimating LVEDP.8

Statistical analysis

Statistical analysis utilized SPSS 28, presenting data as mean ± SD, median, or percentage. Echocardiographic parameters were compared to catheterization, with significance at $p < 0.05$.[9]

3. Results

The study compared 27 hypertensive (HTN) and 23 normotensive patients (total N=50). Mean age was similar between groups. Sex distribution (male: HTN 63.0%, normotensive 69.6%) and smoking prevalence (HTN 51.9%, normotensive 43.5%) did not differ significantly. Overall age, sex, and smoking status were comparable between groups. [Table 1].

Table 1. Patient demographic data Stratification according to HTN status

	HTN (N=27)	Normotensive (N=23)	Total (N=50)	p value
Age				0.527[1]
Mean (SD)	53.9 (6.9)	52.4 (9.2)	53.2 (8.0)	
Range	40.0 - 67.0	37.0 - 68.0	37.0 - 68.0	
BMI				0.008[1]
Mean (SD)	30.7 (3.1)	28.1 (3.3)	29.5 (3.4)	
Range	21.9 - 35.0	22.0 - 34.0	21.9 - 35.0	
sex				0.623[2]
Male	17.0 (63.0%)	16.0 (69.6%)	33.0 (66.0%)	
DM				0.042[2]
Yes	11.0 (40.7%)	16.0 (69.6%)	27.0 (54.0%)	
Smoking				0.555[2]

Yes	14.0 (51.9%)	10.0 (43.5%)	24.0 (48.0%)

The study confirmed preserved left ventricular function with an M-Mode EF% of 62.7 ± 5.5% and a Modified Simpson EF% of 59.1 ± 5.4%. Doppler analysis showed a peak E-wave of 86.1 ± 19.2 cm/s, E/A ratio of 2.0 ± 0.9, and deceleration time of 177.2 ± 26.3 ms. Tissue Doppler Imaging indicated diastolic dysfunction with an E/E' ratio of 15.8 ± 5.9, particularly in hypertensive patients. [Table 2].

Table 2. 2D Echo doppler data of the patients studied.

	Total patients)n=50(	
	Mean (SD)	Range
Functional assessment		
M-Mode EF (%)	62.7 (5.5)	54.0 - 75.0
Modified Simpson EF (%)	59.1 (5.4)	50.0 - 69.0
PW trans mitral inflow		
Peak E wave (cm/s)	86.1 (19.2)	42.0 - 120.0
Peak A wave (cm/s)	50.2 (20.7)	23.0 - 105.0
E/A ratio	2.0 (0.9)	0.4 - 3.9
E Decel. time (ms)	177.2 (26.3)	127.0 - 250.0
Tissue Doppler Imaging (TDI)		
Peak E` (cm/s)	5.9 (1.7)	2.9 - 9.1
Peak A` (cm/s)	5.7 (1.5)	1.8 - 11.5
E/E` ratio	15.8 (5.9)	6.9 - 31.6

The study utilized 2D speckle tracking echocardiography to assess left atrial function. The mean LA reservoir strain was 32.2 ± 6.1%, while conduit and contractile strains were -17.3 ± 6.0% and -14.0 ± 4.1%, respectively. LA volume indices showed a maximum LAVI of 41.3 ± 13.7 ml/m², with total LAEF at 40.8 ± 11.7%, indicating significant atrial dysfunction, particularly in hypertensive patients [Table 3].

Table 3. Speckle Tracking of the studied patients

	Total patients)n=50(	
	Mean (SD)	Range
LA strain		
LA reservoir Strain (%)	32.2 (6.1)	16.0 - 44.0
LA Conduit Strain (%)	-17.3 (6.0)	-33.0 - -4.0
LA Contractile Strain (%)	-14.0 (4.1)	-25.0 - -2.0
LA Indexes		
Max. LAVI (ml/m2)	41.3 (13.7)	26.0 - 85.0
Pre-A LAVI (ml/m2)	32.9 (12.8)	19.0 - 78.0
Minimum (ml/m2)	25.3 (12.6)	11.0 - 70.0
LA function		
Total LAEF (%)	40.8 (11.7)	17.0 - 62.0
Passive LAEF (%)	21.2 (7.7)	7.0 - 44.3
Active LAEF (%)	25.8 (11.1)	9.0 - 52.1

A paired samples t-test was conducted to assess differences between estimated and measured left ventricular end-diastolic pressure (LVEDP) [Table 4].

Table 4. Comparison Between Measured and Estimated L.V.EDP among studied patients

PAIRED SAMPLES T-TEST

			Statistic	P	Mean difference	SE difference
ESTIMATED L.V.EDP	Measured L.V.EDP	Wilcoxon W	81.00	< .001	-0.96	0.12

NOTE. H_a $M_{MEASURE\ 1\ -\ MEASURE\ 2} \neq 0$

The analysis yielded a Wilcoxon W statistic of 81.00 ($p < 0.001$), confirming a significant difference between estimated and measured LVEDP. The mean difference was -0.96 (SE = 0.12), indicating slightly lower estimated values. A strong correlation existed, but hypertensive patients exhibited higher measured LVEDP, suggesting hypertension contributes to elevated LV pressure [Table 5].

Relation between measured and estimated LVEDP in patients with HTN and normal blood pressure showed a strong positive correlation between estimated and measured LVEDP in both groups. The slopes of the lines in Scatterplot of [figure 1] suggest that the estimation method used may be similarly effective across hypertensive and non-hypertensive individuals, though the hypertensive group seems to have slightly higher measured LVEDP values at comparable estimated LVEDP levels, indicating a possible effect of hypertension on LVEDP elevation [figure 1].

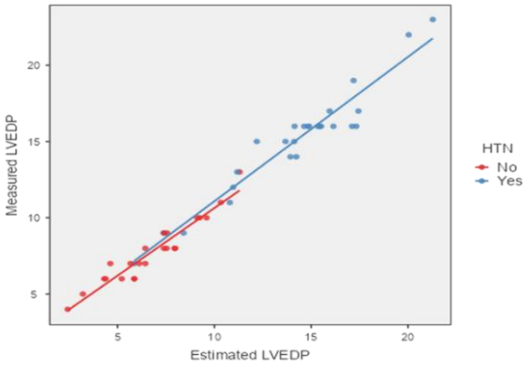

Figure 1. Scatterplot describe the relationship between measured and estimated L.V.EDP in patients with HTN and normal blood pressure.

Table 5. Comparisons of Measured L.V.EDP and Estimated L.V.EDP Between Patients with HTN and Normal Blood Pressures among studied patients.

	HTN (N=27)	Normotensive (N=23)	Total (N=50)	p value
Estimated L.V.EDP				< 0.001[1]
Mean (SD)	14.0 (3.7)	6.8 (2.3)	10.7 (4.8)	
Range	5.9 - 21.3	2.4 - 11.3	2.4 - 21.3	
Measured L.V.EDP				< 0.001[1]
Mean (SD)	14.9 (3.7)	7.8 (2.1)	11.6 (4.7)	
Range	7.0 - 23.0	4.0 - 13.0	4.0 - 23.0	
KT index				< 0.001[1]
Mean (SD)	-0.3 (0.3)	0.3 (0.2)	0.0 (0.4)	
Range	-0.8 - 0.4	-0.0 - 0.7	-0.8 - 0.7	

When comparing hypertensive (HTN) and normotensive groups, the HTN group had a significantly higher estimated LVEDP (14.0 ± 3.7 mmHg) compared to the normotensive group (6.8 ± 2.3 mmHg, $p < 0.0011$) [*Table 6*].

Table 6. Comparisons of Two-dimensional Echo doppler data Between Patients with HTN and Normal Blood Pressures among studied patients.

	Normotensive (N=23)	HTN (N=27)	Total (N=50)	p value
EF % (M-MODE)				0.544[1]
Mean (SD)	63.3 (5.9)	62.3 (5.3)	62.7 (5.5)	
Range	54.0 - 75.0	54.0 - 72.0	54.0 - 75.0	
EF% (modified Simpson)				0.770[1]
Mean (SD)	59.3 (5.6)	58.9 (5.3)	59.1 (5.4)	
Range	50.0 - 69.0	50.0 - 67.0	50.0 - 69.0	
Peak E wave cm/s				0.271[1]
Mean (SD)	89.4 (15.9)	83.3 (21.6)	86.1 (19.2)	
Range	45.0 - 120.0	42.0 - 117.0	42.0 - 120.0	
Peak A wave cm/s				0.100[1]
Mean (SD)	45.0 (15.7)	54.7 (23.5)	50.2 (20.7)	
Range	23.0 - 78.0	23.0 - 105.0	23.0 - 105.0	
E: A Ratio				0.163[1]
Mean (SD)	2.2 (0.8)	1.9 (0.9)	2.0 (0.9)	
Range	0.7 - 3.9	0.4 - 3.9	0.4 - 3.9	
E Decel. Time(ms)				0.213[1]
Mean (SD)	172.2 (17.8)	181.5 (31.5)	177.2 (26.3)	
Range	150.0 - 220.0	127.0 - 250.0	127.0 - 250.0	
Peak E_ cm/s				< 0.001[1]
Mean (SD)	7.0 (1.5)	5.0 (1.2)	5.9 (1.7)	
Range	3.7 - 9.1	2.9 - 8.0	2.9 - 9.1	
Peak A_ cm/s				0.409[1]
Mean (SD)	5.5 (1.2)	5.9 (1.8)	5.7 (1.5)	
Range	3.0 - 8.2	1.8 - 11.5	1.8 - 11.5	
E/E_ ratio				0.007[1]
Mean (SD)	13.4 (3.9)	17.9 (6.6)	15.8 (5.9)	
Range	6.9 - 24.1	7.0 - 31.6	6.9 - 31.6	

Similarly, measured LVEDP was significantly elevated in hypertensive patients (14.9 ± 3.7 mmHg) versus normotensive individuals (7.8 ± 2.1 mmHg, $p < 0.0011$). The Kawasaki-Tanaka (KT) index, an echocardiographic predictor of LVEDP, showed a significant difference between groups.

The HTN group had a KT index of -0.3 ± 0.3, whereas the normotensive group had a KT index of 0.3 ± 0.2 ($p < 0.0011$). These findings suggest that the KT index correlates well with increased LVEDP in hypertensive patients.

Left ventricular ejection fraction remained preserved across both groups, with no significant differences in M-Mode EF% ($p = 0.5441$) or Modified Simpson's EF% ($p = 0.7701$). However, hypertensive patients exhibited impaired diastolic function, with lower peak E' velocity ($p < 0.0011$) and a higher E/E' ratio ($p = 0.0071$).

Hypertension significantly impacted left atrial strain and function. The HTN group demonstrated lower LA reservoir strain (28.0 ± 4.3%) compared to the normotensive group (37.1 ± 3.7%, $p < 0.0011$). LA conduit strain (-14.3 ± 4.6%) and contractile strain (-11.7 ± 3.3%) were also reduced in hypertensive patients compared to normotensive controls (-20.8 ± 5.6% and -16.7 ± 3.2%, respectively, $p < 0.0011$) [Table 7].

Table 7. Comparisons of 2D speckle tracking data of Left atrial Between Patients with HTN and Normal Blood Pressures among studied patients.

	Normotensive (N=23)	HTN (N=27)	Total (N=50)	p value
LA Reservoir Strain %				< 0.001[1]
Mean (SD)	37.1 (3.7)	28.0 (4.3)	32.2 (6.1)	
Range	30.0 - 44.0	16.0 - 37.0	16.0 - 44.0	
LA Conduit Strain %				< 0.001[1]
Mean (SD)	-20.8 (5.6)	-14.3 (4.6)	-17.3 (6.0)	
Range	-33.0 - -14.0	-27.0 - - 4.0	-33.0 - - 4.0	
LA Contractile Strain %				< 0.001[1]
Mean (SD)	-16.7 (3.2)	-11.7 (3.3)	-14.0 (4.1)	
Range	-25.0 - -10.0	-16.0 - - 2.0	-25.0 - - 2.0	
Maximum LAVI (ml/m2)				< 0.001[1]
Mean (SD)	31.3 (3.0)	49.9 (13.4)	41.3 (13.7)	
Range	26.0 - 36.0	32.0 - 85.0	26.0 - 85.0	
Pre-AC LAVI (ml/m2)				< 0.001[1]
Mean (SD)	23.6 (2.6)	40.9 (12.7)	32.9 (12.8)	
Range	19.0 - 29.0	24.0 - 78.0	19.0 - 78.0	
Minimum LAVI (ml/m2)				< 0.001[1]
Mean (SD)	15.6 (2.6)	33.6 (11.8)	25.3 (12.6)	
Range	11.0 - 22.0	15.0 - 70.0	11.0 - 70.0	
Total LAEF %				< 0.001[1]
Mean (SD)	49.4 (6.9)	33.5 (9.9)	40.8 (11.7)	
Range	38.3 - 62.0	17.0 - 55.5	17.0 - 62.0	
Passive LAEF %				0.006[1]
Mean (SD)	24.3 (6.0)	18.5 (8.0)	21.2 (7.7)	
Range	15.6 - 40.0	7.0 - 44.3	7.0 - 44.3	
Active LAEF%				< 0.001[1]
Mean (SD)	33.6 (8.7)	19.1 (8.3)	25.8 (11.1)	
Range	17.3 - 52.1	9.0 - 37.5	9.0 - 52.1	

Hypertensive patients exhibited significantly larger left atrial volumes, with maximum LAVI (49.9 vs. 31.3 ml/m², $p < 0.0011$), pre-atrial contraction LAVI (40.9 vs. 23.6 ml/m², $p < 0.0011$), and minimum LAVI (33.6 vs. 15.6 ml/m², $p < 0.0011$), indicating increased atrial remodeling.

Hypertension significantly impairs left atrial function, with hypertensive patients exhibiting lower total (33.5% vs. 49.4%, $p < 0.0011$), passive (18.5% vs. 24.3%, $p = 0.0061$), and active LAEF (19.1% vs. 33.6%, $p < 0.0011$), indicating reduced strain, increased volume, and compromised emptying fractions.

4. Discussion

Hypertension is a major contributor to HFpEF, causing LV hypertrophy and LA dysfunction, necessitating advanced echocardiographic assessment for early detection and management.

Previous studies suggest that hypertension contributes to HFpEF by impairing LV diastolic

filling and inducing LA structural changes, which indicate LV dysfunction severity. Assessing LA function is crucial for early detection. Advances in 2D speckle tracking echocardiography enable early identification of LA-LV abnormalities in hypertensive patients before significant structural or functional deterioration occurs.[10]

Our study, which included 50 patients with normal ejection fraction (25 hypertensive, 25 normotensive), had a mean participant age of 53.2 years, with 66% of the cohort being male. Comorbidities were common, with diabetes present in 54% of participants, hypertension in 52%, and smoking history in 48%. The mean BMI was 29.5 kg/m², and the mean body surface area (BSA) was 1.9 m². Invasive left heart catheterization was used to measure LV end-diastolic pressure (LVEDP), which ranged from 4 to 23 mmHg, with a mean of 11.6 mmHg. A comprehensive echocardiographic assessment of LA function was conducted using volumetric analysis, speckle tracking, and Doppler-based methods. The mean LV ejection fraction was 62.7% using M-mode and 59.1% using Simpson's method. Doppler-derived trans-mitral inflow velocities showed an average E-wave velocity of 86.1 cm/s and an A-wave velocity of 50.2 cm/s, with an E/A ratio of 2.0 and a deceleration time of 177.2 ms. Tissue Doppler Imaging revealed an E′ velocity of 5.9 cm/s, an A′ velocity of 5.7 cm/s, and an E/E′ ratio of 15.8.

The study results revealed significant differences in LV diastolic function between hypertensive and normotensive patients. Specifically, hypertensive patients exhibited significantly higher estimated LVEDP ($p < 0.0011$), measured LVEDP ($p < 0.0011$), and KT index ($p < 0.0011$), indicating worse LV diastolic function. Conversely, hypertensive patients had significantly lower passive LAEF% ($p = 0.0061$), indicating impaired left atrial relaxation, as well as lower active LAEF% ($p < 0.0011$), total LAEF% ($p < 0.0011$), and active LAEF/minimum left atrial volume index (LAVI) ($p < 0.0011$), suggesting decreased LA ejection fraction. A paired samples t-test was performed to compare estimated and measured LVEDP, yielding a Wilcoxon W statistic of 81.00 and a p-value of <0.001, indicating a statistically significant difference. The mean difference was -0.96, with a standard error of 0.12. Despite statistical significance, the clinical relevance of this discrepancy is minimal.

The comparison between hypertensive (N=27) and normotensive (N=23) groups demonstrated significant differences in peak E′ wave velocity, E/E′ ratio, LA reservoir strain, LA conduit strain, LA contractile strain, maximum LAVI, pre-A LAVI, minimum LAVI, total LAEF, passive LAEF, and active LAEF. The hypertensive group exhibited reduced LA reservoir strain (28.0 ± 4.3%), conduit strain (-14.3 ± 4.6%), and contractile strain (-11.7 ± 3.3%), along with increased maximum, pre-A, and minimum LAVI values (49.9 ± 13.4 ml/m², 40.9 ± 12.7 ml/m², and 33.6 ± 11.8 ml/m², respectively). Additionally, hypertensive patients demonstrated lower total LAEF (33.5 ± 9.9%), passive LAEF (18.5 ± 8.0%), and active LAEF (19.1 ± 8.3%) compared to normotensive patients. However, there were no significant differences between the two groups regarding LV ejection fraction (EF%), peak E-wave velocity, peak A-wave velocity, E/A ratio, or E-wave deceleration time. These findings strongly suggest that hypertension is associated with impaired LA function and mechanics, characterized by reduced strain, increased volume, and decreased emptying fractions.

Our findings are in agreement with the study by Masanori Kawasaki, which introduced the kinetics-tracking index (KT index) as a novel ultrasound-based predictor of pulmonary capillary wedge pressure (PCWP) and LVEDP in patients without significant mitral valve disease, using speckle tracking echocardiography (STE). Kawasaki's validation study defined the KT index in a training cohort of 50 patients and subsequently tested it in a cohort of 196 patients. The KT index, calculated as log10 (active left atrial emptying fraction/minimum LAV index), demonstrated robust correlations with PCWP and LVEDP ($r = 0.86$ and $r = 0.92$ in training and testing cohorts, respectively), outperforming traditional echocardiographic markers such as the E/E′ ratio. This study emphasized the clinical utility of the KT index in managing heart failure, particularly HFpEF, by providing a non-invasive and reliable method for estimating PCWP and LVEDP, which may improve diagnostic and therapeutic approaches.[8]

Our findings also align with those of Flemming Javier Olsen et al., whose study was published in The International Journal of Cardiovascular Imaging in May 2021. This study examined the relationship between left atrial function and LVEDP in 43 patients undergoing coronary angiography. Among these patients, 65% exhibited elevated LVEDP, suggesting widespread diastolic dysfunction. The study assessed total, active, and passive LAEF as functional measures, finding that total and active LAEF correlated significantly with LVEDP, while LA volume measurements lacked a strong association. Notably, passive LAEF emerged as the strongest predictor of elevated LVEDP, underscoring its value as a non-invasive marker of diastolic dysfunction. These findings highlight the critical

role of LA function in diagnosing and managing heart failure in patients with non-dilated left atria, advocating for the integration of these functional parameters into routine clinical assessments to enhance diagnostic accuracy and treatment strategies.[11]

Additionally, our results are consistent with a 2018 study by Bassam Hennawy, published in The Egyptian Heart Journal, which investigated early LA dysfunction in hypertensive patients using STE at Ain Shams University Hospital. This case-control study enrolled 50 hypertensive patients and 50 normotensive controls with normal LA volume indices. Despite normal LA size, hypertensive patients exhibited significantly reduced LA function, as indicated by lower total LA stroke volume, LA expansion index, and global peak atrial longitudinal strain (PALS). A further association was noted between decreased global PALS and diabetes mellitus, advanced left ventricular diastolic dysfunction, older age, higher systolic blood pressure, increased body mass index, and greater left ventricular mass index. The study highlighted the value of STE in detecting early LA dysfunction even when LA enlargement was absent, reinforcing the need for early echocardiographic surveillance in hypertensive patients to prevent complications such as atrial fibrillation and stroke. These findings support the importance of proactive monitoring and timely interventions for hypertensive individuals at risk of heart failure.[12]

4. Conclusion

Assessment of left atrial (LA) function using 2D echocardiography and speckle tracking reliably estimates LVEDP, aiding early diastolic dysfunction diagnosis. Findings confirm a direct correlation between hypertension and elevated LVEDP, supporting LA function as a valuable echocardiographic marker for evaluating diastolic function, particularly in hypertensive patients with normal ejection fraction.

Disclosure

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