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ASSOCIATION OF LEFT ATRIAL VOLUME INDEX WITH DIASTOLIC DYSFUNCTION IN HYPERTENSIVE PATIENTS: A CROSS-SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL

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Abstract

Introduction: *Longstanding hypertension frequently leads to an underrecognized condition of left ventricular diastolic dysfunction (LVDD), which often precedes heart failure (HF). Left atrial volume index (LAVI), a surrogate for the cumulative effect of elevated left ventricular filling pressure, has been proposed as an echocardiographic marker of LVDD.* ***Objectives***: *This study examined the association between LAVI and the presence and severity of LVDD in hypertensive patients.* ***Methodology***: *In this cross-sectional study, transthoracic echocardiography was performed on 217 hypertensive patients. LAVI was measured by the biplane area–length method and indexed to body surface area. Diastolic function was classified as normal or Grade I–III LVDD using the 2016 ASE/EACVI multiparametric algorithm. Group differences, correlations, multivariable linear and logistic regression, and receiver operating characteristic (ROC) analyses were performed.* ***Results***: *The mean age was 59.78 ± 14.87 years, and LVDD was present in 148 patients (68.2%). Mean LAVI was 30.85 ± 5.76 mL/m² in participants with normal diastolic function, compared with 29.50 ± 5.70, 44.37 ± 3.05, and 45.04 ± 2.82 mL/m² in Grade I, II, and III LVDD, respectively. LAVI did not differ between normal function and Grade I ($p = 0.361$) but was substantially higher in Grade II/III than in the normal/Grade I groups ($p < 0.001$). LAVI correlated strongly with E/e′ ($r = 0.771$, $p < 0.001$) and TR velocity ($r = 0.754$, $p < 0.001$). In multiple linear regression, E/e′ ($\beta = 0.771$, $p < 0.001$), LA diameter ($\beta = 0.579$, $p < 0.001$), and TR velocity ($\beta = 0.322$, $p < 0.001$) were independently associated with LAVI. In binary logistic regression, E/e′ (OR = 1.660, 95% CI: 1.352–2.040, $p < 0.001$) was independently associated with LVDD, while BMI showed an inverse association (OR = 0.940, 95% CI: 0.890–0.993, $p = 0.026$). LAVI showed moderate discriminatory ability for LVDD (AUC = 0.704, 95% CI: 0.637–0.771), with 61.5% sensitivity and 66.7% specificity at a cutoff of 34.0 mL/m².*
Conclusion: *In hypertensive patients, LAVI is associated with more advanced grades of LVDD and correlates with Doppler markers of elevated*

filling pressure. Its discriminatory ability for LVDD is moderate, supporting its role as one component of a multiparametric assessment rather than a standalone diagnostic test.

INTRODUCTION

Hypertension (HTN) stands as one of the world's foremost public health concerns, playing a significant role in cardiovascular disease and death (1). Global estimates from 2019 put the number of adults between ages 30 and 79 affected by high blood pressure at around 1.28 billion. When left untreated or poorly managed, elevated blood pressure substantially raises the likelihood of ischemic heart disease, kidney disease, heart failure, and Stroke. Since hypertension often produces no noticeable symptoms for extended periods, catching signs of cardiovascular organ damage early is critical (2).

Hypertension is also a major health concern in Pakistan. National data have reported substantial variation in the prevalence of HTN among Pakistani adults, with estimates ranging from approximately 26% to 44% (3). Inadequate diagnosis, treatment, and blood-pressure control remain important challenges. A multicenter study involving more than 9,000 participants reported that although 48.6% of individuals with hypertension had been diagnosed, only 34.5% were receiving antihypertensive treatment and 12.3% had controlled blood pressure (4).

Sustained elevation of blood pressure imposes chronic mechanical overload on the left ventricle and activates key neurohormonal pathways, most notably the sympathetic nervous system and the RAAS, both of which mediate structural and functional cardiac remodeling (5). (Figure1) LV hypertrophy represents one of the most well-characterized manifestations of this remodeling process. Although hypertrophy initially serves as a compensatory adaptation to increased afterload, its progressive development, together with concomitant myocardial fibrosis, leads to increased LV wall stiffness and impaired myocardial relaxation, ultimately contributing to the development of diastolic dysfunction (LVDD) (6).

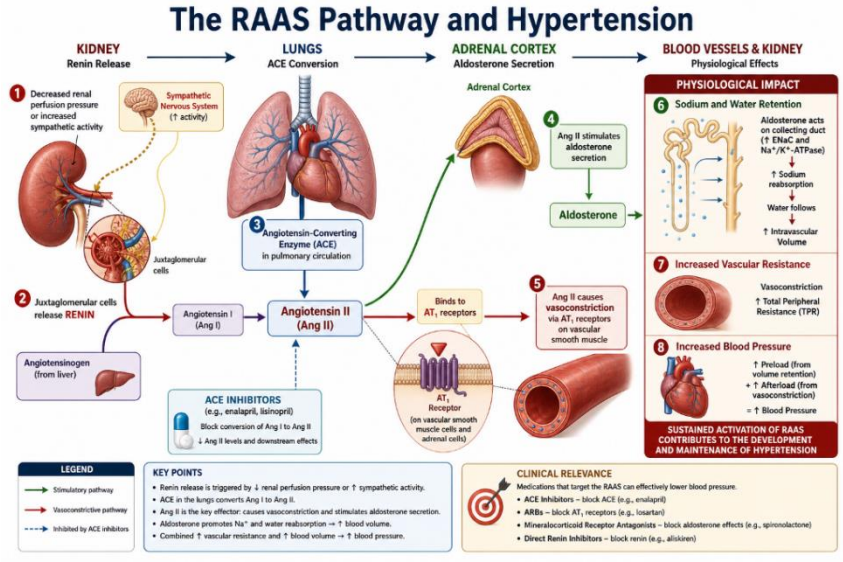


Figure 1: Mechanism of Hypertension

LVDD constitutes an important indication of hypertensive heart disease and may precede the onset of overt heart failure and systolic impairment. Impaired myocardial relaxation coupled with increased ventricular stiffness leads to elevated LV filling pressures, which are subsequently transmitted retrograde to the LA and pulmonary vasculature. This hemodynamic

cascade contributes to LA enlargement, pulmonary hypertension, and, ultimately, the progression to clinical heart failure (7).

Echocardiography provides a non-invasive method for detecting subclinical hypertensive cardiac remodeling. Two-dimensional and Doppler echocardiography can simultaneously evaluate LV structure, systolic and diastolic function, and LA volume (8). Among these parameters, LAVI is particularly relevant because LA enlargement reflects the cumulative effect of chronically elevated LV filling pressures (9).

The LA responds to chronic pressure overload through structural and functional remodeling (10). In hypertension, increased left ventricular stiffness and impaired relaxation require higher filling pressures to maintain ventricular filling. These elevated pressures are transmitted to the LA, producing progressive atrial remodeling, fibrosis, and enlargement. Thus, left atrial enlargement may serve as an indicator of chronic rather than transient abnormalities in ventricular filling (11).

LAVI is calculated by dividing the LA volume by the body surface area and provides a standardized measure of left atrial size. The normal LAVI has been established by the ASE 2015 guidelines on cardiac chamber measurement and is ≤34 ml/m^2 (12). Elevated LAVI has likewise been associated with adverse cardiovascular outcomes, including atrial fibrillation, stroke, heart failure, and cardiovascular mortality (13).

Assessment of diastolic function has evolved substantially with the development of Doppler and tissue-Doppler echocardiography. Mitral inflow parameters such as the velocities of E and A, E/A ratio provide information regarding ventricular relaxation and filling. Other parameters, including E/e′, deceleration time, IVRT, and TR velocity, provide complementary information regarding ventricular filling and associated hemodynamic changes. The 2025 ASE/EACVI recommendations established a multiparametric approach to the assessment and grading of LV diastolic function (14).

Previous research has demonstrated an association between LAVI and diastolic dysfunction. Studies have reported progressive increases in LAVI with worsening grades of LVDD, supporting its role as an indicator of chronic elevation of LV filling pressure (13). Literature also identifies evidence linking LAVI with invasively measured filling pressures and demonstrating its complementary diagnostic value alongside E/e′ and other Doppler parameters (15–19).

Despite the established relationship between hypertension, diastolic dysfunction, and left atrial remodeling, limited local evidence exists regarding the association between LAVI and the severity of LVDD among hypertensive patients in Khyber Pakhtunkhwa.

Materials and methods:

This cross-sectional study was conducted in the Cardiology Department of District Headquarters (DHQ) Hospital, Swabi. A minimum sample size of 217 participants was calculated using the formula: $n = Z^2 \times p(1-p) / d^2$, where Z=1.96 at a 95% confidence level, p= 0.83 (20), and d= 5% margin of error.

Participants were enrolled via consecutive non-probability sampling from the Cardiology Outpatient Department and the Echocardiography Section.

Adult patients aged 20–70 years were eligible for inclusion if they had known hypertension, LVEF ≥50%, good-quality echocardiographic images, and were willing to provide written informed consent. Non-hypertensive individuals were excluded. Patients with LVEF <50%, valvular heart disease, pacemaker-dependent ventricular activation, cardiomyopathies, congenital heart disease, atrial fibrillation or sustained atrial arrhythmias, acute myocardial infarction, previous cardiac surgery, pregnancy, or inability or refusal to provide informed consent were excluded. Ethical clearance was secured from the institutional ethics committee before conducting the study. Confidentiality and privacy of participants were maintained throughout the study, and participation was entirely voluntary.

Definitions. Hypertension: BP ≥140/90 mmHg (repeated reading), hypertension diagnosis or under treatment with antihypertensive. Controlled BP: < 140/90 mmHg at visit. LVH:

LV mass index (Devereux formula) indexed to BSA >115g/m² (men) / >95g/m² (women) (21).
BP Measurement: sitting, ≥ 5 minutes rest, automated oscillometric device calibrated, correct cuff size, 2 readings 1-2 minutes apart, average (22).
Echo assessment: Transthoracic echo by trained cardiologist/technologist (Canon Aplio A with phased array probe), with the patient in left lateral decubitus, using standard parasternal/apical views, and averaging over 3 sinus-rhythm cycles.
The LA volume was obtained using the biplane area-length method (apical 4- and 2-chamber views) at end-systole (pre-mitral-valve-opening frame), with the endocardium traced excluding pulmonary veins, LA appendage, and mitral annular plane area. Calculated as $(8/3\pi) \times (A4C \times A2C)/L$, indexed to BSA (Du Bois) → LAVI. Classified per ASE/EACVI: normal ≤34, mild 35–41, moderate 42–48, severe >48 mL/m² (23).
Doppler: Mitral E/A velocities and deceleration time, via PWD, at leaflet tips; septal/lateral e′ via TWD, averaged for E/e′ ratio. The velocity of the TR peak was measured by IVRT, and the velocity of the TR peak was measured by CWD using multiple windows. LVEF was calculated using biplane Simpson's technique.
Diastolic function classification: Per the guidelines of the ASE/EACVI 2016 guidelines, using 5 criteria: septal e′ <7 cm/s, lateral e′ <10 cm/s, average E/e′ >14, TR velocity >2.8 m/s, LAVI >34 mL/m². Dysfunction exists when more than half of the criteria are positive; it doesn't exist when more than half of the criteria are negative, and it's indeterminate when exactly half (24).
Grading: Grade I (E/A ≤0.8, E ≤50 cm/s); Grade II (E/A ≤0.8 with E >50 cm/s, or E/A 0.8–2.0 with ≥2 of E/e′, TR velocity, LAVI positive); Grade III (E/A ≥2.0).
Data were collected using a pre-designed, structured, and validated data collection proforma developed specifically for the study. Blood pressure was assessed during clinical examination, while LAVI and LVH were assessed by echocardiography. The grade of LVDD was classified according to the recorded echocardiographic measurements. Data were analyzed with IBM SPSS Statistics, version 22.0.

Results:
A total of 217 hypertensive patients were enrolled. Participants had a mean age of 59.78 ± 14.87 years, a mean BMI of 26.51 ± 6.58 kg/m², and a mean hypertension duration of 7.90 ± 4.10 years. Women constituted 54.8% (119/217) of the study population and men 45.2% (98/217). Regarding residence, 53.9% (117/217) of participants were from urban areas and 46.1% (100/217) from rural areas. Mean systolic and diastolic blood pressures were 148.56 ± 17.54 mmHg and 93.08 ± 8.86 mmHg, respectively. Controlled blood pressure was documented in 109 participants (50.2%). Diabetes mellitus was present in 121 participants (55.8%), ischemic heart disease in 111 (51.2%), and previous stroke in 113 (52.1%). Among participants, 77 (35.5%) were current smokers, 58 (26.7%) former smokers, and 82 (37.8%) non-smokers (Table 1).
On echocardiography, the mean left atrial size of 39.87 ± 7.45 mm, mean LAVI was 34.92 ± 8.49 mL/m², and mean LVEF of 56.06 ± 6.64%. The mean E/e′ ratio was 10.92 ± 4.59, mean IVRT was 84.88 ± 25.28 ms, mean TR velocity was 2.67 ± 0.45 m/s, and mean DT was 189.16 ± 34.47 ms. LVH was detected in 112 (51.6%) of patients. (Table 2)
Diastolic dysfunction was identified in 148 participants (68.2%), while 69 (31.8%) had normal diastolic function. Among those with diastolic dysfunction, 77 (35.5%) had Grade I, 31 (14.3%) had Grade II, and 40 (18.4%) had Grade III dysfunction. Regarding LAVI, 103 participants (47.5%) had normal LAVI (≤34 mL/m²), whereas 114 (52.5%) had increased LAVI (>34 mL/m²): 53 (24.4%) mild, 48 (22.1%) moderate, and 13 (6.0%) severe enlargement (Table 3).
Mean LAVI was 30.85 ± 5.76 mL/m² in participants with normal diastolic function, compared with 29.50 ± 5.70, 44.37 ± 3.05, and 45.04 ± 2.82 mL/m² in those with Grade I, II, and III dysfunction, respectively (Table 3). On post-hoc testing, LAVI did not differ significantly between the normal-function and Grade I group ($p = 0.361$). Marked differences were evident between the combined normal/Grade I group and the combined Grade II/Grade III group ($p < 0.001$), whereas Grade II and Grade III did not differ

significantly (p = 0.946). Overall, mean LAVI was significantly higher in participants with diastolic dysfunction than in those with normal function (36.81 ± 8.90 vs 30.85 ± 5.76 mL/m², p < 0.001) (Table 3).

Correlation analysis revealed significant associations between LAVI and several echocardiographic indices of diastolic function (Table 4). Strong positive correlations were observed with LA diameter (r = 0.832, p < 0.001), E/e′ (r = 0.771, p < 0.001), and TR velocity (r = 0.754, p < 0.001); a moderate positive correlation was observed with the E/A ratio (r = 0.596, p < 0.001). Moderate negative correlations were noted with deceleration time (r = −0.535, p < 0.001) and IVRT (r = −0.551, p < 0.001). No significant correlations were detected between LAVI and BMI, duration of hypertension, SBP, or DBP (Table 4).

In multiple linear regression analysis, E/e′ ratio (β=0.771, p<0.001), LA size (β=0.579, p<0.001), and TR velocity (β=0.322, p<0.001) were significant predictors of LAVI. Age, BMI, duration of hypertension, SBP, DBP, LVEF, DT, and IVRT were not significantly associated with LAVI. (Table 4)

The inclusive binary logistic regression model was statistically significant (χ^2=78.098, df=9, p<0.001). Of the predictors assessed, the E/e′ ratio was the strongest independent predictor of LVDD (OR=1.660, 95% CI: 1.352–2.040, p<0.001), and BMI was independently associated with LVDD as well, showing a protective direction of effect (OR=0.940, 95% CI: 0.890–0.993, p=0.026). Age, duration of HTN, SBP, LVH, DM, and BP control did not independently predict LVDD in this model.

ROC curve analysis showed that LAVI possessed statistically significant discriminatory power for identifying LVDD, yielding an AUC of 0.704 (95% CI: 0.637–0.771, p<0.001). At an optimal cutoff of 34.0 mL/m², LAVI achieved a sensitivity of 61.5% and a specificity of 66.7%.

Table 1. Baseline Sociodemographic and Clinical Profile of Study Participants

Characteristic	Category / Summary	n (%) / Mean ± SD
Age (years)	Mean ± SD	59.78 ± 14.87
Body mass index (kg/m²)	Mean ± SD	26.51 ± 6.58
Sex	Male	98 (45.2)
	Female	119 (54.8)
Place of residence	Urban	117 (53.9)
	Rural	100 (46.1)
BP Controlled	Yes	109 (49.8)
	No	108(50.2)
Diabetes mellitus	Yes	121(55.8)
	No	96 (44.2)
Ischemic Heart Disease	Yes	111(51.2)
	No	106(48.8)
SBP(mmHg)	Mean ± SD	148.56 ± 17.54
DBP(mmHg)	Mean ± SD	93.08 ± 8.86
Smoking	Current smoker	77 (35.5)
	Former smoker	58 (26.7)
	Non-smoker	82 (37.8)

Table 2: Echocardiographic characteristics (n = 217)

Variable	Mean ± SD / n (%)
LA Size (mm)	39.87 ± 7.45
LAVI (mL/m²)	34.92 ± 8.49
LVEF (%)	56.06 ± 6.64

E Velocity (cm/s)	75.25±28.94
A Velocity (cm/s)	62.06±14.95
E/e′ Ratio	10.92 ± 4.59
IVRT (ms)	84.88±25.28
TR velocity (m/s)	2.67±0.45
DT (ms)	189.16±34.47
LVH	
Present	105 (48.4%)
Absent	112 (51.6%)

Table 3: Distribution & Comparison of LAVI and group-based DD grade among Hypertensive Patients

	n	%/Mean ± SD (mL/m²)	p-value
LAVI Severity			
Normal (≤34 mL/m²)	103	47.5	
Mild Increase (35–41 mL/m²)	53	24.4	
Moderate Increase (42–48 mL/m²)	48	22.1	
Severe Increase (>48 mL/m²)	13	6.0	
Diastolic Function			
Normal	69	30.85 ± 5.76	
Grade I	77	29.50 ± 5.70	
Grade II	31	44.37 ± 3.05	
Grade III	40	45.04 ± 2.82	
Diastolic Dysfunction (overall)	148	36.81 ± 8.90	**<0.001**
Normal vs diastolic dysfunction			**<0.001**
Normal vs Grade I (post-hoc)			**0.361**
Grade II vs Grade III (post-hoc)			**0.946**
Normal/Grade I vs Grade II/III (post-hoc)			**< 0.001**

Table 4: Correlation of the LAVI with Selected Clinical and Echocardiographic Variables

Variable	r	Correlation	p-value
Age	-0.20		>0.05
BMI	0.007		>0.05
Duration of HTN	0.061		>0.05
SBP	0.005		>0.05
DBP	-0.014		>0.05
LA Diameter	**0.832**	**Positive**	**<0.001**
LVEF	0.391		>0.05
E/e′ Ratio	**0.771**	**Positive**	**<0.001**
TR velocity	**0.754**	**Positive**	**<0.001**
DT	**-0.535**	**Negative**	**<0.001**
IVRT	**-0.551**	**Negative**	**<0.001**
LAVI	**0.832**	**Positive**	**<0.001**

DISCUSSION

The present study included a predominantly middle-aged to elderly hypertensive population, with a mean duration of hypertension of 7.90 ± 4.10 years. Prolonged exposure to hypertension may result in progressive left ventricular remodeling, impaired relaxation, increased LV filling pressures, and subsequent left atrial remodeling over time (25).

The prevalence of LV diastolic dysfunction (LVDD) was 68.2% in this study. This finding is comparable with the 74.6% prevalence reported among patients at Khyber Teaching Hospital, Peshawar (26). The relatively high prevalence observed in both studies may reflect the increased cardiovascular risk profile of hospital-based hypertensive populations. The present prevalence was also comparable to the 69.03% reported by Tauqeer et al. among hypertensive patients in Pakistan (27).

Mean LAVI differed significantly across diastolic function categories ($p < 0.001$); however, the pattern was not a uniform stepwise increase. LAVI was similar in participants with normal function and Grade I LVDD (30.85 ± 5.76 vs 29.50 ± 5.70 mL/m², $p = 0.361$) and in Grade II versus Grade III (44.37 ± 3.05 vs 45.04 ± 2.82 mL/m², $p = 0.946$), whereas Grade II/III values were markedly higher than those of the normal/Grade I groups ($p < 0.001$). The more accurate interpretation, therefore, is that LAVI was substantially higher in Grade II/III dysfunction than in normal/Grade I categories, rather than increasing significantly with every grade of severity. The lack of difference between normal function and Grade I is physiologically plausible: early (Grade I) diastolic impairment may not produce sufficient chronic elevation of left atrial pressure to cause structural enlargement, whereas the persistently elevated filling pressures of Grade II/III disease promote progressive atrial dilation (28). Our values are broadly consistent with those of Pritchett et al., who reported LAVI rising from 23 ± 6 mL/m² in normal function to 48 ± 12 mL/m² in Grade III disease, and El Aouar et al., who documented 21 ± 4 to 50 ± 5 mL/m² across categories (29).

LAVI correlated strongly with E/e′ and TR velocity and moderately with the E/A ratio, deceleration time, and IVRT, consistent with its role as an integrator of chronic filling-pressure elevation (30,31). The strong correlation between LAVI and LA diameter ($r = 0.832$) is expected, as both quantify atrial size. When participants were dichotomized, mean LAVI was higher in those with LVDD than in those with normal function, supporting the association between atrial remodeling and abnormal ventricular filling documented in hypertensive cohorts (27,32).

No significant correlations were observed between LAVI and BMI, hypertension duration, SBP, or DBP, suggesting that in treated hypertensive patients LAVI reflects structural and hemodynamic burden more closely than current blood pressure or demographic indices (20), others have reported differing results (21). Possibly reflecting differences in treatment status and population characteristics. Similarly, increased LAVI (>34 mL/m²) was not associated with diabetes, smoking, blood-pressure control, ischemic heart disease, or stroke in this cohort, and no association was found between LAVI and LVH ($p = 0.965$), in contrast to Cuspidi et al. (33). Differences in LVH definitions, antihypertensive treatment, and disease duration may explain the discrepancy.

The lack of a significant difference between normal function and Grade-I DD may indicate that early diastolic dysfunction does not necessarily produce sufficient chronic elevation in left atrial pressure to cause substantial structural enlargement. In contrast, persistent elevation of filling pressures in Grade-II and Grade-III dysfunction may promote progressive atrial dilation, making LAVI more useful for identifying advanced rather than early diastolic dysfunction (28).

ROC analysis demonstrated that LAVI had moderate discriminatory ability for detecting DD, with an AUC of 0.704 (95% CI: 0.637–0.771; $p<0.001$). The optimal cutoff was 34.0 mL/m², with 61.5% sensitivity and 66.7% specificity. Importantly, the empirically derived cutoff corresponded to the established ASE/EACVI threshold for increased LAVI (34). The moderate diagnostic performance is

consistent with the concept that LAVI should be interpreted as one component of a multiparametric assessment of diastolic function rather than as an isolated diagnostic marker.

The AUC of 0.704 observed for LAVI was lower than the 0.885 AUC reported for left atrial strain by Miljković et al. in discriminating elevated left atrial pressure among treated hypertensive patients with DD (35). This difference suggests that functional measures such as left atrial strain may identify early atrial abnormalities that are not yet reflected by increased atrial volume.

The inverse association between BMI and LVDD (OR 0.940) should be interpreted cautiously. In a cross-sectional design, this may reflect residual confounding, selection effects, or the underestimation of atrial enlargement when left atrial volume is indexed to body surface area in overweight individuals, a recognized limitation of BSA indexing. Similar inverse associations have been discussed as an "obesity paradox" in cardiovascular cohorts (36). But the present data cannot establish causality, and the finding requires confirmation in prospective studies.

Conclusion

In this cross-sectional study of hypertensive patients, LAVI was associated with more advanced grades of diastolic dysfunction (Grade II/III) and correlated with Doppler markers of elevated left ventricular filling pressure, principally E/e′. LAVI showed moderate discriminatory ability for LVDD at the guideline-based cutoff of 34 mL/m² and should be interpreted as one component of a multiparametric assessment. The cross-sectional design precludes causal inference, and the observed inverse BMI–LVDD association requires prospective confirmation.

Limitations

This study is subject to several limitations. Its cross-sectional design precludes establishing causal or temporal relationships among LAVI, E/e′, and diastolic dysfunction. The single-center setting may further limit the generalizability of the findings. Additionally, indexing LAVI to body surface area may underestimate left atrial enlargement in overweight patients. Finally, some of the observed associations involving LAVI may partly reflect mathematical or definitional overlap rather than independent physiological relationships.

Recommendations

LAVI should be routinely assessed in hypertensive patients as an important marker of left atrial remodeling and diastolic dysfunction. The ASE/EACVI cutoff of 34 mL/m² may be applied in clinical practice. However, LAVI should not be used alone because of its moderate sensitivity and specificity (61.5% and 66.7%); it should be interpreted alongside e′, E/e′, and TR velocity using a multiparametric approach. Patients with Grade II/III DD or significant LA enlargement should receive closer follow-up due to their potential risk of HFpEF. The unexpected inverse BMI–DD relationship should be interpreted cautiously.

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Author Contributions

All authors contributed equally to the study's conceptualization, methodology, data curation, validation, and manuscript preparation, and approved the final version.

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None.

Data Availability Statement

Data are available from the corresponding author upon reasonable request.

Declarations

Conflict of Interest

None

Ethical Approval and Informed Consent

Ethical approval was obtained from the Institutional Review Board of DHQ Teaching Hospital, Swabi (Ref. No. 1258/E-7). All participants provided individual informed consent prior to enrollment, and the study adhered to relevant ethical procedures and institutional requirements.

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