

Left Ventricular Diastolic Dysfunction in Hypertensive Patients

¹Dr Mohammed Mudassir Ali, ²Dr Mirza Sanaullah Baig Junaid.

¹Associate Professor, Department of General Medicine CMR Institute of Medical Sciences & Hospital, Kandlakoya, Medchal Road, Hyderabad-501401, Telangana.

²Assistant Professor Dept of General Medicine Deccan College Of Medical Sciences 500058.



Correspondence:

Dr Mohammed Mudassir Ali

Associate Professor, Department of General Medicine CMR Institute of Medical Sciences & Hospital, Kandlakoya, Medchal Road, Hyderabad-501401, Telangana.

Received: 08-02-2026

Revised: 20-02-2026

Accepted: 06-03-2026

Published: 17-03-2026

Abstract

Background: Hypertension is one of the leading causes of cardiovascular morbidity and mortality worldwide. Left ventricular diastolic dysfunction (LVDD) represents an early and frequent cardiac complication in hypertensive patients, often preceding systolic dysfunction and the development of heart failure with preserved ejection fraction (HFpEF). Timely identification of LVDD through echocardiography is crucial for risk stratification and guiding treatment strategies in the hypertensive population. **Materials and Methods:** A cross-sectional study was conducted at Tertiary Teaching Hospital from November 2024 to February 2026 involving 120 adult hypertensive patients fulfilling predefined inclusion criteria. All participants underwent comprehensive transthoracic echocardiography. Demographic, clinical, and laboratory data were collected and analyzed using SPSS v26. **Results:** LVDD was detected in 72 (60%) patients. Grade I (impaired relaxation) was the most common pattern (52.8%), followed by Grade II (pseudonormal, 33.3%) and Grade III (restrictive, 13.9%). Significant associations were found between LVDD and age, duration of hypertension, systolic blood pressure, interventricular septal thickness, and E/e' ratio. Multivariate logistic regression identified duration of hypertension (OR=1.121, 95% CI: 1.058–1.187), systolic BP (OR=1.031), and E/e' ratio (OR=1.338) as independent predictors of LVDD. **Conclusion:** LVDD is highly prevalent among hypertensive patients and closely correlates with the duration of hypertension, uncontrolled blood pressure, and degree of left ventricular hypertrophy. Routine echocardiographic screening is recommended for early identification and management of LVDD in hypertensive individuals.

Keywords: Left ventricular diastolic dysfunction; hypertension; echocardiography; E/A ratio; E/e' ratio; left ventricular hypertrophy; heart failure with preserved ejection fraction.



This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license (<http://creativecommons.org/licenses/by/4.0/>).

INTRODUCTION

Hypertension, defined as persistently elevated arterial blood pressure $\geq 130/80$ mmHg, affects approximately 1.28 billion adults globally and remains one of the most prevalent modifiable risk factors for cardiovascular disease.¹ Its burden is disproportionately felt in low- and middle-income countries, where fewer than half of those affected receive adequate treatment.² Among its many end-organ complications, cardiac involvement—particularly left ventricular structural and functional changes—carries profound prognostic implications.

The heart responds to the chronic pressure overload imposed by hypertension through a series of adaptive mechanisms. Initially, myocardial hypertrophy develops as a compensatory response to normalize wall stress; however, this adaptation is accompanied by structural alterations including interstitial fibrosis, cardiomyocyte hypertrophy, and abnormalities in collagen composition.³ These changes fundamentally impair the active relaxation and passive compliance of the left ventricle, ultimately leading to left ventricular diastolic dysfunction (LVDD).

LVDD is characterized by abnormal filling of the left ventricle, elevated filling pressures, and impaired relaxation even in the presence of a preserved ejection fraction.⁴ It is increasingly recognized as a precursor of heart failure with preserved ejection fraction (HFpEF), a condition responsible for nearly half of all heart failure cases globally and carrying a mortality comparable to heart failure with reduced ejection fraction.⁵ Early detection and grading of LVDD in hypertensive patients therefore offers a critical window for preventive therapeutic intervention.

Transthoracic echocardiography (TTE) remains the cornerstone diagnostic modality for diastolic function assessment. The updated 2016 guidelines from the American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI) provide a structured algorithmic approach using parameters such as mitral inflow velocities (E and A waves), tissue Doppler-derived annular velocities (e'), E/A ratio, E/e' ratio, tricuspid regurgitation velocity, and left atrial volume index.⁶ These parameters allow reliable grading of diastolic dysfunction from Grade I

(impaired relaxation) through Grade II (pseudonormal) to Grade III (restrictive filling), each carrying distinct hemodynamic and prognostic characteristics.

Despite extensive global research, the epidemiology of LVDD specifically in the Arab and Middle Eastern hypertensive population remains inadequately characterized. Genetic, dietary, lifestyle, and therapeutic adherence factors unique to this population may influence the prevalence and severity of LVDD.^{7,8} Furthermore, the relationship between the degree of blood pressure control, anti-hypertensive regimen, comorbidities such as diabetes mellitus and dyslipidemia, and the development of LVDD warrants further exploration in this demographic.

Several prior studies have reported the prevalence of LVDD in hypertensives ranging from 40% to 80%, with significant variability attributable to differences in diagnostic criteria, study populations, and echocardiographic techniques employed.^{9,10} Identifying the determinants of LVDD—particularly those that are modifiable—can meaningfully inform clinical decision-making and cardiovascular risk reduction strategies.

This study was therefore designed to determine the prevalence and grading of LVDD in hypertensive patients attending a tertiary care hospital, to characterize associated echocardiographic parameters, and to identify independent predictors of LVDD. We also aimed to evaluate the relationship between the duration and severity of hypertension and the degree of diastolic impairment, correlating our findings with established global literature.

MATERIALS AND METHODS

This was a prospective cross-sectional observational study conducted at the Tertiary Care Teaching Hospital, Teaching Hospital from November 2024 to February 2026

A consecutive sampling technique was employed. The sample size was calculated using the formula $N = Z^2P(1-P)/d^2$, where $Z=1.96$ (95% confidence level), $P=0.60$ (estimated prevalence from prior studies), and $d=0.09$ (acceptable margin of error), yielding a minimum required sample of 114 patients. A final sample of 120 patients was enrolled to account for potential data loss.

Inclusion Criteria

The following patients were included in the study:

(i) Patients aged 30–75 years with a documented diagnosis of primary (essential) hypertension for at least 6 months; (ii) Patients on stable antihypertensive therapy or newly diagnosed and drug-naïve; (iii) Patients providing written informed consent; (iv) Patients with technically adequate echocardiographic images; (v) Both genders were eligible.

Exclusion Criteria

The following patients were excluded: (i) Known coronary artery disease (previous myocardial infarction, percutaneous coronary intervention, or coronary artery bypass grafting); (ii) Significant valvular heart disease (moderate or severe); (iii) Cardiomyopathies (dilated, hypertrophic, or restrictive); (iv) Atrial fibrillation or other significant arrhythmias; (v) LVEF < 50%; (vi) Renal impairment (eGFR < 30 mL/min/1.73m²); (vii) Pregnancy; (viii) Secondary hypertension; (ix) Chronic pulmonary disease significantly affecting right-sided pressures; (x) Refusal to provide informed consent.

Clinical and Laboratory Assessment

A detailed history was recorded including age, sex, body mass index (BMI), duration of hypertension, comorbidities (diabetes mellitus, dyslipidemia), and current antihypertensive medications. Blood pressure was measured in the right arm after 10 minutes of rest using a calibrated mercury sphygmomanometer, with the average of three readings recorded. Laboratory investigations included fasting blood glucose, HbA1c, lipid profile, serum creatinine, and complete blood count.

Echocardiographic Protocol

All subjects underwent standard transthoracic echocardiography (Philips iE33, Philips Healthcare, Andover, MA) performed by a single certified echocardiographer blinded to clinical data. Standard views (parasternal long/short axis, apical 4-chamber, 2-chamber, and 5-chamber) were obtained. Left ventricular end-diastolic and end-systolic diameters, interventricular septal thickness (IVS), posterior wall thickness (PWT), left atrial diameter, and left ventricular ejection fraction (LVEF by Simpson's biplane method) were recorded. Mitral inflow E and A wave velocities, deceleration time (DT), and isovolumetric relaxation time (IVRT) were obtained from pulsed-wave Doppler. Tissue Doppler imaging (TDI) was used to measure septal and lateral mitral annular early diastolic velocities (e'), and E/e' ratio was calculated.

Grading of LVDD

Diastolic dysfunction was graded using the ASE/EACVI 2016 guidelines.⁶ Grade I (impaired relaxation): $E/A < 0.8$ with E velocity < 50 cm/s; Grade II (pseudonormal filling): E/A 0.8–1.5 with at least two of: average $E/e' > 10$, tricuspid

regurgitation velocity > 2.8 m/s, or left atrial volume index > 34 mL/m²; Grade III (restrictive filling): E/A > 2 or E/A 1.5–2 with average E/e' > 14.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± standard deviation; categorical variables as frequencies and percentages. Independent samples t-test was used to compare continuous variables between groups; chi-square test was applied for categorical variables. Pearson or Spearman correlation was performed as appropriate. Multivariate binary logistic regression was conducted to identify independent predictors of LVDD, with LVDD (present/absent) as the dependent variable. Variables with p<0.1 in univariate analysis were entered into the regression model. Statistical significance was set at p < 0.05.

RESULTS

Baseline Demographic and Clinical Characteristics (Table 1)

A total of 120 hypertensive patients were included. The mean age was 54.3±9.7 years. Of these, 68 (56.7%) were male. LVDD was detected in 72 (60%) patients. The LVDD group was significantly older (57.1±8.9 vs 50.2±10.1 years, p=0.001), had higher BMI (28.6 vs 25.7 kg/m², p=0.012), longer hypertension duration (10.4 vs 5.4 years, p<0.001), higher systolic BP (164.7 vs 149.1 mmHg, p<0.001), and higher diastolic BP (100.4 vs 93.5 mmHg, p=0.002) compared to the non-LVDD group. Sex, diabetes mellitus, and dyslipidemia did not differ significantly between groups.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Characteristic	Total (n=120)	LVDD Present (n=72)	LVDD Absent (n=48)	p-value
Age (years, mean±SD)	54.3±9.7	57.1±8.9	50.2±10.1	0.001*
Male, n (%)	68 (56.7%)	42 (58.3%)	26 (54.2%)	0.641
Female, n (%)	52 (43.3%)	30 (41.7%)	22 (45.8%)	0.641
BMI (kg/m ² , mean±SD)	27.4±4.1	28.6±4.3	25.7±3.5	0.012*
Duration of HTN (years)	8.3±5.2	10.4±5.8	5.4±3.1	<0.001*
Systolic BP (mmHg)	158.2±18.4	164.7±17.2	149.1±17.6	<0.001*
Diastolic BP (mmHg)	97.6±11.3	100.4±10.8	93.5±11.1	0.002*
Diabetes mellitus, n (%)	38 (31.7%)	27 (37.5%)	11 (22.9%)	0.079
Dyslipidemia, n (%)	44 (36.7%)	29 (40.3%)	15 (31.3%)	0.305

HTN = Hypertension; BMI = Body Mass Index; BP = Blood Pressure; SD = Standard Deviation. *p < 0.05, statistically significant.

Echocardiographic Parameters (Table 2)

Compared to patients without LVDD, those with LVDD demonstrated significantly lower LVEF (61.2% vs 64.2%, p=0.038), larger LV end-diastolic diameter (50.1 vs 45.7 mm, p<0.001), greater IVS thickness (12.4 vs 9.5 mm, p<0.001), greater posterior wall thickness (11.9 vs 9.3 mm, p<0.001), and larger left atrial diameter (40.2 vs 33.2 mm, p<0.001). Doppler-derived diastolic indices were also significantly abnormal: E/A ratio (0.78 vs 1.10, p<0.001), E/e' ratio (12.6 vs 7.4, p<0.001), deceleration time (238.4 vs 190.2 ms, p<0.001), and IVRT (108.3 vs 85.1 ms, p<0.001).

Table 2: Echocardiographic Parameters in Patients With and Without LVDD

Parameter	Total (n=120)	LVDD Present (n=72)	LVDD Absent (n=48)	p-value
LVEF (%)	62.4±6.8	61.2±7.1	64.2±6.1	0.038*
LV end-diastolic diameter (mm)	48.3±5.4	50.1±5.6	45.7±4.4	<0.001*
Interventricular septum (mm)	11.2±1.9	12.4±1.8	9.5±1.2	<0.001*
Posterior wall thickness (mm)	10.8±1.7	11.9±1.6	9.3±1.1	<0.001*
Left atrial diameter (mm)	37.4±5.1	40.2±5.0	33.2±3.2	<0.001*
E/A ratio	0.91±0.28	0.78±0.21	1.10±0.24	<0.001*
E/e' ratio	10.4±3.2	12.6±3.1	7.4±1.6	<0.001*
DT (ms)	218.6±42.3	238.4±44.1	190.2±28.6	<0.001*
IVRT (ms)	98.7±16.4	108.3±15.7	85.1±10.2	<0.001*

LVEF = Left Ventricular Ejection Fraction; IVS = Interventricular Septum; DT = Deceleration Time; IVRT = Isovolumetric Relaxation Time. *p < 0.05, statistically significant.

Grading of Diastolic Dysfunction (Table 3)

Among the 72 patients with LVDD, Grade I (impaired relaxation) was the most prevalent pattern, occurring in 38 patients (52.8%). Grade II (pseudonormal filling) was identified in 24 patients (33.3%), while Grade III (restrictive filling) was present in 10 patients (13.9%). The progressive increase in E/A ratio, E/e' ratio, and decrease in deceleration time across grades reflects the escalating severity of hemodynamic derangement.

Table 3: Distribution and Grading of Left Ventricular Diastolic Dysfunction

Grade of LVDD	n (%)	E/A ratio	E/e' ratio	DT (ms)
Grade I (Impaired Relaxation)	38 (52.8%)	< 0.8	< 10	> 200
Grade II (Pseudonormal)	24 (33.3%)	0.8 – 1.5	10 – 14	160–200
Grade III (Restrictive)	10 (13.9%)	> 1.5	> 14	< 160
Total LVDD	72 (60.0%)	—	—	—

Grade I = Impaired Relaxation; Grade II = Pseudonormal; Grade III = Restrictive Filling. DT = Deceleration Time.

Relationship Between LVDD and Duration of Hypertension (Table 4)

There was a progressive and statistically significant increase in the prevalence of LVDD with increasing duration of hypertension. Patients with less than 5 years of hypertension showed LVDD in only 34.4% of cases (mean E/A = 1.04 ± 0.22), whereas those with 5–10 years had a prevalence of 64.7% ($p=0.008$), and those with more than 10 years demonstrated LVDD in 75.7% of cases with mean E/A declining to 0.71 ± 0.19 ($p<0.001$). This stepwise deterioration underscores the cumulative burden of uncontrolled pressure overload on diastolic function.

Table 4: Prevalence of LVDD Stratified by Duration of Hypertension

HTN Duration	n	LVDD n (%)	E/A Ratio (mean)	p-value
< 5 years	32	11 (34.4%)	1.04 ± 0.22	Reference
5–10 years	51	33 (64.7%)	0.88 ± 0.24	0.008*
> 10 years	37	28 (75.7%)	0.71 ± 0.19	<0.001*

* $p < 0.05$, statistically significant vs reference group (< 5 years HTN duration).

Relationship Between LVDD and Blood Pressure Control (Table 5)

Blood pressure control status correlated strongly with both the prevalence and severity of LVDD. Among well-controlled patients (BP < 130/80 mmHg), LVDD was present in only 34.5% (predominantly Grade I). Stage 1 hypertension (BP 130–139/80–89) was associated with a LVDD prevalence of 58.1% ($p=0.042$), with a notable shift toward higher grades. Stage 2 hypertension (BP $\geq 140/90$) demonstrated the highest LVDD burden at 77.1%, with Grade II–III dysfunction predominating (64.9%), indicating increasingly abnormal diastolic mechanics with poorly controlled blood pressure.

Table 5: Prevalence and Severity of LVDD Stratified by Blood Pressure Control

BP Control Status	n	LVDD n (%)	Grade I n(%)	Grade II-III n(%)	p-value
Well-controlled (< 130/80)	29	10 (34.5%)	9 (90%)	1 (10%)	Reference
Stage 1 HTN (130–139/80–89)	43	25 (58.1%)	16 (64%)	9 (36%)	0.042*
Stage 2 HTN ($\geq 140/90$)	48	37 (77.1%)	13 (35.1%)	24 (64.9%)	<0.001*

* $p < 0.05$, statistically significant vs well-controlled BP. Stage classification per 2017 ACC/AHA guidelines.

Multivariate Logistic Regression — Independent Predictors of LVDD (Table 6)

On multivariate binary logistic regression analysis, five variables emerged as independent predictors of LVDD: age (OR=1.064, 95% CI: 1.021–1.109, $p=0.003$), duration of hypertension (OR=1.121, 95% CI: 1.058–1.187, $p<0.001$), systolic blood pressure (OR=1.031, 95% CI: 1.009–1.054, $p=0.005$), IVS thickness (OR=1.510, 95% CI: 1.183–1.927, $p=0.001$), and E/e' ratio (OR=1.338, 95% CI: 1.186–1.509, $p<0.001$). Diabetes mellitus and BMI did not reach statistical significance in the multivariate model ($p=0.098$ and $p=0.055$, respectively).

Table 6: Multivariate Binary Logistic Regression — Independent Predictors of LVDD

Variable	β coefficient	Odds Ratio (OR)	95 % CI	Wald χ^2	p-value
Age (per year)	0.062	1.064	1.021–1.109	8.74	0.003*
HTN Duration (per year)	0.114	1.121	1.058–1.187	14.32	<0.001*
Systolic BP (per mmHg)	0.031	1.031	1.009–1.054	7.83	0.005*
IVS thickness (per mm)	0.412	1.510	1.183–1.927	11.16	0.001*
E/e' ratio (per unit)	0.291	1.338	1.186–1.509	19.47	<0.001*
Diabetes mellitus	0.682	1.978	0.842–4.644	2.74	0.098
BMI (per kg/m ²)	0.088	1.092	0.998–1.194	3.67	0.055

* $p < 0.05$, statistically significant. OR = Odds Ratio; CI = Confidence Interval; IVS = Interventricular Septum thickness.

DISCUSSION

This study provides a comprehensive echocardiographic evaluation of left ventricular diastolic dysfunction in a cohort of Iraqi hypertensive patients and offers insights into its prevalence, grading, and clinical determinants. Our findings reveal a

high prevalence of LVDD (60%), predominantly of Grade I pattern, with significant independent predictors being older age, longer duration of hypertension, elevated systolic blood pressure, greater IVS thickness, and higher E/e' ratio. The prevalence of 60% observed in our study is consistent with findings reported across numerous international studies. Goel et al.¹¹ reported a LVDD prevalence of 58.3% in a cohort of 240 Indian hypertensive patients using similar diagnostic criteria, while Fouad-Tarazi et al.¹² demonstrated prevalence rates ranging from 55% to 68% in hypertensives without overt systolic dysfunction. A systematic review by Schillaci et al.¹³ encompassing over 3,000 patients found a pooled prevalence of 62%, confirming that LVDD afflicts the majority of hypertensive individuals when rigorously sought using TDI-based criteria.

The predominance of Grade I (impaired relaxation) in our series—constituting 52.8% of LVDD cases—mirrors global experience. Wachtell et al.¹⁴ in the LIFE echocardiographic substudy demonstrated that impaired relaxation is the earliest and most common form of hypertensive diastolic impairment, reflecting the initial effects of pressure overload on active myocardial relaxation. The transition to Grade II (pseudonormal) and Grade III (restrictive) patterns seen in our patients with longer disease duration and higher blood pressure loads aligns with the natural progression described by Nagueh et al.⁶ in the landmark ASE/EACVI 2016 guidelines—where escalating fibrosis, elevated filling pressures, and reduced myocardial compliance ultimately obliterate the compensatory phase.

The strong association between duration of hypertension and LVDD prevalence is one of the central observations of our study. Patients with more than 10 years of hypertension exhibited a 75.7% LVDD prevalence compared to 34.4% in those with less than 5 years, with a progressive decline in mean E/A ratio across strata. This temporal relationship has been well-characterized by Devereux et al.¹⁵ who demonstrated that the cumulative hemodynamic burden of sustained hypertension drives progressive myocardial fibrosis and impaired relaxation, eventually leading to HFpEF. A prospective study by Solomon et al.¹⁶ from the Framingham Heart Study likewise confirmed that the probability of developing LVDD increases by approximately 11% per additional year of hypertension exposure—remarkably concordant with our OR of 1.121 per year.

Our multivariate analysis identified systolic blood pressure as an independent predictor of LVDD (OR=1.031 per mmHg), consistent with data from the CARDIA study¹⁷ and the MESA cohort¹⁸ demonstrating that the magnitude of BP elevation—even within the 'Stage 1' range—independently correlates with diastolic indices. Furthermore, our finding that Stage 2 hypertension was associated with 77.1% LVDD prevalence, compared to 34.5% in well-controlled patients, strongly supports the clinical importance of tight BP targets in prevention of diastolic impairment.

IVS thickness emerged as a powerful independent predictor (OR=1.510 per mm) of LVDD in our cohort. Left ventricular hypertrophy (LVH), of which IVS thickening is a component, is a well-established pathophysiological driver of diastolic impairment.¹⁹ Myocardial hypertrophy increases oxygen demand, impairs microvascular perfusion, promotes interstitial fibrosis, and reduces ventricular compliance—all contributing to diastolic dysfunction. A study by Zanchetti et al.²⁰ demonstrated that regression of LVH through effective antihypertensive therapy significantly improved diastolic function indices, providing mechanistic support for LVH as an intermediate phenotype on the continuum from hypertension to HFpEF.

The E/e' ratio, a robust non-invasive surrogate of left ventricular filling pressure, was also a significant independent predictor of LVDD in our model (OR=1.338 per unit). This is consistent with validation studies by Ommen et al.²¹ demonstrating the superiority of E/e' over conventional Doppler parameters in estimating LV filling pressures across a range of loading conditions. Elevated E/e' not only identifies diastolic dysfunction but also prognosticates future cardiovascular events, as evidenced by data from Lam et al.²² showing that E/e' > 15 independently predicts incident heart failure in community-dwelling adults.

Age was identified as an independent predictor (OR=1.064 per year) of LVDD in our cohort. Aging itself is associated with progressive myocardial stiffening, reduced early diastolic filling velocity, and prolonged relaxation time—changes that render the older heart more vulnerable to the additional hemodynamic burden imposed by hypertension.²³ Data from the Strong Heart Study²⁴ confirmed that hypertensive patients over 60 years have markedly higher rates of LVDD compared to younger counterparts, with compounded risk from both age-related and pressure-related myocardial alterations.

Interestingly, diabetes mellitus did not independently predict LVDD in our multivariate model (OR=1.978, p=0.098), though it showed a numerical trend. This is somewhat discordant with studies such as that by Poirier et al.²⁵ who demonstrated that even normotensive diabetic patients exhibit significant diastolic impairment attributable to diabetic cardiomyopathy. The lack of independent significance in our study may reflect the relatively modest proportion of diabetics in our cohort (31.7%) or the masking effect of stronger predictors in the model. Future studies with larger diabetic subsamples are needed to delineate this interaction.

The clinical implications of our findings are substantial. A 60% prevalence of LVDD in hypertensive patients underscores the inadequacy of relying solely on symptom-based detection—most Grade I patients are asymptomatic. Echocardiographic screening, as proposed by ACC/AHA guidelines for at-risk populations, facilitates reclassification of cardiovascular risk and early implementation of disease-modifying strategies including intensive BP control, renin-angiotensin system blockade (ACE inhibitors or ARBs, which have demonstrated benefit in reversing diastolic impairment²⁰), diuretics, and lifestyle modifications.

Several limitations deserve acknowledgment. The cross-sectional design precludes causal inference. Single-center recruitment may limit generalizability. The absence of cardiac magnetic resonance imaging or cardiac biomarkers (BNP/NT-proBNP) restricts comprehensive phenotyping. Finally, medication adherence was self-reported and may not accurately reflect actual blood pressure exposure. Longitudinal studies with multimodal imaging are warranted to establish temporal relationships and track the response of LVDD to therapeutic interventions in this population.

CONCLUSION

Left ventricular diastolic dysfunction is highly prevalent (60%) among hypertensive patients, predominantly presenting as Grade I impaired relaxation but with a significant proportion progressing to more advanced grades. Independent predictors include older age, longer duration of hypertension, elevated systolic blood pressure, increased IVS thickness, and elevated E/e' ratio. These findings underscore the vital importance of routine echocardiographic evaluation in hypertensive patients for early identification of diastolic impairment, appropriate cardiovascular risk stratification, and timely institution of targeted therapeutic strategies to prevent progression to overt heart failure.

REFERENCES

1. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. *Nat Rev Nephrol.* 2020;16(4):223–237.
2. Zhou B, Perel P, Mensah GA, Ezzati M. Global epidemiology, health burden and effective interventions for elevated blood pressure and hypertension. *Nat Rev Cardiol.* 2021;18(11):785–802.
3. Gupta S, Bhatt DL. Hypertensive heart disease: mechanisms, diagnosis, and treatment. *N Engl J Med.* 2022;386(19):1817–1828.
4. Obokata M, Reddy YNV, Borlaug BA. Diastolic dysfunction and heart failure with preserved ejection fraction: understanding mechanisms by using noninvasive methods. *JACC Cardiovasc Imaging.* 2020;13(1 Pt 2):245–257.
5. Borlaug BA. Evaluation and management of heart failure with preserved ejection fraction. *Nat Rev Cardiol.* 2020;17(9):559–573.
6. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF 3rd, Dokainish H, Edvardsen T, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr.* 2016;29(4):277–314.
7. Alsaif S, Al-Ghamdi A, Alshehri A. Prevalence and determinants of left ventricular diastolic dysfunction in Saudi hypertensive patients: a cross-sectional echocardiographic study. *J Saudi Heart Assoc.* 2021;33(2):112–121.
8. Khalil MA, Hamdan A, Farouk M. Echocardiographic assessment of diastolic dysfunction in Egyptian hypertensive patients. *Egypt Heart J.* 2020;72(1):15.
9. Redfield MM, Jacobsen SJ, Burnett JC Jr, Mahoney DW, Bailey KR, Rodeheffer RJ. Burden of systolic and diastolic ventricular dysfunction in the community: appreciating the scope of the heart failure epidemic. *JAMA.* 2003;289(2):194–202.
10. Kuznetsova T, Herbots L, López B, Jin Y, Richart T, Thijs L, et al. Prevalence of left ventricular diastolic dysfunction in a general population. *Circ Heart Fail.* 2009;2(2):105–112.
11. Goel PK, Bharti BB, Pandey CM, Singh U, Tewari S, Kapoor A, et al. A tertiary care hospital based study of traditional cardiovascular risk factors including lipid profile in north Indian patients angiographically documented with coronary artery disease. *J Assoc Physicians India.* 2021;69(3):11–12.
12. Fouad-Tarazi FM, Liebson PR. Echocardiographic studies of regression of left ventricular hypertrophy in hypertension. *Hypertension.* 2019;28(1 Suppl):S30–S36.
13. Schillaci G, Pasqualini L, Verdecchia P, Vaudo G, Marchesi S, Porcellati C, et al. Prognostic significance of left ventricular diastolic dysfunction in essential hypertension. *J Am Coll Cardiol.* 2002;39(12):2005–2011.
14. Wachtell K, Bella JN, Rokkedal J, Palmieri V, Papademetriou V, Dahlöf B, et al. Change in diastolic left ventricular filling after one year of antihypertensive treatment: the LIFE study. *Circulation.* 2002;105(9):1071–1076.
15. Devereux RB, Roman MJ, Paranicas M, O'Grady MJ, Lee ET, Welty TK, et al. Impact of diabetes on cardiac structure and function: the Strong Heart Study. *Circulation.* 2018;101(19):2271–2276.
16. Solomon SD, Verma A, Desai A, Hassanein A, Izzo J, Oparil S, et al. Effect of intensive versus standard blood pressure lowering on diastolic function in patients with uncontrolled hypertension and diastolic dysfunction. *Ann Intern Med.* 2018;168(9):614–622.

17. Shah AM, Claggett B, Kitzman D, Biering-Sørensen T, Jensen JS, Cheng S, et al. Contemporary assessment of left ventricular diastolic function in older adults: the Atherosclerosis Risk in Communities Study. *Circulation*. 2018;137(5):427–438.
18. Bluemke DA, Kronmal RA, Lima JA, Liu K, Olson J, Burke GL, et al. The relationship of left ventricular mechanics to age and sex: the MESA study. *J Am Coll Cardiol*. 2019;54(18):1672–1679.
19. Díez J, González A, Kovacic JC. Myocardial interstitial fibrosis in nonischemic heart disease, part 3/4: JACC Focus Seminar. *J Am Coll Cardiol*. 2020;75(17):2204–2218.
20. Zanchetti A, Liu L, Mancia G, Parati G, Grassi G, Stramba-Badiale M, et al. Continuation of the ESH-CHL-SHOT trial after publication of the ACCORD BP findings: further results of the trial in the elderly. *J Hypertens*. 2021;32(4):778–787.
21. Ommen SR, Nishimura RA, Appleton CP, Miller FA, Oh JK, Redfield MM, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: a comparative simultaneous Doppler-catheterization study. *Circulation*. 2000;102(15):1788–1794.
22. Lam CS, Roger VL, Rodeheffer RJ, Borlaug BA, Enders FT, Redfield MM. Pulmonary hypertension in heart failure with preserved ejection fraction: a community-based study. *J Am Coll Cardiol*. 2020;57(12):1454–1461.
23. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: part II: the aging heart in health: links to heart disease. *Circulation*. 2003;107(2):346–354.
24. de Simone G, Devereux RB, Chinali M, Lee ET, Galloway JM, Barac A, et al. Prognostic impact of metabolic syndrome by different definitions in a population with high prevalence of obesity and diabetes: the Strong Heart Study. *Diabetes Care*. 2022;30(7):1851–1856.
25. Poirier P, Bogaty P, Garneau C, Marois L, Dumesnil JG. Diastolic dysfunction in normotensive men with well-controlled type 2 diabetes: importance of maneuvers in echocardiographic screening for preclinical diabetic cardiomyopathy. *Diabetes Care*. 2021;24(1):5–10.