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Original Article

Age-Related Alterations in Left Ventricular Diastolic Function Assessed by Doppler and Tissue Doppler Echocardiography: A Cross-Sectional Study

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ABSTRACT

Background: Left ventricular diastolic dysfunction (LVDD) is an early manifestation of myocardial aging and a precursor of heart failure with preserved ejection fraction (HFpEF).

Aim: To evaluate age-related changes in LVDD using echocardiography.

Methods: This cross-sectional study included 204 participants aged 30–70 years. Transthoracic echocardiography was performed according to ASE/EACVI guidelines using Doppler and tissue Doppler imaging. Statistical analysis included Chi-square test, multivariate logistic regression, and ROC curve analysis.

Results: LVDD prevalence increased from 30.4% (30–39 years) to 84.6% (60–70 years). Age was significantly associated with LVDD ($p = 0.001$) and was an independent predictor (AOR = 2.18, 95% CI: 1.42–3.35, $p = 0.001$). Hypertension (AOR = 1.89) and BMI (AOR = 1.36) were also significant predictors. ROC analysis showed good predictive ability of age (AUC = 0.82) with an optimal cut-off of 52 years.

Conclusion: Age is a strong independent predictor of LVDD, and echocardiography enables early detection of subclinical dysfunction.

Keywords: Left ventricular diastolic dysfunction; Echocardiography; Aging; Tissue Doppler imaging; HFpEF; Cardiovascular risk.

Introduction:

Left ventricular diastolic function represents a critical determinant of overall cardiac performance, reflecting the ability of the myocardium to actively relax and accommodate venous return during diastole. In normal physiology, early diastolic filling is driven by active myocardial relaxation and elastic recoil, followed by atrial contraction contributing to late ventricular filling. With advancing age, these mechanisms progressively deteriorate, leading to impaired ventricular compliance and altered filling dynamics [3,5]. Left ventricular diastolic dysfunction (LVDD) is increasingly recognized as an early manifestation of myocardial aging and a key pathophysiological precursor to heart failure with preserved ejection fraction (HFpEF), which accounts for nearly half of all heart failure cases globally [2,4]. LVDD is characterized by impaired relaxation, increased myocardial stiffness, and elevated left ventricular filling pressures, often occurring in the absence of overt systolic dysfunction [2].

Age-related cardiac remodeling involves complex structural, cellular, and molecular alterations, including myocardial interstitial fibrosis, increased collagen deposition, reduced myocardial compliance, endothelial dysfunction, impaired calcium reuptake within cardiomyocytes, and ventricular–arterial stiffening [4,5]. These changes collectively contribute to progressive deterioration of diastolic performance, even in apparently healthy individuals. Echocardiography remains the cornerstone non-invasive modality for evaluation of diastolic function [3]. Conventional transmitral Doppler parameters, such as E and A wave velocities and deceleration time, provide initial assessment of filling patterns; however, these indices are significantly influenced by loading conditions. Tissue Doppler imaging (TDI), particularly measurement of early diastolic mitral annular velocity (E’), offers a more load-independent and sensitive marker of myocardial relaxation abnormalities [3]. The integration of Doppler and TDI parameters, as recommended by the American Society of Echocardiography and European Association of Cardiovascular Imaging (ASE/EACVI), allows for accurate grading of diastolic dysfunction [3]. Despite growing evidence on age-related cardiovascular changes, there remains a paucity of region-specific echocardiographic data evaluating the progressive nature of LVDD in South Indian populations. Understanding age-specific diastolic alterations is essential for early identification of individuals at risk of HFpEF and for guiding preventive cardiovascular

strategies [1]. Therefore, the present study aims to evaluate age-related changes in left ventricular diastolic function using Doppler and tissue Doppler echocardiography and to identify the association between age and LVDD in an adult South Indian cohort.

Materials and Methods:

Study Design and Setting: This prospective cross-sectional analytical study was conducted in the Department of Cardiology at Aarupadai Veedu Medical College and Hospital over a period of six months. The study aimed to evaluate age-related alterations in left ventricular diastolic function using comprehensive echocardiographic assessment in an adult South Indian population.

Study Population: A total of 204 consecutive participants aged between 30 and 70 years were enrolled from both outpatient and inpatient cardiology services. All participants provided written informed consent prior to participation. The study protocol was approved by the Institutional Human Ethics Committee, and all procedures were conducted in accordance with ethical standards

Inclusion Criteria:

Participants were included in the study if they met the following criteria:

1. Age between 30 and 70 years
2. Willingness to participate in the study
3. Provision of written informed consent

Exclusion Criteria

Participants were excluded if they had any of the following conditions:

1. History of myocardial infarction
2. Significant valvular heart disease
3. Chronic kidney disease
4. Refusal or inability to participate

Detailed Echocardiographic Examination:

Transthoracic echocardiography was performed using a Mindray diagnostic ultrasound system with standardized imaging protocols. All measurements were obtained in accordance with the American Society of Echocardiography and European Association of Cardiovascular Imaging (ASE/EACVI) guidelines to ensure accuracy, reproducibility, and standardization of diastolic function assessment.

Doppler Echocardiographic Assessment

Left ventricular diastolic function was initially evaluated using pulsed-wave Doppler transmitral inflow from the apical four-chamber view. The following parameters were measured: Early diastolic peak velocity (E wave), Late diastolic peak velocity (A wave), E/A ratio, Deceleration time (DT). These parameters reflect left ventricular filling dynamics but are influenced by preload and loading conditions.

Tissue Doppler Imaging (TDI)

Tissue Doppler imaging was performed at the septal and lateral mitral annulus to obtain myocardial velocities: Early diastolic velocity (E'), Late diastolic velocity (A').

Reduced E' velocity was considered a sensitive marker of impaired myocardial relaxation and early diastolic dysfunction. The E/E' ratio was used as an indirect estimate of left ventricular filling pressures.

Grading of LVDD

Left ventricular diastolic dysfunction (LVDD) was defined and classified using an integrated ASE/EACVI algorithm incorporating: Mitral inflow Doppler patterns, Tissue Doppler velocities, E/E' ratio, Left atrial size. LVDD was categorized into: Grade I: impaired relaxation pattern, Grade II: pseudonormal filling pattern, Grade III: restrictive filling pattern. This multiparametric approach improves diagnostic accuracy compared to single-parameter assessment

Statistical Analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 21. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The association between age and LVDD was assessed using the Chi-square test.

Multivariate logistic regression analysis was performed to identify independent predictors of LVDD after adjusting for confounders such as body mass index, sex, and hypertension. Receiver operating characteristic (ROC) curve analysis was used to evaluate the predictive ability of age for LVDD and to determine an optimal cut-off value. A p-value < 0.05 was considered statistically significant.

Results

Baseline Characteristics of Study Population:

A total of 204 participants aged between 30 and 70 years were included in the analysis. The mean age of the study population was 53 ± 10 years, and the mean body mass index (BMI) was 27 ± 3 kg/m². Males constituted 55% of the study population, while females accounted for 45%. Hypertension was present in 42.2% of participants, and 16.7% demonstrated evidence of left ventricular systolic dysfunction. These baseline characteristics indicate a moderately high cardiovascular risk profile within the study cohort (Table-1).

Parameter	Value
Total participants	204
Age (years)	53 ± 10
BMI (kg/m²)	27 ± 3
Gender (Male)	55%
Gender (Female)	45%
Hypertension	42.2%
LV systolic dysfunction	16.7%

TABLE 1: Baseline Characteristics of Study Population

Age-wise Distribution of Left Ventricular Diastolic Dysfunction:

The prevalence of LVDD demonstrated a progressive increase with advancing age. In the 30–39 years age group, LVDD prevalence was 30.4%, which increased to 48% in the 40–49 years group, 71.4% in the 50–59 years group, and reached 84.6% in the 60–70 years group. This pattern demonstrates a strong age-dependent escalation in diastolic dysfunction burden. Chi-square analysis demonstrated a statistically significant association between age and LVDD (χ^2 = 35.868, df = 3, p = 0.001). The results indicate that advancing age is significantly associated with increased prevalence of diastolic dysfunction (Table-2).

Age Group (years)	LVDD Prevalence (%)
30–39	30.4%
40–49	48.0%
50–59	71.4%
60–70	84.6%

TABLE 2: Age-wise Distribution of LVDD

Echocardiographic Parameter Trends:

Progressive deterioration in diastolic parameters was observed with increasing age. There was a gradual reduction in E/A ratio and E' velocity, along with prolongation of deceleration time (DT), suggesting impaired myocardial relaxation and increased ventricular stiffness. The E/E' ratio showed a rising trend with age, indicating progressively increased left ventricular filling pressures. On multivariate logistic regression analysis, after adjusting for potential confounders including body mass index, sex, and hypertension, age remained a strong independent predictor of left ventricular diastolic dysfunction. Increasing age was significantly associated with higher odds of developing LVDD. Age: Adjusted Odds Ratio (AOR) = 2.18, 95% Confidence Interval (CI) = 1.42–3.35, p = 0.001, BMI: AOR = 1.36, 95% CI = 1.05–1.78, p = 0.019, Hypertension: AOR = 1.89, 95% CI = 1.21–2.94, p = 0.004, Sex (Male): AOR = 1.27, 95% CI = 0.84–1.93, p = 0.248. These findings indicate that age is the most significant independent determinant of LVDD, followed by hypertension and BMI, while sex does not demonstrate a statistically significant independent association after adjustment (Table-3).

Variable	AOR	95% CI	p-value
Age	2.18	1.42–3.35	0.001
Hypertension	1.89	1.21–2.94	0.004
BMI	1.36	1.05–1.78	0.019
Sex (Male)	1.27	0.84–1.93	0.248

TABLE 3: Multivariate Logistic Regression Analysis

ROC curve analysis was performed to evaluate the predictive ability of age for identifying left ventricular diastolic dysfunction. The area under the curve (AUC) for age was found to be 0.82, indicating good discriminatory ability.

Parameter	Value
AUC	0.82
Cut-off age	52 years
Sensitivity	78%
Specificity	74%
Positive Predictive Value	81%
Negative Predictive Value	70%

TABLE 4: ROC Curve Analysis for Age Predicting LVDD

The optimal cut-off value of age for predicting LVDD was identified as 52 years, with the following diagnostic performance: Sensitivity: 78%, Specificity: 74%, Positive predictive value (PPV): 81%, Negative predictive value (NPV): 70%. These results suggest that age has strong discriminatory performance in identifying individuals at risk of diastolic dysfunction, and an age threshold of approximately 50–52 years may be clinically useful for targeted echocardiographic screening (Table-4).

Discussion

This study demonstrates a clear and progressive increase in left ventricular diastolic dysfunction (LVDD) with advancing age, underscoring the strong association between myocardial aging and impaired diastolic performance. The findings indicate that age is an independent determinant of LVDD even after adjustment for key cardiovascular risk factors including body mass index, hypertension, and sex, suggesting that intrinsic myocardial aging plays a central role in the pathophysiology of diastolic impairment.

The observed age-related deterioration in diastolic function can be attributed to well-established structural and functional myocardial changes. With advancing age, there is progressive myocardial interstitial fibrosis, increased collagen deposition, reduced elastin content, and adverse extracellular matrix remodeling. These alterations result in increased ventricular stiffness and reduced compliance, thereby impairing early diastolic relaxation. In addition, age-associated abnormalities in intracellular calcium handling, particularly reduced SERCA-2a activity, contribute to delayed myocardial relaxation and prolongation of isovolumic relaxation time. Collectively, these mechanisms lead to elevated left ventricular filling pressures and progressive diastolic dysfunction [4,5]. The marked increase in LVDD prevalence beyond the fifth decade observed in this study is consistent with large epidemiological evidence demonstrating that diastolic dysfunction becomes increasingly common with age, even in the absence of overt cardiovascular disease. This supports the concept that LVDD represents an early subclinical stage within the continuum toward heart failure with preserved ejection fraction (HFpEF), which is now recognized as a major contributor to heart failure-related morbidity and hospitalization worldwide [2,4].

From a diagnostic standpoint, the combined use of Doppler and tissue Doppler imaging in accordance with ASE/EACVI guidelines enhances the robustness of LVDD assessment. Tissue Doppler-derived E' velocity provides a relatively load-independent index of myocardial relaxation and enables earlier detection of diastolic impairment compared with conventional transmitral inflow parameters. The application of a multiparametric, guideline-based approach reduces diagnostic variability and improves classification accuracy of diastolic dysfunction [3,6,7].

The multivariate analysis further confirms that age is the strongest independent predictor of LVDD, exceeding the influence of traditional risk factors such as hypertension and body mass index. This finding highlights that while comorbid conditions contribute to diastolic impairment, intrinsic myocardial aging remains the dominant determinant of diastolic decline. In addition, ROC curve analysis supports the clinical utility of age as a practical screening marker, demonstrating acceptable discriminatory performance for identifying individuals at risk of LVDD.

From a clinical perspective, these findings have important implications for preventive cardiology. Early identification of subclinical diastolic dysfunction in middle-aged individuals may facilitate timely intervention through optimization of blood pressure control, weight management, and lifestyle modification, thereby potentially delaying or preventing progression to HFpEF. This supports the rationale for incorporating echocardiographic screening in individuals beyond 50 years of age, particularly in high-risk populations. Overall, this study reinforces the concept that LVDD represents not only a pathological condition but also a continuum of physiological cardiovascular aging. Early detection using echocardiography plays a pivotal role in risk stratification and may contribute to reducing the future burden of heart failure in aging populations.

Conclusion

Left ventricular diastolic dysfunction is strongly and independently associated with advancing age. Multivariate analysis demonstrated that age is a significant predictor of LVDD, with additional contributions from hypertension and body mass index. ROC analysis identified age as a good discriminator for LVDD, with an optimal cut-off of approximately 52 years. These findings suggest that age-related myocardial remodeling plays a central role in diastolic impairment and support the utility of echocardiographic screening in middle-aged and elderly populations for early detection of subclinical dysfunction and prevention of progression to HFpEF.

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