Original Article

Prevalence of Doppler-Derived Left Ventricular Diastolic Dysfunction Among Newly Diagnosed Hypertensive Patients

IG Abubakar[1], F Buba[1,2], AI Oyati[3,4], MA Talle[1,2], CO Anjorin[1]

[1]Department of Medicine, Cardiology Unit, University of Maiduguri Teaching Hospital, Nigeria, [2]Department of Medicine, Faculty of Clinical Sciences, College of Medical Sciences, University of Maiduguri, Nigeria, [3]Department of Medicine, Faculty of Clinical Sciences, College of Medical Sciences, Ahmadu Bello University, Zaria, [4]Department of Medicine, Cardiology Unit, Ahmadu Bello University Teaching Hospital, Shika, Nigeria

Abstract

Background: The initial sign of hypertensive heart disease (HHD) is left ventricular diastolic dysfunction (LVDD), which is caused by remodeling of the left ventricle and left atrium, resulting in impaired relaxation of the left ventricle. LVDD is also partly due to left ventricular hypertrophy (LVH). If left untreated, LVDD can progress to diastolic heart failure and systolic heart failure. In Western countries, the prevalence of LVDD in long-term hypertensive patients ranges from 40.3% to 60%, but it is more common among hypertensive Nigerians. Since systemic hypertension can be asymptomatic in the early stages, it is important to evaluate LVDD early and control blood pressure to slow down its progression. **Aims and Objectives:** The study aims to highlight the prevalence of LVDD and to determine the stages of LVDD among newly diagnosed hypertensive patients at the University of Maiduguri Teaching Hospital (UMTH). **Method:** The study design is a hospital-based, cross-sectional, observational study. The study population consists of 352 consecutive treatment Naïve hypertensive adult patients aged 18 years and above who presented to the Cardiology Clinic of UMTH from June 2019 to June 2021. The study used the diagnostic criteria for LVDD and LVH which were based on the American Society of Echocardiography and the European Association of Cardiovascular Imaging. **Results:** A total of 352 newly diagnosed hypertensive patients were recruited, with a mean age of 50.9 ± 11.8 years, and 54.3% were female. The majority of patients (63.6%) were overweight or obese, with a mean body mass index (BMI) of 28.5 ± 4.6 kg/m^2. The mean systolic blood pressure (SBP) was 155.7 ± 16.9 mmHg, and the mean diastolic blood pressure (DBP) was 92.8 ± 10.8 mmHg. LVDD was found in 58.5% of the patients, with stage 1 LVDD being the most common (42.6%), followed by stage 2 LVDD (15.9%). The prevalence of LVDD was significantly higher in females compared to males. Patients with LVDD were significantly older and had higher BMI, higher systolic and DBP, higher pulse pressure, higher LAVI, and higher LVMI compared to those without LVDD ($P < 0.05$). **Conclusion:** LVDD is highly prevalent among newly diagnosed hypertensive patients, with stage 1 being the most common. Female gender, older age, higher BMI, higher blood pressure, higher LAVI, and higher LVMI were significant predictors of LVDD. Early detection and appropriate management of LVDD may help to prevent adverse cardiovascular outcomes in hypertensive patients.

Keywords: *2D echocardiography, left ventricular diastolic dysfunction, left ventricular hypertrophy, treatment Naïve hypertensive*

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Address for correspondence: Dr. IG Abubakar, Department of Medicine, Cardiology Unit, University of Maiduguri Teaching Hospital, Nigeria.
E-mail: dr.galtimari@gmail.com

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Background

Systemic hypertension is the leading cause of major cardiovascular morbidities such as hypertensive heart disease (HHD), renal disease, stroke, and peripheral arterial disease (PAD),[1-3] and about 46% of adults aged 25 years and older Africans are affected by hypertension.[4] The prevalence of systemic arterial hypertension among Nigerian adults over the age of 18 years is 27.8% and the number of people with hypertension is estimated to be 39.1 million by the year 2030.[5,6] Most Nigerians who are newly diagnosed hypertensive have already developed hypertension target organ damage TOD due to poor screening habits and lack of awareness.[7,8]

HHD is a constellation of morphological changes in the left ventricle, left atrium, and coronary arteries as a result of chronic systemic arterial hypertension; these changes include left ventricular hypertrophy (LVH), left ventricular diastolic dysfunction (LVDD), heart failure (HF), coronary artery disease (CAD), and atrial fibrillation (AF) with the key entity being left ventricular hypertrophy.

LVDD is an early manifestation of HHD. Untreated and uncontrolled systemic arterial hypertension may cause a progression of mild LVVD to severe LVDD, and may even lead to diastolic heart failure and systolic heart failure[9]. The prevalence of LVDD among long-term hypertensive patients in Western countries ranges between 40.3% and 60%;[10] it is more prevalent in hypertensive Nigerians[11,12] Systemic hypertension and its consequent complication like LVDD could be asymptomatic at an early stage, therefore, early evaluation for the LVDD and optimal blood pressure (BP) control is necessary to retard the progression.

LVDD is determined by the Doppler interrogation as recommended by the American Society of Echocardiography and the European Association of Cardiovascular Imaging. It is a non-invasive and reproducible method for the evaluation and monitoring of hypertensive heart disease (HHD).[9,13]

There is a paucity of data on Doppler-derived LVDD among never-treated hypertensive patients in northeastern Nigeria. Therefore, we intended to highlight the prevalence of LVDD and to determine the stages of LVDD among newly diagnosed hypertensive patients at the University of Maiduguri Teaching Hospital (UMTH).

Method

Study design

It is a hospital-based, cross-sectional, observational study. The study population was made up of 352 consecutive treatments Naïve hypertensive adult patients aged 18 years and above who presented to the Cardiology Clinic of UMTH from June 2019 to June 2021.

Subjects' inclusion criteria include adults of age 18 years and above, with average office blood pressure (OBP) ≥140/90 mmHg. The exclusion criteria include hypertensive patients on antihypertensive drugs, valvular heart diseases, estimated glomerular filtration rate (eGFR) of <60 ml\min\1.72 m^2, and diabetes mellitus as well as poor echocardiographic image qualities.

Biodata, anthropometric and clinical details

A well-structured questionnaire was used to collect data from eligible patients after obtaining written, informed consent and ethical approval from the Health Research and Ethics Committee of the UMTH. Information obtained using the questionnaire included sociodemographic parameters, and anthropometric parameters which include height and weight were measured using the Health Care Scale Adult with Height Measurement Standard (Model: RGZ-160), patients stood erect with minimal clothing and bare feet then the weight and the height was recorded by the investigator. BMI and body surface area (BSA) were calculated from the weight and height.[14,15]

Blood pressure measurement

Office BP measurements were performed using Accoson® sphygmomanometer and stethoscope. The BP was measured while the patients were in a sitting position, the arm at the level of the heart using an appropriate-size cuff; cuff length and width of 80% and 40% of arm circumference, respectively, deflating the cuff at ≤2 mm Hg/sec. The systolic and DBP were determined using the first and the fifth Korokoff sounds. Three different BPs were taken about five minutes apart and the average was used. Pulse pressure (PP) and mean arterial BP (MABP) were calculated from the SBP and the DBP.

Biochemistry

Capillary blood glucose was measured using Glucometer (one touch ultra mini ACCU-CHEK®Aviva).[16-18] Venous blood specimens were obtained via venipuncture for blood urea nitrogen and creatinine in a sterile plain bottle at the hospital's central chemical pathology laboratory where the blood chemistry auto-analyzer model cobas 311 analyzer (F. Hoffman-La Roche Ltd) was used.

Echocardiography

Echocardiographic examinations were performed using KT-LM 200HDPE (Siemens Acuson X300, Siemen medical solution, USA) ultrasound systems equipped with the appropriate two-dimensional transthoracic probe. Participants were evaluated in the left lateral decubitus position and images were acquired from standard parasternal, and apical windows using second-harmonic two-dimensional imaging.

Mitral inflow velocities were assessed from the apical 4-chamber (A4C) view with a pulsed wave. Doppler by placing a 1–2 mm sample volume between the tips of the mitral leaflets during diastole. From the mitral inflow profile, the E and A-wave velocity, E-deceleration time (DT), and E/A velocity ratio were measured. Doppler tissue imaging was used to measure È velocities by placing a 1–2 mm sample volume in the septal and lateral mitral annulus and the average È was considered. Tricuspid peak gradient (TRPG) was estimated from the peak tricuspid regurgitation velocity, which was observed using continuous wave (CW) Doppler interrogation from an A4C view. It was then converted to the peak pressure by the modified Bernoulli equation; (TRPG = 4V2).[13]

Left atrial volume (LAV) was determined using planimetry of the left atrium (LA) in apical 4-chamber view and apical 2-chamber (A2C) view to obtaining two LA areas; A4C area A1 and A2C area A2, respectively. The measurement of the LA diameter was done by measuring the distance from the annular plane to the superior border of the left atrial in the A4C view. The LAV was determined using the formula; LAV = [(0.85 × A1 × A2)/L] and the left atrial volume index (LAVI) was determined by dividing LAV by BSA.[19,20]

Using M mode, the left ventricular dimensions, interventricular septal thickness (IVST), and left ventricular posterior wall thickness (LVPWT) were measured in the parasternal long axis (PSLAX) window. We used the Devereux regression formula to determine left ventricular mass (LVM); LVM = 1.04 ([LVID + PWTd + IVSTd]^3 - [LVID]^3) - 13.6 g.[21] The left ventricular mass index (LVMI) was determined by dividing LVM by BSA.[20] The LVMI of >115 g/m^2 and >95 g/m^2 were considered as LVH for males and females, respectively.[20]

The patients were stratified as stage one LV diastolic dysfunction was determined using standard echocardiographic parameters that include E/A velocity ratio <0.8 with normal LV filling pressure (E/È ≤8). Stage 2 LVDD was defined based on the E/A ratio between 0.8 and 2.0 and E/E' ≥9 but <13 and the diagnostic criteria for LVDD stage 3 were E/E' ≥13 and E/A >2.[22,23] Those that did not meet the above criteria were excluded.

Data analysis

Statistical analysis was carried out using the Statistical Package for Social Science (SPSS) software version 26 (Chicago, Illinois, USA). The continuous variables following the normality test using the Kolmogorov–Smirnov test were expressed as mean ± SD. The student's *t*-test was used to compare the mean ± SD of the treatment Naïve hypertensive patients with normal LV diastolic function (NLVDF) and the treatment Naïve hypertensive patients with LVDD to determine statistical significance. The categorical variables were expressed as absolute values and percentages. Analysis of variance (ANOVA) one way was used to determine the difference among LV diastolic function phenotypes. A *P* value of ≤0.05 was considered significant.

Results

The total number of treatment Naïve hypertensive patients screened for the study was 352; 52 patients were excluded due to (1) 25 patients having CKD, (2) 16 poor echocardiographic images, and (3) 11 patients having indeterminate LV diastolic function. 300 treatment Naïve hypertensive patients were analyzed of which 114 (38%) were those treatment Naïve hypertensive (TNH) patients with NLVDF while 186 (62%) were those TNH patients with LVDD. The males were 72 (63.2%) in the NLVDF and 108 (58.2%) in the LVDD whereas the female constituted 42 (36.8%) and 78 (41.9%) among the TNH with NLVDF and the TNH with LVDD groups, respectively. The patients

Table 1: The demographic and clinical characteristics of the study population

Variables	NLVDF (*n*=114)	LVDD (*n*=186)	*P*
Male	72 (63.2%)	108 (58.1%)	
Female	42 (36.8%)	78 (41.9%)	
Age (years)	40.9±11.9	46.1±10.5	0.031
Height (m)	1.68±0.07	1.69±0.08	0.377
Weight (kg)	72.2±14.9	72.9±16.3	0.828
BMI (kg/m^2)	25.6±5.0	25.5±5.9	0.925
BSA (m^2)	1.86±0.32	1.82±0.20	0.537
PR (beats/min)	79.4±7.6	85.7±15.5	0.008
SBP (mm Hg)	148.6±11.7	151.0±8.2	0.286
DBP (mm Hg)	93.9±3.8	93.4±5.2	0.532
PP (mm Hg)	54.7±10.6	57.5±8.1	0.162
MABP (mm Hg)	112.2±5.5	112±5.0	0.728

BMI: body mass index; BSA: body surface area; PR: pulse rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; PP: pulse pressure; MABP: mean arterial blood pressure

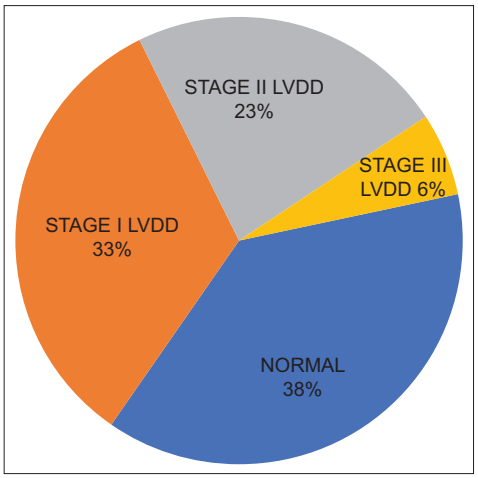

Figure 1: Distribution of treatment Naïve hypertensive patients in relation to the LV diastolic function

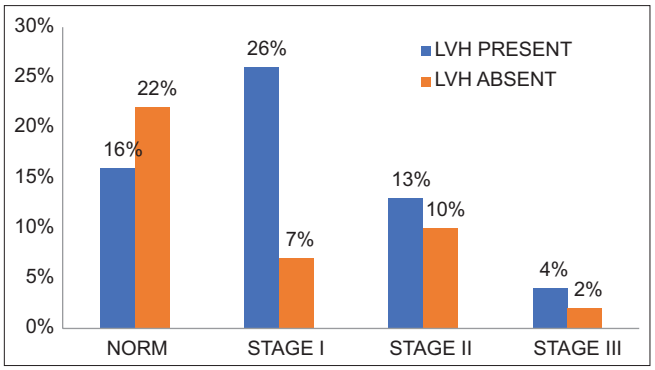

Figure 2: Distribution of left ventricle diastolic function phenotypes in relation to LVH

Table 2: The Echocardiographic characteristics of the study of population

Variables	NLVDF (*n*=114)	LVDD (*n*=186)	*P*
LVEDd (mm)	48.4±8.1	47.3±5.9	0.452
LVEDS (mm)	32.1 ±	31.4±6.3	0.647
LVEF (%)	67.6±8.9	67.1±11.5	0.783
IVSDd (mm)	9.5±1.6	10.7±2.2	0.002
LVPWDd (mm)	9.8±2.5	11.4±2.2	0.002
IVSs (mm)	13.7±3.0	15.5±3.0	0.005
LVPWDd (mm)	13.9±3.3	15.4±3.1	0.019
LVM (g)	194.1±69.7	232.0±68.9	0.01
LVMI (g/m^2)	110.0±40.7	127.3±35.7	0.034
RWT	0.40±0.11	0.48±0.12	0.002
LAV (ml)	32.2±7.8	36.9±14.2	0.035
LAVI (ml/m^2)	17.2±4.2	20.3±12.3	0.013
E vel. (m/s)	0.70±0.23	0.70±0.20	0.915
A vel. (m/s)	0.59±0.12	0.65±0.16	0.015
Dec E T (ms)	202.5±47.6	192.4±36.0	0.266
E/A	1.21±0.37	1.13±0.56	0.330
È. IVS	0.12±0.03	0.08±0.03	0.001
È. LW	0.16±0.24	0.08±0.02	0.047
È (m/s)	0.11±0.03	0.08±0.02	<0.001
E/È	6.2±1.17	8.3±2.58	<0.001
TR Vmax (m/s)	1.34±0.64	1.49±0.71	0.280
TR Pg. (mm Hg)	8.28±8.15	11.0±9.85	0.189

LVEDd: left ventricle diastolic diameter; LVEDd: left ventricle systolic diameter; LVEF left ventricular ejection fraction; IVSDd: intraventricular septum diastolic diameter; LVPWDd: posterior wall diastolic diameter; IVSSd: intraventricular septum systolic diameter; LVPWSd: posterior wall systolic diameter; LVM: left ventricular mass; LVMI: left ventricular mass index; RWT: relative wall thickness; LAV: left atrial volume; LAVI: left atrial volume index; E vel: early mitral flow velocity; A vel: late mitral flow velocity; Dec T E: early mitral flow deceleration time; È IVS: early diastolic velocity of septal mitral annulus; È LW: early diastolic velocity of lateral mitral annulus; È: early diastolic mitral annular velocity derived from averaged velocities of the lateral and septal curves; TR: tricuspid regurgitation maximum velocity; TR Pg.: tricuspid regurgitation peak gradient

were older in the LVDD group (40.9 ± 11.9) than the NLVF group (46.1 ± 10.5) (P = 0.031). There was no significant difference between the two groups in their weight, height, body mass index (BMI), and BSA, which is summarized in Table 1.

There was no significant difference in the SBP between the two groups, (148.6 ± 11.7 versus 151.0 ± 8.2, P = 0.286), in the DBP (93.9 ± 3.8 versus 93.4 ± 5.2, P = 0.532), the PP (54.7 ± 10.6 versus 57.5 ± 8.1, P = 0.162) and the MABP (112.2 ± 5.5 versus 112 ± 5.0, P = 0.728). Pulse rate (PR) was significantly higher among treatment Naïve hypertensive patients with LVDD (85.7 ± 15.5) than the treatment Naïve hypertensive NLVDF group (79.4 ± 7.6) (P = 0.008).

Table 2 demonstrates the echocardiographic characteristics of the study population, the LVIDd in the two groups were similar as well as the LVEF. There was a significant difference in the IVSTd, LVPWTD, IVSTS, and LVPWTS. The LVM (13.9 ± 3.3 versus 15.4 ± 3.1, P = 0.019), LVMI (110.0 ± 40.7 versus 127.3 ± 35.7, P = 0.034), and RWT (0.40 ± 0.11 versus 0.48 ± 0.12, P = 0.002) were significantly higher in the TNH with LVDD group than in the TNH with NLVDF group.

The mean of LAV (32.2 ± 7.8 versus 36.9 ± 14.2, P = 0.035) and the LAVI (17.2 ± 4.2 versus 20.3 ± 12.3, P = 0.013) were significantly higher in the TNH patients with LVDD than in the TNH with NLVDF. The È was significantly higher in the NLVDF (0.11 ± 0.03) than the LVDD (0.08 ± 0.02) group (P = <0.001) while the E/È (6.2 ± 1.17 versus 8.3 ± 2.58, P = <0.001), and mitral inflow A velocity (0.59 ± 0.12 versus 0.65 ± 0.16, P = 0.017) were higher in the LVDD group than the NLVDF.

Overall, 38% of the treatment Naïve hypertensive patients had NLVDF. Grade 1 LVDD was found in 33% of the patient whereas grade 2 was observed in 23% of the study population. Grade 3 LVDD was observed

Table 3: The Left ventricular diastolic function indices of the study population

Variable	Normal (*n*=114)	Stage I LVDD (*n*=99)	Stage II LVDD (*n*=69)	Stage III LVDD (*n*=18)	*F*	Anova *P*
E vel. (m/s)	0.70±0.22	0.61±0.17	0.79±0.10	0.70±0.20	4.53	0.005
A vel.(m/s)	0.59±0.12	0.73±0.13	0.59±0.13	0.63±0.15	13.6	<0.001
E/A	1.2±0.37	0.79±0.37	1.4±0.41	1.9±0.52	21.2	<0.001
Dec E T (ms)	202.5±47.6	197.27±32.8	189.9±43.3	175.3±11.5	1.01	0.391
È (m/s)	0.11±0.03	0.08±0.02	0.07±0.04	0.07±0.04	16.45	<0.001
E/È	6.2±1.2	8.1±2.4	9.3±1.5	14.1±2.1	22.86	<0.001
TR vel. (m/s)	1.34±0.64	1.49±0.69	1.49±0.79	1.60±0.66	0.380	0.768

E vel: early mitral flow velocity; A vel: late mitral flow velocity; Dec T E: early mitral flow deceleration time; È: early diastolic mitral annular velocity derived from averaged velocities of the lateral and septal curves; TR: tricuspid regurgitation maximum velocity

in only 6% of the patients which is summarized in Figure 1.

The mean E velocity was found to be significantly different across the phenotypes of LVDF (P = 0.005), similarly, the mean A velocity was also found to be a statistically significant difference across the LVDF phenotype ($P \leq 0.001$). The mean of A/E was observed to be significantly different across the groups ($P \leq 0.001$). The mean È and the mean E/È were found to be statically different across the LVDF phenotypes ($P \leq 0.001$), as summarized in Table 3.

Figure 2 depicts the distribution of LVDF phenotypes with the presence of Left Ventricular Hypertrophy (LVH), (48) 42% of the patients with NLVDF had LVH whereas (66) 58% of the NLVDF had no LVH. Of those with grade 1 LVDD, (75) 76% had LVH while (24) 24% had no LVH. In grade 2 LVDD, (33) 48% and (36) 52% were observed amongst those with LVH and those without LVH respectively. Similarly, (6) 33% of the LVDD grade 3 had LVH while (12) 67% of the LVDD grade 3 was observed in those without LVH.

Discussion

The main findings of this study include (1) a high prevalence of LVDD was observed in never-treated hypertensive patients. (2) The LVDD was more common among treatment Naïve hypertensive patients with LVH than those without LVH.

Our finding shows that treatment Naïve hypertensive patients with LVDD are older than hypertensive with normal LVDF. The LVDD is observed with increasing age and is probably due to prolongation of LV isovolumic relaxation time with advancing age and more certainly due to increased LV stiffness.[13,24]

The prevalence of LVDD among treatment Naïve hypertensive patients was observed to be 62% (186 out of 300) which is similar to the finding by Adamu *et al.*[11] in a study conducted among newly diagnosed hypertensive patients in North central, Nigeria. In south-eastern Nigeria, Ike *et al.*[12] reported a higher prevalence of 82.86%, this is explained by the fact that the participants in Ikes study were long-term hypertensive patients. A lower prevalence of 35.6% (36 out of 102) was reported in hypertensive patients in the Middle East among Saudi nationals by Sameer Al-Ghamdi *et al.*[25] Similarly, in Asia, the prevalence of 44% was reported by Mohamed *et al.*[26] among the Malay hypertensive population. This is probably due to racial variations like other cardiovascular complications of hypertension which are established to be more severe in black than other races.

LVDD is a condition in which the heart is unable to relax and fill properly during diastole, leading to impaired cardiac function. LV diastolic dysfunction is a continuum of disease that progresses from mild disease to a more advanced form and evolved from one grade 1 LVDD through grade 2 LVDD and finally to grade 3 LVDD.[13,27]

In this study, the prevalence of LVDD is relatively high among treatment Naïve hypertensive patients. We found that grade 1 LVDD was the most common type of LVDD observed, affecting one-third of the treatment Naïve hypertensive patients (33%). Grade 2 LVDD was less frequent than grade 1, affecting around one-fourth of the patients (23%), while grade 3 LVDD was observed in only 6% of the patients. This indicates that LVDD is a progressive condition, with more severe grades being less common than milder forms. We also observed that the mean age tends to increase as the LVDD grade progresses. Stage III has a higher mean age compared to both Stage I and Stage II. This in keeping with earlier study.[26]

These results are consistent with previous research that has shown a high prevalence of LVDD in hypertensive patients.[11] Hypertension is a known risk factor for the development of LVDD, which is characterized by abnormalities in the relaxation of the left ventricle during diastole. LVDD is associated with an increased risk of cardiovascular events, including HF, stroke, and cardiovascular mortality.[28]

These findings underscore the importance of early detection and management of hypertension to prevent or delay the development of LVDD. It is also important to note that LVDD can be asymptomatic in its early stages,[29] making it challenging to diagnose without appropriate screening. Therefore, routine echocardiography screening in hypertensive patients may be necessary to detect and monitor the progression of LVDD.

Based on our findings, LVH is more commonly found in treatment Naïve hypertensive patients with LVDD than in those with NLVDF. Specifically, only 16% of treatment Naïve hypertensive patients with NLVDF had LVH, compared to 22% of hypertensive patients with grade 1 LVDD.

However, suggesting that LVH may be a contributing factor to the development of LVDD in hypertensive patients. It is also interesting to note that while the prevalence of LVH was less frequent in grade 2 and 3 LVDD, it (LVDD) was still higher than in patients without LVH, suggesting that LVH may still play a role in the development of more severe forms of LVDD.

LVH is a well-established consequence of hypertension and is believed to be a compensatory response to increased pressure and workload on the heart. Over time, however, LVH can lead to impaired diastolic function, which can in turn lead to LVDD. The exact mechanisms by which LVH contributes to the development of LVDD are not fully understood but may involve changes in myocardial structure and function, alterations in calcium handling, and changes in extracellular matrix composition.[30-32]

Overall, our findings suggest that LVH may be an important factor to consider in the development and progression of LVDD in treatment Naïve hypertensive patients. Further research is needed to better understand the mechanisms underlying this relationship and to determine whether targeting LVH may be an effective strategy for preventing or treating LVDD in this population.

Conclusion

The prevalence of LVDD is high among treatment—Naive hypertensive patients. The most common form of LVDD seen in these patients is grade 1 LVDD, which is a mild form of the condition. Additionally, LVH is frequently observed among patients with LVDD. This co-occurrence suggests that LVDD and LVH may share common underlying mechanisms and may serve as indicators of cardiovascular disease.

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Conflicts of interest

There are no conflicts of interest.

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