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Association between left ventricle diastolic dysfunction and circadian blood pressure pattern in arterial hypertension

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Abstract

Objective To assess the relationship between left ventricular diastolic dysfunction (LVDD) and circadian blood pressure (BP) pattern in arterial hypertension (AH).

Methods A total of 96 patients with AH of I-II grades were examined. Transthoracic echocardiography was performed using a high-end ultrasound device GE Vivid 7 Pro (USA). 24-hour Ambulatory BP Monitoring was carried out using a Microlife Watch BP 03 device on the non-dominant arm. Statistical analysis of the obtained results was carried out using the Statistica 10.0 application package.

Results After echocardiography all patients were divided into two groups. The first group is patients with AH and LVDD ($n=37$), second group is patients with AH without LVDD ($n=59$). The dipper pattern of systolic BP (SBP) was significantly more common in patients in the second group ($p=0.014$). The non-dipper pattern of diastolic BP (DBP) was significantly more common in patients of group 1 ($p=0.047$). The odds ratio of LVDD developing in patients with AH and a non-dipper pattern of SBP and DBP was 2.907 (95% confidence interval (CI) 1.233–6.852) and 3.329 (95% CI 1.346–8.234), respectively.

Conclusion A non-dipper pattern of SBP and DBP was significantly associated with LVDD and may be a therapeutic target for preservation of the diastolic function.

Keywords Arterial hypertension, Left ventricular diastolic dysfunction, Echocardiographic parameters, Non-dipper pattern

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Background

Left ventricular (LV) diastolic dysfunction (LVDD) is characterized by alterations in LV diastolic filling, which may include impairments in myocardial relaxation and abnormal distensibility of the myocardium [1]. LVDD is a strong predictor of cardiovascular events and incident heart failure (HF) [1]. In observational cohort analysis (n = 122087) LVDD was diagnosed in 25,385 patients, which accounted for 20.8%. Over a median follow-up of 3.4 years, 2938 (2.4%) patients were hospitalized for stroke or transient ischemic attack (TIA): 1732 (1.8%) in patients without LVDD and 842 (4.6%), 290 (4.9%), and 74 (5.5%) in patients with grade I, II, and III LVDD, respectively ($p < 0.001$). Each worsening grade of LVDD was associated with an 80% increase in the risk of stroke or TIA (hazard ratio (HR) = 1.80, 95% confidence interval (CI) 1.72–1.88, $p < 0.001$) [2]. Another study examined the effect of LVDD based on the risk of myocardial infarction (MI) (n = 129,476). LVDD was present in 17.6% of the patients (13.6% Grade I, 3.6% Grade II, 0.4% Grade III). Impaired function of LV was an independent predictor of MI (Grade I: HR = 1.48, 95% CI 1.33–1.66; Grade II: HR = 1.84, 95% CI 1.57–2.16; Grade III: HR = 2.90, 95% CI 1.98–4.25) [3]. In a study by Tsang T.S. et al., the presence of LVDD was a predictor of nonvalvular atrial fibrillation. Abnormal relaxation, pseudonormal, and restrictive LV diastolic filling were associated with HR of 3.33 (95% CI 1.5–7.4, p 0.003), 4.84 (95% CI 2.05–11.4, p 0.001), and 5.26 (95% CI 2.3–12.03, p 0.001), respectively, when compared with normal diastolic function [4]. Zhou D. et al. studied the effect of LVDD on the risk of major adverse cardiac events (MACE) (MI, coronary revascularization procedures, HF, stroke, all-cause mortality) in patients with arterial hypertension (AH). The study included 283 hypertensive patients. LVDD was diagnosed in 12.3%. During mean follow-up of 5.4 years, there were 26.6% patients with MACE in the LVDD group at baseline, 9.9% patients with MACE in the group with normal diastolic function. LVDD independently predicted MACE (HR = 2.50, 95% CI 1.20–5.25, p = 0.032) in a model including age, sex, diastolic blood pressure (BP) (DBP), and E/e′ ratio (c-statistics 0.805) [5].

AH has been reported as the most important risk factor for LVDD in the community [1]. High BP may induce LVDD through several potential mechanisms, including hemodynamic and non-hemodynamic factors [1]. Chronic pressure overload leads to the development of LV hypertrophy (LVH). Progressive hypertrophy and fibrotic changes in the heart lead to progressive diastolic dysfunction, ultimately leading to elevated left-sided filling pressures [6]. Among non-hemodynamic factors in the development of LVDD in patients with AH, the role of age, obesity, diabetes mellitus, and chronic kidney disease has been proven [1].

The search for predictors of LVDD continues, and therefore, the study of the influence of different BP profiles on the risk of developing LVDD seems highly relevant.

Objective

To assess the relationship between LVDD and circadian BP pattern in AH.

Methods

Between September 2023 and April 2025, we conducted a single center trial. Our study involved 96 outpatients with grades I-II AH. The average age of the subjects was 60.0 [57.0; 63.0] years. There were 46 men (47.9%) and 50 women (52.1%). The average body mass index (BMI) was 30.6 [27.8; 34.2] kg/m^2. The duration of hypertension history was 14.0 [10.0; 22.0] years.

Patients with AH of grade III, secondary hypertension, ischemic heart disease, non-coronary myocardial diseases, heart defects, heart rhythm disturbances (ventricular extrasystole above Lown class 2, atrial fibrillation, ventricular tachycardia, WPW syndrome), chronic HF with LV ejection fraction (EF) less than 50%, chronic kidney disease with a glomerular filtration rate of 60 ml/min/1.73 m^2 and below, liver dysfunction and diabetes mellitus, using of benzodiazepine or benzodiazepine receptor agonists, anticonvulsants, antipsychotic medications, or antidepressant medications were excluded from the study.

Clinical characteristics (age, sex, smoking, medical history, antihypertensive medication history) were collected using a standardized questionnaire by trained community staff. Waist circumference (WC), hip circumference (HC), weight and height were measured in participants wearing light clothing and standing with no shoes. BMI was calculated as BMI = weight/height (kg/m^2). Body surface area (BSA) was calculated as BSA =(Weight $^{0.425}$ × Height $^{0.725}$) × 0.007184 [7]. Office BP and heart rate was measured after resting for 5 min.

Fasting blood samples were collected to analyze plasma glucose, creatinine, lipid profile, alanine aminotransferase, aspartate aminotransferase, and serum uric acid. The glomerular filtration rate was estimated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.

The identification of risk factors included the assessment of the incidence of smoking, obesity, hypercholesterolemia, and hyperuricemia. Individuals were considered smokers if they were past or current smokers. BMI ≥ 30 kg/m^2 indicated obesity [8]. Hypercholesterolemia was determined when the total cholesterol level was >4.9 mmol/L and/or hypolipidemic therapy was used [8]. Hyperuricemia was defined as an increase in uric acid level of ≥ 360 μmol/L [8].

Transthoracic Echocardiography (Echo-CG) was performed using a high-end ultrasound device GE Vivid 7 Pro (USA). The following parameters were assessed: left atrium (LA) diameter (LAD), LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), and LV EF. The LA volume (LAV) was determined and then indexed to the BSA and height to the second power. The normal value of LAV/BSA is 34 ml/m^2 or less for both sexes [9]. LA dilation was determined with LAV/height^2 >18.5 ml/m^2 in men and >16.5 ml/m^2 in women [9]. The presence of LVH was assessed using the most commonly used LV mass (LVM) indices – to the BSA and height to the power of 2.7. The LVM/BSA of >115 g/m^2 and >95 g/m^2 were considered as LVH for males and females, respectively. LVH was determined at LVM/height$^{2.7}$ in men over 50 g/$m^{2.7}$ and in women over 47 g/$m^{2.7}$ [9].

LVDD was diagnosed in accordance with the Russian clinical guidelines for chronic HF 2020 [10]. The following parameters were recorded: transmitral early and late diastolic peak velocity (E and A, m/s), and lateral and septal early diastolic mitral annular velocity (lateral e' and septal e', cm/s). The average e' (e'), the ratio E/A, E/e'$_{lat}$, E/e'$_{sept}$ and E/e'$_{avg}$ were calculated. When assessing the state of LV diastolic function, one should first of all focus on the E/A ratio. If the E/A ratio is ≤ 0.8 and the E is ≤ 50 cm/sec, the patient has LVDD grade I. If the E/A ratio is >2, the patient had severe grade III LVDD. In all other cases, three other criteria are used: (1) E/e'>14; (2) LAV/BSA >34 ml/m^2; (3) maximum tricuspid regurgitation (TR) jet velocity >2.8 m/s. If there were at least two criteria, this meant that the patient had LVDD grade II. If there was no more than one criterion, the patient was classified as having LVDD grade I [10].

24-hour Ambulatory BP Monitoring (ABPM) was carried out using a Microlife Watch BP 03 device on the non-dominant arm. The device was programmed to measure BP at 30-min intervals during the day and at 60-min intervals during the night. Based on the decrease in mean night time BP compared to mean day time BP, patients are classified as dippers (>10% – 20%), non-dippers (>0% – 10%), reverse-dippers (≤ 0%), and extreme dippers (>20%) [11]. Night time was recorded as the period between the time when the patient went to bed and the time when the patient woke up the next morning according to his diary.

Statistical analysis was conducted using the Statistica 10.0 application program package. The results are presented as the median (Me) and interquartile range [LQ; UQ] for continuous variables or the number (%) for dichotomous variables. The Mann – Whitney U test was used to compare two independent groups. The category distributions between groups was compared using Pearson's χ^2 homogeneity criterion. In the case of two compared groups and two categories, the Yates correction for Pearson's χ^2 criterion was used. If the conditions for employing Pearson's chi-squared homogeneity criterion were not met, Fisher's exact test was employed. If this criterion indicated the presence of statistically significant differences between the groups, then pairwise comparisons of the distributions were then carried out using the Holm correction for p-values. Odds ratios (OR) were determined from four-field contingency tables using an online calculator [12]. At $p < 0.05$, the differences were considered statistically significant.

Results

After echo-CG all patients were divided into two groups. The first group is patients with AH and LVDD ($n = 37$) (94.6% Grade I, 5.4% Grade II), second group is patients AH without LVDD ($n = 59$). The presented groups are comparable in terms of demographic, clinical, and laboratory characteristics of the participants (Table 1).

No significant differences were found in the groups by smoking status, obesity, hypercholesterolemia, hyperuricemia (p˃0.05).Overall, several patients had long standing hypertension with a median duration of 14.0 years and elevated BP levels despite stable antihypertensive therapy.

In terms of antihypertensive therapy and statins using, patients in both groups were comparable (Table 2).

The echo-CG results are presented in Table 3. Statistically significant differences in LA size were found between the groups. When assessing the LAV/BSA, chamber dilation was detected in 25 patients (67.6%) with LVDD, while in patients with normal LV function, no patient showed LA enlargement ($p = 0.0000$). LA dilation according to the LA volume/height^2 was detected in 33 (89.2%) and 47 (76.7%) patients in groups 1 and 2, respectively; however, the differences between the groups were not statistically significant ($p = 0.271$). The incidence of LVH according to the LVM/BSA in patients with AH and LVDD was statistically significantly higher compared to patients with normal diastolic function (73.0 and 52.5%, respectively, χ^2=3.97, $p = 0.046$). According to the LVM/height$^{2.7}$ the proportion of individuals with LVH in groups 1 and 2 was 78.4% and 69.5%, respectively ($p > 0.05$).

The results of ABPM in the groups of subjects are presented in Table 4. The groups were comparable in terms of daily, daytime, and nighttime values of SBP and DBP.

In patients with LVDD, the distribution of patients by the nature of the daily SBP profile was as follows: dipper – 11 (29.7%), non-dipper – 20 (54.1%), extreme-dipper – 2 (5.4%), reverse-dipper – 4 (10.8%). In patients without LVDD, the number of dippers in SBP was 36 (61.0%), non-dippers – 17 (28.8%), extreme -dippers – 5 (8.5%), reverse-dipper – 1 (1.7%).

Table 1 General characteristics of the study groups

Parameter	Group 1 (*n* = 37)	Group 2 (*n* = 59)	*P*
Age, year	60.0 [58.0; 62.0]	60.0 [56.0; 63.0]	0.886
Female gender, n (%)	20 (54.1)	30 (50.8)	0.760
Duration AH, years	15.0 [10.0; 20.0]	13.0 [10.0; 22.0]	0.763
AH grade I, n (%)	13 (35.1)	26 (44.1)	0.386
AH grade II, n (%)	24 (64.9)	33 (55.9)	0,386
Cigarette smoking, n (%)	13 (35.1)	21 (35.6)	0.862
BMI, kg/m^2	32.3 [27.8; 35.0]	30.1 [27.7; 32.7]	0.228
Obesity, n (%)	22 (59.5)	30 (50.8)	0.539
WC, cm	107.0 [100.0; 115.0]	103.0 [97.0; 110.0]	0.166
HC, cm	114.0 [107.0; 125.0]	112.0 [107.0; 119.0]	0.527
WHR	0.92 [0.88; 0.97]	0.92 [0.87; 0.96]	0.754
SBP, mmHg	140.0 [130.0; 146.0]	138.0 [130.0; 150.0]	0.835
DBP, mmHg	90.0 [82.0; 90.0]	86.0 [80.0; 90.0]	0.507
HR, bpm	72.0 [64.0; 76.0]	72.0 [68.0; 76.0]	0.690
TC, mmol/L	5.4 [4.6; 6.1]	5.7 [5.1; 6.6]	0.145
Hypercholesterolemia, n (%)	31 (83.8)	54 (91.5)	0.407
HDL-C, mmol/L	1.5 [1.3; 1.7]	1.5 [1.1; 1.8]	0.979
LDL-C, mmol/L	3.3 [2.6; 3.8]	3.6 [3.0; 4.1]	0.070
TG, mmol/L	1.4 [1.2; 1.9]	1.5 [1.0; 1.9]	0.532
Glucose, mmol/L	4.3 [3.8; 4.9]	4.3 [3.8; 4.9]	0.665
Uric acid, µmol/L	314.0 [270.0; 371.0]	339.0 [292.0; 376.0]	0.611
Hyperuricemia, n (%)	12 (32.4)	22 (37.3)	0.791
Creatinine, µmol/L	83.0 [76.0; 92.0]	85.0 [79.0; 95.0]	0.382
GFR, ml/min/1.73 m^2	76.0 [67.0; 82.0]	72.0 [64.0; 82.0]	0.365
ALAT	28.0 [21.0; 32.0]	27.0 [22.0; 35.0]	0.889
ASAT	28.0 [22.0; 34.0]	26.0 [22.0; 32.0]	0.331

Abbreviations ALAT alanine aminotransferase, *ASAT* aspartate aminotransferase, *GFR* glomerular filtration rate, *HDL-C* high-density lipoprotein cholesterol, *HR* Heart Rate, *LDL-C* low-density lipoprotein cholesterol, *TC* total cholesterol, *TG* triglycerides

Table 2 Pharmacological therapy in groups

Drug Class	Group 1 (*n* = 37)	Group 2 (*n* = 59)	*P*
Statins, n (%)	9 (24.3)	12 (20.3)	0.837
ACE Inhibitor, n (%)	17 (45.9)	25 (42.4)	0.731
ARB, n (%)	15 (40.5)	22 (37.3)	0.750
Diuretics, n (%)	6 (16.2)	8 (13.6)	0.720
BB, n (%)	16 (43.2)	29 (49.2)	0.572
CCB, n (%)	6 (16.2)	14 (23.7)	0.378
AHT at the time of inclusion in the study:			
Absent	0 (0.0)	5 (8.5)	0.069
Monotherapy	12 (32.4)	21 (35.6)	0.751
Combination Therapy	25 (67.6)	33 (55.9)	0.257

Abbreviations ACE Angiotensin Converting Enzyme, *AHT* Antihypertensive Therapy, *ARB* Angiotensin II Receptor Blockers, *BB* Beta Blockers, *CCB* Calcium Channel Blockers

Table 3 Echocardiographic parameters of the study group

Parameter	Group 1 (*n* = 37)	Group 2 (*n* = 59)	*P*
LAD, mm	41.0 [38.0; 43.0]	37.0 [35.0; 39.0]	0.00006
LAV/BSA, ml/m^2	36.0 [32.0; 38.0]	29.0 [25.0; 30.0]	0.0000
LAV/height^2, ml/m^2	25.0 [22.3; 27.7]	19.5 [18.0; 21.6]	0.0000
Lateral e', sm/s	8.0 [6.0; 10.0]	10.0 [8.0; 11.0]	0.005
Septal e', sm/s	5.9 [5.1; 7.0]	7.1 [6.0; 8.6]	0.0009
E/e'$_{lat}$	8.1 [6.6; 9.5]	7.5 [6.7; 8.6]	0.172
E/e'$_{sept}$	10.1 [8.5; 12.0]	10.0 [8.6; 11.1]	0.237
E/e'$_{avg}$	9.2 [7.5; 11.0]	8.9 [8.0; 9.8]	0.219
LV EDD, mm	51.6 [47.0; 55.0]	49.0 [46.5; 52.0]	0.087
LV ESD, mm	32.0 [30.0; 34.0]	31.0 [29.0; 32.8]	0.055
LV EDV, ml	128.0 [100.0;148.0]	113.0 [100.0; 132.0]	0.109
LV ESV, ml	40.0 [34.0; 49.0]	37.0 [30.0; 42.0]	0.059
LV stroke volume, ml	85.0 [68.0; 96.0]	78.0 [66.0; 89.0]	0.230
LV EF, %	66.0 [64.0; 69.0]	68.0 [65.0; 71.0]	0,079
IVS, mm	13.0 [11.0; 14.0]	12.0 [11.0; 13.5]	0,063
LV PWT, mm	12.0 [10.0; 13.0]	11.0 [10.0; 12.0]	0.079
LVM, g	247.0 [202.0; 285.0]	219.0 [183.0; 254.0]	0.015
LVM/BSA, g/m^2	117.0 [106.0;137.0]	109.0 [96.3; 126.0]	0,044
LVM/height$^{2.7}$, g/m^{2,7}	57.4 [51.2; 70.0]	53.4 [47.0; 63.8]	0.040
Peak E, m/s	0.65 [0.50; 0.77]	0.70 [0.60; 0.80]	0.057
Peak A, m/s	0.76 [0.62; 0.87]	0.75 [0.64; 0.90]	0.946
E/A ratio	0.80 [0.68; 1,0]	1,0 [0.80; 1.10]	0.060
TR jet velocity, ms	2.4 [2.2; 2.5]	2.3 [2.2; 2.4]	0.034

AbbreviationsIVS Interventricular Septal thickness, *LV PWT* Left Ventricular Posterior Wall Thickness

Table 4 ABPM indicators in the groups of subjects examined

Parameter	Group 1 (*n* = 37)	Group 2 (*n* = 59)	*P*
SBP Daily, mmHg	122.0 [117.0; 135.0]	125.0 [115.0; 131.0]	0.898
DBP Daily, mmHg	75.0 [71.0; 85.0]	78.0 [71.0; 82.0]	0.946
SBP Day time, mmHg	130.0 [117.6; 137.0]	131.0 [121.0; 138.0]	0.442
DBP Day time, mmhg	78.2 [74.0; 87.0]	81.0 [76.0; 87.0]	0.228
SBP Night time, mmHg	116.0 [106,0; 134,0]	113,0 [106,0; 123.0]	0.202
DBP Night time, mmHg	72.0 [63.0; 81.0]	69.0 [62.0; 73.0]	0.078
Systolic Dipping, %	5.9 [1.7; 10.7]	11.7 [8.6; 16.2]	0.00003
Diastolic Dipping, %	9.0 [4.1; 14.5]	16.3 [12.4; 20.4]	0.00004

In patients with LVDD, the distribution of patients by the nature of the daily DBP profile was as follows: dipper – 13 (35.1%), non-dipper – 17 (46.0%), extreme dipper – 4 (10.8%), reverse dipper – 3 (8.1%). In patients without LVDD, the number of dippers in DBP was detected in 31 (52.6%) of patients, 12 (20.3%) with a non-dipper profile, 16 (27.1%) – with an extreme-dipper profile. The reverse-dipper profile was absent in group 2.

When comparing the groups by the distribution of daily SBP and DBP profiles, statistically significant differences were found (p = 0.005 and p = 0.002, respectively). When pairwise comparing the types of daily SBP profiles between groups 1 and 2, statistically significant differences were found in the frequency of dippers (p = 0.014). The incidence of non-dippers for SBP tended to be higher in patients with LVDD (p = 0.054). At the same time, the daily non-dipper profile for DBP is significantly more common in group 1 (p = 0.047).

The OR of LVDD developing in patients with AH and a non-dipper pattern for SBP was 2.907 (95% CI 1.233–6.852), while for dipper it was 0.270 (95% CI 0.112–0.650). The OR of LVDD developing in hypertensive patients and a non-dipper pattern for DBP was 3.329 (95% CI 1.346–8.234). The presence of a dipper pattern for DBP did not reduce the risk of LVDD developing in patients with AH (OR = 0.489, 95% CI 0.210–1.141)

Discussion

In our study, LVDD was detected in 38.5% of hypertensive patients, which is consistent with the results of other studies.In the study performed by Al-Ghamdi S., LVDD was diagnosed in 35.6% of patients with AH [13]. In the CARE NORTH study, the incidence of LVDD among hypertensive participants was 43.7%. Grade I LVDD was observed in 24.4% whereas grade II LVDD was found in 19.3% of the patients. None of the studied patients were characterized by grade III LVDD [14]. Among newly diagnosed hypertensive Nigerians LVDD was found in 62% of the patients (grade I – 33%, grade II – 23%, grade III – 6%) [15]. Thus, the incidence of LVDD varies from 36% to 62%, which can be explained by various factors, including age, female gender, obesity, diabetes mellitus, duration of AH, as well as the use of different diagnostic criteria for the diagnosis of LVDD [14].

Several patients had long standing hypertension with elevated BP despite stable anti hypertensive therapy, which could influence the association between circadian BP patterns and LVDD.Our work has established that patients with AH and LVDD are significantly more likely to have LVH and LA dilation, which is consistent with the results of other studies. Abubakar I.G. et al. showed a significant difference in the LVM/BSA (110.0 ± 40.7 vs. 127.3 ± 35.7, p = 0.034) and the LAV/BSA (17.2 ± 4.2 vs. 20.3 ± 12.3, p = 0.013) in hypertensive patients with normal LV diastolic function and LVDD respectively [15]. Masugata H. et al. reported that E/e′ indicated a progressive worsening of LV diastolic function from hypertensive patients with normal geometry to patients with concentric remodelling, and then to patients with eccentric and concentric LVH (10.2 ± 3.0, 10.5 ± 3.5, 11.1 ± 3.6 and 13.4 ± 5.4, respectively) [16]. Pritchett A. et al. demonstrated that the LAV/BSA increased with worsening LVDD: 23 ± 6 ml/m^2 (normal diastolic function), 25 ± 8 ml/m^2 (grade I LVDD), 31 ± 8 ml/m^2 (grade II LVDD), 48 ± 12 ml/m^2 (grade III LVDD) [17].

Our study established a relationship between LVDD and the non-dipper 24-hour BP profile. Aydin M. et al. reported that non-dipper patient had worse parameters reflecting diastolic function compared to dippers in AH: the transmitral E-wave decreased (0.55 ± 0.2 vs 0.62 ± 0.2 m/s, p < 0.05), transmitral A-wave increased (211 ± 44 vs 196 ± 42 ms, p < 0.05), E-wave deceleration times increased (0.77 ± 0.1 vs 0.70 ± 0.1 m/s, p < 0.01), the transmitral E/A ratio decreased (0.78 ± 0.1 vs 0.86 ± 0.2, p < 0.05) and the isovolumic relaxation time (112 ± 15 vs 105 ± 14 m/s, p < 0.05) [18]. Yeter E. et al. studied diastolic function in dippers and non-dippers with prehypertension. The transmitral E-wave, transmitral A-wave, E-wave deceleration times and transmitral E/A ratio were not significantly different between the dipper and non-dipper groups. But the septal e' (9.2 ± 2.3 vs 7.0 ± 1.2, p < 0.01), lateral e' (7.23 ± 1.84 vs 8.25 ± 2.36, p < 0.05), E/e' septal (9.02 ± 2.44 vs 11.77 ± 3.09, p < 0.01), E/e' lateral (11.4 ± 2.4 vs 10.2 ± 2.4, p < 0.05) were significantly different between the groups [19]. However, according to the results of another study, carried out by Erdogan et al. showed that diastolic function parameters were similar between dippers and non-dippers [20]. It should be noted that in our study, patients were divided into groups depending on the presence or absence of LVDD, whereas in the above-mentioned studies, the division into groups was based on the type of daily BP profile (dipper or non-dipper). Our group of authors assessed the circadian BP pattern of SBP and DBP separately whereas the studies Aydin M. et al. and Erdogan et al. included only those with the same daily profile of SBP and DBP. In these studies, the authors do not provide results on the frequency of occurrence of LVDD in groups, but only evaluate parameters reflecting diastolic function.

Recently, chronotherapy has received increased attention as a means of improving control of nocturnal hypertension and a non-dipping BP pattern [21]; its use in patients with LVDD and non-dipping circadian BP profile will improve diastolic function.

Conclusion

In summary, our results showed that the 24-hour non-dipper profile was significantly associated with LVDD. This study provides a foundation for further investigations aimed at investigating the use of hypertension chronotherapy to improve LV diastolic function.

Limitations

This study has several limitations that should be considered in the interpretation of our results. First, this is a single-center study with a limited number of patients

involved. This sample size limitation is due to the difficulty of predicting the required sample power in advance due to the absence of previous large studies demonstrating the relationship between LVDD and the non-dipper circadian BP profile. Extrapolating these results to the general population needs validation from larger multicentre studies. Second, a significant limitation of the study conducted is its cross-sectional nature. It is necessary to conduct prospective observation of patients with AH and a non-dipper circadian BP profile in order to detect LVDD during dynamic observation. Third, It should be noted that the median BMI of participants is 30 kg/m^2.Therefore, these findings may primarily apply to hypertensive patients with elevated body weight.It should also be noted that patients were not screened for obstructive sleep apnea syndrome at the time of inclusion in the study.However, it is known that none of the study participants received CPAP-therapy.

Abbreviations

ABPM	Ambulatory blood pressure monitoring
AH	Arterial hypertension
BMI	Body mass index
BP	Blood pressure
BSA	Body surface area
CI	Confidence interval
DBP	Diastolic blood pressure
Echo-CG	Echocardiography
HC	Hip circumference
HF	Heart failure
HR	Hazard ratio
LA	Left atrium
LAD	Left atrium diameter
LAV	Left atrial volume
LV	Left ventricular
LVDD	Left ventricular diastolic dysfunction
LVEDD	Left ventricular diastolic dysfunction
LVEDV	Left ventricular end-diastolic volume
LV EF	Left ventricular ejection fraction
LVESD	Left ventricular end-systolic diameter
LVESD	Left ventricular end-systolic volume
LVH	Left ventricular hypertrophy
LVM	Left ventricular mass
MACE	Major adverse cardiac events
MI	Myocardial infarction
OR	Odds ratios
OSAS	Obstructive sleep apnea syndrome
SBP	Systolic blood pressure
TIA	Transient ischemic attack
TR	Tricuspid regurgitation
WC	Waist circumference
WHR	Waist to hip ratio

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Authors' contributions
IUE– statistical processing, text editing and interpretation of the results obtained; NVB– concept and design of the article, text editing, interpretation of the results obtained; LNS – collection of the results, text writing; SJNS– collection of the results, text writing; SLE – collection of the results, preparing tables; APV – collection of the results, text writing; LVY- interpretation of the results obtained, literature review. All authors have read and approved the final version of the manuscript for publication.

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Data availability
All data generated and analysed during this study are included in the article.

Declarations

Ethics approval and consent to participate
The study protocol was approved by the Biomedical Ethics and Deontology Committee of Grodno State Medical University (protocol No. 1 of 11.01.2021). An informed written consent was obtained from all the patients. This study adheres to the principles in the declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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