

Can childhood white coat hypertension affect left ventricular mass?

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Abstract

Background

The aim of this study is to investigate whether children with white coat hypertension (WCH) have evidence of left ventricular geometrical changes.

Methods

A total of 237 (161 male) patients and 122 (83 male) controls with a mean age of 12.69 ± 3.34 years were included in the study. Patients were divided into two main groups as normal weight ($n=191$) and obese ($n=168$) based on body mass index (BMI). Each group were further divided into three groups based on their blood pressure profile as hypertension (HT), WCH and control. All participant has been performed echocardiography, WCH and HT groups has been also performed ambulatory blood pressure monitoring (ABPM) and biochemical analysis. Cardiac geometry was categorized as Concentric Left Ventricular Hypertrophy [cLVH; increased left ventricular mass index (LVMI) and increased relative wall thickness (RWT)], Eccentric LVH (eLVH; increased LVMI and normal RWT), Concentric Geometry (CG; normal LVMI and increased RWT)

Results

In the present study, LV geometrical changes were observed as 36.2%, 22%, and 15% in the obese groups [HT/Obese ($n=94$), WCH/Obese ($n=41$), and Control/Obese ($n=33$), respectively, $p=0.04$]. However, it was 26.3%, 11.1%, and 1% in the non-obese groups [HT/Non-Obese ($n=57$), WCH/Non-Obese ($n=45$), and Control/Non-Obese ($n=89$), respectively, $p=0.000$] (Linear-by-linear association for obese 0.027 and for non-obese 0.000).

Conclusion

WCH can be another risk factor for cardiovascular disease. WCH can cause LV geometric changes and can be considered a pre-hypertensive intermediate state. The target-organ damage can manifest in WCH patients, especially those who are obese, or who have non-dipping BP patterns or family history

Introduction

Both obesities induced left ventricular hypertrophy (LVH) and hypertension (HT) induced LVH have several risk factors for adverse cardiovascular events in adulthood [1, 2]. Some research involving adults has found that white coat hypertension (WCH) may be an independent risk factor for increased LVMI and that patients with WCH may have an elevated long-term incidence of stroke [3, 4]. Few studies have evaluated LVMI in children with WCH [5–8]. The aim of this study is to investigate whether children with WCH have evidence of left ventricular geometrical changes in both obese and non-obese populations.

Methods

This retrospective observational study performed hypertensive children admitted to the Pediatric Nephrology Department of Bahçeşehir University's Medical Park Göztepe Hospital. The local ethics committee of Bahçeşehir University's Faculty of Medicine (25.02.2021/22481095-020-348) approved the study. Informed consent was provided from each of the participants and parents.

All hypertensive patients had diagnosed accordance with a scientific statement from the American Heart Association [9]. The thresholds for HT in children are based on the American Academy of Pediatrics Hypertension Guidelines [10]. Normotension for those 13 years and older is defined as $< 120/80$ mmHg in addition to having a 24-hr systolic blood pressure (SBP) and 24-hr diastolic blood pressure (DBP) load < 25 th % as confirmed by ABPM. As their office BP was high and ABPM was normal, these patients were considered to have white coat hypertension (WCH). The mean values of arterial pressure, systolic load and diastolic load, and dipping status were evaluated by ABPM. The BP load and dipping were interpreted according to the concerned literature [11–13]. The body mass index (BMI) ≥ 95 th percentile for age and gender is defined obesity, while BMI between the 85 to the 95th percentile for age and gender is defined overweight. The BMI less than the 85th percentile for age and sex is defined non-obesity [14]. Left ventricular hypertrophy (LVH) in children is indicated as a left ventricular mass index (LVMI) that is greater than 95th percentile for age and gender [15].

Patients were divided into two main groups as normal weight and obese based on BMI. Each group were further divided into three groups based on their blood pressure profile as HT, WCH and control. Patients who applied to the Pediatric Cardiology Department due to murmur etiology and were diagnosed with an innocent murmur were assigned to control of three groups based on age, gender, and BMI.

The following assessments were collected for all patients: weight, height, BMI, blood and urine chemistry parameters (plasma creatinine, urea, electrolytes, uric acid, total cholesterol, triglycerides, fasting plasma glucose, urinalysis, urine culture, first morning urine albumin, and creatinine ratio) and left ventricular mass index (LVMI). Newly diagnosed and previously untreated patients were included in the study population. Exclusion criteria were: (i) the existence of any chronic disease or need for chronic pharmacological treatment such as chronic kidney disease (CKD), diabetes, chronic heart disease, congenital kidney, and urinary tract abnormality, (ii) infections in the 6 weeks prior enrollment, (iii) use of any medications during the study or in the prior 6 weeks, (iv) the licensed athletes, and (v) missing information.

Ambulatory blood pressure monitoring

24-h ABPM was performed using SCHILLER BR-102 plus monitors (Schiller, Switzerland). The device measured BP readings every 30 min from 22:00 to 07:00, and every 20 min from 07:00 to 22:00. Patients' own declaration determined the wake and sleep periods for ABPM and BP parameters were studied using SCHILLER software. A proper sized cuff was put on the nondominant arm, and information regarding the use of the device were provided to the child and parents.

Echocardiography

An echocardiographic analysis was applied using a Vivid 3 device with a 3-MHz transducer. A whole transthoracic echocardiographic examination of cardiac anatomy and function were performed for every patient. Measurements of left ventricular end-systolic dimension (LVESd), left ventricular end-diastolic dimension (LVEDD), interventricular septal thickness (IVSd), and posterior wall thickness (LVPWd) were made in M-mode in the parasternal long-axis view complying with the American Society of Echocardiography recommendations [16]. Measurements were repeated two times, and the mean was figured out. The LVM was calculated according to the Devereux formula $[0.8 \times \{1.04 \times [(LVEDd + LVPWd + IVSd)^3 - LVEDd^3] + 0.6 \text{ g}]$. The LVMI was calculated by normalizing the LVM by height to the power of 2.7 [16]. A LV relative wall thickness (RWT) of >0.42 indicates concentric geometry [18]. LVH was described as $LVMI \geq 95$ th percentile accordingly age and sex [10, 19]. Cardiac geometry was categorized on the basis of LVMI and RWT into 4 subgroups: Concentric LVH (cLVH), Eccentric LVH (eLVH), Concentric Geometry (CG), and Normal Geometry (NG). The cLVH was defined as both increased LVMI and increased RWT, eLVH was defined as increased LVMI and normal RWT, CG was defined as normal LVMI and increased RWT, and NG is normal LVMI and normal RWT.

Statistical analysis

All statistical calculations were conducted using SPSS for Windows 24.0. We compared the three groups using one-way ANOVA for normally distributed data and by Kruskal-Wallis for non-normally distributed data ($p < 0.05$). Pairwise comparison was performed using the Post-hoc Bonferroni for normally distributed parameter tests ($p < 0.01$). Pairwise comparison was performed using the independent samples Kruskal Wallis test for non-normal distribution. The pair groups comparison was performed using the independent-samples T Test (if normally distributed data) and the Mann-Whitney U (if non-normally distributed data). The chi-square test was used for the comparison of categorical data. The counts (percentage) expression are used for discrete variables, mean (standard deviation) expression are used for continuous variables with normal distribution and median (interquartile ranges; Q1–Q3) expression are used for continuous variables with non-normal distribution. Statistical significance was defined as $p < 0.05$.

Results

Clinical and demographic characteristics

A total of 237 (161 male) patients and 122 (83 male) controls with a mean age of 12.86 ± 2.91 and 12.6 ± 3.01 years were included in the study. Of these, 94 were hypertensive obese (HT/O), 41 were white coat hypertensive obese (WCH/O), 57 were hypertensive normal weight (HT/non-O), and 45 were white coat hypertensive normal weight (WCH/Non-O) according to ABPM readings. In addition, according to office blood pressure measurements, 33 obese normotensive and 89 normal weight normotensive children (C/O vs C/Non-O) were included in control groups.

There were no significant differences between the three obese groups (HT/O, WCH/O, C/O) in terms of age, gender, and BMI. Also, there were no significant differences between the two obese patients's groups (HT/O, WCH/O) for number of symptoms, and symptom duration. The same findings were existed in non-obese groups (Table 1). There was an increased incidence of family cardiovascular disease history (such as HT, diabetes mellitus, dyslipidemia, hereditary kidney or endocrine disease, stroke, ischemic heart disease and neurocutaneous syndrome) in the hypertensive and WCH groups than the control groups.

Office and ambulatory blood pressure monitoring

In obese groups, the mean office systolic blood pressure measurement was higher in the patient groups than control group (HT/O~WCH/O>C/O, $p < 0.000$). There were no differences in mean diastolic blood pressure between obese groups. In normal weight groups,

both mean office systolic and diastolic blood pressure measurement were higher in patient groups than control groups (HT/Non-O~WCH/Non-O > C/Non-O, respectively $p = 0.000$ vs 0.003) (Table 2a).

According to ABPM measurements, there were no significant differences in dipping systole, dipping diastole, and pulse between HT/O vs WCH/O and HT/Non-O vs WCH/Non-O groups (Table 2b).

Echocardiography

There were significant differences in RWT between obese groups ($p=0.001$). According to the pairwise comparison, RWT was higher in the HT/O group than the C/O group ($p=0.008$). There were no differences in other pairwise comparisons.

In normal weight groups, LVM z-score, LVMI $\text{g}/\text{m}^{2.7}$ and LVMI $\text{g}/\text{m}^{2.7}$ z-score were different between groups. According to the pairwise comparison, all three parameters were higher in the HT/Non-O group than the C/Non-O group ($p=0.002$, 0.000 , 0.000 , respectively). (Table 3).

In comparison of normal geometry and other geometrical changes (CG, eLVH, cLVH), there were significant differences in both obese and normal weight groups ($p=0.004$ and 0.000 , respectively). In obese groups, abnormal cardiac geometric changes were 36.2%, 22%, and 15%, respectively, for HT/O, WCH/O, and C/O (Linear-by-linear association was 0.027). In normal weight groups, abnormal cardiac geometric changes were 26.3%, 11.1%, and 1%, respectively, for the HT/Non-O, WCH/Non-O, and C/Non-O (Linear-by-linear association was 0.000).

Biochemistry

There were no significant differences in biochemical parameter between groups (Table 4)

Discussion

The aim of this study was to investigate whether children with WCH have evidence of left ventricular geometrical changes in both obese and non-obese populations. The major findings from the study population are as follows: the rate of cardiac geometric abnormality decreased linearly according to having HT, WCH or normal blood pressure. In addition, a family history of cardiovascular disease, and therefore genetic disposition, appears to be an important risk factor in patients with both hypertension and WCH. Although the ABPM mean values of those with WCH were normal, their non-dipping status was similar to those diagnosed with HT.

It is questioned whether the WCH phenotype is innocent. In research by Stabouli et al., among 85 children (27% obese) who were evaluated for suspected hypertension and underwent ABPM, 11 (12.9%) had WCH and 21.7% of these patients were obese. The LVMI was calculated by dividing left ventricular mass by height^{2.7}. No significant differences were found in the LVMI between normotensives, white-coat hypertensives, masked hypertensives, and hypertensives [5]. Stabouli et al. also determined that children with WCH had greater BMIs than those with confirmed normotension. In a study by McNiece, 32 WCH patients (mean age 12.4 ± 2.5 years) were found to have LVH with a prevalence of 9.4%. However, nearly half of these patients were obese, and LVH was defined as $\text{LVMI} > 51 \text{ g}/\text{m}^{2.7}$ [7]. Lande's study included groups of normotensives, WCH, and sustained hypertensives. Each group was sex, age, and BMI matched and had 27 patients. LVH was defined as $\text{LVMI g}/\text{m}^{2.7} \geq$ the 95th percentile. Although no LVH was found in any subject in the normotensive or WCH groups, the mean LVMI of the WCH group was significantly higher than the normotensives [8]. This result suggests that WCH may be intermediate between that of normotensives and sustained hypertensives for hypertensive end-organ effects. Pall's study investigated normotensive, WCH, and sustained HT groups. In this study, 47 WCH patients aged 16.3 ± 1.1 years were evaluated for LVH. LVH was defined as $\text{LVMI g}/\text{m}^{2.7} \geq$ the 95th percentile. BMI was higher in the HT and WCH groups compared to the normotensives. While there were no differences between the LVMI of the normotensive and WCH groups, six (12.7%) cases in the WCH group were diagnosed with LVH [20]. These studies did not differentiate the obesity effect, possibly due to the fact that they had low patient numbers. In Kavey's research, 62 WCH patients were evaluated for LVMI and 58% of them were obese. For LVH defined as $\text{LVMI} > 51 \text{ g}/\text{m}^{2.7}$, it was found to be 13.6%. [6]. LVH is not unique to HT, can also be induced by obesity. [21]. Therefore, groups in the present research design were planned according to BMI.

Abnormal cardiac geometric changes, defined as concentric geometry, eccentric LVH, and concentric LVH within this present study, were found to be 22% in the obese WCH group and 11.1% in the normal weight WCH group, whereas in HT groups, the frequency of geometric changes was found to be 36.2% for obese and 26.3% for normal weight. A LVH (concentric or eccentric) rate of 12% in the WCH/O group and 8.8% in the WCH/Non-O group was found. These findings clearly demonstrate the impact of obesity on those with WCH. It is known that while pressure overload such as HT, predisposes increased LVMI or RWT, volume overload predisposes eLVH. In the present study, CG and cLVH rates were 20.2%, 7.3%, and 6% in the obese groups (HT/O, WCH/O, and C/O, respectively). However they were 15.8%, 6.7%, and 0% in the non-obese groups (HT/Non-O, WCH/Non-O, and C/Non-O, respectively). A linear relationship was also found from the HT group to the WCH and control groups. Similar to Lande's results, this finding supports the idea that WCH is an intermediate pre-HT condition for both obese and non-

obese subjects but is more pronounced in the obese. The current study supports the need to screen patients with WCH for cardiac end-organ damage. Accurately identifying WCH children at risk is important for indications of antihypertensive pharmacological therapy.

In a Swedish study, Westerståhl et al. re-examined 30 WCH schoolchildren after a median follow-up of 9.3 years [22]. Seven had sustained HT. They used LVH defined as LVMI > 115 g/m² for men and > 95 g/m² for women. They found BMI and LVMI were higher in HT patients than the normotensives, but there were no significant differences (23.5 vs 29.7 kg/m² p = 0.057 and 83.5 vs 93.1 g/m² p = 0.26, respectively). In addition, they found LVH in 2 (6%) of 23 patients who still had WCH after follow-up, and in 2 (28.5%) of 7 patients with HT. Their results support the importance of following children with WCH for the early diagnosis of hypertension.

Results such as those indicated above also demonstrate that there is a need to better understand why some WCH patients have LVH and there is need to identify their risk factors. Recently, Miyashita et al. re-evaluated 89 patients with WCH and a median age of 13.9 years after a 14-month median interval with ABPM [23]. Fifty-five percent of patients were obese and LVH was out of the scope of this study. On their follow-up ABPM, 23% had progressed to ambulatory hypertension and 8% to ambulatory prehypertension. They found no differences for BMI z score and obesity between hypertension, prehypertension, and normotensives. They indicated that patients with WCH aged between 12 and 17 years who had a daytime SBP index ≥ 0.9 were associated with progressing to HT. In the present study, ABPM averages of those with WCH were normal, however their non-dipping statuses were similar to those with a diagnosis of HT. Ultradian rhythms are biological rhythms that have more than one cycle per day such as heart rate, respiratory rate, and bowel activity. These rhythms are usually caused by external and behavioral stimuli lead to sympathetic activity. It has been hypothesized that the mechanism of LVH in children with WCH is associated with frequent increases in BP in response to stress, which may result in increased LVM [24]. In research by Litwin, BP and heart rate rhythm analyses were performed in 129 hypertensive children, 54 children with WCH, and 146 healthy subjects. BMI in the WCH group was higher than the healthy group. Their main finding was that children with HT and WCH have changed rhythmicity patterns of circadian BP and HR than the healthy children [25]. This supports the fact that the circadian rhythm of cardiovascular functions, which are functions of the central sympathetic centers, is disturbed during the night in WCH patients. Nocturnal non-dipping is independently related to end organ damage and cardiovascular risk [26–29]. In Litwin's study, 26% of WCH were non-dippers; however, the present research found 65.9% of WCH/O and 55.6% of WCH/non-O were systolic non-dippers. Nocturnal non-dipper patterns were correlated with the central sympatho-adrenergic drive as indicated by catecholamine excretion. These findings support the idea that some patients with WCH have disturbed nocturnal circadian rhythms and that this may be a risk factor for LVH. Because of the low number of WCH patients, the parameters between patients with and without LVH [WCH/non-O (5/40) and WCH/O (9/32)] could not be compared.

In the present study, both HT and WCH patients had higher percentages of family cardiovascular history than control groups. In general, a person with two or more first-degree relatives with high BP has a 3.8-fold increased risk for increased BP before the age of 55. As well, it has been documented that the genetic contribution to essential HT is 25–60% [30]. It is not known on what basis patients with a similar family history will develop the HT or WCH phenotype. Epigenetic changes may also have an important role in the heritability of the polygenic nature of HT.

Limitations

The main limitation of this research is that the low number of WCH patients with LVH prevented the comparison of parameters between WCH patients with and without LVH. Having greater patient numbers could have also resulted in this research providing more precise answers. Another limitation is the use of retrospective analysis.

Conclusion

ABPM is an important tool for differentiating HT and WCH. It has been known that ABPM values more closely correlate with LVMI [26]. Besides this, WCH can be another risk factor for CVD. This research determined that 19.5% of WCH/O and 6.7% of WCH/non-O patients had LVH. This is the first study to differentiate more clearly the effect of obesity on WCH, with the results indicating that target-organ damage can manifest in WCH patients, especially those who are obese, or who have non-dipping BP patterns or family history.

Declarations

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Ethics approval, Consent, Data, Material and/or Code are available

Authors' contribution statements.

The study has not been presented before.

Disclosure of conflict of interest : None

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Statement and Declarations

We disclosed financial or non-financial interests that are directly or indirectly related to the work submitted for publication.

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Tables

Table 1 Epidemiologic data of obese and normal weight group

Parameter	Hypertensive obese n= 94	WCH obese n= 41	Normotensive obese n=33	p	Parameter	Hypertensive normal weight n=57	WCH normal weight n=45	Normotensive normal weight n=89	p
Age (year)	12.19 (±3.1)	13.34 (±3.06)	12.16 (±2.95)	0.06	Age (year)	12.56 (±3.82)	13.36 (±3.35)	13.04 (±2.06)	0.332
Gender (M, no, %)	64 (68.1%)	30 (73.2%)	23 (69%)	0.55	Gender (M, no, %)	36 (63.2%)	31 (68.9%)	60 (67.4%)	0.92
Weight (kg)	68.93 (±23.87)	75.5 (±24.94)	67.38 (±20.94)	0.058	Weight (kg)	50.57 (±20.34)	53.82 (±17.66)	49.82 (±14.01)	0.056
Weight z score	2.21 (1.57-2.85)	2.24 (1.57-2.91)	2.01 (1.23-2.6)	0.063	Weight z score	0.52 [(-0.48)-1.03]	0.33 [(-0.33)-0.83]	-0.16 [(-0.36)-0.43]	0.071
Height (cm)	156 (±18.73)	162 (±19.57)	158.4 (±15.57)	0.206	Height (cm)	154 (±21.33)	158 (±21.27)	158 (±12.72)	0.290
Height sds	0.97 [(-0.01)-1.57]	0.86 (0.21-1.56)	1.01 (0.01-1.67)	0.056	Height sds	0.57 [(-0.27)-1.41]	0.35 [(-0.38)-1.11]	0.48 (0.22-0.73)	0.742
BMI (kg/m ²)	27.1 (±5.02)	28.13 (±5.64)	26.92 (±3.64)	0.061	BMI (kg/m ²)	20.05 (±3.73)	20.6 (±3.04)	19.73 (±2.6)	0.059
BMI sds	2.04 (±0.71)	1.97 (±0.64)	1.95 (±0.66)	0.065	BMI sds	0.10 (±0.86)	0.13 (±0.91)	0.08 (±0.98)	0.06
Number of Symptom	3.6 (±2.81)	3.07 (±2.77)			Number of Symptom	3.01 (±2.2)	2.9 (±2.9)		
Symptom duration (month)	6 (2-12)	3 (1-12)			Symptom duration (month)	4(1-12)	3 (1-9)		
Family history of CVD	79.8% (75)	85% (34)	42.4%(14)	0.000	Family history of CVD	78.6% (44)	57.8% (25)	33.7%(30)	0.000
<p>BMI: body mass index, CVD: Cardiovascular disease, M:male, WCH: White coat hypertension</p> <p>The comparison of the three groups was performed by one-way ANOVA for normal distribution data and by Kruskal-Wallis for non-normal distribution data (p<0.05).</p> <p>a Comparison of Family history of CVD by Pearson Chi-Square</p>									

Table 2a Office Blood pressure data of obese and normal weight group

Parameter	Hypertensive obese n= 94	WCH obese n= 41	Normotensive obese n=33	p	p*	p**	p***
Office SBP (mmHg)	130 (120-140)	128 (116-140)	106 (104-107)	0.000	0.960	0.000	0.000
Office DBP (mmHg)	71.6 (±11.9)	69.6 (±10.9)	68 (±6.7)	0.646	0.578	0.354	0.935
Parameter	Hypertensive normal weight n=57	WCH normal weight n=45	Normotensive normal weight n=89	p	p*	p**	p***
Office SBP (mmHg)	126 (114-132)	127 (112-130)	110 (100-122)	0.000	0.480	0.000	0.000
Office DBP (mmHg)	70.5 (±10.6)	70.3 (±12.3)	66 (±5.6)	0.003	0.989	0.007	0.023
<p>SBP: Systolic blood pressure, DBP: Diastolic blood pressure, ABPM: Ambulatory blood pressure monitoring, HT: Hypertension, WCH: White coat hypertension</p> <p>The comparison of the office blood pressure of the three groups was performed by one-way ANOVA for normal distribution data and by Kruskal-Wallis for non-normal distribution data (p<0.05). The pairwise comparison were performed by Post-hoc Benferroni for normal distribution parameter test (p*, p**, and p*** <0.01). The comparison of ABPM was performed by T-Test for normal distribution and by Mann-Whitney Test for non-normal distribution</p> <p>The comparison between hypertensive obese/normal weight and WCH obese/normal weight was shown with p*,</p> <p>The comparison between hypertensive obese/ normal weight and Normotensive obese/ normal weight was shown with p**,</p> <p>The comparison between WCH obese/ normal weight and normotensive obese/ normal weight was shown with p***.</p>							

Table 2b ABPM data of obese and normal weight group

Parameter	Hypertensive obese	WCH obese	p	Hypertensive normal weight	WCH normal weight	p
ABPM results	n= 94	n= 41		n=57	n=45	
Mean SBP (mmHg)	134 (128.2-140.7)	119 (110-124)	0.000	132 (124-137)	117 (110-123)	0.000
Mean SBP z score	2.41 (1.88-2.41)	0.5 [(-0,09)-0.95]	0.000	2.05 (1.75-2.41)	0.28 [(-0.03)-0.92]	0.000
Systolic index	1.04 (1.02-1.09)	0.92 (0.87-0.95)	0.000	1.03 (1.01-1.08)	0.92 (0.82-0.96)	0.000
Mean DBP (mmHg)	77.21 (±8.34)	66.55 (±5.84)	0.000	76.5 (±8.19)	67.6 (±7.88)	0.000
Diastolic index	1 (0.94-1.05)	0.85 (0.78-0.9)	0.000	1.01 (0.94-1.04)	0.88 (0.78-0.96)	0.000
Daytime SBP (mmHg)	136 (128-144)	121 (112-128)	0.000	135 (126-141)	122 (116-129)	0.000
Daytime DBP (mmHg)	80 (73-85)	69 (64-76)	0.000	79 (74-83.7)	71 (65-77)	0.000
Nighttime SBP (mmHg)	129 (124-135)	111.5 (102.7-120)	0.000	127 (118-132)	110 (106-122)	0.000
Nighttime DBP (mmHg)	69 (64-76)	61.5 (56.7-68.5)	0.000	71 (65-79)	64 (59-68)	0.003
Systolic load daytime (%)	55.2 (±28.46)	10.54 (±10.76)	0.000	51.05 (±28.04)	12,5 (±11.8)	0.000
Diastolic load daytime (%)	31 (±25)	10.74 (±13.4)	0.000	30.9 (±24.98)	10.95 (±11.02)	0.034
Systolic load nighttime (%)	66.11 (±27.9)	12.35 (±7.04)	0.000	54.57 (±29.69)	11.52 (±8.64)	0.002
Diastolic load nighttime (%)	35.5 (16-60)	10 (3-20)	0.000	30 (10-70.7)	16 (2-22)	0.037
Dipping Systol (%)	%31 (29)	%34.1 (14)	0.541	36.8 % (21)	44.4 % (20)	0.233
Dipping Diastol (%)	52.1% (49)	43.9% (18)	0.674	57,7% (30)	46.6% (21)	0.707
Pulse (/min)	84 (77-88)	80 (74.4-86.2)	0.676	84.5 (76.2-91.7)	81 (76-86)	0.346
HT type						
Isolated Systolic HT	66% (62)			64.9% (37)		
Systolic + Diastolic HT	34% (32)			35.1% (20)		
SBP: Systolic blood pressure, DBP: Diastolic blood pressure, ABPM: Ambulatory blood pressure monitoring, HT: Hypertension, WCH: White coat hypertension						
The comparison of ABPM was performed by T-Test for normal distribution and by Mann-Whitney Test for non-normal distribution(p<0.05).						

Table 3 Echocardiography data of obese and normal weight group

Parameter	Hypertensive obese n= 94	WCH obese n= 41	Normotensive obese n=33	p	Hypertensive normal weight n=57	WCH normal weight n=45	Normotensive normal weight n=89	p
EF (%)	67.23 (±8.9)	69.14 (±3.8)	67.41 (±4)	0.50	65.61 (±4.48)	68.61 (±3.3)	67.27 (±0.63)	0.243
FS (%)	37.26 (±3.11)	39.17 (±5.2)	37.5 (±4.5)	0.32	38.54 (±7.02)	38.16 (±4.42)	38 (±1.59)	0.881
LVM (g)	120.53 (±50.9)	133 (±45.35)	106.32 (±27.3)	0.107	96.65 (48-148)	75.85 (42-188)	86.95 (47-175)	0.976
LVM z score	1.09 (±0.87)	1.05 (±0.83)	0.75 (±0.48)	0.238	0.39 (±1.08)	0.18 (±1.04)	-0.21 (±0.84)	0.002
LVMI g/m ²	70.19 (±19.09)	71.01 (±16.16)	64.65 (±13.06)	0.10	70.93 (±19.6)	68.77 (±20.16)	63.64 (±12.65)	0.159
LVMI g/m ^{2.7}	35.38 (±8.15)	35.03 (±7.37)	31.83 (±5.76)	0.07	30.8 (±7.09)	29.33 (±5.76)	26.43 (±5.59)	0.000
LVMI g/m ^{2.7} z score	0.99 (±0.99)	0.81 (±0.85)	0.61 (±0.86)	0.134	0.43 (±0.97)	0.08 (±0.88)	-0.28 (±0.81)	0.000
RWT	0.37 (±0.1)	0.34 (±0.04)	0.31(±0.03)	0.001	0.36 (±0.08)	0.31 (±0.66)	0.30 (±0.02)	0.059
Normal Geometry (NG)	63.8% (60)	78% (32)	85% (28)	0.04	73.7% (42)	88.9% (40)	99% (88)	0.000
Concentric geometry (CG)	5.3% (5)	2.4% (1)	3% (1)	Linear-by-linear association 0.027	10.5% (6)	4.4% (2)		Linear-by-linear association 0.000
Eccentric LVH (eLVH)	16% (15)	14.6% (6)	9% (3)		10.5% (6)	4.4% (2)	1% (1)	
Concentric LVH (cLVH)	14.9% (14)	4.9% (2)	3% (1)		5.3% (3)	2.3% (1)		
<p>EF:Ejection Fraction, FS:Fractional Shortening, LVM: Left Ventricular Mass, LVMI: Left Ventricular Mass Index, RWT: Relative wall thickness, LVH: Left Ventricular Hypertrophy, WCH: White coat hypertension</p> <p>The comparison of the three groups was performed by one-way ANOVA for normal distribution data and by Kruskal-Wallis for non-normal distribution data (p<0.05). The pairwise comparison were performed by Post-hoc Benferroni for normal distribution parameter test (p<0.01). The pairwise comparison were performed by Independent samples Kruskal Wallis test for non-normal distribution.</p> <p>^a Comparison of NG and other (CG, eLVH, cLVH) by Pearson Chi-Square</p>								

Table 4 Biochemistry data of obese and normal weight group

Parameter	Hypertensive obese n= 94	WCH obese n= 41	p	Hypertensive normal weight n=57	WCH normal weight n=45	p
Fasting glucose (mg/dl)	89.21 (±7.33)	84.97(±8.07)	0.09	89.13 (±7.56)	78.87 (±5.7)	0.543
Total cholesterol (mg/dl)	171.86(±31.65)	180.02(±25.96)	0.544	157 (±28.63)	162 (±40)	0.09
LDL (mg/dl)	108 (±24.1)	110,9(±29.5)		85 (±24.47)	91 (±30.4)	
HDL (mg/dl)	52.23 (±14.57)	51.33 (±11.48)		60.41 (±28.3)	54.76(±12.2)	
Creatinine (mg/dl)	0.65 (±0.12)	0.68 (±0.15)	0.432	0.65 (±0.14)	0.64 (±0.13)	0.06
BUN (mg/dl)	12.32 (±3.41)	11.54 (±4.14)	0.667	12.43 (±2.9)	11.53 (±3.54)	0.876
Uric acid (mg/dl)	5.38 (±1.42)	5.97 (±2.23)	0.06	4.97 (±1.28)	4.21 (±1.06)	0.654
LDL: Low density cholesterol, HDL: High density cholesterol, BUN: Blood urea nitrogen, WCH: White coat hypertension						
The comparison of the three groups was performed by one-way ANOVA for normal distribution data (p<0.05).						

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