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ASSOCIATION BETWEEN EXCESS BODY WEIGHT, LEFT VENTRICULAR REMODELING, AND CIRCADIAN BLOOD PRESSURE PROFILE IN MIDDLE-AGED MEN WITH ARTERIAL HYPERTENSION

Botirkhon Salikhov Ravshanovich
Independent Researcher, Military Medical Institute
Tashkent, Uzbekistan
botirhoncardio@mail.ru
orcid.org/0009-0008-4760-0448

Abstract

Background. Excess body weight is one of the most important modifiable risk factors for the development of arterial hypertension and cardiovascular complications. Increasing body mass index (BMI) is associated with adverse myocardial remodeling, disruption of the circadian blood pressure (BP) pattern, and progressive target organ damage. However, the relationship between excess body weight, left ventricular (LV) geometry, and the 24-hour BP profile in men has not been sufficiently investigated.

Objective. To evaluate the association between excess body weight, left ventricular remodeling, and abnormalities of the circadian blood pressure profile in middle-aged men with arterial hypertension.

Materials and Methods. This cross-sectional study included 73 male aged 20-45 years with grade I-II arterial hypertension. All participants underwent anthropometric assessment, transthoracic echocardiography with evaluation of LV structural and geometric parameters, and 24-hour ambulatory blood pressure

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monitoring (ABPM). According to BMI, participants were classified into three groups: normal weight, overweight, and class I obesity.

Results. The prevalence of concentric LV remodeling and abnormal circadian BP patterns increased progressively with increasing BMI. Patients with class I obesity demonstrated a significantly higher prevalence of the non-dipper BP profile and a lower prevalence of the physiological dipper pattern. Echocardiographic evaluation also revealed a stepwise increase in interventricular septal thickness, left ventricular posterior wall thickness, left ventricular mass, and left ventricular mass index with increasing body weight.

Conclusion. Excess body weight in men with arterial hypertension is associated with more pronounced left ventricular remodeling and disturbances of the circadian blood pressure profile. These findings emphasize the importance of a comprehensive therapeutic strategy that includes not only achieving target blood pressure levels but also effective weight management to slow the progression of target organ damage and reduce long-term cardiovascular risk.

Keywords: arterial hypertension; overweight; obesity; left ventricular remodeling; left ventricular geometry; ambulatory blood pressure monitoring; circadian blood pressure profile;

Introduction

Excess body weight and obesity are among the most important modifiable risk factors for the development of arterial hypertension and cardiovascular disease. According to recent epidemiological evidence, the global prevalence of obesity continues to increase steadily, making it one of the leading public health challenges worldwide. An elevated body mass index (BMI) has been consistently associated with a progressively higher risk of arterial hypertension, target organ damage, heart failure, stroke, and premature cardiovascular mortality. The pathogenesis of obesity-related hypertension is multifactorial and involves activation of the renin-angiotensin-aldosterone system and the sympathetic

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nervous system, chronic low-grade inflammation, endothelial dysfunction, insulin resistance, and impaired sodium homeostasis. Collectively, these mechanisms contribute to increased vascular resistance, greater hemodynamic load on the myocardium, and subsequent cardiovascular remodeling [7,10].

Current clinical guidelines recognize weight reduction as one of the key components of comprehensive management of arterial hypertension in patients with overweight and obesity. Even a modest weight loss of 5-10% has been shown to produce clinically meaningful reductions in blood pressure, improve metabolic parameters, attenuate neurohumoral activation, and reduce the risk of cardiovascular complications. In addition to pharmacological therapy, lifestyle modification-including a healthy diet, sodium restriction, regular aerobic physical activity, and long-term weight management-constitutes an essential component of treatment. The implementation of this comprehensive approach may slow the progression of myocardial remodeling, enhance the effectiveness of antihypertensive therapy, and improve long-term cardiovascular outcomes [7,3].

Despite substantial advances in understanding the relationship between obesity and arterial hypertension, the characteristics of left ventricular structural remodeling and abnormalities of the circadian blood pressure profile in men remain insufficiently investigated. Despite substantial advances in understanding the relationship between obesity and arterial hypertension, the characteristics of left ventricular structural remodeling and abnormalities of the circadian blood pressure profile in men remain insufficiently investigated. Early identification of myocardial remodeling and abnormalities of the circadian blood pressure profile is essential for accurate cardiovascular risk stratification, prevention of target organ damage, and optimization of therapeutic and preventive strategies [4,5].

Therefore, the aim of the present study was to evaluate the association between excess body weight, left ventricular remodeling, and abnormalities of the circadian blood pressure profile in middle-aged men with arterial hypertension.

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Materials and Methods

A cross-sectional observational comparative study was conducted including 73 men aged 20-45 years with grade I-II arterial hypertension and stage I hypertensive disease. According to body mass index (BMI), calculated using the World Health Organization (WHO) criteria [9], participants were stratified into three groups: normal weight (n = 10), overweight (n = 37), and class I obesity (n = 26). The mean age of the participants was 34.7 ± 6.1, 36.7 ± 4.4, and 37.3 ± 5.3 years, respectively, with no statistically significant differences in age between the groups ($p > 0.05$).

The exclusion criteria were female sex, secondary hypertension, type 1 or type 2 diabetes mellitus, coronary artery disease, chronic heart failure, congenital or acquired valvular heart disease, cardiac arrhythmias interfering with accurate echocardiographic assessment, chronic kidney disease, decompensated liver or respiratory diseases, malignant neoplasms, and any other conditions that could independently influence myocardial structure and function or ambulatory blood pressure monitoring parameters.

Echocardiographic Assessment

Transthoracic echocardiography was performed using a Samsung RS85 ultrasound system (Samsung Medison, Republic of Korea) in accordance with the recommendations of the American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI). Standard two-dimensional, M-mode, and Doppler imaging were used to evaluate left ventricular dimensions, wall thickness, left ventricular mass, and ventricular geometry.

Left ventricular mass (LVM) was calculated using the Devereux formula recommended by the ASE/EACVI guidelines. The left ventricular mass index (LVMI) was calculated by indexing LVM to body surface area. Left ventricular geometry was classified based on the combined assessment of LVMI and relative wall thickness (RWT), calculated as follows:

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RWT = (2 × LVPWT) / LVEDD

where LVPWT is the left ventricular posterior wall thickness and LVEDD is the left ventricular end-diastolic diameter.

According to the ASE/EACVI recommendations, four patterns of left ventricular geometry were identified: normal geometry (normal LVMI and RWT ≤ 0.42), concentric remodeling (normal LVMI and RWT > 0.42), eccentric hypertrophy (increased LVMI and RWT ≤ 0.42), and concentric hypertrophy (increased LVMI and RWT > 0.42). For men, an LVMI >115 g/m² was considered indicative of left ventricular hypertrophy, whereas an RWT value >0.42 defined increased relative wall thickness [2].

Ambulatory Blood Pressure Monitoring

Twenty-four-hour ambulatory blood pressure monitoring (ABPM) was performed using the Schiller BP-102 system (Schiller AG, Switzerland) according to current international recommendations. Mean 24-hour, daytime, and nighttime systolic and diastolic blood pressure values were analyzed together with the nocturnal blood pressure decline (dipping index).

Based on the nocturnal BP decline, participants were classified into four circadian blood pressure patterns: dippers (physiological nocturnal BP reduction of 10-20%), non-dippers (reduction <10%), over-dippers (reduction >20%), and reverse dippers (risers), characterized by the absence of a physiological nocturnal BP decline or an increase in nighttime blood pressure (dipping index <0%). The latter two patterns were considered the most unfavorable circadian blood pressure profiles because of their well-established association with target organ damage and increased cardiovascular risk [7].

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as the mean ± standard deviation (SD). The normality of data distribution was assessed using

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the Shapiro-Wilk test. Normally distributed variables were compared using one-way analysis of variance (ANOVA) followed by Tukey's post hoc multiple-comparison test, whereas non-normally distributed variables were analyzed using the Kruskal-Wallis test. Categorical variables were compared using the Pearson χ^2 test or Fisher's exact test, as appropriate. A two-sided p-value <0.05 was considered statistically significant.

Results

Left ventricular remodeling represents one of the earliest manifestations of target organ damage in arterial hypertension and reflects the degree of chronic hemodynamic overload imposed on the myocardium. Analysis of left ventricular geometric patterns demonstrated statistically significant differences among the study groups according to body mass index (Table 1).

Normal left ventricular geometry was observed most frequently in patients with normal body weight (90.0%) and progressively decreased with increasing body mass index, accounting for 73.0% of patients with overweight and 65.4% of those with class I obesity ($p = 0.041$). In contrast, the prevalence of concentric remodeling increased gradually from 10.0% in the normal-weight group to 24.3% and 34.6% in the overweight and class I obesity groups, respectively ($p = 0.038$). Concentric left ventricular hypertrophy was not observed in the study cohort, whereas eccentric hypertrophy was identified in only one patient (2.7%) with overweight.

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Table 1. Left Ventricular Geometric Patterns in Middle-Aged Men with Arterial Hypertension According to Body Mass Index

Variable	Normal Weight (n = 10)	Overweight (n = 37)	Class I Obesity (n = 26)	p value
Normal LV geometry	9 (90.0%)	27 (73.0%)	17 (65.4%)	0.041
Concentric remodeling	1 (10.0%)	9 (24.3%)	9 (34.6%)	0.038
Concentric hypertrophy	0	0	0	-
Eccentric hypertrophy	0	1 (2.7%)	0	0.999

Data are presented as n (%). Intergroup comparisons were performed using Pearson's χ^2 test. Fisher's exact test was applied when expected cell counts were <5. **LV**, left ventricle.

Overall, these findings indicate that increasing BMI is associated with a progressive transition from normal left ventricular geometry toward concentric remodeling, whereas concentric hypertrophy was not observed in this cohort.

Analysis of the circadian blood pressure profile demonstrated statistically significant differences among the study groups (Table 2). With increasing body mass index, the proportion of patients exhibiting the physiological dipper pattern progressively decreased. The dipper profile was observed in 60.0% of participants with normal body weight, compared with 51.4% of those with overweight and only 19.2% of patients with class I obesity (p = 0.012).

Conversely, the prevalence of the pathological non-dipper pattern increased with increasing BMI, being identified in 40.0% of participants with normal body

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weight, 40.5% of those with overweight, and 65.4% of patients with class I obesity (p = 0.031). The reverse-dipper (riser) pattern was observed exclusively in participants with excess body weight and occurred most frequently in the class I obesity group (15.4%), whereas it was not detected among participants with normal body weight. The over-dipper pattern was identified in only one patient (2.7%) with overweight and had no substantial impact on the overall distribution of circadian blood pressure profiles.

Table 2. Circadian Blood Pressure Patterns in Middle-Aged Men with Arterial Hypertension According to Body Mass Index

Circadian BP Profile	Normal Weight (n = 10)	Overweight (n = 37)	Class I Obesity (n = 26)	p value
Dipper	6 (60.0%)	19 (51.4%)	5 (19.2%)	0.012
Non-dipper	4 (40.0%)	15 (40.5%)	17 (65.4%)	0.031
Over-dipper	0	1 (2.7%)	0	0.999
Reverse-dipper (Riser)	0	2 (5.4%)	4 (15.4%)	0.041

Data are presented as n (%). Intergroup comparisons were performed using Pearson's χ^2 test. Fisher's exact test was applied when expected cell counts were <5. **BP**, blood pressure.

Taken together, increasing BMI was associated with a progressive shift from the physiological dipper pattern toward adverse circadian blood pressure profiles, characterized predominantly by an increased prevalence of the non-dipper and reverse-dipper patterns.

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Echocardiographic evaluation demonstrated progressive changes in the structural and geometric characteristics of the left ventricle with increasing body mass index (Table 3). Parameters reflecting left ventricular chamber size, including left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), stroke volume, and left ventricular ejection fraction (LVEF), showed no significant differences among the study groups (all $p > 0.05$), indicating preserved left ventricular systolic function regardless of body weight.

In contrast, echocardiographic markers of myocardial remodeling increased progressively across BMI categories. Interventricular septal thickness, left ventricular posterior wall thickness, left ventricular mass (LVM), and left ventricular mass index (LVMI) all increased progressively across the BMI categories. The most pronounced structural changes were observed in patients with class I obesity, in whom the mean LVM reached 173.9 ± 8.9 g and the mean LVMI 90.8 ± 7.1 g/m², both of which were significantly higher than the corresponding values observed in participants with normal body weight ($p < 0.001$).

A similar trend was observed for relative wall thickness (RWT), which increased progressively from 0.388 ± 0.030 in participants with normal body weight to 0.417 ± 0.030 in those with class I obesity ($p = 0.006$). Taken together, these findings indicate that increasing body mass index is associated with progressive structural remodeling of the left ventricle despite preserved systolic myocardial function.

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Table 3. Echocardiographic Characteristics of the Left Ventricle in Middle-Aged Men with Arterial Hypertension According to Body Mass Index

Variable	Normal Weight (n = 10)	Overweight (n = 37)	Class I Obesity (n = 26)	p value
LVEDD, cm	4.56 ± 0.33	4.70 ± 0.29	4.80 ± 0.20	0.084
LVESD, cm	3.04 ± 0.40	2.91 ± 0.28	3.08 ± 0.38	0.211
LVEDV, mL	104.5 ± 10.8	107.1 ± 9.4	108.6 ± 9.1	0.328
LVESV, mL	35.4 ± 7.8	33.7 ± 7.4	37.9 ± 7.1	0.192
Stroke volume, mL	72.9 ± 9.1	73.0 ± 8.9	72.4 ± 7.8	0.917
Left ventricular ejection fraction, %	67.8 ± 5.5	68.0 ± 4.7	65.3 ± 5.6	0.146
Interventricular septal thickness, cm	0.93 ± 0.07	0.99 ± 0.06	1.02 ± 0.09	0.009
Left ventricular posterior wall thickness, cm	0.91 ± 0.08	0.95 ± 0.05	1.01 ± 0.06	0.004
Left ventricular mass, g	160.1 ± 9.6	167.7 ± 8.8	173.9 ± 8.9	<0.001
Left ventricular mass index, g/m²	79.8 ± 5.6	85.6 ± 7.4	90.8 ± 7.1	<0.001
Relative wall thickness	0.388 ± 0.030	0.406 ± 0.020	0.417 ± 0.030	0.006

Data are presented as mean ± standard deviation (SD). Intergroup differences were assessed using one-way analysis of variance (ANOVA). P values represent the statistical significance of differences among the study groups.

Abbreviations: LVEDD, left ventricular end-diastolic diameter; **LVESD**, left ventricular end-systolic diameter; **LVEDV**, left ventricular end-diastolic volume; **LVESV**, left ventricular end-systolic volume; **LVEF**, left ventricular ejection fraction; **IVS**, interventricular septum; **LVPWT**, left ventricular posterior wall thickness; **LVM**, left ventricular mass; **LVMI**, left ventricular mass index; **RWT**, relative wall thickness.

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Overall, increasing BMI was associated with progressive left ventricular wall thickening and increases in left ventricular mass and relative wall thickness, while left ventricular chamber dimensions and systolic function remained unchanged across the study groups.

Discussion

The present study demonstrates that increasing body mass index is associated with progressive alterations in left ventricular structure and abnormalities of the circadian blood pressure profile in middle-aged men with arterial hypertension. As BMI increased, the prevalence of normal left ventricular geometry and the physiological dipper pattern progressively declined, whereas the frequencies of concentric remodeling and adverse circadian blood pressure profiles, particularly the non-dipper and reverse-dipper patterns, increased. These findings reinforce the concept that excess body weight contributes to early hypertension-mediated target organ damage even during the initial stages of arterial hypertension [7].

The observed increase in the prevalence of concentric left ventricular remodeling among participants with excess body weight has a well-established pathophysiological basis. Excess adipose tissue promotes chronic activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system, together with persistent low-grade systemic inflammation, endothelial dysfunction, and insulin resistance. In addition, obesity is associated with increased circulating blood volume and elevated cardiac afterload, leading to progressive left ventricular wall thickening, increased myocardial mass, and the development of concentric remodeling. These findings are consistent with previous studies demonstrating that obesity is an independent determinant of cardiac structural remodeling, irrespective of blood pressure levels [6,8].

An additional important finding of the present study is the progressive deterioration of the circadian blood pressure profile with increasing BMI. The proportion of participants exhibiting the physiological dipper pattern decreased

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progressively, whereas the prevalence of the non-dipper and reverse-dipper patterns increased across BMI categories. Previous studies have demonstrated that impaired nocturnal blood pressure decline is associated with more severe target organ damage, accelerated left ventricular hypertrophy and remodeling, vascular dysfunction, and an increased risk of cardiovascular events, independent of office blood pressure values. Therefore, abnormalities of the circadian blood pressure profile may serve as an additional independent marker of adverse cardiovascular prognosis in patients with hypertension and obesity [7,8].

Several limitations of this study should be acknowledged. First, this was a single-center study with a relatively small sample size, which may limit the generalizability of the findings. Second, only middle-aged men were included; therefore, the results cannot be directly extrapolated to women or the general population. Furthermore, the cross-sectional design precludes establishing causal relationships between excess body weight, left ventricular remodeling, and abnormalities of the circadian blood pressure profile. Nevertheless, the relatively homogeneous study population minimized the influence of potential confounding factors and enabled a more accurate assessment of the association between excess body weight and early cardiovascular remodeling in patients with arterial hypertension. Future prospective, multicenter studies involving larger and more diverse populations are warranted to validate these findings and further elucidate the causal relationship between excess body weight, left ventricular remodeling, and abnormalities of the circadian blood pressure profile.

Conclusions

1. In middle-aged men with arterial hypertension, increasing body mass index is associated with progressive left ventricular remodeling, characterized by a reduced prevalence of normal left ventricular geometry and an increased prevalence of concentric remodeling.

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2. Increasing body mass index is accompanied by progressive deterioration of the circadian blood pressure profile, manifested by a decline in the prevalence of the physiological dipper pattern and an increased frequency of the pathological non-dipper and reverse-dipper patterns, both of which are associated with elevated cardiovascular risk.
3. Echocardiographic assessment demonstrated progressive increases in interventricular septal thickness, left ventricular posterior wall thickness, left ventricular mass, and left ventricular mass index while left ventricular systolic function remained preserved, indicating the development of early structural myocardial remodeling associated with increasing body mass index.
4. These findings underscore the importance of a comprehensive management strategy for patients with arterial hypertension and excess body weight that includes not only achievement of target blood pressure levels but also active weight management to prevent the progression of myocardial remodeling and reduce the risk of cardiovascular complications.

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