Acta Scientific Gastrointestinal Disorders (ISSN: 2582-1091)

Volume 4 Issue 9 September 2021

Research Article

Peculiarities of Myocardial Remodeling in Young Obsessive Women with Arterial Hypertension

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Received: August 11, 2021

Published: August 23, 2021

Abstract

Background/Aims: Overweight and obesity play a significant role in the development of many noncommunicable diseases, leading to a reduction in life expectancy and adversely affecting its quality. A number of studies have proven a close relationship between high blood pressure and abdominal obesity. In adult patients, it has been proven that obesity leads to an increase in the mass of the left ventricular myocardium, impaired myocardial function, which are significant predictors of unfavorable outcomes of cardiovascular diseases. The aim of the study was to investigate left ventricular (LV) structural changes in overweight and obese young women with arterial hypertension (AH).

Design and Methods: In the study, according to inclusion criteria, 108 young women of 18 - 45 years old were included. Three groups were identified: the women with obesity without AH, the women with obesity and 1st degree of AH, the women with obesity and 2nd degree of AH. All patients underwent anthropometry (height and weight) with calculation of body mass index (BMI), echocardiography was performed according to a standard procedure with calculation of LV structural indexes. The LV geometry phenotypes were determined.

Results: Echocardiography LV dimensions (posterior wall thickness, interventricular septal thickness, diastolic and systolic LV diameter), as well as LVM and LVMI were higher in obese women compared to control group. LV hyper-trophy (LVH) developed in 61,9% obese patients without AH and in 76,4% and 78,1% patients with obesity and AH. Normal LV geometry was found in 23,8% patients with obesity, concentric remodeling - in 14,3% cases, 35,7% patients had concentric LVH, and 26,2% - eccentric LVH. In obese patients with 1st degree of AH the distribution of various types of LV remodeling was as follows: 17,6%/6%/52,9%/23,5%, respectively, and in obese patients with 2nd degree of AH - 12,5%/9,4%/53,1%/25%, respectively.

Conclusion: Since young age overweight and obesity are risk factors for LVH and the development of various LV geometry phenotypes. Markers of myocardial remodeling is an affordable way of early cardiovascular risk stratification in overweight and obese young women.

Keywords: Overweight; Obesity; Left Ventricular Hypertrophy; Left Ventricular Geometry

Introduction

Obesity is one of the largest noncommunicable epidemics. The main reason for the increase in the number of people with AO is considered to be a change in the lifestyle of a modern person towards the use of high-calorie food and lack of physical activity. The prevalence of AO, according to different authors, is 5 - 30% [1]. In

the United States, more than 30% of the adult population is obese, in the countries of the WHO European Region, half of the adult population and one in five children are overweight, and a third of them are already obese, while the number of such persons is growing rapidly [2]. According to the World Health Organization (WHO), in 2016, over 340 million children and adolescents aged 5 to 19 years were diagnosed with overweight or obesity [3].

Overweight and obesity play a significant role in the development of many noncommunicable diseases, leading to a reduction in life expectancy and adversely affecting its quality. Overweight causes more than one million deaths in Europe every year [4]. According to the results of the conducted studies, Russia is among the most disadvantaged countries in Europe in terms of the prevalence of obesity. Obesity is spreading especially rapidly among women. Among obese patients, a special category is formed by young women with overweight, formed after childbirth. Numerous studies show that it is the period of pregnancy that can be the moment of triggering the metabolic syndrome in women, one of the main components of which is abdominal obesity [5]. A number of multicenter studies have shown a close relationship between hypertension and abdominal obesity (AO) [1]. It has been shown that metabolic disorders underlie the increase in blood pressure, dyslipidemia, and type 2 diabetes mellitus. Overweight increases the risk of developing type 2 diabetes mellitus, atherosclerosis and coronary heart disease [6,7].

Myocardial hypertrophy is the most common variant of cardiac remodeling, as cardiomyocytes increase in size compensatory in order to maintain cardiac output. Re-modeling of the left ventricular (LV) myocardium includes a progressive increase in the mass of the LV myocardium (LVMM), an increase in the volume of heart cavities, as well as a change in its geometric characteristics [7-9]. AO is an independent pathological factor in myocardial remodeling [10]. There are two main reasons for remodeling in obesity - hemodynamic and metabolic [11]. Metabolic predisposition lies in the direct effect of regulatory metabolites, the deposition of adipocytes between myocardial fibers with a possible direct toxic effect. Excessive accumulation of adipose tissue occurs due to an increase in their size, which, taking into account the expression of insulin receptors on adipocytes, leads to a decrease in the density of these receptors and the formation of insulin resistance (IR), which, in turn, causes compensatory hyperinsulinism. IR reduces the level of insulin entering the cell, followed by energy deficiency in insulin dependent tissues and compensatory stimulation of the sympathoadrenal system with activation of the renin-angiotensin-aldosterone system, followed by an increase in arterial pressure (BP) and the formation of compensatory hypertrophy due to an increase in the load on the myocardium. It was found that LV remodeling, especially its concentric type, increases the likelihood of arrhythmias, in particular, high-grade ventricular extrasystoles. LV concentric hypertrophy is associated with the severity of cardiac arrhythmias (HRV). Eccentric remodeling, in turn, contributes to the development of relative coronary insufficiency [12,13]. Obesity, in turn, is a modifiable, independent risk factor for coronary artery disease, ventricular dysfunction, heart failure, and arrhythmias [14]. In adult patients, it has been proven that obesity is associated with an increase in left ventricular (LV) myocardial mass, systolic and diastolic dysfunction, which are significant predictors of unfavorable outcomes of cardiovascular diseases (CVD) [15].

Aim of the Study

The aim of this work is to investigate the effect of abdominal obesity and hypertension on myocardial remodeling in young women.

Materials and Methods

In accordance with the set goal, 108 women aged 18 to 45 years with signs of abdominal obesity were examined. The control group consisted of 24 young women with normal waist circumference without cardiovascular diseases.

Inclusion criteria were: age of women from 18 to 45 years, BMI more than 25 kg/m^2, signed informed consent.

The exclusion criteria were the presence of endocrine (except for exogenous constitutional obesity), symptomatic arterial hypertension (AH), organic cerebral diseases, acute inflammatory or exacerbation of chronic inflammatory diseases in the previous 2 weeks during a comprehensive examination. The study was terminated when the patients refused, the circumstances that hindered the patient's participation in the study were identified, and administrative problems.

All patients underwent anthropometric screening, including the determination of BMI. Body mass index (BMI) was calculated using

the Quetelet formula: body weight/height^2 (kg/m^2). At the same time, BMI 18.5 - 24.9 kg/m^2 was taken for normal body weight, BMI 25.0 - 29.9 kg/m^2 was regarded as excess body weight, and BMI ≥ 30 kg/m^2 was taken for obesity. The degree of abdominal obesity (AO) was assessed on the basis of measurements of waist circumference (WC) ≥ 80 cm (women), WC/hip circumference (HC) > 0.85 (women). To exclude polycystic ovary syndrome (PCOS), a gynecologist was consulted. All patients underwent a standard biochemical blood test: serum lipid spectrum indicators (total cholesterol (CS), TG, HDL cholesterol and low-density lipoprotein cholesterol (LDL)), fasting plasma glucose (FPG). The visceral obesity index (VAI - Visceral adiposity index) was calculated: (WC/39.68 + (1.88 x BMI)) x TG/1.03 x 1.31/HDL cholesterol. The VAI is < 2.52 for women under 30 and < 2.23 for women between the ages of 30 and 42 and indirectly indicates impaired adipose tissue distribution. The HOMA-IR index was determined by the formula: fasting serum insulin (μU/ml) x fasting blood glucose (mmol/l)/22.5; normal HOMA-IR is < 2.52 [16]. The structural and functional parameters of the heart were assessed using echo and Doppler echocardiography (EchoCG) using a GE Vivid 7 Dimension ultrasound diagnostic apparatus. The linear and volumetric indicators of the left ventricle (LV) were measured: end systolic dimension/volume (ESD/ESV), end diastolic dimension/volume (EDD/EDV). The calculation of the LV myocardial mass (LVMM) was carried out on the basis of linear measurements obtained in the M-mode under the control of the B-mode. The American Echographic Society (ASE) recommends for the assessment of LVM a formula based on linear measurements and an LV model in the form of an elongated ellipsoid of rotation, which takes into account the thickness of the posterior wall in diastole (TPWd) and the thickness of the interventricular septum in diastole (TIVSd) [17] LVMM = 0.8x (1.04x [(EDD + TPWd + TIVSd) x3- (KDD)x3) +0.6 (g). LVMM is considered elevated in women with a value greater than 67 - 162g.

LV myocardial mass index (LVMMI) was defined as the ratio of LVMM (g) to body surface area. LVMMI > 43 - 95 g/m^2 in women were considered as signs of LV hypertrophy (LVH). The relative wall thickness of the LV (RWT) was calculated using the formula: 2xTPWd/EDD. Values > 0.45 determined the severity of concentric LVH. The ejection fraction (EF) was determined by the Simpson method, EF = (EDV-ESD)/EDV (≥ 50%). To assess LV diastolic function, a study of transmitral blood flow was performed in a pulsed Doppler mode. The maximum rate of early diastolic filling of the LV was calculated (E, m/s); the rate of early relaxation of the myocardium (Eт, m/s), the ratio of the rates of the early diastolic flow (E/Eт), the maximum rate of LV filling in atrial systole (A, m/s); the ratio of the maximum velocities of the transmitral flow (E/A), the time of deceleration of the early filling flow (DT, ms); LV isovolumetric relaxation time (IVRT, ms). The LA volume (Vla) was measured using the Simpson method (for women, the norm is 22-52 ml). Diastolic myocardial stress (DMS) reflects the normal nature of preload at a value of ≤ 140. DMS = diastolic blood pressure (DBP) x EDD/4 x TPWd x (1 + TPWd/EDD) dyn/cm^2 [18].

Statistical processing was performed using Microsoft Excel 2010 and Statistica 7.0 software. All data were presented as arithmetic mean values and mean error (M ± m). To establish the significance of differences in the comparison groups, Student's t test and Pearson's fit test (χ^2) were used. The pairwise relationship between continuous and independent features was determined by using regression models with the definition of odds ratios (OR, odds ratio) for significant predictors with a 95% confidence interval (CI). Differences were considered significant at $p < 0.05$.

Results and Discussion

When analyzing cardiometabolic risk factors in all groups of patients, an increased TG level was recorded, which was confirmed by a significant increase in the atherogenic index (Table 1). The TG level directly correlated with the indirect indicator of AO (WC/HC) in patients of all groups (respectively: r = 0.62, r = 0.65 and 0.79; $p < 0.001$). When comparing groups in terms of carbohydrate profile indices in obese patients, a significant trend was revealed of an increase in the level of HPN and HOMA-IR ($p < 0.001$) with an increase in WC/HC (r = 0.78, r = 0.79 and r = 0, 80; $p < 0.001$). The values of the integral indicator “visceral adipose tissue function” - the VAI index, recorded within the normal range in the control group, increased 1.7 times in obese patients, and 2.8 - 3 times in patients with AH, indicating significant increase in cardiovascular risk ($\chi^2 = 86.934$; $p < 0.01$).

Analysis of the structural and functional parameters of the LV (Table 2) revealed that not only the presence of overweight (group I), but also AH - (II and III groups of patients) was accompanied by a significant increase in BMI, which was directly associated with metabolic risk factors (WC/HC and VAI index; r = 0.47; $p < 0.05$ and r = 0.83; $p < 0.001$). An increase in pre-load was characterized

Indicators	Control group (n = 24)	Obesity without hypertension group I (n = 42)	Obesity and AH grade 1 II group (n = 34)	Obesity and AH grade 2 Group III (n = 32)
cholesterol, mmol/l	3,74 ± 0,4	5,46 ± 0,35**	5,58 ± 0,26**	5,94 ± 0,32**
HDL cholesterol, mmol/l	1,26 ± 0,1	1,16 ± 0,09	1,11 ± 0,06	1,1 ± 0,05
LDL cholesterol, mmol/l	2,34 ± 0,1	3,26 ± 0,14**	3,42 ± 0,19**	3,86 ± 0,25**
Atherogenic index	1,46 ± 0,09	2,96 ± 0,12***	4,22 ± 0,18***	4,41 ± 0,17***
TG, mmol/l	1,16 ± 0,18	3,25 ± 0,14***	3,46 ± 0,22***	3,65 ± 0,22***
GPn, mmol/l	5,1 ± 0,1	5,64 ± 0,08**	6,48 ± 0,18***	6,55 ± 0,19***
Impaired glucose tolerance, mmol/l	5,4 ± 0,13	6,45 ± 0,22**	6,98 ± 0,21**	6,89 ± 0,25**
Insulin, μU/ml	6,9 ± 0,42	9,6 ± 0,49**	19,6 ± 0,66***	19,9 ± 0,82***
HOMA IR	1,54 ± 0,19	2,56 ± 0,15**	5,16 ± 0,34***	5,25 ± 0,31***
VAI	1,65 ± 0,18	2,84 ± 0,26*	4,63 ± 0,25***	4,92 ± 0,29***

Table 1: Indicators of lipid and carbohydrate profile, VAI index (M ± m) in young women with obesity and arterial hypertension.

Note: * - $p < 0.05$, ** - $p < 0.01$, *** - $p < 0.001$ - reliability of differences in groups I, II, III in relation to the control.

by an increase in MSD in patients with hypertension, the value of which exceeded the obtained data not only in patients with normal weight, but also with overweight ($p < 0.001$). The MSd value correlated with the VAI index ($r = 0.63$; $p < 0.01$). The increase in the pre-load increase coincided with the increase in EDV ($r = 0.57$; $p < 0.01$) and Vla ($r = 0.75$; $p < 0.01$). These factors formed various types of LV remodeling (Figure 1). Most obese patients (46.3%) had concentric LVH due to LV overload by both volume and pressure, a slightly smaller percentage (26.9%) was eccentric LVH. The analysis of LV diastolic function revealed the following features: the least pronounced violations were noted in group I of patients, where the type with impaired relaxation was predominant. With the addition of hypertension, the restrictive type of myocardial relaxation begins to prevail, characterized by the shortest slowing down of early LV filling, which apparently contributed to an increase in pressure in the LV and a significant increase in Vla (Table 3).

Younger obesity is associated with both immediate and long-term health risks. As shown in the Bogalusa study, childhood obesity is associated with LVM in adults, and LVH is strongly and independently correlated with CVD and mortality [19]. In our work, LVH was diagnosed in 57.1% of obese patients without an increase in blood pressure, which is comparable with the data of other studies. The pathogenesis of LVH in obesity is based on increased metabolic activity due to excess adipose tissue, which leads to an increase in cardiac output and total blood volume to meet metabolic needs. This compensatory process causes structural changes and cardiac dysfunction [20]. The study of the effect of high blood pressure and obesity on LVMM showed that these conditions are independent factors that increase the likelihood of LVH and have an additive effect on the development of LVH [21,22]. Some researchers associate obesity and abdominal circumference with impaired diastolic function, and hypertrophy and concentric remodeling of the myocardium - with hypertension [23]. The most common

Figure 1: Types of left ventricular remodeling in young obese women.

Indicators	Control group (n = 24)	Obesity without hypertension group I (n = 42)	Obesity and AH grade 1 II group (n = 34)	Obesity and AH grade 2 Group III (n = 32)
End diastolic dimension, mm	49,5 ± 0,7	52,5 ± 0,5***	54,9 ± 0,3***	58,5 ± 0,6***
End systolic dimension, mm	25,1 ± 0,6	29,2 ± 0,7***	34,8 ± 0,5***	38,6 ± 1,1***
End diastolic volume, ml	115,2 ± 7,4	136,8 ± 4,8***	161,3 ± 7,6***	171,5 ± 4,2***
End systolic volume, ml	31,2 ± 1,9	56,4 ± 4,6***	65,2 ± 2,2***	69,7 ± 3,1***
Interventricular septum, mm	7,6 ± 0,23	11,8 ± 0,17***	12,9 ± 0,21***	14,6 ± 0,25***
Thickness of the posterior wall in diastole (TPWd), mm	7,7 ± 0,29	13,6 ± 0,29***	12,6 ± 0,73***	16,2 ± 0,48***
Left atrium, mm	26,1 ± 1,2	29,8 ± 2,3	38,9 ± 1,4	43,5 ± 1,6*
Left ventricular myocardium mass, g	119,8 ± 7,9	202,4 ± 7,8***	215,8 ± 9,9***	246,3 ± 5,9***
Left ventricular myocardial mass index, g/m^2	68,4 ± 4,9	116,4 ± 8,1***	129,6 ± 9,5***	135,1 ± 6,6***
Relative wall thickness	0,31 ± 0,008	0,51 ± 0,04***	0,44 ± 0,09***	0,54 ± 0,05***
Ejection fraction,%	66,4 ± 0,5	57,1 ± 2,56***	59,2 ± 2,72***	58,4 ± 1,36***

Table 2: LV structural and functional parameters (M ± m) in young women with obesity and arterial hypertension.

Note: * - p < 0.05, ** - p < 0.01, *** - p < 0.001 - reliability of differences in groups I, II, III in relation to the control.

Indicators	Control group (n = 24)	Obesity without hypertension group I (n = 42)	Obesity and AH grade 1 II group (n = 34)	Obesity and AH grade 2 Group III (n = 32)
Normal	15 (62,5%)	-	-	-
Hypertrophic type DD	9 (37,5%)	13 (30,9%)	8 (23,5%)	6 (18,8%)
DTE, м	225,1 ± 1,6	239,4 ± 4,62*	236,8 ± 4,7*	242,4 ± 3,9*
IVRT, мс	102,2 ± 0,81	109,6 ± 3,42	108,1 ± 3,1	110,1 ± 2,8*
E/A	0,87 ± 0,02	0,73 ± 0,05*	0,75 ± 0,04*	0,74 ± 0,06*
$E/E_т$	6,2 ± 0,39	10,56 ± 0,32**	10,5 ± 0,46**	11,2 ± 0,35**
Pseudo-normal type DD	-	18 (42,9%)	14 (41,2%)	13 (40,1%)
DTE, м	-	148,2 ± 2,12	146,8 ± 4,1	148,8 ± 3,2
IVRT, мс	-	82,4 ± 2,26	81,6 ± 3,1	84,1 ± 3,2
E/A	-	1,25 ± 0,05	1,36 ± 0,01*	1,38 ± 0,03*
$E/E_т$	-	12,1 ± 0,4	12,5 ± 0,3	12,8 ± 0,4
Restrictive type of DD	-	11 (26,2%)	12 (35,3%)	13 (40,1%)
DTE, м	-	136,1 ± 2,4	139,8 ± 1,7	141,3 ± 1,5
IVRT, мс	-	62,1 ± 2,2	57,9 ± 2,2	58,9 ± 2,2
E/A	-	1,9 ± 0,05	2,1 ± 0,06*	2,3 ± 0,05**
$E/E_т$	-	16,06 ± 0,14	16,4 ± 0,3	16,1 ± 0,2
Left Atrial Volume, ml/m^2	22,4 ± 0,6	28,92 ± 0,5	29,8 ± 0,5	32,6 ± 0,4

Table 3: LV diastolic function (M ± m) in young women with obesity and arterial hypertension.

Note: * - p < 0.05, ** - p < 0.01, *** - p < 0.001 - reliability of differences in groups II, III in relation to group 1.

changes are concentric hypertrophy and concentric remodeling [24]. It is known that eccentric hypertrophy in adults is associated with the development of heart failure, while concentric remodeling and concentric hypertrophy are associated with heart attacks and impaired coronary circulation [25].

The process of LV remodeling is assessed not only by the degree of LV hypertrophy and dilatation, but also by the nature of diastolic function. This study showed the association of MMI with risk factors for obesity, the predominance of concentric LVH in obese patients, which apparently contributes to the more rapid formation of fibrosis and the development of restrictive diastolic dysfunction of the myocardium [26]. It is likely that impaired relaxation/filling of the myocardium increases MSD, overflows Vla, which has been demonstrated both in patients with both overweight and the development of hypertension.

Conclusion

Based on the analysis of structural-geometric and functional parameters of LV remodeling, it was revealed that in obese young patients, concentric LVH with concomitant restrictive diastolic dysfunction, a significant increase in MMI, preload value and Vla. The results of the study indicate that obesity in patients with hypertension contributes to a significant increase in cardiometabolic risk factors, exacerbates cardiac remodeling and negatively affects the function of the heart muscle.

Acknowledgments

The authors are grateful to the hospital administration, staff of the policlinics unit, the participants.

Conflict of Interest

No conflict of interest to declare.

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