CHARACTERISTICS OF HEMODYNAMIC PARAMETERS IN OBESE PATIENTS WITH STABLE EXERTIONAL ANGINA

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***Abstract**: Obesity is a primary, alterable risk factor for heart disease, closely linked to the onset and worsening of stable angina during exertion. Excess body fat can cause adverse alterations in cardiac structure, impair myocardial relaxation (diastolic function), disrupt endothelial function, and increase the workload on the cardiovascular system. These changes significantly affect systemic hemodynamics and contribute to the development and progression of cardiovascular disease. Nevertheless, the specific nature of these circulatory adjustments in obese individuals experiencing stable exertional angina has not been thoroughly studied.*

***Keywords:** systemic hemodynamics, left ventricle, ischemic heart disease, body mass index, coronary hemodynamics, obesity.*

Background: Due to their high rates of morbidity, disability, and mortality, cardiovascular diseases continue to rank among the most significant medical and social health issues globally [11]. 4.205 million people received their first diagnosis of circulatory system disorders in 2014, a 12.6% rise from 2010 [8]. During the same period, ischemic heart disease was newly diagnosed in 984.3 thousand individuals, reflecting a 39% increase compared with 2010. Of these cases, 449.2 thousand were diagnosed with stable angina, nearly twice the number reported in 2010 [6,8]. In the Russian Federation, circulatory system disorders were responsible for 52.9% of all fatalities in 2014 [5]. The working-age population showed the biggest rise in cardiovascular mortality. In 2014, circulatory system diseases accounted for 31.3% of deaths among working-age men and 23.9% among working-age women [8].

Several large-scale studies have demonstrated a close association between myocardial ischemia and atherosclerotic stenosis of the coronary arteries, with the extent of coronary involvement determining disease severity and clinical prognosis [4]. In recent years, however, an increasing number of patients have presented with typical anginal symptoms and positive stress test results despite the absence of significant atherosclerotic lesions in the coronary arteries [3]. The detrimental effect of myocardial ischemia is determined not only by the presence of anginal pain but also by the duration and severity of impaired myocardial perfusion [2].

As a consequence, myocardial contractile dysfunction, which is not always detectable by conventional echocardiography, may lead to significant impairment of left ventricular systolic function [7]. According to the myocardial helical architecture concept, ischemic heart disease initially affects the subendocardial myocardial fibers, resulting in early alterations of left ventricular diastolic mechanics before overt systolic dysfunction develops [1,10]. Progressive left ventricular dysfunction associated with chronic ischemic heart disease is a major contributor to the development of chronic heart failure and is reported in approximately 56% of patients with ischemic heart disease in the Russian Federation [9].

Conventional echocardiography remains the standard method for assessing myocardial function; however, its evaluation of myocardial contractility is partly subjective and influenced by preload, afterload, valvular function, and heart rate [7]. Therefore, comprehensive assessment of coronary, intracardiac, and systemic hemodynamics using modern diagnostic techniques in patients with stable exertional angina is essential for improving risk stratification, predicting adverse cardiovascular outcomes, and optimizing clinical management.

Objective: To evaluate the characteristics of central and systemic hemodynamic parameters in patients with stable exertional angina and obesity.

Materials and research methods. The study was conducted at the Clinic of Bukhara State Medical Institute and the Bukhara Regional Cardiology Dispensary. A total of **193 patients** were enrolled, including **110 patients** with obesity and stable exertional angina (Canadian Cardiovascular Society functional class I–II), who constituted the main group, and **83 patients** with stable exertional angina (functional class I–II) without obesity, who served as the comparison group.

Participants were considered ineligible if they were younger than 18 years or had acute coronary syndrome, heart failure, severe cardiac arrhythmias, advanced hepatic or renal dysfunction, systemic connective tissue disease, malignant neoplasms, endocrine disorders, or were pregnant.

Results and discussions.

The study was initiated by analyzing the gender distribution of the enrolled patients.

Table 1.

Gender Characteristics of Patients in the Main and Comparison Groups (n = 193)

Gender	**Main Group** (Stable exertional angina, CCS class I–II + obesity) *(n = 110)*		**Comparison Group** (Stable exertional angina, CCS class I–II without obesity) *(n = 83)*	
	абс	%	абс	%
Female	39	20,2%	47	24,3%
Male	71	36,8%	36	18,7%
Total	110	57%	83	43%

In the main group, **39 (20.2%)** patients were female and **71 (36.8%)** were male, whereas the comparison group included **47 (24.3%)** female and **36 (18.7%)** male patients. Overall, the main group comprised **110 (57.0%)** patients and the comparison group **83 (43.0%)** patients. Male patients outnumbered female patients by **1.8-fold** in the main group, while female patients were **1.3-fold** more frequent than male patients in the comparison group.

Subsequently, the distribution of obesity grades and the anthropometric characteristics of the study groups were evaluated.

Table 2.

Distribution of Obesity Grades and Anthropometric Characteristics of the Study Groups

Parameter	**Main Group (Stable Exertional Angina + Obesity)** *(n = 110)*	**Comparison Group (Stable Exertional Angina without Obesity)** *(n = 83)*
Body mass index (kg/m²)	34,6±2,8	26,1±1,9
Waist circumference (cm)	108,4±6,7	88,6±5,4
Overweight	18 (16,4%)	29 (34,9%)
Class I obesity	42 (38,2%)	-
Class II obesity	47 (42,7%)	-
Class III obesity	21 (19,1%)	-

According to the results presented in **Table 2**, anthropometric parameters were significantly higher in obese patients with stable exertional angina than in the comparison group. In the main group, the mean body mass index (BMI) was **34.6 ± 2.8 kg/m², ** with **Class I and Class II obesity**

predominating over Class III obesity. **Class II obesity** was the most frequent, observed in **47 (42.7%)** patients, indicating that increased body weight is associated not only with anthropometric changes but also with metabolic disturbances. **Class III obesity** was identified in **21 (19.1%)** patients and reflects a greater hemodynamic burden imposed on the cardiovascular system.

The accumulation of adipose tissue, particularly visceral fat, promotes activation of neurohumoral pathways, including the sympathetic nervous system and the renin–angiotensin–aldosterone system, resulting in increased peripheral vascular resistance. These alterations increase cardiac workload and myocardial oxygen demand, thereby contributing to the progression of myocardial ischemia.

The mean waist circumference in the main group reached **108.4 ± 6.7 cm**, confirming the presence of abdominal obesity, which is closely associated with insulin resistance, endothelial dysfunction, and chronic low-grade inflammation.

These findings suggest that the degree of obesity should be regarded not only as an anthropometric characteristic but also as an early predictor of adverse hemodynamic alterations in patients with stable exertional angina.

Subsequently, arterial blood pressure parameters were compared between the study groups. The results of this analysis are presented in **Table 3.**

Table 3.

Analysis of Arterial Blood Pressure Parameters in the Study Groups

Parameter	**Main Group (Stable Exertional Angina + Obesity)** ***(n = 110)***	**Comparison Group (Stable Exertional Angina without Obesity)** ***(n = 83)***
Systolic blood pressure (mmHg)	148,6±12,4	132,5±10,8
Diastolic blood pressure (mmHg)	91,4±7,2	82,6±6,4
Arterial hypertension, n (%)	76 (69,1%)	34 (40,9%)
Pulse pressure (mmHg)	57,2±8,1	49,9±6,7

According to the findings presented in **Table 3**, patients with obesity and stable exertional angina exhibited significantly higher arterial blood pressure values than those in the comparison group. In the main group, the mean systolic blood pressure was **148.6 ± 12.4 mmHg**, while the mean diastolic blood pressure was **91.4 ± 7.2 mmHg**. In the comparison group, the corresponding values were **132.5 ± 10.8 mmHg** and **82.6 ± 6.4 mmHg**, respectively. Compared with the comparison group, both systolic and diastolic blood pressure values were approximately **1.1-fold higher** in obese patients, indicating an increased hemodynamic load on the cardiovascular system associated with obesity.

Arterial hypertension was identified in **76 (69.1%)** patients in the main group and **34 (40.9%)** patients in the comparison group, demonstrating a substantially higher prevalence among obese patients with stable exertional angina. These findings suggest that the coexistence of obesity and stable exertional angina exerts a synergistic adverse effect on systemic hemodynamics.

Persistently elevated systolic blood pressure increases left ventricular afterload, promoting left ventricular hypertrophy, increased myocardial mass, and the development of diastolic dysfunction.

The mean pulse pressure was **57.2 ± 8.1 mmHg** in the main group and **49.9 ± 6.7 mmHg** in the comparison group. The higher pulse pressure observed in obese patients reflects reduced arterial elasticity and may adversely affect coronary perfusion.

Subsequently, a comparative analysis of echocardiographic parameters was performed in both study groups, and the results are presented in **Table 4.**

Table 4.

Comparative Analysis of Echocardiographic Parameters

Parameter	**Main Group (Stable Exertional Angina + Obesity) *(n = 110)***	**Comparison Group (Stable Exertional Angina without Obesity) *(n = 83)***
Left ventricular ejection fraction (LVEF, %) *Reference range: 55–70%*	52,8±4,6	60,1±3,9
Left ventricular mass (g)	214±18	176±15
Left ventricular wall thickness (mm)*Reference range: 9–11 mm*	12,6±1,1	10,4±0,9
E/A ratio*Reference range: 0.8–2.0*	0,78±0,09	1,05±0,12
Left atrial diameter (mm)*Reference range: 20–40 mm*	41,5±3,2	36,8±2,9

According to the data presented in **Table 4**, echocardiographic examination made it possible to identify structural and functional changes in the hearts of obese patients with stable exertional angina.

In the main group, the decrease in left ventricular ejection fraction indicated reduced myocardial functional reserve, with a mean value of **52.8 ± 4.6%,** whereas in the comparison group the mean value was **60.1 ± 3.9%.**

In the main group, the mean left ventricular myocardial mass was **214 ± 18 g**, and its increase can be explained by the cardiac remodeling process developing under the influence of obesity and arterial hypertension. Although these changes are initially compensatory in nature, they subsequently lead to the development of diastolic dysfunction and heart failure. In the comparison group, the mean left ventricular myocardial mass was **176 ± 15 g**, which was within the normal range, i.e., normal for men and slightly increased for women, indicating that myocardial mass was considerably lower and healthier than in the main group.

The mean left ventricular wall thickness in the main group was **12.6 ± 1.1 mm**, indicating thickening of the ventricular wall, i.e., myocardial hypertrophy. This condition is most commonly observed in patients with long-standing arterial hypertension. In the comparison group, the mean left ventricular wall thickness was **10.4 ± 0.9 mm**, indicating that myocardial wall thickening had not yet developed.

In the main group, the mean **E/A ratio** was **0.78 ± 0.09**, indicating left ventricular diastolic dysfunction (ratio below 0.8). In the comparison group, the mean **E/A ratio** was **1.05 ± 0.12**, indicating that cardiac function remained within the normal range (normal value above 0.8). In particular, a decrease in this ratio reflects impaired left ventricular relaxation.

In the main group, the mean left atrial diameter was **41.5 ± 3.2 mm**, indicating that because of impaired left ventricular relaxation (E/A = 0.78), excessive pressure was imposed on the left atrium, resulting in its enlargement.

In the comparison group, the mean left atrial diameter was **36.8 ± 2.9 mm**, which was within the normal range.

One of the main objectives of the study was the analysis of systemic hemodynamic parameters (**Table 5**).

Table 5.

Comparative Analysis of Systemic Hemodynamic Parameters

Parameter	**Main Group (Stable Exertional Angina + Obesity)** ***(n = 110)***	**Comparison Group (Stable Exertional Angina without Obesity)** ***(n = 83)***
Systolic blood pressure (mmHg)	148,6±12,4	132,5±10,8
Diastolic blood pressure (mmHg)	91,4±7,2	82,6±6,4
Heart rate (beats/min)	82,4±7,1	74,8±6,3
Stroke volume (mL)	68,5±5,7	75,4±6,1
Cardiac output (L/min)	5,6±0,5	5,2±0,4
Cardiac index (L/min/m²)	2,6±0,3	3,1±0,3
Total peripheral vascular resistance	1680±190	1320±160

According to the results presented in **Table 5**, patients with obesity and stable exertional angina demonstrated significant alterations in systemic hemodynamics.

In the main group, the mean systolic arterial blood pressure was **148.6 ± 12.4 mmHg**, while the mean diastolic arterial blood pressure was **91.4 ± 7.2 mmHg**, indicating that the elevated blood pressure values reflect an increased hemodynamic load on the cardiovascular system associated with obesity. In the comparison group, the mean systolic arterial blood pressure was **132.5 ± 10.8 mmHg**, and the mean diastolic arterial blood pressure was **82.6 ± 6.4 mmHg**. These findings showed that both systolic and diastolic arterial blood pressure values were **1.1-fold higher** in the main group than in the comparison group. In obesity, the increase in adipose tissue volume leads to an increase in circulating blood volume, thereby increasing the demand for cardiac output. The increase in left ventricular stroke volume initially represents a compensatory mechanism; however, its long-term persistence results in excessive functional stress on the myocardium.

The mean total peripheral vascular resistance was **1680 ± 190** in the main group and **1320 ± 160** in the comparison group. This parameter was **1.3-fold higher** in the main group than in the comparison group. Increased total peripheral vascular resistance is linked to endothelial dysfunction, loss of vascular wall elasticity, and sympathetic nervous system activation, collectively leading to impaired coronary flow reserve.

Conclusion.

This research indicates that obesity significantly affects hemodynamic measures in individuals experiencing stable exertional angina. Compared to non-obese individuals, obese patients displayed more significant changes in both central and systemic hemodynamics, along with reduced left ventricular diastolic function. These alterations intensified with greater degrees of obesity, highlighting the negative influence of excess body weight on the heart's function. A thorough evaluation of hemodynamic parameters offers valuable diagnostic and prognostic insights, enabling the early detection of individuals with a heightened risk of cardiovascular events.These findings support the implementation of individualized preventive and therapeutic strategies aimed at improving cardiac function, reducing cardiovascular complications, and enhancing the quality of life in obese patients with stable exertional angina.

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