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DYNAMICS OF CHARACTERISTICS OF THE GLYCEMICAL PROFILE OF PATIENTS WITH CORONARY HEART DISEASE AND OBESITY AFTER TREATMENT

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Summary

Introduction. Cardiovascular diseases (CVD) have the status of an epidemic, as they have high levels of prevalence and are the main cause of disability and mortality both in Ukraine and in the world and cause a significant increase in health care costs. Among all CVDs, coronary heart disease (CHD) ranks first. Many studies confirm a high percentage of CHD among all CVDs, especially in comorbidity with hyperlipidemia and obesity. Therefore, when treating CHD disease with obesity, it is necessary to take into account the effectiveness of the applied pharmacological agents and determine the dynamics of pharmacological intervention.
The aim. To determine the dynamics of indicators of the glycemic profile in the comorbid course of CHD and obesity after the treatment.
Materials and methods. It was conducted a randomized controlled single-center prospective study case-control, which is based on the analysis of the results of 130 people aged 25-85 were examined, who were divided into 3 groups: 70 persons (main group) with CHD on the background of obesity and 35 people with isolated coronary artery disease (comparison group) and control group (25 practically healthy people). The studied groups were randomized by age and gender.
Results. Before treatment, a probable predominance of daily glucose levels was determined in patients main group to the patients comparison group and controls. According to the results of the glucose tolerance test (GTT), an improbable excess of fasting glucose levels and after a glucose load was determined in CHD with obesity (respectively 5.64±1.92 and 7.08±2.25 mmol/l) compared to the isolated of CHD (respectively 5, 15±2.22 (p=0.791) and 6.20±3.15 (p=0.403) mmol/l) and control group (respectively 5.32±0.49 (p=0.685) and 5.42±0, 51 (p<0.001) mmol/l). After treatment, recovery of blood glucose levels was determined.
Conclusions. It was established that the characteristics of the dynamics of glucose metabolism indicators can be used as an indicator of the effectiveness of the treatment in the comorbidity of obesity and CHD. The obtained results indicate that the characteristics of glucose metabolism in the comorbidity of CHD and obesity must be taken into account to ensure therapeutic and preventive measures.

Key words: **coronary heart disease, obesity, dynamics, glycemical profile, comorbidity.**

INTRODUCTION

Recently, cardiovascular diseases (CVD) have the status of an epidemic, as they have high levels of prevalence and are the main cause of disability and mortality both in Ukraine and in the world and cause a significant increase in health care costs [1-8]. According to research results, in 2017, CVD caused 17.8 million of all global deaths [4, 8], and in 2019-6.2 million deaths of people aged 30-70 years [4]. According to forecasts of WHO specialists, the mortality rate from CVD will increase to 24.1-24.3 million by 2030 [9].

Scientists indicate that the main pathogenetic mechanisms of CVD progression are atherosclerotic lesions, which provoke the development of coronary heart disease (CHD), cerebrovascular diseases, venous thromboembolism, and diseases of peripheral vessels (mostly in most cases CHD). In addition, etiological factors of CVD are hyperlipidemia, hypertension,

diabetes, obesity, smoking, and low physical activity (more than 90.0% of CVD risks) [10]. At the same time, many studies confirm a high percentage of CHD among all CVDs, especially in comorbidity with hyperlipidemia and obesity [11].

It has been confirmed that obesity causes pathophysiological changes that are associated with the development of vascular events, resulting in the development of CHD and other CVD [12]. The risks of developing CVD in obese patients are likely to double compared to those of normal body weight (odds ratio (OR) 2.0; [95.0% confidence intervals (CI) 1.7-2.4]; p<0.0001); five times – with obesity of the 1st degree (OR 4.5 [95.0% CI 3.5-5.8]; p<00001) and almost 15 times – with obesity of the 2nd and 3rd degrees (combined OR 14.5 [95.0% CI 10.1-21.0]; p<0.0001). The risks of developing CHD with obesity of the II and III degrees increase by 2.2 times (OR 2.2 [95.0% CI 1.9-2.6]) [13].

Therefore, when treating CHD with obesity, it is necessary to take into account the effectiveness of the applied pharmacological agents and determine the dynamics of pharmacological intervention.

THE AIM

To determine the dynamics of indicators of the glycemic profile in the comorbid course of CHD and obesity after the treatment.

MATERIALS AND METHODS

Conducted a randomized controlled single-center prospective study case-control, which is based on the analysis of the results of 130 people 25-85 were examined, who were divided into 3 groups: 70 persons (main group) with CHD on the background of obesity and 35 people with isolated CHD (comparison group). In the main group, the average age was 63.6±8.8 years, most of them (72.9%) were female compared to respondents (27.1%) who were male. Among the patients of the main group, there was a majority of patients with I (26 (37.1%)) degree of obesity, compared to II (24 (34.3%)) and III (20 (28.6%)). In the comparison group, the average age was 69.7±7.9 years and, on the contrary, the majority (68.6%) were men compared to women (31.4%). The control group (25 practically healthy people, randomized by age and gender) had an average age of 59.8±14.6 years and the majority (77; 59.2%) consisted of women in contrast to men (10; 40.8%). The statistical difference according to the studied age-sex characteristics was established at a statistically significant level ($p \leq 0.001$).

All respondents before the start of the study were fully informed about the voluntariness of their participation in this study and the complete confidentiality of the information received from them, and had comprehensive written information about the main purpose and objectives of our study and its duration and substance. The respondents surveyed by us took part in the study entirely of their own free will, which was confirmed by their personal signature in the relevant informed consent. Each of the research subjects was personally fully informed about his responsibilities and rights in the conducted research and the complete possibility to end his participation in the research at any time without any consequences for him and explanation of the reasons for his actions. Inclusion criteria were: reaching 18 years of age, presence (main group) CHD on the background of obesity, isolated CHD (comparison group) or absence CHD and obesity (control group), consent to participate in the study. Exclusion criteria were: not reaching 18 years of age, absence (main and comparison group) CHD and obesity or presence (control group) of CHD and obesity, absence: diffuse and focal diseases, diabetes and other endocrine pathology, allergic reactions, systemic connective tissue diseases, acute and chronic inflammatory diseases of internal organs, severe decompensated somatic pathology, psychiatric and oncological diseases, acute cardiovascular disorder, thyrotoxic crisis, acute and significant decompensation of carbohydrate metabolism; availability: unsatisfactory physical condition, pregnancy and breastfeeding, chronic alcoholism, refusal to participate in the study and refusal to comply with all prescriptions.

In the treatment of CHD and obesity, all patients (70 (100.0%)) received statins and metabolic agents; also prescribed antiplatelet agents (67 (95.7%) persons), diuretics (54 (77.1%)); β-blockers (49 (70.0%)) and angiotensin II receptor blockers (44 (62.9%)); calcium channel blockers (32 (32.9%)); nitrates (27 (38.6%)); ACE inhibitors (18 (25.7%)); hypoglycemic agents (10 (14.3%)); cardiac glycosides (7 (10.0%)) and anticoagulants (4 (5.7%)). Metabolic drugs were prescribed to all patients with isolated coronary artery disease (35 (100.0%) persons); most were prescribed antiplatelet agents (34 (97.1%)); statins (33 (94.3%)); diuretics (30 (85.7%)); β-blockers (24 (68.6%)) and angiotensin II receptor blockers (23 (65.7%)); ACE inhibitors (16 (45.7%)); nitrates (12 (34.3%)); calcium channel blockers (11 (31.4%)); hypoglycemic (6 (17.1%)) and anticoagulant (5 (14.3%)) agents and cardiac glycosides (3 (8.6%)).

The diagnosis of CHD was established according to current guidelines [14, 15]. The diagnosis of obesity was determined according to the recommendations EASO (2017) [16] and NIH (2000) [17].

Determination of indicators of carbohydrate metabolism (fasting venous blood glucose, glycemic profile, and the oral glucose tolerance test (GTT), and others) was carried out using generally accepted methods.

Medical-statistical calculation. The distribution of qualitative and quantitative signs was carried out graphically visually and with the help of the Kolmogorov-Smirnov and Lilliefors test for normality and Shapiro-Wilk's test of normality. During the assessment, it was established that there were significant differences from the normal nature of the distribution, so further calculations were carried out using non-parametric medical and statistical methods.

Thus, when characterizing the central tendency and variability of quantitative (continuous or interval) traits, the mean value (M) and standard square deviation (SD, σ) were determined: M ± SD.

The probability of differences in the obtained quantitative characteristics in two mutually independent groups was determined using the Mann-Whitney U-test, and in mutually dependent groups, the Wilcoxon matched-pairs signed-ranks T-test was used.

The threshold value of the level of probability of all calculated features was taken as 0.05 (p = 0.05) with the indication of the exact value of the level of reliability «p» with three decimal places. When carrying out multiple comparisons of the obtained characteristics, the Bonferroni correction was applied to correct the confidence level. Statistical calculations were performed in IBM SPSS 25.0 (trial version) for Windows.

The work is a fragment of research work of The Department of Internal Medicine № 2, Clinical Immunology and Allergology named after academician L. T. Malaya Kharkiv National Medical University «Predicting the course, improving diagnosis and treatment of coronary artery disease and hypertension in patients with metabolic disorders» (№ state registration 0120U102025), deadline: 2020-2025. Project leader – Head of the Department of Internal Medicine № 2, Clinical Immunology and Allergology named after academician L. T. Malaya Kharkiv National Medical University, Doctor of Medical Sciences, professor Pavlo G. Kravchun.

RESULTS

We determined the dynamics of indicators of the glycemic profile and the oral GTT among patients of the main group, patients of the comparison group, and individuals of the control group. In patients of the main group, these indicators were analyzed depending on the degree of obesity. Before treatment, based on the results of the study of the glycemic profile of the subjects and the oral GTT, were determined gradual increases in glucose levels during the day compared to the level obtained on an empty stomach and some decrease in these levels until 9 p.m. At the same time, it was noted mainly probable expected predominance of daily glucose levels in CHD compared to controls. Also, slightly disturbed on an empty stomach glycemia was determined in the group of patients with CHD and obesity (≥5.6≤6.1 mmol/l on an empty stomach and <6.7 at 9 a.m.). Thus, in patients with CHD with obesity, on an empty stomach glucose was determined at the level of 5.62 ± 1.42 mmol/l and at 9 a.m. – 5.76 ± 2.04 – Table 1.

Table 1

Characteristics of the glycemic profile, M ± SD

Characteristics of the glycemic profile		Research groups			p_{1-2}	p_{1-3}	p_{2-3}
		main (*n* = 70)	comparison (*n* = 35)	control (*n* = 25)			
Glucose, mmol/l	9:00	5.76 ± 2.04	5.06 ± 2.38	4.86 ± 0.68	0.575	0.016	0.267
	13:00	6.64 ± 2.67	5.07 ± 3.45	4.87 ± 0.63	0.954	<0.001	0.002
	17:00	6.26 ± 1.90	5.74 ± 2.65	4.90 ± 0.70	0.900	<0.001	<0.001
	21:00	5.96 ± 1.69	5.31 ± 2.29	5.02 ± 0.63	0.814	0.004	0.014
	6:00	5.62 ± 1.42	5.02 ± 2.21	4.92 ± 0.66	0.206	0.005	0.372
GTT, mmol/l	1 measurement	5.64 ± 1.92	5.15 ± 2.22	5.32 ± 0.49	0.791	0.685	0.994
	2 measurement	7.08 ± 2.25	6.20 ± 3.15	5.42 ± 0.51	0.403	<0.001	0.001

Notes: the probability of differences: p_{1-2} – in the comparison of the indicators of the main and the comparison groups; p_{1-3} – in the comparison of indicators of the main and control groups; p_{2-3} – in the comparison of indicators of the comparison group and the control group.

It was determined that for all daily indicators, glucose levels significantly prevailed in CHD against the background of obesity. Thus, blood glucose level on an empty stomach was 5.62 ± 1.42 mmol/l, improbably (p=0.206) exceeding the levels in isolated CHD (5.02 ± 2.21) and significantly (p=0.005) – the control group (4.92 ± 0.66). At 9 a.m., there was an expected increase in glucose levels with the same significant excess in CHD and obesity compared with isolated CHD and controls (5.76 ± 2.04; 5.06 ± 2.38 (p=0.575) and 4.86 ± 0.68 (p=0.016) m mol/l). At 1 p.m., an increase in glucose levels to 6.64 ± 2.67 mmol/l was noted in the main group, improbably (p=0.954) exceeding the indicators of the comparison group (5.07 ± 3.45) and probably (p<0.001) – the control group (4.87 ± 0.63). At 5 p.m., maximum daily glucose levels were noted with the same excess in patients with CHD and obesity (6.26 ± 1.90 mmol/l) improbably (p=0.900) compared to isolated CHD (5.74 ± 2.65) and probably (p<0.001) with control (4.90 ± 0.70). At 9 p.m., happened

a significant decrease in glucose levels of the subjects with higher levels in the main group (5.96 ± 1.69 mmol/l) improbably compared with the group with isolated CHD (5.31 ± 2.29; p=0.814) and probably with the control group (5.02 ± 0.63; p=0.004) – Table 1.

According to the values of oral GTT, no deviations from the normative indicators were found in the comparison group. An improbable excess on an empty stomach glucose levels (1st measurement) and after a glucose load (2nd measurement) was determined among patients with CHD and obesity (5.64 ± 1.92 and 7.08 ± 2.25 mmol/l, respectively) compared to the comparison group (respectively 5.15 ± 2.22; p=0.791 and 6.20 ± 3.15; p=0.403) and control group (respectively 5.32 ± 0.49; p=0.685 and 5.42 ± 0.51; p< 0.001) – Table 1.

According to the obtained levels of the daily glycemic profile, depending on the degree of obesity, the study determined some excess of the normative indicators (unlikely, most of them in II and III degrees) – Table 2. A slightly disturbed on an empty stomach glycemia (≥5.6≤6.1 mmol/l on an empty stomach and <6.7 at 9 a.m.) was also recorded in II degree (on an empty stomach glucose level was recorded at the level of 6.00 ± 1.37 mmol/l and at 9 a.m. – 5.84 ± 1.81) – Table 2.

Table 2

Characteristics of the glycemic profile of the subjects of the main group depending on the degree of obesity, M ± SD

Characteristics of the glycemic profile		Degrees of obesity			p_{1-2}	p_{1-3}	p_{2-3}
		I (*n* = 26)	II (*n* = 24)	III (*n* = 20)			
Glucose, mmol/l	9:00	5.54 ± 1.79	5.84 ± 1.81	5.99 ± 2.62	0.726	0.936	0.654
	13:00	6.52 ± 2.88	6.80 ± 2.56	6.63 ± 2.64	0.409	0.665	0.732
	17:00	6.36 ± 1.93	6.35 ± 1.88	6.03 ± 1.94	0.953	0.842	0.436
	21:00	6.03 ± 2.35	5.80 ± 1.20	5.85 ± 1.13	0.232	0.542	0.509
	6:00	5.41 ± 1.53	6.00 ± 1.37	5.43 ± 1.29	0.071	0.626	0.389
GTT, mmol/l	1 measurement	5.49 ± 1.78	5.73 ± 1.74	5.75 ± 2.34	0.749	1.000	0.646
	2 measurement	7.45 ± 3.00	7.48 ± 1.75	6.13 ± 1.20	0.360	0.058	0.003

Notes: the probability of differences: p_{1-2} – in the comparison of indicators for I and II degrees of obesity; p_{1-3} – in the comparison of indicators for I and III degrees of obesity; p_{2-3} – in the comparison of indicators for II and III degrees of obesity.

An improbable predominance glucose levels on an empty stomach was found in II degree of obesity (6.00 ± 1.37 mmol/l) compared to III and I (respectively, 5.43 ± 1.29; p=0.389 and 5.41 ± 1.53; p=0.071). At 9 a.m., there was an increase in glucose levels with an improbable advantage at III degree obesity (5.99 ± 2.62 mmol/l) compared to II (5.84 ± 1.81; p=0.654) and I (5.54 ± 1.79; p=0.936). At 1 p.m., an increase in glucose levels was also recorded, with a corresponding improbable advantage at II degree (6.80 ± 2.56 mmol/l) compared to III (6.63 ± 2.64; p=0.732) and I (6.52 ± 2.88; p=0.409); after which, at 5 p.m., a decrease in these levels was ascertained with incredibly lower values at III degree (6.03 ± 1.94) compared to II (6.35 ± 1.88; p=0.436) and I (6.36 ± 1.93; p=0.842); as well as at 9 p.m., when their decrease was also noted with lower values at III degree (5.85 ± 1.13) compared to II (5.80 ± 1.20; p=0.509) and I (6.03 ± 2.35; p=0.542) – Table 2.

GTT profiles were within normative limits with an improbable predominance on an empty stomach (1st measurement) at III (5.75 ± 2.34 mmol/l) and II (5.73 ± 1.74) degrees compared to I (5.49 ± 1.78; respectively, p=1.000 and p=0.749). It should be noted that patients with II and I degrees probably reacted to the glucose load (2nd measurement) with a greater increase in blood glucose (7.48 ± 1.75 and 7.45 ± 3.00 mmol/l, respectively) compared to III (6.13 ± 1.20; respectively, p=0.003 and p=0.058) – Table 2.

According to the dynamics of the glycemic profile of patients with CHD and obesity after the treatment in the main group, a decrease in blood glucose levels compared to the pretreatment level was noted, and in the comparison group – a slight increase (both groups practically equalized); although, exceeding the values of their daily levels was not recorded. As before the treatment, after it, an increase in daily glucose levels was noted in both groups until 5 p.m., and then their gradual decrease until 9 p.m. – Table 3.

Unbelievably, after treatment with isolated CHD, the daily glucose levels slightly exceeded the indicators in the main group: at 6 a.m., respectively, 5.21 ± 1.40 and 5.39 ± 1.10 (p=0.536) mmol/l; at 9 a.m. – 5.42 ± 1.52 and 5.46 ± 1.52 (p=0.802) mmol/l; at 1 p.m. – 6.10 ± 1.53 and 5.88 ± 1.30 (p=0.463) mmol/l; at 5 p.m. – 6.25 ± 1.31 and 5.88 ± 1.23 (p=0.081) mmol/l and at 9 p.m. – 5.96 ± 1.46 and 5.72 ± 1.33 (p = 0.278) mmol/l – Table 3.

In the main group, on the background of treatment, in the dynamics compared to the up to treatment level, a decrease in daily glucose levels was noted, and in the comparison group, on the contrary, there was a slight increase: at 6 a.m. – by 0.23 (p=0.039) and 0.19 (p=0.253) mmol/l; at 9 a.m. – by 0.3 (p=0.029) and 0.36 (p=0.395) mmol/l; at 1 p.m. – by 0.76 (p=0.010) and 1.03 (p=0.073) mmol/l; at 5 p.m. – by 0.38 (p=0.139) and 0.51 (p=0.365) mmol/l and at 9 p.m. – by 0.24 (p=0.270) and 0.65 (p=0.630) mmol/l – Table 3.

Table 3

Characteristics of the glycemic profile of the subjects after treatment, M ± SD

Characteristics of the glycemic profile		Research groups				p_1	p_2	p_3
		main (n = 70)		comparison (n = 35)				
		indicator	dynamics	indicator	dynamics			
Glucose, mmol/l	9:00	5.46 ± 1.52	-0.3	5.42 ± 1.52	+0.36	0.802	0.029	0.395
	13:00	5.88 ± 1.30	-0.76	6.10 ± 1.53	+1.03	0.463	0.010	0.073
	17:00	5.88 ± 1.23	-0.38	6.25 ± 1.31	+0.51	0.081	0.139	0.365
	21:00	5.72 ± 1.33	-0.24	5.96 ± 1.46	+0.65	0.278	0.270	0.630
	6:00	5.39 ± 1.10	-0.23	5.21 ± 1.40	+0.19	0.536	0.039	0.253
GTT, mmol/l	1 measurement	5.37 ± 1.29	-0.27	5.38 ± 1.39	+0.23	0.927	0.217	0.527
	2 measurement	6.36 ± 1.14	-0.72	6.35 ± 1.16	+0.15	0.970	0.001	0.207

Notes: the probability of differences: p_1 – in the comparison of the indicators of the main and the comparison group after treatment; p_2 – in the comparison of the indicators of the main group before and after treatment; p_3 – in the comparison of indicators of the comparison group before and after treatment.

According to the levels of oral GTT, a decrease in glucose levels after treatment was also noted compared to the pretreatment level in the main group (1st measurement improbable (p=0.217) by 0.27 and probable (p=0.001) 2st measurement by 0.72 mmol/l) and an increase in the comparison group (1st measurement – improbable (p=0.527) by 0.23 and improbable (p=0.207) 2st measurement – by 0.15 mmol/l). At the same time, GTT indicators according to the respective profiles in the main and comparison groups were improbably compared and amounted to 5.37 ± 1.29 and 5.38 ± 1.39 (p=0.927) mmol/l for the 1st measurement, respectively, and for the 2nd measurement – 6.36 ± 1.14 and 6.35 ± 1.16 (p=0.970) mmol/l, respectively. It should be noted that after treatment in both groups noted impaired glucose tolerance (1st measurement <6.1 and 2st measurement – ≥6.1 and <10.0 mmol/l) according to GTT – Table 3.

According to the dynamics of the daily levels of the glycemic profile and oral GTT in the main group, depending on the degree of obesity after treatment, the better dynamics of lowering blood glucose levels was observed in II and I degrees of obesity compared to III (in which some increase was also noted, which indicates the negative impact of significant obesity on glucose levels and possible influence on the course of CHD itself). Also, the negative effect of obesity on the glucose level was determined by an increase in glucose at 9 a.m. and its decrease after at 1 p.m. at the III degree of obesity. At the II and I degrees of obesity, a decrease in glucose levels was noted after 1 p.m. – 5 p.m. Yes, the advantage of daily glucose levels was recorded in all profiles at the III degree of obesity compared to II and I: at 6 a.m. – 5.86 ± 1.21 and 5.86 ± 1.21 respectively 5.20 ± 1.02 (p=0.035) and 5.21 ± 1.01 (p=0.058) mmol/l; at 9 a.m. – 5.87 ± 1.87 and 5.37 ± 1.49 (p=0.511) and 5.22 ± 1.19 (p=0.444); at 1 p.m. – 6.42 ± 1.41 and 5.71 ± 1.28 (p=0.036) and 5.63 ± 1.15 (p=0.054); at 5 p.m. – 6.08 ± 1.28 and 5.62 ± 1.21 (p=0.171) and 5.96 ± 1.22 (p=0.368) and at 9 p.m. – 6.26 ± 1, 45 and 5.50 ± 1.20 (p=0.035) and 5.50 ± 1.28 (p=0.044) – Table 4.

According to the indicators of oral GGT, there was also a predominance of glucose levels in the III degree of obesity (respectively, the 1st measurements and 2nd measurements 5.52 ± 1.36 and 6.27 ± 1.16 mmol/l) compared to the II degree of obesity (respectively, 5.27 ± 1.35 (p=0.532) and 6.22 ± 0.83 (p=0.777) mmol/l) and I degree of obesity (respectively 5.37 ±1.21 (p=0.938) and 6.55 ± 1.36 (p=0.578) mmol/l). It was noted the presence of impaired glucose tolerance (1st measurement <6.1 and 2nd measurement – ≥6.1<10.0 mmol/l) at all degrees of obesity – Table 4.

According to the dynamics of the indicators of the glycemic profile of patients in the main group, depending on the degree of obesity, after the treatment compared to the pre-treatment level, in general, positive dynamics were noted. Thus, at the 2nd and 1st degrees of obesity, at 6 a.m., there was a decrease in glucose levels probably (p=0.001) by 0.8 and improbably (p=0.419) by 0.2 mmol/l; at 9 a.m. – respectively, improbable (p=0.126) by 0.47 and improbable (p=0.061) by 0.32 mmol/l; at 1 p.m. – probable (p=0.004) by 1.09 and improbable (p=0.147) by 0.89 mmol/l; at 5 p.m. – unlikely (p=0.060) by 0.73 and unlikely (p=0.305) by 0.40 mmol/l and at 9 p.m. – likely (p=0.012) by 0.3 and unlikely (p=0.275) by 0.53 mmol/l. At the same time, with III degree of obesity after treatment, an increase in glucose levels was noted: at 6 a.m., at 5 p.m. and at 9 p.m. (respectively by 0.43 (p=0.329); 0.05 (p=0.345) and 0.41 (p=0.030) mmol/l) and a decrease – at 9 a.m. and at 1 p.m. (by 0.12 (p=0.706) and 0.21 (p=0.875) mmol/l, respectively). According to the indicators of the GTT in the dynamics, a decrease in glucose levels was determined in II and I degrees of obesity (respectively, the 1st measurement by 0.46 (p=0.161) and 0.12 (p=0.615) mmol/l and the 2nd measurement – by 1,26 (p=0.001) and 0.9 (p=0.076) mmol/l; and with III degree of obesity – a decrease according to the 1st measurement – by 0.23 (p=0.950) mmol/l and an increase according to the 2nd measurement – by 0.14 (p=1,000) mmol/l – Table 4.

Table 4

Characteristics of the glycemic profile of the subjects of the main group depending on the degree of obesity after treatment, M ± SD

Characteristics of the glycemic profile		Degrees of obesity			p_{1-2}	p_{1-3}	p_{2-3}
		I (n = 26)	II (n = 24)	III (n = 20)			
Glucose, mmol/l	9:00	5.22 ± 1.19	5.37 ± 1.49	5.87 ± 1.87	0.779	0.444	0.511
	13:00	5.63 ± 1.15	5.71 ± 1.28	6.42 ± 1.41	0.946	0.054	0.036
	17:00	5.96 ± 1.22	5.62 ± 1.21	6.08 ± 1.28	0.606	0.368	0.171
	21:00	5.50 ± 1.28	5.50 ± 1.20	6.26 ± 1.45	0.930	0.044	0.035
	6:00	5.21 ± 1.01	5.20 ± 1.02	5.86 ± 1.21	0.801	0.058	0.035
GTT, mmol/l	1 measurement	5.37 ± 1.21	5.27 ± 1.35	5.52 ± 1.36	0.648	0.938	0.532
	2 measurement	6.55 ± 1.36	6.22 ± 0.83	6.27 ± 1.16	0.868	0.578	0.777
dynamics					p_1	p_2	p_3
Glucose, mmol/l	9:00	-0.32	-0.47	-0.12	0.061	0.126	0.706
	13:00	-0.89	-1.09	-0.21	0.147	0.004	0.875
	17:00	-0.40	-0.73	+0.05	0.305	0.060	0.345
	21:00	-0.53	-0.30	+0.41	0.275	0.012	0.030
	6:00	-0.20	-0.80	+0.43	0.419	0.001	0.329
GTT, mmol/l	1 measurement	-0.12	-0.46	-0.23	0.615	0.161	0.950
	2 measurement	-0.90	-1.26	+0.14	0.076	0.001	1.000

Notes: the probability of differences: p_{1-2} – in the comparison of indicators for I and II degrees of obesity after treatment; p_{1-3} – in the comparison of indicators for I and III degrees of obesity after treatment; p_{2-3} – in the comparison of indicators for II and III degrees of obesity after treatment; p_1 – in the comparison of indicators in the 1st degree of obesity before and after treatment; p_2 – in the comparison of indicators for II degree of obesity before and after treatment; p_3 – in the comparison of indicators for III degree of obesity before and after treatment.

DISCUSSION

Our results regarding that which, that characteristics of the dynamics of glucose metabolism indicators can be used as an indicator of the effectiveness of the treatment in the comorbidity of obesity and CHD are completely consistent with other conducted studies.

Thus, Evstratova Y. N. and Mukhannad Alshbul [18] in a study of 70 patients with stable angina of functional class I-II complicated by abdominal obesity (mean age of patients 60 ± 5 years) obtained results that indicated the presence of a state of impaired glucose tolerance with clinical manifestations of CHD and obesity. As a result of the study, it was found a significant excess blood glucose on an empty stomach up to 6.5 ± 0.4 mmol/l ($p<0.050$) and an excess of blood glucose 2 hours after glucose load up to 10.0 ± 0.7 mmol/l ($p<0.05$).

Mankovsky G. B. [19] in the examination of 89 patients with CHD (46 men, 43 women, mean age 62+3.6years) using the determination of glucose in venous blood plasma on an empty stomach found an increase in the studied indicator, sufficient for diagnosing diabetes mellitus in 16% of the examined. Also, when conducting a glucose tolerance test, 16% of patients found an increased level of glucose in the blood plasma 2 hours after taking glucose.

Lapovets L. Ye. et al [20] examined 30 patients with verified CHD (20 patients with CHD without obesity (15 men, 5 women) and 10 patients with CHD and obesity (9 men, 1 woman), mean age was 55 ± 2 years) and 20 practically healthy persons (control group), randomized by sex and age. As a result of the study, they obtained data that the presence of glucose in the blood plasma of patients with CHD exceeded the normal values by 76% ($p<0.05$); and in patients with CHD, aggravated by obesity, it was aggravated by glucose, exceeding the control value by 56% ($p<0.05$).

Kadykova O. I. [21] investigated the positive effect of therapy on glucose levels. 42 patients with coronary artery disease and obesity were studied, who were divided into two subgroups: subgroup 1-22 patients with coronary artery disease and obesity, who received enalapril at a daily dose of 20 mg, carvedilol at a daily dose of 50 mg, and spironolactone at a dose of 50 mg per day; Subgroup 2-20 patients with coronary heart disease and obesity who received lisinopril at a daily dose of 20 mg, nebivalol at a daily dose of 10 mg and eplerenone at a dose of 50 mg per day. According to the results of a comprehensive study, a decrease in glucose levels was established from 4.43 ± 0.07 mmol/l to 4.39 ± 0.05 mmol/l (1 subgroup) and to 4.33 ± 0.06 mmol/l (2 subgroup) – by 0.9% and 2.26%, respectively.

CONCLUSIONS

On the basis of the conducted studies, it was established that the characteristics of the dynamics of glucose metabolism indicators can be used as an indicator of the effectiveness of the treatment in the comorbidity of obesity and CHD. An increase in the indicators of glucose metabolism in the blood serum characterizes a significant relationship with the development of such a comorbidity, which indicates a significant influence of the characteristics of glucose metabolism on the pathogenesis of the comorbidity of obesity and CHD. The obtained results indicate that the characteristics of glucose metabolism in the comorbidity of CHD and obesity must be taken into account to ensure therapeutic and preventive measures.

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COMPLIANCE WITH ETHICS REQUIREMENTS

The ethical approval was obtained from Bioethics Committee of the Kharkiv National Medical University. All patients provided written consent to participate in research in accordance with the recommendations of the Ethics Committees for Biomedical Research, Ukrainian Health Legislation and the Declaration of Helsinki of 2000.

LITERATURE

1. Zhou D., Xi B., Zhao M., Wang L., Veeranki S. P. Uncontrolled hypertension increases risk of all-cause and cardiovascular disease mortality in US adults: the NHANES III Linked Mortality Study. Sci Rep. 2018. Vol. 8(1). P. 9418. doi: https://doi.org/10.1038/s41598-018-27377-2
2. Yusuf S., Joseph P., Rangarajan S., Islam S., Mente A., Hystad P., Brauer M., Kutty V. R., Gupta R. et al. Modifiable risk factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study. Lancet. 2020. Vol. 395(10226). P. 795-808. doi: https://doi.org/10.1016/S0140-6736(19)32008-2
3. Liu S., Li Y., Zeng X., Wang H., Yin P., Wang L., Liu Y., Liu J., Qi J. et al. Burden of Cardiovascular Diseases in China, 1990-2016: Findings From the 2016 Global Burden of Disease Study. JAMA Cardiol. 2019. Vol. 4(4). P. 342-352. doi: https://doi.org/10.1001/jamacardio.2019.0295
4. Mensah G. A, Roth G. A., Fuster V. The Global Burden of Cardiovascular Diseases and Risk Factors: 2020 and Beyond. J Am Coll Cardiol. 2019. Vol. 74(20). P. 2529-2532. doi: https://doi.org/10.1016/j.jacc.2019.10.009
7. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020. Vol. 396(10258). P. 1204-1222. doi: https://doi.org/10.1016/S0140-6736(20)30925-9
8. GBD 2017 DALYs and HALE Collaborators. Global, regional, and national disability-adjusted life-years (DALYs) for 359 diseases and injuries and healthy life expectancy (HALE) for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2018. Vol. 392(10159). P. 1859-1922. doi: https://doi.org/10.1016/S0140-6736(18)32335-3
9. Roth G. A., Mensah G. A., Johnson C. O., Addolorato G., Ammirati E., Baddour L. M., Barengo N. C., Beaton A. Z., Benjamin E. J. et al. Global Burden of Cardiovascular Diseases Writing Group. Global Burden of Cardiovascular Diseases and Risk Factors, 1990-2019: Update From the GBD 2019 Study. J Am Coll Cardiol. 2020. Vol. 76(25). P. 2982-3021. doi: https://doi.org/10.1016/j.jacc.2020.11.010
10. GBD 2016 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2017. Vol. 390(10100). P. 1345-1422. doi: https://doi.org/10.1016/S0140-6736(17)32366-8
11. Global BMI Mortality Collaboration, Di Angelantonio E., Bhupathiraju Sh. N. et al. Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents. Lancet. 2016. Vol. 388(10046). P. 776-86. doi: https://doi.org/10.1016/S0140-6736(16)30175-1
12. Global Burden of Metabolic Risk Factors for Chronic Diseases Collaboration (BMI Mediated Effects), Lu Y., Hajifathalian K., Ezzati M., Woodward M., Rimm E. B., Danaei G. Metabolic mediators of the effects of body-mass index, overweight, and obesity on coronary heart disease and stroke: a pooled analysis of 97 prospective cohorts with 1·8 million participants. Lancet. 2014. Vol. 383(9921). P. 970-83. doi: https://doi.org/10.1016/S0140-6736(13)61836-X

13. Kivim ki M., Kuosma E., Ferrie J. E., Luukkonen R., Nyberg S. T., Alfredsson L., Batty G. D., Brunner E. J., Fransson E., et al. Overweight, obesity, and risk of cardiometabolic multimorbidity: pooled analysis of individual-level data for 120 813 adults from 16 cohort studies from the USA and Europe. Lancet Public Health. 2017. Vol. 2(6). P. e277-e285. doi: https://doi.org/10.1016/S2468-2667(17)30074-9
14. Knuuti J., Wijns W., Saraste A., Capodanno D., Barbato E., Funck-Brentano C., Prescott E., Storey R. F., Deaton C. et. al. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. Eur Heart J. 2020. Vol. 41(3). P. 407-477. doi: https://doi.org/10.1093/eurheartj/ehz425
15. Gulati M., Levy P. D., Mukherjee D., Amsterdam E., Bhatt D. L., Birtcher K. K., Blankstein R., Boyd J., Bullock-Palmer R. P. et. al. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021. Vol. 144(22). P. e368-e454. doi: https://doi.org/10.1161/CIR.0000000000001029
16. Hebebrand J., Holm J. C., Woodward E., Baker J. L., Blaak E., Durrer Schutz D., Farpour-Lambert N. J., Fr hbeck G., Halford J. G. C. et. al. A proposal of the European Association for the Study of Obesity to Improve the ICD-11 Diagnostic Criteria for Obesity Based on the Three Dimensions Etiology, Degree of Adiposity and Health Risk. Obes Facts. 2017. Vol. 10(4). P. 284-307. doi: https://doi.org/10.1159/000479208
17. National Institutes of Health, NHLBI Obesity Education Initiative, National Heart, Lung, and Blood Institute, North American Association for the Study of Obesity. The Practical Guide Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. The Practical Guide Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. 2000:94 p. URL: https://www.nhlbi.nih.gov/files/docs/guidelines/prctgd_c.pdf
18. Евстратова И. Н., Муханнад Алшбул. Физическая реабилитация больных ишемической болезнью сердца с нарушением толерантности к глюкозе и абдоминальным ожирением. Науковий часопис Національного педагогічного університету імені М. П. Драгоманова. Серія 15: Науково-педагогічні проблеми фізичної культури (фізична культура і спорт). 2015. Вип. 10. С. 55-58.
19. Маньковский Г. Б. Диагностика нарушений углеводного обмена у больных с ишемической болезнью сердца. Медичні перспективи. Вип. 23(2). С. 46-52. doi: https://doi.org/10.26641/2307-0404.2018.2.133698
20. Лаповець Л. Є., Башта Г. В., Щурко М. М., Ткачук С. О. Особливості змін кліренсу креатиніну при ішемічній хворобі серця, ускладненій ожирінням. Вісник проблем біології і медицини.2019. Вип. 3. С. 143-146. doi: https://doi.org/10.29254/2077-4214-2019-3-152-143-146
21. Кадикова О. І. Диференційований підхід до використання стандартної терапії хронічної серцевої недостатності у хворих на ішемічну хворобу серця й ожиріння. Журнал клінічних та експериментальних медичних досліджень. 2018. Т. 6, № 1. С. 113-120.

REFERENCES

1. Zhou, D., Xi, B., Zhao, M., Wang, L., Veeranki, S. P. (2018). Uncontrolled hypertension increases risk of all-cause and cardiovascular disease mortality in US adults: the NHANES III Linked Mortality Study. Sci Rep., 8(1), 9418. doi: https://doi.org/10.1038/s41598-018-27377-2
2. Yusuf, S., Joseph, P., Rangarajan, S., Islam, S., Mente, A., Hystad, P., Brauer, M., Kutty, V. R., Gupta, R., Wielgosz, A., AlHabib, K. F., Dans, A., Lopez-Jaramillo, P., Avezum, A., Lanas, F., Oguz, A., Kruger, I. M., Diaz, R., Yusoff, K., Mony, P., Chifamba, J., Yeates, K., Kelishadi, R., Yusufali, A., Khatib, R., Rahman, O., Zatonska, K., Iqbal, R., Wei, L., Bo, H., Rosengren, A., Kaur, M., Mohan, V., Lear, S. A., Teo, K. K., Leong, D., O'Donnell, M., McKee, M., Dagenais, G. (2020). Modifiable risk factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study. Lancet, 395(10226), 795-808. doi: https://doi.org/10.1016/S0140-6736(19)32008-2
3. Liu, S., Li, Y., Zeng, X., Wang, H., Yin, P., Wang, L., Liu, Y., Liu, J., Qi, J., Ran, S., Yang, S., Zhou, M. (2019). Burden of Cardiovascular Diseases in China, 1990-2016: Findings From the 2016 Global Burden of Disease Study. JAMA Cardiol, 4(4), 342-352. doi: https://doi.org/10.1001/jamacardio.2019.0295
4. Mensah, G. A, Roth, G. A., Fuster, V. (2019). The Global Burden of Cardiovascular Diseases and Risk Factors: 2020 and Beyond. J Am Coll Cardiol, 74(20), 2529-2532. doi: https://doi.org/10.1016/j.jacc.2019.10.009
7. GBD 2019 Diseases and Injuries Collaborators (2020). Global burden of 369 diseases and injuries in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019.

Lancet, 396(10258), 1204-1222. doi: https://doi.org/10.1016/S0140-6736(20)30925-9
8. GBD 2017 DALYs and HALE Collaborators (2018). Global, regional, and national disability-adjusted life-years (DALYs) for 359 diseases and injuries and healthy life expectancy (HALE) for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet, 392(10159), 1859-1922. doi: https://doi.org/10.1016/S0140-6736(18)32335-3
9. Roth, G. A., Mensah, G. A., Johnson, C. O., Addolorato, G., Ammirati, E., Baddour, L. M., & Fuster, V., GBD-NHLBI-JACC Global Burden of Cardiovascular Diseases Writing Group (2020). Global Burden of Cardiovascular Diseases Writing Group. Global Burden of Cardiovascular Diseases and Risk Factors, 1990-2019: Update From the GBD 2019 Study. J Am Coll Cardiol, 76(25), 2982-3021. doi: https://doi.org/10.1016/j.jacc.2020.11.010
10. GBD 2016 Risk Factors Collaborators (2017). Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet, 390(10100), 1345-1422. doi: https://doi.org/10.1016/S0140-6736(17)32366-8
11. Global BMI Mortality Collaboration, Di Angelantonio E., Bhupathiraju Sh. N. et al. Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents. Lancet. 2016. Vol. 388(10046). P. 776-86. doi: https://doi.org/10.1016/S0140-6736(16)30175-1
12. Global Burden of Metabolic Risk Factors for Chronic Diseases Collaboration (BMI Mediated Effects), Lu, Y., Hajifathalian, K., Ezzati, M., Woodward, M., Rimm, E. B., Danaei, G. (2014). Metabolic mediators of the effects of body-mass index, overweight, and obesity on coronary heart disease and stroke: a pooled analysis of 97 prospective cohorts with 1·8 million participants. Lancet, 383(9921), 970-83. doi: https://doi.org/10.1016/S0140-6736(13)61836-X
13. Kivim ki, M., Kuosma, E., Ferrie, J. E., Luukkonen, R., Nyberg, S. T., Alfredsson, L., & Jokela, M. (2017). Overweight, obesity, and risk of cardiometabolic multimorbidity: pooled analysis of individual-level data for 120 813 adults from 16 cohort studies from the USA and Europe. Lancet Public Health, 2(6), e277-e285. doi: https://doi.org/10.1016/S2468-2667(17)30074-9
14. Knuuti, J., Wijns, W., Saraste, A., Capodanno, D., Barbato, E., Funck-Brentano, C., & Bax, J. J., ESC Scientific Document Group (2020). 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. Eur Heart J, 41(3), 407-477. doi: https://doi.org/10.1093/eurheartj/ehz425
15. Gulati, M., Levy, P. D., Mukherjee, D., Amsterdam, E., Bhatt, D. L., Birtcher, K. K., & Shaw, L. J. (2021). 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation, 144(22), e368-e454. doi: https://doi.org/10.1161/CIR.0000000000001029
16. Hebebrand, J., Holm, J. C., Woodward, E., Baker, J. L., Blaak, E., Durrer-Schutz, D., & Yumuk, V. A. (2017). A proposal of the European Association for the Study of Obesity to Improve the ICD-11 Diagnostic Criteria for Obesity Based on the Three Dimensions Etiology, Degree of Adiposity and Health Risk. Obes Facts, 10(4), 284-307. doi: https://doi.org/10.1159/000479208
17. National Institutes of Health, NHLBI Obesity Education Initiative, National Heart, Lung, and Blood Institute, North American Association for the Study of Obesity (2000). The Practical Guide Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. The Practical Guide Identification, Evaluation, and Treatment of Overweight and Obesity in Adults, 94 p. URL: https://www.nhlbi.nih.gov/files/docs/guidelines/prctgd_c.pdf
18. Evstratova, Y. N., Mukhannad Alshbul (2015). Fyzycheskaia reabylytatsyia bolnыkh yshemycheskoi bolezniu serdtsa s narushenyem tolerantnosty k hliukoze y abdomynalnым ozhyrenyem [Physical rehabilitation of patients with ischemic heart disease with impaired glucose tolerance and abdominal obesity]. Naukovyi chasopys Natsionalnoho pedahohichnoho universytetu imeni M. P. Drahomanova. Seriia 15: Naukovo-pedahohichni problemy fizychnoi kultury (fizychna kultura i sport) [Scientific journal of the National Pedagogical University named after M. P. Drahomanov. Series 15: Scientific and pedagogical problems of physical culture (physical culture and sports)], 10, 55-58. (In Russian).
19. Mankovskyi, H. B. (2018). Dyahnostyka narushenyi uhlevodnoho obmena u bolnыkh s yshemycheskoi bolezniu serdtsa [Diagnosis of impaired carbohydrate metabolism in patients with ischemic heart disease]. Medychni perspektyvy [Medical perspectives], 23(2), 46-52. doi: https://doi.org/10.26641/2307-0404.2018.2.133698 (In Ukrainian).
20. Lapovets, L. Ye., Bashta, H. V., Shchurko, M. M., Kachuk, S. O. (2019). Osoblyvosti zmin klirensu kreatyninu pry ishemichnii khvorobi sertsia, uskladnenii ozhyrinniam [Peculiarities of changes in creatinine clearance in coronary heart disease complicated by obesity]. Visnyk problem biolohii i medytsyny [Herald of problems of biology and medicine], 3(152), 143-146. doi: https://doi.

org/10.29254/2077-4214-2019-3-152-143-146 (In Ukrainian).
21. Kadykova, O. I. (2018). Dyferentsiiovanyi pidkhid do vykorystannia standartnoi terapii khronichnoi sertsevoi nedostatnosti u khvorykh na ishemichnu khvorobu sertsia y ozhyrinnia [A differentiated approach to the use of standard therapy for chronic heart failure in patients with coronary heart disease and obesity]. J. Clin. Exp. Med. Res, 6(1), 113-120. (In Ukrainian).

Резюме

Вступ. Серцево-судинні захворювання (ССЗ) мають статус епідемії, оскільки мають високі рівні поширеності та є основною причиною інвалідності та смертності як в Україні, так і у світі та спричиняють значне зростання витрат на охорону здоров'я. Серед усіх ССЗ ішемічна хвороба серця (ІХС) займає перше місце. Багато досліджень підтверджують високий відсоток ІХС серед усіх ССЗ, особливо при супутній патології з гіперліпідемією та ожирінням. Тому, при лікуванні ІХС з ожирінням необхідно враховувати ефективність застосовуваних фармакологічних засобів і визначати динаміку фармакологічного втручання.
Мета. Визначити динаміку показників глікемічного профілю при коморбідному перебігу ІХС та ожиріння після проведеного лікування.
Матеріали та методи. Проведено рандомізоване контрольоване одноцентрове проспективне дослідження випадок-контроль, яке базується на аналізі результатів обстеження 130 осіб 25-85 років, яких було розподілено на 3 групи: 70 осіб (основна група) з ІХС на фоні ожиріння, 35 осіб з ізольованою ІХС (група порівняння) та контрольна група (25 практично здорових осіб). Досліджувані групи були рандомізовані за віком і статтю.
Результати. Перед початком лікування було визначено ймовірне переважання добових рівнів глюкози у пацієнтів основної групи над пацієнтами групи порівняння та контролю. За результатами глюкозотолерантного тесту при ІХС з ожирінням встановлено перевищення рівня глюкози натще та після навантаження глюкозою (відповідно 5,64±1,92 та 7,08±2,25 ммоль/л) порівняно з ізольованою ІХС (відповідно 5,15±2,22 (p=0,791) та 6,20±3,15 (p=0,403) ммоль/л) та контрольної групи (відповідно 5,32±0,49 (p=0,685) та 5,42±0,51 (p<0,001) ммоль/л. /л). Після лікування було визначено відновлення рівня глюкози в крові.
Висновки. Встановлено, що характеристика динаміки показників метаболізму глюкози може бути використана як показник ефективності лікування при коморбідності ожиріння та ІХС. Отримані результати свідчать про необхідність урахування особливостей метаболізму глюкози при коморбідності ІХС та ожиріння для забезпечення лікувально-профілактичних заходів.

Ключові слова: **ішемічна хвороба серця, ожиріння, динаміка, глікемічний профіль, коморбідність.**