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ОЦЕНКА УГЛЕВОДНОГО ОБМЕНА У ЖЕНЩИН С ИШЕМИЧЕСКОЙ БОЛЕЗНЬЮ СЕРДЦА В ЗАВИСИМОСТИ ОТ НАЛИЧИЯ МЕТАБОЛИЧЕСКОГО СИНДРОМА

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АННОТАЦИЯ

Сердечно-сосудистые заболевания остаются ведущей причиной смертности среди женщин во всем мире, при этом ишемическая болезнь сердца (ИБС) является одной из наиболее распространённых форм сердечно-сосудистой патологии и вносит значительный вклад в показатели заболеваемости и смертности. Распространённость метаболического синдрома существенно возрастает после наступления менопаузы и ассоциируется с инсулинорезистентностью, ожирением, дислипидемией и артериальной гипертензией. Нарушения углеводного обмена рассматриваются как один из ключевых механизмов, связывающих метаболический синдром и ИБС и способствующих повышению риска неблагоприятных сердечно-сосудистых исходов.В исследование были включены 168 женщин старше 40 лет с подтверждённой ишемической болезнью сердца. В зависимости от наличия метаболического синдрома (МС) пациентки были разделены на две группы: основную группу составили 96 женщин с ИБС и сопутствующим МС, группу сравнения – 72 женщины с ИБС без МС. Для оценки состояния углеводного обмена определяли уровень глюкозы крови натощак, инсулина сыворотки крови, гликированного гемоглобина (HbA1c), а также индекс инсулинорезистентности HOMA-IR.Наличие МС у женщин с ИБС ассоциировалось с выраженными нарушениями углеводного обмена, включая гипергликемию, гиперинсулинемию, повышение индекса HOMA-IR и уровня HbA1c. Полученные результаты свидетельствуют о важной роли инсулинорезистентности в развитии кардиометаболических нарушений и подчёркивают необходимость раннего выявления и коррекции нарушений углеводного обмена у женщин с ИБС.

Ключевые слова: ишемическая болезнь сердца, метаболический синдром, углеводный обмен, инсулинорезистентность, HOMA-IR.

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ASSESSMENT OF CARBOHYDRATE METABOLISM IN WOMEN WITH ISCHEMIC HEART DISEASE ACCORDING TO METABOLIC SYNDROME STATUS

ANNOTATION

Cardiovascular diseases remain the leading cause of death among women worldwide, with ischemic heart disease (IHD) representing one of the most prevalent forms of cardiovascular pathology and a major contributor to morbidity and mortality. The prevalence of metabolic syndrome increases significantly after menopause and is associated with insulin resistance, obesity, dyslipidemia, and hypertension. Disturbances in

carbohydrate metabolism are considered key mechanisms linking metabolic syndrome and IHD, contributing to an increased risk of adverse cardiovascular outcomes. The study included 168 women aged over 40 years with confirmed ischemic heart disease. Depending on the presence of metabolic syndrome (MS), the patients were divided into two groups: the main group comprised 96 women with IHD and concomitant MS, while the comparison group included 72 women with IHD without MS. Carbohydrate metabolism was evaluated by measuring fasting blood glucose, serum insulin, glycated hemoglobin (HbA1c), and the homeostasis model assessment of insulin resistance (HOMA-IR). Metabolic syndrome in women with ischemic heart disease is associated with significant disturbances in carbohydrate metabolism, including hyperglycemia, hyperinsulinemia, elevated HOMA-IR, and increased HbA1c levels. These findings highlight the important role of insulin resistance in the development of cardiometabolic disorders and emphasize the need for early detection and correction of carbohydrate metabolism abnormalities in women with IHD.

Keywords: ischemic heart disease, metabolic syndrome, carbohydrate metabolism, insulin resistance, HOMA-IR

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YURAK ISHEMIK KASALLIGI BO‘LGAN AYOLLARDA METABOLIK SINDROM MAVJUDLIGIGA QARAB UGLEVOD ALMASHINUVINI BAHOLASH

ANNOTATSIYA

Yurak-qon tomir kasalliklari butun dunyoda ayollar o‘limining yetakchi sabablaridan biri bo‘lib qolmoqda. Yurak ishemik kasalligi (YuIK) yurak-qon tomir patologiyalarining eng ko‘p uchraydigan shakllaridan biri bo‘lib, kasallanish va o‘lim ko‘rsatkichlariga sezilarli hissa qo‘shadi. Metabolik sindromning tarqalishi menopauzadan keyin sezilarli darajada ortadi va insulinrezistentlik, semizlik, dislipidemiya hamda arterial gipertenziya bilan bog‘liq. Uglevod almashinuvining buzilishlari metabolik sindrom va YuIKni o‘zaro bog‘lovchi asosiy mexanizmlardan biri sifatida qaraladi hamda noxush yurak-qon tomir hodisalari xavfining oshishiga olib keladi.Tadqiqotga tasdiqlangan yurak ishemik kasalligi bo‘lgan 40 yoshdan katta 168 nafar ayol kiritildi. Metabolik sindrom (MS) mavjudligiga qarab bemorlar ikki guruhga ajratildi: asosiy guruhni YuIK va MS bo‘lgan 96 nafar ayol, taqqoslash guruhini esa YuIK bo‘lgan, ammo MS aniqlanmagan 72 nafar ayol tashkil etdi. Uglevod almashinuvi holatini baholash uchun och qoringa qondagi glyukoza, qon zardobidagi insulin, glikatsiyalangan gemoglobin (HbA1c) miqdori hamda insulinrezistentlikning HOMA-IR indeksi aniqlandi.YuIK bo‘lgan ayollarda MSning mavjudligi uglevod almashinuvining yaqqol buzilishlari, jumladan, giperglikemiya, giperinsulinemiya, HOMA-IR indeksi va HbA1c darajasining oshishi bilan bog‘liq bo‘ldi. Olingan natijalar insulinrezistentlikning kardiometabolik buzilishlar rivojlanishidagi muhim rolini ko‘rsatadi hamda YuIK bo‘lgan ayollarda uglevod almashinuvi buzilishlarini erta aniqlash va korreksiya qilish zarurligini ta’kidlaydi.

Kalit so‘zlar: yurak ishemik kasalligi, metabolik sindrom, uglevod almashinuvi, insulinrezistentlik, HOMA-IR.

Introduction: Cardiovascular diseases remain the leading cause of death among women, accounting for approximately 30% of all fatalities and significantly exceeding the combined mortality from all types of cancer [22]. Ischemic heart disease (IHD) is one of the leading forms of cardiovascular pathology and contributes substantially to morbidity and mortality worldwide [4,5,23]. In recent years, the prevalence of cardiometabolic risk factors among women has increased, highlighting the growing medical and social significance of this problem. Although IHD has long been considered a predominantly male disease, contemporary studies have demonstrated its high prevalence among women and the elevated risk of adverse cardiovascular outcomes, particularly in older age groups [2,11]. In women, clinical manifestations of obstructive IHD occur on average 7–10 years later compared to men. In addition, regardless of age, women are less likely to have pronounced focal stenosis of the coronary arteries [15].

Menopause is associated with an increased risk of coronary heart disease and a worse prognosis in women with pre-existing cardiovascular disease. After menopause, the prevalence of metabolic syndrome increases significantly, reaching values 2-3 times higher than in premenopausal women [15].

The study of the effect of menopause on the risk of cardiovascular disease began as part of the Framingham Heart Study. In a study that included 2,873 women under the age of 55, it was shown that the incidence of cardiovascular disease in women before menopause was lower compared to women after menstrual function stopped [12].

Menopause is accompanied by a complex of hormonal and metabolic changes that have a significant impact on carbohydrate metabolism and cardiovascular risk in women. Decreased ovarian function and decreased estrogen production lead to changes in energy metabolism, redistribution of adipose tissue, and decreased sensitivity of peripheral tissues to insulin [17,19,25]. Consequently, the likelihood of developing insulin resistance, impaired glucose tolerance, and type 2 diabetes mellitus is increased. One of the most significant risk factors for the development of cardiovascular diseases in women is metabolic syndrome (MS), which is a combination of abdominal obesity, arterial hypertension, dyslipidemia and carbohydrate metabolism disorders [2,5,20].

Current research shows that women with MS have a significantly higher cardiovascular risk compared to women without metabolic disorders.

An important pathogenetic link that unites MS and IHD is carbohydrate metabolism disorders. Insulin resistance, hyperinsulinemia and chronic hyperglycemia contribute to the development of endothelial dysfunction, chronic inflammation and atherosclerotic vascular damage [9,14,25].

Hormonal changes in the menopausal period are accompanied by an increase in body fat mass, mainly due to its abdominal and visceral components. Visceral obesity is accompanied by an increased production of pro-inflammatory cytokines, free fatty acids and reactive oxygen species, which contributes to the development of insulin resistance [18].

According to the Framingham study, a 4.5 kg increase in body weight is associated with an increase in systolic blood pressure of about 4.4 mm Hg. in men and by 4.2 mm Hg. in women [26].

The results of numerous studies indicate a direct relationship between weight gain and overall mortality. It has been established that obesity grade I is associated with a 3-fold increase in the risk of developing T2DM, obesity grade II with a 5-fold increase, and obesity grade III with an approximately 10-fold increase compared with individuals with normal body weight [26].

According to the European Prospective Investigation into Cancer and Nutrition (EPIC)-InterAct, early menopause is associated with a

32% increased risk of developing T2DM. In addition, women after oophorectomy were 57% more likely to develop type 2 diabetes compared to women with preserved ovarian function [18].

Type 2 diabetes mellitus is one of the most common complications of metabolic syndrome. The development of T2DM is primarily driven by insulin resistance, which reduces tissue sensitivity to insulin and impairs glucose uptake and utilization, thereby contributing to chronic hyperglycemia. Abdominal obesity further aggravates these metabolic disturbances through increased production of free fatty acids and pro-inflammatory cytokines, which adversely affect insulin signaling pathways [1,4,9].

A decline in the anti-atherogenic properties of HDL cholesterol and an increase in lipoprotein(a) levels during menopause are associated with an elevated cardiovascular risk in women. At the same time, progressive abdominal obesity and the associated disturbances in carbohydrate metabolism contribute to the development of T2DM, which further increases the risk of cardiovascular complications [7,16,28].

Insulin resistance is one of the central pathogenetic mechanisms of metabolic syndrome and is considered an important factor in the development of cardiovascular diseases. This condition is characterized by reduced sensitivity of peripheral tissues to the biological action of insulin, resulting in the need for increased insulin secretion by pancreatic β-cells to maintain normal glucose levels. In the early stages, compensatory hyperinsulinemia helps preserve normoglycemia; however, as metabolic disturbances progress, impaired glucose tolerance develops, followed by T2DM [18,21].

Impaired insulin signaling is accompanied by reduced glucose uptake in skeletal muscle and adipose tissue, increased lipolysis, elevated concentrations of free fatty acids, and activation of pro-inflammatory pathways. These changes contribute to the development of endothelial dysfunction, oxidative stress, and atherosclerotic vascular damage [9,17,26].

Visceral adipose tissue exhibits high metabolic activity and serves as a major source of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α). Chronic low-grade inflammation disrupts insulin signaling pathways and further reduces tissue sensitivity to insulin, thereby contributing to the progression of insulin resistance [9,24,27].

Hyperglycemia is one of the main manifestations of carbohydrate metabolism disorders in metabolic syndrome and reflects a progressive decline in tissue sensitivity to insulin. Excess glucose concentrations promote the formation of advanced glycation end products (AGEs), which damage the vascular wall, enhance inflammatory responses, and accelerate the progression of atherosclerosis [9].

Currently, fasting glucose and HbA1c levels are considered important markers of cardiometabolic risk. Therefore, the timely detection and correction of hyperglycemia are important strategies for preventing the progression of MS and IHD [1,25].

Chronic hyperinsulinemia exerts adverse effects not only on carbohydrate metabolism but also on lipid metabolism. Elevated insulin concentrations are associated with increased hepatic triglyceride synthesis, reduced HDL-C levels, and increased concentrations of atherogenic LDL-C. As a result, dyslipidemia develops, which is one of the major components of MS [4,6,28].

Excess insulin concentrations contribute to the activation of the sympathetic nervous system, sodium and water retention, increased vascular tone, and the development of arterial hypertension [3].

The onset of menopause is associated with atherogenic changes in the lipid profile, including a 10–15% increase in LDL-C and triglyceride levels, as well as a slight decrease in HDL-C levels [15,21,28].

Obesity, caused by excessive accumulation of adipose tissue, is accompanied by adipocyte hypertrophy and the development of chronic low-grade inflammation. Increased secretion of TNF-α and IL-6 contributes to the development of insulin resistance by disrupting insulin signaling pathways. The development of insulin resistance is one of the key mechanisms underlying MS, which includes arterial hypertension, dyslipidemia, and hyperglycemia [8,13,26].

Insulin resistance and atherogenic dyslipidemia contribute to the progression of atherosclerosis, whereas oxidative stress and excessive production of reactive oxygen species exacerbate endothelial dysfunction and vascular wall damage. These processes increase the risk of arterial hypertension, myocardial infarction, and stroke [2,8].

Modern therapy for MS is aimed at correcting the major pathogenetic abnormalities, including insulin resistance, dyslipidemia, arterial hypertension, and obesity. Alongside traditional medications such as metformin, thiazolidinediones, statins, and antihypertensive agents, GLP-1 receptor agonists and SGLT-2 inhibitors have gained increasing importance in recent years due to their pronounced metabolic and cardioprotective effects. A comprehensive treatment approach can improve metabolic parameters, reduce cardiovascular risk, and slow the progression of MS-related complications [9,10].

The purpose of the study: To assess carbohydrate metabolism parameters in women with ischemic heart disease according to metabolic syndrome status.

Materials and methods. A total of 168 women aged over 40 years were examined at the Samarkand Regional Branch of the Republican Specialized Scientific and Practical Medical Center of Cardiology. All patients had a confirmed diagnosis of ischemic heart disease (IHD) and were undergoing inpatient treatment at the time of the study. Depending on the presence of metabolic syndrome (MS), the patients were divided into two groups: the main group consisted of 96 women with IHD and concomitant MS, while the comparison group included 72 women with IHD without metabolic syndrome.

Carbohydrate metabolism was assessed by determining fasting blood glucose, serum insulin, glycated hemoglobin (HbA1c), and the homeostasis model assessment of insulin resistance (HOMA-IR). Fasting plasma glucose was measured using standard biochemical methods, while HbA1c levels were used to evaluate long-term glycemic control. Insulin resistance was assessed using the HOMA-IR index calculated according to the following formula: fasting insulin (μU/mL) × fasting glucose (mmol/L) / 22.5.

Statistical analysis was performed using standard biomedical statistical methods. Quantitative variables were expressed as mean ± standard deviation (M±SD). Differences between groups were evaluated using Student's t-test. A p-value of less than 0.05 was considered statistically significant.

Results. The study results demonstrated that disorders of carbohydrate metabolism were significantly more pronounced in women with IHD and metabolic syndrome (MS) compared with patients without metabolic syndrome.

Table 1.

Comparative analysis of carbohydrate metabolism parameters in women with IHD according to the presence of metabolic syndrome

Parameters	IHD + MS (n=96)	IHD without MS (n=72)	p-value
Fasting blood glucose, mmol/L	6.54 ± 2.94	4.67 ± 0.72	<0.001
Insulin, μU/mL	14.27 ± 3.22	8.66 ± 1.99	<0.001
HOMA-IR index	3.88 ± 1.56	1.82 ± 0.56	<0.001
HbA1c, %	6.3 ± 0.5	5.2 ± 0.4	<0.001
Type 2 diabetes mellitus, n (%)	32 (33.3%)	0 (0.0%)	—

Thus, the fasting blood glucose level in the main group was 6.54±2.94 mmol/L, whereas in the comparison group it was significantly lower, amounting to 4.67±0.72 mmol/L ($p<0.001$).

Serum insulin levels also differed significantly between the study groups. In patients with IHD and metabolic syndrome, the insulin concentration was 14.27±3.22 μU/mL, which was significantly higher than that observed in women without MS (8.66±1.99 μU/mL; $p<0.001$).

Assessment of insulin resistance revealed a significant increase in the HOMA-IR index in the main group. The mean HOMA-IR value was 3.88±1.56 compared with 1.82±0.56 in the comparison group ($p<0.001$), indicating the presence of pronounced insulin resistance in patients with MS.

A similar pattern was observed for glycated hemoglobin (HbA1c). In women with IHD and metabolic syndrome, the HbA1c level was 6.3±0.5%, whereas in patients without metabolic syndrome it was 5.2±0.4% ($p<0.001$).

Additional analysis showed that T2DM was diagnosed in 32 of 96 women with IHD and MS, accounting for 33.3% of the study population. In contrast, no cases of T2DM were identified among patients with IHD without MS. These findings indicate a substantially higher prevalence of carbohydrate metabolism disorders in women with MS.

The mean fasting glucose level in patients with T2DM was 9.41±3.50 mmol/L, which markedly exceeded the reference range and indicated significant impairment of glycemic control. It should be noted that several patients demonstrated particularly high glucose levels, reflecting decompensated carbohydrate metabolism and clinically significant metabolic disturbances.

Table 2.

Distribution of women with ischemic heart disease and metabolic syndrome according to the presence of T2DM and insulin resistance

Parameter	T2DM		Insulin resistance	
	(+)	(–)	HOMA-IR >2,7	HOMA-IR ≤2,7
n=96	32	64	79	17
%	33,3	66,7	82,3	17,7

As shown in Table 2, among 96 women with IHD and metabolic syndrome, T2DM was diagnosed in 32 patients (33.3%), whereas 64 patients (66.7%) did not have T2DM. Insulin resistance (HOMA-IR >2.7) was identified in 79 patients (82.3%), while 17 patients (17.7%) had HOMA-IR ≤2.7. These findings demonstrate a high prevalence of insulin resistance among women with IHD and metabolic syndrome.

Thus, the presence of MS in women with IHD was associated with more pronounced disturbances in carbohydrate metabolism, including elevated fasting glucose, hyperinsulinemia, increased HOMA-IR values, higher HbA1c levels, and a greater prevalence of T2DM. These findings indicate significant impairment of glucose metabolism and highlight the important role of insulin resistance in the development of cardiometabolic disorders in this category of patients.

Conclusions: Women with IHD and concomitant metabolic syndrome exhibited significantly more pronounced disturbances in carbohydrate metabolism compared with patients without metabolic syndrome. The presence of metabolic syndrome was associated with higher fasting blood glucose, insulin, HOMA-IR, and HbA1c levels. These findings indicate the presence of marked insulin resistance and impaired glucose metabolism in women with IHD and metabolic syndrome, highlighting the importance of early detection and correction of carbohydrate metabolism disorders in this high-risk patient population.

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