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НОВЫЙ ДЕНЬ В МЕДИЦИНЕ

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FEATURES OF THE COURSE OF ISCHEMIC DISEASE HEART IN METABOLIC SYNDROME *(Literature review)*

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✓ ***Resume***

Probability of developing coronary heart disease (CHD) in patients with metabolic syndrome (MS) significantly increased. Complications of coronary artery disease develop in the presence of MS earlier than in others. IHD against the background of MS and/or diabetes mellitus (DM) is more malignant, up to painless myocardial infarction. IHD associated with glycemic disorders is more likely to result in to unstable angina, myocardial infarction, various arrhythmias. For this category patients develop congestive heart failure faster, diffuse coronary artery disease arteries, including the distal parts of the coronary bed. The presence of MS and DM is a risk factor during cardiac surgery. The effectiveness of therapy for this complication is known largely depends on the timeliness of its detection, which makes it particularly relevant and the importance of the problem of studying risk factors and early diagnosis of metabolic disorders.

Key words: ischemic heart disease; diabetes; metabolic syndrome.

METABOLIK SINDROMI BO ‘LGANLARDA YURAK ISHEMIK KASALLIGINING O‘ZIGA XOS XUSUSIYATLARI *(Adabiyotlar sharhi)*

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✓ ***Rezyume***

Metabolik sindromi (MS) bo‘lgan bemorlarda yurak ishemik kasalligi rivojlanish ehtimoli sezilarli darajada oshdi. Yurak ishemik kasalligining asoratlari MS bo ‘lganlarda MS bo ‘lmaganlarga qaraganda erta rivojlanadi. MS hamda 2-tip qandli diabet (QD) fonida YuIK ko'proq yomon oqibatlarni keltirib chiqaradi va og'riqsiz miyokard infarkti rivojlanishigacha olib keladi. YuIK glikemik kasalliklar bilan bog'liq bo'lganda ko'proq nostabil stenokardiya, miyokard infarkti, turli aritmiyalar kabi og‘ir oqibatlarga olib keladi. Ushbu toifa bemorlarda qonning dimlanishi natijasida surunkali yurak yetishmovchiligi koronar arteriyalarning diffuz zararlanishlari, jumladan koronar tomirlarning distal qismlari zaralanishlari tez rivojlanadi. Metabolik sindrom va YuIKlari oqibatida yuzaga kelgan asoratlarni samarali davolash ko'p jihatdan uning o'z vaqtida tashxislanishiga bog'liq bo‘lib, bu esa uni xavf omillarini o'rganish va metabolik buzilishlarni erta tashxislash muammosining ahamiyatini ko‘rsatib beradi.

Kalit so'zlar: yurak ishemik kasalligi; qandli diabet; metabolik sindrom.

ХАРАКТЕРИСТИКА ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА У ЛЮДЕЙ С МЕТАБОЛИЧЕСКИМ СИНДРОМОМ *(обзор литературы)*

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✓ ***Резюме***

Вероятность развития ишемической болезни сердца (ИБС) у пациентов с метаболическим синдромом (МС) значительно повышена. Осложнения ИБС развиваются при наличии МС раньше, чем в других случаях. ИБС на фоне МС и/или сахарного диабета (СД) протекает более злокачественно – вплоть до безболевых инфарктов миокарда. ИБС, ассоциированная с расстройством гликемии, чаще приводит к нестабильной стенокардии, инфаркту миокарда, различным нарушениям ритма. У данной категории больных быстрее развивается застойная сердечная недостаточность, диффузное поражение коронарных артерий, включая дистальные участки коронарного русла. Наличие МС и СД является фактором риска при хирургических вмешательствах на сердце. Эффективность терапии данного осложнения в зна-

чительной мере зависит от своевременности его выявления, что придает особую актуальность и значимость проблеме изучения факторов риска и ранней диагностики метаболических нарушений.

Ключевые слова: ишемическая болезнь сердца; сахарный диабет; метаболический синдром.

Relevance

In modern literature, the term "metabolic syndrome" (MS) is widely used. Its prevalence among the general population reaches 15% [1], and among the population aged 40–60 years it is recognized MCs are already observed in 20–25% of cases [2]. The basis of MS is hyperinsulinemia, which in turn leads to the development of dyslipidemia, the formation of arterial hypertension (AH) and obesity [3]. Metabolic syndrome is a combination of risk factors for the development of type 2 diabetes mellitus (DM) and cardiovascular diseases (CVD), which include abdominal-visceral obesity, dyslipoproteinemia, hypertension, insulin resistance with compensatory hyperinsulinemia, as well as hyperuricemia, changes in the homeostasis system pre-thrombotic orientation [4, 5]. The classic variant of MS is a combination of hypertension, abdominal obesity, dyslipidemia, and impaired carbohydrate tolerance. Often, the course of MS is accompanied by “secondary” signs: hyperuricemia, increased blood clotting, and hypofibrinolysis [3–6]. One of the most important arguments for the use of the term "metabolic syndrome" in clinical practice and the expansion of its scope is the prevalence among the adult population. The frequency of occurrence depends on the geographical, ethnic characteristics of the population, gender, age, and the nature of the carbohydrate metabolism disorder [4, 7, 8]. The prevalence of MS in Europe is 25-35% among the adult population, over the age of 60 years, the proportion of people with MS increases to 42-43.5%. Approximately 47 million US citizens have been diagnosed with MS, with a prevalence of 24% in men and 23.4% in women [9]. In Russia, large-scale epidemiological studies on the spread of MS have not been conducted. It is known that the presence of MS in parents increases the risk of its development in children. According to international data, the incidence of MS in childhood is 4–12% of the population and is significantly higher in obese children and adolescents [10].

According to Russian scientists, every third obese child has signs of MS [11]. It is obvious that the increase in the incidence of obesity in children and adolescents, as well as the prevalence of disorders of carbohydrate and fat metabolism, make the study of MS in children and adolescents increasingly important.

According to the criteria of the International Diabetes Association (International diabetes Federation), MS is diagnosed in children over the age of 10 years in the presence of abdominal obesity and two or more of the following criteria: increased triglyceride levels, decreased high-density lipoprotein (HDL), hypertension, increased blood glucose levels [10, 11]. There is an opinion that more important in the development of coronary heart disease (CHD) is not the fact of obesity itself, which is certainly a risk factor, but the lack of sufficient physical activity in most overweight patients [5]. Other authors talk about the need to take into account in studies the status of smoking and the presence of chronic obstructive pulmonary disease in lean patients, which could be powerful factors for poor prognosis [12]. Moreover, weight loss in most diseases is initially a powerful factor in poor prognosis [10]. Back in 1947, J. Vague identified two variants of the distribution of adipose tissue - android (male, " apple-shaped ") and gynoid (female, "pear-shaped") and noted the most common combination of android type of obesity with type 2 diabetes, coronary artery disease, gout [8, 13]. Many studies have

shown an increase in the incidence of CVD in patients with MS. It is important that with an increase in the number of available MS components, the incidence rate increases [1, 2]. Among patients with MS, the prevalence of diseases of the cardiovascular system is twice as high and approximately 5 times more likely to develop them during 4.5 years of follow-up compared with individuals who have not been diagnosed with concomitant MS [3,13-15]. post hoc analysis of the results of large studies on the use of statins (4S, AFCAPS / TEXCAPS and WOSCOPS) revealed a 30% increase in the risk of developing coronary artery disease and a 40-50% increase in its main complications (fatal and non-fatal myocardial infarction, sudden cardiac death and unstable angina pectoris) in for 5 years in people with MS compared with people without MS [6, 16, 17]. According to the State Research Center for Preventive of medical science (GNITsPM), in the Russian Federation, almost 10 million of the able-bodied population suffer from coronary artery disease, more than a third of them have angina pectoris [7]. The frequency of detection of angina pectoris clinic depends on age: the incidence in women increases from 0.1–1% at the age of 45–54 years to 10–15% at 65–74 years of age, the same trend can be traced in men: from 2–5% at 45 years old - 54 years old up to 10-20% at 65 years old - 74 years old. In most European

In some countries, the prevalence of angina pectoris is 20,000–40,000 per 1 million populations [3]. The Framingham study showed that exertional angina is the first symptom of coronary artery disease in men in 40.7% of cases, in women - in 56.5%. Of great importance is the fact that only 40–50% of patients are aware of their disease and receive appropriate treatment, while in 50–60% the disease goes unrecognized. Patients with a combination of MS and stable angina pectoris die from coronary artery disease twice as often as those without MS. The data of GNITsPM showed that life expectancy in men suffering from coronary artery disease, on average, is 8 years less compared to those who do not have this pathology. Most of the epidemiological studies conducted are aimed at studying the prevalence of MS in the adult population. Currently, in countries with a high standard of living, signs of MS occur in 10-25% of the population. The presence of MS increases the incidence of sudden death and the development of CVD by 2–4 times, and the risk of developing type 2 diabetes increases by 5–9 times [10, 18]. It has been convincingly shown that hypertension in combination with obesity in the vast majority of cases precedes the development of coronary circulation disorders [2, 10, 17].

According to the Framingham Study, in 70% of men and 60% of women, the development of hypertension is closely related to the presence of obesity: for every 4.5 kg of body weight, systolic blood pressure (BP) increases by 4.5 mm Hg. Art. [9]. An annual increase in the number of patients with coronary heart disease and MS is recorded, the presence of which indicates deep metabolically interrelated atherothrombotic disorders in the body [3, 14, 15]. The CHD clinic is caused by a critical narrowing of the lumen of the coronary arteries due to the progression of atherosclerosis. The nature of the course of angina largely depends on the severity and nature of the progression of coronary artery damage, underlying coronary insufficiency. Although the nature of the course of angina pectoris in some cases remains unchanged for many years, in others it has a clear increase in symptoms. Regardless of the nature of the course of IHD, angina pectoris of any functional class, including for the first time emerged, can suddenly acquire a rapidly progressive course and lead to myocardial infarction. It is known that angina pectoris occurs when there is a lack of supply by the coronary arteries of the corresponding zones of the myocardium with an adequate volume of oxygen-enriched blood and the need for the myocardiumin oxygen. Anginal attack in angina pectoris is a direct manifestation of myocardial ischemia, as a result of which there is an accumulation of underoxidized metabolites in the heart muscle [4]. As myocardial ischemia progresses, the blood pH in the coronary sinus decreases, intracellular potassium is lost, and instead of lactate utilization, its increased production begins. In most cases, this is reflected on the ECG in the form of pathological changes, the mechanical performance of the ventricles is disturbed. The main factors that determine myocardial oxygen demand are heart rate (HR), myocardial contractility, and systolic tension or systolic blood pressure. With an increase in any of these indicators against the background of reduced coronary blood flow, an angina attack develops. Thus, almost any physical stress in patients with hemodynamically significant stenosis of the native arteries causes an angina attack, which, as a rule, stops at rest. If an attack occurs at rest, the subjective sensation of pain is usually accompanied by an increase in heart rate and an increase in blood pressure, in most cases significant. If the attack is not stopped, these shifts form the basis for the inclusion of a potentially fatal biofeedback: the higher the blood pressure and heart rate, the greater the discrepancy between myocardial oxygen demand and the degree of its provision. In the presence of MS, it triples the probability of death from diseases of the cardiovascular system [19]. It is known that patients suffering

from DM have a more pronounced atherosclerotic change in the coronary bed with a predominance of multivessel lesions [1, 6, 20]. In the work of F. Mouquet et al. it was shown that in patients with occlusion of the coronary arteries in people with MS, compared with people without MS and DM, the degree of development of the collateral bed is less (p = 0.005) [17]. Insulin, the so-called atherogenic hormone, accelerates the formation of atherosclerotic plaques at the tissue level [15]. A number of studies show that CAD in patients with MS is more malignant, leading to the development of myocardial infarction and chronic heart failure [16, 18]. The features of the course of atherosclerosis in IHD and MS include more frequent development of various complications, in particular rhythm disturbances. Rhythm disturbances are also more common in the combination of coronary artery disease and MS [1, 3, 17, 21].

Currently, more and more scientific research confirms give that one of the main mechanisms leading to the development of cardiovascular complications in patients with MS is the specific hormonal activity of visceral adipose tissue, whose adipocytes are normally located not only in the abdominal region, but also around the heart, kidneys, liver, blood vessels, and there is its excessive development in this category of patients [15]. Until a consensus is reached on as the primary cause of metabolic disorders in the pathogenesis of MS. According to G. Reaven's hypothesis, MS is caused by an unfavorable combination of genetic factors and lifestyle. The trigger mechanism in the chain of metabolic disorders is insulin resistance, which implies a violation of insulin-mediated glucose utilization in the liver, skeletal muscles and adipose tissue [22]. Thus, depending on the nature of insulin action in three important body systems, a pathological basis for the development of clinical manifestations of MS is formed [6, 16]. Insulin resistance is a decrease in the sensitivity of target tissues to the action of insulin at its sufficient concentration [13].

There are both genetic factors associated with “breakdowns” at the receptor and post-receptor levels, as well as external ones. Among the exogenous factors that stimulate the appearance and progression of insulin resistance, hypodynamia, excessive, excessively high-calorie nutrition, increased activity of the sympathetic nervous system (frequent stress), smoking, but primarily visceral obesity is considered [13, 23]. Visceral adipose tissue is characterized by a number of features. It is highly vascularized, well innervated, and communicates directly with the portal vein. Abdominal adipocytes have a high density of 3-adrenergic receptors that regulate the intensity of lipolysis, corticosteroid and androgen receptors and at the same time a low content of insulin and 2-adrenergic receptors. Such structural-receptor features of visceral adipose tissue determine its high sensitivity to the lipolytic effect of catecholamines and tolerance to the anti-lipolytic effect of insulin [9, 19]. When lipolysis is activated, a large amount of free fatty acids (FFA) is formed in the intra-abdominal adipose tissue, which enter the liver through the portal vein and then into the systemic circulation [12, 20]. FFAs in the liver activate gluconeogenesis, increasing glucose production, and FFAs that enter the systemic circulation reduce the sensitivity of the receptor apparatus of target cells to the action of insulin, which disrupts the transport of glucose into cells and leads to the development of hyperglycemia. At the same time, high concentrations of FFAs reduce the excretion of insulin by the liver from the bloodstream, which ultimately leads to the appearance of systemic hyperinsulinemia [13]. As insulin resistance develops, hyperinsulinemia increases to the point where the pancreas is unable to increase insulin secretion. After decompensation of B -cell function pancreatic secretion of insulin begins to lag behind the severity tissue insulin resistance, leading to to impaired glucose tolerance, the ending of which is the development of SD type 2 [15]. It is known that adipose tissue is source of energy in the body, and performs endocrine functions. Adipocytes secrete more than 50 biologically active substances, which in turn affect the activity of metabolic processes in many organs directly or indirectly through neuroendocrine mechanisms through pituitary hormones, insulin, catecholamines [1, 20, 24]. Adipokines have a direct effect on metabolism lipids, glucose homeostasis, processes inflammation, coagulation, immunity, angiogenesis, bone formation, tumor growth [7].

Free fatty acids are also a substrate for synthesis by the liver. very low lipoprotein (VLDL) and low density (LDL), rich triglycerides (TG), which causes development of hyperlipidemia with increased the content of TG in the blood plasma [23]. The amount of high-density lipoprotein (HDL) decreases, because for their formation requires phospholipids, which are released from VLDL and LDL during their lipolysis, although their elimination is reduced under conditions of insulin resistance [16]. As a result, the developing dyslipoproteinemia has an atherogenic character, since HDL deficiency reduces the body's ability to eliminate excess cholesterol from cells through the reverse transport of cholesterol

to the liver [15]. Based on the time scale, evolution, a significant part of modern society in a record short period has switched to a high-calorie diet with lack of natural earlier periods starvation and significant expenditure of muscle energy. As a result, our contemporary, first of all, a man, who usually has less developed adipose tissue than a woman, pays IHD and myocardial infarction for overeating combined with incomplete use their muscle strength [7, 23].

Studying the features of the course of MS is of tremendous clinical importance, since this condition is reversible, that is, with appropriate treatment, it is possible to achieve complete leveling or at least a decrease the severity of its main manifestations. After all, MS is a precursor of such pathologies as type 2 diabetes and atherosclerosis, which in turn directly increases mortality in the population [23]. In the PubMed search engine in 2014 was more than 6000 publications were identified, in the title of which the term "metabolic" was mentioned syndrome", however, the mechanisms of MS are not fully understood. Known data on the increase in the level C-reactive protein (CRP) in MS and its decrease with decreasing mass index bodies [5]. The results accumulated in this fields of knowledge show that the concentration of insulin in blood plasma directly affects all components of an atherosclerotic plaque: the lipid core, collagen, foamy macrophages, and proliferating smooth muscle cells. Against the backdrop of hyperinsulinemia, stimulation of cell proliferation, increased synthesis of endogenous cholesterol and triglycerides due to the effect of insulin on lipogenic enzymes: glucose-6-phosphate dehydrogenase and 3-hydroxyacetyl-CoA dehydrogenase [23]. Also insulin enhances the synthesis of collagen, insulin-like growth factor-1, causes hyperfibrinogenemia and an increase in the activity of an inhibitor of tissue plasminogen activator type 1 [2].

Conclusion

Thus, following evolutionary genetic patterns, the debut of successive disorders in MS is the development of chronic hyperinsulinemia in response to systematic overnutrition. Next comes the overflow of fat tissues with lipids, which in turn leads to a protective reaction of the cell - a decrease in the number of insulin receptors, the development of dyslipidemia and hyperglycemia, and finally - the deposition of lipids in the vascular wall.

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