BODY WEIGHT AN OBESITY PREVENTION IN DIFFERENT CATEGORIES OF HYPERGLYCEMIA

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Solutions to the problems of improving the population's health do not lose their relevance. The emphasis made on the prevention and treatment of obesity, cardiovascular diseases, on the reduction of cardiac mortality contributed to the development of differentiated preventive measures. The concept of "Metabolic Syndrome" has combined into a single complex interdependent metabolic disorders that form the basis of the pathogenesis of various diseases (atherosclerosis, hypertension, ischemic heart disease, obesity, type 2 diabetes mellitus, etc.). The possibilities of preventing and treating each of the diseases that make up the metabolic syndrome are determined by the success of the prevention and treatment of the metabolic syndrome itself.

ГИПЕРГЛИКЕМИЯНИНГ ТУРЛИ ДАРАЖАЛАРИДА СЕМИЗЛИК ПРОФИЛАКТИКАСИ ВА ТАНА ВАЗНИНИНГ ҲОЛАТИ

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Аҳоли саломатлигини яхшилаш ва муаммоларини ҳал этиш ҳозирга кунда ўз долзарблигини сақлаб келмоқда. Семизлик, юрак қон-томир касалликларини олдини олиш ва даволашга оид дифференциаллашган профилактик чораларини ишлаб чиқиш амалга оширилмоқда. Метаболик синдром концепсияси турли хил касалликлар (атеросклероз, гипертония, юрак ишемик касаллиги, семириш, диабетнинг иккинчи тури ва бошқалар) Патогенезининг асосини ташкил этадиган ягона мураккаб ўзаро боғлиқ метаболик касалликларга бирлашди. Метаболик синдромни ташкил этувчи

касалликларнинг ҳар бирини олдини олиш ва даволаш имкониятлари метаболик синдромнинг олдини олиш ва даволашнинг мувафаққияли асоси ҳисобланади.

СОСТОЯНИЕ МАССЫ ТЕЛА И ПРОФИЛАКТИКА ОЖИРЕНИЯ ПРИ РАЗЛИЧНЫХ КАТЕГОРИЯХ ГИПЕРГЛИКЕМИИ

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Решения задач оздоровления населения не теряют своей актуальности. Сделанные акценты на предупреждение и лечение ожирения, сердечно-сосудистых заболеваний, на снижение сердечной смертности способствовали разработке дифференцированных профилактических мероприятий. Концепция «Метаболический синдром» объединила в единый комплекс взаимозависимые нарушения метаболизма, составляющие основу патогенеза разных заболеваний (атеросклероз, АГ, ИБС, ожирение, сахарный диабет 2 типа и др.). Возможности предупреждения и лечения каждого из заболеваний, составляющих метаболический синдром, определяются успешностью предупреждения и лечения самого метаболического синдрома.

Obesity remains one of the most common diseases in the population of both economically developed and developing countries. Up to 15-32% of the inhabitants of these states are overweight. Despite the use of new approaches in prevention and treatment, the number of obese people is not only not decreasing, but, on the contrary, is progressively increasing, especially among women. The leaders in this regard are the United Arab Republic, Germany, and the USA. Until now, the real results of the ongoing treatment of obesity remain unsatisfactory. WHO considers Obesity as a non-communicable epidemic affecting millions of people on all continents [3, 2, 11, 20, 21]. Obesity the importance of the problem is determined by the threat of disability among patients of a relatively young age and a decrease in life expectancy. The occurrence of such diseases as atherosclerosis, type 2 diabetes mellitus (DM-2), osteoarthritis, gout, cholelithiasis, reproductive dysfunction, varicose veins of the lower extremities, hemorrhoids is associated with obesity. The likelihood of occurrence of both cardiovascular and other diseases increases with increasing body weight and age [1, 2, 3, 6]. Obesity is understood as a heterogeneous disease in which excess fat

deposition in the body can be either an independent polyetiological disease or a symptom of various other pathological conditions. Among the factors contributing to the occurrence of dermatitis, a special role is assigned to a hereditary predisposition. In families where both parents are obese, children are most likely to be obese (up to 82%). In the presence of obesity only in the father or only in the mother, the probability of obesity decreases (up to 39% and 52%, respectively). At the same time, unfavorable environmental factors (unhealthy lifestyle) also play a role in the realization of genetic predisposition [24]. In addition to genetic factors, other independent risk factors should be identified that determine the development of obesity: demographic (age, gender, ethnicity), socio-economic (education, profession), mental and behavioral (dietary habits, physical activity, alcohol, smoking, stress). It can be considered that obesity is the result of a long-term imbalance in the energy balance, when excess energy intake from food (overeating, excessive consumption of fatty foods) exceeds energy expenditure (low physical activity), usually in persons with a hereditary predisposition. This simplified scheme assumes the participation in this process of a number of still insufficiently studied intermediate mechanisms (for example, the influence of intestinal microflora, etc.) [9, 10, 11, 25]. In the pathogenesis of obesity, a special place is occupied by the adipose tissue itself, which not only plays the role of an energy reserve, but also has endo-, auto- and paracrine functions. Adipose tissue produces biologically active substances that have a variety of effects on the activity of metabolic processes in tissues and various systems, either directly or indirectly through the neuro-endocrine system, interacting with pituitary hormones, catecholamines, and insulin. These active producers of adipose tissue include: leptin, interleukin-1.6 (IL-1.6), tumor necrosis factor-α (TNF), tissue plasminogen activator-1 inhibitor (ITAP-1), angiotensinogen, which stimulates protein acetylation, regulators of lipoprotein metabolism - lipoprotein lipase (LPL), hormone-sensitive lipase (HPL), cholesterol ester transfer protein. In turn, the function of adipose tissue is influenced by catecholamines, corticosteroids, insulin. Adipose tissue is directly related to the regulation of systemic inflammation by the release of proinflammatory cytokines (IL-1,6, TNF) [8, 9, 12, 24]. In the regulation of energy metabolism, the system of the central nervous system, hypothalamus-adipose tissue, is of great importance. Leptin is an active factor in this system. It is assumed that the regulation of leptin activity ensures the conservation of energy reserves and, in conditions of prolonged excess food intake, contributes to an increase in the mass of adipose tissue. obesity is characterized by increased production of leptin [1, 3, 8, 9, 12]. It was found that the risk of developing diseases associated with obesity depends not only on the degree of excess body

weight, the age of the patient, the duration of the disease, but also on the nature of the distribution of fat. It turned out that complications are much more often detected in patients with predominant accumulation of fat in the abdominal cavity (abdominal, or visceral, or android, or upper obesity, or obesity of the "apple" type) than in patients with predominant fat deposition in the subcutaneous tissue on the buttocks and hips and in patients with general (mixed) obesity [2, 3].

The use of computed tomography or magnetic resonance imaging, which more accurately reflects the topography of fat distribution, made it possible to find that with AO, a large accumulation of visceral adipose tissue can be both with overweight (with obesity) and with normal body weight (without signs of general obesity). It has been shown that all AO variants have the highest risk of complications in comparison with gluteofemoral obesity. AO is accompanied by insulin resistance and hyperinsulinemia , which, in turn, are predictors of DM-2. Moreover, it was found that excessive accumulation of visceral adipose tissue is combined with an atherogenic lipoprotein profile of blood plasma - hypertriglyceridemia, an increase in the level of low-density lipoprotein cholesterol, and a decrease in high-density lipoprotein cholesterol (HDL C). At the same time, hypercoagulable tendencies in the blood coagulation system are also revealed, creating preconditions for thrombus formation, combined with subclinical chronic inflammation [7, 11, 17, 23]. The biological mechanisms of the formation of various types of fat distribution are not fully understood. The development of AO is associated with a change in the metabolism of glucocorticosteroids in adipose tissue. Under conditions of IR and GI, the conversion of cortisone into cortisol increases, the activity of which is stimulated by insulin. The hormone has a local effect on adipose tissue, activating the differentiation of stromal cells into adipocytes, intracellular accumulation of lipids, as well as redistribution of adipose tissue with predominant accumulation in the omentum and mesentery, which have a high level of blood supply. There is also a connection between the distribution of fat and testosterone content, in particular, for patients with AO, an increase in the content of free testosterone and a decrease in the content of globulin binding the sex hormone are characteristic [18, 24, 25]. Interest in the study of the pathogenetic significance of AO has become noticeably heightened in connection with the development of the concept of metabolic "syndrome X" (MS), proposed by Reaven G. MS combined a complex of interdependent disorders of carbohydrate and fat metabolism, as well as mechanisms of blood pressure regulation, endothelial function. AO is one of the main components of MS along with IR, GI, impaired glucose tolerance (IGT), arterial hypertension (AH), atherogenic dyslipidemia. The clinical significance of MS is determined by a combination of risk factors that create preconditions for

the aggressive development of atherosclerosis, its complications, diabetes mellitus-2 and a number of other pathological processes dependent on dysmetabolism. The essence of AO should now be considered within the framework of the MS. Both genetic and external factors, including the peculiarities of the patients' lifestyle, play a role in the development of MS, as well as AO [1, 3, 4, 21, 24, 25]. If we consider obesity as a possible component of MS, then the general principles of its differentiation should be taken into account. There are different options for classifying obesity. They reflect the increase in body weight, the type of obesity (primary, secondary), the severity of obesity (three or four degrees), the stage of obesity (stable, progressive). To determine the degree of excess body weight, there are various methods for calculating the "theoretical" or "ideal" body weight. In recent studies, classifications of obesity by etiology, by the type of adipose tissue deposition, and by body mass index are more often used [6, 8].

Classification of obesity according to the etiological principle:

✓ alimentary-constitutional (i.e. obesity - as an independent disease);

✓ symptomatic (- endocrine, - hypothalamic, - iatrogenic); by type of adipose tissue deposition:

✓ abdominal, - gluteal-femoral, - general (mixed);

✓ body mass index: Quetelet's body mass index (BMI) (the ratio of body mass in kg to the square of height in meters) - objectifies the degree of accumulation of adipose tissue. As a criterion for "ideal" body weight, BMI indicators were taken from 18.5 to 24.9, overweight - from 25.0 to 29.9, obesity - from 30.0 and above. The higher the index, the higher the risk of concomitant diseases. Another criterion for high risk is abdominal obesity (AO). To determine AO in everyday medical practice, two indicators are used: a) the ratio of the waist circumference at the level of the navel to the circumference of the thighs at the level of the gluteal fold in (with AO OT / OB> 0.9 in men and> 0.85 in women) or waist circumference in cm (with AO OT> 102 cm in men and> 88 cm in women). A more accurate method for assessing AO (topography of adipose tissue distribution) can be considered computed and magnetic resonance imaging [17]. Clinically, the differentiation of abdominal and general obesity seems to be the first step in the diagnosis of MS.

Abdominal obesity and disorders of carbohydrate and lipid metabolism. In a complex complex of metabolic disorders in MS, IR should be attributed to the most significant signs of violation

1. Type of impairment of fat deposition BMI, kg / (m) 2 Risk of concomitant diseases Deficiency of body weight 140/90 mm Hg. Art.

2) general obesity with a BMI> 30 kg / (m) ² or a sign of AO - the ratio of the waist / hip circumference> 0.9 for men and> 0.85 for women.

3) dyslipidemia - an increase in the level of TG in plasma > 1.7 mmol / L and / or low HDL C <0.9 mmol / L for men and <1.0 mmol / L for women.

4) microalbuminuria> 20 μg / min

5) impaired carbohydrate metabolism in the form of impaired glucose tolerance (IGT) or SD-2 [1, 22].

The second principle of diagnostics was developed by experts of the US educational program on cholesterol in the third report in 2001. There is no need to determine IR to detect MS. The presence of three of the five components listed below makes it possible to diagnose MS:

1) waist circumference (WC) as a marker of abdominal obesity> 102 cm in men and> 88 cm in women

2) AH> 130/85 mm Hg. Art

3) a decrease in HDL C levels below 1.04 mmol / l for men and below 1.23 mmol / l for women

4) an increase in the level of triglycerides> 1.69 mmol / l

5) fasting hyperglycemia> 6.1 mmol / L [1, 22].

Initial medical examination in a polyclinic or in a hospital: complaints, lifestyle, diet, hereditary burden, concomitant diseases (accounting for endocrine diseases, SD-2), physical examination, determination of blood pressure, heart rate, OT. Signs of AO (WC> 88 cm in women and> 102 cm in men) in combination with hypertension (> 130/85 mm Hg) are a sufficient reason for a preliminary diagnostic version of MS, especially if the patient has any manifestations of atherosclerosis. It should be borne in mind that general obesity can exist for a long time only as a risk factor for MS. Stage II. Additional examination to clarify the type of obesity or MS: blood plasma lipids (OH, X LDL, X HDL, TG), fasting glucose and 2 hours after loading 75 g of glucose, BMI, ECG, EchoCG. Diagnostic criteria for impaired glucose tolerance are glucose levels <7.0 mmol / L in venous blood plasma and <6.1 mmol / L in capillary blood on an empty stomach and> 7.8 - <6.1 mmol / L in venous blood plasma and <5.6 mmol / L in capillary blood; 2 hours after a load of 30 kg / (m) ², moderate dyslipidemia, the presence of AO, NTG or CD-2, ischemic heart disease, myocardial infarction, stroke. Variants of clinical manifestations of MS. Despite the clinical diversity inherent in MS, a number of researchers identify its most common variants [1, 21]. The evaluation criteria and the number of such options are not the same for different authors. At the same time, it is worthwhile to distinguish three clinical variants of MS based on the absence or presence of dyslipidemia and impaired glucose tolerance (or type 2 diabetes mellitus) [21].

1. Combination of AO with hypertension and dyslipidemia.
2. Combination of AO with AH, dyslipidemia and with NTG (or CD-2).
3. Combination of AO with AG and with NTG (or SD-2).

It should be emphasized that AO and AH in MS are the most common combination. The peculiarities of biochemical changes must be taken into account both in the diagnosis and in the determination of the treatment program. Treatment of MS The main goal of treatment of MS should be considered to reduce the total risk of cardiovascular diseases by correcting its components - a decrease in AO, AH, IR, GI, indicators of impaired fat and carbohydrate metabolism. Since the excessive accumulation of adipose tissue is one of the main pathogenetic factors in the formation of IR and GI, the leading place in the complex treatment of MS should be taken by measures aimed at reducing body weight and abdominal obesity, at correcting nutrition and increasing physical activity. This lifestyle modification is considered the first, the main line of treatment for both MS and general obesity without signs of MS [6, 8, 10, 21, 25,].

Correction of overweight and obesity. The program for reducing body weight and AO includes two sets of measures. 1. Non-drug measures: - patient education, psychological preparation, keeping a food diary, - diet, - changing eating habits, giving up bad habits, - physical training. 2. Medical treatment. Non-drug measures can be combined with drug treatment at any stage of the disease.

When determining a rational low-calorie diet, one must proceed from the following provisions: 1). The diet is made taking into account the age, body weight, gender, level of physical activity, dietary habits of the patient; 2). Limiting the consumption of refined, quickly digestible carbohydrates (sugar, honey, candy, cake). Complex carbohydrates (vegetables, fruits) should make up at least 50% of the diet; 3). Limiting fat intake to 25-30% of the daily calorie intake; four). Introduction to the daily diet of at least 25 g of fiber (wholemeal bread, beans, oats). Fiber promotes the excretion of neutral stearins in the feces and a decrease in plasma cholesterol levels; five). Limiting the daily intake of table salt to 4-5 g. Long-term salt restriction helps to reduce blood pressure in hypertension and regression of LV hypertrophy. It should be borne in mind that with severe hypertension and hyper-TG, alcohol consumption worsens the course of MS. In other cases, it is allowed to take 20-40 g of pure alcohol per day (approximately 50 g of vodka or 150 g of dry wine). Small doses of alcohol increase the level of antiatherogenic HDL C [1, 21].

Correction of carbohydrate metabolism disorders.

To correct IR, metformin (from the biguanide group) is usually used, which has a positive effect on tissue IR by increasing glucose uptake by skeletal

muscles. It has the ability to: a) reduce the rate of absorption of carbohydrates in the small intestine, b) reduce appetite, c) improve intracellular glucose transport.

Metformin is used in patients with MS both without diabetes mellitus-2, and in the detection of DM-2 [12]. In recent years, along with metformin, acarbose has been used, which has shown good efficacy and safety in patients with impaired glucose tolerance [12, 14]. New drugs that improve the sensitivity of cells to insulin include the group of PPAR-γ receptor agonists. Along with a decrease in IR, these drugs contribute to the normalization of blood pressure, have endothelium-protective, anti-inflammatory and antithrombotic effects. The next generation of the drug, an agonist of PPAR-α receptors, activates fatty acid oxidation and helps to reduce hypertriglyceridemia [19]. Clinical trials of these drugs have not yet been completed.

Conclusion. Solutions to the problems of improving the health of the population do not lose their relevance. The emphasis made on the prevention and treatment of obesity, cardiovascular diseases, on the reduction of cardiac mortality contributed to the development of differentiated preventive measures. The related research has noticeably changed the pre-existing ideas about the essence of the diseases mentioned. The concept of "Metabolic Syndrome" has combined into a single complex interdependent metabolic disorders that form the basis of the pathogenesis of various diseases (atherosclerosis, hypertension, ischemic heart disease, obesity, type 2 diabetes mellitus, etc.). It became obvious that the possibilities of preventing and treating each of the diseases that make up the metabolic syndrome are determined by the success of the prevention and treatment of the metabolic syndrome itself. There are a number of problems on the way to improving the prevention and treatment of metabolic syndrome. The first of them is the complexity of its diagnosis in the working conditions of a practical doctor. In recent years, there has been a need for targeted examination of a large number of patients. In these conditions, it becomes especially important to define an accessible diagnostic program (with criteria that are feasible in the primary health care setting), which would allow not only to isolate patients with metabolic syndrome, but also to carry out long-term controlled treatment and prevention. To a certain extent, these requirements are met by the recommendations of experts from the US Cholesterol Education Program (ATP III, 2001). The accumulated experience suggests that the ATP III recommendations make it possible to detect metabolic syndrome by doctors of different specialties without using special complex laboratory tests. Providing early diagnosis and especially subsequent long-term controlled treatment opens up new prospects for health-improving activities and large-scale epidemiological studies.

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